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EDITORIAL

What can we do about our doctor shortage?

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The Aotearoa New Zealand doctor shortage: current context and strategies for retention

Eva G D Hitchon, Kate Eggleston, Roger T Mulder, Richard J Porter, Katie M Douglas

There is a shortage of doctors in Aotearoa New Zealand. Many New Zealand-trained doctors choose to leave for better renumeration, and those who stay are at risk of burnout due to unsustainable working conditions. This article examines the context behind the migration of New Zealand-trained doctors and proposes several strategies for retention as potential solutions to the underlying problem.

Epidemiology of giant cell arteritis in Waikato, Aotearoa New Zealand

Philippa van Dantzig, Vicki Quincey, Jason Kurz, Caroline Ming, Sujatha Kamalaksha, Douglas White

Giant cell arteritis is an inflammatory condition where there is inflammation in the blood vessel wall that can decrease the blood supply to organs in the body. The most concerning implication of this is a reduction of blood supply to the eye, with permanent visual loss. Timely treatment is thus extremely important to prevent this. Understanding how common this disease is in the community is vital when we advocate to the government for Specialist Services and new medications used internationally to treat this condition.

The broader health benefits of optimised dietary thresholds proposed for type 2 diabetes prevention in Aotearoa New Zealand: simulation modelling

Andrew N Reynolds, Christine L Cleghorn, Jim I Mann

We have demonstrated the potential wide-ranging benefits of modifying present dietary intakes in Aotearoa towards healthier intakes, providing evidence to reinforce and extend current dietary guidelines. However, the population dietary changes required for the realisation of these benefits are unlikely to be achieved without the creation of a sustainable food environment that ensures the availability, at affordable cost, of health-promoting foods, and discourages the consumption of dietary promotors of ill health. Successive governments have consistently held back from developing a national food strategy that includes the legislative and policy measures needed to change the current food environment. Investment now in such a strategy would have benefits for the environment and climate, in addition to health and the economy, for this, and subsequent generations.

Performance of a fast-track pathway for giant cell arteritis in Waikato, Aotearoa New Zealand

Philippa van Dantzig, Douglas White, Jason Kurz, Caroline Ming, Sujatha Kamalaksha, Vicki Quincey

Giant cell arteritis is a disease with inflammation in the wall of blood vessels that can reduce blood form to important organs. A feared complication is permanent vision loss. Rapid investigation and treatment are thus very important. A fast-track pathway streamlines the investigation of these patients to ensure that those with the disease get a rapid diagnosis and those without the diagnosis are not exposed to unnecessary steroid treatment while waiting to rule out the disease.

Temporal trends of transport-related injuries on New Zealand roads

Siobhan Isles, Michael Keane, Joanna F Dipnall, Ben Beck

Reducing deaths and serious injury on New Zealand roads continues to be a goal for successive

governments. This work looks at how injury rates have changed over time. People who ride motorcycles are more likely to be injured today than they were 5 years ago, particularly riders aged 10–19 years, and riders aged over 50 years. The injury rates for cyclists and pedestrians have not changed; however, more people are using these modes of transport now. People who are in cars, both drivers and occupants, are less likely to be injured today than previously.

The impact of living with migraine disease in Aotearoa New Zealand

Susan M Garrett, Fiona Imlach

This study reports on a survey of 530 people that explored the impact of living with migraine in Aotearoa New Zealand. Almost half of respondents to the survey had severe migraine-related disability, indicating a significant impact on daily life. Migraine disease impacted on physical health, mental health, ability to work or study at full capacity, ability to engage and commit to social activities and led people to spend much time and money on trying to find a "cause" or "cure", which was often fruitless. Improved support for people with migraine is needed, including more awareness and education about migraine disease, recognition of migraine as a complex, disabling neurological condition, more flexible and accommodating work and education, better support for people unable to work full-time or at all and increased access to effective treatment options.

Can imaging determine if a rotator cuff tear is traumatic?

Jessica Mowbray, Khalid Mohammed

Determining the acuity of a rotator cuff tear is important for patients and their ability to claim for ACC-funded treatment. This review examines a number of signs that can be detected on imaging of the shoulder (including X-rays, ultrasound, magnetic resonance imaging and computed tomography scans) and investigates which of these signs suggest if a rotator cuff tear is an acute traumatic tear or is more indicative of chronic degeneration of the rotator cuff.

Improving community antibiotic prescribing to keep antibiotics working in Aotearoa New Zealand

Mark G Thomas

All antibiotic use gradually makes the bacteria that live on and in us more resistant to antibiotic medicines. That happens whether the antibiotics were prescribed to cure a life-threatening infection or in the forlorn hope that they might speed recovery from a cold, whether the course was finished or not and whether the antibiotics were prescribed by a doctor or passed on by a friend. The more we use antibiotics, the more our bacteria become resistant and much harder to treat. We have high rates of antibiotic use in Aotearoa New Zealand, and relatively high rates of antibiotic resistance. The most effective remedy for this problem is to dramatically reduce our use of antibiotics for those infections in which they provide no benefit: the very common, self-limiting, viral infections, of the nose, throat, ears and sinuses. People in nations with low rates of antibiotic use don't expect antibiotic treatment for these coughs and colds. If we follow the example of these nations we will help to ensure that antibiotics remain effective for future generations of Kiwis.

Bariatric surgery in end-stage kidney disease—removing a barrier to transplantation

Mary J Baker, Michael W C Booth, Jason P Roberston, Janak R de Zoysa

We describe the case of a patient who had bariatric surgery. She has no complications from the surgery, and this allowed her to come off dialysis and have a successful kidney transplant. We discuss personal benefits and cost savings from having bariatric surgery.

Are two shocks better than one? Aotearoa New Zealand emergency medical services implement a new defibrillation strategy: implications for around nine patients per week

Bridget Dicker, Sarah Maessen, Andy Swain, Elena Garcia, Tony Smith

Emergency medical services (EMS) encounter up to nine patients per week in cardiac arrest suffering from refractory ventricular fibrillation (VF) or refractory ventricular tachycardia (VT). These conditions are challenging to treat, with standard methods failing to revive up to 20% of these patients despite repeated defibrillator shocks. A breakthrough treatment called double sequential external defibrillation (DSED) has emerged, where patients receive two rapid succession shocks every two minutes instead of one. A successful trial in Canada showcased improved survival rates, prompting Aotearoa New Zealand to become the first country outside Canada to adopt DSED in January 2024. The introduction of this novel treatment is being closely monitored to assess its impact on patient survival, offering hope for those with refractory VF or VT.

Pre-hospital, pre-antibiotic blood cultures for patients with suspected sepsis—a feasibility study

Aileen Harwood, Scott Pearson, Julia Howard, Nicole Jones, Rosie Greenlees, Charlotte Broms, Sharon J Gardiner, Simon C Dalton

Sepsis (sometimes called blood poisoning or mate whakatāoke) is a life-threatening emergency where antibiotics should be given as quickly as possible. This can make it hard to find the bacteria causing the infection if blood culture tests are taken after the antibiotics. Until now it has been difficult to collect the blood culture tests in the ambulance. We developed an approach to overcome the barriers to this and showed that you could get much higher rates of positive blood cultures if you collect them in the ambulance just before antibiotics are given, compared to waiting until the patient arrives in the emergency department. Knowing what bacteria is causing the infection can help improve the care of patients with sepsis.

Should menstrual cycle data be collected during suspected suicide autopsies?

Angie Hoskin, Sarah K McKenzie, Emily B Cooney, Gabrielle Jenkin

This letter explores gender differences in suicide rates, noting that while men are more likely to die by suicide, women attempt it more frequently. It highlights the scarcity of research on women's suicidal behaviors, particularly regarding menstruation's impact on mental health. The authors discuss how the stigma surrounding menstruation contributes to delays in diagnoses and inadequate treatment. They question the role of autopsies in understanding the relationship between menstrual cycles and suicidal behaviour. By incorporating this information into autopsy procedures, we may uncover valuable insights into the intersection between menstruation and suicide. This could lead to more tailored suicide prevention strategies and improved healthcare practices, particularly in addressing the unique needs of women.

The Aotearoa New Zealand doctor shortage: current context and strategies for retention

Eva G D Hitchon, Kate Eggleston, Roger T Mulder, Richard J Porter, Katie M Douglas

ABSTRACT

The international migration of health professionals has been an ongoing issue with the medical workforce in Aotearoa New Zealand. There are many reasons why New Zealand-trained doctors choose to leave. Often it has been to gain overseas experience, with many eventually returning to New Zealand; however, this has now changed, with increasing numbers not returning. Little has been done to combat this developing problem, amidst an increasingly competitive global market for health professionals. There is public and political concern about the current shortage and uneven distribution of doctors, particularly because this has fostered unsustainable working conditions, which diminishes the provision of safe healthcare in this country. This article examines the context behind the migration of New Zealand-trained doctors and proposes several strategies for retention as potential solutions to the underlying problem.

Many countries are short of healthcare workers, thousands of whom have left healthcare since the COVID-19 pandemic. The migration of doctors is a particular challenge to Aotearoa New Zealand. Although the New Zealand health system is regarded highly among its international counterparts, there is a shortage of doctors and other health workers. In a report last year by Te Whatu Ora – Health New Zealand, New Zealanders learnt, for the first time, the extent of these staffing issues. The estimated shortfall was 1,700 doctors, a gap that is projected to increase to 3,400 by 2032.[1] Many New Zealand doctors are choosing to move overseas to practise. The latest medical workforce data reports that the number of New Zealand-trained doctors practising in Australia alone is 2,187.[2] This article outlines and discusses the reasons which may influence New Zealand-trained doctors to move overseas. An overview of strategies for doctor retention is provided, demonstrating the potential next steps to addressing the issue of migration.

The workforce

Historically, government funding of medical school places in New Zealand has been inconsistent. Prior to this year, the last major increase in student numbers took place after the 2008 election when National promised an additional 200 places across the two medical schools. The intended increase was in anticipation of the growing population demand for health services over the coming decades.[3] Once elected, however, National's plan to boost the medical school intake was reduced, eventually funding 175 places by the end of 2015. Subsequent appeals for an increase were declined until 2023, when the Labour Government approved funding for 50 additional places, increasing the total to 589.

Poor planning for the future medical workforce has resulted in low medical graduation rates. This shortage is especially visible in rural areas of New Zealand and in specialties such as general practice.[4] In addition, the workforce is aging, with many doctors in their mid- to late-career considering opportunities for career breaks and retirement. Overall, there is no longer an adequate number of doctors to care for and meet the increasingly complex medical and socio-cultural demands in New Zealand. The COVID-19 pandemic highlighted this mismatch.

Loss of the workforce

While many young doctors have traditionally left the country to gain overseas experience, increasing numbers of younger and older doctors are leaving because the current working conditions created by these shortages are not sustainable. Excessive workloads, significant on-call duties, long hours and unpaid administrative tasks are some reasons which contribute to burnout.[5] Other factors include feeling undervalued and frustrated with a controlling management culture.[6] High rates of burnout are associated with an intention

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to leave the workforce.[7] A study conducted by the Association of Salaried Medical Specialists (ASMS) in 2016 reported that half of the senior doctors and dentists who responded reported symptoms of burnout.[6] Unsurprisingly, burnout is more common in areas of the workforce that face severe shortages. The recent GP workforce survey report found that nearly 80% of GP respondents had experienced some burnout, with high levels being reported by 48%.[8] Issues linked with mental health and wellbeing among medical students have also been documented. Among medical students studying at the University of Otago, Christchurch Campus last year (2023), "severe" to "extremely severe" levels of stress were reported by 19% of participants.[9] Similar levels of depression and anxiety were reported by 20% and 30% of students, respectively—a concerning finding for individuals at the start of their medical career.

Alongside difficulties with working conditions is the issue of remuneration. In New Zealand and internationally, literature highlights low wages as a driver for doctors leaving the country in which they trained.[4,10] This has been the primary motivation behind strike action across the country, with doctors expressing frustration with Te Whatu Ora – Health New Zealand for their refusal to provide a salary that aligns with inflation. General practitioners are becoming increasingly disheartened by the lack of pay parity with doctors practising in hospital-based disciplines. The result is that doctors feel under-valued. This, along with higher salaries in Australia and other countries, has encouraged doctors to leave.

Most locally trained doctors who are practising overseas do so in Australia.[2] The Australian health system is appealing for a number of reasons: it recognises New Zealand qualifications, it provides doctors the opportunity to work in well-resourced hospitals and there is greater earning potential.[10] The average salary for New Zealand doctors is less than 60% of the Australian average.[11]

What is the fundamental importance of New Zealand-trained doctors?

The importance of locally trained doctors cannot be overstated. Ideally, the New Zealand medical workforce should reflect the socio-cultural demographics of New Zealand society.[12] For decades, however, the New Zealand health system has been dependent on overseas-trained doctors to compensate for the gaps in our workforce. International medical graduates (IMGs) offer a short-term solution for the staffing shortages across the country. Out of OECD countries, New Zealand has the second highest dependency on overseas-trained doctors (second only to Israel), with IMGs making up 42% of the workforce.[13]

Overseas-trained doctors are less likely to remain in the country compared with New Zealand doctors. Currently, around 60% of IMGs leave within 2 years of registration, with significant costs to the New Zealand health system.[2] New Zealand-trained doctors stay longer, suggesting that a locally trained workforce is more sustainable. Other issues with the IMG workforce include a lack of familiarity with the New Zealand healthcare system, potential language barriers, the moral question of employing doctors from developing countries who need them and uncertainty around the provision of culturally appropriate care for Māori.[14]

The latter is particularly important. Medical education in New Zealand places significant emphasis on cultural training for students. Māori experience poorer health outcomes and shorter life expectancies compared with most other population groups in New Zealand.[15] In response to these inequities, both medical schools (Otago, Auckland) now have a core curriculum domain of Hauora Māori, with a focus on community-based teaching and cultural immersion.[16] For example, at Otago, second year medical students experience a week-long Hauora Māori immersion programme, which includes marae-based learning about Māori health models and the Hui process to help guide culturally safe interactions with future patients. Te reo Māori and concepts of tikanga are incorporated into courses. Students at both medical schools also receive teaching in Pacific health. This involves in-context learning directly from Pacific communities about their worldview and health models. Such teaching is vital to ensure that students are equipped with the specific knowledge and skills to improve Indigenous health and reduce inequalities. IMGs are unlikely to have received this culturally relevant training.

There is also an ongoing issue with representation in the workforce. Both medical schools in New Zealand are committed to training more Māori and Pacific doctors through dedicated entry pathways and ongoing support for these students. A similar entry scheme is in place for students from rural backgrounds. The goal is a representative workforce, one that shares cultural values and beliefs with that of the diverse communities of New Zealand and strengthens the belief that the healthcare services are serving them. This is harder to

achieve with a heavy reliance on IMGs. While there are benefits from the skills and experience of an imported workforce, there are also practical and socio-cultural advantages to training New Zealanders and retaining them.

Strategies for retention—what works?

Research into retention has been relatively limited, with most research focussing on reasons for leaving, rather than how to improve retention.[17] Studies that explicitly examine retention strategies for doctors tend to be predominantly concerned with GP or rural workforces. However, the principles behind this research can still be applied.

Better pay: Evidence suggests that pay is linked to retention, with higher income correlating with an intention to stay among medical practitioners.[17] Ideally, doctors should be paid in a way which reflects the ongoing challenges and pressures associated with working in healthcare, such as high workload, frequent on-call duties and long hours. Fair pay, which takes into account these factors, is a key determinant of retention. Remuneration is cited as one of the most important factors that would influence a doctor's decision to continue working in the public health sector.[18] It is important to note, however, the relationship between pay and retention is complex.[17] Financial compensation, alone, may not provide adequate incentive to stay if poor working conditions persist.[19,20]

Other financial incentives, such as loan repayments, bonding for work in hard-to-staff areas and lump sum payments, appear to be less effective, and more closely associated with recruitment rather than long-term retention.[21] This is shown by the limited success of New Zealand's Voluntary Bonding Scheme, which since 2009 offered a restricted number of payments to doctors in exchange for service in hard-to-staff areas. The only review of the scheme, in 2012, reported that more than half of graduates had opted out after just 3 years.[22]

Overseas recruitment: In the short-term, there will be continued reliance on IMGs to supplement our workforce, especially for GPs and rural areas. Maintaining adequate and stable staffing might reduce the current issues associated with burnout, hours worked, on-call commitments and workload.[21] With better working conditions, we could see improved retention across the country. Given the length of medical training in New Zealand, the short-term goal must be recruiting more doctors from overseas. In 2023, 942 IMGs registered to practise in New Zealand, which is insufficient. Amidst a global shortage of healthcare workers, development of strategies to increase recruitment of IMGs include removal of unnecessary barriers to visas and registration, incentives to attract IMGs to hard-to-staff areas and ensuring appropriate pay.[14] Alongside this, we need to invest in ongoing education to ensure overseas doctors are fit to practise within the cultural context of New Zealand.

Local training: In the long-term, increased capacity to train New Zealand doctors is required. There is no shortage of young New Zealanders wanting to train as medical doctors, as evidenced by the highly competitive entry pathways into Auckland and Otago medical schools. What limits the number of graduates we currently produce is the number of places available for these students. Investment in training more doctors by increasing the number of medical school places for New Zealand citizens and residents seems obvious. But simply increasing the number of students overall will not solve the demographic and geographic imbalances in our workforce. Further investment in increasing Māori and Pacific medical workforces should be a priority, as part of our obligation to Te Tiriti and to help ensure culturally safe working conditions. Training students from rural backgrounds is also important, as research has shown that doctors of rural origin are more likely to stay and work in rural areas long-term.[20] Any increase in the number of medical school places would need to be mirrored by a corresponding increase in the number of postgraduate clinical placements and vocational training posts.[14]

Conclusion

In summary, the doctor shortage in New Zealand needs urgent attention. We need to train more, retain more and recruit more to work our way out of this problem. We need to train more doctors, as well as attract doctors from overseas amidst a global medical worker shortage. Existing evidence suggests that issues around retention are a systemic problem with no simple solution. In addition, we need to try and ensure these doctors are appropriately supported and remunerated and have a working environment that is safe and sustainable. It is impossible to achieve this without significant investment into the future of our medical workforce.

COMPETING INTERESTS
Nil.

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REFERENCES
1. Te Whatu Ora – Health New Zealand. Health Workforce Plan 2023/24 [Internet]. Wellington (NZ): Te Whatu Ora – Health New Zealand; 2023 [cited 2024 Feb]. Available from: https://www.tewhatuora.govt.nz/assets/Publications/Health-Workforce-Plan/FINAL-HEALTH-WORKFORCE-PLAN_3-July-2023.pdf
2. Te Kaunihera Rata o Aotearoa – Medical Council of New Zealand. The New Zealand Medical Workforce in 2022 [Internet]. Wellington (NZ): Medical Council of New Zealand; 2022 [cited 2024 Feb]. Available from: https://www.mcnz.org.nz/assets/Publications/Workforce-Survey/64f90670c8/Workforce-Survey-Report-2022.pdf
3. Crampton P. Does New Zealand need a third medical school? N Z Med J. 2017;130(1453):11-16.
4. Yow A, Ozanne A, Audas R. Carousel and conveyor belt: the migration of doctors in New Zealand. Labour Ind. 2015;25(3):1-16. doi: 10.1080/10301763.2015.1081479.
5. Association of Salaried Medical Specialists. Over the edge: Findings of the 2022 survey of the future intentions of senior doctors and dentists [Internet]. Wellington (NZ): ASMS; 2023 [cited 2023 Dec]. Available from: https://asms.org.nz/wp-content/uploads/2023/03/Over-the-Edge-Future-Intentions-of-the-SMO-Workforce-March-2023.pdf
6. Association of Salaried Medical Specialists. "Tired, worn-out and uncertain": Burnout in the New Zealand public hospital senior medical workforce [Internet]. Wellington (NZ): ASMS; 2016 [cited 2023 Dec]. Available from: https://asms.org.nz/wp-content/uploads/2022/05/Tired-worn-out-and-uncertain-burnout-report.pdf
7. Gunja MZ, Gumas ED, Williams II RD, et al. Stressed Out and Burned Out: The Global Primary Care Crisis–Findings from the 2022 International Health Policy Survey of Primary Care Physicians [Internet]. New York, NY (US): The Commonwealth Fund; 2022 [cited 2023 Dec]. Available from: https://www.commonwealthfund.org/publications/issue-briefs/2022/nov/stressed-out-burned-out-2022-international-survey-primary-care-physicians
8. The Royal New Zealand College of General Practitioners. 2022 Workforce Survey [Internet]. Wellington (NZ): The Royal New Zealand College of General Practitioners; 2023 [cited Dec 2023]. Available from: https://www.rnzcgp.org.nz/resources/data-and-statistics/2022-workforce-survey/
9. Douglas KM, Jordan J, Porter RJ, Huria T. Development of a brain training intervention for student mental health. HRC NZ Activation Grant 2022.
10. Lynch AC. So why are New Zealand-trained doctors leaving? N Z Med J. 2008;121(1273):6-8.
11. Groom M, Riley H, Nana G. Comparison of salaries of Medical Specialists, Australia and New Zealand [Internet]. Wellington (NZ): Business and Economic Research Ltd; 2019 [cited Dec 2023]. Available from: https://www.berl.co.nz/sites/default/files/2019-11/BERL%20Report%20Final%20November.pdf
12. Crampton P, Weaver N, Howard A. Holding a mirror to society? The sociodemographic characteristics of the University of Otago's health professional

students. N Z Med J. 2012;125(1361):12-28.
13. OECD. Health at a Glance 2023: OECD Indicators [Internet]. Paris (FR): Organisation for Economic Co-operation and Development; 2023 [cited Dec 2023]. Available from: https://doi.org/10.1787/19991312.
14. Manatū Hauora – Ministry of Health. Fit for purpose and for practice [Internet]. Wellington (NZ): Manatū Hauora – Ministry of Health; 2006 [cited 2023 Dec].
15. Dowell A, Crampton P, Parkin C. The first sunrise: an experience of cultural immersion and community health needs assessment by undergraduate medical students in New Zealand. Med Educ. 2001;35(3):242-9. doi: 10.1046/j.1365-2923.2001.00772.x.
16. McKimm J, Wilkinson T, Poole P, Bagg W. The current state of undergraduate medical education in New Zealand. Med Teach. 2010;32(6):456-60. doi: 10.3109/0142159X.2010.486427.
17. Darbyshire D, Brewster L, Isba R, et al. Retention of doctors in emergency medicine: a scoping review of the academic literature. Emerg Med J. 2021;38(9):663-672. doi: 10.1136/emermed-2020-210450.
18. Association of Salaried Medical Specialists. A Less Public Place: A survey of ASMS members on reasons for working part-time outside of the public health system [Internet]. Wellington (NZ): ASMS; 2023 [cited 2023 Dec]. Available from: https://asms.org.nz/wp-content/uploads/2023/08/A-Less-Public-Place-FINAL-1.0.pdf
19. Marchand C, Peckham S. Addressing the crisis of GP recruitment and retention: a systematic review. Br J Gen Pract. 2017;67(657):227-237. doi: 10.3399/bjgp17X689929.
20. Kumar S, Clancy B. Retention of physicians and surgeons in rural areas-what works? J Public Health (Oxf). 2021;43(4):689-700. doi: 10.1093/pubmed/fdaa031.
21. Buykx P, Humphreys J, Wakerman J, Pashen D. Systematic review of effective retention incentives for health workers in rural and remote areas: towards evidence-based policy. Aust J Rural Health. 2010;18(3):102-9. doi: 10.1111/j.1440-1584.2010.01139.x.
22. Association of Salaried Medical Specialists. ASMS submission on the Voluntary Bonding Scheme 2017 to Health Workforce New Zealand [Internet]. Wellington (NZ): ASMS; 2016 [cited Dec 2023]. Available from: https://asms.org.nz/wp-content/uploads/2022/05/Submission-to-the-Ministry-of-Health-on-Voluntary-Bonding-Scheme.pdf

Epidemiology of giant cell arteritis in Waikato, Aotearoa New Zealand

Philippa van Dantzig, Vicki Quincey, Jason Kurz, Caroline Ming, Sujatha Kamalaksha, Douglas White

ABSTRACT

AIM: Giant cell arteritis (GCA) is the most common primary vasculitis in adults over 50 years of age. Our primary objective was to assess the incidence and prevalence of GCA in Waikato in a bid to deepen our understanding of the epidemiology of GCA in Aotearoa New Zealand.

METHODS: From January 2014 to December 2022, cases of GCA were identified prospectively and retrospectively through temporal artery ultrasound request lists and temporal artery biopsy histology reports. Using electronic health records, data were collected retrospectively on patient demographics and clinical features. These were used to calculate the incidence, prevalence and standardised mortality ratio (SMR) of GCA in Waikato.

RESULTS: There were 214 patients diagnosed with GCA over the 9-year period. The majority of patients were European (93.9%, 201/214) with Māori patients being significantly younger than European patients. The mean annual incidence of clinical GCA was 14.7 per 100,000 people over 50 years (95% confidence interval [CI] 12.7–16.6). The SMR was 1.18 (95% CI 0.83–1.52).

CONCLUSION: This is the largest study to date on the epidemiology of GCA in Aotearoa New Zealand. The incidence of GCA is comparable to other studies performed in Aotearoa New Zealand and appears to be stable over time. GCA is uncommon in Māori, Pacific peoples and Asian ethnic groups.

Giant cell arteritis (GCA) is the most common primary vasculitis in adults over 50 years of age. It is a granulomatous vasculitis and classically involves the aorta and its branches, the distribution of which helps to classify it as either cranial or extra-cranial.[1] Symptoms include temporal headaches, visual loss, scalp sensitivity, jaw claudication and limb claudication. A feared complication of untreated disease is permanent vision loss.

Ethnicity may be a risk factor for GCA, with Scandinavian countries reporting the highest incidence rates.[2] It appears to be much less common in Asian and African American patients.[3–7] Age also appears to be a risk factor, with peaking incidence in the 70–79-year age group with a female predominance.[7]

Investigation of GCA has historically been with temporal artery biopsy (TAB), which has been the gold standard. Colour Doppler ultrasound of the temporal and axillary arteries (CDUS) has been used with increasing frequency over the last decade depending on the availability and expertise in local centres. Ultrasound features of GCA include the halo sign (a hypoechoic ring around the lumen of the temporal or axillary artery) or vessel wall thickening that can lead to stenosis or occlusion of a blood vessel.[8,9] Computed tomography (CT), magnetic resonance imaging (MRI) and F-fluorodeoxyglucose positron emission tomography with computed tomography (FDG-PET) supplement these investigations, particularly in extra-cranial large vessel vasculitis.

The American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) released new classification criteria in 2022.[10] Patients must be 50 years or older with additional clinical criteria of polymyalgic symptoms, vision loss, jaw or tongue claudication, scalp tenderness and abnormal examination of the temporal artery. Investigations with inflammatory markers, TAB, CDUS and FDG-PET are used in the criteria. Patients with six or more points reach a classification for GCA.

The foundation of treatment for GCA is corticosteroids, and while untreated disease is associated with significant morbidity, its treatment is also associated with morbidity for patients.[11] Newer steroid-sparing treatments include tocilizumab, an interleukin-6 inhibitor, or janus kinase (JAK) inhibitors. In resource-limited countries like Aotearoa New Zealand, appreciation of the epidemiology of a condition is crucial when advocating for government support for these evolving therapies, which are currently not routinely available for our patients.

Aotearoa New Zealand is a country of 4.6 million people with 16 regions. As of the 2018

Census, there are 70.2% Europeans (made up mostly of New Zealand Europeans [NZE] with a smaller group of Other Europeans), 16.5% Māori, 15.1% Asian and 8.1% Pacific peoples, along with smaller proportions of other nationalities. Waikato is the fourth largest region in Aotearoa New Zealand, making up 9.5% of the population.[12] Our primary objective was to examine the incidence of GCA in Waikato, which, given its large population, is likely to be reflective of the epidemiology of GCA nationally.

Methods

Beginning in 2013, a new process was developed for handling suspected cases of GCA in the Waikato Region. General practitioners, physicians and those likely to encounter suspected cases were asked to discuss cases with the on-call rheumatologist. Cases were therefore identified prospectively. In addition, a search of ultrasonography lists in the radiology department and a histology search of all temporal artery biopsies performed from January 2014 to December 2022 were screened for patients suspected of having GCA.

The electronic records were reviewed, including clinic letters, primary care referrals, ultrasound request forms, laboratory results, discharge summaries and electronic prescriptions. Data were collected regarding patient demographics, clinical and laboratory features of their GCA, investigations, treatment and mortality. Ethnicity was reported in conjunction with the Statistics New Zealand method of reporting and if patients identified as two ethnicities, both were noted.

Cases of GCA were defined as the final clinical diagnosis recorded by the rheumatologist or ophthalmologist. Where a patient had been evaluated more than once over the time period, only the original evaluation was retained for analysis.

CDUS involved ultrasound of both temporal and axillary arteries. Positive CDUS was classified as the halo sign and was indeterminate if only vessel wall thickening was noted. TAB was classified as positive if there was evidence of active inflammation and the pathology report supported a diagnosis of GCA.

Statistical analysis

Descriptive data are presented as frequencies for categorical variables. Continuous variables are presented as mean with standard deviation (SD). Where symptoms were not reported, this was reported as missing data; thus, percentage calculations are of valid percent rather than total percent. T-tests were used to compare mean age at diagnosis. Chi-squared tests were used to compare deaths between ethnicities. Incidence was calculated by examining the number of new instances of GCA in proportion to the population of the Waikato Region for each year over the duration of the study. Population counts for the Waikato Region were provided by age groups from Statistics New Zealand, a centralised government agency that collects and manages official statistical data for Aotearoa New Zealand. Yearly population projections for the Waikato Region are mostly based around 2018 Census data.[13] After monitoring incoming patients for GCA over a span of 9 years and meticulously tracking occurrences of deaths, prevalence was calculated by determining the total number of individuals with GCA at the end of the observation period and expressing this count as a proportion of the total population under surveillance (i.e., Waikato Region and people over 50 years old). The mean annual mortality rate was calculated from the yearly death count among the changing total population of total known cases of GCA in the Waikato Region. Moreover, a standardised mortality ratio (SMR) was derived comparing the observed deaths among cases of GCA versus what would be expected using the age-specific rates of the surrounding population. Analyses were conducted in SPSS and R software. All significance tests were two-tailed and values of $p<0.05$ were considered significant.

Ethics statement

National ethics approval was granted by HDEC (reference: 2023 EXP 15448) and there was a local assessment through the Waikato Hospital who also approved the project (RD023025), which included review by Te Puna Oranga Māori Consultation Research Review Committee.

Results

Cases of GCA and demographics

From January 2014 to December 2022, a total of 761 evaluations took place for patients with suspected GCA and were referred for either CDUS, TAB or both. Of these, 214 individuals had a final diagnosis of GCA. The majority of the patients with GCA were managed by rheumatology (65.9%), followed by ophthalmology (17.8%), a combination

of both specialities (8.5%) and other specialities (usually general medicine or neurology; 7.9%). Most patients were female (68.2%) with a mean age of 74.0 (SD 8.3) years.

Patients with GCA were most commonly of European background in 93.9% (201/214), Māori 7.9% (17/214) and Asian 0.5% (1/214). The remaining patients (2.8%) included other minorities or patients who did not state an ethnicity. There were no cases of GCA in patients of Pacific peoples descent. Seventeen patients identified as having both European and Māori ethnicity; thus, the total percentage is greater than 100%.

The mean age of European patients with GCA (74.2 [SD 8.3] years) was significantly older than those identifying as Māori (70.1 [SD 8.9] years) (p=0.047).

Table 1 identifies the baseline clinical characteristics of patients with GCA. There was a mean duration of symptoms of 26.4 (SD 29.5) days at the time of referral or initial review.

Analysis of moving averages for annual numbers of cases of GCA showed a statistically significant peak in 2019 and a smaller one in 2021.

Diagnostic tests

In patients diagnosed with GCA, CDUS was performed in 167/214 (78.0%). Of these ultrasounds performed, 17 (10.2%) were positive with a halo sign, 88 (52.7%) were negative and 62 (37.1%) were indeterminate with vessel wall thickening noted.

TAB was performed in 168 (78.5%) of the patients with GCA and 67 (39.9%) had positive histology reported. The ACR/EULAR 2022 classification criteria for GCA were positive in 83.6% of patients.

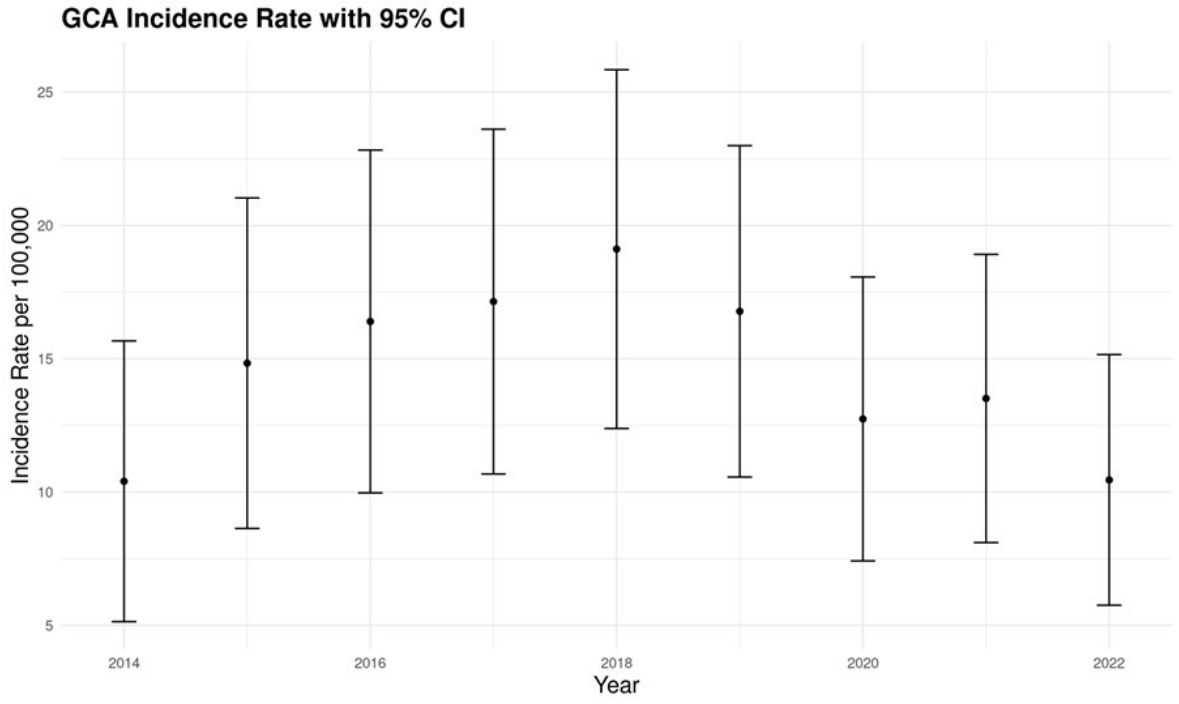
The sensitivity and specificity of CDUS compared to clinical diagnosis was 10.2% (95% confidence interval [CI] 6.0–15.8%) and 99.8% (95% CI 98.9–99.9%) respectively. The sensitivity and specificity of a non-negative CDUS (i.e., either a positive or indeterminate result) was 47.3% (95% CI 39.5–55.2%) and 91.2% (95% CI 88.3–93.5%) respectively.

Table 1: Clinical characteristics of patients with GCA.

Clinical symptoms	No. (valid %)
Headache	183/205 (89.3)
Unilateral headache	86/205 (42.0)
Scalp sensitivity	103/155 (66.5)
Jaw claudication	92/181 (50.8)
Visual symptoms (any)	93/183 (50.8)
Typical symptoms (i.e., AION, CRAO)	24/214 (11.2)*
Diplopia	14/214 (6.5)*
PMR	73/142 (51.4)
Temporal artery tenderness	82/142 (57.7)
ESR mean (SD) mm/hour	46.2 (29.6)
CRP mean (SD) mg/L	68.7 (70.1)
Platelets above normal range	69/208 (33.2)
Haemoglobin below normal range	66/208 (31.7)
ACR/EULAR 2022 classification criteria ≥6	179/214 (83.6%)

*Reported using total population.
AION = anterior ischaemic optic neuropathy; CRAO = central retinal artery occlusion; PMR = polymyalgia rheumatica; ESR = erythrocyte sedimentation rate; CRP = c-reactive protein; platelet ULN >400x10^9/L; haemoglobin LLN<115g/L (women) and <130g/L (men); ACR = American College of Rheumatology; EULAR = European League Against Rheumatism.

Figure 1: GCA annual incidence.



GCA = giant cell arteritis; CI = confidence interval.

For TAB, the sensitivity and specificity compared to clinical diagnosis was 39.9% (95% CI 32.4–47.7%) and 100.0% (95% CI 97.5–100.0%) respectively. For the ACR/EULAR 2022 classification criteria, the sensitivity was 83.6% (95% CI 78.0–88.3) and specificity was 53.1% (95% CI 48.7–57.5%).

Eleven patients with GCA had an FDG-PET scan performed looking for large vessel vasculitis (LVV). There was evidence of LVV in five patients (45.5%) with none (0/5) of these patients having a positive CDUS and one (1/3) having a positive TAB.

Two patients with GCA had neither a CDUS nor a TAB. Their data had been collected prospectively as part of the CDUS referral process. Both had significant referral delays where the treating rheumatologist decided against further tests but the case was clinically consistent with GCA.

Incidence and prevalence of GCA in Waikato

The incidence of GCA was calculated for cases identified with TAB and also for cases identified clinically (i.e., on the experience of the rheumatologist with or without supporting imaging or histological data). The mean annual incidence (MAI) of biopsy positive GCA was 4.6 per 100,000 people over 50 years (95% CI 3.5–5.7). The MAI of clinical GCA was 14.7 per 100,000 people over 50 years (95% CI 12.7–16.6). The annual incidence for clinical GCA is reflected in Figure 1. The confidence intervals are overlapping and thus there does not appear to be any change in the annual incidence rate over the 9-year period. The estimate of the lower bound of prevalence of clinically positive GCA was 94.6 per 100,000 people over 50 years.

COVID-19

Aotearoa New Zealand had an initial lockdown period due to the COVID-19 pandemic in March–May 2020 and a subsequent lockdown in August–September 2021, when there was community transmission. During these periods, the service remained in place where patients and physicians had access to CDUS and TAB. Reviews occurred via telephone consultation unless the patient was unwell, in which case a face-to-face review was organised.

Mortality

There were 47 deaths in the cohort over the study period in patients with GCA. The mean

age at time of death was 81.1 (SD 7.5) years with a range of 64–95 years of age. Females made up 59.6% of deaths. Mean time to death from referral was 29.1 (SD 23.6) months. The mean annual mortality rate was 38.4 per 1,000 cases over 50 years. The SMR was 1.18 (95% CI 0.83–1.52). Most deceased patients were of European descent (42/47, 89.4%) with four deaths in Māori patients (8.5%) and one not stated. The difference between deaths between Europeans and Māori was not statistically significant (p=0.27).

Infection was the most common cause of death, accounting for 16.7% (8/48) of deaths, followed by malignancy in 12.5% (6/48), cerebrovascular disease in 12.5% (6/48) and cardiovascular disease in 10.4% (5/48). Half of these deaths due to infection (4/8) occurred prior to the onset of the COVID-19 pandemic and of the four infection-related deaths in patients occurring in 2020 onwards, none were positive for COVID-19 at the time of their death. In addition to causes of mortality already described, there was one patient who died from an aortic dissection, one patient from venous thromboembolism (VTE) and two from peptic ulcer disease.

Discussion

This is the largest epidemiological study on GCA in Aotearoa New Zealand published to date and certainly the first to document the epidemiology of GCA in Waikato.

We report a MAI for clinically diagnosed GCA of 14.7 per 100,000 people over 50 years and a lower incidence of TAB-positive GCA of 4.6 per 100,000 people over 50 years. This latter finding is likely due to only 78.5% of GCA patients having a TAB performed, and only 39.9% being positive. There are three studies to date looking at the incidence of GCA in Aotearoa New Zealand and they cover Otago,[14] Canterbury[15] and Counties Manukau.[16] Collectively, these report a MAI of biopsy-positive GCA of 10.5–12.73 per 100,000 people over 50 years[14,15] and a MAI of clinically positive GCA of 11.4–15 per 100,000 people over 50 years old.[15,16]

Data on sensitivity and specificity for TAB and CDUS in Aotearoa New Zealand are only available in one of the three studies (Counties Manukau[16]), with a slightly higher sensitivity for TAB of 57% and CDUS 26%. Specificity is similar with 100% for TAB and 97% for CDUS.

This unity between four epidemiology studies in Aotearoa New Zealand supports the notion that cases of GCA are largely stable over the last decade. Certainly, over our study period in Waikato, cases of GCA did not appear to be rising.

World-wide, pooled incidences of GCA are 10.00 (95% CI 9.22–10.78) cases per 100,000 over 50 years, which is lower than our study but likely reflects the combination of high-risk countries (Scandinavian), moderate-risk countries (European, American) with low-risk countries (Asian) in different proportions to what we see in Aotearoa New Zealand.[2] It is likely, given Aotearoa New Zealand is made up predominantly of Europeans, that the epidemiology of GCA in our country will reflect other European countries.

The prevalence of GCA in our study was 94.6 per 100,000 over 50 years. This is also higher than the pooled prevalence of 51.7 per 100,000 people over 50 years from the meta-analysis by Li et al.,[2] yet very similar to the prevalence of 87.9 per 100,000 people over 50 years reported by a European study.[17] It is likely, given Aotearoa New Zealand is made up predominantly of Europeans, that the epidemiology of GCA in our country will reflect other European countries. While the methods of the study were not dedicated to prevalence calculations, we believe that the duration of the study, life expectancy and average age of admittance means it is possible to make an estimate on the lower bound for prevalence.

Our study has similar baseline demographics compared to other New Zealand studies that have noted a female predominance of 65.5–71%[14–16] and mean age of 72.8–74.2 years.[14,15]

Despite Māori and Pacific peoples making up 23.9% and 4.5% of the Waikato population respectively,[12] only 7.0% of our patients with GCA were Māori and none were of Pacific peoples origin. Other Aotearoa New Zealand studies have also demonstrated that GCA is uncommon in Māori and Pacific peoples.[15,16] One factor contributing to this may be the lower life expectancy in these groups[12] and the known risk of age and GCA.[7] Asians make up 9.5% of the population of Waikato, yet we noted very few Asians with GCA, a finding consistent with the very low incidence in this group noted by other studies.[4]

Our study documented lower sensitivities of TAB and CDUS compared to those in Counties Manukau[16] and other world-wide studies.[18–22] TAB is a difficult investigation to acquire in Waikato Hospital due to local referral issues and an under-resourced vascular department, where biopsies commonly occur outside of the optimal window. This may account for the lower positive biopsy rates. The lower sensitivity of CDUS was also seen in the Counties Manukau study and the cause

remains unclear.[16] A separate study analysing the efficacy of a fast-track pathway to investigate GCA in Waikato has noted that early commencement of corticosteroids results in fewer positive CDUS[23] scans, which has been noted in other studies as well.[6] However, early corticosteroids are mandatory to prevent complications of disease. We also suspect that ultrasonographers have under-reported some positive CDUS scans, describing findings as "suggestive of GCA" rather than noting the presence of a halo sign. This has been noted on a separate retrospective review of several CDUS scans used in the data collection; however, the significance remains unclear as static ultrasound appears inferior to real-time ultrasound, though not formally evaluated in GCA.[24]

The ACR/EULAR 2022 classification criteria also performed at lower sensitivity and specificity in our study compared to a recent review of their performance in the United States,[22] which noted a sensitivity of 92.6% and specificity of 71.8%.

The mortality rate was 38.4 per 1,000 people over 50 years old. This compares to a pooled annual mortality rate of 20.4 (95% CI 17.8–23.0) cases per 1,000 people over 50 years.[2] While higher than the pooled mortality rate, it is similar to some of the European studies included in this meta-analysis and thus is probably consistent given our predominantly European population.

Our study noted no increase in the SMR for patients with GCA, which is concordant with literature. Several meta-analyses looking at all-cause mortality in GCA compared to the general population have found that the SMR is not increased with GCA.[25,26] However, on subgroup analysis in these studies, there appears to be an increase in mortality if patients are recruited through a hospital setting.[26] An increase in mortality is also noted during the first 2 years of treatment, which disappears after 10 years.[27,28]

From literature, the leading cause of death in GCA is cardiovascular disease followed by cerebrovascular disease, infection and malignancy.[26] Our study showed causes of death were due to infection, followed by malignancy and cerebrovascular disease, and lastly by cardiovascular disease. This discrepancy may be due to the low numbers of deaths (n=47) and shorter duration of follow-up for patients recruited in the latter half of the study period, which may not reflect causes of death for patients who died later than 2023. Despite the study period crossing the COVID-19 pandemic, death due to COVID-19 was uncommon and unlikely to explain the higher number of infection-related deaths. Over half of deceased patients had a time from referral to death of under 2 years (53.2%), consistent with the known increased mortality risk noted in the first 2 years.[27,28]

We acknowledge that patients being investigated in the private healthcare system were not captured in this study and that the private health community in Aotearoa New Zealand does account for a significant portion of the rheumatology workforce.[29] The access to CDUS in the Waikato private sector is limited and unlikely to occur at short notice except via the Waikato fast-track pathway, which was set up in 2013 to enable rapid access (i.e., same day or next working day) to CDUS for patients. Thus, most patients in the private sector are referred to the public system and would be captured in this dataset. It is unlikely that patients living out of Waikato would be investigated by the Waikato Hospital because access to healthcare was usually strictly limited to District Health Board zones. This may not be the case for private cases of GCA, which may have crossed boundaries. Overall, it is likely that we have recorded most cases of GCA over a 9-year period. Other minor limitations of this study include its retrospective nature along with some missing data due to inadequate documentation of clinical symptoms.

Conclusion

The epidemiology of GCA in Waikato is comparable to other epidemiological studies in Aotearoa New Zealand, reflecting a stability in the incidence of GCA nationally over the past decade. GCA appears to be less common in Māori, Pacific peoples and Asian patient groups and occurs at a younger age in Māori patients compared to European. We noted no increase in mortality, with common causes of mortality being infection followed by malignancy and cerebrovascular disease.

COMPETING INTERESTS
None.
Philippa van Dantzig has been employed part-time by the Waikato Hospital (Te Whatu Ora) for 12 months in a research position to carry out this research project among others, as well as perform a clinical role. There is no other specific funding towards the project.

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REFERENCES
1. Simon S, Ninan J, Hissaria P. Diagnosis and management of giant cell arteritis: Major review. Clin Exp Ophthalmol. 2021;49(2):169-85. doi: 10.1111/ceo.13897.
2. Li KJ, Semenov D, Turk M, Pope J. A meta-analysis of the epidemiology of giant cell arteritis across time and space. Arthritis Res Ther. 2021;23(1):82. doi: 10.1186/s13075-021-02450-w.
3. Smith CA, Fidler WJ, Pinals RS. The epidemiology of giant cell arteritis. Report of a ten-year study in Shelby County, Tennessee. Arthritis Rheum. 1983;26(10):1214-9. doi: 10.1002/art.1780261007.
4. Kobayashi S, Yano T, Matsumoto Y, et al. Clinical and epidemiologic analysis of giant cell (temporal) arteritis from a nationwide survey in 1998 in Japan: the first government-supported nationwide survey. Arthritis Rheum. 2003;49(4):594-8. doi: 10.1002/art.11195.
5. Yu E, Chang J. Giant Cell Arteritis: Updates and Controversies. Front Ophthalmol. 2022;2. https://doi.org/10.3389/fopht.2022.848861.
6. Pinnell J, Tiivas C, Mehta P, Dubey S. Corticosteroids reduce vascular ultrasound sensitivity in fast- track pathways (FTP): results from Coventry Multi-Disciplinary FTP for cranial Giant Cell Arteritis. Scand J Rheumatol. 2023;52(3):283-92. doi: 10.1080/03009742.2022.2051279.
7. Gonzalez-Gay MA, Vazquez-Rodriguez TR, Lopez-Diaz MJ, et al. Epidemiology of giant cell arteritis and polymyalgia rheumatica. Arthritis Rheum. 2009;61(10):1454-61. doi: 10.1002/art.24459.
8. Rinagel M, Chatelus E, Jousse-Joulin S, et al. Diagnostic performance of temporal artery ultrasound for the diagnosis of giant cell arteritis: a systematic review and meta-analysis of the literature. Autoimmun Rev. 2019;18(1):56-61. doi: 10.1016/j.autrev.2018.07.012.
9. Jianu DC, Jianu SN, Dan TF, et al. Ultrasound Technologies and the Diagnosis of Giant Cell Arteritis. Biomedicines. 2021;9(12):1801. doi: 10.3390/biomedicines9121801.
10. Ponte C, Grayson PC, Robson JC, et al. 2022 American College of Rheumatology/EULAR classification criteria for giant cell arteritis. Ann Rheum Dis. 2022;81(12):1647-53. doi: 10.1136/ard-2022-223480.
11. Curtis JR, Westfall AO, Allison J, et al. Population-based assessment of adverse events associated with long-term glucocorticoid use. Arthritis Rheum. 2006;55(3):420-6. doi: 10.1002/art.21984.
12. Statistics New Zealand. 2018 Census place summaries: New Zealand [Internet]. 2018 [cited 2023 Jul 2]. Available from: https://www.stats.govt.nz/tools/2018-census-place-summaries/new-zealand.
13. Statistics New Zealand, NZ.Stat. Subnational population estimates (RC, SA2), by age and sex, at 30 June 1996-2023 (2023 boundaries). [cited 2023 Sep 13]. Available from: https://nzdotstat.stats.govt.nz/wbos/Index.aspx?DataSetCode=TABLECODE7979#.
14. Abdul-Rahman AM, Molteno AC, Bevin TH. The epidemiology of giant cell arteritis in Otago, New Zealand: a 9-year analysis. N Z Med J. 2011;124(1329):44-52.
15. Lyne SA, Ruediger C, Lester S, et al. Giant cell arteritis: A population-based retrospective cohort study exploring incidence and clinical presentation in Canterbury, Aotearoa New Zealand. Front

Med (Lausanne). 2022;9:1057917. doi: 10.3389/fmed.2022.1057917.
16. Nagarajah R, Gupta R, Kumar S. Diagnostic use of ultrasound in giant cell arteritis in Counties Manukau District Health Board, New Zealand. Rheumatol Adv Pract. 2022;6(2):rkac040. doi: 10.1093/rap/rkac040.
17. Catanoso M, Macchioni P, Boiardi L, et al. Incidence, Prevalence, and Survival of Biopsy-Proven Giant Cell Arteritis in Northern Italy During a 26-Year Period. Arthritis Care Res (Hoboken). 2017;69(3):430-8. doi: 10.1002/acr.22942.
18. Sebastian A, Tomelleri A, Kayani A, et al. Probability-based algorithm using ultrasound and additional tests for suspected GCA in a fast-track clinic. RMD Open. 2020;6(3):e001297. doi: 10.1136/rmdopen-2020-001297.
19. Moreel L, Betrains A, Doumen M, et al. Diagnostic yield of combined cranial and large vessel PET/CT, ultrasound and MRI in giant cell arteritis: A systematic review and meta-analysis. Autoimmun Rev. 2023;22(7):103355. doi: 10.1016/j.autrev.2023.103355.
20. Nakajima E, Moon FH, Junior NC, et al. Accuracy of Doppler ultrasound in the diagnosis of giant cell arteritis: a systematic review and meta-analysis. Adv Rheumatol. 2023;63(1):5. doi: 10.1186/s42358-023-00286-3.
21. Rubenstein E, Maldini C, Gonzalez-Chiappe S, et al. Sensitivity of temporal artery biopsy in the diagnosis of giant cell arteritis: a systematic literature review and meta-analysis. Rheumatology (Oxford). 2020;59(5):1011-20. doi: 10.1093/rheumatology/kez385.
22. Molina-Collada J, Castrejón I, Monjo I, et al. Performance of the 2022 ACR/EULAR giant cell arteritis classification criteria for diagnosis in patients with suspected giant cell arteritis in routine clinical care. RMD Open. 2023;9(2):e002970. doi: 10.1136/rmdopen-2022-002970.
23. van Dantzig P, White D, Kurz J, et al. Performance of a fast-track pathway for giant cell arteritis in Waikato, Aotearoa New Zealand. N Z Med J. 2024 Mar 22;137(1592):31-42.
24. Van Holsbeke C, Yazbek J, Holland TK, et al. Real-time ultrasound vs. evaluation of static images in the preoperative assessment of adnexal masses. Ultrasound Obstet Gynecol. 2008;32(6):828-31. doi: 10.1002/uog.6214.
25. Lee YH, Song GG. Overall and cause-specific mortality in giant cell arteritis : A meta-analysis. Z Rheumatol. 2018;77(10):946-51. doi: 10.1007/s00393-018-0440-7.
26. Hill CL, Black RJ, Nossent JC, et al. Risk of mortality in patients with giant cell arteritis: A systematic review and meta-analysis. Semin Arthritis Rheum. 2017;46(4):513-9. doi: 10.1016/j.semarthrit.2016.08.015.
27. Baslund B, Helleberg M, Faurschou M, Obel N. Mortality in patients with giant cell arteritis. Rheumatology (Oxford). 2015;54(1):139-43. doi: 10.1093/rheumatology/keu303.
28. Mohammad AJ, Nilsson JÅ, Jacobsson LT, et al. Incidence and mortality rates of biopsy-proven giant cell arteritis in southern Sweden. Ann Rheum Dis. 2015;74(6):993-7. doi: 10.1136/annrheumdis-2013-204652.
29. Harrison AA, Tugnet N, Taylor WJ. A survey of the New Zealand rheumatology workforce. N Z Med J. 2019;132(1507):70-6.

The broader health benefits of optimised dietary thresholds proposed for type 2 diabetes prevention in Aotearoa New Zealand: simulation modelling

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ABSTRACT

AIM: Optimised dietary thresholds for type 2 diabetes prevention exist; however, they likely have additional benefits beyond diabetes prevention. We have modelled the effects of the proposed dietary thresholds on Health-Adjusted Life Years (HALY), health inequities and health system cost in Aotearoa New Zealand.

METHODS: We created a national diet scenario using the optimised thresholds and compared it with current intakes using an established multistate life table. The primary model considered change in outcome from increasing intakes of fruits, vegetables, nuts and seeds while decreasing red meat and sugar-sweetened beverages. A separate secondary nutrient-based model considered change due to increasing whole grains and yoghurt while decreasing refined grains, potatoes and fruit juice. Both models considered the direct non-weight mediated associations between diet and disease.

RESULTS: In the primary model, adopting the dietary thresholds produced clear benefit to Aotearoa New Zealand in terms of HALY (1.2 million years [95%UI 1.0–1.5]), and a health system cost saving of $17.9 billion (95%UI 13.6–23.2) over the population life course. HALY gain was at least 1.8 times higher for Māori than non-Māori. The secondary model indicated further gains in HALY for all population groups and health systems costs.

CONCLUSION: These striking benefits of altering current dietary intakes provide strong evidence of the need for change. Such change requires government commitment to an overarching food strategy in Aotearoa New Zealand to build supportive food environments that enable healthy choices at affordable prices.

What we eat and the nutrients in food are important determinants of non-communicable disease risk.[1] There has long been convincing evidence on an increased risk of cardiovascular diseases in those with high intakes of dietary sodium and saturated fatty acids, and for a protective effect of dietary fibre, whole grains, vegetables, fruit and n-6 unsaturated fatty acids.[1–3] Dietary fibre and whole grains have also been associated with a reduced risk of type 2 diabetes and colorectal cancer.[4,5] More recently, red and processed meat intakes have been shown to be promotive factors in the development of colorectal cancer,[6] while nut, seed and legume intakes protect against cardiovascular disease.[7,8]

Escalating rates of type 2 diabetes worldwide[9] have prompted an in-depth evaluation of the role of dietary factors in this disease. O'Hearn and colleagues[5] have very recently utilised three global data sets to examine the relative importance of 11 dietary factors in 184 countries by considering both body weight mediated and direct non-weight mediated associations between diet and disease. Grain processing (whole grain intakes were protective, refined grain intakes were associated with increased risk) and red and processed meat intakes were found to be the major contributors to the global diet-related burden of type 2 diabetes incidence.[5] Based on this modelling of the global data, the authors concluded that 70.3% (68.8–71.8%) or 14.1 (13.8–14.4) million new cases of type 2 diabetes in 2018 were attributable to the 11 dietary factors studied.[5] For Aotearoa New Zealand this translated to 7,055 (6,459–7,615) potentially avoidable new diagnoses of type 2 diabetes in 2018.[5] These recent findings are compatible with advice from current guidelines[10] and the results of randomised controlled trials, which indicate that dietary interventions can more than halve the risk of progression from prediabetes to type 2 diabetes.[11]

While these results are striking, they do not acknowledge that a type 2 diabetes-protective diet would also have broader health benefits, likely in terms of cardiovascular health and the mitigation of some cancers. To better understand the true scope of health benefits with dietary change, we have used the same 11 dietary thresholds applied by O'Hearn et al.[5] to consider the broader health consequences of changing dietary intakes in Aotearoa New Zealand beyond type 2 diabetes incidence. Our aim in doing so is to provide local context to these recent global findings,[5] and further discussion on changing our food environment here in Aotearoa New Zealand to support healthier lives.

Method

Simulation model parameters

We used a proportional multistate life table model (DIET MSLT), following established methods.[12,13] This model[14] has been used in previous projects to model interventions that are individually targeted,[15] modify the food environment[16] or are theoretical.[17,18]

Within the model, the 2011 Aotearoa New Zealand population (4.4 million) is divided into 5-year age group cohorts, modelled as four separate sex by ethnic populations, and simulated until death or the year 2121, whichever is the earlier.[14] Non-communicable disease outcomes (cardiovascular diseases, osteoarthritis, type 2 diabetes and colorectal, lung, oesophageal, head and neck, and ovarian cancers) were included in the model as they related to dietary factors.[1] Disease rates used in the multistate life tables of the DIET MSLT model were taken from a range of sources.[14] Relative risks for the associations between the relevant dietary factors (vegetables, fruit, sugar-sweetened beverages, nuts and seeds, red meat and processed meats) with non-communicable disease outcomes were obtained from the Global Burden of Disease study.[1] Only the direct effects of the dietary change were considered, and not those mediated by body weight due to differences in energy derived from the scenario diet and current intakes.

The current scenario was modelled in Microsoft Excel using Ersatz (EpiGear International), which enables calculation of uncertainty intervals around a point estimate. The scenarios were run with 2,000 iterations (Monte Carlo simulations) to produce 95% uncertainty intervals. Each of these simulations involved a random draw from the probability density function for the parameters specified with uncertainty within the model. Health-Adjusted Life Years (HALY) and health system costs were discounted at 3%.[19] Detailed modelling methods are available in the model's technical report.[14]

Scenarios modelled

We worked with the current Adult Nutrition Survey data (2008/09),[20] divided into 340 food and beverage categories, and showed as an average intake weight for four population groups (Māori women, Māori men, non-Māori women and non-Māori men). There were 122 food and beverage categories identified that related to the 11 optimal intake levels proposed by O'Hearn et al.[5] Intakes of these food and beverage categories were adjusted in the relative proportions that they were consumed per population group, so that the overall intake weight of each food group met the proposed weight, as shown in **Table 1**.

Of the other food and beverage categories, the average amounts of 148 categories were not modified, as these categories did not relate to the 11 proposed dietary thresholds shown in **Table 1** (e.g., egg, poultry or milk). The 70 remaining food and beverage categories were mixed dishes containing at least one of the food groups shown in **Table 1**. In a conservative approach we did not modify the amounts of mixed dishes, as we could not be confident in the ratios of each ingredient (e.g., "bacon and egg pie" would contain processed meat and refined wheat, but also egg). The most common dietary factors in mixed meals that were not modified were refined rice and wheat, processed meat and unprocessed meat.

Two scenarios were modelled. The primary model considered the direct associations between 6 of the 11 dietary factors with non-communicable disease outcomes (processed meat, unprocessed meat, sugar-sweetened beverages, fruits, non-starchy vegetables, nuts and seeds). The DIET MSLT does not include disease associations with yoghurt, starchy vegetables, fruit juice or refined and whole grains. This required a second scenario to be considered within a nutrient-based model that looked at fibre and sodium intakes when reducing refined grains, starchy vegetables and fruit juice while increasing whole grain or yoghurt intakes in line with the proposed thresholds. As the multistate life table methods assess multiple risk factors onto multiple diseases simultaneously, including the effect of increasing life expectancy on future disease incidence, it is

Table 1: Current and proposed optimal intake levels of 11 dietary factors.

Dietary factor (n categories modified)	Current intakes (grams)	Proposed intakes (grams)
Whole grains (8)	27.6	90
Yoghurt (5)	21.2	87.1
Processed meat (15)	37.6	0
Unprocessed meat (25)	64.9	14.3
Sugar-sweetened beverages (7)	183.3	0
Potatoes (9)	103.9	0
Refined rice and wheat (19)	121.7	0
Fruits (15)	150.1	300
Non-starchy vegetables (12)	119.8	300
Nuts and seeds (6)	4.5	20.3
Fruit juices (1)	46.1	0

*Current intakes are an adult population average from the Adult Nutrition Survey (2008/09) of 4,721 participants in Aotearoa New Zealand.

not possible to simply add the effects of these two scenarios together. Instead, the effects on outcomes from the two scenarios are distinct from each other.

Simulation model outcomes

The primary outcome was change in health as measured by HALY. Secondary health outcomes were indicators of health inequities by ethnicity (Māori and non-Māori),[21] and costs or cost savings to the healthcare system[14] when each scenario was compared with current dietary intakes in Aotearoa New Zealand. Health inequity by ethnicity was considered by showing the health impact to Māori and non-Māori separately and in additional analyses where the higher background rates of mortality and morbidity for Māori were adjusted down to be the lower rates experienced by non-Māori in Aotearoa New Zealand.[21]

The healthcare system's costs or cost savings were calculated based on differences in rates of diet-related disease between Business as Usual (BAU) and the intervention.[14] Costs from hospitalisations, inpatient procedures, outpatients, pharmaceuticals, laboratories and expected primary care usage were used to calculate disease, age and sex-specific health system costs. Costs from individually linked data for publicly funded (and some privately funded) health events (2006 to 2010) were sourced from the New Zealand HealthTracker database for all diseases except diabetes, which was sourced through the Virtual Diabetes Register (VDR).[14]

Role of the funding source

This project was unfunded.

Results

Current dietary intakes in Aotearoa New Zealand provide an average of 1,954g food and beverages (other than water), and 8.6MJ of energy per day. The diet modified to meet the proposed intake levels of 11 food groups provided an average of 1,860g food and beverages (other than water), and 7.6MJ of energy per day. These differences in dietary volume and energy intake do not influence the observed health results, which only consider the direct non-weight mediated associations between diet and disease that are independent of energy balance.

The primary model of health outcomes due to six of the 11 proposed optimal intakes are shown

in **Table 2**. The proposed optimal intakes scenario resulted in substantial HALY gains when compared with current intakes. HALY gain was higher for men than women (1.3 times), and higher for Māori than non-Māori (1.8 times). Per capita, HALY gains were higher again for Māori when their higher background rates of mortality and morbidity were adjusted for under the equity analysis (2.5 times), suggesting a reduction in health inequities with adoption of the proposed intakes.

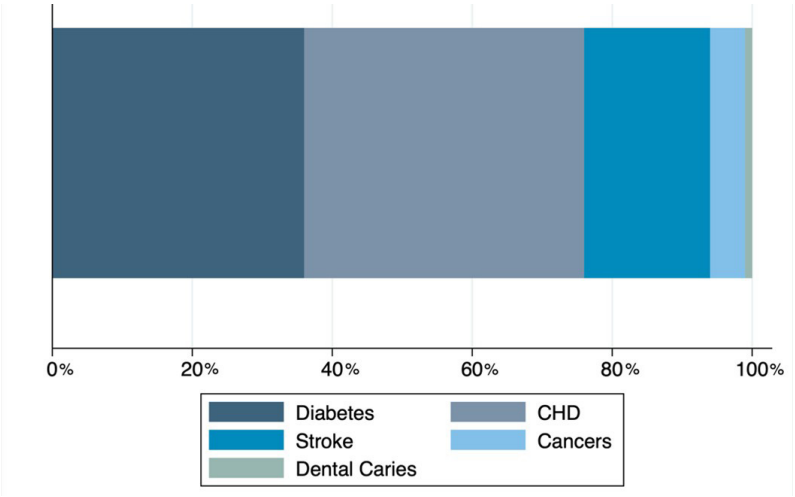
The secondary nutrient-based model considering the change in fibre and sodium content when meeting the proposed intakes of yoghurt, starchy vegetables, fruit juice, refined and whole grains are shown in **Table 3**. These results indicate a similar pattern of additional benefits, with health gains for Māori 1.3 times higher than for non-Māori when looking at age-standardised per capita health gain. This ratio increases to 1.9 times higher when using the equity analysis results.

Figure 1 shows the relative contribution of major non-communicable diseases included in the model to the HALY gain observed in the primary analyses. The major contributors to the HALY gain when moving dietary intakes towards the proposed thresholds were coronary heart disease, type 2 diabetes and stroke.

Discussion

We have modelled the direct non-weight mediated associations between diet and disease to identify the broad and systemic health effects should dietary intakes in Aotearoa New Zealand change to meet recently suggested optimised dietary thresholds for 11 food groups. These thresholds were identified in a global risk assessment analysis of the impact of a suboptimal diet on type 2 diabetes of both direct and weight-mediated associations between diet and disease;[5] however, our analyses indicate far greater health benefits beyond type 2 diabetes prevention are likely should the national diet change towards these thresholds. Health gains were greater for Māori than non-Māori, suggesting these dietary changes may reduce health inequities, while the healthcare system's savings were around NZ$18 billion over the life course of the 2011 cohort, should the dietary changes be maintained. While the potential benefits of altering present dietary intakes are striking, they are also expected. Evidence of health benefits with improved dietary intakes has long existed; however, changing population intakes is not an individual responsibility, but instead requires strong and sustained government commitment. Such action has not been shown in Aotearoa New Zealand. What is needed is an overarching national food strategy in Aotearoa New Zealand to build supportive food environments that enable healthy food choices at affordable prices. Alongside health, a national food strategy in Aotearoa New Zealand should also encompass the environmental and economic aspects of food production.

Figure 1: Contribution of major non-communicable diseases to the HALY gain observed in the primary analyses.



*Cancer burden shown here relates to colorectal, lung, oesophageal, head and neck, and ovarian cancers.

Table 2: Health and health system cost savings when adopting 6 of the 11 optimal intake thresholds (processed meat, unprocessed meat, sugar-sweetened beverages, fruits, non-starchy vegetables, nuts and seeds).

	Non-Māori	Māori	Māori	Ethnic groups combined	
	Health gains: HALYs	Health gains: HALYs	Equity analysis health gains: HALYs	Health gains: HALYs	Net health system cost savings (NZ$ billion)
Total	900,100 (716,900 to 1,101,200)	289,600 (241,400 to 342,900)	409,400 (337,300 to 489,700)	1,189,700 (961,600 to 1,447,000)	$17.9 (13.6 to 23.2)
Men	522,500	155,500	218,100	678,000	$10.8
Women	377,700	134,100	191,300	511,800	$7.1
Per capita	241.3 (299.3)	429.6 (554.4)	607.2 (785.8)	270.1	$4,068

Results in brackets in the total population row are 95% Uncertainty Intervals. Per capita results are HALYs per 1,000 people and NZ$ per adult, with results in brackets presenting age-standardised data.

Table 3: Health and health system cost savings for fibre and sodium differences when adopting the other 5 of the 11 optimal intake thresholds (yoghurt, starchy vegetables, fruit juice, refined grains and whole grains).

	Non-Māori	Māori	Māori	Ethnic groups combined	
	Health gains: HALYs	Health gains: HALYs	Equity analysis health gains: HALYs	Health gains: HALYs	Net health system cost savings (NZ$ million)
Total	17,700 (6,600 to 30,400)	3,800 (1,300 to 6,500)	5,500 (1,800 to 9,900)	21,500 (9,400 to 36,000)	$241.8 (113.0 to 384.7)
Men	10,900	1,700	2,500	12,600	$140.9
Women	6,700	2,100	3,000	8,800	$100.8
Per capita*	4.7 (5.7)	5.6 (7.4)	8.1 (10.7)	4.9	$54.9

Results in brackets in the total population row are 95% Uncertainty Intervals. Per capita results are HALYs per 1,000 people and NZ$ per adult, with results in brackets presenting age-standardised data.

The current work highlights the difference between providing optimised dietary thresholds and a reference diet. Our results indicate a 1MJ on average reduction in daily energy intake when adopting the optimised dietary thresholds, a change in energy intake which would likely have unaccountable compensatory measures in eating behaviours. To avoid this uncertainty in the modelling, we only considered the directly mediated associations between diet and disease rather than body weight mediated associations. The nutritional completeness when adopting the optimised dietary thresholds and their viability to supply in current food systems were also not considered when proposed.[5] In contrast, a reference diet like the EAT-Lancet diet,[22] proposed in 2019, which appears similar to the optimised dietary thresholds (increased minimally processed whole grain, vegetable, fruit, nut, seed and legume intake, decreased red and processed meat and sugar-sweetened beverage intake) was modelled for nutritional completeness, including energy intake, as well as the systems change required to deliver such a diet at a population level.

Recent modelling of the adoption of the Eat-Lancet diet in Aotearoa New Zealand, alongside an Aotearoa New Zealand diet optimised for health and climate impact, showed similar health gains to the current study (1.4 million Quality-Adjusted Life Years [QALY]).[23] QALY and HALY gains can be interpreted similarly for these studies. Other recent studies have examined the potential benefits of red and processed meats' replacement with five different scenarios, with similar health gains seen when current red and processed meat intakes were replaced with minimally plant based meat alternatives (1.3 million QALYs).[18] Previous modelling has shown 1.0 million QALY gains when the Aotearoa New Zealand population was modelled to follow the current dietary guidelines, with these gains increasing to 1.5 million QALYs when the Aotearoa New Zealand population was also modelled to be vegan.[17] All previous analyses align in showing that population shifts away from the current diet and towards higher intakes of dietary fibre, whole grains, vegetables, fruit and n-6 unsaturated fatty acids and lower intakes of dietary sodium and saturated fatty acids will result in appreciable benefits to health in Aotearoa New Zealand.

The strengths of the current analyses are that they utilise optimal dietary thresholds recently identified in one of largest studies of their kind,[5] consider the benefits of dietary change on much broader health metrics than just type 2 diabetes incidence and rely on data relevant to Aotearoa New Zealand. The most important limitation is that the dietary intake data are derived from the 2008/09 Adult Nutrition Survey, our most recent nationally representative dietary assessment. As the global food supply has moved towards more processed and ultra-processed foods,[24] intakes of the 2008/09 Adult Nutrition Survey data may be closer to the proposed thresholds than actual current intakes, and therefore underestimate the health benefits of meeting them. Furthermore, the current DIET MSLT model does not incorporate the disease associations for yoghurt, fruit juice, starchy vegetables, whole and refined grains, so the effects of all 11 optimised dietary threshold could not be shown together. Instead, two scenarios are shown, where a second nutrient-based model was run to consider sodium and fibre changes when meeting optimised thresholds for yoghurt, fruit juice, starchy vegetables, whole grains and refined grains. Given the more recent understanding of the importance of whole grain intakes in human health,[4,25–26] and existing uncertainty on the effects of refined grain intakes,[27] this second scenario is a likely gross underestimation of the health benefits in Aotearoa New Zealand when changing grain intakes.

Aotearoa New Zealand has a long track record of nutrition recommendations that have been issued and updated by first the Department, and then Manatū Hauora – Ministry of Health. The most recent Eating and Activity Guidelines[28] would, if fully implemented, go some way towards achieving the targets which past modelling[17] has shown would achieve appreciably improved health and environmental outcomes. However, it is widely acknowledged that a sustainable food environment that ensures the availability, at affordable cost, of health promoting foods and discourages the consumption of dietary promotors of ill health is an essential requisite to achieve meaningful changes in population dietary habits. Regrettably, national policies likely to promote such an environment are absent in Aotearoa New Zealand.

We have demonstrated that the potential wide-ranging benefits of modifying present dietary intakes in Aotearoa New Zealand may be even greater than suggested previously, and that such changes could contribute to achieving equity in health outcomes. Our findings provide evidence for reinforcing and extending current dietary guidelines. However, the population dietary changes required for the realisation of

these benefits are unlikely to be achieved without the creation of a sustainable food environment that ensures the availability, at affordable cost, of health promoting foods and discourages the consumption of dietary promotors of ill health. Successive governments have consistently held back from developing a national food strategy that includes the legislative and policy measures needed to change the current food environment. Investment now in such a strategy would have benefits for the environment and climate, in addition to health and the economy, for this, and subsequent generations.

COMPETING INTERESTS
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REFERENCES
1. Forouzanfar MH, Afshin A, Alexander LT, et al. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016;388(10053): 1659-1724. doi: 10.1016/S0140-6736(16)31679-8.
2. Reynolds AN, Hodson L, De Souza R, et al. Saturated fat and trans-fat intakes and their replacement with other macronutrients: a systematic review and meta-analysis of prospective observational studies. Geneva: World Health Organization; 2022.
3. Afshin A, Sur PJ, Fay KA, et al. Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2019;393(10184): 1958-1972. doi: 10.1016/S0140-6736(19)30041-8.
4. Reynolds A, Mann J, Cummings J, et al. Carbohydrate quality and human health: a series of systematic reviews and meta-analyses. Lancet. 2019;393(10170):434-445. doi: 10.1016/S0140-6736(18)31809-9.
5. O'Hearn M, Lara-Castor L, Cudhea F, et al. Incident type 2 diabetes attributable to suboptimal diet in 184 countries. Nat Med. 2023;29(4):982-995. doi: 10.1038/s41591-023-02278-8.
6. Vieira AR, Abar L, Chan DSM, et al. Foods and beverages and colorectal cancer risk: a systematic review and meta-analysis of cohort studies, an update of the evidence of the WCRF-AICR Continuous Update Project. Ann Oncol. 2017;28(8):1788-1802. doi: 10.1093/annonc/mdx171.
7. Becerra-Tomás N, Paz-Graniel I, W C Kendall C, et al. Nut consumption and incidence of cardiovascular diseases and cardiovascular disease mortality: a meta-analysis of prospective cohort studies. Nutr Rev. 2019;77(10):691-709. doi: 10.1093/nutrit/nuz042.
8. Viguiliouk E, Glenn AJ, Nishi SK, et al. Associations between Dietary Pulses Alone or with Other Legumes and Cardiometabolic Disease Outcomes: An Umbrella Review and Updated Systematic Review and Meta-analysis of Prospective Cohort Studies. Adv Nutr. 2019;10(Suppl_4):S308-S319. doi: 10.1093/advances/nmz113.
9. Ong KL, Stafford LK, McLaughlin SA, et al. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2023;402(10397):203-34.
10. Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD). Evidence-based European recommendations for the dietary management of diabetes. Diabetologia. 2023;66(6):965-985. doi:

10.1007/s00125-023-05894-8.
11. Uusitupa M, Khan TA, Viguiliouk E, et al. Prevention of Type 2 Diabetes by Lifestyle Changes: A Systematic Review and Meta-Analysis. Nutrients. 2019;11(11):2611. doi: 10.3390/nu11112611.
12. Blakely T, Moss R, Collins J, et al. Proportional multistate lifetable modelling of preventive interventions: concepts, code and worked examples. Int J Epidemiology. 2020;49(5):1624-1636. doi: 10.1093/ije/dyaa132.
13. Briggs ADM, Cobiac LJ, Wolstenholme J, Scarborough P. PRIMEtime CE: a multistate life table model for estimating the cost-effectiveness of interventions affecting diet and physical activity. BMC Health Serv Res. 2019;19(1):485. doi: 10.1186/s12913-019-4237-4.
14. Cleghorn C, Blakely T, Nghiem N, et al. Technical Report for BODE^3 Intervention and DIET MSLT models, Version 1. Wellington (NZ): Department of Public Health, University of Otago; 2017. Available from: https://www.otago.ac.nz/__data/assets/pdf_file/0015/330009/technical-report-for-bode-diet-intervention-and-multistate-lifetable-models-670797.pdf.
15. Cleghorn C, Wilson N, Nair N, et al. Health benefits and cost-effectiveness from promoting smartphone apps for weight loss: multistate life table modeling. JMIR Mhealth Uhealth. 2019;7(1):e11118. doi: 10.2196/11118.
16. Cleghorn C, Blakely T, Mhurchu CN, et al. Estimating the health benefits and cost-savings of a cap on the size of single serve sugar-sweetened beverages. Prev Med. 2019;120:150-156. doi: 10.1016/j.ypmed.2019.01.009.
17. Drew J, Cleghorn C, Macmillan A, Mizdrak A. Healthy and Climate-Friendly Eating Patterns in the New Zealand Context. Environ Health Perspect. 2020;128(1):017007. doi: 10.1289/EHP5996.
18. Reynolds AN, Mhurchu CN, Kok ZY, Cleghorn C. The neglected potential of red and processed meat replacement with alternative protein sources: Simulation modelling and systematic review. EClinicalMedicine. 2022;56:101774. doi: 10.1016/j.eclinm.2022.101774.
19. Sanders GD, Neumann PJ, Basu A, et al. Recommendations for conduct, methodological practices, and reporting of cost-effectiveness analyses: Second panel on cost-effectiveness in health and medicine. JAMA. 2016;316(10):1093-103. doi: 10.1001/jama.2016.12195.
20. University of Otago and Ministry of Health. Methodology Report for the 2008/09 New Zealand Adult Nutrition Survey. Wellington (NZ): Ministry of Health; 2011 [cited 2023 Sep 5]. Available from: https://www.health.govt.nz/publication/methodology-report-2008-09-nz-adult-nutrition-survey.
21. McLeod M, Blakely T, Kvizhinadze G, Harris R. Why equal treatment is not always equitable: the impact of existing ethnic health inequalities in cost-effectiveness modeling. Popul Health Metr. 2014;12:15. doi: 10.1186/1478-7954-12-15.
22. Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems. Lancet. 2019;393(10170):447-492. doi: 10.1016/S0140-6736(18)31788-4.
23. Cleghorn C, Nghiem N, Ni Mhurchu C. Assessing the Health and Environmental Benefits of a New Zealand Diet Optimised for Health and Climate Protection. Sustainability. 2022;14(21):13900. doi: 10.3390/su142113900.
24. Baker P, Machado P, Santos T, et al. Ultra-processed foods and the nutrition transition: Global, regional and national trends, food systems transformations and political economy drivers. Obes Rev. 2020;21(12):e13126. doi: 10.1111/obr.13126.
25. Reynolds AN, Akerman A, Kumar S, et al. Dietary fibre in hypertension and cardiovascular disease management: systematic review and meta-analyses. BMC Med. 2022;20(1):139. doi: 10.1186/s12916-022-02328-x.
26. Reynolds AN, Akerman AP, Mann J. Dietary fibre and whole grains in diabetes management: Systematic review and meta-analyses. PLoS Med. 2020;17(3):e1003053. doi: 10.1371/journal.pmed.1003053.
27. Gaesser GA. Perspective: Refined Grains and Health: Genuine Risk, or Guilt by Association? Adv Nutr. 2019;10(3):361-371. doi: 10.1093/advances/nmy104.
28. Manatū Hauora – Ministry of Health. Eating and Activity Guidelines for New Zealand Adults: Updated 2020 [Internet]. Wellington (NZ): Manatū Hauora – Ministry of Health; 2020 [cited 2023 Sep 5]. Available from: https://www.health.govt.nz/publication/eating-and-activity-guidelines-new-zealand-adults.

Performance of a fast-track pathway for giant cell arteritis in Waikato, Aotearoa New Zealand

Philippa van Dantzig, Douglas White, Jason Kurz, Caroline Ming, Sujatha Kamalaksha, Vicki Quincey

ABSTRACT

AIMS: Giant cell arteritis (GCA) is the most common primary vasculitis in adults over 50 years of age. To facilitate early diagnosis and reduce harms from corticosteroids and temporal artery biopsies, fast-track pathways have been established. We review the benefits of the fast-track pathway set up in Waikato, Aotearoa New Zealand.

METHODS: Patients were collected prospectively as part of the fast-track pathway from 2014 to 2022. Their records were then reviewed retrospectively to collect data on clinical features, investigations and treatment.

RESULTS: There were 648 individual patients over the study period who had a colour Doppler ultrasound (CDUS) of the temporal arteries. There were 17 true positive CDUS, giving a sensitivity of 10.3% (95% confidence interval [CI] 6.3–15.5%) and specificity of 99.8% (95% CI 99.1–100%). Patients with GCA and a positive scan had significantly fewer steroids than those with GCA and a negative scan (p=0.0037). There were 376 patients discharged after a CDUS who did not have a diagnosis of GCA, resulting in reduced corticosteroid and temporal artery biopsy exposure.

CONCLUSIONS: This is a real-life study that reflects the benefits of fast-track pathways in Aotearoa New Zealand to patients and healthcare systems. It also shows the effect of corticosteroids on positive CDUS, an important consideration when setting up an fast-track pathway.

Giant cell arteritis (GCA) is a large vessel vasculitis, which is the most common primary vasculitis in adults over 50 years of age. Ischaemic complications can arise, including vision loss that can be permanent. Early accurate diagnosis and prompt treatment is therefore critical.

Historically, temporal artery biopsy (TAB) has been the primary means of diagnosis; however, in recent years, there has been focus on colour Doppler ultrasound (CDUS) of the temporal and axillary arteries with particular interest in its low cost and availability.[1,2] Numerous meta-analyses support the performance of CDUS in the diagnosis of giant cell arteritis.[1,3–9]

Fast-track pathways in GCA aim to have a risk assessment by a specialist (usually a rheumatologist or ophthalmologist) performed at the time of referral with a CDUS organised for the same day or next working day. Decisions regarding further tests and the need to continue corticosteroids are made rapidly, reducing harms of treatment or investigations to patients. The benefits of fast-track pathways include less vision loss, reduced time to diagnosis and fewer TAB requests.[2,10–13] Fast-track pathways also support primary care physicians, who have highlighted access issues in the rapid investigation of patients with suspected GCA.[14]

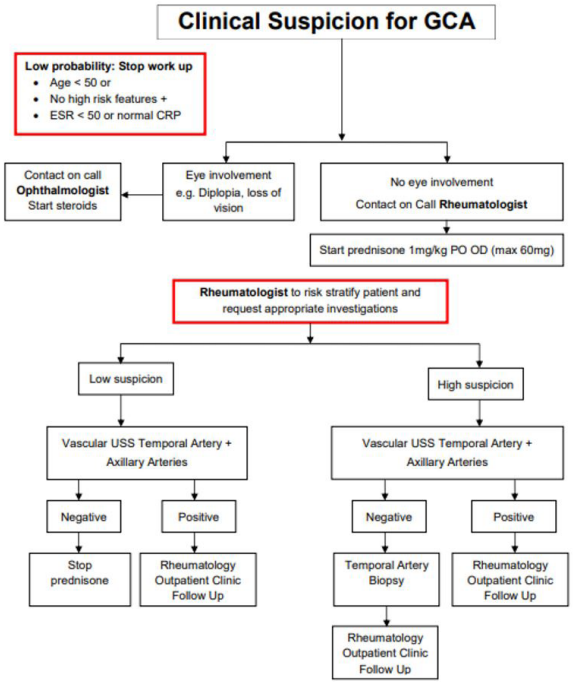
Most of the data on fast-track pathways do not take into consideration "real-world" practice. This is where our study is beneficial in reflecting the practical aspects of implementing a fast-track pathway for GCA in the hope of guiding other healthcare centres in the design of their own pathways.

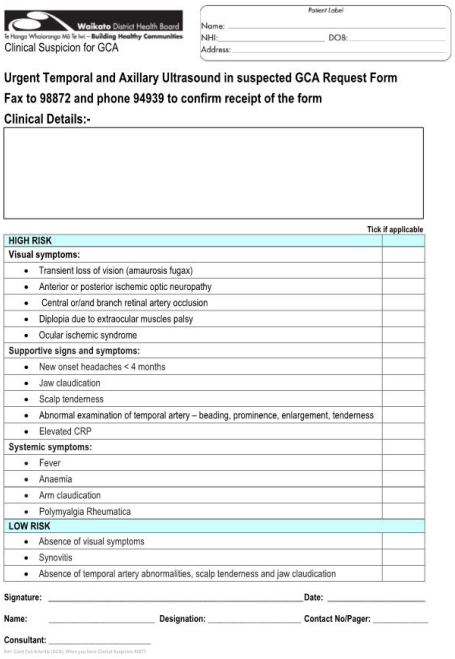
Methods

The Rheumatology Department at the Waikato Hospital set up a GCA fast-track pathway at the end of 2013 (Figure 1) in collaboration with ophthalmologists. Patients suspected of having GCA were referred via telephone from primary care physicians, specialists or inpatient teams to either Ophthalmology (if visual symptoms were present) or Rheumatology (if no visual symptoms).

If the risk of GCA was assessed as sufficient, a CDUS of the temporal arteries was requested, which would usually be done on the day of or next day after the referral. If the referral was made out-of-hours, corticosteroids would be commenced at

Figure 1: The protocol for the GCA fast-track pathway at Waikato Hospital, Aotearoa New Zealand.




Waikato District Health Board
Te Hanga Whaioranga Mō Te Iwi – Building Healthy Communities
Clinical Suspicion for GCA

Patient Label
Name:
NHI: DOB:
Address:

Urgent Temporal and Axillary Ultrasound in suspected GCA Request Form
Fax to 98872 and phone 94939 to confirm receipt of the form
Clinical Details:-

	Tick if applicable
HIGH RISK	
Visual symptoms:	
• Transient loss of vision (amaurosis fugax)	
• Anterior or posterior ischemic optic neuropathy	
• Central or/and branch retinal artery occlusion	
• Diplopia due to extraocular muscles palsy	
• Ocular ischemic syndrome	
Supportive signs and symptoms:	
• New onset headaches < 4 months	
• Jaw claudication	
• Scalp tenderness	
• Abnormal examination of temporal artery – beading, prominence, enlargement, tenderness	
• Elevated CRP	
Systemic symptoms:	
• Fever	
• Anaemia	
• Arm claudication	
• Polymyalgia Rheumatica	
LOW RISK	
• Absence of visual symptoms	
• Synovitis	
• Absence of temporal artery abnormalities, scalp tenderness and jaw claudication	

Signature: **Date:**
Name: **Designation:** **Contact No/Pager:**
Consultant:

NB: GCA = giant cell arteritis; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; PO = oral; OD = daily; USS = ultrasound scan.

the discretion of the on-call specialist. Following the CDUS, the specialist would make a decision regarding further investigation (i.e., TAB) and on appropriate follow-up.

Ultrasound examinations were performed by experienced vascular ultrasonographers with postgraduate- or master's-level qualifications. The protocol involved scanning both temporal and axillary arteries. Ultrasounds early in the study period were performed using the GE Logiq 9 or Philips IU22 ultrasound system with transducers of operating frequency at 3–9MHz or 5–17MHz. Over the course of the study period, newer ultrasound systems and transducers were used. By 2021, there was an upgrade to GE Logiq 10 and Philips EPIQ (with higher frequency transducers of 4–18MHz or 3–12MHz). When scanning temporal or axillary arteries, the highest frequency transducer for adequate penetration was chosen.

Cases in the fast-track pathway have been collected prospectively from January 2014 using the referral request for CDUS. The requests from January 2014 to December 2022 were reviewed retrospectively to gather data on clinical symptoms, laboratory results and to generate a list of all cases in the fast-track pathway. The electronic records for each case were searched to collect information from referrals, results of investigations, treatment received by patients and final clinical diagnoses noted by treating physicians. In order to capture all patients going through the fast-track pathway, we also searched ultrasonography lists for patients who had a CDUS requested over this period. If the CDUS occurred alone or prior to a TAB, it was considered part of the fast-track pathway. TAB lists were also collected for the 3 years prior to the pathway to appreciate the rate of biopsy use prior to implementation of the pathway.

Ethnicities have been reported in conjunction with Statistics New Zealand (Stats NZ) reporting,[15] and where patients identified with two different ethnicities (commonly Māori and New Zealand European), both were counted, thus giving a total percent as greater than 100%. This is consistent with how Stats NZ report their census data.

Given the pre-test probability of GCA was not often clear from the records, a risk score was applied, dividing patients into risk categories using an externally validated probability score established by Ing et al.[16] This includes age, gender, clinical symptoms (i.e., new headache, temporal artery tenderness or reduced pulse, jaw or tongue claudication, diplopia or typical

vision loss) with erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and elevated platelets above upper limit of normal. Based on these factors, a "probability of having GCA" score was generated, organising patients into very low risk (<2.7%), low risk (<7%), moderate risk (<23%), high risk (<43%) and very high risk (>43%). This score appears to assist in the triage of patients with suspected GCA, though not without fault.[16,17]

CDUS was classified as positive if the halo sign was present (defined as a hypoechoic ring around the lumen of the temporal or axillary artery) and indeterminate if only wall thickening was noted according to the radiology report. TAB was classified as positive if there was evidence of active inflammation consistent with GCA and the histopathologist's report was supportive of active GCA. If there was a suggestion of possible past arteritis, this was not included as a positive TAB.

Statistical analysis

Descriptive data are presented as frequencies for categorical variables. Continuous variables are presented as a mean with standard deviation (SD) and a median with interquartile range (IQR). Where symptoms were not reported, this was reported as missing data; thus, percentage calculations are a valid percent rather than a total percent. Student *t*-Tests or Welch's *t*-Test were performed where appropriate, as were non-parametric tests (Mann–Whitney U test). All analyses were conducted in IBM SPSS 29 (New York, United States). All significance tests were two-tailed and p-values of less than 0.05 were considered significant.

Ethics statement

National ethics approval was granted by HDEC (Reference: 2023 EXP 15448) and there was a local assessment through the Waikato Hospital who also approved the project (RD023025), which included review by Te Puna Oranga Māori Consultation Research Review Committee.

Results

Between January 2014 and December 2022, there were 664 patients who were referred through the fast-track pathway, with 648 individual patients. There were 16 duplicate episodes that have been excluded from analysis but are detailed in the Appendix.

Baseline characteristics

Patients managed through the fast-track pathway had a mean (SD) age of 70.5 (11.0) years and 69.3% were female. The age range was 17 to 96 years, with 25 patients (3.8%) being under 50 years of age. Ethnicity, clinical and laboratory features are detailed further in Table 1.

Of those referred through the fast-track pathway, 511/648 (78.8%) patients were managed by Rheumatology, 39/648 (6.0%) by Ophthalmology, 45/648 (6.9%) by a combination of both and 53/648 (8.2%) by other teams, which included General Medicine or Neurology.

Colour Doppler ultrasound

All patients in the fast-track pathway had CDUS performed. Out of 648 CDUS scans, 18 (2.8%) were reported as positive with the halo sign, 102/648 (15.7%) had abnormal vessel wall thickening noted and were thus labelled as indeterminate and 528/648 (81.5%) were negative. Axillary involvement was noted with vessel wall thickening (no axillary halos noted) in 52/648 (8.0%) of patients. A final diagnosis of GCA was made in 166/648 (25.6%) of patients.

Figure 2 illustrates the flow of patients through the pathway, outlining investigations performed. It shows how patients exited the pathway if they did not have GCA.

For patients with a halo sign (all of which were in the temporal artery), 17/18 (94.4%) had a final diagnosis of GCA. There was one patient with a halo sign on CDUS but clinical review assessed them as not having GCA. Out of the 102 patients with an indeterminate CDUS, 60/102 (58.8%) had a final diagnosis of GCA. Out of those with a negative CDUS, 89/528 (16.8%) had a final diagnosis of GCA. The sensitivity and specificity of CDUS compared to different reference standards are summarised in Table 2.

Non-specific vessel wall thickening was noted in 102 patients (i.e., indeterminate scans) and in 21/102 (20.5%) of these patients, this was supportive enough for a final diagnosis of GCA without a TAB needing to be performed. A further 61/102 (59.8%) patients required a TAB, of which 20/61 (32.8%) were positive and thus labelled as GCA, 41/61 (67.2%) were negative and of these, 19/41 (46.3%) were diagnosed with GCA. Thus, in total, 60/102 (58.8%) of indeterminate CDUS were associated with a diagnosis of GCA.

The sensitivity of a non-negative CDUS (i.e., either halo sign present or vessel wall thickening) was 46.7% (95% confidence interval [CI] 39.1–54.3%)

Table 1: Clinical characteristics of patients in the fast-track pathway.

	FTP (n=648)
Ethnicity no. pts (%)	
European	553 (85.3)
Māori	63 (9.7)
Pacific peoples	7 (1.1)
Asian	9 (1.4)
MELAA	4 (0.6)
Other	10 (1.5)
Not stated	10 (1.5)
Total	(101.1)
Clinical features	
Days of symptoms—median (IQR)	14.0 (6–30)
Symptoms—no./valid no. (valid %)	
Headache (any)	565/617 (91.6)
Headache (unilateral)	288/617 (46.7)
Scalp sensitivity	278/461 (60.3)
Jaw claudication	162/486 (33.3)
Visual symptoms (any)	219/488 (44.9)
Typical visual symptoms (AION, PION, CRAO)	21/648 (3.2)‡
Diplopia	20/648 (3.1)‡
PMR symptoms	171/375 (45.6)
Temporal artery abnormality†	252/392 (64.3)
Laboratory features—no./valid no. (valid %)	
Haemoglobin g/L	
<115 (women)	66/426 (15.5)
<130 (men)	65/176 (37.0)
Platelets >400 (%) x109/L	101/616 (16.4)

Table 1 (continued): Clinical characteristics of patients in the fast-track pathway.

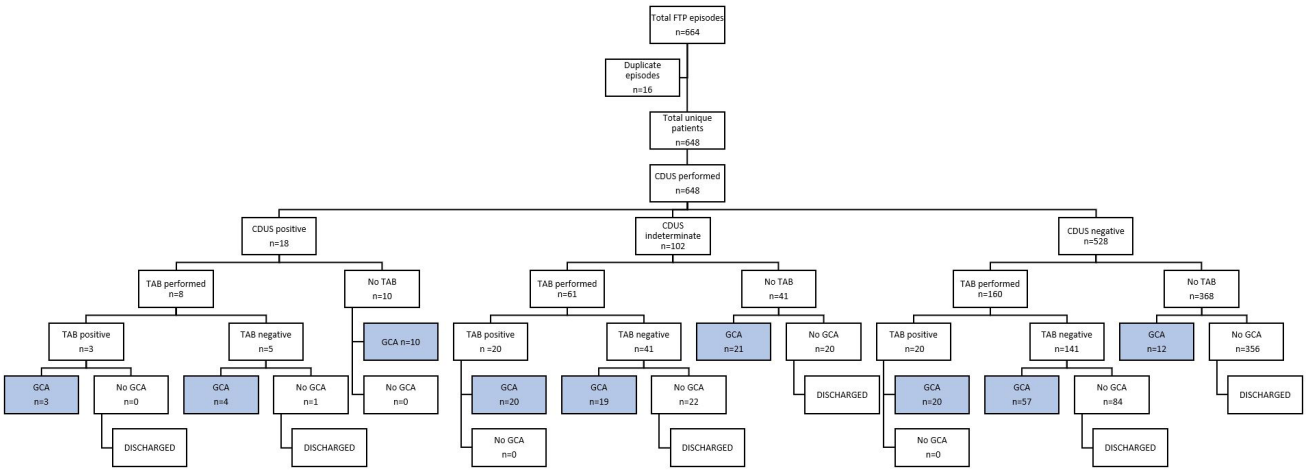
ESR mm/hour	
mean (SD)	30.6 (26.5)
median (IQR)	23.5 (10–41)
CRP mg/L	
mean (SD)	40.9 (66.0)
median (IQR)	10.0 (2.5– 53.0)
ACR 2022 criteria score—no. (%)	
6 or more	365/638 (57.2)
Less than 6	273/638 (42.8)
Risk using Ing risk score[16] n. pts (valid %) n=503	
Very low <2.7%	37/503 (7.4)
No. with GCA (% of risk group)	2 (5.4)
Low <7, 2.7%	152/503 (30.2)
No. with GCA (% of risk group)	10 (6.6)
Moderate <23, 7.0%	171/503 (34.0)
No. with GCA (% of risk group)	34 (19.8)
High <43, 23.0%	69/503 (13.7)
No. with GCA (% of risk group)	30 (43.5)
Very high ≥43%	74/503 (14.7)
No. with GCA (% of risk group)	53 (71.6)

FTP = fast-track pathway; MELAA = Middle Eastern, Latin America, African; IQR = interquartile range; AION = anterior ischaemic optic neuropathy; PION = posterior ischaemic optic neuropathy; CRAO = central retinal artery occlusion; PMR = polymyalgia rheumatica; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; ACR = American College of Rheumatology; GCA = giant cell arteritis.
†Temporal artery abnormality—either decreased pulse or tenderness.
‡% calculated as number of typical symptoms out of the total number of patients in this pathway, rather than the number of variables collected.

and specificity was 91.1% (95% CI 88.3–93.4%). The positive predictive value was 64.2% (95% CI 55.4–72.4%) and negative predictive value was 83.3% (95% CI 80.0–86.3%).

Corticosteroids were started in 73.5% of patients. The mean (SD) was 13.8 (55.6) days of corticosteroids prior to CDUS and the median (IQR) was 1 (1–4) day. For patients with a final diagnosis of GCA, those with a positive CDUS had a mean (SD) duration pre-ultrasound of 0.7 (SD 1.1) days and a median (IQR) of 0 (0–1.3) days. This compared to a mean (SD) of 14.2 (56.2) days and median (IQR) of 1 (0–4) days in patients with a negative CDUS (p=0.007). When patients who were already on corticosteroids for polymyalgia rheumatica were excluded from analysis, the mean and median did not change for the positive CDUS group but was reduced to a mean (SD) 3.1 (13.1) and median (IQR) 1 (0–3) days for the negative CDUS group, which remained statistically different between the two groups (p=0.022).

Figure 2: Flowchart of patients through the Waikato Giant Cell Arteritis fast-track pathway.



NB: FTP = fast-track pathway; CDUS = colour Doppler ultrasound; TAB = temporal artery biopsy; GCA = giant cell arteritis.

Table 2: Sensitivity and specificity of colour Doppler ultrasound compared to different reference standards.

Reference	Sensitivity	Specificity	Positive predictive value	Negative predictive value
	% (95% confidence interval)			
Clinical diagnosis	10.3 (6.3–15.5)	99.8 (99.1–100)	94.4 (77.7–99.7)	76.5 (73.1–79.7)
Temporal artery biopsy	7.1 (1.8–17.5)	97.3 (94.3–99.0)	37.5 (11.0–71.0)	82.4 (77.0–87.0)
ACR 2022 criteria ≥6	4.7 (2.8–7.1)	99.6 (98.4–100.0)	94.4 (77.7–99.7)	43.9 (40.0–47.8)

ACR = American College of Rheumatology.

TAB

TAB was performed in 229/648 patients and was positive in 42/229 (18.3%) patients. The mean (SD) size of biopsy was 16.0 (9.8) mm. The British Society of Rheumatology guidelines recommend a biopsy length of at least 10mm.[18] In this cohort, 48/229 patients (21.1%) had a TAB length of less than 10mm, with 5/48 (10.4%) of these biopsies being positive.

Time to TAB was mean (SD) 14.5 (20.6) days with a median (IQR) of 10 (5–17) days. Duration of corticosteroids prior to TAB was mean (SD) 25.1 (46.1) days with a median (IQR) of 12 (4–25.5) days. The sensitivity and specificity of TAB with clinical diagnosis as a reference standard were 34.4% and 100% respectively.

TAB was avoided in 43/648 (6.6%) of patients with GCA after having a CDUS performed.

Prior to the pathway being established (i.e., 2011 to 2013), the mean annual incidence of all TAB requests was 28.1 (95% CI 23.0–33.3) per 100,000 people over 50 years. This reduced to

21.5 (95% CI 19.1–23.8) per 100,000 people over 50 years. Given the slight overlap of CIs, this was not statistically significant but was clearly trending downwards in conjunction with the introduction of the fast-track pathway.

Patients discharged from the pathway

Focussing on patients exiting the pathway, there were 376/648 patients (58.0%) who were discharged after a CDUS who did not have a final diagnosis of GCA. These patients had a mean (SD) of 15.6 (66.9) days and a median (IQR) of 1 (0–4) days of corticosteroids. Patients without GCA but who had a TAB and a CDUS performed had a mean (SD) duration of corticosteroids of 23.7 (SD 36.6) and a median of 12 (3–27) days. This is significantly longer than those patients who were discharged after only a CDUS ($p<0.001$). When patients who were previously on corticosteroids for polymyalgia rheumatica were excluded, the mean (SD) and median (IQR) for the CDUS only group was 3.0 (16.0) days and 0 (0–2) days respectively. In the CDUS and TAB group, this was a mean (SD) of 12.4 (12) and median 9 (2–19) days, remaining a statistically significant difference ($p<0.001$).

COVID-19 pandemic

Aotearoa New Zealand had an initial lockdown period due to the COVID-19 pandemic in March–May 2020, and a subsequent lockdown in August–September 2021 when there was community transmission. During these periods, the service remained in place where patients and physicians had access to colour Doppler ultrasound and TAB. Reviews occurred via telephone consultation unless the patient was unwell, in which case a face-to-face review was organised.

Discussion

This real-world study of the Waikato GCA fast-track pathway is the largest cohort published to date, alongside the study by Pinnell et al.[19] who had 620 patients. The benefit of a fast-track pathway for GCA is clear with significant numbers of patients avoiding TAB, an invasive and costly investigation for both patients and healthcare systems.[1] This was evident for low-risk patients without GCA who had a non-positive CDUS result. This could also be appreciated by the down-trending rate of TAB requests with implementation of the pathway. The reduction in exposure to corticosteroids by only having CDUS to investigate GCA rather than needing a TAB was significant. This would likely translate to reduced corticosteroid toxicity for patients.

We note that our data is reflective of TAB access in Aotearoa New Zealand but may not be as significant a finding in other centres with rapid access to TAB. Rheumatology patients with suspected GCA are referred to vascular surgery, an under-resourced service where waiting times to TAB are often outside of the optimal window. For a portion of patients with a higher probability of GCA, the positive or indeterminate CDUS result was supportive enough to commit to the diagnosis and avoid the need for biopsy.

This study helps assess the performance of colour Doppler ultrasound in a real-world setting where, due to practical and safety reasons, corticosteroids are commenced at the time of referral. The sensitivity in our study is significantly lower than that reported in numerous meta-analyses. Table 3 provides a comparison of our data to other meta-analyses. Corticosteroid use appears to significantly decrease the chance of a positive colour Doppler ultrasound and may be part of the reason for our lower sensitivity. Pinnell et al. also performed a real-world study that had a lower sensitivity for colour Doppler ultrasound. They demonstrated the impact that corticosteroids had on detecting a positive CDUS and an increase in sensitivity when ultrasound was performed without corticosteroids.[19]

Corticosteroids appear to contribute to the disappearance of the halo sign.[1,20–22] Hauenstein et al.[20] noted that if colour Doppler ultrasound was performed on the first day of corticosteroid treatment, the sensitivity of the ultrasound was 88%. It dropped to 50% after 2–4 days of corticosteroids and 50% if patients had more than 4 days of corticosteroids.

While the number of positive colour Doppler ultrasounds is small, there is a larger number with increased thickening of the blood vessel wall. It remains unknown if any of these would have manifested a halo sign if corticosteroids had been withheld until after the ultrasound was performed. In the development of our fast-track pathway in Waikato, urgent corticosteroid treatment is mandatory to avoid consequences. Our protocol design and the restraints on our healthcare system cannot always guarantee a same-day CDUS.

Despite this emerging association, corticosteroid exposure may not entirely explain the discrepancy in the number of positive colour Doppler ultrasounds in our study compared

Table 3: Meta-analyses on the performance of colour Doppler ultrasound in GCA.

Study		Sensitivity (%)	Specificity (%)
Clinical diagnosis as reference standard			
Duftner 2018[3]		77	96
Sebastian 2021[4]		67	95
Moreel 2023[5]		80	95
	(including large vessels)	95	96
Nakajima 2023[6]		76	93
	(including axillary arteries)	86	95
Temporal artery biopsy as reference standard			
Karassa 2005[7]		69	82
Duftner 2018[3]		70	84
Rinagel 2019[8]		68	81
Sebastian 2021[4]		63	90
ACR criteria 1990 as reference standard			
Karassa 2005[7]		55	94
Arida 2010[9]		68	91
Current study			
	Clinical diagnosis	10.3	99.8
	Temporal artery biopsy	7.1	97.3
	ACR 2022 criteria	1.3	90.0

GCA = giant cell arteritis; ACR = American College of Rheumatology.

to others, and the reasons are probably multifactorial. There may be a larger number of low-risk patients entering the pathway, which reduces the number of true GCA cases and is reflected by 37.6% of patients being categorised as very low risk or low risk through the prediction score. Of note, Sebastian et al.[4] and Melville et al.[2] had similar risk profiles in their study and yet had 37.6% and 30.2% positive scans respectively. The total number of cases of clinically diagnosed GCA in our study was 19.7%, which is smaller than to Sebastian et al.'s and Melville et al.'s studies of 25% and 34.1% respectively.[2,4]

Technical factors including ultrasound machines and probes may play a role given that Aotearoa New Zealand has a resource-limited healthcare system with ultrasonographers using older, less advanced equipment at the start of the study period. Our ultrasonographers are experienced in vascular ultrasound; however, it remains unclear

as to how this compares to experts internationally, specifically trained in the features of GCA on ultrasound. We are currently undertaking a retrospective audit on the ultrasounds to look for any features of GCA that had not been reported in the final report and will use this to further improve the fast-track pathway.

The majority of patients in this cohort are referred to Rheumatology with smaller proportions of referrals to Ophthalmology. Other studies have not noted this discrepancy between specialties.[19] Our fast-track pathway protocol recommends that patients with any vision symptoms are referred to Ophthalmology. Rheumatology had 25% of their patients reporting any vision symptoms, suggesting some of their patients should be seen by Ophthalmology instead.

As we reflect on the implementation of this fast-track pathway in Waikato, we can visualise potential improvements. The fact that 3.8% of patients were under 50 years raises the question that many low-risk patients were entering the pathway, perhaps inappropriately. There is a significant proportion (189/648 [37.6%]) of patients who are low or very low risk entering the pathway, though 12/189 (6.3%) of these had a final diagnosis of GCA. It is difficult to know if this reflects the weaknesses of prediction scores for GCA or the entry of too many low-risk patients to the pathway. As a real-world study, physicians have varying levels of confidence in excluding GCA in low-risk patients and this is reflected in our data. We must note that when the pathway was developed in 2013, in order to validate the safety, efficacy and accuracy of the pathway for the investigation of GCA, all patients regardless of risk needed to go through the pathway. The CDUS was a new test to the department and caution was exercised. Given the pathway is now well established, review of entry criteria and exploring other prediction tools would be appropriate to reduce unnecessary patients going through. Clearly, ultrasound access has improved but access to TAB is delayed and more focus could be on improving this aspect of the pathway.

We acknowledge other limitations to this study. It is a retrospective study and there was missing data due to inadequate documentation. We have not included patients from the private health community in Aotearoa New Zealand. However, the private sector does not have the same rapid access to colour Doppler ultrasound as this pathway does; thus, most patients would have entered the public health system to access the pathway. Lastly, we acknowledge that there are no conventional pathway data to provide a true reflection of the benefit that this pathway has had on the Waikato community.

Conclusion

Fast-track pathways using temporal artery colour Doppler ultrasound in the investigation of GCA are beneficial to patients and our healthcare systems. There is a reduction in the number of temporal artery biopsies required and subsequent reduction in exposure to corticosteroids in patients without GCA. Corticosteroid exposure, while often mandatory in preventing serious complications, appears to reduce the sensitivity of colour Doppler ultrasound and remains an issue to consider when designing a fast-track pathway. Reflection around entry criteria to such pathway is also crucial.

COMPETING INTERESTS
Nil.
Philippa van Dantzig has been employed part-time by the Waikato Hospital (Te Whatu Ora) for 12 months in a research position to carry out this research project among others, as well as perform a clinical role. There is no other specific funding towards the project.

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REFERENCES
1. Luqmani R, Lee E, Singh S, et al. The Role of Ultrasound Compared to Biopsy of Temporal Arteries in the Diagnosis and Treatment of Giant Cell Arteritis (TABUL): a diagnostic accuracy and cost-effectiveness study. Health Technol Assess. 2016;20(90):1-238. doi: 10.3310/hta20900.
2. Melville AR, Donaldson K, Dale J, Ciechomska A. Validation of the Southend giant cell arteritis probability score in a Scottish single-centre fast-track pathway. Rheumatol Adv Pract. 2021;6(1):rkab102. doi: 10.1093/rap/rkab102.
3. Duftner C, Dejaco C, Sepriano A, et al. Imaging in diagnosis, outcome prediction and monitoring of large vessel vasculitis: a systematic literature review and meta-analysis informing the EULAR recommendations. RMD Open. 2018;4(1):e000612. doi: 10.1136/rmdopen-2017-000612.
4. Sebastian A, Tomelleri A, Kayani A, et al. Probability-based algorithm using ultrasound and additional tests for suspected GCA in a fast-track clinic. RMD Open. 2020;6(3):e001297. doi: 10.1136/rmdopen-2020-001297.
5. Moreel L, Betrains A, Doumen M, et al. Diagnostic yield of combined cranial and large vessel PET/CT, ultrasound and MRI in giant cell arteritis: A systematic review and meta-analysis. Autoimmun Rev. 2023;22(7):103355. doi: 10.1016/j.autrev.2023.103355.
6. Nakajima E, Moon FH, Junior NC, et al. Accuracy of Doppler ultrasound in the diagnosis of giant cell arteritis: a systematic review and meta-analysis. Adv Rheumatol. 2023;63(1):5. doi: 10.1186/s42358-023-00286-3.
7. Karassa FB, Matsagas MI, Schmidt WA, Ioannidis JP. Meta-analysis: test performance of ultrasonography for giant-cell arteritis. Ann Intern Med. 2005;142(5):359-69. doi: 10.7326/0003-4819-142-5-200503010-00011.
8. Rinagel M, Chatelus E, Jousse-Joulin S, et al. Diagnostic performance of temporal artery ultrasound for the diagnosis of giant cell arteritis: a systematic review and meta-analysis of the literature. Autoimmun Rev. 2019;18(1):56-61. doi: 10.1016/j.autrev.2018.07.012.
9. Arida A, Kyprianou M, Kanakis M, Sfikakis PP. The diagnostic value of ultrasonography-derived edema of the temporal artery wall in giant cell arteritis: a second meta-analysis. BMC Musculoskelet Disord. 2010;11:44. doi: 10.1186/1471-2474-11-44.
10. Farah Z, Gulati M, Matheou M, et al. THU0344 A rheumatology led pathway for the initial management of GCA improves diagnostic outcomes compared to the generalist. Ann Rheum Dis. 2017;76(Suppl 2):333-4.
11. Diamantopoulos AP, Haugeberg G, Lindland A, Myklebust G. The fast-track ultrasound clinic for early diagnosis of giant cell arteritis significantly reduces permanent visual impairment: towards a more effective strategy to improve clinical outcome in giant cell arteritis? Rheumatology (Oxford). 2016;55(1):66-70. doi: 10.1093/rheumatology/kev289.
12. Monti S, Bartoletti A, Bellis E, et al. Fast-Track Ultrasound Clinic for the Diagnosis of Giant Cell Arteritis Changes the Prognosis of the Disease

but Not the Risk of Future Relapse. Front Med (Lausanne). 2020;7:589794. doi: 10.3389/fmed.2020.589794.
13. Patil P, Williams M, Maw WW, Achilleos K, et al. Fast track pathway reduces sight loss in giant cell arteritis: results of a longitudinal observational cohort study. Clin Exp Rheumatol. 2015;33(2 Suppl 89):S-103-6.
14. Helliwell T, Muller S, Hider SL, et al. Challenges of diagnosis and management of giant cell arteritis in general practice: a multimethods study. BMJ Open. 2018;8(2):e019320. doi: 10.1136/bmjopen-2017-019320.
15. Stats New Zealand. 2018 Census place summaries: New Zealand. [cited 2023 Jul 2]. Available from: https://www.stats.govt.nz/tools/2018-census-place-summaries/new-zealand.
16. Ing EB, Lahaie Luna G, Toren A, et al. Multivariable prediction model for suspected giant cell arteritis: development and validation. Clin Ophthalmol. 2017;11:2031-42. doi: 10.2147/OPTH.S151385.
17. Jasim M, Magan P, Patel F, et al. Can a Giant Cell Arteritis (GCA) Risk Stratification Score Be Helpful in Clinical Practice? Cureus. 2022;14(12):e32310. doi: 10.7759/cureus.32310.
18. Dasgupta B; Giant Cell Arteritis Guideline Development Group. Concise guidance: diagnosis and management of giant cell arteritis. Clin Med (Lond). 2010;10(4):381-6. doi: 10.7861/clinmedicine.10-4-381.
19. Pinnell J, Tiivas C, Mehta P, Dubey S. Corticosteroids reduce vascular ultrasound sensitivity in fast- track pathways (FTP): results from Coventry Multi-Disciplinary FTP for cranial Giant Cell Arteritis. Scand J Rheumatol. 2023;52(3):283-92. doi: 10.1080/03009742.2022.2051279.
20. Hauenstein C, Reinhard M, Geiger J, et al. Effects of early corticosteroid treatment on magnetic resonance imaging and ultrasonography findings in giant cell arteritis. Rheumatology (Oxford). 2012;51(11):1999-2003. doi: 10.1093/rheumatology/kes153.
21. Schmidt WA, Kraft HE, Vorpahl K, et al. Color duplex ultrasonography in the diagnosis of temporal arteritis. N Engl J Med. 1997;337(19):1336-42. doi: 10.1056/NEJM199711063371902.
22. Karahaliou M, Vaiopoulos G, Papaspyrou S, et al. Colour duplex sonography of temporal arteries before decision for biopsy: a prospective study in 55 patients with suspected giant cell arteritis. Arthritis Res Ther. 2006;8(4):R116. doi: 10.1186/ar2003.

Appendix

Table 4: Duplicate episodes in the fast-track pathway with reasons for subsequent episode.

Number of patients (n=16)	**Reason for duplicate episode**		
Patients n=3	Repeat GCA-like symptoms with negative tests and no clinical diagnosis of GCA.		
Patients n=3	Repeat investigation to look for objective evidence in patients with a diagnosis of GCA. One of these had a subsequent positive CDUS after previous negative CDUS (difference between scans was 918 days).		
Patients n=7	Repeat testing to look for recurrence of GCA in patients who already had a diagnosis of GCA.		
Possible missed diagnosis n=3	See descriptions below.		
	1st episode	**2nd episode**	**Final outcome**
Patient 1	Negative CDUS. No GCA.	380 days later. Negative CDUS + negative TAB.	GCA after 2nd episode.
Patient 2	Headache, CDUS indeterminate. TAB negative. No GCA.	572 days later. Positive CDUS. No TAB done.	GCA after 2nd episode.
Patient 3	Negative CDUS.	6 years later. Negative CDUS. Negative TAB.	GCA after 2nd episode.

Temporal trends of transport-related injuries on New Zealand roads

Siobhan Isles, Michael Keane, Joanna F Dipnall, Ben Beck

ABSTRACT

AIM: This observational study aimed to investigate temporal trends in transport-related injuries in New Zealand by mode of transport and explore whether specific population groups and localities have a relatively higher incidence of injury. These trends provide insight into changes in injury patterns from road trauma.

METHODS: A retrospective study of hospitalised road trauma in New Zealand was conducted between 1 July 2017 to 30 June 2021. Data were obtained from the National Minimum Dataset of hospital admissions and the New Zealand Trauma Registry (NZTR). Road trauma was identified using ICD-10 coding, and major trauma using Abbreviated Injury Scale (AIS) coding. Analysis included road trauma by mode, ethnicity, rurality and population rates. Statistical analysis included Interrupted Time Series (ITS) analysis to account for the impact of COVID-19 on road trauma.

RESULTS: Over the 4-year period there were 20,607 incidents of transport-related injury that resulted in admission to a New Zealand hospital. Of these, 14.5% (2,992) involved injuries that were classified as major trauma. Car occupants accounted for 62% of hospitalisations, followed by motorcyclists (23%), pedestrians (9%) and pedal cyclists (4%). Temporal trends showed no reduction in injuries from cars, pedal cyclists and pedestrian injuries, but an increase in motorcycling injuries. Māori had an age-standardised incidence rate almost 3.5 times higher than the rate for Asian peoples.

CONCLUSION: The increases in motorcycling injuries and no changes in pedestrian and cycling injuries, as well as demographic variation, highlight the need to focus on vulnerable road users. Effective and targeted initiatives on vulnerable road users will support objectives to reduce deaths and serious injury on New Zealand roads. Enhanced exposure data is needed for vulnerable road users to account for mobility changes over time. Linked data across population-based datasets is an important asset that enhances our understanding of road traffic injuries and allows evidence-based countermeasures to be developed.

Death and serious injury involving transport are important metrics for monitoring and evaluating road safety initiatives. New Zealand has a higher rate of road fatalities than comparable countries[1] and, after a downward trend in fatalities and serious injuries since the mid-1980s, the number of people killed or seriously injured on New Zealand roads has been increasing since 2013.[2] To reverse this trend, investment in road safety strategies have been launched with the aim to reduce annual deaths and serious injury caused by road transport.[3] The focus of these strategies are motorised modes of personal transport such as cars and motorbikes, and also public transport, cycling and walking. Policies are influenced by the successes observed in countries such as the Netherlands and Sweden, which have changed approaches from being an individual's responsibility to take a systems-approach to addressing road safety issues. The "safe system" approach is focussed on three principles: people make mistakes, roads and vehicles need to be designed to minimise the impact of crashes and road safety is a shared responsibility.[4]

Accurate and comprehensive data is important to monitoring performance against targets, evaluating road safety interventions and setting future priorities. However, complete transport injury information is not centralised in any single source. While serious car crashes in New Zealand are typically documented in the Crash Analysis System,[5] moderate and minor road incidents, particularly those involving a non-motorised mode of transport such as cycling and walking, are not always captured by police.[6]

Covering a 4-year period using data from hospital admission and the New Zealand Trauma Registry (NZTR), the present study investigated temporal trends in injury-causing crashes in New Zealand and explored whether specific population groups have an increased incidence of injury. These trends provide insight into changes in injury patterns from road trauma.

The study period included the COVID-19 pandemic and resultant changes to daily life following the introduction of the COVID-19 Alert System. This study explored the impact of the lockdown on injury rates during and after the restrictions.

Methods

This observational study of hospitalised patients in New Zealand used existing data from national collections and no additional information was obtained from individuals. The study covers the period between 1 July 2017 to 30 June 2021.

Data sources

Hospitalisations were identified within the Hospital Events National Minimum Dataset (NMDS),[7] a national collection of all hospital admission data. Using ICD-10-AM[8] external cause codes, which are linked to individual hospital events, admissions coded as being caused by road traffic injury were identified. Road traffic injury was defined as "traffic" codes in the range V010 to V899, as well as a small number of X and Y codes. Non-traffic codes (e.g., mountain biking) were excluded.

Events were excluded if the patient was discharged home from the emergency department, or the patient was admitted to hospital over 14 days after the crash, or the admission was arranged and the clinical specialty was Maternity Services, or no overnight stay occurred, or the clinical specialty was Mental Health Services or arranged or waitlist.

Mode of transport was identified using the ICD-10-AM coding in the NMDS and categorised into car (driver or passenger, and passenger cars only), motorcycle (driver or passenger), pedal cycle and pedestrian. The last category was "Other", and included any mechanised vehicle not classified as a car such as a truck, van, light or bus, and incidents involving horses or other animals on a road.

Major trauma incidents were linked to the hospitalisation dataset using National Health Index (NHI) in the NZTR,[9] a population-based registry that collects data on all major trauma patients who have a threat to life and are admitted to acute hospitals. Matching cases between the NMDS and the NZTR enabled stratification analyses by major and non-major trauma.

Statistics New Zealand population projections for relevant periods and for all population subgroups and rural/urban classifications were used. The 2013 Census estimated population for Māori was used as the reference population.

Prioritised ethnicity data associated with the NHI was used.

Rural/urban classification is based on University of Otago's Geographic Classification of Health[10] using the patient's domicile code of residence and Census data. This does not reflect where the injury occurred.

The study period included the COVID-19 pandemic and the introduction of the lockdowns that were first put in place from 26 March 2020 to 27 April 2020, with strong restrictions on transport. Legally, individuals had to stay home other than for essential personal movement. These restrictions resulted in a dramatic decrease in traffic volume to around 15% of usual rates during the lockdown period.[11] Auckland city was subject to further lockdowns at other periods during the pandemic.

The Health and Disability Ethics Committee approved this study (2022 EXP 12993) and the Data Governance Groups of the NZTR and Manatū Hauora – Ministry of Health approved the use of their respective data.

Statistical analysis

Age standardisation was applied to all ethnicity rates, with 95% confidence intervals calculated using the Dobson method.[12] Incidence rates were calculated using the 2022 Census data and presented as an event rate per 100,000 population, as robust exposure data were unavailable for all modes of transport. Temporal trends were not analysed for pedestrians and cyclists due to insufficient numbers.

Due to the disruption to usual transport activity by the COVID-19 restrictions in 2020, an interrupted time series analysis (ITSA) was used to determine changes in the number of incidents over the study period.[13] The ITSA models accounted for seasonality and were compared to the counterfactual to investigate if the disruption altered the post-COVID trends. Two models were used for the modes of transport with sufficient incidents to analyse: one for motorcycle, and one for car occupant. Further details are described in the Appendices. Analysis was performed using R statistical software[14] and statistical significance was defined as $p < 0.05$.

Results

Over the 4-year period there were 20,607 incidents of transport-related road trauma that resulted in a hospital admission in New Zealand. Of these, 14.5% (2,992) involved injuries that were classified as major trauma.

Car occupants accounted for 62% of incidents, followed by motorcyclists (23%), pedestrians (9%) and pedal cyclists (4%) (Table 1). Males made up 63% of the cohort and the median age was

Table 1: Age, ethnicity, sex and period of road trauma cohort by mode of transport, 2017/2018–2020/2021. Q1= first quartile, Q3= third quartile.

		Mode of transport			
	All road trauma	**Car**	**Motorcycle**	**Pedal cycle**	**Pedestrian**
N =	20,607	12,664	4,709	910	1,802
Age (years), median (Q1–Q3)	38 (23, 59)	36 (22–60)	41 (26–55)	48 (28–60)	39 (20–64)
Ethnicity					
Māori	5,097 (25%)	3,355 (26%)	1,077 (23%)	115 (13%)	441 (24%)
Pacific peoples	1,287 (6.2%)	898 (7.1%)	187 (4.0%)	24 (2.6%)	149 (8.3%)
Asian peoples	1,516 (7.4%)	1,005 (7.9%)	192 (4.1%)	50 (5.5%)	217 (12%)
European/ other	12,707 (62%)	7,406 (58%)	3,253 (69%)	721 (79%)	995 (55%)
Sex					
Female	7,707 (37%)	5,886 (46%)	620 (13%)	205 (23%)	760 (42%)
Male	12,900 (63%)	6,778 (54%)	4,089 (87%)	705 (77%)	1,042 (58%)
Period					
2017/2018	5,256 (26%)	3,341 (26%)	1,093 (23%)	223 (25%)	478 (27%)
2018/2019	5,353 (26%)	3,272 (26%)	1,183 (25%)	257 (28%)	497 (28%)
2019/2020	4,784 (23%)	2,939 (23%)	1,130 (24%)	201 (22%)	379 (21%)
2020/2021	5,214 (25%)	3,112 (25%)	1,303 (28%)	229 (25%)	448 (25%)

Note: "Other" mode of transport was excluded because of low numbers.

Figure 1: Monthly count of road trauma hospitalisations by mode of transport, 2017/2018–2020/2021. Note: Y-axis uses a logarithmic scale.

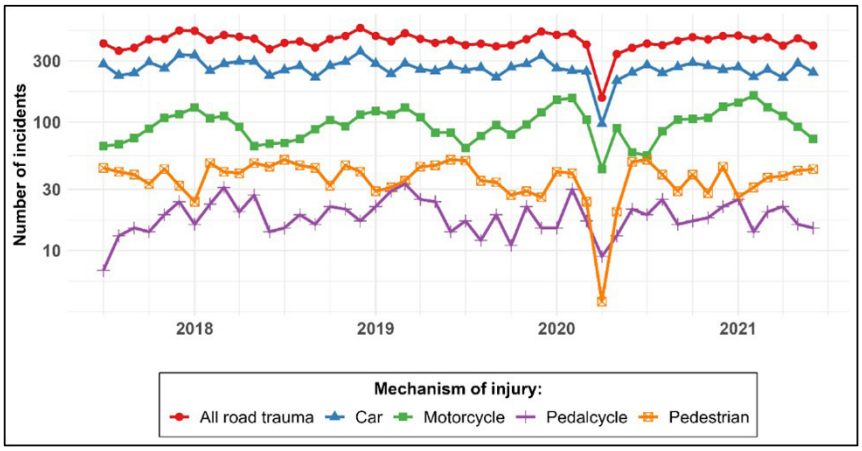


Table 2: Incidence rate of transport-related hospitalisations per 100,000 people by event type and mode of transport, 2017/2018–2020/2021. Parentheses contain 95% confidence interval.

Injury severity	2017/2018	2018/2019	2019/2020	2020/2021
Non-major trauma hospitalisation	94.5 (91.8–97.3)	93.8 (91.1–96.6)	82.7 (80.2–85.3)	88.3 (85.7–91.0)
Major trauma	14.9 (13.8–16.0)	15.9 (14.8–17.0)	14.0 (12.9–15.0)	15.7 (14.6–16.8)
Mode of transport				
Car	69.6 (67.2–72.0)	67.1 (64.8–69.4)	59.4 (57.2–61.6)	62.1 (59.9–64.3)
Motorcycle	22.8 (21.4–24.2)	24.3 (22.9–25.7)	22.8 (21.5–24.2)	26.0 (24.6–27.4)
Pedal cycle	4.6 (4.1–5.3)	5.3 (4.6–6.0)	4.1 (3.5–4.7)	4.6 (4.0–5.2)
Pedestrian	10.0 (9.1–10.9)	10.2 (9.3–11.1)	7.7 (6.9–8.5)	8.9 (8.1–9.8)
Other	2.5 (2.1–3.0)	3.0 (2.5–3.5)	2.7 (2.3–3.2)	2.4 (2.0–2.9)
Total	**109.5 (106.5–112.5)**	**109.7 (106.8–112.7)**	**96.6 (93.9–99.4)**	**104.0 (101.2–106.9)**

Table 3: Incidence rate of transport-related hospitalisations per 100,000 people by rural urban classification and ethnicity, 2017/2018–2020/2021. Parentheses contain 95% confidence interval.

Rural Urban Classification	Rate per 100,000 (95% confidence interval)
Urban 1	92.0 (90.3–93.7)
Urban 2	108.9 (105.5–112.3)
Rural 1	127.8 (123.3–132.4)
Rural 2	131.8 (125.0–138.9)
Rural 3	198.3 (179.7–218.3)
Ethnicity	**Age-standardised rate per 100,000 (95% confidence interval)**
Māori	155.9 (151.6–160.2)
Pacific peoples	94.0 (88.9–99.3)
Asian peoples	45.1 (42.6–47.6)
European/other	90.7 (88.8–92.6)

38 years (IQR: 25, 59). Those of European/other ethnicities made up 62% of incidents, followed by Māori (25%), Asian peoples (7.4%) and Pacific peoples (6.2%).

A sharp decline in deaths and serious injury in early 2020 was observed and coincided with the period of the COVID-19 Alert Level 4 lockdown, which was in place from March 2020 to April 2020 (Figure 1).

The incidence rate of all transport-related hospitalisation was lowest in 2019/2020, at 96.6 per 100,000 people (95% CI: 93.6–99.4), although motorcycle incidents were highest in 2020/2021 than in any other period prior (Table 2).

The rate of transport-related hospitalisation varied between age groups within different modes of transport. Those aged 80 years and older had the highest rate of incidents as car occupants, followed by those aged 20–24 years. Pedal cyclist incidents were highest for those aged 55–59 years. An increase in the incidence rate of motorcycling injuries was observed for males aged 10–19 and 60–69 years (Appendix Table 1).

Asian peoples had a substantially lower rate of transport-related hospitalisation than other ethnicity groups, with around half the incidence rate of NZ European/other for both injury severity categories (Table 3). Māori had an age-standardised incidence rate almost 3.5 times higher than the rate for Asian peoples.

Incidence rates for road trauma hospitalisation were higher in people living in rural areas relative to people living in urban areas. People living in the remotest rural areas (rural 3) had over twice the rate of hospitalisation (198.3, 95% CI: 179.7–218.3) than those living in the densest urban areas (92.0, 95% CI: 90.3–93.7; Table 3).

Car model

The seasonal model best captures the car injury hospitalisations patterns for both the pre-COVID and post-COVID periods (refer red line predictions in Figure 2). Again, the sharp drop-off of car injury hospitalisations at the start of the COVID-19 period appeared to be only a minor interruption as the expected pre-COVID seasonal pattern returned.

During the pre-COVID period, there was no change over time in the number of car occupant hospitalisation injuries (IRR= 1.000, 95% CI: 0.999–1.000, p= 0.326). The car model detected a fall in the rate of car injury hospitalisations of between 3% to 68% into the initial week of COVID-19 (IRR= 0.565, 95% CI: 0.320–0.997, p= 0.049). However, the average car rate returned to the counterfactual by first week into the post-COVID period, with no difference between the post-COVID and counterfactual detected (IRR= 1.032, 95% CI: 0.918–1.161, p= 0.595) (Appendix Table 2).

Motorcycle model

The seasonal model captured the seasonal pattern in motorcycle hospitalisation counts for both the pre-COVID and post-COVID periods, as seen by the predictions in Figure 3. The sharp drop-off of motorcycle-related hospitalisations at the start of the COVID-19 period appeared to be only a minor interruption as the expected pre-COVID seasonal pattern returned, albeit initially lower than the counterfactual model, but returned by the end of the period.

During the pre-COVID period there was an increase in the number of weekly motorcycle injury hospitalisations (*IRR*= 1.001, *95% CI:* 1.000–

Figure 2: Predicted outcome from seasonally adjusted and non-seasonally adjusted car model, with counterfactual scenario (dashed lines).

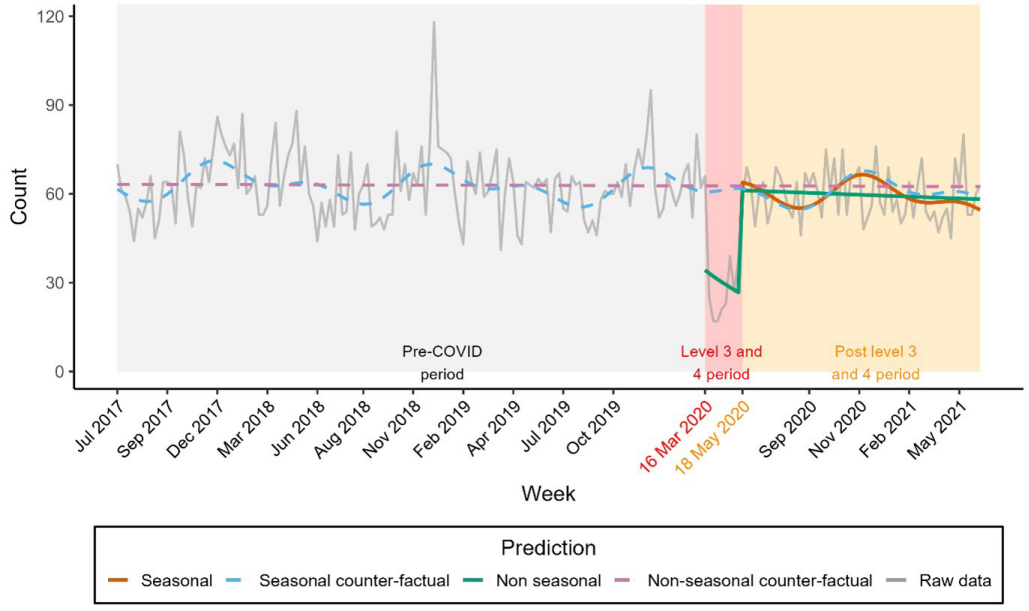
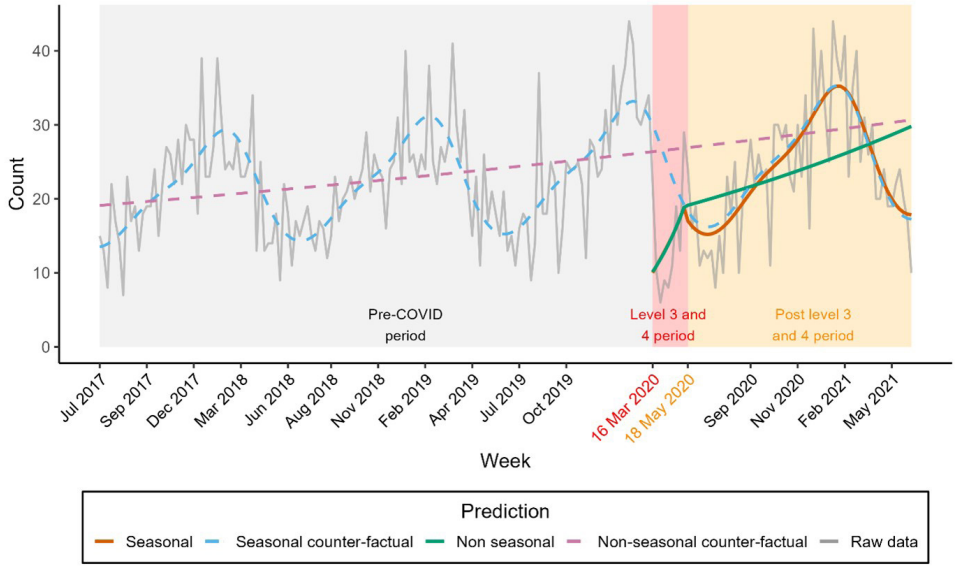


Figure 3: Predicted outcome from seasonally adjusted and non-seasonally adjusted motorcycle model, with counterfactual scenario (dashed lines).



1.002, p= 0.009). As expected, the COVID-19 period was associated with a large decrease in the count of motorcycle injury hospitalisations into the first week of between 33% to 83% (IRR= 0.334, 95% CI: 0.167–0.667, p= 0.002), which was an initial deviation from the counterfactual. However, post-COVID, motorcycle hospitalisations were similar to the predicted rate using data from a pre-COVID period. (Appendix Table 3).

Discussion

This study has highlighted the difference in rates across modes, and across ethnicity groups, and has described the impact of COVID-19. The results are unique to the New Zealand context, although many results are consistent with the findings in international studies of this nature.[15–17]

The rate of motorcycle injury hospitalisations increased over the study period and were over 10% higher in 2020/2021 than in 2017/2018, and largely occurred in men. This is despite investment in initiatives to reduce motorcycle injuries, such as rider competency training.[18] The incidence rate is nearly double for motorcycle injury for those who live rurally compared to urban domicile, but to the best of our knowledge there is scant research to understand why the difference occurs. The results suggest two quite distinct demographic groups who are being injured on motorcycles: males aged between 10–19 and 60–69 years, where significant increases in the rate of injury were observed, and males aged between 20–59 that had high rates of injury. Motorcycle exposure data shows a reduction of 18 million kilometres over the 4 years of this study.[19] This suggests that while motorcycling injuries are increasing, that increase is *not* necessarily associated with increased exposure.

The converse pattern is observed for pedal cyclists. The number of cycling injuries has remained stable, but the number of cyclists has increased, at least in the urban areas on key cycling routes.[20–22] One possible explanation is that the infrastructure spending by local councils and the transport agencies to build dedicated cycle lanes separated from cars and other vehicles may be successful in reducing cycling injuries. Another possibility is that cycling injuries are not being captured well in the data, which is discussed further below.

There is distinct variation in the patterns of transport-related injury by ethnicity. We found that Asian peoples have a much lower rate of transport-related road trauma. There is a dearth of research to provide insight into what influences among Asian peoples lead to this low rate of injury. While Asian peoples may be more urban than the total population, this does not explain the difference, as both major and non-major trauma rates in Asian peoples are approximately half of the rates in the urban 1 category.

A study by Randal et al. showed a faster rate of increase of transport-related deaths and serious injury for Māori than non-Māori between 2014 and 2017.[23] Our study shows this upward trend has continued unabated over the subsequent 4 years after their study. There is a substantial long-term negative impact on Māori health at a similar level due to the effects of tobacco and obesity.[24] The relationship between age, rurality and Māori ethnicity indicate there are specific geographical locations where serious injury is particularly high.

The differences of hospitalisation rate across ethnicities, sex and age groups provide some impetus to the aims of *Road to Zero*. Given the evidence that there is variation between some groups within the population on the magnitude of the 40% target, this implies that the aims are achievable in principle, and that a focus on reducing inequities is a worthy endeavour.

Strengths and weaknesses

The use of ICD-10-AM external cause codes alone appears to be insufficient for identifying all relevant hospitalisations that occurred during the study period. The coding system distinguishes between "traffic" and "non-traffic" events, and analysis suggests hospitalised pedal cyclists and pedestrians not involved in a collision with another vehicle are often coded as "non-traffic" events, even if the incident occurred on a public road. All but one of the 910 pedal cycle incidents captured in this study have an external cause code indicating a collision with another vehicle. The NMDS collects information on place of occurrence (which can be identified as a road/street or highway) of injury and external cause codes for falls, which taken together could be used to identify transport incidents that have been coded as "non-traffic" in future studies. Previous work investigating pedestrian injuries in Victoria, Australia using this method identified approximately 65 times more pedestrian falls than were present in the Victorian Police report Road Crash Data.[25]

Better exposure data, and agreement on how to interpret that data, is needed for all modes of transport to understand whether changes over time can be attributed to changes in usage. Differences between modes with respect to the number of kilometres, travel time, number of trips and vehicle registrations contribute to analysis of exposure rates. Notwithstanding this limitation, trend analysis, such as analysed in this paper, is important particularly in the context of reducing deaths and serious injuries for all road users.

Conclusions

This study has highlighted critical differences in road trauma rates and explored trends over time. The increases in motorcycling injuries and no changes in car, pedestrian and cycling injuries, as well as demographic variation, highlight the need to focus on vulnerable road users. Effective and targeted initiatives for vulnerable road users will support the aims to reduce deaths and serious injury on New Zealand roads. More clearly defined exposure data is needed to provide an accurate denominator. Linked data across population-based datasets is an important asset that enhances our understanding of road traffic injuries and allows evidence-based countermeasures to be developed.

COMPETING INTERESTS
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REFERENCES
1. World Health Organization. Global Status Report on Road Safety 2018 [Internet]. Geneva (CH): World Health Organization; 2018 [cited 2023 Jun 9]. Available from; https://apps.who.int/iris/handle/10665/276462.
2. Ministry of Transport. Safety — Road deaths [Internet]. NZ: Ministry of Transport; 2023 [cited 2023 Apr 21]. Available from: https://www.transport.govt.nz/statistics-and-insights/safety-road-deaths/.
3. Waka Kotahi NZ Transport Agency. Road to Zero [Internet]. NZ: Waka Kotahi NZ Transport Agency; 2023 [cited 2023 Apr 19]. Available from: https://www.nzta.govt.nz/safety/what-waka-kotahi-is-doing/nz-road-safety-strategy/.
4. International Transport Forum. Zero Road Deaths and Serious Injuries: Leading a Paradigm Shift to a Safe System. OECD; 2016. doi:10.1787/9789282108055-en.
5. Waka Kotahi NZ Transport Agency. Crash Analysis System (CAS) [Internet]. NZ: Waka Kotahi NZ Transport Agency; 2023 [cited 2023 Jun 9]. Available from: https://www.nzta.govt.nz/safety/partners/crash-analysis-system/.
6. Koorey G, Sim P, Diorni G. How do we measure harm in transport? [Internet]. NZ: Transportation Group New Zealand; 2023 [cited 2023 Jun 9]. Available from: https://viastrada.nz/sites/default/files/2023-04/Transportation2023-ThinkPiece-KooreyEtAl-revised.pdf.
7. Te Whatu Ora – Health New Zealand. National Minimum Dataset (hospital events) [Internet]. Wellington (NZ): Ministry of Health; 2023 [cited 2013 Apr 21]. Available from: https://www.health.govt.nz/nz-health-statistics/national-collections-and-surveys/collections/national-minimum-dataset-hospital-events.
8. Te Whatu Ora – Health New Zealand. Introduction to Classification – ICD-10-AM/ACHI/ACS [Internet]. Wellington (NZ): Ministry of Health; 2023 [cited 2013 Apr 21]. Available from: https://www.health.govt.nz/nz-health-statistics/classification-and-terminology/icd-10-am-achi-acs.
9. National Trauma Network. New Zealand Trauma Registry [Internet]. Wellington (NZ): National Trauma Network; 2024 [cited 2023 Apr 21]. Available from: https://www.majortrauma.nz/nz-mtr/.
10. Whithead J, Davie G, de Graaf B, et al. Defining rural in Aotearoa New Zealand: a novel geographic classification for health purposes. N Z Med J. 2022;135(1559):24-40.
11. Automobile Association. Road report: The impact Covid-19 had on motoring in New Zealand [Internet]. NZ: AA; 2020 [cited 2023 Apr 21]. Available from: https://www.aa.co.nz/membership/aa-directions/driver/road-report-the-impact-covid-19-had-on-motoring-in-new-zealand/.
12. Dobson AJ, Kuulasmaa K, Eberle E, Scherer J. Confidence intervals for weighted sums of Poisson parameters. Stat Med. 1991;10(3):457-462. doi: 10.1002/sim.4780100317.
13. Bernal JL, Cummins S, Gasparrini A. Interrupted time series regression for the evaluation of public health interventions: a tutorial. Int J Epidemiol. 2017;46(1):358-355. doi: 10.1093/ije/dyw098.
14. R Core Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing [Internet]. Vienna (AT): R Foundation; 2024 [cited 2023 Jun 9]. Available from: https://www.R-project.org/.
15. Leijdesdorff HA, Gillissen S, Schipper IB, Krijnen P. Injury Pattern and Injury Severity of In-Hospital Deceased Road Traffic Accident Victims in The Netherlands: Dutch Road Traffic Accidents Fatalities. World J Surg. 2020;44(5):1470-1477. doi: 10.1007/s00268-019-05348-6.
16. Fagerlind H, Harvey L, Humburg P, et al. Identifying individual-based injury patterns in multi-trauma road users by using an association rule mining method. Accid Anal Prev. 2022;164:106479. doi:

10.1016/j.aap.2021.106479.
17. Koch DA, Hagebusch P, Lefering R, et al. Changes in injury patterns, injury severity and hospital mortality in motorized vehicle accidents: a retrospective, cross-sectional, multicenter study with 19,225 cases derived from the TraumaRegister DGU®. Eur J Trauma Emerg Surg. 2023;49(4):1917-1925. doi: 10.1007/s00068-023-02257-1.
18. Ride Forever [Internet]. NZ: Ride Forever; 2024 [cited 2014 Apr 19]. Available from: https://www.rideforever.co.nz/.
19. Te Manatū Waka Ministry of Transport. RD017 Vehicle kilometers travelled by motorcycle [Internet]. NZ: Ministry of Transport; 2021/2017 [cited 2023 Apr 19]. Available from: https://www.transport.govt.nz/statistics-and-insights/road-transport/.
20. Cycling in Christchurch. Christchurch Cycling Counter [Internet]. Christchurch (NZ): Cycling in Christchurch; 2022 [cited 2023 Apr 19]. Available from: http://cyclingchristchurch.co.nz/topic/cycle-numbers/.
21. Bike There. Monthly biking stats [Internet]. Wellington (NZ): Wellington City Council; 2023 [cited 2023 Apr 19]. Available from: https://www.bikethere.org.nz/monthly-biking-stats/.
22. Auckland Transport. Monthly cycle monitoring, 2023 monthly cycle monitoring report [Internet]. Auckland (NZ): Auckland Transport; 2023 [cited 2023 Apr 21]. Available from: https://at.govt.nz/cycling-walking/research-monitoring/monthly-cycle-monitoring/.
23. Waka Kotahi NZ Transport Agency. Māori road safety outcomes report [Internet]. NZ: Waka Kotahi NZ Transport Agency; 2021 [cited 2023 Apr 21]. Available from: https://www.nzta.govt.nz/resources/maori-road-safety-outcomes-report/.
24. Randal E, Shaw C, McLeod M, et al. The Impact of Transport on Population Health and Health Equity for Māori in Aotearoa New Zealand: A Prospective Burden of Disease Study. Int J Environ Res Public Health. 2022;19(4):2032. doi: 10.3390/ijerph19042032.
25. Oxley J, O'Hern S, Burtt D, Rossiter B. Falling while walking: A hidden contributor to pedestrian injury. Accid Anal Prev. 2018;114:77-82. doi: 10.1016/j.aap.2017.01.010.
26. Zeileis A, Kleiber C, Jackman S. Regression Models for Count Data in R. J Stat Softw. 2008;27(8). doi: 10.18637/jss.v027.i08.
27. Bhaskaran K, Gasparrini A, Hajat S, et al. Time series regression studies in environmental epidemiology. Int J Epidemiol. 2013;42(4):1187-1195. doi: 10.1093/ije/dyt092.
28. Stock JH, Watson MW. Introduction to Econometrics. 3rd ed. US: Addison-Wesley; 2011.
29. Nelson M. Tests and Confidence Intervals on Directly Standardized Rates for Several Methods [Internet]. R package version 1.0.0; 2022. [cited 2023 Jun 9]. Available from: https://CRAN.R-project.org/package=dsrTest.
30. Hothorn T, Bretz F, Westfall P. Simultaneous Inference in General Parametric Models. Biom J. 2008;50(3):346-363. doi: 10.1002/bimj.200810425.

Appendices

Appendix Table 1: Incidence rate of motorcycle transport-related hospitalisations per 100,000 people by age group, 2017/2018–2020/2021. Parentheses contain 95% confidence interval.

	Period			
Age (years)	**2017/2018**	**2018/2019**	**2019/2020**	**2020/2021**
0–9	1.3 (0.5–2.5)	1.7 (0.9–3.1)	3.0 (1.8–4.7)	1.7 (0.9–3.1)
10–19	16.5 (13.5–20.0)	17.5 (14.4–21.1)	19.1 (15.9–22.8)	23.8 (20.2–27.9)
20–29	38.2 (33.7–43.1)	39.0 (34.5–43.9)	31.2 (27.2–35.7)	36.0 (31.7–40.8)
30–39	28.0 (24.0–32.5)	28.9 (24.9–33.4)	26.7 (22.9–30.9)	27.2 (23.4–31.4)
40–49	31.4 (27.1–36.1)	30.4 (26.2–35.0)	30.6 (26.4–35.3)	30.4 (26.2–35.0)
50–59	34.4 (30.0–39.4)	37.8 (33.1–42.9)	34.6 (30.2–39.5)	38.7 (34.0–43.9)
60–69	18.7 (15.1–22.9)	25.9 (21.6–30.6)	26.0 (21.8–30.7)	35.1 (30.3–40.5)
70–79	9.9 (6.8–14.0)	11.5 (8.2–15.7)	12.1 (8.8–16.3)	15.0 (11.3–19.5)
80+	7.5 (4.0–12.9)	5.1 (2.3–9.6)	3.3 (1.2–7.1)	11.5 (7.2–17.3)

Appendix Table 2: Estimated incident rate ratios, 95% confidence intervals and p-values for car seasonal and non-seasonal model.

	Seasonal model			**Non-seasonal model**		
Characteristic	**IRR[1]**	**95% CI[1]**	**p-value**	**IRR[1]**	**95% CI[1]**	**p-value**
Pre-COVID period slope change	1.000	0.999, 1.000	0.326	1.000	0.999, 1.001	0.906
COVID-19 period level change	0.565	0.320, 0.997	0.049	0.545	0.306, 0.971	0.040
COVID-19 period slope change	0.967	0.860, 1.088	0.575	0.970	0.862, 1.091	0.608
Post-COVID period level change	2.474	1.352, 4.526	0.003	2.361	1.309, 4.257	0.004
Post-COVID period slope change	1.032	0.918, 1.161	0.595	1.030	0.916, 1.159	0.617
Seasonal parameters (Fourier)						
Fourier 1	0.973	0.939, 1.008	0.131			
Fourier 2	0.963	0.932, 0.995	0.026			
Fourier 3	0.935	0.901, 0.970	0.000			
Fourier 4	1.039	1.001, 1.078	0.042			

[1]IRR = Incidence Rate Ratio, CI = Confidence Interval

Appendix Table 3: Estimated incident rate ratios, 95% confidence intervals and p-values for motorcycle seasonal and non-seasonal model.

	Seasonal model			Non-seasonal model		
Characteristic	**IRR[1]**	**95% CI[1]**	**p-value**	**IRR[1]**	**95% CI[1]**	**p-value**
Pre-COVID period slope change	1.001	1.000, 1.002	0.009	1.002	1.000, 1.005	0.063
COVID-19 period level change	0.334	0.167, 0.667	0.002	0.385	0.196, 0.754	0.006
COVID-19 period slope change	1.144	1.010, 1.296	0.035	1.078	0.953, 1.220	0.232
Post-COVID period level change	0.829	0.490, 1.402	0.483	0.940	0.508, 1.738	0.842
Post-COVID period slope change	0.876	0.773, 0.993	0.039	0.933	0.824, 1.055	0.268
Seasonal parameters (Fourier)						
Fourier 1	0.884	0.843, 0.927	0.000			
Fourier 2	1.051	1.006, 1.097	0.025			
Fourier 3	0.736	0.706, 0.766	0.000			
Fourier 4	0.925	0.889, 0.962	0.000			

[1]IRR = Incidence Rate Ratio, CI = Confidence Interval

Statistical analysis

Motorcycling and car occupant data was collapsed into weekly counts and plotted as a time series graph to establish the appropriate ITSA period segments: pre-COVID period included weeks up to 16 March 2020 and considered the counterfactual; COVID-19 period corresponded to Level 4 and 3 restrictions and included weeks 16 March 2020 to 17 May 2020; post-COVID period included weeks 18 May 2020 to 27 June 2021. Three days were excluded from the start and end of the period due to containing incomplete weekly data. Quasi-Poisson generalised linear models (GLM) were used in the ITSA due to the presence of overdispersion in the count data. The Quasi-Poisson uses the mean regression and variance functions from the standard Poisson model but allows the dispersion parameter to be unrestricted and estimated from the data so the standard errors are scaled appropriately in the presence of overdispersion.[26] The ITSA models of the count of incidents included the time since start of study, dummy variables representing the COVID-19 and post-COVID periods and the interaction of period and time. To account for seasonal trends in the data, a Fourier term was included that allowed for regular seasonal shifts in the number of incidents.[27] Since the presence of autocorrelation and heteroskedasticity of the residuals was detected from model diagnostics, a Newey–West standard error adjustment was made to handle autocorrelation in addition to possible heteroskedasticity with the maximum lag set according to Stock and Watson's[28] rule-of-thumb. Post-estimation model diagnostics included deviance and autocorrelation graphs. The predicted count level change from the first week of the post-COVID period from the pre-COVID period (counterfactual) were performed to establish the potential impact of the preceding COVID-19 period. Predicted counts for each ITSA segment from the models were graphed against the counterfactual. Poisson tests and GLM models using the *stats* package, age standardisation using the method in the *dsrTest* package.[29] Counterfactual comparisons were performed using the *multicomp*[30] package.

The impact of living with migraine disease in Aotearoa New Zealand

Susan M Garrett, Fiona Imlach

ABSTRACT

AIM: To describe the impact of living with migraine disease in Aotearoa New Zealand.

METHODS: Online survey: Migraine in Aotearoa New Zealand Survey (MiANZ) delivered via SurveyMonkey from 22 August 2022 to 7 October 2022. Questions included: socio-demographics, the Migraine Disability Assessment Scale (MIDAS), the impact on work and open-ended questions with free text. Analysis used a mixed method approach.

RESULTS: Five hundred and thirty people from Aotearoa New Zealand (82% female; 77% NZ European/Other). Almost half of respondents had severe disability, measured by the MIDAS. Based on reported headaches days per month, 23% had chronic migraine. Significant impacts were noted on all areas of life. Themes from rich free-text data included "physical impacts", "mental health impacts", "impacts on work (paid/unpaid)", "impacts on social connection" and "impacts of trying to find a cause or cure".

CONCLUSIONS: This is the first reported survey of people with migraine in Aotearoa New Zealand and shows high levels of migraine disability. Greater awareness of the impact of migraine is needed, among the public and in workplaces, where more support and accommodation for workers with migraine could have a positive effect on productivity.

Migraine disease is a complex neurological condition diagnosed using reported symptoms and the criteria of the International Headache Society's International Classification of Headache Disorders (ICHD) (3rd edition).[1] It is estimated to affect one in seven people globally,[2] and affects two to three times as many women as men.[3] From the 2016 and 2019 Global Burden of Disease studies, migraine is the second highest cause of "years of life lived with disability" (YLD) worldwide, but the top cause of YLD among people aged 15–49 years old.[4] In Aotearoa New Zealand, it is estimated that 642,000 people have migraine.[4] Prevalence of migraine is similar for Māori, Pacific peoples and NZ Europeans (15.7%, 16.0% and 14.4% respectively).[5]

Migraine is classified as episodic or chronic, depending on attack frequency. Episodic migraine is defined as having up to 14 headache days/month. In 7–9% of people with migraine, the disease becomes chronic, where headache occurs on 15 or more days a month, for at least three months.[6,7]

Disability from migraine can be measured using tools such as the Migraine Disability Assessment Scale (MIDAS), which uses a set of questions to assess the impact of migraine on daily life. These tools demonstrate the significant impact of migraine on nearly all aspects of life, including employment, education, household work, social and family life, with people with chronic migraine experiencing the highest level of disability.[8]

Little research on the impact of migraine has been undertaken in Aotearoa New Zealand. From the Dunedin Multidisciplinary Health and Development Study in 1998–1999, participants at age 26 were asked about headaches in the last 12 months and 72 (7.3% of the sample) fulfilled the ICHD criteria for migraine. For 39% of these individuals, headache impaired their ability to work "quite a lot" to "very much" and 42% reported a similar high interference with social activities.[9] No further research on the experiences of people with migraine in Aotearoa New Zealand has been published.

To address this gap, Migraine Foundation Aotearoa New Zealand (MFANZ), a charity with the mission of raising awareness of migraine disease and supporting people living with migraine in Aotearoa New Zealand, undertook an online survey in 2022: the Migraine in Aotearoa New Zealand Survey (MiANZ). The aims of this paper were to describe the impact of migraine disease on wellbeing and daily life, using data from the MiANZ survey. Our research questions were:

- How does migraine disease affect people in Aotearoa New Zealand?
- What can be done to mitigate the impact of migraine in Aotearoa New Zealand?

Methods

Study population and recruitment

Participants included anyone with migraine living in Aotearoa New Zealand. The survey was promoted through MFANZ social media and networks, including Health Navigator (now Healthify), Neurological Foundation and New Zealand Pain Society. A link to the survey was placed on the MFANZ website and media articles publicised the survey (GP Pulse, Scoop). From an initial 579 responses, four duplicates were removed. An additional 33 responses were removed as they answered <6% of the survey and did not contribute substantively to the research questions.

The final dataset included people with either a positive Migraine Identification test (ID-Migraine test™)[10] (n=513) or who reported being diagnosed with migraine by a health professional (n=17). A positive ID-Migraine test™ (answering "yes" to at least two of three questions about migraine symptoms) has a sensitivity of 84% and specificity of 76% for migraine.[11]

Survey development, content and delivery

The survey was developed using existing questions where possible: the five question MIDAS,[12] impact on work,[13] other long-term health conditions (including anxiety and depression) and socio-demographic questions (age, gender, ethnicity, employment status) as used by Statistics NZ and Manatū Hauora – Ministry of Health (questionnaire in Appendix). A question on number of headache days per month identified people with chronic and episodic migraine—six respondents who were unsure or didn't know were classed as "episodic".

The MIDAS was scored as follows: mild disability (6–10), moderate disability (11–20) and severe disability (>20). Due to an unforeseen error in the survey, question 4 of the MIDAS was omitted, meaning that the MIDAS scores will underestimate the true disability of respondents.

The survey was delivered online via SurveyMonkey and piloted by six people, most of whom had migraine disease. The survey ran from 22 August 2022 through to 7 October 2022.

Ethical approval was granted by the University of Otago Human Ethics Committee (Ref: D23/156). Information about the survey was provided on the front page; hence, informed consent was inferred by participation in the online survey.

Data analysis

A mixed-methods approach to analysis was used. At the close of recruitment, survey data were exported into Microsoft Excel for cleaning, collation and analysis. Selected demographic data were re-coded (e.g., age-bands, gender, ethnicity). We prioritised ethnicity (Māori, Pacific peoples, Asian, NZ European/Other) for anyone who reported more than one ethnic group. For quantitative data, response frequencies were tabulated with number and percentages calculated where appropriate. Only descriptive statistics were used, due to the self-selecting nature of the sample.

Responses to each free-text question in the survey were initially coded separately, but with overlap between codes across questions; the second stage of coding involved creating themes across questions. Final coding formed a combined dataset of themes.[14] This resulted in five themes, with sub-themes in each. Quotes to demonstrate themes are accompanied by gender, age group and ethnicity of the respondent. Respondents' recommendations for what could be done to improve their situation were categorised into actions targeting society, workplaces and the health system.

Results

Participant characteristics

Table 1 presents the characteristics of the total survey sample and for those with chronic and episodic migraine. The majority were women (82%) and NZ European/Other (77%). Most were in either part-time or full-time employment (70%), with a small number of retirees and students. Almost half of the respondents met the criteria for severe disability.

Survey quantitative results

Although the majority (51.9%) of respondents had 7 days or less of headache a month, nearly a quarter of respondents (22.6%) had chronic migraine, experiencing headache on 15 or more days a month. Of those with chronic migraine, 20% had continuous or nearly continuous headache (4.5% of all respondents) and another 22% had 24 or more days of headache per month (4.9% of all respondents).

Respondents were asked questions (as per the MIDAS) about how often migraine impacted their everyday life. Figure 1 shows that half of respondents hadn't been able to do household work, nearly a third had missed family, social or leisure activities

Table 1: Socio-demographic characteristics of the survey sample by episodic and chronic migraine type.

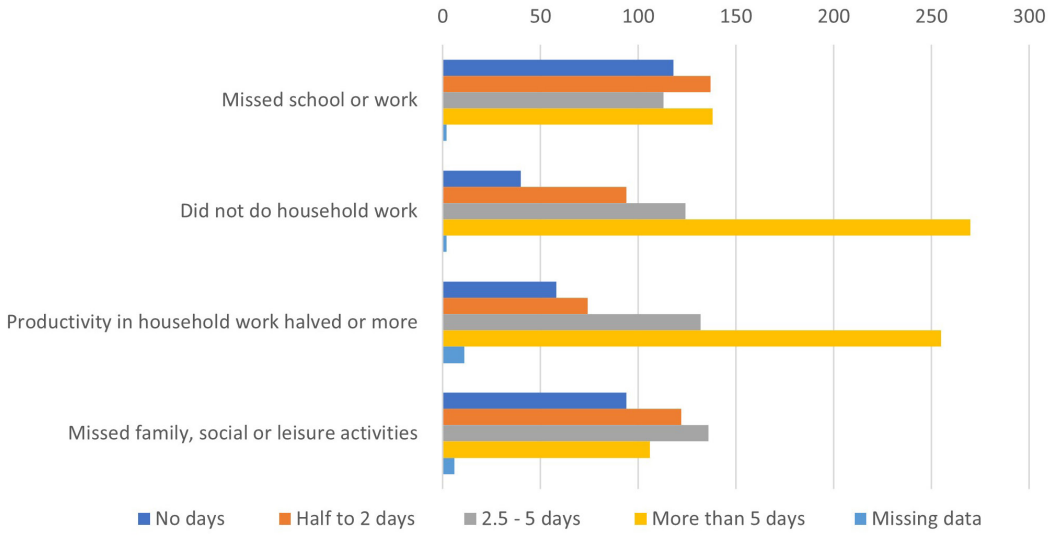
Characteristic	Migraine type					
	Chronic n=118 (22.2%)		Episodic n=412 (77.7%)		Total n=530	
Age-band	**N**	**Col %**	**N**	**Col %**	**N**	**Col %**
<18 years	1	0.8	1	0.2	2	0.4
18–24 years	5	4.2	15	3.6	20	3.8
25–34 years	16	13.6	64	15.5	80	15.1
35–44 years	30	25.4	93	22.6	123	23.2
45–54 years	35	29.7	120	29.1	155	29.2
55–64 years	15	12.7	55	13.3	70	13.2
65+ years	7	5.9	24	5.8	31	5.8
Missing data	9	7.6	40	9.7	49	9.2
Gender						
Female	96	81.4	337	81.8	433	81.7
Male	10	8.5	31	7.5	41	7.7
Another gender[1]	3	2.5	5	1.2	8	1.5
Missing data	9	7.6	39	9.5	48	9.1
Ethnic group						
Māori	7	5.9	32	7.8	39	7.4
Pacific peoples	0	0.0	6	1.5	6	1.1
Asian	2	1.7	21	5.1	23	4.3
NZ European/Other	99	83.9	310	75.2	409	77.2
Missing data	10	8.5	43	10.4	53	10.0
MIDAS Disability Score						
0–5 (little or no)	1	0.8	74	18.0	75	14.2
6–10 (mild)	3	2.5	74	18.0	77	14.5
11–20 (moderate)	11	9.3	105	25.5	116	21.9
>21 (severe)	103	87.3	159	38.6	262	49.4
Self-reported mental health						
Anxiety	50	42.3	120	29.1	170	32.0
Depression	34	28.8	91	22.0	125	23.6

Table 1 (continued): Socio-demographic characteristics of the survey sample by episodic and chronic migraine type.

Employment/education						
Student	1	0.8	13	3.2	14	2.6
Stay at home carer	8	6.8	14	3.4	22	4.2
Retired	6	5.1	22	5.3	28	5.3
Not employed, not looking for work	16	13.6	18	4.4	34	6.4
Not employed/looking for work	6	5.1	4	1.0	10	1.9
Employed part-time	30	25.4	94	22.8	124	23.4
Employed full-time	42	35.6	208	50.5	250	47.2
Missing data	9	7.6	39	9.5	48	9.1

[1]3/8 people who responded with "another gender" indicated their gender: Non-binary, Pansexual, Gender queer (AFAB)

Figure 1: Days of activity missed in the last 3 months because of headaches.



and over a quarter had missed school or work on more than five days in the last three months.

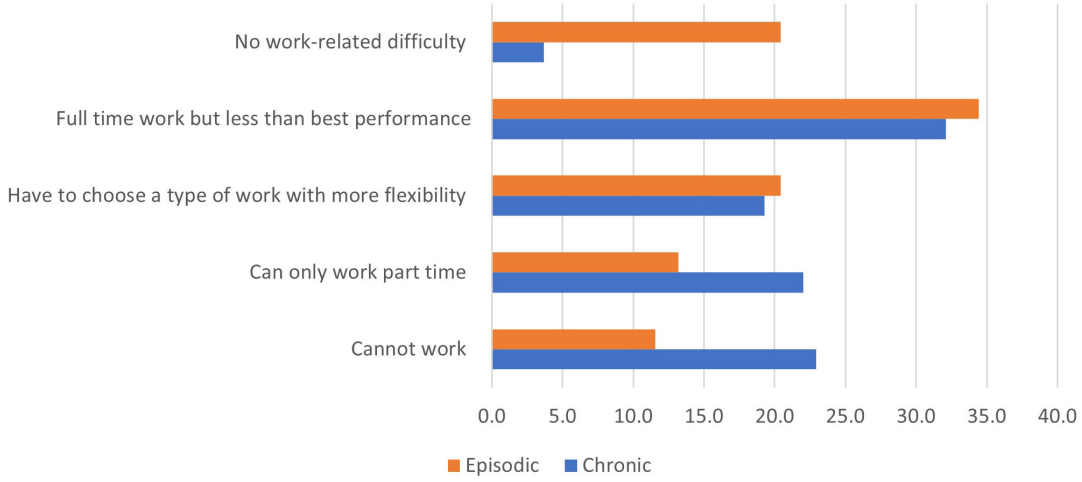
Respondents were asked to indicate the impact of migraine on work (Figure 2). Very few of those with chronic migraine reported no impact. The impact of episodic migraine on work was less, although over a third were working full-time but at less than their best performance. This is consistent with the higher rates of unemployment and lower rates of full-time employment reported by those with chronic migraine in Table 1.

Survey qualitative results

Free-text data came from three open-ended questions: "What could be done to improve your life with migraine?", "Is there anything else you want to tell us about living with migraine in New Zealand?" and a free-text question at the end of the MIDAS. These questions were answered by 437, 254 and 67 respondents respectively.

Themes were identified under the following headings: "physical impacts", "mental health impacts", "impacts on work (paid/unpaid)", "impacts on social connection" and "impacts of trying to find a cause or cure".

Figure 2: Impact of migraine on work, proportion by episodic and chronic migraine type.



Physical impacts

Respondents spoke of significant levels of pain, which could last for hours or days. Pain was likened to no other, prompting thoughts of extreme measures to seek relief.

> *"People don't understand the excruciating pain and think 'it's just a headache'. I've pondered if I could just cut my own head off to make it stop." – 45–54 years, Female, NZ European/Other, 668*

> *"Recently I broke my elbow. That was very painful. The migraines I experience are significantly more painful than breaking my elbow. Migraines are also significantly more disruptive to my life than not being able to use one of my arms. If I could cut off one of my arms and never experience migraines again I would do it in a heartbeat." – 35–44 years, Male, NZ European/Other, 149*

Migraine attacks include other physical symptoms, which can be as debilitating and distressing as the headache. Respondents reported loss of coherent speech, paralysis on one side of the body, auras (visual disturbance and even loss of vision), pins and needles, sensory sensitivity (photophobia, phonophobia) and nausea with or without vomiting.

> *"My symptoms can sometimes make me look like I've had a stroke because I can't talk or walk properly, with pain so severe I am beyond crying." – 45–54 years, Male, NZ European/Other, 355*

Mental health impacts

The effect of migraine on everyday activities could lead to anxiety and depression, with words such as "misery", "devastating" and "ruining life" used to describe the impact. Going without a migraine attack for short periods could invoke feelings of happiness or relief.

> *"It sucks. It ruins my life. It changes plans, disrupts routine, creates stress. It makes me miserable." – 25–34 years, Female, Asian, 473*

Constant worry about when the next migraine attack was going to arise and how that would impact planned events could induce high levels of anxiety. Participants used combat terms such as "threat", "attack" and "live in fear", giving migraine disease an enemy-like persona, something to be battled against, or to be fearful of, making living with migraine possibly akin to "shell shock" or post-traumatic stress disorder.

> *"I think sometimes the fear of a migraine is the worst … I have no one to call to drive me to the doctor for an injection when I am really ill, and I end up in bed all day in extreme pain … as a single mother it makes me feel very vulnerable." – 45–54 years, Female, NZ European/Other, 097*

Those living with chronic migraine describe only living half a life, feeling isolated, trapped, lonely and useless.

> *"The toll migraine headaches take on a person's mental health is devastating and severely overlooked. You feel useless and worthless because you can't provide." – 35–44 years, Female, Māori, 461*

Impacts on work (paid and unpaid)

Many people noted their inability to work or study, or reduced ability to work due to migraines: "unable to work", "I don't work or study at the moment", "not able to work because of them", "I don't work now because of my migraines", "haven't worked for a year, too many migraines", "I had to quit my job 2 years ago". As well as the obvious reasons for not being able to work (frequency of migraines and needing to lie in bed for days), the negative impact of migraine on cognition and fatigue also reduced people's ability to function productively at work.

> *"Don't work. Migraines interfere with cognitive function almost daily." – 45–54 years, Female, NZ European/Other, 116*

Those who did work spoke about being "lucky" to have flexible work schedules, reducing work hours to accommodate migraine or choosing work where regular attendance was not required. A cycle was noted of pain limiting the ability to work, trying to keep working despite impairment and then using any spare time to catch up and recover. Some people relied on acute medication to continue working.

> *"I have so much pain regularly and I still have to show up and drug up. I was in a senior executive job and would push through until the times I couldn't and then I would be in bed for 3 days in the dark." – 45–54 years, Female, NZ European/Other, 599*

As well as impacting paid work, people talked about the effects on household tasks, parenting and being a responsible partner. They worried about the impact on other family members, with partners having to do extra and children being affected.

> *"I have had to give up my job. I can no longer drive. I don't cook. I often can't bathe. I rarely do any housework. I'm dependent on my partner for everything." – 45–54 years, Female, NZ European/Other, 963*

> *"I can't imagine how single parents with migraines cope!! It affects the state of one's house. It's just impossible to keep up with housework. It restricts your children's lives." – 45–54 years, Female, NZ European/Other, 116*

Impacts on social connection and time

A common theme was respondents' lack of ability to commit to social events and feeling like they were letting people down when migraine attacks kept them at home.

> *"I don't tend to make plans to do things because I can hardly ever follow through." – 25–34 years, Another gender, NZ European/Other, 759*

The concept of migraine "stealing life away" was common. People talked of "lost days to migraines" and time you can never get back. The days lost to migraine attacks were also a hindrance to social contact, as good days were spent catching up on work or activities that were unable to be completed on migraine days. Avoiding triggers (lack of sleep, alcohol, places with bright lights, noise or strong smells) interfered with the ability to attend social occasions when these triggers were an integral part of socialising.

> *"I rarely socialise because if I don't have a migraine, I'm catching up on all the things I've neglected when I've been experiencing one…" – 45–54 years, Female, NZ European/Other, 116*

Impact of trying to find a "cause" or "cure"

People spent a significant amount of effort, time, research and money trying to find a cure, or something that would reduce the frequency or intensity of migraine attacks. They struggled with potential triggers, which could be overwhelming and often fruitless, when triggers could not be consistently identified or avoided. The time and financial investment into activities and therapies to "get better" or at least maintain a level of wellbeing could be prohibitive and restrictive.

> *"It's expensive—privately funding*

Table 2: Recommendations for supporting people with migraine.

Recommendations for supporting people with migraine		Supporting quote or information
Societal	More understanding, education and awareness for the general public	*"More awareness in how debilitating it is; you can't just drink more water, rest, etc. It lasts days and it is not just the pain, though that can be very bad, [it's] that other symptoms come with it."* *"I feel like I live with a made-up illness, constantly having to over-explain myself and try to convince people that it's a real condition."*
	Recognition of migraine as a significant, complex neurological condition causing disability	*"People do not understand and think it's just a headache. It is not seen as a serious neurological disease."* *"If it were considered a disability, I feel like I would be treated differently."*
	Better support for people who can't work full-time or at all because of migraine (e.g., a benefit for people with a health condition or disability)	*"I'm unable to work, yet don't qualify for a sickness benefit."* *"Disability financial support (with a working partner I am ineligible for any support), as migraines are so debilitating that I feel incapable of working."*
Workplace	Better sick and annual leave entitlements	*"Ability to have sick days when needed without guilt."* *"Migraine leave for work. It takes a few days for me to get back to normal."*
	Accommodation for people with migraine to manage their environment to avoid triggers.	*"Natural light in workspaces. Migraine-specific occupational health workplace assessments of computers and workstations/lighting."* *"Working with my workplace to reduce triggers such as exhaust fumes."*
	More understanding and awareness of migraine disease in workplaces	*"Workplaces see migraines as simply a headache and mine tends to put pressure on me to work despite my migraines being completely debilitating."* *"Employer … insisted I get a medical certificate any time I had one. Despite the cost and difficulty to do so while experiencing a migraine."*
	Provide flexible work and education options	*"I work from home so that removes the biggest issue, as I can just sleep when I have a migraine and work flexibly."* *"Having flexible teachers who understand how debilitating they are and help with setting work to catch up at home."*
Health system	Access to more treatment options	*"Get new drugs that mean people can work … We would rather work and pay taxes and pay back the cost of those drugs than be sick and costing via healthcare, welfare and more."*

CGRP meds [calcitonin gene-related peptide, new medication not currently funded in New Zealand] or having to give up work ... Plus physio, psychologist, supplements." – 45–54 years, Female, NZ European/Other, 823

"I had a decade of experimenting to find food triggers that were never identified. In the end I gave up, figuring why add misery to pain." – 55–64 years, Male, Māori, 706

How to improve life for people with migraine

Survey respondents were asked to comment on "What could be done to improve your life with migraine?". Responses were grouped into suggestions for what would help at a societal level, in workplaces and the health system (Table 2).

Discussion

People living with migraine in Aotearoa New Zealand reported significant levels of migraine disability and impacts on all areas of life. Impacts were particularly pronounced for people with chronic migraine who experienced higher levels of disability and more restrictions on employment. For all respondents, impacts on household chores, family, social or leisure activities and work/study were pronounced.

From the qualitative data, impact went beyond dealing with the physical pain and other symptoms associated with migraine attacks, and affected mental health, social connections and ability to work. Other impacts included the financial and time burden of researching and accessing treatments and therapists who might offer a "cure" or at least help with minimising the intensity or frequency of migraine attacks.

The themes identified in this study are similar to those found in a systematic review of psychosocial difficulties of people with migraine,[15] which included general physical and mental health, pain, fatigue, emotional problems, difficulties at work, social functioning and global disability.

People with migraine commonly state that they would be better partners and parents without migraine and report a reduced ability to do household chores, missed family or social activities and negative impacts on parenting.[16,17] Studies consistently show that migraine affects family, partners and children of people with the disease.[18] These impacts were also noted by our survey respondents.

Research from a large longitudinal survey of people with migraine in the US found that those with chronic migraine were less likely to be in paid employment than those with 3 or fewer headache days/month and who missed more hours of work per week.[19] Many other studies report marked productivity impacts for people with migraine, which are worse for those with chronic migraine.[20,21]

The impact of migraine on mental health is complex. Depression and anxiety have bidirectional relationships with migraine disease, and both are at least twice as common in people with migraine than the general population,[22,23] consistent with our findings. Comorbid mood disorders are associated with reduced quality of life and increased disability in people with migraine and can also increase the risk of migraine becoming chronic.[24]

However, the negative impact of migraine on mental health extends beyond comorbid mental health diagnoses. The anticipatory anxiety surrounding migraine, where people fear attacks due to their unpredictable nature, is common and can cause significant distress.[25] Isolation, guilt and feelings of hopelessness are other emotions described in qualitative research on the impact of migraine[18] and in our population.

Searching for a cause or a cure was also noted in our analysis. People with migraine want to understand what triggers their attacks, so they can avoid triggers and reduce their attacks. However, the evolving science around migraine triggers and the migraine prodrome now suggests that many factors that have previously been considered triggers (diet, emotions, environmental factors) are much more likely to be early symptoms of an impending migraine attack.[26] An excessive preoccupation with potential trigger avoidance can reduce quality of life and provoke anxiety in people with migraine[18] and is unlikely to be effective.[26]

This is one of the first studies to gather data about the experience of migraine in Aotearoa New Zealand. However, this is not a representative sample of people with migraine in Aotearoa New Zealand, but a self-selected, opportunistic sample recruited largely through social media and online channels, likely not frequented by high numbers of Māori or other ethnic minorities and with more severe disease than the general migraine population (e.g., 23% of respondents had chronic migraine compared to a population rate of approximately 8%).[24]

A broad range of actions is needed to reduce the impact of migraine disease in Aotearoa New Zealand, starting with a wider societal understanding that migraine is a complex neurological condition. Poor understanding of migraine leads to stigma and exacerbates migraine-related disability.[27]

For people with migraine who are unable to work at their full capacity, more supportive work environments could help reduce work-related stress and improve productivity. For people who are unable to work at all, recognising migraine as a disability could allow more people to access benefits and contributions towards treatment costs.

Further research is needed to understand the cost-effectiveness and other implications of some of the recommendations listed in Table 2, and to explore in detail medication use and related overuse issues.

Conclusions

Migraine is a complex, disabling neurological condition with wide-ranging physical and mental health impacts, as well as negative effects on work, social and family life. From this survey of people with migraine in Aotearoa New Zealand, levels of migraine disability were high, with the majority experiencing some limitation in work, social and family life. Greater support and awareness of migraine as more than "just a headache" is needed, both among the public and in workplaces.

COMPETING INTERESTS
The authors have no conflicts of interest to declare. Data are not publicly available. Study participants were not asked for permission, and nor was ethical approval granted for data to be shared publicly.

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REFERENCES
1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38:1-211. doi: 10.1177/0333102417738202.
2. Stovner LJ, Hagen K, Linde M, Steiner TJ. The global prevalence of headache: an update, with analysis of the influences of methodological factors on prevalence estimates. J Headache Pain. 2022;23(1):34. doi: 10.1186/s10194-022-01402-2.
3. Ahmad SR, Rosendale N. Sex and Gender Considerations in Episodic Migraine. Curr Pain Headache Rep. 2022;26:505-516. doi: 10.1007/S11916-022-01052-8.
4. Global Burden of Disease Collaborative Network. Global Burden of Disease Study 2019 (GBD 2019) Results [Internet]. Seattle, WA (US): Institute for Health Metrics and Evaluation; 2020 [cited 2022 Nov 3]. Available from: https://vizhub.healthdata.org/gbd-results/.
5. Migraine Foundation Aotearoa New Zealand. New Zealand health survey data – a decade to be released [Internet]. NZ: Migraine Foundation Aotearoa New Zealand; 2022 [cited 2023 Nov 11]. https://www.migrainefoundation.org.nz/blog/post/93215/new-zealand-health-survey-data--a-decade-to-be-released/
6. Adams AM, Serrano D, Buse DC, et al. The impact of chronic migraine: The Chronic Migraine Epidemiology and Outcomes (CaMEO) Study methods and baseline results. Cephalalgia. 2015;35(7):563-78. doi: 10.1177/0333102414552532.
7. May A, Schulte LH. Chronic migraine: risk factors, mechanisms and treatment. Nat Rev Neurol. 2016;12(8):455-464. doi: 10.1038/nrneurol.2016.93.
8. Burch RC, Buse DC, Lipton RB. Migraine: Epidemiology, Burden, and Comorbidity. Neurol Clin. 2019;37(4):631-649. doi: 10.1016/j.ncl.2019.06.001.
9. Waldie KE, Poulton R. The burden of illness associated with headache disorders among young adults in a representative cohort study. Headache. 2002;42(7):612-9. doi: 10.1046/j.1526-4610.2002.02148.x.
10. Lipton RB, Dodick D, Sadovsky R, et al. ID Migraine validation study. A self-administered screener for migraine in primary care: The ID Migraine validation study. Neurology. 2003;61(3):375-82. doi: 10.1212/01.wnl.0000078940.53438.83.
11. Cousins G, Hijazze S, Van de Laar FA, Fahey T. Diagnostic accuracy of the ID Migraine: a systematic review and meta-analysis. Headache. 2011;51(7):1140-8. doi: 10.1111/j.1526-4610.2011.01916.x.
12. Stewart WF, Lipton RB, Kolodner K. Migraine disability assessment (MIDAS) score: relation to headache frequency, pain intensity, and headache symptoms. Headache. 2003;43(3):258-65. doi: 10.1046/j.1526-4610.2003.03050.x.
13. Young WB, Park JE, Tian IX, Kempner J. The stigma of migraine. PLoS One. 2013;8(1):e0054074. doi: 10.1371/journal.pone.0054074.
14. Braun V, Clarke V, Boulton E, et al. The online survey as a qualitative research tool. Int J Soc Res Methodol. 2021;24(6):641-654. doi: 10.1080/13645579.2020.1805550.
15. Raggi A, Giovannetti AM, Quintas R, et al. A systematic review of the psychosocial difficulties relevant to patients with migraine. J Headache Pain. 2012;13(8):595-606. doi: 10.1007/s10194-012-0482-1.
16. Lipton RB, Bigal ME, Kolodner K, et al. The family impact of migraine: population-based studies in the USA and UK. Cephalalgia. 2003;23(6):429-440. doi: 10.1046/j.1468-2982.2003.00543.x

17. Lipton RB, Buse DC, Adams AM, et al. Family Impact of Migraine: Development of the Impact of Migraine on Partners and Adolescent Children (IMPAC) Scale. Headache. 2017;57(4):570-585. doi: 10.1111/head.13028.
18. Estave PM, Beeghly S, Anderson R, et al. Learning the full impact of migraine through patient voices: A qualitative study. Headache. 2021;61(7):1004-1020. doi: 10.1111/head.14151.
19. Stewart WF, Wood GC, Manack A, et al. Employment and work impact of chronic migraine and episodic migraine. J Occup Environ Med. 2010;52(1):8-14. doi: 10.1097/JOM.0b013e3181c1dc56.
20. Gerth WC, Carides GW, Dasbach EJ, et al. The multinational impact of migraine symptoms on healthcare utilisation and work loss. Pharmacoeconomics. 2001;19(2):197-206. doi: 10.2165/00019053-200119020-00006.
21. Steiner TJ, Stovner LJ, Jensen R, et al. Lifting the Burden: The Global Campaign against Headache. Migraine remains second among the world's causes of disability, and first among young women: findings from GBD2019. J Headache Pain. 2020;21(1):137. doi: 10.1186/s10194-020-01208-0.
22. Karimi L, Wijeratne T, Crewther SG, et al. The Migraine-Anxiety Comorbidity Among Migraineurs: A Systematic Review. Front Neurol. 2021;11:613372. doi: 10.3389/fneur.2020.613372.
23. Lampl C, Thomas H, Tassorelli C, et al. Headache, depression and anxiety: associations in the Eurolight project. J Headache Pain. 2016;17:59. doi: 10.1186/s10194-016-0649-2.
24. May A, Schulte LH. Chronic migraine: risk factors, mechanisms and treatment. Nat Rev Neurol. 2016;12(8):455-64. doi: 10.1038/nrneurol.2016.93.
25. Peres MFP, Mercante JPP, Tobo PR, et al. Anxiety and depression symptoms and migraine: a symptom-based approach research. J Headache Pain. 2017;18(1):37. doi: 10.1186/s10194-017-0742-1.
26. Martinelli D, Pocora MM, De Icco R, et al. Triggers of migraine: where do we stand? Curr Opin Neurol. 2022;35(3):360-366. doi: 10.1097/WCO.0000000000001065.
27. Parikh SK, Young WB. Migraine: Stigma in Society. Curr Pain Headache Rep. 2019;23(1):8. doi: 10.1007/s11916-019-0743-7.

Appendix 1: Migraine in Aotearoa New Zealand survey

Migraine in Aotearoa New Zealand

Survey information

This survey is to understand the burden and impact of migraine in Aotearoa New Zealand. It includes questions about treatments you've tried, health services you've used and any issues or challenges living with migraine has on your professional and personal life.

We are seeking participants who currently live in New Zealand who have been diagnosed with migraine or have symptoms that are consistent with migraine disease. These include:

- pain on one side of the head
- pain that lasts 4 hours to 3 days if not treated
- throbbing or pulsing pain, usually moderate to severe and often worse with routine activity such as walking or climbing stairs
- sensitivity to light, sound and/or smell
- nausea and vomiting.

This survey is being run by Migraine Foundation Aotearoa New Zealand. Migraine Foundation Aotearoa New Zealand is the only registered charity in New Zealand supporting people living with migraine. Our mission is to raise awareness of the impact of migraine disease and support people living with migraine in Aotearoa New Zealand.

All responses are anonymous and remain confidential.

The survey will take around 20 minutes to complete.

Migraine identification

Do you have migraine?

These questions help identify people who have migraine disease.

1. Have you had a headache in the last 3 months?

- Yes
- No
- Don't know

2. Has a headache limited your activities for a day or more in the last 3 months? (Activities includes work, study, play or other things you need to do in the day)

- Yes
- No
- Don't know

3. Are you nauseated or sick to your stomach when you have a headache?

- Yes
- No
- Don't know

4. Does light bother you when you have a headache?

- Yes
- No
- Don't know

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

About your migraine disease

5. How old were you when you had your first migraine attack?

- 0–9 years
- 10–14 years
- 15–19 years
- 20–24 years
- 25–29 years
- 30–39 years
- 40–49 years
- 50–59 years
- 60 or older
- Don't know/unsure

6. How old were you when you were diagnosed with migraine by a health professional?

- 0–9 years
- 10–14 years
- 15–19 years
- 20–24 years
- 25–29 years
- 30–39 years
- 40–49 years
- 50–59 years
- 60 years or older
- Don't know/unsure
- Not diagnosed by a health professional

7. Does anyone else in your family have migraine?

- Yes
- No
- Don't know/unsure

Migraine disability assessment scale

These questions help measure the impact of headaches on your life. They are based on an international set of questions that have been tested and used in migraine drug trials.

8. On average, on how many days a month do you have a headache? (If a headache lasted more than 1 day, count each day.)

- 0–7 days per month
- 8–14 days per month
- 15–23 days per month
- >/=24 days per month
- Continuous/nearly continuous (essentially no headache-free time)
- Don't know/unsure

9. On average, how painful were these headaches on a scale of 0–10? (where 0=no pain at all, and 10=pain as bad as it can be.)

0 5 10

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

Please answer the following questions about ALL of the headaches you have had over the last 3 months. Select zero if you did not have the activity in the last 3 months.

It can be hard to remember what happened in the last 3 months, so your best guess is fine.

10. On how many days in the last 3 months did you miss work or school because of your headaches?
11. On how many days in the last 3 months did you not do household work (such as housework, home repairs and maintenance, shopping, caring for children and relatives) because of your headaches?
12. How many days in the last 3 months was your productivity in household work reduced by half or more because of your headaches? (Do not include days you counted in question 3 where you did not do household work.)
13. On how many days in the last 3 months did you miss family, social or leisure activities because of your headaches?

The total MIDAS score can be used to define four grades of migraine-related disability with grade I for "little or no disability" (0–5); grade II for "mild disability" (6–10); grade III for "moderate disability" (11–20); and grade IV for "severe disability" (≥21).

Note: one question was missed in the survey:

How many days in the last 3 months was your productivity at work or school reduced by half or more because of your headaches? (Do not include days you counted in question 1 where you missed work or school.)

Self-rated health

14. In general, would you say your health is:

- Excellent
- Very good
- Good
- Fair
- Poor

Acute treatments

This section asks about what treatments you use when you get a migraine attack.

15. Do you or have you used paracetamol to treat your migraine attacks?

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

16. On how many days in the last month have you used paracetamol for a migraine attack?
    0 30
17. Do you or have you used non-steroidal anti-inflammatories (NSAIDs) to treat your migraine attacks?
    e.g., Aspirin, Ibuprofen (Nurofen, Brufen, Advil), diclofenac (Voltaren), naproxen (Naprosyn, Naprogesic, Noflam), celecoxib (Celebrex), meloxicam (Mobic)—including tablets that combine NSAIDs with paracetamol.

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

18. On how many days in the last month have you used NSAIDs for a migraine attack?
    0 30
19. Do you or have you used sumatriptan (Imigran, Imitrex) to treat your migraine attacks?

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

20. On how many days in the last month have you used sumatriptan for a migraine attack?
    0 30
21. Do you or have you used rizatriptan (Maxalt, Rizamelt) to treat your migraine attacks?

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

22. On how many days in the last month have you used rizatriptan for a migraine attack?
    0 30
23. Do you or have you used opioids to treat your migraine attacks? e.g., tramadol (Tramal), codeine (including combined with paracetamol in Panadeine or ibuprofen in Nurofen Plus), Oxycodone.

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

24. On how many days in the last month have you used opioids for a migraine attack?
    0 30
25. Do you or have you used anti-emetics (anti-nausea medications) to treat your migraine attacks? e.g., metoclopramide (Maxolon), ondansetron, prochlorperazine (Stemetil, Buccastem)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

26. Which of the following non-medication treatments have or do you use to treat your migraine attacks?

- Caffeine
- Occipital nerve block

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

- Neurostimulation device e.g., TENS machine
- Ginger e.g., tablets, tea
- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

Preventive treatment

There are many medicines that can be taken to prevent migraine attacks. This section asks whether you have or would like to try preventive medicines and why you might have stopped taking them.

27. Which of the following anti-depressants have you used to prevent migraine attacks?

- Amitriptyline (Amirol)
- Nortriptyline (Norpress)
- Venlafaxine (Effexor)
- Fluoxetine (Prozac)
- Other (please specify)

Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

28. Which of the following anti-epileptic medications have you used to prevent migraine attacks?

- Topiramate (Topamax)
- Sodium valproate (Epilim)
- Gabapentin (Neurontin)
- Lamotrigine (Lamictal)
- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

29. Which of the following anti-hypertensive or cardiac medications have you used to prevent migraine attacks?

- Nadolol (Corgard)
- Metoprolol (Lopressor)
- Propranolol (Inderal)
- Verapamil (Isoptin)
- Candesartan (Candesar)
- Lisinopril (Zestril)

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

30. Which of the following migraine-specific medications have you used to prevent migraine attacks?

- Pizotifen (Sandomigran)
- Erenumab (Aimovig)
- Galcanezumab (Emgality)
- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

31. Which of the following hormone treatments have you used to prevent migraine attacks?

- Melatonin
- Estrogen, with or without progesterone e.g., hormone replacement therapy, combined oral contraceptive pill
- Progesterone on its own e.g., progesterone-only oral contraceptive, depot provera, progestin implant or intrauterine device/IUD
- Testosterone
- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

32. Which of the following supplements have you used to prevent migraine attacks?

- Magnesium
- Riboflavin (vitamin B2)
- Coenzyme Q10
- Feverfew
- Ginger
- Butterbur
- Other (please specify)

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

33. Which of the following injections have you used to prevent migraine attacks?

- Botulinum toxin A (Botox) injections
- Occipital nerve block
- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

34. Which of the following non-medication approaches have you used to prevent migraine attacks?

- Neurostimulation device e.g., TENS machine
- Meditation or mindfulness practice
- Yoga or tai chi
- Biofeedback
- Acupuncture
- Massage
- Cold therapy e.g., ice packs, cold baths
- Other (please specify)

Currently use	Previously used—stopped because of side effects	Previously used—stopped because it didn't work	Previously used—stopped for another reason	Never used—would like to try	Never used—don't want to try

35. Aimovig, Emgality, Ajovy and Vyepti are a new class of migraine prevention medication developed specifically to target migraine (calcitonin gene-related peptide or CGRP monoclonal antibodies). They have fewer side effects than most other preventive medications. Only Aimovig and Emgality are currently available in New Zealand.
If you have ever tried one of these, please tell us about your experience.
If you haven't, please tell us why you would or wouldn't try one in the future.

Healthcare use

This section asks about health professionals you have seen to help your management of migraine disease.

36. Which of the following health professionals have you seen about migraine?

- Primary care/GP
- Neurologist
- Emergency department or urgent care physician
- Osteopath

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

- Chiropractor
- Pain specialist
- Physiotherapist
- Nutritionist/dietitian
- Occupational therapist
- Dentist
- Pharmacist
- Acupuncturist
- Naturopath
- Massage therapist
- Optician or eye specialist
- Other (please specify)

Seen in the last 12 months	Seen in the past (>12 months ago)	Never seen—would like to	Never seen—don't want to

37. How would you rate the knowledge of migraine and treatment options in the health professionals you have seen? (If you have seen more than one, rate the one you have seen most recently)

- Primary care/GP
- Neurologist
- Emergency department or urgent care physician
- Osteopath
- Chiropractor
- Pain specialist
- Physiotherapist
- Nutritionist/dietitian
- Occupational therapist
- Dentist
- Pharmacist
- Acupuncturist
- Naturopath
- Massage therapist
- Optician or eye specialist
- Other (please specify)

Excellent	Very good	Good	Fair	Poor	Not applicable/ haven't seen

38. Have you ever wanted to see a health professional for migraine but were unable to?

- Yes
- No

39. Which health professional(s) were you unable to see for migraine?
40. Why were you unable to see a health professional for migraine? (multiple responses allowed)

- It was too expensive
- Waiting time to be seen was too long
- Unable to get or was declined an appointment
- Service not available where I live
- Had no transport to get there

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

- Difficult to take time off work
- Could not arrange childcare or care for a dependent
- Other (please specify)

41. What could be done to improve your life with migraine?

Co-morbidities

The next question is about long-term health conditions. A long-term health condition is a physical or mental illness or condition that has lasted, or is expected to last, for more than six months. The symptoms may come and go or be present all the time.

42. Which, if any, of the following long-term conditions have you been diagnosed with and currently have (in addition to migraine)? Please select all that apply

- Anxiety
- Arthritis
- Asthma
- Depression
- Epilepsy
- Fibromyalgia
- Heart disease
- Hypertension/high blood pressure
- Insomnia
- Irritable bowel syndrome
- Low back pain
- Stroke
- I do not currently have any other long-term health conditions
- Other (please specify)

Stigma

43. How often do you hide or minimise migraine symptoms for fear of being judged or misunderstood?

- Always
- Often
- Sometimes
- Rarely
- Never

44. How often do you feel judged or misunderstood because of your migraine disease by your:

- Spouse or partner
- Family
- Friends
- Workplace
- School/place of education or training
- Health professional
- Other (please specify)

Always	Often	Sometimes	Rarely	Never	Not applicable/ don't know

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

45. Is there anything else you want to tell us about living with migraine in New Zealand?

Demographics

The final questions are about you.

46. How old are you?
47. What is your gender?

- Male
- Female
- Another gender
  Please specify

48. Which ethnic group or groups do you belong to?

- New Zealand European
- Māori
- Samoan
- Cook Island Māori
- Tongan
- Niuean
- Chinese
- Indian
- Don't know
- Refused
- Other (please specify)

49. Where do you live?

- Northland
- Auckland
- Waikato
- Bay of Plenty
- Gisborne
- Hawke's Bay
- Taranaki
- Manawatū-Whanganui
- Wellington
- Tasman
- Nelson
- Marlborough
- West Coast
- Canterbury
- Otago
- Southland
- Other (please specify)

50. What is your current employment status?

- Employed full-time
- Employed part-time
- Retired
- Student
- Stay at home carer (e.g., of children, parents)

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

- Not employed, looking for work
- Not employed, not looking for work

51. What is the impact of migraine on your ability to work? (if you are not currently working, imagine trying to work with your current migraine condition)

- Cannot work
- Can only work part time
- Have had to choose a type of work with more flexibility
- Full-time work but less than best performance
- No work-related difficulties

52. In the last 12 months, what are all the ways that you yourself got income? Please do not count loans, including student loans

- Wages, salaries, commissions, bonuses etc, paid by an employer
- Self-employment, or business you own and work in
- Interest, dividends, rent, other investments
- Regular payments from ACC or a private work accident insurer
- NZ Superannuation or Veteran's Pension
- Other superannuation, pensions, annuities (other than NZ Superannuation, Veteran's Pension or War Pension)
- Jobseeker Support
- Sole Parent Support
- Supported Living Payment
- Student allowance
- Other government benefits, government income support payments, war pensions, or paid parental leave
- Other sources of income
- No source of income during that time
- Don't know

53. What is the total income that your household got from all sources, before tax or anything was taken out of it, in the last 12 months?

- Zero income or loss
- $1–$20,000
- $20,001–$30,000
- $30,001–$50,000
- $50,001–$70,000
- $70,001–$100,000
- $100,001 or more
- Don't know

54. Do you have health or medical insurance?

- Yes
- No
- Don't know/unsure

Have more to say?

Migraine is under-recognised in every way—in funding, research, diagnosis, treatment and understanding. Telling your story about living with migraine sheds light on this disease, reduces stigma, raises awareness and helps with advocacy.

Appendix 1 (continued): Migraine in Aotearoa New Zealand survey.

Question title

55. If you would like to find out more about telling your story, please leave your contact details and we will get in touch with you. These details will be kept separate from your survey responses and will not be shared beyond Migraine Foundation Aotearoa New Zealand.

Name
Email

Thanks for taking part in our survey!

We will use your responses to advocate for better treatment and support for people with migraine in New Zealand.

Please forward the survey on to other people you know with migraine who would like to contribute (this is the link: https://www.surveymonkey.com/r/XNSTFM5)

For more information about migraine in New Zealand, visit our website https://www.migrainefoundation.org.nz/

For questions or feedback about the survey, please email info@migrainefoundation.org.nz

Can imaging determine if a rotator cuff tear is traumatic?

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ABSTRACT

AIM: We reviewed the last decade of literature to update a previous publication on this topic by the senior author. In New Zealand, traumatic causation has implications for entitlement for treatment though the Accident Compensation Corporation (ACC). Acuity and chronicity may also be relevant in determining repairability.

METHODS: Literature was reviewed regarding acromial morphology, greater tuberosity (GT) cysts, acromiohumeral interval (AHI), fatty degeneration and atrophy, acromioclavicular (AC) arthrosis, tendinopathy, bursal changes and other features.

RESULTS: Some factors can be considered normal for those middle aged and older, including AC arthrosis, type 1 and 2 acromion and tendinopathy. Some factors may indicate acuity, including haemorrhage and debris, GT oedema, mid-substance soft tissue tear, kinking of the tendon and isolated complete subscapularis tears. Other factors may be associated with chronicity, including significant fatty degeneration, positive tangent sign for atrophy, anterior GT cysts, type 3 acromion, critical shoulder angle (CSA) >35 degrees and acromial index (AI) <0.7.

CONCLUSION: A multitude of factors on imaging may infer, to a varying degree, the likelihood of acuity or chronicity. The patient history is also of importance in determining causation.

In 2010, we published a paper with the same title in the *New Zealand Medical Journal*.[1] The impetus for that review was the importance of this topic in defining entitlement for Accident Compensation Corporation (ACC) funded treatment in New Zealand. The 2010 publication has been used and cited in medicolegal reviews and guidelines including the ACC document *Rotator Cuff Tears: Consideration Factors for ACC Cover*.[2]

In this paper we review the last decade of literature on this topic and update the conclusions of our 2010 paper. We performed an online search of PubMed, Embase, Medline and the Cochrane Library between 2011 and 2021. Each of the radiographic categories were searched with "imaging" AND "chronicity of rotator cuff tear"; for example, "acromion morphology" AND "imaging" AND "chronicity of rotator cuff tear". This search yielded 14,747 results. Duplicates were excluded and titles and abstracts were screened to determine suitability for inclusion in the paper. In total, 63 articles were included in the final review.

We anticipate this will continue to be a useful resource for clinicians, advocates and the ACC in the complex field of determining causation in rotator cuff tears. Understanding the radiological features of chronicity is also helpful for clinicians' decision making, as it may influence repairability.

Acromion morphology

Historical

Our last review from 2010 found that many studies were not designed to assess the role of acromial morphology in the causation of rotator cuff pathology, and that acromial morphology was unreliably and inconsistently assessed by radiographs. Previous research was conflicting as to the role of acromial morphology and rotator cuff disease; however, studies reported that patients with a large subacromial spur were more likely to have rotator cuff pathology.[3]

Recent research

In 2006, Nyffeler et al. described the acromial index (AI), which was a measure of the lateral extension of the acromion relative to the glenoid.[4] It was postulated that a large AI predisposes to the development of degenerative rotator cuff tears. The supraspinatus must exert a greater force in order to counteract the more vertical pull of the middle fibres of the deltoid as a consequence of a large AI. The study demonstrated a significant correlation between an increased AI and the presence of degenerative rotator cuff tears when compared with patients with arthritis and an intact cuff and age-matched controls.[4] This has been further supported by Morelli et al., who

demonstrated in a systematic review that an AI of greater than 0.695 has the greatest predictive value for a non-traumatic rotator cuff tear.[5]

However, the AI does not account for either glenoid tilt or the presence of degenerative changes, which may falsely alter the overall measurement. Moor et al. has recently reconceptualised the relationship between scapular morphology and rotator cuff pathology with the critical shoulder angle (CSA), which is the angle that combines the measurements of inclination of the glenoid and the lateral extension of the acromion. In their study of 298 patients there was a significant association between an increased CSA >35 degrees and the presence of degenerative rotator cuff tears, while a CSA <30 degrees was associated with the development of osteoarthritis.[6] The CSA demonstrated excellent inter- and intra-observer reliability; however, it is important to note this study excluded acute rotator cuff tears.

In 2019, a systematic review by Morelli et al. analysed the effects of acromial morphological type (as described by Bigliani et al.), the AI and the prevalence of rotator cuff tears.[5] Those individuals with a Bigliani type 3 (hooked) acromion were three times more likely to have a degenerative rotator cuff tear compared with a type 1 or 2 acromion. A further systematic review of 34 studies by Andrade et al. from 2019 has supported the previously published work on the CSA, AI and the acromial type, finding moderate evidence to suggest an association between a CSA >35 degrees, AI >0.7, a type 3 acromion and the presence of a degenerative rotator cuff tear.[7]

Liu et al. reported on a novel radiographic measure, the acromion–greater tuberosity impingement index (ATI), and its association with degenerative rotator cuff pathology.[8] This index is calculated by dividing the distance from the centre of rotation (COR) of the humeral head to the greater tuberosity (GT) by the distance from the COR to the underside of the acromion. A cut-off value of 0.965 on magnetic resonance imaging (MRI) and 0.865 on X-rays was able to discriminate between the presence and absence of subacromial impingement, with a higher ATI associated with a degenerative rather than a traumatic tear.

Similar to our previous review in 2010, a recent update on os acromiale by You et al. has not determined a correlation between the presence of an os acromiale and rotator cuff tears.[9]

Conclusion

The recent evidence suggests that those individuals with a type 3 acromion, a CSA of greater than 35 degrees and a larger AI are more likely to have a degenerative rotator cuff tear than those without these morphological features.

Reduced acromiohumeral distance (AHD)

Historical

The AHD was first described by Golding in 1962 as a radiographic measurement that could be used to assess for rotator cuff disease on radiographs.[10] This represents the distance between the undersurface of the acromion and the superior aspect of the humeral head. Recent research has validated the AHD as a reliable and reproducible radiographic parameter with the use of standardised X-rays, and it has been noted that the position of the arm can alter the AHD with a neutral rotation recommended.[11,12] An AHD of <7mm is abnormal and associated with full thickness tears of the rotator cuff or multiple tendon tears, and up to 71% of patients with an AHD <7mm will show fatty atrophy on an MRI scan.[12] Our previous study concluded that the available evidence demonstrated a reduced AHD of <7mm on standardised X-rays is reliable, reproducible and associated with rotator cuff tears with both fatty atrophy and symptoms that may be of more than several years duration.

Recent research

The AHD in the normal population is reported to be between 7 to 14mm with a decreased AHD associated with rotator cuff tears, an increased rate of irreparable rotator cuff tears, higher rates of re-tear and increased fatty degeneration of the muscles.[14,15] Chuang et al. also demonstrated an inverse relationship between the AHD and the degree of tendon retraction, with each 1mm increase in the ADH being associated with a decreasing chance of developing advanced tendon retraction.[16] Although previous studies have demonstrated a good inter- and intra-observer reliability, McCreesh et al. highlighted the importance of utilising standardised radiographs to measure AHD in order to improve the reliability of the measurement, with up to 7mm variability reported using non-standardised radiographs.[17] Sanguanjit et al. demonstrated a significant difference in the AHD between supine and upright radiographs, with the average AHD

on supine radiographs being 1.3mm lower. The group found that an upright AHD of <7mm and a supine AHD of <6.5 were both 100% specific for a full thickness rotator cuff tear, with an accuracy of 64% and 66% respectively, making this a useful measure for ruling out a full thickness tear.[18]

The measurement of AHD on MRI scans and its correlation with plain radiographs has been increasingly investigated. Mirzayan et al. demonstrated a high degree of correlation between plain radiographs and MRI for the measurement of AHD in patients with Hamada 3–5, while a large variability was seen between the imaging modalities in Hamada 1 and 2 patients, with a tendency towards a larger AHD on the plain films. The group also noted that while the AHD on plain radiographs correlated with the degree of supraspinatus fatty degeneration, MRI measures of AHD correlated with both supraspinatus and infraspinatus fatty degeneration.[19] The AHD on average has been found to decrease by 1.7mm on MRI scans when compared with upright plain radiographs, and as such the cutoff of <7mm is not applicable to MRI measurements.[18]

de Oliveira França et al. investigated the influence of gravity on the measurement of the AHD on MRI scans. The group concluded that the location of the tear rather than the presence of gravity was more significantly associated with the degree of humeral head migration, with the posterosuperior and antero-posterosuperior tears being more closely associated with a reduced AHD compared to superior or anterosuperior tendon tears.[20]

Conclusion

A reduced AHD is associated with chronic rotator cuff degeneration and has high intra- and inter-observer reliability when measured on standardised shoulder X-rays. However, caution must be exercised in the use of computed tomography (CT) and MRI, as the AHD has been shown to be significantly smaller than values on radiographs.

Acromioclavicular (AC) joint arthrosis

Historical

In 1972, Neer mentioned that excrescences on the undersurface of the AC joint may potentially impinge on the rotator cuff and contribute to rotator cuff tears. Our previous review revealed an extremely high prevalence of AC arthritis on imaging studies. Shubin et al. demonstrated AC joint arthrosis in 68% of those under 30 years and in 93% in those over 30 years, and likewise Needell et al. found AC joint arthrosis to correlate more closely with age rather than the development of rotator cuff tears.[21,22]

Recent research

Choo et al. found, in their review of 146 patients, similar rates of rotator cuff degeneration and rotator cuff tears between those with symptomatic and asymptomatic AC joint arthrosis. Furthermore, inferior AC joint osteophytes, subchondral cysts, AC joint articular surface irregularities and fluid within the AC joint were not associated with the presence of rotator cuff tears and were instead age-related changes.[23]

Previous research had suggested that inferior spurs on the distal clavicle may be a causative factor in the development of degenerative rotator cuff tears. Oh et al., in a prospective, randomised, controlled trial of rotator cuff tear repairs in the context of asymptomatic AC joint arthrosis, demonstrated no difference in the re-tear rates if a distal clavicle excision was performed at the time of surgery or not. The group concluded that the AC joint degenerative changes did not cause damage to the rotator cuff; however, it is important to note that the follow-up in this study was only 1 year, and thus these results might change with longer-term follow-up.[24]

Case reports have commented on the very rare occurrence of AC joint cysts, which are associated with massive rotator cuff tears. Type 1 cysts are isolated cysts that are limited to the AC joint with no communication with the glenohumeral joint. Type 2 cysts are dependent on complete rupture or tear of the rotator cuff musculature and are associated with cuff tear arthropathy.[26–28]

Conclusion

AC joint arthrosis is a common finding that is unlikely to be directly involved in the development of rotator cuff tears. However, a type 2 AC joint cyst is highly suggestive of a massive rotator cuff tear and advanced cuff tear arthropathy, although this is a rare occurrence with an incidence of only 1%.

GT changes

Historical

Our previous study concluded that while cysts in the GT are common, the location of the cyst is very important. Anterior cysts showed a strong

correlation with rotator cuff tears, with 48% of patients demonstrating full thickness degenerative rotator cuff tears. Previous research has highlighted the importance of CT and MRI scans in being able to carefully characterise the location of these cysts, and has also suggested there is no strong correlation between GT cortical thickening or subcortical sclerosis and the development of degenerative rotator cuff tears.

Recent research

In 2017, Chin et al. examined the accuracy of plain radiographs in diagnosing rotator cuff pathology in 50 consecutive patients undergoing shoulder arthroscopy for presumptive rotator cuff pathology. The group examined 11 radiographic parameters: acromial pathology (acromial spur, reduced acromiohumeral space, sourcil sign, acromial acetabularisation, os acromiale); humeral head pathology (GT cortical irregularity, sclerosis, cysts, humeral head rounding); and reduced acromiohumeral interval (AHI) of <6mm. The strongest predictors of rotator cuff pathology were tuberosity irregularity, tuberosity sclerosis and cystic changes. Furthermore, 100% of patients with a >50% posterosuperior rotator cuff tear demonstrated three or more of the 11 radiographic signs, with the humeral head signs being more closely correlated with rotator cuff pathology than acromial changes.[28] Chuang et al. also demonstrated a significant correlation between GT sclerosis and irregularity (spurring) and the degree of tendon retraction, with these signs also correlating with a more advanced Patte stage.[29]

Regarding GT cysts, Suluova et al.[30] studied the correlation between MRI findings of GT cysts and the occurrence of rotator cuff pathology on shoulder arthroscopy. The study found that cyst size correlated with age; however, cyst location was unrelated to patient age. Anterior cysts were more common (56%) and strongly associated with rotator cuff pathology, while posterior and lesser tuberosity (LT) cysts were less common and unrelated to either patient age or rotator cuff pathology.[30] Furthermore, a strong correlation was noted between the MRI and arthroscopy findings with a sensitivity of 96% and specificity of 87%.[30] This is further supported by Gwark et al., who demonstrated an odds ratio of 11.43 and 7.71 for anterior GT cysts and the presence of a supraspinatus and infraspinatus tendon tear respectively.[31] Similarly, Pan et al. studied the association between GT cysts and rotator cuff pathology in 105 consecutive patients with painful shoulders undergoing arthroscopy.[32] Anterior cysts were commonly associated with rotator cuff pathology, while posterior and bare area cysts were not. The positive predictive value for GT cysts in the context of a painful shoulder for those over 50 years was 74% and for those over 60 years was 85% for the presence of a rotator cuff tear.[32]

The association between the presence of LT cysts and the presence of subscapularis degeneration has been less clear, with Wissman et al. demonstrating a very strong correlation between LT cysts and subscapularis tears, while Cho et al. were unable to support this finding, instead noting the presence of a small pit about the LT correlated with subscapularis tendon pathology.[33,34]

Conclusion

The presence of changes in the GT on standardised true glenohumeral X-rays can be a highly predictable method of predicting rotator cuff pathology. The presence of cysts in the anterior GT strongly correlates with both supraspinatus and infraspinatus tears, while cysts within the LT are yet to be definitively linked to subscapularis tears. Furthermore, GT irregularity and sclerosis are also highly predictive of rotator cuff pathology, particularly in conjunction with the presence of anterior cystic change.

Changes in rotator cuff tear size with time

If a patient has a known historical rotator cuff tear, what is the likely change with time in the condition of the rotator cuff and what is the likelihood of developing cuff tear arthropathy? Cuff tear arthropathy is graded according to the Hamada classification, which is based primarily on reduced AHI as follows:

- Grade 1—AHI >6mm
- Grade 2—AHI < or equal to 5mm
- Grade 3—Acetabularisation of the acromion
- Grade 4—Narrowing of the glenohumeral joint
- Grade 5—Humeral head collapse

Two recent key papers have reported a higher rate of progression than previously reported in literature. Ranebo et al. reported on the 22-year follow-up outcomes for 69 patients that underwent a subacromial decompression alone with

evidence of rotator cuff tearing at the time of surgery.[35] At the index operation there were 45 partial thickness and 24 full thickness tears. Eighty-seven percent of the full thickness tears showed evidence of progression, with 74% showing signs of cuff tear arthropathy (Hamada 2 or more) and 30% showing Hamada 4b (severe arthropathy). For partial thickness rotator cuff tears there was a 42% rate of tear progression; however, only 7% demonstrated radiographic evidence of rotator cuff tear arthropathy. Chalmers et al., in their 8-year follow-up of 138 patients, demonstrated that within the first 5 years there was increasing superior migration of the humeral head and Hamada grade, which then plateaued from 5 to 8 years.[36] As the study had a control group, a partial thickness group and a full thickness group they were able to show that size of the tear did not correlate with the amount of progression of the Hamada grade; however, the presence/absence of a tear did correlate.[36] Despite advancing proximal humeral migration, severe arthritic changes were rarely seen.

Moosmayer et al. reported on the outcomes of 50 patients with asymptomatic full thickness rotator cuff tears managed non-operatively for a duration of up 3 years.[37] The group demonstrated that those patients who became symptomatic, on average, had a larger tear size than those patients who remained asymptomatic. Furthermore, a correlation between the development of symptoms and higher grades of muscular atrophy and fatty degeneration was found on MRI scans.[37] Fucentese et al. followed 24 patients with isolated full thickness rotator cuff tears who declined operative intervention. At an average of 42 months of follow-up they noted overall no significant change in tear size, but the degree of fatty infiltration did significantly progress; however, no patient had Goutallier >2 at final follow-up.[38] Both of these studies, however, are limited by shorter follow-up than the previous studies.

Conclusion

The recent literature supports that full thickness rotator cuff tears have a higher risk for progression to cuff tear arthropathy at longer-term follow-up compared with partial thickness rotator cuff tears.

Fatty muscle degeneration

Historical

Goutallier et al. introduced the concept of fatty degeneration of the rotator cuff in 1989, and developed a 5-grade staging system, which noted that rotator cuff tears were associated with fatty muscle degeneration.[39] This group were also the first to demonstrate a highly negative correlation between fatty infiltration of the infraspinatus and surgical outcome. Several natural history outcome studies have noted poorer surgical outcomes and higher re-tear rates for patients with pre-operative fatty infiltration and muscular atrophy, particularly Goutallier grade 2 or higher. Although often used interchangeably, fatty infiltration and muscular atrophy are two distinct phenomena, the pathophysiology of which is not yet fully understood. The tangent sign, as described by Zanetti et al., is an indicator of muscle atrophy.[40] The tangent sign is evaluated on the sagittal plane at the most lateral image where the scapular spine is in contact with the scapular body. The tangent sign is positive if the supraspinatus does not reach above this line.[1] Our previous review highlighted that while CT and MRI may both be used for the evaluation of rotator cuff fatty infiltration and muscle atrophy, the inter-observer reliability remains relatively poor; however, this can be improved through evaluation on the axial plane images.[41]

Recent research

Rotator cuff atrophy and fatty infiltration are two distinct phenomena and are both independent predictors of outcome for rotator cuff surgery.[42,43] Fatty infiltration represents a chronic change in the muscle and may signal a poor biological capacity to heal and poor mechanical properties that can make repair difficult or reduce the chance of a repair remaining intact.[44,45] A large retrospective review of 1,688 patients by Melis et al. demonstrated that fatty infiltration of the supraspinatus is associated with increasing age, duration of symptoms prior to presentation and the number of tendons involved.[42,44,45] On average, grade 2 fatty degeneration occurred after 4 years and severe fatty infiltration after 6 years. For traumatic tears the progression of fatty infiltration was on average 1 year faster. Melis et al. also demonstrated the correlation between the degree of fatty infiltration and muscle atrophy. Significant muscle atrophy with a positive tangent sign occurred on average 4.5 years after the onset of symptoms.[44] Hebert-Davies et al. likewise highlighted the chronic nature of fatty infiltration and atrophy, with the appearances of these phenomena being present on average 1 year and 1.4 years after the

onset of symptoms for supraspinatus and infraspinatus respectively.[46]

Chitkara et al. reviewed the reliability of coronal imaging vs sagittal imaging for the accurate assessment of the fatty infiltration.[47] The original Goutallier method utilises the most lateral point where the scapular spine is in contact with the scapular body. However, with tendon tears there is retraction of the muscle belly with increased chronicity, and as such the sagittal image may not be examining the greatest diameter of the muscle but rather the musculotendinous junction and is thus over-calling the degree of muscle atrophy and fatty infiltration. Their study of 50 patients (30 with tears and 20 without tears) found that with sagittal imaging compared to coronal plane imaging, there was a discrepancy in the Goutallier grade 62% of the time, with the sagittal plane tending to over-call the degree of fatty infiltration.[47]

Lee et al. reported on the use of the IDEAL MRI sequence, which is accurately able to assess the intra-muscular fat fraction.[48] Although it has been mainly used for research purposes, this study shows that this sequence is highly reproducible and clinically feasible. The group found that the supraspinatus fat fraction was highly associated with the size of the tear and the degree of tendon retraction, with a fat fraction of 3.7% with no tear, 6.8% with a partial tear, 15.7% with large tears and 16.1% with massive tears.[48] Furthermore, an increasing fat fraction was seen with increasing tendon retraction, and it was noted that in general the fat fraction in muscle increased 0.15% per year independent of a tear of any size.[48]

Conclusion

Fatty infiltration and muscle atrophy are two distinct processes that occur in tandem in the setting of a chronic rotator cuff. These findings tend to be associated with a tear that has been present for >6 months duration, and they increase with increasing age, duration of symptoms and number of tendons involved in the tear. Fatty infiltration and muscle atrophy do not improve after surgical treatment; however, surgery may halt further progression. The inter-observer reliability of the Goutallier classification is low to moderate and the use of sagittal images may overstate the degree of fatty infiltration. As such, use of the coronal image or the axial image may better reflect the true degree of fatty infiltration. Furthermore, advances in scanning with the IDEAL MRI sequence may become more routine in assessing the intramuscular fat fraction.

Tendon retraction

Historical

At the time of our previous publication, little had been reported on the association between the degree of tendon retraction and the acuity of a rotator cuff tear. Previous studies suggested that retraction of a torn tendon to the glenoid does not occur within 12 weeks of injury.

Recent research

Loew et al. investigated features on MRI and radiographs that might distinguish between the acute traumatic tear and the chronic tear.[49] They found that between the two groups there was no significant difference in the degree of tendon retraction; however, the "traumatic group" were twice as likely to display a "*crinkling, undulating appearance of the peripheral end of the torn muscle, described as kinking*", with a specificity of 68% and sensitivity of 64%.[49] Furthermore, the traumatic group of tears were most frequently found to have signal enhancement on T2 sequences at the tendomuscular transition (indicating haemorrhage or oedema), which was only observed in one of the 25 non-traumatic tears. Such an oedema was found to have a positive predictive value of 93.7% and negative predictive value of 72.7% for an acute rotator cuff tear.[49]

Walcott et al. reported a case series of transtendinous rotator cuff tears in a cohort of athletic patients.[50] All patients had a clear history of trauma and a short duration of symptoms. The group concluded that such tendon tears occur in the context of trauma, specifically an axial load; for example, a fall onto an abducted arm.[50]

A sonographic study by Artul et al. looking to distinguish between acute vs chronic tears found a significant association between the thickness of the torn tendon and the acuity of symptoms.[51] A thick torn tendon is more frequently found in the acute tears, with a proposed mechanism being the tendon oedema in the acute setting. No threshold for tendon thickness was given, however.[51]

Park et al. created a scoring system to determine if a rotator cuff tear was likely to be repairable. A supraspinatus tendon length of <15mm was associated with a non-repairable rotator cuff tear and reflects chronicity of injury.[52]

Conclusion

Current literature suggests that the degree of tendon retraction is not a reliable indicator of the acuity of the tear. Rather, the presence of kinking

of the tendon, musculotendinous oedema and the location of the tear (transtendinous) may be more useful guides to helping distinguish an acute rotator cuff tear.

Bursal changes and glenohumeral effusion

Historical

To date, little has been published regarding the association between bursal thickening and signs of non-traumatic rotator cuff tears. In our previous report, Teefey et al. investigated the sonographic difference between acute and chronic tears, with the key element being the history of a traumatic injury. Bursal thickening was defined as over 2mm, and this study found that a mid-substance tear or the presence of bursal fluid in the context of a rotator cuff tear were more commonly present in the acute setting.[53]

Recent research

Little has been reported regarding the macroscopic qualities of synovitis and their correlation with rotator cuff disease. In 2015, Jo et al. proposed a macroscopic and microscopic classification for synovitis in both the glenohumeral joint and the subacromial space. Interestingly, they found that glenohumeral joint synovitis was more clearly linked with rotator cuff disease than synovitis and inflammatory change within the subacromial space.[54] This study was further supported by Kim et al., who found that the degree of glenohumeral synovitis correlated with the duration of symptoms more closely than subacromial synovitis.[55] They suggested considering the chronic rotator cuff tear as a "pan joint pathology", like that of knee osteoarthritis.

Loew et al. compared MRI findings in those with a clear history of trauma and those without. They found that extensive effusion in the subacromial bursa occurred with similar incidences between the traumatic and non-traumatic groups (84% vs 79%).[49] They also noted that glenohumeral joint effusion tended to be more frequent in the traumatic group compared with the non-traumatic group, and while this is seemingly in contrast with the above findings, these were MRI findings rather than arthroscopic findings.[49]

Artul et al. performed a sonographic study aiming to distinguish between the acute and the chronic tear. This group found that acute tears were more likely to have significant biceps peri-tendon oedema when compared with the atraumatic tears, although no threshold for what constitutes "significant oedema" has currently been published. The group did find that a subdeltoid effusion correlated more closely with the acute tears; however, if this was the only positive feature on sonographic examination it was only just on the edge of significance.[51]

Conclusion

Assessment of the degree of glenohumeral synovitis may be more indicative of the chronicity of a tear when compared with subacromial changes. It would appear that subacromial bursal changes are unable to reliably distinguish between the acute and the chronic rotator cuff tear, unless there is extensive bursal fluid and debris. Furthermore, although there is an association between significant biceps peri-tendon oedema in the setting of an acute tear, there currently exists no clear threshold for what degree of oedema would most closely correlate with an acute rotator cuff tear.

Partial thickness tears

Historical

Our last report highlighted the complex nature of the aetiology of partial thickness rotator cuff tears. While trauma has been shown to be associated with both articular and bursal sided tears, such tears have also been demonstrated in young athletes.

Recent research

Much work has been done in recent years to determine the natural history of the partial thickness rotator cuff, and to determine the outcomes of surgical intervention; however, there remains little research with regards to determining the underlying cause for a partial thickness tear.

Tsuchiya et al., in their systematic review of partial thickness tears, demonstrated that partial thickness tears do progress over time but at a relatively slow rate in the short- to intermediate-term follow-up, on average 0.26% per month.[55] This review found no significant difference in the rate of progression to full-thickness tears over time, between the symptomatic and asymptomatic groups. Yamamoto et al. prospectively followed non-operatively treated partial thickness rotator cuffs. There were only 17 traumatic tears in the cohort of 174 shoulders, and on average 47% of partial thickness tears progressed, with the highest rate of progression in the medium-sized

tears.[57] Trauma was a risk factor for the progression of medium-sized tears.

McMonagle et al., in a review on the utility of MRI in the shoulder, showed that the sensitivity and specificity for partial thickness tears was lower than that for full thickness tears (63.6% and 91.7% vs 92.1% and 92.9%), with articular sided tears being significantly more common compared with bursal sided tears.[58]

Shibata et al. compared the arthroscopic findings of acute and degenerative partial thickness and full thickness rotator cuff tears in patients over 65 years with a Goutallier grade <2. While the MRI appearances of the tears were similar, on arthroscopic repair the traumatic tears were significantly less stiff when attempting to mobilise the tear, and the tension of the repair was less when compared with the degenerative tears.[59]

Conclusion

The aetiology of partial thickness tears is complex in nature. Despite the recent interest in the study of partial thickness tears, most research focusses on natural history or surgical outcome. The natural history of a partial thickness tear is that of slow progression. Imaging is unable to clearly distinguish between a traumatic or degenerative underlying cause

Tendinopathy

Historical

Our previous report on rotator cuff imaging noted that tendinopathy is a complex topic, with literature suggesting a multifactorial pathogenesis resulting from intrinsic, extrinsic and environmental factors. Intrinsic degeneration may result from chronic overload of the tendon, while extrinsic compression from subacromial impingement was also linked to the development of tendinopathy. Along with the multifactorial aetiology, patient age was shown to be a factor related to tendinopathy, with the peak incidence occurring in the fifth to seventh decades.

Recent research

Recent research on tendinopathy has focussed on trying to determine the underlying pathological processes that occur to cause tendinopathy. Proposed mechanisms include a combination of both intrinsic and extrinsic mechanisms.

Excessive tissue load has been reported as a significant causative factor in the development of rotator cuff tendinopathy, as reflected by the fact that tendinopathy occurs more frequently in the dominant limb and in occupations/sports with high rates of upper-limb loading.[60]

Rotator cuff tendinopathy remains a common clinical diagnosis. Frost et al. reported a high rate of rotator cuff tendinopathy in both patients with subacromial impingement (55%) and those without any symptoms of shoulder dysfunction (52%), with an increasing incidence observed in both groups with increasing age.[61] A more recent study found that 96% of asymptomatic men were reported to have some form of structural abnormality identifiable on ultrasound, including subacromial bursal thickening, supraspinatus tendinosis and supraspinatus tears.[62]

Thickening of the rotator cuff tendons is noted on MRI and ultrasound, and this can cause a relative decrease in the subacromial space and thus worsen any external impingement.[61] Histopathologic examination of tissue from rotator cuff tears demonstrated abnormal tendons features in 97% of cases, with animal models demonstrating that tendinopathic changes induce specific gene upregulation that causes tissue metaplasia and reduces the load to failure of the tendon.[63] Furthermore, serum levels of reactive oxygen species have been noted to be higher in patients with tendinopathy compared to those without, suggesting oxidative stress plays a role in tendon damage.[64]

Conclusion

The current evidence has shown a link between increasing age and the occurrence of tendinopathy. Imaging may show evidence of tendon thickening and areas of relative hyper- or hypo-vascularity. Biochemical studies have demonstrated abnormal tendon features with tissue metaplasia and evidence of inflammatory and oxidative stress. We are unable to determine if a rotator cuff tear in the setting of tendinopathy is purely due to degeneration or if acute trauma may play a role.

Conclusions

The following can be considered normal findings with increasing age and are not related to acuity or chronicity of rotator cuff tears:

- Type 1 and type 2 acromion[6,8]
- AC joint arthrosis[23,24]
- Tendinopathy[60–62]

The following have no definite association with causation of rotator cuff tears:

- Os acromiale[9]
- Posterior and bare area GT cysts[30,31]
- Calcific tendinosis[1]

The following are associated with the risk of evolution of rotator cuff tears:

- AI >0.7[5,6,8]
- CSA >0.35[7,8]

The following are associated with chronic rotator cuff tears:

- Type 3 acromion[6,8]
- AHI <7mm[14,15,16,19]
- Goutallier fatty degeneration grade 2+[42,44,45,46,48]
- Positive tangent sign[44,46]
- Length of torn supraspinatus tendon <15mm[52]
- Anterior GT cysts[28,30,31,32]

The following are associated with acuity and trauma of rotator cuff tears:

- Bone oedema of GT[1,65]
- Mid-substance rotator cuff tears[50]
- Kinked appearance of the tendon tear[49]
- Large effusion[49]
- Bursal haematoma and/or debris[51]

COMPETING INTERESTS
None.

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REFERENCES
1. Mohammed KD, Wilkinson B, Nagaraj C. Can imaging determine if a rotator cuff tear is traumatic? N Z Med J. 2010 Dec 17;123(1327):99-113.
2. Codman EA, Akerson IB. THE PATHOLOGY ASSOCIATED WITH RUPTURE OF THE SUPRASPINATUS TENDON. Ann Surg. 1931;93(1):348-59. doi: 10.1097/00000658-193101000-00043.
3. Ogawa K, Yoshida A, Inokuchi W, Naniwa T. Acromial spur: relationship to aging and morphologic changes in the rotator cuff. J Shoulder Elbow Surg. 2005;14(6):591-8. doi: 10.1016/j.jse.2005.03.007.
4. Nyffeler RW, Werner CM, Sukthankar A, et al. Association of a large lateral extension of the acromion with rotator cuff tears. J Bone Joint Surg Am.2006;88(4):800-805. doi: 10.2106/JBJS.D.03042.
5. Morelli KM, Martin BR, Charakla FH, et al. Acromion morphology and prevalence of rotator cuff tear: a systematic review and meta-analysis. Clin Anat. 2019;32(1):122-130. doi: 10.1002/ca.23309.
6. Moor BK, Bouaicha S, Rothenfluh DA, et al. Is there an association between the individuals anatomy of the scapula and the development of rotator cuff tears or osteoarthritis of the glenohumeral joint?: A radiological study of the critical shoulder angle. Bone Joint J. 2013;95-B(7):935-41. doi: 10.1302/0301-620X.95B7.31028.
7. Andrade R, Correia AL, Nunes J, et al. Is Bony Morphology and Morphometry Associated With Degenerative Full-Thickness Rotator Cuff Tears? A Systematic Review and Meta-analysis. Arthroscopy. 2019;35(12):3304-3315.e2. doi: 10.1016/j.arthro.2019.07.005.
8. Liu HX, Xu XX, Xu DL, et al. The acromion-greater tuberosity impingement index: A new radiographic measurement and its association with rotator cuff pathology. J Orthop Surg (Hong Kong). 2020 Jan-Apr;28(1):2309499020913348. doi: 10.1177/2309499020913348.
9. You T, Frostick S, Zhang WT, et al. Os Acromiale: Reviews and Current Perspectives. Orthop Surg. 2019 Oct;11(5):738-744. doi: 10.1111/os.12518.
10. Golding FC. The shoulder--the forgotten joint. Br J Radiol. 1962 Mar;35:149-58. doi: 10.1259/0007-1285-35-411-149.
11. Gruber G, Bernhardt GA, Clar H, et al. Measurement of the acromiohumeral interval on standardized anteroposterior radiographs: a prospective study of observer variability. J Shoulder Elbow Surg. 2010 Jan;19(1):10-3. doi: 10.1016/j.jse.2009.04.010.
12. Nové-Josserand L, Edwards TB, O'Connor DP, Walch G. The acromiohumeral and coracohumeral intervals are abnormal in rotator cuff tears with muscular fatty degeneration. Clin Orthop Relat Res. 2005 Apr;(433):90-6. doi: 10.1097/01.blo.0000151441.05180.0e.
13. Saupe N, Pfirrmann CW, Schmid MR, et al. Association between rotator cuff abnormalities and reduced acromiohumeral distance. AJR Am J Roentgenol. 2006 Aug;187(2):376-82. doi: 10.2214/AJR.05.0435.
14. Park SH, Choi CH, Yoon HK, et al. What can the radiological parameters of superior migration of the humeral head tell us about the reparability of massive rotator cuff tears? PLoS One. 2020 Apr 16;15(4):e0231843. doi: 10.1371/journal.pone.0231843.
15. Razmjou H, Palinkas V, Christakis M, et al. Reduced acromiohumeral distance and increased critical shoulder angle: implications for primary care clinicians. Phys Sportsmed. 2020 Sep;48(3):312-319. doi: 10.1080/00913847.2019.1703475.
16. Chuang HC, Hong CK, Hsu KL, et al. Radiographic greater tuberosity spurs and narrow acromiohumeral intervals are associated with advanced retraction of the supraspinatus tendon in patients with symptomatic rotator cuff tears. JSES Int. 2020 Nov 26;5(1):77-82. doi: 10.1016/j.jseint.2020.09.015.
17. McCreesh KM, Crotty JM, Lewis JS. Acromiohumeral distance measurement in rotator cuff tendinopathy: is there a reliable, clinically applicable method? A systematic review. Br J Sports Med. 2015 Mar;49(5):298-305. doi: 10.1136/

bjsports-2012-092063.
18. Sanguanjit P, Apivatgaroon A, Boonsun P, et al. The differences of the acromiohumeral interval between supine and upright radiographs of the shoulder. Sci Rep. 2022 Jun 7;12(1):9404. doi: 10.1038/s41598-022-13632-0.
19. Mirzayan R, Donohoe S, Batech M, et al. Is there a difference in the acromiohumeral distances measured on radiographic and magnetic resonance images of the same shoulder with a massive rotator cuff tear? J Shoulder Elbow Surg. 2020 Jun;29(6):1145-1151. doi: 10.1016/j.jse.2019.10.020.
20. de Oliveira França F, Godinho AC, Ribeiro EJ, et al. Evaluation of the acromiohumeral distance by means of magnetic resonance imaging umerus. Rev Bras Ortop. 2016 Feb 4;51(2):169-74. doi: 10.1016/j.rboe.2016.01.008.
21. Shubin Stein BE, Ahmad CS, Pfaff CH, et al. A comparison of magnetic resonance imaging findings of the acromioclavicular joint in symptomatic versus asymptomatic patients. J Shoulder Elbow Surg. 2006 Jan-Feb;15(1):56-9. doi: 10.1016/j.jse.2005.05.013.
22. Needell SD, Zlatkin MB, Sher JS, et al. MR imaging of the rotator cuff: peritendinous and bone abnormalities in an asymptomatic population. AJR Am J Roentgenol. 1996 Apr;166(4):863-7. doi: 10.2214/ajr.166.4.8610564.
23. Choo HJ, Lee SJ, Kim JH, et al. Can symptomatic acromioclavicular joints be differentiated from asymptomatic acromioclavicular joints on 3-T MR imaging? Eur J Radiol. 2013 Apr;82(4):e184-91. doi: 10.1016/j.ejrad.2012.10.027.
24. Oh JH, Kim JY, Choi JH, et al. Is arthroscopic distal clavicle resection necessary for patients with radiological acromioclavicular joint arthritis and rotator cuff tears? A prospective randomized comparative study. Am J Sports Med. 2014 Nov;42(11):2567-73. doi: 10.1177/0363546514547254.
25. Tanaka S, Gotoh M, Mitsui Y, et al. A Case Report of an Acromioclavicular Joint Ganglion Associated with a Rotator Cuff Tear. Kurume Med J. 2017 Apr 13;63(1.2):29-32. doi: 10.2739/kurumemedj.MS6300002.
26. Purohit S, Keny S, Raja B, Marathe N. Massive acromio-clavicular joint ganglion cyst associated with cuff tear arthropathy and acromioclavicular joint arthritis with normal functional shoulder-A case report. J Clin Orthop Trauma. 2019 May-Jun;10(3):522-525. doi: 10.1016/j.jcot.2019.03.001.
27. Hiller AD, Miller JD, Zeller JL. Acromioclavicular joint cyst formation. Clin Anat. 2010 Mar;23(2):145-52. doi: 10.1002/ca.20918.
28. Chin K, Chowdhury A, Leivadiotou D, et al. The accuracy of plain radiographs in diagnosing degenerate rotator cuff disease. Shoulder Elbow. 2019 May;11(1 Suppl):46-51. doi: 10.1177/1758573217743942.
29. Chuang HC, Hong CK, Hsu KL, et al. Radiographic greater tuberosity spurs and narrow acromiohumeral intervals are associated with advanced retraction of the supraspinatus tendon in patients with symptomatic rotator cuff tears. JSES Int. 2020 Nov 26;5(1):77-82. doi: 10.1016/j.jseint.2020.09.015.
30. Suluova F, Kanatli U, Ozturk BY, et al. Humeral head cysts: association with rotator cuff tears and age. Eur J Orthop Surg Traumatol. 2014 Jul;24(5):733-9. doi: 10.1007/s00590-013-1247-5.
31. Gwark JY, Park TS, Park HB. Association between the location of tuberosity cysts and rotator cuff tears: A comparative study using radiograph and MRI. J Orthop Surg (Hong Kong). 2019 Jan-Apr;27(1):2309499019825762. doi: 10.1177/2309499019825762.
32. Pan YW, Mok D, Tsiouri C, Chidambaram R. The association between radiographic greater tuberosity cystic change and rotator cuff tears: a study of 105 consecutive cases. Shoulder and Elbow. 2011 Aug;3(4):205-209.
33. Wissman RD, Ingalls J, Hendry D, et al. Cysts within and adjacent to the lesser tuberosity: correlation with shoulder arthroscopy. Skeletal Radiol. 2012 Sep;41(9):1105-10. doi: 10.1007/s00256-012-1366-9.
34. Cho JH, Han KJ, Lee DH, et al. Pit above the lesser tuberosity in axial view radiography. Knee Surg Sports Traumatol Arthrosc. 2015 Feb;23(2):370-5. doi: 10.1007/s00167-013-2546-4.
35. Ranebo MC, Björnsson Hallgren HC, Norlin R, et al. Clinical and structural outcome 22 years after acromioplasty without tendon repair in patients with subacromial pain and cuff tears. J Shoulder Elbow Surg. 2017 Jul;26(7):1262-1270. doi: 10.1016/j.jse.2016.11.012.
36. Chalmers PN, Salazar DH, Steger-May K, et al. Radiographic progression of arthritic changes in shoulders with degenerative rotator cuff tears. J Shoulder Elbow Surg. 2016 Nov;25(11):1749-1755. doi: 10.1016/j.jse.2016.07.022.
37. Moosmayer S, Gärtner AV, Tariq R. The natural course of nonoperatively treated rotator cuff tears: an 8.8-year follow-up of tear anatomy and clinical outcome in 49 patients. J Shoulder Elbow Surg. 2017 Apr;26(4):627-634. doi: 10.1016/j.jse.2016.10.002.
38. Fucentese SF, von Roll AL, Pfirrmann CW, et al. Evolution of nonoperatively treated symptomatic

isolated full-thickness supraspinatus tears. J Bone Joint Surg Am. 2012 May 2;94(9):801-8. doi: 10.2106/JBJS.I.01286.
39. Goutallier D, Bernageau J, Patte D. Assessment of the trophicity of the muscles of the ruptured rotator cuff by CT scan. In: Post M, Morrey B, Hawkins R, editors. Surgery of the Shoulder. St. Louis, MO: Mosby; 1990. p. 11-13.
40. Zanetti M, Gerber C, Hodler J. Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. Invest Radiol. 1998 Mar;33(3):163-70. doi: 10.1097/00004424-199803000-00006.
41. Williams MD, Lädermann A, Melis B et al. Fatty infiltration of the supraspinatus: a reliability study. J Shoulder Elbow Surg. 2009;18(4):581-7. doi: 10.1016/j.jse.2008.12.014.
42. Deniz G, Kose O, Tugay A, et al. Fatty degeneration and atrophy of the rotator cuff muscles after arthroscopic repair: does it improve, halt or deteriorate? Arch Orthop Trauma Surg. 2014 Jul;134(7):985-90. doi: 10.1007/s00402-014-2009-5.
43. Kuzel BR, Grindel S, Papandrea R, Ziegler D. Fatty infiltration and rotator cuff atrophy. J Am Acad Orthop Surg. 2013 Oct;21(10):613-23. doi: 10.5435/JAAOS-21-10-613.
44. Melis B, DeFranco MJ, Chuinard C, Walch G. Natural history of fatty infiltration and atrophy of the supraspinatus muscle in rotator cuff tears. Clin Orthop Relat Res. 2010 Jun;468(6):1498-505. doi: 10.1007/s11999-009-1207-x.
45. Barry JJ, Lansdown DA, Cheung S, et al. The relationship between tear severity, fatty infiltration, and muscle atrophy in the supraspinatus. J Shoulder Elbow Surg. 2013 Jan;22(1):18-25. doi: 10.1016/j.jse.2011.12.014.
46. Hebert-Davies J, Teefey SA, Steger-May K, et al. Progression of Fatty Muscle Degeneration in Atraumatic Rotator Cuff Tears. J Bone Joint Surg Am. 2017 May 17;99(10):832-839. doi: 10.2106/JBJS.16.00030.
47. Chitkara M, Albert M, Wong T, et al. Rotator Cuff Fatty Infiltration Are Coronal Images More Helpful for Characterization than Sagittal Images? Bull Hosp Jt Dis (2013). 2016 Jun;74(2):130-4.
48. Lee S, Lucas RM, Lansdown DA, et al. Magnetic resonance rotator cuff fat fraction and its relationship with tendon tear severity and subject characteristics. J Shoulder Elbow Surg. 2015 Sep;24(9):1442-51. doi: 10.1016/j.jse.2015.01.013.
49. Loew M, Magosch P, Lichtenberg S, et al. How to discriminate between acute traumatic and chronic degenerative rotator cuff lesions: an analysis of specific criteria on radiography and magnetic resonance imaging. J Shoulder Elbow Surg. 2015 Nov;24(11):1685-93. doi: 10.1016/j.jse.2015.06.005.
50. Walcott ME, Daniels SD, Sinz NJ, et al. Traumatic full-thickness transtendinous rotator cuff tears: a case series. J Shoulder Elbow Surg. 2017 Jan;26(1):62-67. doi: 10.1016/j.jse.2016.04.023.
51. Artul S, Habib G. Ultrasonographic clues for acuity/chronicity of rotator cuff tear. Eur J Rheumatol. 2017 Dec;4(4):260-263. doi: 10.5152/eurjrheum.2017.17047.
52. Park I, Kang JS, Lee HA, et al. A Novel Reparability Assessment Scoring System for Full-Thickness Rotator Cuff Tears. Orthop J Sports Med. 2020 Aug 7;8(8):2325967120940979. doi: 10.1177/2325967120940979.
53. Teefey SA, Middleton WD, Bauer GS, et al. Sonographic differences in the appearance of acute and chronic full-thickness rotator cuff tears. J Ultrasound Med. 2000 Jun;19(6):377-8; quiz 383. doi: 10.7863/jum.2000.19.6.377.
54. Jo CH, Shin JS, Kim JE, Oh S. Macroscopic and microscopic assessments of the glenohumeral and subacromial synovitis in rotator cuff disease. BMC Musculoskelet Disord. 2015 Sep 30;16:272. doi: 10.1186/s12891-015-0740-x.
55. Kim DH, Bae KC, Choi JH, et al. Chronicity is associated with the glenohumeral synovitis in patients with a rotator cuff tear. J Orthop Res. 2021 Oct;39(10):2226-2233. doi: 10.1002/jor.24941.
56. Tsuchiya S, Davison EM, Rashid MS, et al. Determining the rate of full-thickness progression in partial-thickness rotator cuff tears: a systematic review. J Shoulder Elbow Surg. 2021 Feb;30(2):449-455. doi: 10.1016/j.jse.2020.08.022.
57. Yamamoto N, Mineta M, Kawakami J, et al. Risk Factors for Tear Progression in Symptomatic Rotator Cuff Tears: A Prospective Study of 174 Shoulders. Am J Sports Med. 2017 Sep;45(11):2524-2531. doi: 10.1177/0363546517709780.
58. McMonagle J, Vinson E. MRI of the shoulder: Rotator cuff. Applied Radiology. 2012 Apr; 41(4):20-28.
59. Shibata T, Izaki T, Nishio J, et al. Are there differences in arthroscopic and histological features between traumatic and degenerative rotator cuff tears in elderly patients? A prospective dual-center analysis. J Orthop Surg Res. 2022 Apr 7;17(1):206. doi: 10.1186/s13018-022-03100-w.
60. Varacallo M, El Bitar Y, Mair SD. Rotator Cuff Tendonitis. 2023 Aug 4. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–.
61. Frost P, Andersen JH, Lundorf E. Is supraspinatus pathology as defined by magnetic resonance imaging associated with clinical sign of shoulder

impingement? J Shoulder Elbow Surg. 1999 Nov-Dec;8(6):565-8. doi: 10.1016/s1058-2746(99)90090-3.
62. Lewis J, McCreesh K, Roy JS, Ginn K. Rotator Cuff Tendinopathy: Navigating the Diagnosis-Management Conundrum. J Orthop Sports Phys Ther. 2015 Nov;45(11):923-37. doi: 10.2519/jospt.2015.5941.
63. Dean BJF, Dakin SG, Millar NL, et al. Review: Emerging concepts in the pathogenesis of tendinopathy. Surgeon. 2017 Dec;15(6):349-354. doi: 10.1016/j.surge.2017.05.005.
64. Cong GT, Lebaschi AH, Camp CL, et al. Evaluating the role of subacromial impingement in rotator cuff tendinopathy: Development and analysis of a novel murine model. J Orthop Res. 2018 Oct;36(10):2780-2788. doi: 10.1002/jor.24026.
65. McCauley TR, Disler DG, Tam MK. Bone marrow edema in the greater tuberosity of the humerus at MR imagng: association with rotator cuff tears and traumatic injury. Magn Reson Imaging. 2000;18(8):979-84. doi: 10.1016/s0730-725x(00)00201-0.

Improving community antibiotic prescribing to keep antibiotics working in Aotearoa New Zealand

Mark G Thomas

ABSTRACT

Antibiotic resistance, principally a consequence of the human use of antibiotics dispensed in the community, is a relentlessly growing threat to human health in Aotearoa New Zealand. Reducing the prescription of antibiotics for conditions in which they confer no benefit is the most effective method of slowing the spread of antibiotic resistance. In Aotearoa New Zealand, as in many other nations, antibiotic "treatment" of acute respiratory tract infections is the most important component of unnecessary antibiotic use.

Because of the ethnic inequities in the incidence and consequences of infectious diseases in Aotearoa New Zealand, Māori and Pacific patients should receive antibiotic treatment more frequently than patients of other ethnicities. However, Māori and Pacific people who present to their doctor with conditions that do not require antibiotic treatment deserve the same excellent treatment as anyone else and should not be prescribed an antibiotic when it will provide no benefit. Setting annual goals for reductions in community antibiotic dispensing has been an effective method to encourage sustained improvements in antibiotic prescribing in other nations, and may help to quickly reduce inappropriate antibiotic prescribing in Aotearoa New Zealand.

Antibiotic resistance poses a threat to human health in Aotearoa New Zealand

Antibiotic resistance is widely expected to pose a steadily growing threat to human health in the coming decades.[1–4] Recent examples of the rapid spread of antibiotic resistance in Aotearoa New Zealand include mupirocin resistance in *Staphylococcus aureus*[5] and ciprofloxacin resistance in *Neisseria gonorrhoeae*.[6] During the 1990s, approximately 200,000 15g tubes of "Bactroban" (mupirocin) were dispensed annually, approximately equivalent to one tube per 17 residents of Aotearoa New Zealand each year. In 8 years, the prevalence of mupirocin resistance in strains of *S. aureus* tested by the Auckland community laboratory went from <5% to >20%.[5] Between 2000 and 2012, the proportion of isolates of *N. gonorrhoeae* in Aotearoa New Zealand that were resistant to ciprofloxacin increased from less than 2% to more than 40%.[6] Similar rapid increases in the prevalence of ciprofloxacin resistance occurred in many other nations.

Other important examples include the rapid spread of extended-spectrum beta-lactamase producing (ESBL+ve) *Escherichia coli, Klebsiella pneumoniae* and related Gram-negative bacteria. Seven ESBL+ve bacteria were isolated from patients in Aotearoa New Zealand in 1998; by 2012, more than 5,000 ESBL+ve bacteria were isolated each year.[7] These bacteria are resistant to most readily available oral antibiotics, and commonly require treatment with meropenem or another closely related carbapenem antibiotic, administered intravenously.[8] However, resistance to meropenem is also increasing rapidly. The number of people in Aotearoa New Zealand who had a meropenem-resistant *E. coli, K. pneumoniae* or related bacterium isolated increased from one person in 2009 to 223 people in 2023.[9]

There are very limited options for treating these carbapenem-resistant bacteria. Once strains of *E. coli, K. pneumoniae* and *P. aeruginosa* with resistance to carbapenems spread widely in Aotearoa New Zealand, as they have elsewhere, we will be faced with large numbers of patients with common infections such as pyelonephritis and cholecystitis that are almost untreatable and therefore likely to be fatal. The future is illustrated by a 2014–2015 survey of long-term care facilities across the United States, which reported that 24.6% (946/3,846) residents were colonised with carbapenem-resistant *K. pneumoniae*.[10] Infections with these bacteria were four times more likely to be fatal than infections with *K. pneumoniae* that were carbapenem susceptible.[11]

The risk of infections caused by extensively

resistant organisms will have severe implications for the use of the many medical treatments that increase patients' risk of severe infections, such as chemotherapy for haematologic cancers. It will also have implications for many surgical procedures, such as organ transplantation, that rely on the ability to successfully treat post-operative infections. A report commissioned by the United Kingdom's [UK] prime minister in 2014 predicted that by 2050 world-wide deaths attributable to antimicrobial resistance would surpass those attributable to cancer.[12]

The magnitude of the antibiotic resistance threat differs greatly between nations, with the threat expected to be greatest in those nations that have had very high rates of antibiotic use in the preceding decades. In developed countries, antibiotics dispensed by community pharmacies comprise 85–95% of all antibiotic consumption, with antibiotics dispensed for hospital inpatients comprising the remaining 5–15%.[13] Figure 1 shows the rates of community antibiotic dispensing, measured in defined daily doses (DDDs)/1,000 population/day, during 2018 for a range of European nations[14] and Aotearoa New Zealand.[15] In Greece, Spain, France, Poland and Aotearoa New Zealand the rates of community antibiotic dispensing were more than twice as high as they were in Sweden, Austria and the Netherlands.

High rates of antibiotic use in Aotearoa New Zealand have resulted in high rates of antibiotic resistance

The national rate of community dispensing of beta-lactam antibiotics (penicillins and cephalosporins) during 2018 in Aotearoa New Zealand was 12.9 DDDs/1,000 population/day, more than four times the rate in the Netherlands (2.9), and more than twice the rates in Norway (5.6) and Sweden (5.8)[14,15] (Figure 2a). A likely consequence of the high national rate of use of beta-lactam antibiotics in Aotearoa New Zealand in recent decades is that in 2018 methicillin-resistant *S. aureus* (MRSA) comprised 12.7% of all *S. aureus* isolates in Aotearoa New Zealand,[16] a much higher prevalence than in the Netherlands (1.2%), Sweden (1.9%) or Norway (0.9%).[17] In Belgium, Greece, France and Spain the rates of community beta-lactam antibiotic use were comparable or greater than those in Aotearoa New Zealand,[14] and the prevalence of MRSA in some of these nations was even higher than in Aotearoa New Zealand.[17] If we continue to have high rates of beta-lactam antibiotic use, the prevalence of MRSA in Aotearoa New Zealand is likely to increase to be similar to the high prevalence in Greece, Spain and Italy. Our ability to successfully treat *S. aureus* infections will fall.

The high national rate of community use of beta-lactam antibiotics in recent decades in Aotearoa New Zealand has also led to a high prevalence of reduced susceptibility to penicillin in *Streptococcus pneumoniae* in Aotearoa New Zealand (24%),[16] similar to the prevalence in Spain (18.5%) and France (29.1), and very much higher than the prevalence in the Netherlands, Norway and Sweden[17] (≤5%) (Figure 2b). The reduced susceptibility of *S. pneumoniae* to penicillin has important implications for the treatment of patients with bacterial meningitis and other invasive infections. The same trend of reduced susceptibility to penicillin, secondary to high rates of beta-lactam use, has also been observed in strains of *Neisseria meningitidis* isolated in Aotearoa New Zealand.[18]

The example of ciprofloxacin clearly shows the other side of this story. In recent decades, Aotearoa New Zealand has had a low national rate of community use of ciprofloxacin and other quinolone antibiotics (0.4 DDDs/1,000 population/day).[15] The low rate of use of quinolone antibiotics in Aotearoa New Zealand is the likely cause of the very low prevalence of ciprofloxacin resistance in *E. coli* (0.6%)[16] (Figure 3). In nations with high rates of use of quinolone antibiotics, such as Greece (2.9), Spain (2.7), Italy (2.7) and Bulgaria (2.8),[14] the prevalence of ciprofloxacin resistance in *E. coli* is very much higher (approximately 30–40%).[17]

Antibiotic use in animals is a minor contributor to antibiotic resistance in humans in Aotearoa New Zealand

Antibiotic use in farming has undoubtedly made a significant contribution to the emergence of antibiotic resistance. However, in most developed countries, including Aotearoa New Zealand, the rate of antibiotic use in humans (total kg antibiotic dispensed/total kg human biomass) greatly exceeds that in animals (total kg antibiotic dispensed/total kg farmed animal biomass), and antibiotic use in animals is generally thought to contribute relatively little to the rates of antibiotic resistance in human pathogens.[19] The total weight of antibiotics used in animals and in humans was

Figure 1: Community antibiotic dispensing in 16 European nations and Aotearoa New Zealand during 2018.[14,15]

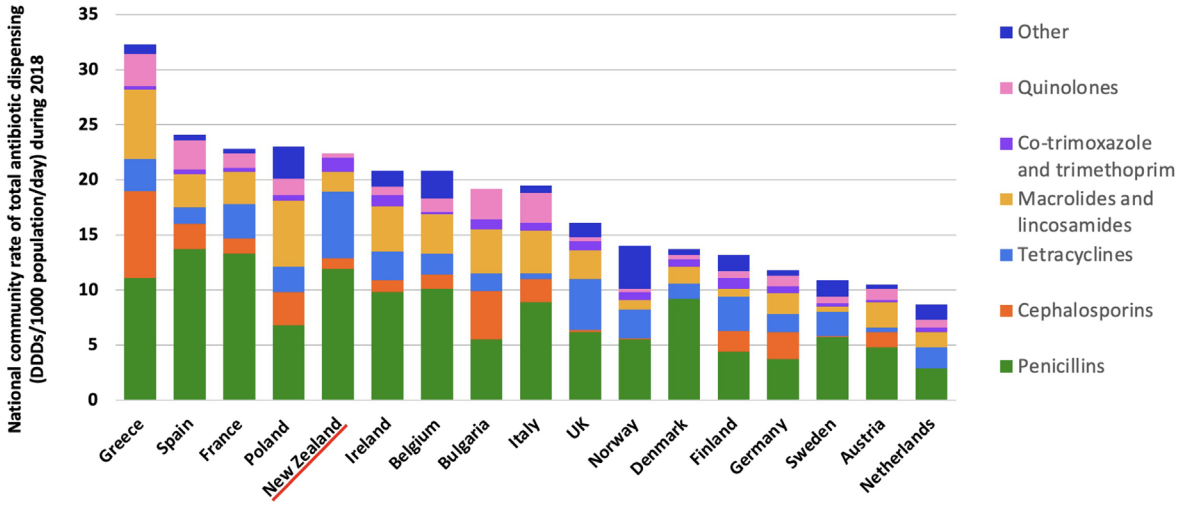


Figure 2: Prevalence of methicillin-resistance in *S. aureus* (a), and of reduced susceptibility to penicillin in *S. pneumoniae* (b), in relation to rates of community dispensing of beta-lactam antibiotics (penicillins plus cephalosporins)

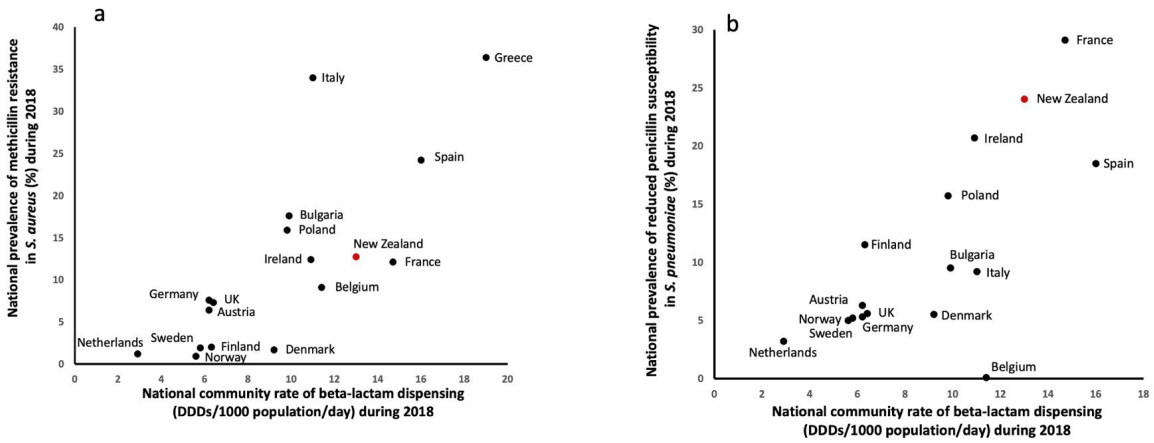


Figure 3: Prevalence of ciprofloxacin resistance in *E. coli* in relation to rates of community dispensing of quinolone antibiotics in 16 European nations and Aotearoa New Zealand during 2018.[14–17]

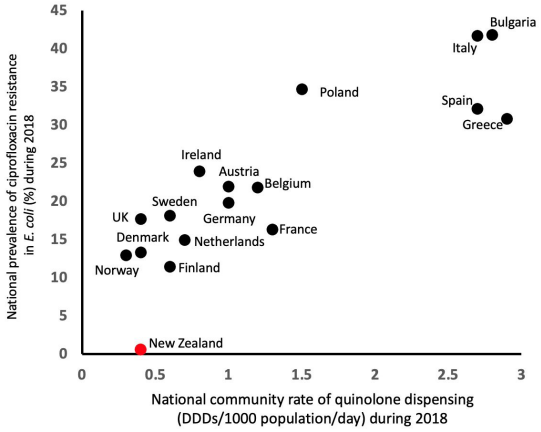


Figure 4: The proportion of consultations for an acute upper respiratory tract infection at 111 general practices in Aotearoa New Zealand during 2014 that were associated with dispensing of an antibiotic during the subsequent 7 days. Each column represents one general practice. An antibiotic was dispensed to at least 50% of patients at approximately two thirds of practices.[25]

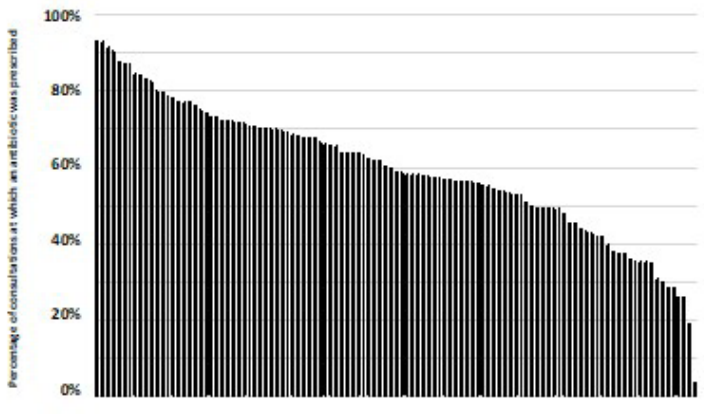
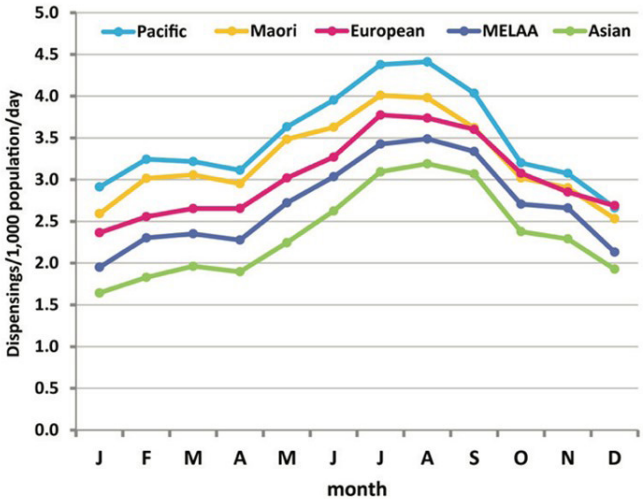


Figure 5: Rate of community antibiotic dispensing in relation to ethnicity and season in Aotearoa New Zealand during 2015.[26]



estimated for 30 countries, including Aotearoa New Zealand, during 2012. The rate of antibiotic use in animals in Aotearoa New Zealand was very low in comparison to the rates in other countries for which data was available.[20] In 2015, the New Zealand Veterinary Association published an ambitious goal that "*By 2030 New Zealand Inc. will not need antibiotics for the maintenance of animal health and wellness*".[21] Between 2017 and 2022, total antibiotic use in animals or plants in Aotearoa New Zealand fell by 42% from 71,361kg to 41,033kg.[22]

Reducing community antibiotic use that provides nil or minimal health benefit is a wise way to slow the spread of antibiotic resistant bacteria

All antibiotic use helps to select for the survival and spread of antibiotic-resistant bacteria within a community. However, it is only the inappropriate use of antibiotics that we should try to reduce. There are some good reasons why community rates of antibiotic use are higher in Aotearoa New Zealand than in many other nations. For example,

in Aotearoa New Zealand a relatively high proportion of the population are young, and a high proportion suffer socio-economic deprivation.

However, there is plenty of evidence that in all communities in Aotearoa New Zealand a large proportion of antibiotic use provides nil or minimal benefit. In developed countries, antibiotic "treatment" of people with viral respiratory tract infections comprises the largest proportion of inappropriate antibiotic dispensing.[23,24] A 2014 study reported that an antibiotic was dispensed following 61% (31,082/50,691) of consultations for acute respiratory tract infections at 111 general practices in Aotearoa New Zealand[25] (Figure 4). In comparison with many other countries, this is a very high rate of antibiotic prescribing for patients with acute respiratory tract infections.

A commonly used measure of the relative amount of inappropriate prescribing of antibiotics for people with acute respiratory tract infections is the proportional increase in dispensing of antibiotics during the colder months (April–September) when compared with dispensing of antibiotics during the warmer months (October–March). Total per capita antibiotic dispensing was 26% higher in the winter months than in the summer months in Aotearoa New Zealand during 2014–2015[26] (Figure 5). The winter increase in antibiotic dispensing was broadly similar regardless of ethnicity: 35% in Asian people, 31% in Middle Eastern, Latin American and African people, 29% in Pacific people, 28% in Māori people and 25% in European people.[26]

Much lower winter increases in antibiotic dispensing are seen in some other nations such as Denmark (12%) and the UK (14%).[26] These data strongly suggest that there is a clear opportunity to achieve significant reductions in inappropriate antibiotic dispensing in all ethnic groups in Aotearoa New Zealand.

Large UK studies strongly suggest that reducing community antibiotic consumption is safe

It is likely that most doctors will be concerned that any reduction in their per capita rate of antibiotic prescribing will create an increased risk of adverse outcomes for their patients. However, this is an understandable misconception. A study of 45 million person-years of observation at 610 UK general practices during 2005 to 2014 evaluated the safety of a policy to reduce antibiotic prescribing for respiratory tract infections in primary care. Slightly more than 50% of patients who presented with a respiratory tract infection were prescribed an antibiotic. The study suggested that, for a practice with 7,000 registered patients, a 10% reduction in the rate of antibiotic prescribing for patients presenting with respiratory tract infections would result in one more case of pneumonia each year, and one more case of peritonsillar abscess each decade.[23] A subsequent larger study of 66 million person-years of follow-up at 706 UK general practices during 2002 to 2017, by the same UK team, found no association between rates of antibiotic prescribing for any indication and subsequent risk of serious bacterial infections. Patients who attended practices that prescribed antibiotics less frequently did not have a higher rate of serious bacterial infections. This study reported that the number needed to treat to prevent one episode of sepsis was almost 30,000 in children aged 0–4, and was >250 in men aged ≥85 years, and >350 in women aged ≥85 years.[27]

A recent Aotearoa New Zealand study also suggests that reducing antibiotic treatment is safe

There was an overall 36% reduction in the number of antibiotic prescriptions dispensed in the community during weeks 15–20 in 2020—early in the COVID-19 epidemic—compared to the same 5-week periods in 2017, 2018 and 2019. The magnitude of the reductions during 2020 when compared with 2019 was 29% in Māori, 44% in Pacific people, 47% in Asian people and 25% in people of all other ethnicities. These large reductions in community antibiotic dispensing were not associated with any subsequent increase in admissions to hospital for pneumonia, peritonsillar abscess or rheumatic fever.[28]

Community antibiotic dispensing has been reducing in Aotearoa NZ in recent years

The per capita annual rate of community antibiotic dispensing increased steadily (by about 5% per year) in Aotearoa New Zealand until 2015, but since then it has declined. The average annual decline in Aotearoa New Zealand between 2015 and 2018 (4.6%) was broadly similar to the magnitude of the decline seen during the same period in Australia (6.5%), Denmark (3.6%) and the UK (3.2%)[15] (Figure 6).

The rate of decline was greater in children aged

Figure 6: Rates of total community antibiotic dispensing in Aotearoa New Zealand and other countries during 2013–2018.[15]

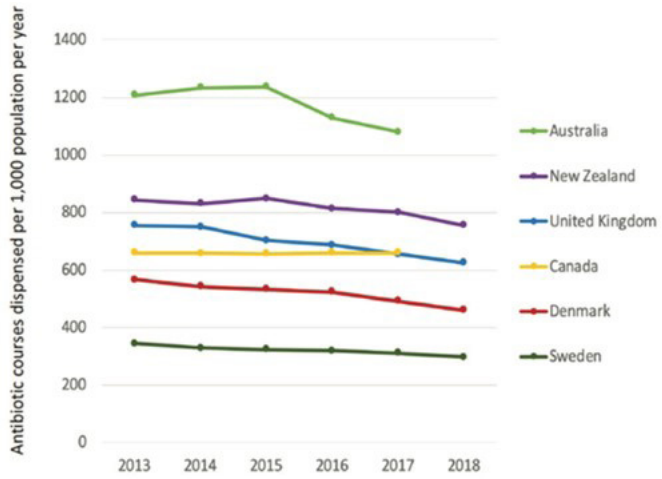


Figure 7: Rates of total community antibiotic dispensing in relation to patients' (a) ethnicity and (b) level of socio-economic deprivation (1 = least deprived, 5 = most deprived) in Aotearoa New Zealand during 2013–2018.[15]

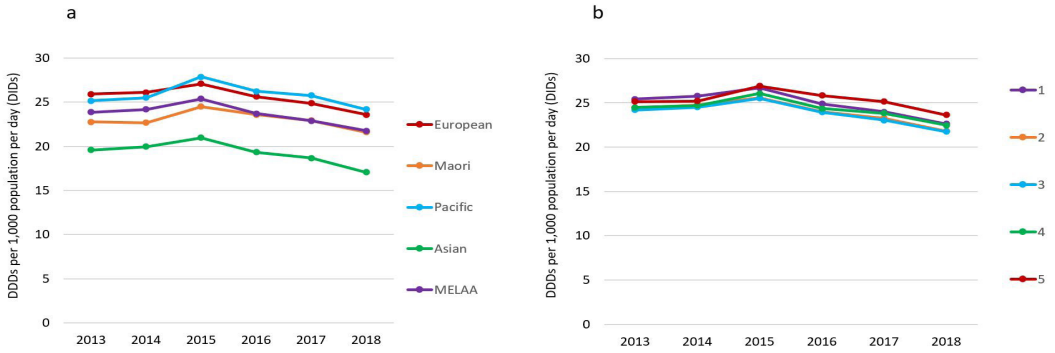


Figure 8: Percentage change between 2015 and 2018 in the rate of community antibiotic dispensing for each DHB (a) and each PHO (b).[15]

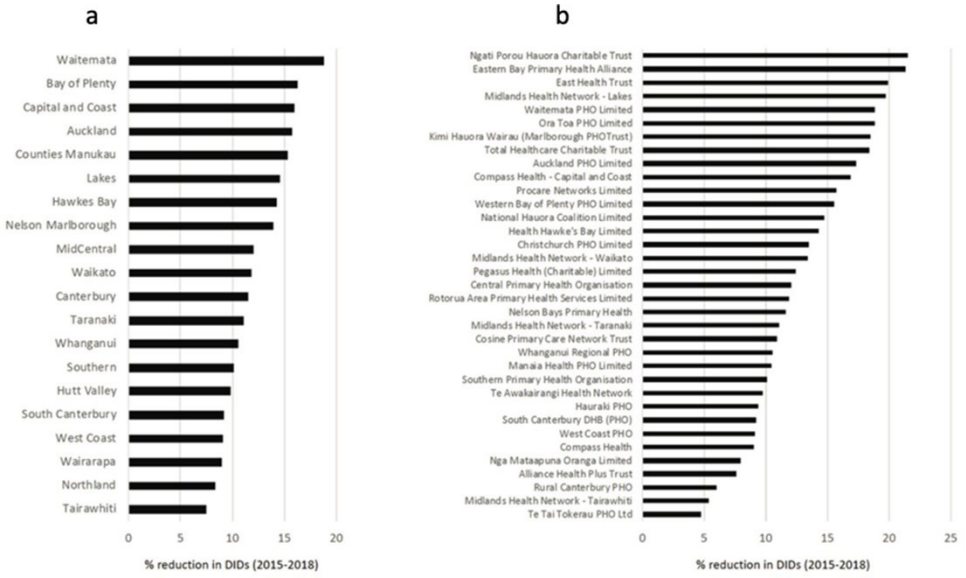
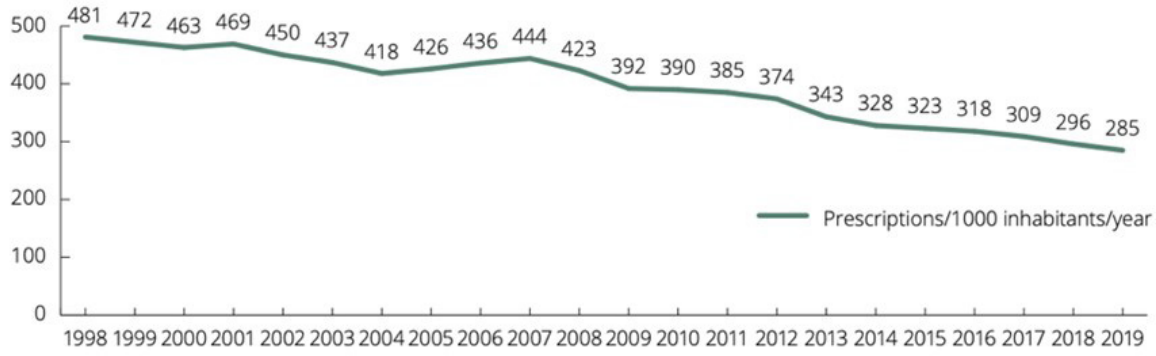


Figure 9: Swedish annual rate of community antibiotic dispensing 1998–2019.[32]



0–4 years than in other age groups. The rate of decline was similar regardless of patients' ethnicity or level of socio-economic deprivation (Figure 7).[15]

The rate of decline in total community antibiotic dispensing varied considerably with regard to primary health organisations (PHO), and to a lesser degree with regard to District Health Board (DHB)[15] (Figure 8).

The large declines in rates of community antibiotic dispensing in all PHOs and all DHBs between 2015 and 2018 suggest that there is a widespread, sustained awareness of the safety, and of the benefits, of reducing inappropriate antibiotic dispensing in Aotearoa New Zealand.

Recommending goals for reductions in community antibiotic dispensing has been very successful in France, England and Sweden

In 2002, the French National Health Insurance launched a long-term, nation-wide campaign to decrease total antibiotic use in the community by 25%. During the next 5 years there was a 26.5% reduction in the number of antibiotic prescriptions.[29]

In 2015, the National Health Service England stated that it would financially reward clinical commissioning groups (CCGs; the English "equivalent" of DHBs) for improvements in the quality of the services they provide in primary care. To be eligible for the Quality Premium payment there needed to be a ≥1% reduction in the number of antibiotic prescriptions compared with the number of prescriptions for the same CCG in 2013–2014, and a ≥10% reduction in the number of broad-spectrum antibiotic prescriptions compared with the number for the same CCG in 2013–2014. Twenty-three months after the intervention there had been an overall 8.2% decrease in total antibiotic prescriptions, and an overall 18.9% decrease in broad-spectrum antibiotic prescriptions.[30]

In 2009, when approximately 400 community antibiotic prescriptions were dispensed per 1,000 inhabitants each year, the Swedish antimicrobial stewardship programme set a national target of fewer than 250 antibiotic prescriptions dispensed per 1,000 inhabitants per year.[31] In 2020, this target was achieved in 19 out of 21 regions[32] (Figure 9).

As previously discussed, and as illustrated in Figure 6, the annual rates of antibiotic dispensing in Aotearoa New Zealand during 2013–2018 were approximately 800 per 1,000 inhabitants—2.7 times higher than in Sweden (approximately 300) and approximately 1.5 times higher than in Denmark (approximately 500).[15] Some might suggest that the differences in antibiotic dispensing rates between these two Scandinavian nations and Aotearoa New Zealand relate to the differences in the rates of deprivation in each of these nations. However, in Aotearoa New Zealand in recent years the level of community antibiotic consumption has hardly differed at all in relation to patients' level of socio-economic deprivation[15] (Figure 7b). The least deprived in Aotearoa New Zealand, as well as the most deprived in Aotearoa New Zealand, all have rates of community antibiotic dispensing that are very much higher than the average rates in Sweden and Denmark.

Improved community antibiotic prescribing can reduce health inequities in Aotearoa New Zealand

The rates of admission to hospital for an infectious disease during 1989–2008 were approximately

twice as high in Māori and Pacific people as in people of other ethnicities in Aotearoa New Zealand,[33] and the rates of admission to hospital for a first episode of rheumatic fever during 2000–2018 were 24 times higher for Pacific and 12 times higher for Māori people than for people of European and other ethnicities.[34] Reducing rates of community antibiotic prescribing for serious bacterial infections, and for *S. pyogenes* pharyngitis, would be certain to worsen health outcomes for Māori and Pacific people. Instead, we need to increase rates of community antibiotic prescribing for all Māori and Pacific people with conditions that require antibiotic treatment.[25]

However, Māori and Pacific people who present to their doctor with conditions that do not require antibiotic treatment deserve the same high-quality treatment as anyone else, and should not be prescribed an antibiotic when it will provide no benefit. As previously discussed, and as illustrated in Figure 5, antibiotic prescribing for Māori and Pacific people increases dramatically during the winter, as it does for people of other ethnicities. Reducing this antibiotic treatment of people with winter upper respiratory tract infections, whether they are Māori or Pacific peoples or other, should be a goal for all general practitioners.

Finally, the fact that the Ngāti Porou Hauora Charitable Trust and the Eastern Bay Primary Health Alliance were the two PHOs with the greatest reductions in rates of community antibiotic dispensing during 2015–2018[15] (Figure 7) suggests that Māori health organisations, and the communities that they serve, see advantages in reducing inappropriate antibiotic prescribing. The fact that community antibiotic dispensing reduced by approximately 22% over a period of 3 years in both these PHOs suggests that the doctors serving these communities have found many opportunities to improve their prescribing practices.

Conclusion

Continued high rates of community antibiotic dispensing will lead to ever higher rates of antibiotic resistance and worse health outcomes for people with infectious diseases in Aotearoa New Zealand in the coming decades. Comparisons with other developed nations suggest that total community antibiotic dispensing could safely be reduced by at least 25% in Aotearoa New Zealand, principally by reducing antibiotic prescribing for people with self-limiting respiratory tract infections, regardless of ethnicity.

However, there is a pressing need to increase the rates of antibiotic treatment for Māori and Pacific people with infectious diseases that do benefit from antibiotic treatment. Despite the incidence of many treatable infectious diseases being disproportionately high in Māori and Pacific people, they currently do not receive commensurately higher rates of community antibiotic prescribing. Therefore, when caring for Māori and Pacific people, healthcare providers need to increase their antibiotic prescribing for those conditions in which antibiotic treatment will be helpful, and decrease their prescribing for those conditions in which antibiotic treatment provides no benefit.

Healthcare providers should take every opportunity to educate their patients about the need for changes in antibiotic prescribing in Aotearoa New Zealand. Reducing the frequency with which patients with self-limiting respiratory tract infections present to their healthcare providers will have benefits for patients and for healthcare providers. Patients will also benefit from increased knowledge of those conditions for which antibiotic treatment is beneficial.

COMPETING INTERESTS
None.

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REFERENCES

1. Royal New Zealand College of General Practitioners. Antibiotics and antimicrobial resistance: avoiding a post-antibiotic era [Internet]. 2015 May 3 [cited 2023 Oct 5]. Available from: https://www.rnzcgp.org.nz/gpdocs/New-website/Advocacy/05.2015-Antibiotics-and-antimicrobial-resistance-Policy-brief-1.pdf.
2. Royal Society Te Apārangi. Antimicrobial Resistance [Internet]. 2017 [cited 2023 Oct 5]. Available from: https://www.royalsociety.org.nz/what-we-do/our-expert-advice/all-expert-advice-papers/antimicrobial-resistance/.
3. Manatū Hauora – Ministry of Health and Manatū Ahu Matua – Ministry for Primary Industries. New Zealand Antimicrobial Resistance Action Plan [Internet]. 2017 [cited 2023 Oct 5]. Available from: https://www.health.govt.nz/system/files/documents/publications/new-zealand-antimicrobial-resistance-action-plan.pdf.
4. Office of the Prime Minister's Chief Science Advisor. Kotahitanga: Uniting Aotearoa against infectious disease and antimicrobial resistance [Internet]. 2021 [cited 2023 Oct 5]. Available from: https://www.pmcsa.ac.nz/files/2022/06/OPMCSA-AMR-Full-report-FINAL-V3-PDF.pdf.
5. Upton A, Lang S, Heffernan H. Mupirocin and Staphylococcus aureus: a recent paradigm of emerging antibiotic resistance. J Antimicrob Chemother. 2003;51(3): 613-7. https:// doi: 10.1093/jac/dkg127.
6. The Institute of Environmental Science and Research Ltd (ESR). Sexually Transmitted Infections in New Zealand: Annual Surveillance Report 2016. Porirua, New Zealand: ESR; 2019 [cited 2024 Mar 6]. Available from: https://www.esr.cri.nz/media/plmffoo2/final_2016_sti_annualreport-1.pdf.
7. Blakiston M, Heffernan H, Roberts S, Freeman J. The clear and present danger of carbapenemase-producing Enterobacteriaceae (CPE) in New Zealand: time for a national response plan. N Z Med J. 2017;130(1454):72-9.
8. Freeman JT, McBride SJ, Nisbet MS, et al. Bloodstream infection with extended-spectrum beta-lactamase-producing Enterobacteriaceae at a tertiary care hospital in New Zealand: risk factors and outcomes. Int J Infect Dis. 2012;16(5):e371-4. doi: 10.1016/j.ijid.2012.01.008.
9. Institute of Environmental Science and Research (ESR). Antimicrobial Resistance (AMR): Acquired carbapenemsae producing organisms [Internet]. [cited 2024 Mar 7]. Available from: https://www.esr.cri.nz/our-research/nga-kete/infectious-disease-intelligence/antimicrobial-resistance-amr/.
10. Chen HY, Jean SS, Lee YL, et al. Carbapenem-Resistant Enterobacterales in Long-Term Care Facilities: A Global and Narrative Review. Front Cell Infect Microbiol. 2021 Apr 23;11:601968. doi: 10.3389/fcimb.2021.601968.
11. Livorsi DJ, Chorazy ML, Schweizer ML, et al. A systematic review of the epidemiology of carbapenem-resistant Enterobacteriaceae in the United States. Antimicrob Resist Infect Control 2018;7:55. doi: 10.1186/s13756-018-0346-9.
12. Review on Antimicrobial Resistance. Antimicrobial Resistance: Tackling a crisis for the health and wealth of nations [Internet]. 2014 [cited 2023 Oct 5]. Available from: hhttps://wellcomecollection.org/works/rdpck35v.
13. Duffy E, Ritchie S, Metcalfe S, et al. Antibacterials dispensed in the community comprise 85%-95% of total human antibacterial consumption. J Clin Pharm Ther. 2018;43(1):59-64. doi: 10.1111/jcpt.12610.
14. European Centre for Disease Prevention and Control. Antimicrobial consumption - Annual Epidemiological Report for 2018 [Internet]. Stockholm: ECDC; 2019 [cited 2023 Oct 25]. Available from: https://www.ecdc.europa.eu/en/publications-data/surveillance-antimicrobial-consumption-europe-2018.
15. Thomas M, Tomlin A, Duffy E, Tilyard M. Reduced community antibiotic dispensing in New Zealand during 2015-2018: marked variation in relation to primary health organisation. N Z Med J. 2020;133(1518):33-42.
16. Institute of Environmental Science and Research Ltd (ESR). Antimicrobial susceptibility data from hospital and community laboratories, 2018 [Internet]. [cited 2023 Oct 25]. Available from: https://www.esr.cri.nz/assets/1Reports/

Surveillance-Datasets/General-antimicrobial-susceptibility-data/antimicrobial-susceptibility-data-2018-esr.pdf.
17. European Centre for Disease Prevention and Control. Surveillance of antimicrobial resistance in Europe 2018 [Internet]. Stockholm: ECDC; 2019 [cited 2023 Oct 25]. Available from: https://www.ecdc.europa.eu/en/publications-data/surveillance-antimicrobial-resistance-europe-2018.
18. Broom M, Best E, Heffernan H, et al. Outcomes of adults with invasive meningococcal disease with reduced penicillin susceptibility in Auckland 2004-2017. Infection. 2023;51:425-32. https://doi: 10.1007/s15010-022-01897-6.
19. Ma F, Xu S, Tang Z, et al. Use of antimicrobials in food animals and impact of transmission of antimicrobial resistance on humans. Biosafety and Health 2021;3:32-8. http://dx.doi.org/10.1016/j.bsheal.2020.09.004
20. Hillerton JE, Irvine CR, Bryan MA, et al. Use of antimicrobials for animals in New Zealand, and in comparison with other countries. New Zealand Veterinary Journal. 2017;65(2):71-77. http://dx.doi.org/10.1080/00480169.2016.1171736.
21. Scoop. Veterinarians set antibiotic goals for animals [Internet]. New Zealand Veterinary Association; 2015 Jul 21 [cited 2023 Oct 25]. Available from: https://www.scoop.co.nz/stories/SC1507/S00059/veterinarians-set-antibiotic-goal-for-animals.htm.
22. Manatū Ahu Matua – Ministry for Primary Industries. 2022 Antibiotic Agricultural Compound Sales Analysis [Internet]. 2023 [cited 2023 Oct 25]. Available from: https://www.mpi.govt.nz/dmsdocument/59719-2022-Antibiotic-Agricultural-Compound-Sales-Analysis.
23. Gulliford MC, Moore MV, Little P, et al. Safety of reduced antibiotic prescribing for self-limiting respiratory tract infections in primary care: cohort study using electronic health records. BMJ. 2016;354:i3410. https://doi: 10.1136/bmji3410.
24. Harris AM, Hicks LA, Qaseem A, et al. Appropriate Antibiotic Use for Acute Respiratory Tract Infection in Adults: Advice for High-Value Care From the American College of Physicians and the Centers for Disease Control and Prevention. Ann Intern Med. 2016 Mar 15;164(6):425-34. doi: 10.7326/M15-1840.
25. Thomas M, Whyler N, Tomlin A, Tilyard M. Ethnic disparities in community antibacterial dispensing in New Zealand-is current antibacterial dispensing for Māori and Pacific people insufficient or excessive, or both? N Z Med J. 2019;132(1505):100-4.
26. Whyler N, Tomlin A, Tilyard M, Thomas M. Ethnic disparities in community antibacterial dispensing in New Zealand, 2015. N Z Med J. 2018;131(1480):50-60.
27. Gulliford MC, Charlton J, Boiko O, et al. Safety of reducing antibiotic prescribing in primary care: a mixed-methods study. Southampton (UK): NIHR Journals Library; 2021 May.
28. Duffy E, Thomas M, Hills T, Ritchie S. The impacts of New Zealand's COVID-19 epidemic response on community antibiotic use and hospitalisation for pneumonia, peritonsillar abscess and rheumatic fever. Lancet Reg Health West Pac. 2021;12:100162. doi: 10.1016/j.lanwpc.2021.100162.
29. Sabuncu E, David J, Bernède-Bauduin C, et al. Significant reduction of antibiotic use in the community after a nationwide campaign in France, 2002-2007. PLoS Med. 2009;6(6):e1000084. doi: 10.1371/journal.pmed.1000084.
30. Balinskaite V, Johnson AP, Holmes A, Aylin P. The Impact of a National Antimicrobial Stewardship Program on Antibiotic Prescribing in Primary Care: An Interrupted Time Series Analysis. Clin Infect Dis. 2019 Jul 2;69(2):227-232. doi: 10.1093/cid/ciy902.
31. Mölstad S, Löfmark S, Carlin K, et al. Lessons learnt during 20 years of the Swedish strategic programme against antibiotic resistance. Bull World Health Organ. 2017;95(11):764-73. doi: 10.2471/BLT.16.184374.
32. Government Offices of Sweden. Swedish Strategy to Combat Antibiotic Resistance: 2020–2023 [Internet]. 2020 [cited 2023 Oct 25]. Available from: https://cdn.who.int/media/docs/default-source/antimicrobial-resistance/amr-spc-npm/nap-library/swedish-strategy-to-combat-antimicrobial-resistance-2020-2023.pdf?sfvrsn=dacbb55f_1&download=true.
33. Baker MG, Barnard TL, Kvalsvig A, et al. Increasing incidence of serious infectious diseases and inequalities in New Zealand: a national epidemiological study. Lancet. 2012;379(9821):1112-19. doi: 10.1016/S0140-6736(11)61780-7.
34. Bennett J, Zhang J, Leung W, et al. Rising Ethnic Inequalities in Acute Rheumatic Fever and Rheumatic Heart Disease, New Zealand, 2000-2018. Emerg Infect Dis. 2021 Jan;27(1):36-46. doi: 10.3201/eid2701.191791.

Bariatric surgery in end-stage kidney disease—removing a barrier to transplantation

Mary J Baker, Michael W C Booth, Jason P Robertson, Janak R de Zoysa

Kidney transplantation (KT) allows better survival, improved quality of life and lower costs than dialysis for patients with end-stage kidney disease (ESKD).[1] Assessment of suitability for KT involves synthesis of physical, psychological and behavioural issues. Obesity is viewed as a contraindication to KT, with a cut-off body mass index (BMI) of 40kg/m^2 being used for entry onto the national New Zealand Kidney Allocation Scheme (NZKAS) and many renal transplant centres.[1] Elevated BMI is associated with increased post-operative complications such as delayed graft function, wound infection, wound dehiscence and prolonged hospital stay.[1] Dietary education and intervention are resource intensive and often ineffective for lowering BMI in patients on renal replacement therapy.[2] Paradoxically, patients with an elevated BMI on haemodialysis have better outcomes than those with a lower BMI;[3] however, KT provides clear survival benefit over dialysis even in obese patients.[4]

Here we described the case of a patient on dialysis who underwent bariatric surgery allowing progression to KT.

Case report

A 43-year-old Tongan lady presented to hospital in September 2009 with coughing, dyspnoea and oedema for 1 month. Her BMI was 46kg/m^2 (height 1.59m, weight 117kg). She was found to have new kidney impairment with a serum creatinine of 1060µmol/L, potassium of 5.3mmol/L, haemoglobin of 84g/L, haematuria, proteinuria and normal-sized kidneys on ultrasound. Her creatinine was 115µmol/L in November 2008. An autoimmune screen and viral serology were normal. Renal biopsy showed an immune-complex glomerulonephritis with interstitial nephritis and 30% tubulointerstitial scarring. She was commenced on emergent dialysis and corticosteroids; however, she did not recover kidney function, remaining dialysis-dependent. She progressed to home haemodialysis 4 days a week. Despite regular specialist dietitian input, her BMI never fell to less than 40kg/m^2 (weight range 114kg–123kg). She was referred to the bariatric service in December 2020. She had gastric sleeve surgery in June 2021, which was uncomplicated. By September 2022, her BMI fell to 34kg/m^2 (weight 87.3kg) and she was accepted onto the NZKAS. Between referral and listing she had intensive input from the bariatric and renal service, with over 75 visits. She received a deceased donor KT in October 2022 and remains free of dialysis, on immunosuppression, with a creatinine of 133µmol/L in October 2023 (weight 87.6kg).

Discussion

Obesity is a worldwide epidemic. The Aotearoa New Zealand Health Survey 2020/2021 identified that over one in three adults were obese, with prevalence more common in Pacific peoples (71.3%), Māori (50.8%) and European (31.9%) than in Asian (18.5%) adults.[5] This represents a dramatic increase over the last 15 years, when just over a quarter of adults were obese (26.5% 2006/2007 to 34.3% 2019/2020). Obesity is common in patients on dialysis and is one of the most common reasons for not entering onto the kidney transplant list. In our district, we currently have 61 patients active on the NZKAS with 41 other patients having a raised BMI preventing listing. A systematic review of literature has demonstrated that bariatric surgery in ESKD patients has similar post-operative weight loss to that of the general population;[6] however, it is associated with increased peri-operative mortality and cardiac events.[7] The annual cost of dialysis is estimated at NZ$115,712. Sleeve gastrectomy and related follow-ups are estimated to cost $23,000, while the first year's costs of transplantation is $107,361 but then reduces to $10,000–$15,000 annually from the

second year onwards.[8] Thus, within 2 years there are economic benefits from bariatric surgery facilitating renal transplantation. Overseas, bariatric surgery for patients with ESKD is steadily increasing;[9] however, an unanswered question remains whether the risk of bariatric surgery in patients with ESKD outweighs the gain, with the potential for kidney transplantation.

In Aotearoa New Zealand, there is a steady increase in privately and publicly funded bariatric surgery.[10] One issue is whether patients with fewer comorbidities should be prioritised for surgery over higher risk patients, such as those with ESKD;[10] however, this may still disadvantage patients who can gain significant improvement in quality of life and quantity of life. Here we present a patient with ESKD on dialysis who had bariatric surgery and highlight that access to this important procedure may remove a further barrier and allow successful kidney transplantation.

COMPETING INTERESTS
There are no conflicts of interest in the submission of this manuscript.
Verbal consent was obtained from the patient for presentation of this case.

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REFERENCES
1. Pham PT, Pham PA, Pham PC, et al. Evaluation of adult kidney transplant candidates. Semin Dial. 2010 Nov-Dec;23(6):595-605. doi: 10.1111/j.1525-139X.2010.00809.x. PMID: 21175834.
2. DiCecco SR, Francisco-Ziller N. Obesity and organ transplantation: successes, failures, and opportunities. Nutr Clin Pract. 2014 Apr;29(2):171-91. doi: 10.1177/0884533613518585. PMID: 24503157.
3. Port FK, Ashby VB, Dhingra RK, et al. Dialysis dose and body mass index are strongly associated with survival in hemodialysis patients. J Am Soc Nephrol. 2002 Apr;13(4):1061-1066. doi: 10.1681/ASN.V1341061. PMID: 11912267.
4. Gill JS, Lan J, Dong J, et al. The survival benefit of kidney transplantation in obese patients. Am J Transplant. 2013 Aug;13(8):2083-90. doi: 10.1111/ajt.12331. PMID: 23890325.
5. Manatū Hauora –Ministry of Health. New Zealand Health Survey: Annual Data Explorer [Internet]. 2022 [cited 2023 Nov]. Available from: https://minhealthnz.shinyapps.io/nz-health-survey-2021-22-annual-data-explorer.
6. Guggino J, Coumes S, Wion N, et al. Effectiveness and Safety of Bariatric Surgery in Patients with End-Stage Chronic Kidney Disease or Kidney Transplant. Obesity (Silver Spring). 2020 Dec;28(12):2290-2304. doi: 10.1002/oby.23001. PMID: 33230959.
7. Palamuthusingam D, Hawley CM, Pascoe EM, et al. Trends in Rates of Surgery and Postoperative Mortality Among Patients Receiving Chronic Kidney Replacement Therapy: A Population-based Cohort Study. Ann Surg. 2022 Dec 1;276(6):1002-1010. doi: 10.1097/SLA.0000000000005694. PMID: 36052682.
8. Hogan S, Tuano K. Transforming lives and saving money: The golden opportunity of kidney transplants and the system changes needed to lift the numbers [Internet]. New Zealand Institute of Economic Research; 2021 [cited 2023 Nov]. Available from: https://www.kidney.health.nz/resources/files/links/nzier-transforming-lives-and-saving-money-final-report-5-november-2021.pdf.
9. Sheetz KH, Woodside KJ, Shahinian VB, et al. Trends in Bariatric Surgery Procedures among Patients with ESKD in the United States. Clin J Am Soc Nephrol. 2019 Aug 7;14(8):1193-1199. doi: 10.2215/CJN.01480219. PMID: 31345840; PMCID: PMC6682821.
10. Garrett M, Poppe K, Wooding A, Murphy R. Private and Public Bariatric Surgery Trends in New Zealand 2004-2017: Demographics, Cardiovascular Comorbidity and Procedure Selection. Obes Surg. 2020 Jun;30(6):2285-2293. doi: 10.1007/s11695-020-04463-x. PMID: 32166697.

Laboratory findings in acute bacterial meningitis and acute viral encephalitis

Hasan Tahsin Gozdas, Ahmet Dogan

Dear sir,

We read the case report of Wright and Fox-Lewis[1] recently published in the *New Zealand Medical Journal* with great interest. They reported the first case of imported human rabies in Aotearoa New Zealand. Their patient presented with a 3-day history of fever, vomiting and inability to swallow food or fluids. We have a criticism about the empirical treatment of this patient.

Empirical treatment was started for this patient with ceftriaxone, clarithromycin and aciclovir, suspecting acute meningoencephalitis. Blood tests of this patient revealed leukocytosis with neutrophilia and normal C-reactive protein (CRP) level. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis, normal protein and mildly elevated glucose. In CSF examination of acute bacterial meningitis cases, neutrophilic pleocytosis, low glucose and very high protein levels are expected. In addition, a high CRP level is almost always seen in acute bacterial central nervous system (CNS) infection cases. In CSF analysis of acute viral encephalitis cases, lymphocytic pleocytosis, normal glucose and normal or mildly elevated protein levels are expected.[2] Their patient's blood and CSF changes were compatible with acute viral encephalitis rather than acute bacterial meningitis. We wonder whether CSF gram staining was done to discriminate bacterial and viral aetiology. We are of the opinion that indication of antibacterial treatment with ceftriaxone and clarithromycin is not clear in this patient.

COMPETING INTERESTS

No conflict of interest was declared by the authors.

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https://nzmj.org.nz/journal/vol-137-no-1592/laboratory-findings-in-acute-bacterial-meningitis-and-acute-viral-encephalitis

REFERENCES

1. Wright H, Fox-Lewis A. A case of imported rabies in Aotearoa New Zealand. N Z Med J. 2023 Dec 15;136(1587):108-13.
2. Young N, Thomas M. Meningitis in adults: diagnosis and management. Intern Med J. 2018 Nov;48(11):1294-307. doi: 10.1111/imj.14102.

Response to: Laboratory findings in acute bacterial meningitis and acute viral encephalitis

Hamish Wright, Andrew Fox-Lewis

Thank you for your interest in the paper. CSF gram staining was performed, and no organisms were seen. We agree that the patient's blood and CSF changes were not typical of acute bacterial meningitis. Differential diagnoses included acute viral encephalitis. In this case, empirical meningoencephalitis treatment was commenced on day 4 post-symptom onset when the patient was admitted to the local hospital closest to where his vessel was in port. Lumbar puncture and CSF analysis were undertaken on day 5 following transfer to the intensive care unit at the regional tertiary hospital. The CSF was obtained for analysis after approximately 24 hours of antibacterial treatment, polymerase chain reaction testing failed to detect any common viral causes of encephalitis, and the CSF glucose value was confounded by concurrent hyperglycaemia. Given the patient was critically unwell, the clinical decision was made to err on the side of caution and continue empirical treatment for both acute viral encephalitis and a broad range of bacterial causes of meningoencephalitis while awaiting investigations. CSF parameters cannot confidently exclude bacterial meningitis, especially if pre-treated, and 10% of cases of bacterial meningitis have a lymphocytic predominance.[1] The most likely indication for commencing a macrolide antibiotic was ground-glass pulmonary infiltrate that was seen on computed tomography chest imaging on day 4, and was later thought to be non-significant.

It is valuable to emphasise that empiric treatment of meningitis for the most common pathogens normally consists of ceftriaxone with dexamethasone. Vancomycin is added if risk factors are present for infection with strains of *Streptococcus pneumoniae* with reduced susceptibility to ceftriaxone. Benzylpenicillin or amoxicillin are added if risk factors are present for infection with *Listeria monocytogenes*. Acyclovir is often also added to empiric CNS treatment, to cover predominantly for HSV encephalitis.[1]

COMPETING INTERESTS
Nil.

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REFERENCES

1. Young N, Thomas M. Meningitis in adults: diagnosis and management. Intern Med J. 2018 Nov;48(11):1294-307. doi: 10.1111/imj.14102.

Are two shocks better than one? Aotearoa New Zealand emergency medical services implement a new defibrillation strategy: implications for around nine patients per week

Bridget Dicker, Sarah Maessen, Andy Swain, Elena Garcia, Tony Smith

Aotearoa New Zealand emergency medical services (EMS) began routinely using new external defibrillation strategies for the treatment of refractory ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) from January 2024. These strategies are vector change (VC) defibrillation and double sequential external defibrillation (DSED). DSED provides rapid sequential shocks using two defibrillators with the pads attached in two different vectors, one in an anterior-lateral and the other in an anterior-posterior position. Around 20% of patients with ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) do not respond to three or more single defibrillation attempts (i.e., are in refractory VF/VT) and have lower survival rates than those who respond to standard (single defibrillation every two minutes) resuscitation approaches.[1] Consideration of DSED and VC defibrillation (switching defibrillation pads from anterior-lateral to an anterior–posterior position) has been suggested for these patients in the 2023 update of the International Consensus on Cardiopulmonary Resuscitation (ILCOR) and Emergency Cardiovascular Care Science with Treatment Recommendations.[2]

Though the task force noted that DSED was already in use by some EMS, ILCOR recommended against the routine use of DSED in 2020 on the basis that there was a lack of high-quality evidence to support its use.[1] In the updated review, one additional randomised controlled trial was identified that utilised DSED and VC defibrillation strategies,[2,3] leading to an update in guidelines while acknowledging that this was a weak recommendation with low certainty of evidence.[1,2] The cluster-randomised trial was undertaken in Canada with a total of 405 patients randomised to one of three strategies: standard defibrillation with the pads in the usual anterior-lateral position, DSED or VC. Refractory VT/VF was defined as three consecutive shocks being delivered by paramedics or participating fire services, and the primary outcome of the trial was survival to hospital discharge. The trial showed a convincing survival benefit of DSED (31/143 [21.7%], RR 2.21 [1.33–3.67]) and VC (38/125 [30.4%], RR 1.71 [1.01–2.88]) over standard defibrillation 18/135 (13.3%). Limitations in this study are also described in an accompanying editorial to this trial.[4] Post-resuscitation care was not described for trial participants and could vary significantly between groups, and the trial was terminated early, with the power analysis indicating that 930 patients were needed but only 405 were analysed. As well as those recruited for the trial, this included 152 patients from an internal pilot study that had subtly different inclusion criteria to the main trial and did not include patients with pulseless VT, shocks administered by fire personnel or additional EMS services to determine refractory VF, or resuscitations started by non-participating EMS agencies.[4,5]

In Aotearoa New Zealand, EMS work under standing orders called the Clinical Procedures and Guidelines (CPGs).[6] The CPGs are developed by the National Ambulance Sector Clinical Working Group, who have considered this study in depth alongside the ILCOR guidelines and incorporated DSED and VC into the CPGs to become routine practice from January 2024.[6] Prior to being able to use this protocol in practice, EMS personnel must have undertaken face-to-face education, simulation and online training in the performance of DSED and VC. When undertaking DSED, a single operator activates the defibrillators in sequence, with one hand moving from the first defibrillator to the second. The CPGs stipulate that if VF/VT

Table 1: Number of patients from 1 July 2020 to 30 June 2023 that would have met the criteria for DSED or VC and the current rate of survival.

Total n=1,390	**N/N (%)**
Return of spontaneous circulation (ROSC) on hospital handover	
YES	359/1,390 (26%)
Survival to 30 days	
Survived	198/1,390 (14%)

persists beyond three shocks then VC/DSED is to be administered. If one manual defibrillator is present, then VC is administered; if two are present, then DSED is administered. Outside of Canada, Aotearoa New Zealand is the first country to implement these resuscitation treatments as routine protocol in cardiac arrest. As the technique is new to Aotearoa New Zealand and at the forefront of current defibrillation strategies, patient outcomes and implementation are being carefully monitored through transmission of defibrillator recordings for clinical review.

Though the balance of evidence suggests this new strategy may have survival benefits for some patients, one of the key factors that will be monitored during clinical review of DSED/VC cases is any potential impact on no-flow/pauses in cardio-pulmonary resuscitation (CPR) that may occur when co-ordinating the switching of defibrillation pads into the anterior-posterior position for DSED or VC. It is well established that pauses in compressions during CPR have a significant impact on patient survival, and this was one of the key considerations when Aotearoa New Zealand EMS implemented high performance CPR in 2019.[7–9] In addition, key changes over the years in resuscitation guidelines relating to defibrillation strategies have evolved in response to reducing the hands-off chest/no-flow time. In particular, this includes the shift post-2010 from three stacked shocks to the delivery of a single shock, with CPR initiated as soon as possible post-shock.[10,11]

Data from the Aotearoa New Zealand Paramedic care Collection (ANZPaCC), the Aotearoa New Zealand database derived from EMS electronic patient report forms (ePRFs), indicates that Aotearoa New Zealand EMS attended 1,390 patients over a 3-year period (1 July 2020 to 30 June 2023) who would have met the criteria for VC/DSED—approximately nine patients a week (Table 1).

As emphasised in the ILCOR guidelines, evidence for these new external defibrillation strategies is still emerging, and implementation in Aotearoa New Zealand is a key opportunity to understand how they could translate from a controlled research setting to the real world. Our existing survival statistics align closely with the findings of the Cheskes et al. study, revealing a 14% survival rate through standard defibrillation.[3] We are keenly observing whether the adoption of this new strategy can emulate the enhanced survival rates observed in their trial. However, implementing and monitoring this new protocol presents a challenge, as it effectively transforms into a before-and-after observational study that inherently carries limitations, such as observational bias and confounding factors. Considering the findings of the Cheskes et al. study and the recommendations from ILCOR, we concluded that there was a lack of equipoise, making it unethical to randomise patients to "standard care" in this scenario. Furthermore, due to the relatively small number of survivors annually (less than 100) at a national level, a step-wise cluster introduction would not have had sufficient power to detect outcome differences unless carried out over an extended period (several years).

While we are closely reviewing efficacy of this new treatment strategy, we maintain a low threshold to default to previous practice if harm becomes evident.

In conclusion, Aotearoa New Zealand is the first country outside Canada to routinely implement DSED, and this could affect the care of up to nine patients a week.

COMPETING INTERESTS
All of the authors are employees of Hato Hone St John or Wellington Free Ambulance.

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REFERENCES
1. Nolan JP, Maconochie I, Soar J, et al. Executive summary: 2020 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Circulation. 2020;142(16_suppl_1):S2-S27. doi: 10.1161/CIR.0000000000000890.
2. Berg KM, Bray JE, Ng KC, et al. 2023 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: Summary From the Basic Life Support; Advanced Life Support; Pediatric Life Support; Neonatal Life Support; Education, Implementation, and Teams; and First Aid Task Forces. Circulation. 2023;148(24):e187-e280. doi: 10.1161/CIR.0000000000001179.
3. Cheskes S, Verbeek PR, Drennan IR, et al. Defibrillation strategies for refractory ventricular fibrillation. N Engl J Med. 2022;387(21):1947-56. doi: 10.1056/NEJMoa2207304.
4. Sasson C, Haukoos J. Defibrillation after Cardiac Arrest—Is It Time to Change Practice? N Engl J Med. 2022;387(21):1995-6. doi: 10.1056/NEJMe2213562.
5. Drennan IR, Dorian P, McLeod S, et al. DOuble SEquential External Defibrillation for Refractory Ventricular Fibrillation (DOSE VF): study protocol for a randomized controlled trial. Trials. 2020;21(1):977. doi: 10.1186/s13063-020-04904-z.
6. National Ambulance Sector Clinical Working Group. Emergency Ambulance Service Clinical Procedures and Guidelines, Version: 1.0.4.1 [Internet]. NZ: National Ambulance Sector Working Group; 2023 [cited 2023 Nov]. Available from: https://cpg.stjohn.org.nz/tabs/guidelines?_ga=2.229533532.1908072009.1674680333-1630428122.1628726924
7. Deakin CD, Koster RW. Chest compression pauses during defibrillation attempts. Curr Opin Crit Care. 2016;22(3):206-11. doi: 10.1097/MCC.0000000000000310.
8. Bobrow BJ, Clark LL, Ewy GA, et al. Minimally interrupted cardiac resuscitation by emergency medical services for out-of-hospital cardiac arrest. Jama. 2008;299(10):1158-65. doi: 10.1001/jama.299.10.1158.
9. New Zealand Guidelines Group. The Aotearoa New Zealand Model of High Performance CPR [video on the Internet]. Hato Hone St John, Wellington Free Ambulance, Auckland University of Technology (AUT), Fire & Emergency New Zealand. Auckland (NZ): AUT Paramedicine; 2019 [cited 2023 Nov]. Available from: https://youtu.be/7L45-AsO4bA.
10. Rea TD, Helbock M, Perry S, et al. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. Circulation. 2006;114(25):2760-5. doi: 10.1161/CIRCULATIONAHA.106.654715.
11. Nolan JP, Hazinski MF, Billi JE, et al. Part 1: executive summary: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation. 2010;81 Suppl 1(1):e1-e25. doi: 10.1016/j.resuscitation.2010.08.002.

Pre-hospital, pre-antibiotic blood cultures for patients with suspected sepsis—a feasibility study

Aileen Harwood, Scott Pearson, Julia Howard, Nicole Jones, Rosie Greenlees, Charlotte Broms, Sharon J Gardiner, Simon C Dalton

Prompt pathogen identification and effective antimicrobial treatment are critical to prevent mortality for patients with sepsis (mate whakatāoke).[1] In Aotearoa New Zealand, Hato Hone St John (HHStJ) play a crucial role in early sepsis management, with Waikato Hospital data revealing that 74% of sepsis cases enter hospital via HHStJ.[2] St John Clinical Procedures and Guidelines[3] state that individuals over 12 years of age with a provisional sepsis diagnosis and at least one of 12 high-risk factors (e.g., systolic blood pressure ≤90mmHg) may be given empiric antibiotics if time to definitive care is over 30 minutes. This accommodates delays due to both distance and emergency department (ED) ramping. The recommended antibiotics are amoxicillin/clavulanic acid with or without gentamicin depending on likely site of infection, or ceftriaxone if meningococcal disease is suspected.[3] The guidelines do not require blood cultures (BCs) to be taken before antibiotic administration, as would be best practice, due to logistical (e.g., short shelf-life of BC bottles),[3] clinical (e.g., contamination) and financial (e.g., cost of BC bottles) concerns. This diminishes pathogen detection in BCs taken subsequently in hospital.[4] Given this, and the substantial pressures across emergency services, there is value in identifying initiatives that could improve sepsis management, including achieving equitable care for those in remote locations.

Here, in a collaboration between HHStJ and Te Whatu Ora – Waitaha Canterbury Emergency Medicine, Microbiology and Infection Management Services at Christchurch Hospital, we conducted a feasibility study to establish a local approach to pre-hospital pre-antibiotic BC collection. We also aimed to assess rates of pathogen and contaminant identification in BCs drawn by HHStJ and ED. This quality improvement initiative was assessed as exempt from Health and Disability Ethics Committee review.

From 21 October 2022 to 21 April 2023, Waitaha Canterbury HHStJ paramedics were asked to draw one set of BCs on insertion of a peripheral intravenous catheter from patients with suspected sepsis before giving intravenous antibiotics as per their guidelines. Paramedics received training on BC collection via resources developed by HHStJ, a microbiology scientist and an emergency medicine specialist. The resources were placed on the HHStJ online learning platform and included a written BC collection protocol, video demonstration and a skill sheet outlining key points for BC collection.

BC collection kits (specimen bag, aerobic and anaerobic culture bottles, vacutainer and request form) were obtained from ED and replaced as used, thus circumventing the logistical and financial barriers mentioned earlier. At handover, paramedics advised ED that BCs had been taken before transferring them with an HHStJ-labelled request form to the laboratory via pneumatic tube. The BCs were registered to ED for follow-up purposes. ED staff were informed of the pilot but were not given explicit advice to draw repeat BCs.

All HHStJ BCs and any subsequent BCs obtained in ED for each patient episode were compiled, reviewed by a clinical microbiologist and analysed descriptively using Microsoft Excel™. A patient episode denotes a separate hospital presentation, rather than a different patient. An isolate was deemed a contaminant following definitions outlined in the recent national BC audit,[5] or as per clinical microbiologist assessment following clinical review.

Table 1 summarises the results of 135 BCs (85 HHStJ, 50 ED) taken from 80 patients across 85 patient episodes. Forty-one patient episodes had repeat BCs in ED, and nine patient episodes had two sets of BCs in ED. Positive results represent both pathogens and probable contaminants.

Of the 85 BCs drawn by HHStJ, 29 were positive and 56 were negative. Of the 29 positive cultures taken by HHStJ, 17 had repeat BCs drawn in ED,

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Table 1: Overall summary of blood culture (BC) results.

<table>
<tr><td>Hato Hone St John (HHStJ) (n=85)</td><td></td><td colspan="4">Emergency Department (ED) (n=50)*</td></tr>
<tr><td></td><td></td><td colspan="2">First set (n=41)</td><td colspan="2">Second set (n=9)</td></tr>
<tr><td rowspan="5">Positive</td><td rowspan="5">29</td><td rowspan="2">Positive</td><td rowspan="2">5</td><td>Positive</td><td>1</td></tr>
<tr><td>Negative</td><td>0</td></tr>
<tr><td colspan="4"></td></tr>
<tr><td rowspan="2">Negative</td><td rowspan="2">12</td><td>Positive</td><td>0</td></tr>
<tr><td>Negative</td><td>3</td></tr>
<tr><td colspan="6"></td></tr>
<tr><td rowspan="5">Negative</td><td rowspan="5">56</td><td rowspan="2">Positive</td><td rowspan="2">2</td><td>Positive</td><td>0</td></tr>
<tr><td>Negative</td><td>1</td></tr>
<tr><td colspan="4"></td></tr>
<tr><td rowspan="2">Negative</td><td rowspan="2">22</td><td>Positive</td><td>0</td></tr>
<tr><td>Negative</td><td>4</td></tr>
</table>

Each result represents a single patient episode. Positive BC results include both pathogens and contaminants.
*BCs were repeated in the ED in 41 patient episodes, with nine patient episodes having two sets of repeat BCs.

with 12 of these testing negative. Of the 56 negative cultures by HHStJ, 24 had repeat BCs in ED, with two testing positive.

Table 2 shows the distribution of microorganisms isolated. *Escherichia coli* was the most common pathogen, identified in six HHStJ BCs and three ED BCs. *Staphylococcus aureus* was isolated in four patient episodes. One of these was methicillin (but not gentamicin) resistant (MRSA) and was detected only by HHStJ; this patient received gentamicin and amoxicillin/clavulanic acid pre-hospital. Identification of *Enterococcus faecalis* by HHStJ, but not ED, in another patient episode facilitated diagnosis and treatment of endocarditis. These cases demonstrate the utility of pre-hospital, pre-antibiotic BC collection.

There were 10 probable contaminants isolated from nine BCs drawn from seven patient episodes. In two patient episodes, probable contaminants were isolated by both HHStJ and ED. In one of these, *Proteus mirabilis*, *Staphylococcus capitis* and *Corynebacterium striatum* were isolated from the HHStJ BC, and *C. striatum* was isolated from the ED BC. *P. mirabilis* was assessed as a pathogen and the others were assessed as probable contaminants. In the other, two different contaminants were isolated (mixed coagulase negative staphylococci by HHStJ and *S. capitis* by ED). The remaining five contaminants were isolated by HHStJ.

Post hoc statistical analyses using Chi-squared tests showed the proportion of positive blood cultures was 2.0 (95% CI = 0.96 to 4.17, p=0.024) times higher in samples taken by HHStJ (29/85, 34%; 95% CI = 24.9% to 44.7%) compared with the first set taken in ED (7/41, 17%; 95% CI = 8.2% to 31.6%). The proportion of contaminated samples was 1.7 (95% CI = 0.37 to 7.77, p=0.268) times higher in samples taken by HHStJ (7/85, 8%; 95% CI = 3.8% to 16.3%) compared with the first set taken in ED (2/41, 4.9%; 95% CI = 0.5% to 17.0%).

After excluding contaminants, the HHStJ true pathogen positivity rate (22/85; 26%) exceeded both the ED (6/50; 12%) and the overall Waitaha Canterbury (6.4%)[5] true positive rates, likely reflecting the increased pre-test probability seen in community patients with suspected sepsis and the benefit of pre-antibiotic BCs. The true positivity and contamination rates found in HHStJ BCs align

Table 2: Distribution of bacteria isolated from the blood cultures (BCs).

Species	Hato Hone St John (HHStJ) (n=31*)	Emergency department (ED) (n=8)	Total (n=39*)
Escherichia coli	6	3	9
Coagulase negative staphylococci**	6	1	7
Beta-haemolytic streptococci	5	1	6
Staphylococcus aureus	4	0	4
Proteus mirabilis	2	1	3
*Corynebacterium striatum***	1	1	2
Bacteroides fragilis	1	0	1
Citrobacter species	0	1	1
Enterobacter cloacae	1	0	1
Enterococcus faecalis	1	0	1
Leclercia adecarboxylata	1	0	1
Proteus vulgaris	1	0	1
*Streptococcus parasanguinis***	1	0	1
Streptococcus pneumoniae	1	0	1

*Thirty-nine isolates were detected in 37 sets of BCs. One BC taken by HHStJ grew both the pathogen *Proteus mirabilis* and two contaminants *S. capitis* and *C. striatum*. The repeat BC in the ED grew *C. striatum* only.
**Assessed as contaminants.

with similar studies overseas.[6,7]

Pre-hospital antibiotics were administered by HHStJ in 91% (77/85) of patient episodes—57% as amoxicillin/clavulanic acid with gentamicin (44/77), 34% as amoxicillin/clavulanic acid alone (26/77), and the remaining 9% as gentamicin alone (4/77), ceftriaxone alone (2/77) or ceftriaxone with gentamicin (1/77). Antibiotics were not administered after BCs in five cases due to hospital proximity and in three cases for an undocumented reason.

A limitation of our approach was that only one set of BCs was drawn by HHStJ before antibiotics instead of the two recommended as best practice.[8] Moving forward, two sets of BCs could be obtained by HHStJ before antibiotic administration. Alternatively, repeat BCs could be taken in ED when indicated to address any shortfall, although a lower positivity rate should be anticipated. We propose that ongoing audits of this quality improvement initiative are undertaken, including time from pre-hospital BC collection and antibiotic administration, to time to BC collection in ED. We also recommend review of choice of antibiotics for suspected sepsis by HHStJ—a single dose of ceftriaxone 2g likely strikes a pragmatic balance between expected benefit and harms.

Overall, our results confirmed that pre-hospital, pre-antibiotic BCs had a high rate of pathogen detection without clinically significant contamination or logistical concerns. Pathogen detection is critical to guide antibiotic choice and regimen. Provided that BCs can be expeditiously collected, we advocate that they should be taken before antibiotic administration.

We are pleased to report that, based on this study, HHStJ have formally approved national implementation of pre-hospital, pre-antibiotic BC collection for patients with suspected sepsis

(in an email from N Jones, Hato Hone St John Tāmaki Makaurau Auckland [Nicole.Jones@stjohn.org.nz] in Nov 2023). This is a positive step towards achieving equity in sepsis-related care for those who live remotely. The approach used will be modelled on that described here, including supply of BC kits from receiving hospital EDs and working collaboratively with clinicians at the receiving hospital and associated infection-related services.

COMPETING INTERESTS
Nil.

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REFERENCES
1. Rüddel H, Thomas-Rüddel DO, Reinhart K, et al. Adverse effects of delayed antimicrobial treatment and surgical source control in adults with sepsis: results of a planned secondary analysis of a cluster-randomized controlled trial. Crit Care. 2022;26(1):51. doi: 10.1186/s13054-022-03901-9.
2. Walland KM, Howard C, Paul O, et al. Raise the Flag I: the impact of a sepsis quality improvement programme on delivery of a sepsis resuscitation bundle at a tertiary hospital in New Zealand. N Z Med J. 2023;136(1587):75-84.
3. National Ambulance Sector Clinical Working Group. Clinical Procedures and Guidelines: Comprehensive Edition. Section 6.2 Sepsis. NZ: St John New Zealand; 2019 [cited 2023 Oct 19]. Available from: https://www.stjohn.org.nz/globalassets/documents/health-practitioners/clinical-procedures-and-guidelines---comprehensive-edition.pdf.
4. Scheer CS, Fuchs C, Gründling M, et al. Impact of antibiotic administration on blood culture positivity at the beginning of sepsis: a prospective clinical cohort study. Clin Microbiol Infect. 2019;25(3):326-331. doi: 10.1016/j.cmi.2018.05.016.
5. Elvy J, Addidle M, Andersson HS, et al. A national audit of performance standards for blood cultures in Aotearoa New Zealand: opportunities for improvement. N Z Med J. 2022;136(1568):65-71.
6. Mikkelsen VS, Gregers MCT, Justesen US, et al. Pre-hospital antibiotic therapy preceded by blood cultures in a physician-manned mobile emergency care unit. Acta Anaesthesiol Scand. 2021;65(4):540-548. doi: 10.1111/aas.13777.
7. Chippendale J, Lloyd A, Payne T, et al. The feasibility of paramedics delivering antibiotic treatment pre-hospital to 'red flag' sepsis patients: a service evaluation. Br Paramed J. 2018;2(4):19-24. doi: 10.29045/14784726.2018.03.2.4.19.
8. Clinical and Laboratory Standards Institute (CLSI). M47-A Principles and Procedures for Blood Cultures; Approved Guideline. Wayne, PA (US): Clinical and Laboratory Standards Institute; 2007 [cited 2022 Oct]. Available from: https://clsi.org/media/1448/m47a_sample.pdf

Should menstrual cycle data be collected during suspected suicide autopsies?

Angie Hoskin, Sarah K McKenzie, Emily B Cooney, Gabrielle Jenkin

Suicide is a global health concern that necessitates comprehensive, critical, compassionate and evidence-based approaches.[1] Gender differences are stark in suicide trends in New Zealand and globally. While men die by suicide at greater rates, women have higher rates of non-fatal suicide attempts.[2] Despite these reported differences, research focussed on suicidal behaviours among women remains scarce. Considering the release of the New Zealand Government's *Women's Health Strategy* in 2023, it seems timely to reconsider potential contributions of female biology to key health outcomes such as suicidal behaviours.[3]

One area that lacks exploration is the relationship between menstruation and suicide. For many people, physical, psychological and social wellbeing are frequently, and for some, catastrophically, disrupted by menstrual cyclicity.[4] Common symptomatic complaints include pain, dysmenorrhea, anxiety, depression and fatigue, which often signal conditions such as endometriosis, premenstrual syndrome and premenstrual dysphoric disorder (PMDD).[5] PMDD has been associated with post-traumatic stress disorder,[6] decreased quality of life and a significantly heightened risk of suicide.[7]

Menstrual health is under-studied, underserved and stigmatised in many countries, including New Zealand. The stigma related to menstruation varies by culture, and in many cultures can lead to shame, isolation and marginalisation, all of which are known risk factors for suicidal behavior.[8] The lack of scientific literature on menstruation and suicide reflects the historical under-representation of research into issues that predominantly affect women. The relative dearth of such research leads to delays in diagnoses, inadequate pain management and insufficient treatment.[9] This can result in scientific inaccuracies, adverse health outcomes and healthcare cost ineffectiveness,[9] and compounds gender inequities in health outcomes and health service access.

Few studies explore the menstrual status of individuals who die by suicide. To our knowledge, with the exception of one study,[10] all research in the past 30 years on this topic originates solely from India. A critical opportunity for focus is the nature of data collected by pathologists at autopsies. Autopsy findings, when analysed in aggregate, can offer an understanding of community healthcare needs, and can reveal disease patterns within specific populations, illness disparities and critical factors that may inform public health decisions and suicide prevention strategies.[11]

During the scoping phase of a PhD, conversations had with pathologists nationally and internationally have revealed significant variability in the collection of menstrual cycle data during autopsies. Some pathologists reported routinely including menstrual cycle status in their autopsy practice, whereas others considered collection of this data irrelevant to the cause of death, and yet some other pathologists reported never considering it. Interestingly, several global pathologists noted they had observed a trend of menstruation present in suicide cases.

Such variability may reflect broader differences between countries in how deaths are investigated, and the role of a pathologist within disparate judicial health systems. In the New Zealand coronial system, all suspected suicide deaths are referred to the coroner, which retains information that the pathologist collects as part of the investigation to determine if the death was a suicide. As not all countries take this approach, differences in death examination may underlie the variability in menstruation status data collection practices. However, if this were the case, then practices within countries with coronial systems should be relatively homogenous regarding menstruation data collection. Anecdotally, this is not what we observed.

To understand this discrepancy in practice further we initiated an international survey of pathologists asking about their professional views

and practices, not limited to confirmed suicide cases but inclusive of all autopsy procedures where a menstrual cycle may be present. The survey has received over 100 responses from pathology professionals with experience in around 60 countries. Initial survey findings suggest that the intersection between menstruation and suicide is poorly understood. We believe that the diversity of pathology methods in investigating menstrual phase status reflects the systemic ambivalence present regarding menstruation in health research and in general.

As we invite discussion on this under-studied topic, we emphasise the importance of empirical research. The absence of comprehensive, rigorous studies on the menstrual cycle and suicide warrants further exploration. We must inquire whether scientific research can confirm or disprove the potential role of the menstrual cycle in deaths by suicide. In a rapidly evolving healthcare landscape, research on the menstrual cycle and suicide can inform evidence-based healthcare and suicide prevention strategies. We call on thoughtful discourse on this vital yet under-examined subject.

COMPETING INTERESTS
Angie Hoskin received a doctoral scholarship from the University of Otago for the manuscript.

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REFERENCES
1. World Health Organization. Suicide worldwide in 2019: Global Health Estimates [Internet]. Geneva: World Health Organization; 2013 [cited 2023 Dec 6]. Available from: https://iris.who.int/bitstream/handle/10665/341728/9789240026643-eng.pdf.
2. Saunders KE, Hawton K. Suicidal behaviour and the menstrual cycle. Psychol Med. 2006 Jul;36(7):901-12. doi:10.1017/S003291706007392.
3. Minister of Health. Women's Health Strategy [Internet]. Wellington: Ministry of Health; 2023 [cited 2023 Nov 20]. Available from: https://www.health.govt.nz/system/files/documents/publications/womens-health-strategy-oct23.pdf.
4. Critchley HOD, Babayev E, Bulun SE, et al. Menstruation: science and society. Am J Obstet Gynecol. 2020 Nov;223(5):624-664. doi:10.1016/j.ajog.2020.06.004.
5. Gopalan P, Albertini E, Amin P, Curley M, Glance J, Kalia S. Chapter 18: Trauma and Reproductive Health. In: Hutner LA, Catapano LA, Nagle-Yang SM, Williams KE, Osborne LM, editors. Textbook of Women's Reproductive Mental Health. 1st ed. Washington, DC: American Psychiatric Association Publishing; 2022. p.483-518.
6. Pilver CE, Levy BR, Libby DJ, Desai RA. Posttraumatic stress disorder and trauma characteristics are correlates of premenstrual dysphoric disorder. Arch Womens Ment Health. 2011 Oct;14(5):383-93. doi: 10.1007/s00737-011-0232-4.
7. Prasad D, Wollenhaupt-Aguiar B, Kidd KN, et al. Suicidal Risk in Women with Premenstrual Syndrome and Premenstrual Dysphoric Disorder: A Systematic Review and Meta-Analysis. J Womens Health (Larchmt). 2021;30(12):1693-1707. doi: 10.1089/jwh.2021.0185.
8. Chapple A, Ziebland S, Hawton K. Taboo and the different death? Perceptions of those bereaved by suicide or other traumatic death. Sociol Health Illn. 2015;37(4):610-625. doi:10.1111/1467-9566.12224.
9. Merone L, Tsey K, Russell D, Nagle C. Mind the Gap: Reporting and Analysis of Sex and Gender in Health Research in Australia, a Cross-Sectional Study. Womens Health Rep (New Rochelle). 2022 Sep 12;3(1):759-767. doi:10.1089/whr.2022.0033.
10. Vanezis P. Deaths in women of reproductive age and relationship with menstrual cycle phase. An autopsy study of cases reported to the coroner. Forensic Sci Int. 1990 Aug;47(1):39-57. https://doi.org/10.1016/0379-0738(90)90284-6.
11. Warner M, Braun PA, Brown PA. Public Health Impact: How Medicolegal Death Investigation Data Help the Living. Acad Forensic Pathol. 2017 Dec;7(4):xiv–xvi. doi: 10.1177/192536211700700405.

The Pollination of Grasses and Trees in the Auckland Province and the Occurrence of Seasonal Hay-fever.

NZMJ, 1924.

Address given to British Medical Association, Auckland, 5th September, 2023, by T. H. Patterson, H.D.A., *Department of Agriculture, Auckland.*

HAY FEVER—VASOMOTOR RHINITIS.

In a recent work, *Diseases of the Nose and Throat, Dr. Cornelius G. Coakley*, of Columbia University, states that the complaint commonly called hay-fever has in recent years been the subject of widespread investigation both as to its etiology and treatment. "While our knowledge of the subject has been vastly increased, there still remains much work to be done before we can explain all the various manifestations of this condition. We now know that the term 'hay-fever' is a misnomer, as neither hay nor fever are conspicuous factors in the disease." An individual may become sensitised to a given proteid, such as that of the pollen of certain groups of plants, mainly grasses and trees. He further points out that certain individuals, especially those of nervous temperament, become sensitised to pollen proteins or to a proteid substance ingested in certain foods. These persons, on being subjected to test, give a characteristic reaction. An abrasion or scratch is made on the skin, usually on the anterior surface of the forearm, and a solution of the suspected proteid is put on the scratch. If the person is not sensitised to the proteid used in the test no reaction takes place. If, however, he is sensitised, then a positive reaction does take place within a period of from five to fifteen minutes.

In taking a critical view of the available evidence relating to seasonal hay fever some differences of opinion exist on what appears to me to be a matter of fundamental importance. I have never carried out any experiments such as the skin test on the arm referred to above, hence I must quote opinions based on the experimental evidence of medical men.

TWO SCHOOLS OF OPINION. — There are two schools of opinion. In the one we find adherents of what is known as "group reaction." They hold that the pollen of any species of a group, say the graminaceæ or compositæ, will produce a positive reaction if the person tested is sensitive to any one species of the group. This presumes that the pollen of closely-allied families of plants has in common a characteristic structural arrangement of the protein molecule. If a person is sensitive to Timothy grass pollen, for example, he will generally react to the pollen of any true grass.

The opposing school holds that these views are unscientific and unsound. From conclusions drawn from tests made they contend that differential results are obtained from the various species of the plant groups ; yet the general experience is that group reactions hold in most cases. The ordinary pollacine put on the market contains the protein of Timothy grass.

It appears to me from an examination of the evidence available that an individual may be sensitised by the pollen of several members of a group, and the sensitisation depends on the presence of the pollen of the offending plants. The degree of sensitisation to any one species or number of species is probably dependent on the prevalence of one or several of the species causing trouble. Timothy, in the Eastern and Southern United States of America, is very widely distributed, and hence Timothy pollen is the common cause of early hay-fever. A less common species would not cause as much trouble.

To medical men the point at issue is of paramount importance, as attacks of seasonal hay-fever may be warded off by pre seasonal treatment, which consists of a series of subcutaneous injections with solutions of protein prepared from the offending pollen. Some weeks before the expected attack these are administered at regular intervals with gradually increasing concentrations. With grasses, any one pollen extract should cover all cases, if the views of the group-reaction school are correct. A pollen extract of a grass growing in Mexico, for instance, but not in New Zealand, would do quite well for a New Zealand patient. If,

on the other hand, the specific pollen proteid of one species or a group of several, though a limited number of species, is necessary for successful pre-seasonal treatment, then the case is different. For example, the pollen extract of Timothy grass, which is extensively used in America as a sort of blanket prescription for summer hay-fever, may not be effective in New Zealand, where a patient was more exposed to the pollen of, say, ryegrass, cocksfoot, and crested dogstail, and others of our common species of grasses. I am powerless to supplement the views given. The matter, from the medical point of view, must be settled satisfactorily to make progress possible.