NEW ZEALAND
TE ARA TIKA O TE HAUORA HAPORI
MEDICAL JOURNAL

PUBLISHED BY:
PMA PASIFIKA MEDICAL ASSOCIATION Group

Vol. 138 | No. 1614 | 2 May 2025

IN THIS ISSUE:

EDITORIALS

The common good: reviving our social contract to improve healthcare

Health Minister Brown wants to “fix” the system—but risks breaking it further

NEW ZEALAND
TE ARA TIKA O TE HAUORA HAPORI
MEDICAL JOURNAL

PMA PASIFIKA MEDICAL ASSOCIATION Group

Publication information

published by the Pasifika Medical Association Group

The *New Zealand Medical Journal* (*NZMJ*) is the principal scientific journal for the medical profession in New Zealand. The *Journal* has become a fundamental resource for providing research and written pieces from the health and medical industry.

The *NZMJ*'s first edition was published in 1887.
It was a key asset of the New Zealand Medical Association (NZMA) up until July 2022.

It is owned by the Pasifika Medical Association Group (PMAG).

The PMAG was formed in 1996 by a group of Pasifika health professionals who identified a need for an association with the purpose of "providing opportunities to enable Pasifika peoples to reach their aspirations".

ISSN (digital): 1175-8716

Editorial Board

NEW ZEALAND
TE ARA TIKA O TE HAUORA HAPORI
MEDICAL JOURNAL

PMA PASIFIKA MEDICAL ASSOCIATION Group

Publication information

published by the Pasifika Medical Association Group

Further information

ISSN (digital): 1175-8716
Publication frequency: bimonthy
Pubication medium: digital only

To contribute to the *NZMJ*, first read:
nzmj.org.nz/contribute

Other enquiries to

PMA Group
7a Pacific Rise
Auckland 1060
New Zealand

To subscribe to the *NZMJ*, email:

nzmj@pmagroup.co.nz

Full access is available to individual subscribers and does not incur a fee. Institutional subscription is available at the rates below.

All access to the *NZMJ* is by login and password, but IP access is available to institutes.

Further information is available on the *NZMJ* website:
http://www.nzmj.org.nz

If you are a member or a subscriber and have not yet received your login and password, or wish to receive email alerts, please email: nzmj@pmagroup.co.nz

Subscription rates for 2025

Individual		Institute	
New Zealand	Free	New Zealand	$680
International	Free	International	$700

New Zealand rate includes GST. No GST is included in the international rate.

Contents

Editorials

Articles

Viewpoint

Clinical correspondence

100 years ago in the *NZMJ*

Proceedings

Summaries

The common good: reviving our social contract to improve healthcare

Philip Bagshaw, John D Potter, Andrew Hornblow, Susan Bagshaw, Christopher Frampton, Robert Campbell, Ganesh R Ahirao (aka Ganesh Nana), William J Rosenberg, Gilbert O Barbezat, John McCall, Brian Cox, Matthew Roskruge, Frank Kueppers

Successive governments have not fulfilled their responsibility to provide open, adequate healthcare with the idea of achieving good health for all citizens. Their approaches have been to leave more and more responsibility for health in the hands of individual people. Governments need to make a new open contract that accepts their responsibility to provide comprehensive, high standard healthcare for all citizens. After all, that is a major part of their jobs.

Health Minister Brown wants to "fix" the system—but risks breaking it further

Virginia Mills, Lyndon Keene, Harriet Wild

In March, the minister of health announced that long-term, he wants to see as much planned care as possible outsourced to the private sector. This approach risks damaging the public health system further, by shifting much needed resources away from the public system and towards the private system. New Zealand has a limited number of senior doctors. Increasing how much planned care is outsourced to private providers will mean senior doctors leave the public system to work in the private system, which tends to have better pay, easier working conditions and less complex patients. Those who remain working in the public system will face heavier workloads, with fewer colleagues to look after much more high-risk, complex patients. Ultimately, outsourcing planned care will compromise the capacity of the public system to support patients needing acute care, and result in patients needing more complex and costly planned care being left behind.

The effect of pre-operative cardiorespiratory fitness on functional and subjective outcomes following total hip and knee arthroplasty: a single centre, observational study

Brendon H Roxburgh, Holly A Campbell, James D Cotter, Ulla Reymann, Michael JA Williams, David P Gwynne-Jones, Kate N Thomas

Participants with low pre-operative fitness have poorer functional and perceived outcomes following hip or knee arthroplasty. Pre-operative fitness, either measured or estimated, and daily step count are useful clinical tools for predicting functional and subjective recovery following hip or knee arthroplasty. Future work should investigate whether pre-operative optimisation of these predictors translates to improved post-operative functional and subjective recovery, and clinical outcomes.

Delay to diagnosis in childhood bone and joint infection

Sarah Hunter, Elsie Brown, Haemish Crawford, Vanessa Selak, Cameron Grant

Bone and joint infections in children are potentially serious conditions, with the most unwell children requiring intensive care admission and multiple surgeries. Recent review shows that almost half of cases treated in the Auckland region receive an alternative diagnosis when first seeking medical care. Alternatively diagnosed children were more likely to have attended primary or urgent care centres. Getting an alternative diagnosis delayed treatment by 3 days on average. Children experiencing symptoms in the community for longer than a week were more likely to experience spread of infection, requiring more surgery and longer hospitalisations, at higher cost.

Anticoagulation management and poor clinical outcomes in children and young adults following mechanical valve replacement surgery for rheumatic heart disease in Counties Manukau

Prathyusha Tangirala, Bridget Farrant, Rachel Webb

Rheumatic heart disease (RHD) causes significant cardiovascular morbidity and mortality, with persisting inequitably high rates in Māori and Pacific tamariki and rangatahi. Mechanical valve replacement surgery is required for people with severe RHD and requires lifetime anticoagulation. Information regarding anticoagulation and outcomes following mechanical valve replacement surgery for RHD is lacking. We aimed to describe patient characteristics, anticoagulation management and complications in a cohort of tamariki and rangatahi ≤25 in Counties Manukau. Fifty-three patients ≤25 years old were identified. The median age at time of first mechanical valve surgery was 15 years (range 4–23 years). Nineteen percent were Māori, and 81% were Pacific. The most common method of monitoring was via the community laboratory service and general practitioner. Over 70% of persons (38) had ≥1 anticoagulation-related hospitalisation. Most were due to high or low international normalised ratio (INR) levels, but 14% had haemorrhage, 9% stroke, 6% other thromboembolic events and 4% prosthetic valve thrombosis. Five deaths occurred between 2016 and 2021. Urgent efforts are required to improve services for anticoagulation monitoring and management and clinical outcomes in young adults following mechanical valve surgery for RHD.

Electric scooter–related orthopaedic injuries in Wellington

Wing Yung Agnes Chu, Michael T Lee, Ilia Elkinson

Commercially operated e-scooters were introduced to Wellington City in June 2019. In the 2 years prior, there were two e-scooter–related orthopaedic fractures, compared with the 145 fractures sustained over the 2 years since their introduction. Twenty-nine percent of orthopaedic fractures required an operation. Intoxicated drivers were more likely to have open wounds associated with their limb fractures.

Proton pump inhibitors in cirrhosis: a retrospective five-year analysis of increased risks of hepatic decompensation and infections

Abhimati Ravikulan, Natalie Russell, Christin Coomarasamy, Ashok Raj

This study looked at the effects of proton pump inhibitors (PPIs)—commonly used for acid reflux and ulcers—on people with cirrhosis, a serious liver disease. Researchers followed 392 patients for 5 years and found that many were prescribed PPIs without a strong medical reason. Patients taking PPIs had a higher chance of developing infections and complications related to liver failure. However, the study did not find that PPIs significantly increased the risk of death. The results suggest that doctors should be more cautious about prescribing PPIs to cirrhosis patients and should regularly review whether they are necessary.

Applying Indigenous identity definitions in official health statistics: a case study using linked cancer registry data on gastric cancer

Nicole Satherley, Brandon de Graaf, Gabrielle Davie, Sheree Gibb, Andrea Teng, Andrew Sporle

There are two different officially recognised identity definitions for the Māori population (Māori descent and Māori ethnicity). Official health statistics are usually reported by Māori ethnicity but not descent, as health statistics collections such as the New Zealand Cancer Registry (NZCR) do not record Māori descent information. We created new approaches linking different datasets to add Māori descent information to health statistics collections using stomach cancer as an example. These new approaches can be used to produce information on other health and social outcomes for the Māori descent population for the first time.

Cancers potentially attributable to excess body weight in Aotearoa New Zealand from 2019 to 2023

Michael Walsh, Jennifer Brenton-Peters, Olivia Perelini, Karen Bartholomew

Excess body weight (EBW) is associated with a large number of cancers in Aotearoa New Zealand, with 6,962 cases (5.1% of all cancers) between 2019 and 2023, averaging 1,390 per year. The impact is not evenly distributed, with Māori and Pacific peoples, particularly Pacific women, experiencing a higher burden. Women are also more affected than men, largely due to the strong link between EBW and uterine, postmenopausal breast and ovarian cancers. If the prevalence of higher body weight was reduced, hundreds of cancer cases could be prevented each year. Tackling this issue requires a balanced public health approach that strengthens prevention, improves healthcare access and reduces weight stigma, while recognising the wider social and cultural factors that influence body size and health.

Asian health trends in New Zealand from 2002 to 2021, and the case for dedicated research funding

Robert Scragg, Zhenqiang Wu, Sally F Wong

The proportion of the Aotearoa New Zealand population with Asian ancestry is growing, from 17% in the 2023 Census to an expected 26% by 2043. We have recently completed a major report on the health status of Asian people living in New Zealand since 2002, using data from the New Zealand Health Survey. While there have been some improvements, levels of most risk factors—such as fruit and vegetable intake, physical activity, alcohol intake and obesity—have worsened or not improved over the last 20 years, resulting in an elevated risk of diabetes and cardiovascular disease, particularly among South Asians, at levels similar to those for Māori and Pacific. After reviewing the funding of Asian health research by the Health Research Council of New Zealand since 2010, we found a mismatch between the number of funded grants and size of the Asian population in New Zealand (respectively, 2.3% and 17% in 2023). The Health Research Council needs to ring-fence funding for Asian researchers so that Asian researchers have increased resources to research the major health issues that are adversely affecting their communities.

Scurvy in the modern era: a case of vitamin C deficiency with unexplained bruising and anaemia

Akram Shmendi

Vitamin C is an essential nutrient that helps keep our body's cells healthy and protects them from damage. It plays a key role in maintaining strong skin, blood vessels, bones and cartilage, and it also helps wounds heal properly. A lack of vitamin C can lead to a disease called scurvy, which can cause symptoms like easy bruising, bleeding from the gums and other mucous membranes, swollen and inflamed gums and slow healing of cuts and wounds. Getting enough vitamin C through food or supplements is important for overall health and preventing these problems.

The common good: reviving our social contract to improve healthcare

Philip Bagshaw, John D Potter, Andrew Hornblow, Susan Bagshaw, Christopher Frampton, Robert Campbell, Ganesh R Ahirao (*aka Ganesh Nana*), William J Rosenberg, Gilbert O Barbezat, John McCall, Brian Cox, Matthew Roskruge, Frank Kueppers

As a nation, we are becoming increasingly polarised into two philosophical groups, positions that are well described in a recent *New Zealand Herald* op-ed and an ensuing letter to the editor. The first describes how our government is simply allowing, even perhaps encouraging, privatisation to creep forwards through neglect of its philosophical and economic responsibilities for hospital healthcare.[1] A contrary brief response argues that the government is doing its best against an ever-growing demand for an increasingly expensive service.[2] We contend that the resurrection of the social contract between government and governed is a constructive way to bring these two positions together for better national health and wellbeing.

The social contract

The notion of a, sometimes implicit and sometimes explicit, social contract between groups or between sovereign/government and the people, to their mutual advantage, has ancient roots.[3,4] It rose to prominence in the seventeenth century, when it became a pledge from powerful monarchs or governments to protect the security and property of the governed, in return for payment in cash, kind, loyalty and service. It became established by habitual use and in statute, and developed along two separate philosophical lines: i) contractarianism—based on all involved parties being motivated to maximise self-interest,[5] and ii) contractualism—based on morally constrained agreements between those who regard each other as equals, warranting respect.[6] These two social arrangements have been diversely developed to include many types of human interactions and transactions. Across democratic countries, the choice between them is influenced by the prevailing political philosophies and economic policies of the participating parties.

In the commercial world, neoliberal philosophies declare "the market knows best"; thus, the contractarian line is followed with the intention of maximising profits for owners and investors. Here, time frames are largely determined by short-term strategic planning and profit. Conversely, in public healthcare, where altruistic service is central, and some health-education, prevention and early-intervention programmes might take years to complete, a contractualist social contract is justified,[7] and can serve the current conception of "the common good".[8]

Beginning of the welfare state in Aotearoa New Zealand

Until the inception of our universal-access secondary health system in 1938, healthcare was delivered by private providers and charitable institutions. The new system did not cover primary healthcare or adequately address inequitable health and welfare outcomes for Māori, Pacific peoples and all those living in poverty. However, clearly the government of the time was attempting to constructively address their philosophical and economic responsibility for the health and wellbeing of the population. For their part in the partnership with government, the people responded to this responsible contractualist-style leadership by strongly backing the Labour Government involved and by returning it to office at three subsequent elections.[9]

During the second half of the twentieth century, the mix of public and private healthcare here (as in the United Kingdom [UK] and Australia) could be categorised as a liberal health system, where income support and social and health services were only partially funded and were less comprehensive than some other national health systems.[10] Contractarian and contractualist philosophies and economics existed side-by-side in relative harmony, but substantial inequities in wealth, healthcare and welfare outcomes persisted.

Subsequent developments

Since 1990, the social pendulum has swung towards greater contractarian, individualistic immediacy—"it's all about me; it's all about now". Successive governments have followed this social trend and slowly abrogated their responsibility to provide the economic support necessary for the maintenance of the common good and an effectively functioning public healthcare system.[11] Supporters of such contractarian individualism claim that the cost of universal access is prohibitively high.[2] Large international studies have shown, on the contrary, that, in the long-term, universal access to healthcare is the cheapest and most cost-effective system.[12–15] Indeed, investing in healthcare resources across 25 European Union (EU) countries has been shown to yield substantive fiscal multipliers.[12] Unfortunately, it appears some governments either have not believed in such dividends or have erroneously assumed that they are not politically attractive because they would take too long to mature to address short-term political agendas.[12,13] Creeping privatisation in Aotearoa New Zealand is facilitating a decline in government responsibility for comprehensive secondary healthcare.[16]

Constructive policy changes can produce health-system improvement. One study has shown that integrating well accepted and appropriate contractualist philosophy and policy can create a more just and equitable health service.[17] Another has suggested how political, philosophical and economic considerations can be combined to help appropriate healthcare policy making.[18] To the surprise of many, data from both the United States (US) and the EU show that the private healthcare sector is not even more efficient than the public sector; indeed, the reverse is more generally true.[19,20] Nonetheless, it is important to acknowledge that an articulated philosophy to underpin our healthcare system is insufficent without also ensuring that this system is built and maintained at an appropriate level of technical and managerial excellence. The current accelerated trend to greater healthcare privatisation will not work to our long-term advantage and will ultimately benefit only an ever-smaller group of the wealthy and powerful, who simply do not care for the common good.[21,22]

The solution

We suggest that a way forward for us is to again bring into the centre of our public life a positive, widely canvassed, ethically normative social contract—tailored to our local needs and regularly updated—that establishes:

- the philosophical and economic responsibilities of government that have been shown to be efficient and cost-effective elsewhere.[12,13]
- a focus on equity of outcomes rather than equality of access.
- transparency at all clinical, commercial, administrative, managerial and political levels, especially ensuring that contractual details for public works are not kept secret on the grounds of commercial sensitivity.
- short-term political ambitions that are attuned to long-term social needs.
- the reciprocal responsibilities required of our people, particularly a revival of our former egalitarian spirit and, in pursuit of the common good, the necessary industry to increase mutual trust and to make the contract work. These reciprocal responsibilities are reflected in Te Ao Māori values of manaakitanga, kotahitanga, whanaungatanga and kaitiakitanga.

Because twenty-first century governments of all persuasions here have often reneged on their responsibility for leadership, the force for change will necessarily come from our community, our people. They can provide strong, resilient and well-informed advocacy via standing committees of relevant experts and community leaders, including all ethnic, cultural and socio-economically underprivileged groups.

The key challenge involves deciding as a society that we need to re-establish the social contract and to fund the duties and responsibilities that flow from that. The population being older, the non-communicable disease burden being greater and inequity being much more marked are reasons to respond with a system that aims at universal access, not a system that rations on the basis of the ability to pay. Just because tax revenue is constrained under the current income-tax–based system does not mean that there is insufficient wealth in the population to support the needs of an ageing population. A more progressive income tax, a wealth tax, a capital-gains tax or some judicious combination of these can ensure that the healthcare system remains viable into the foreseeable future. Like the social contract, a more equitable taxation system that provides sufficient funding for that social contract is a choice.

Conclusions

Our initial steps in 1938 towards a universal open-access health system were world-leading but imperfect. Subsequent reform should have been focussed on widening the contractualist vision and providing the political and economic support to achieve universal equitable health outcomes. Nearly a century later, we are regressing to an increasingly contractarian philosophy. This will lead us progressively to a US-style health system that is prohibitively expensive, highly inefficient and unacceptably inequitable, and from which we will be unable to extricate ourselves. Right now, we have the opportunity and the capacity to revive the social contract. Do we have the courage and persistence necessary to do so?

COMPETING INTERESTS
PB: Youth Hub Christchurch Trust Board Member.
RC: Chancellor, Auckland University of Technology, and Former Chair, Te Whatu Ora – Health New Zealand.
GA: Chair; Finance, Assurance, and Risk Committee; New Zealand Drug Foundation Te Puna Whakaiti Pāmamae Kai Whakapiri.
WR: Tax Justice Aotearoa Committee member; Chair of Policy Advisory Committee.
SB: Trustee of Canterbury Charity Hospital Trust; Chair of Youth Hub Christchurch Trust.

AUTHOR INFORMATION
Philip Bagshaw: Chair, Canterbury Charity Hospital Trust.
John D Potter: Professor, Centre for Public Health Research, Massey University, Wellington.
Andrew Hornblow: Emeritus Professor, University of Otago Christchurch.
Susan Bagshaw: Senior Lecturer, University of Otago Christchurch; Educator, Collaborative Trust.
Christopher Frampton: Professor, Department of Medicine, University of Otago Christchurch.
Robert Campbell: Former Chair, Te Whatu Ora – Health New Zealand.
Ganesh R Ahirao (aka Ganesh Nana): Former Chair, Productivity Commission Te Kōmihana Whai Hua o Aotearoa; Former Chief Economist, Business and Economic Research Limited.
William J Rosenberg: Visiting Scholar at Victoria University of Wellington Te Herenga Waka; Economist.
Gilbert O Barbezat: Emeritus Professor, University of Otago Dunedin.
John McCall: Professor, Department of Surgery, Faculty of Medical and Health Sciences, The University of Auckland.
Brian Cox: Specialist in Public Health.
Matthew Roskruge: Professor, Associate Dean Māori, Massey Business School, Te Ātiawa and Ngāti Tama.
Frank Kueppers: Department of Urology, Christchurch Hospital, Christchurch.

CORRESPONDING AUTHOR
Philip Bagshaw: Chair, Canterbury Charity Hospital Trust; PO Box 20409, Christchurch 8543, Aotearoa New Zealand. Ph: 03-360-2266. E: philipfbagshaw@gmail.com

URL
https://nzmj.org.nz/journal/vol-138-no-1614/the-common-good-reviving-our-social-contract-to-improve-healthcare

REFERENCES
1. Nahill A. Private healthcare in NZ: We need a transparent, comprehensive and independent review. The New Zealand Herald [Internet]. 2025 14 Jan [cited 2025 Apr 8]. Available from: https://www.nzherald.co.nz/nz/private-healthcare-in-nz-we-need-a-transparent-comprehensive-and-independent-review-art-nahill/ELH3I5KVRJCU5IN2BZCIV67CEE
2. Le Grice H. Letter to the Editor: Private Healthcare. The New Zealand Herald. 2025 Jan 17 [available on request].
3. Wikipedia. Social contract [Internet]. Wikipedia; 2025 Mar 8 [cited 2025 Apr 8]. Available from: https://en.wikipedia.org/wiki/Social_contract
4. Russell B. History of Western Philosophy. 2nd ed. London: Unwin Paperbacks; 1979.
5. Cudd A, Eftekhari S. Contractarianism. In: Edward N Zalta, editor. The Stanford Encyclopedia of Philosophy (Winter 2021 Edition). California (US): Department of Philosophy, Stanford University; 2000 Jun 18; substantive revision 2021 Sep 30 [cited 2025 Apr 8]. Available from: https://plato.stanford.edu/archives/win2021/entries/contractarianism/
6. Ashford E, Mulgan T. Contractualism. In: Edward N Zalta, editor. The Stanford Encyclopedia of Philosophy (Summer 2018 Edition). California (US): Department of Philosophy, Stanford University; 2007 Aug 30; substantive revision 2018 Apr 20 [cited 2025 Apr 8]. Available from: https://plato.stanford.edu/entries/contractualism
7. D'Agostino F, Gaus G, Thrasher J. Contemporary Approaches to the Social Contract. In: Edward N Zalta, Uri Nodelman, editors. The Stanford Encyclopedia of Philosophy (Spring 2024 Edition). California (US): Department of Philosophy, Stanford University; 1996 Mar 3; substantive revision 2021 Sep 27 [cited 2025 Apr 8]. Available from: https://plato.stanford.edu/archives/spr2024/entries/contractarianism-contemporary/
8. Sandel MJ. The Tyranny of Merit; What's Become of the Common Good? London (UK): Penguin Books; 2021.
9. New Zealand Parliament – Pāremata Aotearoa. Governments in New Zealand since 1856 [Internet]. Wellington (NZ): New Zealand Parliament; 2023 Nov 29 [cited 2025 Apr 8]. Available from: https://www.parliament.nz/en/visit-and-learn/mps-and-parliaments-1854-onwards/governments-in-new-zealand-since-1856/
10. Barnett P, Bagshaw P. Neoliberalism: what it is, how it affects health and what to do about it. N Z Med J. 2020;133(1512):76-84.
11. Mills V, Keene L, Roberts J, Wild H. The cost

of everything and the value of nothing: New Zealand's underinvestment in health. N Z Med J. 2024;137(1601):9-13. doi: 10.26635/6965.e1601.
12. Reeves A, Basu S, McKee M, Meissner C, Stuckler D. Does investment in the health sector promote or inhibit economic growth? Global Health. 2013;9(1):43. doi: 10.1186/1744-8603-9-43.
13. Stuckler D, Basu S. The Body Economic: Eight experiments in economic recovery, from Iceland to Greece. London (UK): Penguin Books; 2014.
14. Stuckler D, Reeves A, Loopstra R, et al. Austerity and health: the impact in the UK and Europe. Eur J Public Health. 2017;27(suppl_4):18-21. doi: 10.1093/eurpub/ckx167.
15. Reeves A, McKee M, Stuckler D. The attack on universal health coverage in Europe: recession, austerity and unmet needs. Eur J Public Health. 2015;25(3):364-5. doi: 10.1093/eurpub/ckv040.
16. Bagshaw P, Potter JD, Bagshaw S. The looming spectres of public-private partnerships for hospitals and the resulting decline of government responsibility for comprehensive secondary healthcare in Aotearoa New Zealand. N Z Med J. 2024;137(1590):9-13. doi: 10.26635/6965.e1590.
17. Fritz Z, Cox CL. Integrating philosophy, policy and practice to create a just and fair health service. J Med Ethics. 2020;46(12):797-802. doi: 10.1136/medethics-2020-106853.
18. Oswald M. In a democracy, what should a healthcare system do? A dilemma for public policymakers. Polit Philos Econ. 2013;14(1):23-52.
19. Kruse FM, Stadhouders NW, Adang EM, et al. Do private hospitals outperform public hospitals regarding efficiency, accessibility, and quality of care in the European Union? A literature review. Int J Health Plann Manage. 2018;33(2):e434-e53. doi: 10.1002/hpm.2502.
20. Schlesinger M, Gray BH. How nonprofits matter in American medicine, and what to do about it. Health Aff (Millwood). 2006;25(4):W287-303. doi: 10.1377/hlthaff.25.w287.
21. Piketty T. A Brief History of Equality. Cambridge MA: Harvard University Press; 2022.
22. Riddell R, Ahmed N, Maitland A, et al. Inequality Inc. How corporate power divides our world and the need for a new era of public action [Internet]. UK: Oxfam; 2024 [cited 2025 Apr 8]. Available from: https://policy-practice.oxfam.org/resources/inequality-inc-how-corporate-power-divides-our-world-and-the-need-for-a-new-era-621583/

Health Minister Brown wants to "fix" the system—but risks breaking it further

Virginia Mills, Lyndon Keene, Harriet Wild

In March, the new Health Minister, Simeon Brown, declared in a speech to the BusinessNZ health forum that it is "*time to fix this system*," and announced a raft of measures.[1] For secondary care this included a continued focus on health targets; Te Whatu Ora – Health New Zealand allocating NZ$50 million to carry out 10,579 elective procedures by June 2025 (in-house and outsourced); and an expectation that as much planned care as possible should be outsourced to private hospitals—not as a stop gap, but for the long term.

The health minister's plan to outsource as many elective procedures as possible to the private sector in the long term is unlikely to "fix" the public system. Rather, it is at serious risk of breaking it further, increasing health costs, decreasing productivity and further constraining the ability to provide patients with the care they need.

The challenges facing the public health system have become increasingly clear to the public, with frequent media coverage highlighting workforce shortages, infrastructure deficits, unmet need, overwhelmed emergency departments, ballooning waiting lists, major restructures and high turnover of leadership. Media reported that former Health Minister Dr Shane Reti received a briefing from Te Whatu Ora – Health New Zealand that warned of the rising number and complexity of acute public hospital cases "*in a highly resource-constrained environment*" and explained "*as demand grows for acute and urgent care, available capacity for planned* [elective] *care diminishes*."[2] Public hospital capacity for the latter cannot easily be increased because of workforce shortages and hospital wards frequently operating at 100% occupation rates.[3]

The health system requires more than a superficial "fill it and fix it" approach. In the short term, the minister's goal of 10,579 additional elective procedures by June 2025 is bold, but significant constraints stand in the way of delivery. This includes severe workforce shortages (that impact both public and private sectors),[4] increasing demand for acute care[5] and limited funding (with the NZ$50 million for electives being reallocated from another unknown part of the health system). It is also unclear what marginal capacity is available in the private system to pick up extra elective work, with documented difficulties establishing reliable information on private sector marginal capacity, despite attempts to do so.[6]

If the minister planned to "fix" the public system, such long-term plans involving private providers would not be necessary. The minister will be aware that the private sector is also going through hard times, with recent balance sheet losses. It is not that the private sector is losing customers. Southern Cross, Aotearoa New Zealand's largest health insurer, for example, is inching towards the 1-million-member mark. It is more that the increasing costs and volumes of insurance claims are rising above revenue from premiums.[7–9] The minister's announcement will no doubt be a relief to the private sector.

It is problematic when the private system becomes essential for filling the growing gaps in the public health system. Decades of underinvestment and inadequate planning have brought the public system to its knees, with reduced capacity to tackle elective waiting lists. Given this tragic reality and the urgent need to ensure patients receive treatment with as little delay as possible, it is necessary to involve the private sector in the short term. Taking that beyond a stop-gap measure, however, will contribute to the continuing demise of the public system.

Here's why:

One: Outsourcing public services to private providers can lead to "cream-skimming", where private providers choose patients with less severe conditions and who are less financially risky, leaving the complex and more expensive cases with the public system.[10] This can result in patients

needing more complex and costly treatment getting left behind, exacerbating inequitable health outcomes.

Indeed, the NZ$50 million recently reallocated by the minister to fund an extra 10,579 procedures amounts to less than NZ$5,000 per procedure on average, indicating they will be mostly minor procedures.

"Cream-skimming" is already happening in our system, with evidence private providers are able to choose which patients they will do procedures on, with little regard to patient need or the length of time patients have been waiting.[11] As the balance tips further towards the public system only providing care for the most complex cases (which take more time and money), it will also appear as though so-called "productivity" in the public sector is falling.

Two: We have a finite pool of doctors. Many medical specialists who staff public hospitals also work in the private system. Given the entrenched workforce shortages both here and internationally, outsourcing to the private sector will almost certainly mean pulling more specialists away from the public system. Association of Salaried Medical Specialists (ASMS) analysis shows this is already happening, and the key reasons for specialists leaving the public system to work in private are remuneration, the ability to manage one's own time and workload, and clinical satisfaction.[12] Losing staff from the public sector places additional burden on those left behind, as fewer specialists are available to be on-call and public capacity to cover acute presentations reduces, with negative impacts on patient care. Due to "cream-skimming", the work in public is also increasingly complex, and the pull towards private where specialists can earn more money, take more straightforward cases and not have to participate in call rosters will become more and more enticing.

Three: Outsourcing to the private sector limits medical trainees' exposure to a range of procedures, since they are mostly trained in public hospitals. The more elective procedures go to the private sector, the more difficult it becomes for resident doctors to successfully meet training requirements and the more difficult it becomes to develop skilled and experienced medical specialists. Loss of senior medical officers to the private system will also mean that those who remain in public are stretched even thinner, impacting their ability to train and mentor junior colleagues.

Four: The private system is not subject to the same level of scrutiny and accountability as the public system.[13] Anyone who has attempted to obtain basic data from private providers will know this. International evidence suggests that higher rates of privatisation and outsourcing correspond with poorer health outcomes for patients.[14] In Aotearoa New Zealand, it is an unknown.

Five: Commercial barriers also mean there is scant information about the cost effectiveness of the private system compared with public provision. This raises a fifth issue: that of cost to the taxpayer. One rare study, by the Health Funds Association, compared the average cost of five elective surgical procedures in the two sectors during 2004/2005. It did not account for the likelihood of public hospitals undertaking more complex (and therefore more costly) procedures. Despite this, of the five procedures, all except one was more costly in the private sector than in the public sector.[15]

Today, media reports suggest the costs of outsourcing elective operations are growing,[16] and the Planned Care Taskforce found private providers were seeking "*significant uplifts to historic prices – in some cases … in excess of 20%.*"[6]

Private hospitals also do not carry the same overheads as public hospitals. Public hospitals maintain essential services such as intensive care units and blood banks. When patients treated in private facilities need such services, the public sector picks up the bill.[17]

Six: Private hospitals are not evenly distributed,[18] raising issues of unequal access, especially for regional communities and those in poorer areas. Private hospitals have little incentive to service rural communities where smaller populations mean it is difficult to generate profits or break even.

Seven: As more elective treatment is provided in the private sector, drawing staff away from the public sector, the public capacity to cope with the growing and more complex acute hospital admissions will deteriorate further. The minister appears to have not understood this when he says he wants to see more outsourcing of electives, thereby "*freeing public hospitals for acute needs*."

Genuinely fixing the public health system is imperative if we are to ensure Aotearoa New Zealand is able to deliver on the universal health coverage that was intended in the 1938 *Social Security Act*. This will require real, substantial investment well beyond the funding needed to simply tread water.

We need:

- a comprehensive health workforce plan, including increasing our domestic supply of medical practitioners, and implementing extensive recruitment and retention strategies across the board.
- strong clinical leadership to turn Te Whatu Ora – Health New Zealand into an organisation that prioritises patient care and excellent clinical outcomes, and in which staff are thriving rather than surviving.
- significant investment in hospital infrastructure and equipment, to recover from decades of deferred maintenance and ensure we have facilities fit for future generations and the tools for health practitioners to provide excellent patient care.
- adequate funding to increase access to primary care, so people can receive care early in the community and stay well for longer.

Making such investments will require a shift in mindset. Rather than seeing health spending as a short-term cost, it needs to be understood as a long-term investment to support a healthy society. How we value health is fundamental to the economic success and resilience of Aotearoa New Zealand.[19]

COMPETING INTERESTS
The authors are employed by Toi Mata Hauora, the Association of Salaried Medical Specialists.

AUTHOR INFORMATION
Virginia Mills: Interim Director of Policy and Research, Association of Salaried Medical Specialists, Wellington, New Zealand.
Lyndon Keene: Senior Policy and Research Advisor, Association of Salaried Medical Specialists, Sydney, Australia.
Harriet Wild: Director of Policy and Research, Association of Salaried Medical Specialists, Wellington, New Zealand.

CORRESPONDING AUTHOR
Virginia Mills: Interim Director of Policy and Research, Association of Salaried Medical Specialists, Wellington, New Zealand. E: virginia.mills@asms.org.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/health-minister-brown-wants-to-fix-the-system-but-risks-breaking-it-further

REFERENCES
1. Brown S. Speech to the BusinessNZ Health Forum [Internet]. Wellington (NZ): New Zealand Government; 2025 Mar [cited 2025 Apr 10]. Available from: https://www.beehive.govt.nz/speech/speech-businessnz-health-forum
2. Jones N. Revealed: Why elective surgeries are being cancelled amid record wait list delays. The New Zealand Herald [Internet]. 2024 Sep 2 [cited 2025 Apr 9]. Available from: https://www.nzherald.co.nz/nz/revealed-why-elective-surgeries-are-being-cancelled-amid-record-wait-list-delays/LHT7XNZRINHIRLFHHOI5PAN54Q/
3. Roden J. Hospitals hit 100% occupancy more than 600 times last year. 1News [Internet]. 2023 Feb 9 [cited 2025 Apr 10]. Available from: https://www.1news.co.nz/2023/02/09/hospitals-hit-100-occupancy-more-than-600-times-last-year/
4. Te Whatu Ora – Health New Zealand. Health Workforce Plan 2024 [Internet]. Wellington (NZ): Te Whatu Ora – Health New Zealand; 2024 [cited 2025 Apr 9]. Available from: https://www.tewhatuora.govt.nz/publications/health-workforce-plan-2024
5. Association of Salaried Medical Specialists. Anatomy of a health crisis - by the numbers [Internet]. Wellington (NZ): Association of Salaried Medical Specialists; 2024 [cited 2025 Apr 9]. Available from: https://asms.org.nz/wp-content/uploads/2024/04/ASMS-Anatomy-of-a-Health-Crisis-210416.pdf
6. Planned Care Taskforce. Reset and Restore Plan [Internet]. Wellington (NZ): Te Whatu Ora – Health New Zealand; 2022 Sep [cited 2025 Apr 9]. Available from: https://www.tewhatuora.govt.nz/publications/planned-care-taskforce-reset-and-restore-plan
7. Somers E. Southern Cross records annual deficit with claims up 15% and premiums up 9% [Internet]. Auckland (NZ): JDJL Limited; 2024 Sep 30 [cited 2025 Apr 9]. Available from: https://www.interest.co.nz/insurance/129987/southern-cross-records-annual-deficit-claims-15-and-premiums-9
8. Arneil S. Southern Cross Health Society Group annual results reflect steep increase in demand for private healthcare. Southern Cross [Internet]. 2024 Sep 30 [cited 2025 Apr 9]. Available from: https://www.southerncross.co.nz/news/2024/southern-cross-health-society-group-annual-results-reflect-steep-increase-in-demand
9. Smith M. Health insurer NIB warns profit to fall up to 10pc as NZ claims soar. Financial Review [Internet]. 2024 Nov 12 [cited 2025 Apr 10]. Available from: https://www.afr.com/companies/healthcare-and-fitness/health-insurer-nib-warns-profit-to-fall-up-to-10pc-as-nz-claims-soar-20241112-p5kpvc
10. Cheng TC, Haisken-DeNew JP, Yong J. Cream skimming and hospital transfers in a mixed public-private system. Soc Sci Med. 2015;132:156-64. doi: 10.1016/j.socscimed.2015.03.035.
11. Hill R. Outsourcing being used to pretend hospital wait times are being fixed - doctor. Radio New Zealand [Internet]. 2025 Mar 25 [cited 2025 Apr 9]. Available from: https://www.rnz.co.nz/news/political/545877/outsourcing-being-used-to-pretend-hospital-wait-times-are-being-fixed-doctor
12. Association of Salaried Medical Specialists. A less public place [Internet]. Wellington (NZ): Association of Salaried Medical Specialists; 2023 Aug [cited 2025 Apr 9]. Available from: https://asms.org.nz/wp-content/uploads/2023/08/A-Less-Public-Place-FINAL-1.0.pdf
13. Torpy O. Privatised Hospitals: An Accountability Black Hole [Internet]. NSW (AU): The McKell Institute; 2017 Jun [cited 2025 Apr 9]. Available from: https://mckellinstitute.org.au/research/articles/privatised-hospitals-an-accountability-black-hole/
14. Goodair B, Reeves A. Outsourcing health-care services to the private sector and treatable mortality rates in England 2013-20: an observational study of NHS privatisation. Lancet Public Health. 2022;7:E638-46.
15. Ashton T. The benefits and risks of DHBs contracting

out elective procedures to private providers. N Z Med J. 2010;123(1314):84-91.
16. Mathias S, Vitz E. The rising costs of outsourced healthcare. The Spinoff [Internet]. 2022 Nov 22 [cited 2025 Apr 9]. Available from: https://thespinoff.co.nz/society/22-11-2022/the-rising-costs-of-outsourced-healthcare
17. Penno E, Sullivan T, Barson D, Gauld R. Private choices, public costs: Evaluating cost-shifting between private and public health sectors in New Zealand. Health Policy. 2021;125(3):406-14. doi: 10.1016/j.healthpol.2020.12.008.
18. Ministry of Health – Manatū Hauora. Private Hospitals [Internet]. Wellington (NZ): Ministry of Health – Manatū Hauora; 2024 [cited 2024 Apr 10]. Available from: https://www.health.govt.nz/regulation-legislation/certification-of-health-care-services/certified-providers/private-hospitals
19. World Health Organization. Health for All: transforming economies to deliver what matters [Internet]. Geneva (CH): World Health Organization; 2023 [cited 2025 Apr 10]. Available from: https://www.who.int/publications/m/item/health-for-all--transforming-economies-to-deliver-what-matters

The effect of pre-operative cardiorespiratory fitness on functional and subjective outcomes following total hip and knee arthroplasty: a single centre, observational study

Brendon H Roxburgh, Holly A Campbell, James D Cotter, Ulla Reymann, Michael JA Williams, David P Gwynne-Jones, Kate N Thomas

ABSTRACT

AIM: The aim of this study was to assess the effect of pre-operative cardiorespiratory fitness (peak $\dot{V}O_2$) on physical and subjective recovery from total hip or knee arthroplasty. A secondary aim was to assess the relationship between daily step count or estimated peak $\dot{V}O_2$ via the Duke Activity Status Index (DASI) questionnaire, on post-operative recovery.

METHODS: In this secondary analysis of a prior randomised controlled trial, 51 patients (69 [8] y; 25 female; peak $\dot{V}O_2$: 20.1 [7.8] mL/min/kg) scheduled for total hip (n=23) or knee (n=28) arthroplasty underwent pre-operative assessment (cardiopulmonary exercise testing, physical function tests [30-second sit to stand, timed up and go, knee range of motion]), accelerometry and subjective questionnaire (DASI). Post-operative assessments included length of hospital stay, the Surgical Recovery Scale (SRS) and repeated functional assessments.

RESULTS: A low pre-operative peak $\dot{V}O_2$ (i.e., <15mL/min/kg) was associated with five fewer sit-to-stand reps (95% CI [confidence interval]: 3 to 7; p=0.002), 3,500 fewer daily steps (95% CI: 1,053 to 5,867; p=0.006) and poorer subjective surgical recovery at 7-days (-12 arbitrary units [AU], 95% CI: -3 to -22, p=0.014) and 6-weeks post-operative (-13 AU, 95% CI: -5 to -21; p=0.003). Estimated pre-operative peak $\dot{V}O_2$ using the DASI questionnaire was moderately correlated with post-operative daily step count (r=0.51, p <0.001); post-operative daily step count increased by 500 steps for every 1mL/min/kg increase in estimated peak $\dot{V}O_2$.

CONCLUSION: Pre-operative peak $\dot{V}O_2$ was associated with physical and subjective recovery following total hip or knee arthroplasty. Daily step count and estimated peak $\dot{V}O_2$ via the DASI questionnaire had similar moderate associations with post-operative functional outcomes as directly measured pre-operative peak $\dot{V}O_2$ and may be acceptable alternatives to predict recovery following hip or knee arthroplasty.

Arthroplasty is an effective intervention for treating end-stage osteoarthritis. Despite being a common and relatively safe procedure, there is a wide range of physical function and patient-reported outcomes following arthroplasty.[1,2] Low cardiorespiratory fitness, specifically a low peak oxygen consumption ($\dot{V}O_2$; i.e., <15mL/min/kg), and/or low anaerobic threshold (<11mL/min/kg) are associated with poorer outcomes and recovery following non-cardiac surgery;[3] however, no data are available on the clinical significance of these metrics in patients scheduled for total joint arthroplasty. As cardiorespiratory fitness provides a systemic and highly integrative assessment of physical function and capacity,[4] these pre-operative variables are likely associated with a patient's recovery following total joint arthroplasty (e.g., ability to tolerate surgical stress, early ambulation), as for other surgeries.[5]

Cardiopulmonary exercise testing (CPET) is the gold standard for assessing pre-operative cardiorespiratory fitness.[5] Despite its utility as an objective pre-operative risk stratification tool, CPET is not universally available, as it is resource intensive (e.g., time, expensive equipment, specialised staff, etc.). Furthermore, clinical contraindications may preclude some patients performing the test.[6] While assessing the utility of CPET in patients scheduled for arthroplasty, it would, therefore, also be pertinent to know how other forms of assessment compare as a feasible alternative when CPET is not available or appropriate. For example, objectively measured pre-operative physical activity (e.g., accelerometer or pedometer) is an easily implemented tool that is associated with post-operative complications

in gastrointestinal surgery patients.[7–9] Although physical activity is correlated with peak VO_2, it may represent an index of fitness independent of peak VO_2; that is, daily physical activity has minimal effect on increasing peak VO_2, but has numerous physiological (e.g., breaking sedentary behaviour, maintaining glucoregulation, neuromuscular function) and psychological benefits for the surgical patient.[4] Even simpler to use, the Duke Activity Status Index (DASI) questionnaire provides an estimated peak VO_2 using a person's perceived ability to perform activities of daily living and has been shown to correlate with directly measured peak VO_2 (via CPET) in older adults presenting for major non-cardiac surgery.[10] While these associations show promise in older adults and some peri-operative settings, it remains unclear how well these measures predict recovery from total joint arthroplasty.

Therefore, the aims of this study were to assess: 1) the effect of pre-operative peak VO_2 on functional and subjective recovery at 6-weeks post-operative, and 2) the relationship between other pre-operative assessments, and functional and subjective recovery following total hip or knee arthroplasty.

Materials and methods

Ethics

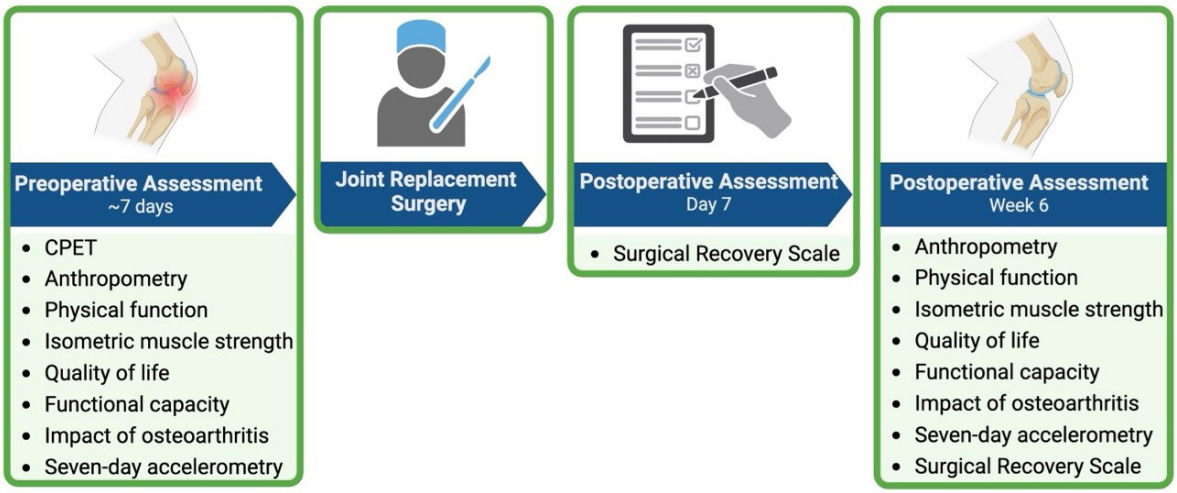
Ethical approval for this study was obtained from the Health and Disability Ethics Committee (18/NTA/148), and the study conformed to the standards set by the Declaration of Helsinki. The trial was registered with the Australian New Zealand Clinical Trials Registry (ACTRN 12618001358235). Written, informed consent was obtained for all participants.

Study design

Participants who completed a prior randomised control trial[11] were invited to participate in this study comparing pre-operative measures of cardiorespiratory fitness and physical and subjective health on functional and subjective recovery from total hip or knee arthroplasty (referred to as hip arthroplasty or knee arthroplasty hereafter). This study uses a subset of data from the previous trial, as the COVID-19 pandemic altered the original intervention timeline. Specifically, participants who completed the 12-week prehabilitation intervention but had not yet scheduled surgery were invited for a pre-operative assessment within a week of their surgical procedure, with post-operative follow-up. Data collection took place from September 2019 to July 2021.

Participants completed a pre-operative assessment (see below for details; Figure 1) within 1 week before undergoing hip or knee arthroplasty. Post-operatively, each participant completed the Surgical Recovery Scale (SRS) on day 7.[12] At 6-weeks post-operative, participants repeated the assessment session.

Figure 1: Schematic representation of assessment schedule.



CPET = cardiopulmonary exercise test.

Study participants

Patients from Dunedin Public Hospital's Orthopaedic department waiting list for hip or knee arthroplasty, who were able to travel to the testing location within 1 week of surgery, were invited to participate in this study.

Participants were excluded if they met any of the following criteria: surgery scheduled and insufficient time to perform assessment; a contraindication to non-physician supervised maximal exercise testing;[13] stable or unstable angina; myocardial infarction within the last 3 months; an implantable cardioverter defibrillator and/or pacemaker; revision arthroplasty; staged bilateral total joint replacement; pathology limiting upper-limb exercise (i.e., shoulder-joint osteoarthritis); and any other medical condition deemed by the study anaesthetist or cardiologist to be a significant risk to study participation.

Experimental procedures

Pre-operative assessment

CPET was performed to measure cardiorespiratory fitness (i.e., peak VO_2, anaerobic threshold) using a previously reported protocol;[14] in brief, the cross trainer was the preferred CPET modality, but arm ergometry was used when this was not feasible (e.g., severe joint pain, joint motion limitations, etc.). A 3–5-minute warm-up was performed, then intensity was increased by 10–20 watts per minute (dependent on participant tolerability and ergometer type) until volitional fatigue. Calibration of gas and flow (Quark CPET; COSMED, Rome, Italy) was conducted per manufacturer instructions before each test. All CPETs were supervised by a registered clinical exercise physiologist (BR). Anthropometric measures of height (stadiometer, Wedderburn WS-HRP, Auckland, New Zealand), body mass (scales, Seca, Hamburg, Germany) and body mass index (BMI; mass [kg]/height^2 [m]) were collected. Physical function was assessed via the 30-second sit to stand test, timed up and go test and knee joint range of motion, following established procedures.[15,16] Subjective measures were assessed using validated questionnaires, including perceived quality of life (12-Item Short Form Survey[17] [SF-12] and EuroQual-5D[18] [EQ-5D]), functional capacity (DASI[19]) and impact of osteoarthritis (Western Ontario and McMaster Universities Osteoarthritis Index[20] [WOMAC]). Physical activity was assessed using accelerometry (activPAL3c, Glasgow, Scotland) in the 7 days preceding surgery.

Post-operative measurements

Seven-days post-operative

Length of stay data were acquired from hospital notes and discharge letters. Participants completed the SRS questionnaire at 7-days post-operative.[12] Participants were asked to complete the questionnaire alone and in a quiet area with no distractions.

Six-weeks post-operative

Participants repeated the pre-operative assessments (except CPET) and the SRS. All assessments were conducted by the same researcher (BR) at Dunedin Hospital and the School of Physical Education, Sport and Exercise Sciences, University of Otago, Dunedin, New Zealand.

Standardisation

To ensure test repeatability, participants were asked to comply with recommendations outlined previously;[11,14] in brief, this included abstaining from cigarette smoking 4 hours prior, caffeinated and alcoholic beverages 12 hours prior and moderate- or high-intensity physical activity 24 hours prior to each CPET. The peri-operative team (surgeon, anaesthetist, surgical nurse and administrative team) were not aware participants were being followed up post-operatively, and study participation did not impact inpatient or outpatient care. Following arthroplasty, all participants were offered standard care rehabilitation as typically provided by the hospital. Initially, rehabilitation entailed 2–5 days of inpatient physiotherapy consisting of weight bearing, joint mobilisation, lower-limb flexibility exercises and stair climbing/descending exercise. Upon discharge from hospital, participants were provided (by the hospital) a generic home exercise programme. The exercise programme primarily consisted of joint range of motion and strengthening exercises. Participants were advised by hospital staff to perform the exercises daily.

Data analysis

CPET data were analysed independently by two researchers (BR & HC), and peak VO_2, anaerobic threshold and oxygen uptake efficiency slope were determined using previously reported methods.[14] Accelerometry data were analysed using manufacturer software (PALbatch v8.10, PAL Technologies, Glasgow, Scotland) and data from only "valid wear days" were included in analysis—an entire 24-hour period with 0 minutes of "non-wear time" was considered valid.

Statistical analysis

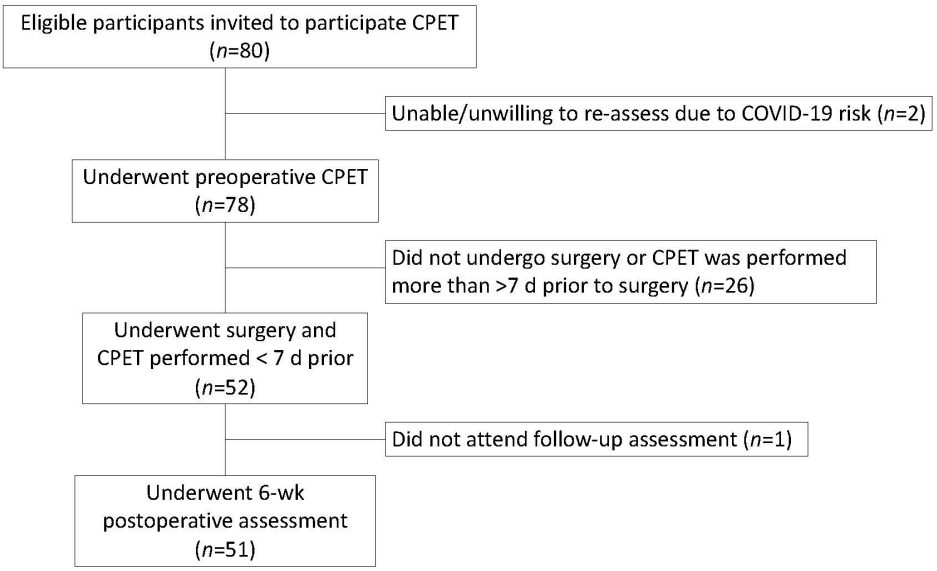
Given the relatively small sample, observational nature, absence of prospective power and large number of simultaneous statistical comparisons, all statistical analyses were exploratory and should be interpreted with caution. Descriptive data are expressed as mean (standard deviation) or number (proportion). To assess the effect of peak VO_2 and the anaerobic threshold on post-operative outcomes, participants were stratified based on previously defined thresholds in non-cardiac surgery (i.e., peak VO_2: 15mL/min/kg; anaerobic threshold: 11mL/min/kg;[3] no previously defined thresholds available for total joint arthroplasty patients). Grouped means were then compared via a repeated-measures Analysis of Variance (ANOVA), with pre-operative osteoarthritis impact as a covariate, or an Independent Samples *t*-test, as appropriate. For the ANOVA, the main effects of Group (low vs high fitness), Time (pre-operative vs post-operative) and the Interaction (Group×Time) were assessed to determine whether the change from pre-operative to post-operative differed by fitness group. Post-hoc testing using Tukey's test was performed if statistical significance was observed (i.e., $p < 0.05$). Categorical data were analysed using the Chi-squared test. Simple linear regression and Pearson's correlation coefficients were used to analyse the relationship between pre-operative variables and post-operative functional and subjective measures. To aid in the interpretation of the correlation coefficients, 0.9–1.0 was considered a very high correlation, 0.7–0.9 high correlation, 0.5–0.7 moderate correlation, 0.3–0.5 low correlation and 0.0–0.3 negligible correlation.[21] Multiple linear regression was performed to assess pre-operative peak VO_2 for predicting functional (daily step count, timed up and go test) and subjective (SRS and WOMAC score) recovery at 6-weeks post-operative. Pre-operative peak VO_2 was modelled with sex, BMI and age; these were selected due to their potential relationship with post-operative recovery while attempting to avoid multicollinearity with other measures. Complete-case analysis was used, excluding participants with missing data on any variable required for a specific analysis. Statistical analysis was performed using R (version 4.1.1, R Development Core Team) and graphed using Prism (v8.0, GraphPad, San Diego, USA).

Results

Participant characteristics

Fifty-one participants completed pre-operative and post-operative assessments and were included in this analysis (Figure 2). All participants survived the surgery, and 6-week recovery period and descriptive characteristics are listed in Table 1. Participants scheduled for knee arthroplasty

Figure 2: Study flow diagram.



CPET = cardiopulmonary exercise test.

Table 1: Descriptive statistics and length of hospital stay for all participants, low fit participants and high fit participants.

Variable—Mean (SD)	Overall (*n*=51)	Low fit (*n*=14)	High fit (*n*=37)
Age (y)	69 (8)	73 (7)	67 (8)
Height (cm)	168 (8)	164 (6)	169 (8)
BMI ($kg.m^{-2}$)	30.9 (5.7)	33.3 (4.9)	30.0 (5.8)
Cardiopulmonary exercise test			
Relative peak VO_2 (mL/min/kg)	20.1 (7.8)	12.4 (1.8)	23.0 (7.2)
Absolute peak VO_2 ($mL{\cdot}min^{-1}$)	1,713 (684)	1,100 (225)	1,944 (656)
Anaerobic threshold (mL/min/kg)	13.0 (5.3)	7.6 (1.3)	15.0 (4.8)
Oxygen uptake efficiency slope	1,892 (740)	1,339 (355)	2,101 (743)
Variable—N (%)			
Male/female	26/25 (51/49)	4/10 (29/71)	22/15 (54/46)
Hip/knee arthroplasty	23/28 (43/55)	8/6 (57/43)	15/22 (41/59)
Ethnicity			
NZ European/European	50 (98)	14 (100)	36 (96)
Cook Islands Māori	1 (1)	0	1 (4)
Comorbidity			
Asthma/COPD	6 (12)	1 (7)	5 (14)
Chronic kidney disease	2 (4)	2 (14)	0
CVD			
Previous myocardial infarct	6 (12)	2 (14)	4 (11)
Atrial arrythmia	2 (4)	1 (7)	1 (3)
Previous stroke	2 (4)	0	2 (5)
Dyslipidaemia	21 (41)	6 (43)	15 (41)
Hypertension	30 (59)	11 (79)	19 (51)
Obesity	25 (49)	11 (79)	14 (38)
Diabetes mellitus/pre-diabetes	9 (18)	3 (21)	6 (16)

SD = standard deviation; BMI = body mass index; COPD = chronic obstructive pulmonary disease; CVD = cardiovascular disease; Hip = Hip arthroplasty participants; Knee = Knee arthroplasty participants; peak VO_2 = peak oxygen consumption.
Data are mean (SD) or as an absolute number with the percentage (%) of the whole. High fit = peak VO_2 >15mL/min/kg; Low fit = peak VO_2 <15mL/min/kg.

had a higher BMI and on average were Class 1 obese.[22] There were no differences in any CPET variables between hip and knee arthroplasty participants, except the oxygen uptake efficiency slope was lower (-418 AU, 95% CI: -823 to -12, p=0.044) in participants scheduled for hip arthroplasty.

Association between pre-operative peak VO_2 and post-operative functional and subjective recovery

Low fit vs high fit participants

Participants were categorised into high fit or low fit based on their peak VO_2 (i.e., >15 or <15mL/min/kg respectively). At 6-weeks post-operative, participants with a pre-operative high fit score performed five more sit to stand repetitions (95% CI: 3 to 7, p=0.002) and performed an additional 3,460 steps/day (95% CI: 1,053 to 5,867, p=0.006) compared to low fit participants (Table 2). Low fit participants decreased their timed up and go test duration to a greater extent but remained 2.2 seconds slower (95% CI: 0.9 to 3.5, p=0.002) at 6-weeks post-operative. Estimated peak oxygen consumption was higher (via the DASI; +4.3mL/min/kg, 95% CI: 1.0 to 7.7, p=0.011), and impact of osteoarthritis (WOMAC; -8.5 AU [95% CI: 0.7 to 16.6, p=0.034]) was less for the high fit group at 6-weeks post-operative compared to the low fit group. High fit participants had a better early (i.e., 7-day post-operative; +12 AU, 95% CI: 3 to 22, p=0.014) and later (i.e., 6-weeks post-operative; +13 AU, 95% CI: 5 to 21, p=0.003) subjective recovery from surgery. Length of hospital stay was not statistically different between groups (0.8 days, 95% CI: -0.4 to 1.9, p=0.176). Analyses using alternate stratification thresholds for peak VO_2 (i.e., low fit ≤14.2mL/min/kg; high fit ≥18.2mL/min/kg),[23] and using the anaerobic threshold (i.e., > or <11 mL/min/kg) did not appreciably alter the statistical significance of these results (Appendix Tables 1 and 2).

Correlational and simple and multiple linear regression analysis

Pre-operative peak VO_2 was moderately correlated with post-operative daily step count (r=0.65, p <0.001) and timed up and go test time (r=-0.56, p <0.001). In regression analysis, for every 1.0mL/min/kg increase in pre-operative peak VO_2, the number of daily steps at 6-weeks post-operative increased by 361 (95% CI: 234 to 488; Table 3); this was slightly lower (209 steps; 95% CI: 32 to 386) in the multiple regression model (Table 3, Appendix Tables 3 and 4). Hip arthroplasty participants had a high correlation with pre-operative peak VO_2 and post-operative daily step count (r=0.73, p <0.001), and participants completed 490 (95% CI: 285 to 695) additional steps per day for each 1.0mL/min/kg increase in pre-operative fitness (p <0.001; Appendix Tables 5 and 6).

Pre-operative peak VO_2 was also a significant predictor of subjective recovery (Table 3; Appendix Tables 7 and 8). For every 1.0mL/min/kg higher pre-operative peak VO_2, surgical recovery scale score at 7-days and 6-weeks post-operative was 0.8 (95% CI: 0.2 to 1.4) and 0.7 (95% CI: 0.3 to 1.1) higher, and this was not statistically different for 6-week post-operative score when controlling for age, sex and BMI. Pre-operative peak VO_2 was not associated with length of hospital stay (p=0.204; Table 3) for either arthroplasty type (p ≥0.350; Appendix Tables 5 and 6).

Non-CPET measures for estimating post-arthroplasty recovery

Participants in the high fit group as estimated by the DASI (estimated peak VO_2 >15mL/min/kg) post-operatively performed five more sit to stand repetitions (95% CI: 3 to 7, p <0.001) and performed an additional 2,026 steps/day (95% CI: 558 to 3,492, p=0.007) compared to low fit participants (i.e., <15mL/min/kg; Table 4). High fit participants had a better early (i.e., 7-days post-operative; +11 AU, 95% CI: 1 to 20, p=0.027) and later (i.e., 6-weeks post-operative; +11 AU, 95% CI: 3 to 19, p=0.007) subjective recovery from surgery. Estimated pre-operative peak VO_2 using the DASI was moderately correlated with post-operative functional recovery (r=0.51, p <0.001; Appendix Table 9). For every 1mL/min/kg increase in estimated pre-operative peak VO_2, participants performed 497 (95% CI: 250 to 744) more steps per day, were 0.3 seconds quicker on the timed up and go test (95% CI: -0.5 to -0.1) and scored 1.1 (95% CI: 0.1 to 2.1) points higher on the SRS at 6-weeks post-operative. Estimated pre-operative peak VO_2 was not associated with length of hospital stay (+0.3 days, 95% CI: -0.7 to 1.4, p=0.511).

Pre-operative daily step count was highly correlated with post-operative step count (r=0.83, p <0.001; Appendix Table 10). Low corelations were observed for pre-operative daily step count with other indices of functional and subjective recovery (r ≤0.43, p ≤0.036).

Table 2: Six-weeks post-operative functional and subjective measures in low and high fit participants.

Variable	Low fit (*n*=14)		High fit (*n*=37)		Group	Time	Group×Time Interaction
	Pre-operative	Post-operative	Pre-operative	Post-operative			
Functional							
Sit to stand (reps)	6 (5)	7 (4)	12 (5)	12 (5)	<0.001	0.894	0.301
Timed up and go (s)	14.5 (5.1)	10.3 (2.6)	10.3 (2.7)	8.1 (1.7)	<0.001	<0.001	0.004
Daily steps (n)	3,641 (1,748)	3,908 (1,569)	6,738 (3,186)	7,368 (4,335)	0.002	0.226	0.622
Knee ROM TJA side (°)	109 (19)	110 (12)	116 (16)	114 (16)	0.378	0.831	0.322
Knee ROM non-TJA side (°)	122 (17)	123 (12)	125 (11)	125 (11)	0.530	0.606	0.897
Subjective							
WOMAC	73 (13)	27 (16)	54 (17)	18 (11)	0.003	<0.001	0.052
DASI—estimated peak $\dot{V}O_2$ (mL/min/kg)	13.9 (2.6)	18.4 (4.0)	18.1 (4.7)	22.4 (5.5)	0.002	<0.001	0.969
SRS—7-days post-operative	-	47 (18)	-	59 (13)	0.014	-	
SRS—6-weeks post-operative	-	66 (14)	-	76 (13)	0.003	-	
Other							
Length of hospital stay (days)	-	4.4 (2.7)	-	3.7 (1.2)	0.176	-	

CPET = cardiopulmonary exercise test; DASI = Duke Activity Status Index (0 worst–58 best); peak $\dot{V}O_2$ = peak oxygen consumption; ROM = range of motion; SRS = Surgical Recovery Scale—higher score is best; TJA = total joint arthroplasty; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index (best 0–94 worst).
Variables are presented as mean (SD) and analysed with a repeated-measures ANOVA test or Independent Samples *t*-test. High fit = peak $\dot{V}O_2$ >15mL/min/kg; Low fit = peak $\dot{V}O_2$ <15mL/min/kg.

Table 3: Simple linear regression analysis of pre-operative peak VO_2 with 6-weeks post-operative assessments (n=51 for all).

Outcome	Slope (95% CI)	R^2	P-value
Functional			
Daily step count	361 (234 to 488)	0.42	**<0.001**
Timed up and go test	-0.2 (-0.4 to 0.1)	0.31	**<0.001**
Subjective			
WOMAC	-0.5 (-0.9 to 0.1)	0.08	**0.049**
SRS—7-days post-operative	0.8 (0.2 to 1.4)	0.13	**0.018**
SRS—6-weeks post-operative	0.7 (0.3 to 1.1)	0.13	**0.009**
Other			
Length of hospital stay	0 (0 to 0)	0.03	0.204

CI = confidence interval; DASI = Duke Activity Status Index; peak VO_2 = peak oxygen consumption; SRS = Surgical Recovery Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index.
Pre-operative peak VO_2 was used as the predictor.

Discussion

Participants who had a low pre-operative peak VO_2 (i.e., <15mL/min/kg) on average performed ~50% (5) fewer sit to stand reps, approximately 60% (3,500) fewer daily steps and took ~2 seconds longer to perform the timed up and go test at 6-weeks post-operative; they also had poorer subjective surgical recovery at 7-days and 6-weeks post-operative. Pre-operative peak VO_2 had the highest correlation with functional and subjective recovery at 6-weeks post-operative. Pre-operative daily step count was highly correlated with post-operative step count; that is, participants who were more active pre-operatively were more likely to be active 6-weeks post-operative. Lastly, estimated pre-operative peak VO_2 using the DASI was moderately correlated with post-operative functional recovery.

These results reinforce previous work highlighting the clinical importance of pre-operative fitness in patients undergoing non-cardiac surgery and underscore the importance of pre-operative fitness as a prognostic surgical indicator for patients scheduled for hip or knee arthroplasty. Additionally, estimated pre-operative fitness (via the DASI or daily step count) may be as effective for predicting recovery from arthroplasty as the gold standard CPET.

What is the effect of pre-operative peak VO_2 on functional and subjective recovery following hip or knee arthroplasty?

In this study a low pre-operative peak VO_2 was associated with poorer functional and subjective recovery from arthroplasty at 6-weeks post-operative. Early and longer-term perceived recovery from surgery was poorer in low fit participants, and despite the surgery, the impact of osteoarthritis remained significantly worse in these participants at 6-weeks post-operative. The rate of functional and subjective recovery was similar for both groups; however, low fit participants had worse pre-operative scores, resulting in worse 6-week post-operative values. Although the timed up and go test was an exception and improved *more* than high fit participants, the difference between groups was still up to twice the minimal clinically important difference (i.e., ~1.2 seconds).[24,25] Of all post-operative outcomes, daily step count at 6-weeks had the highest correlation with pre-operative peak VO_2 (r=0.65,

Table 4: Six-weeks post-operative functional and subjective measures in DASI estimated low fit (<15mL/min/kg) and high fit (>15mL/min/kg) participants.

Variable	Low fit (*n*=18)		High fit (*n*=33)		Group	Time	Group×Time Interaction
	Pre-operative	Post-operative	Pre-operative	Post-operative			
Functional							
Sit to stand (reps)	7 (5)	7 (5)	13 (5)	12 (4)	<0.001	0.856	0.509
Timed up and go (s)	13.1 (4.5)	10.2 (2.4)	9.0 (3.4)	7.9 (1.7)	<0.001	<0.001	0.050
Daily steps (n)	4,592 (2,582)	5,071 (3,141)	6,581 (3,279)	7,132 (4,376)	0.048	0.141	0.917
Knee ROM TJA side (°)	114 (13)	111 (13)	114 (19)	114 (15)	0.929	0.598	0.574
Knee ROM non-TJA side (°)	121 (13)	123 (9)	125 (13)	126 (13)	0.390	0.477	0.532
Subjective							
WOMAC	72 (10)	27 (13)	52 (17)	17 (11)	<0.001	<0.001	0.060
DASI—estimated peak $\dot{V}O_2$ (mL/min/kg)	13.0 (1.2)	20.3 (6.8)	19.4 (3.3)	21.7 (4.8)	<0.001	<0.001	0.003
SRS—7-days post-operative	-	49 (15)	-	60 (14)	0.027	-	
SRS—6-weeks post-operative	-	66 (16)	-	77 (13)	0.007	-	
Other							
Length of hospital stay (days)	-	4.1 (2.1)	-	3.7 (1.4)	0.511	-	

DASI = Duke Activity Status Index (0 worst–58 best); peak $\dot{V}O_2$ = peak oxygen consumption; ROM = range of motion; SRS = Surgical Recovery Scale—higher score is best; TJA = total joint arthroplasty; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index (best 0–94 worst).
Variables are presented as mean (SD) and analysed with a repeated measures ANOVA test or Independent Samples *t*-test.

p <0.001), and pre-operative peak VO_2 was still predictive when controlling for age, sex and BMI; this correlation was slightly higher in hip arthroplasty participants compared to knee arthroplasty participants (r=0.73 vs 0.59). Pre-operative measures of peak VO_2 had a low–moderate correlation with other measures of physical function at 6-weeks post-operative (e.g., timed up and go test, 30-second sit to stand test).

As this is the first study to compare pre-operative peak VO_2 and post-arthroplasty recovery, no comparative evidence is available. However, theoretical models propose that those with higher pre-operative peak VO_2 retain a higher level of functional capacity following surgery,[4] and therefore should be able to ambulate and return to normal activities of daily living earlier, contributing to improved functional and subjective outcomes (e.g., quality of life, satisfaction). The findings of the current study support this hypothesis, with higher fit patients having a quicker perceived surgical recovery at 7-days and 6-weeks post-operative and greater physical function and physical activity at 6-weeks post-operative.

Pre-operative peak VO_2 was not associated with length of hospital stay for either hip or knee arthroplasty participants. Previous studies have reported clinician rated functional capacity (i.e., American Society of Anaesthesiologists [ASA], particularly those III/IV) to be associated with longer hospital stay.[26] Pre-operative peak VO_2 is considered a more discriminate measure of functional capacity; the lack of association in the present study may be attributable to insufficient statistical power.

When compared to other indices of cardiorespiratory fitness (e.g., anaerobic threshold), peak VO_2 had higher correlations with functional and subjective recovery. When using a binary cut-off for anaerobic threshold, the relationships with post-operative recovery were similar to those for peak VO_2. This is reassuring, as maximal effort is not always possible in patients with osteoarthritis; often the test is terminated prematurely due to the lower-limb limitations. Therefore, in practice, when a representative peak VO_2 is unable to be obtained during pre-operative CPET, determining an anaerobic threshold from sub-maximal exercise appears to have a similar ability to predict how well a patient functionally recovers post-operatively. An individual with either a peak VO_2 <15mL·kg^{-1}·min^{-1} or anaerobic threshold <11mL·kg^{-1}·min^{-1} may require more attention pre-operatively to improve cardiorespiratory fitness, or greater care post-operatively, to increase the likelihood of greater physical function after hip or knee arthroplasty.

Are non-CPET pre-operative assessments associated with functional and subjective recovery following hip or knee arthroplasty?

The DASI questionnaire has utility as an alternative to pre-operative CPET for risk stratification.[10] The results from the present study show it may be as effective for stratifying patients as low or high fit and predicting post-operative functional recovery following arthroplasty as CPET. However, there was still a large amount of variability explained by other factors, and similar to CPET it had only low correlations with subjective recovery (i.e., r ≤0.35). Wijeysundera et al.[10] showed the predictive capacity of the DASI for post-operative complication to be as good as CPET and significantly better than a clinician's subjective assessment of risk. Although cardiopulmonary exercise testing can provide additional insight on how patients respond physiologically to stress,[4] an advantage of the DASI is that being questionnaire-based, it can be performed anywhere and does not require the patient to be in the presence of a clinician or other healthcare worker. This has utility in any setting where the public hospital serves a large geographical area, with many pre-operative assessments conducted via telemedicine. Furthermore, it may be useful for triaging patients for pre-operative CPET if resources are scarce, and for identifying high priority patients for prehabilitation. Future adequately powered studies should compare estimated peak VO_2 via the DASI questionnaire with CPET-measured peak VO_2 to confirm these findings and its utility for detecting surgical complications.

We reported that low fit participants had a larger improvement in estimated peak VO_2 from pre-operative to 6-week post-operative assessments than high fit participants (Table 4). A limitation of the DASI is that it has poor ability to detect change in peak VO_2 across time.[11] Although we did not directly measure peak VO_2 at 6-weeks post-operative, it is likely the reported improvement in estimated peak VO_2 is due to improved function from the surgery and ability to tick "yes" to more questions, rather than an improvement in fitness per se.

The strongest correlation with physical activity at 6-weeks post-operative was pre-operative physical activity. This is likely attributable in part to multicollinearity; nevertheless, a higher pre-operative step count was associated with a higher step count

6-weeks post-operatively (r=0.83, p=<0.001), and this is an important and valid measure of functionality. Consistent with previous research,[27–30] the present study highlighted that despite the surgical procedure, there was no difference between pre-operative physical activity and physical activity at 6-weeks post-operative. Essentially, those most active before surgery are also most active after surgery, and one could argue that their severe osteoarthritis diagnosis was not the limiting factor to physical activity. This relationship persists at 6-months,[27,28] 9-months[29] and 1-year post-operative[28,30] in hip and knee arthroplasty recipients.

Despite the high correlation between pre-operative and post-operative daily step count in the present study, previous research has shown limited association with pre-operative daily step count and other functional post-arthroplasty outcomes.[31–33] In particular, there appears to be no clear relationship with time to return to work or subjective recovery,[31,32] but increased pre-operative physical activity may be associated with less post-operative pain.[33] Research in other surgery types has shown a relationship between pre-operative physical activity and outcomes following surgery. Richards et al.[7] reported that in patients aged 65 years and older undergoing major abdominal surgery, those with a median step count <2,500 steps/day had a more than two-fold greater length of hospital stay (14 days vs 6 days, p <0.001) compared to those with a normal step count. Low step count also increased the risk of intensive care unit admission and post-operative complications.

Limitations

The findings of this study must be considered in the context of several limitations. A convenience sample was used for this study and the sample size was relatively small; not all participants recruited into the original RCT could be included, largely due to loss to follow-up related to COVID-19 (e.g., lockdowns, extended surgical waitlisting). Despite this, *post hoc* between-group power for the main physical and subjective variables was high, ranging from 0.86–0.99. Approximately 10% of patients were excluded from the original study due to medical contraindications to CPET. Therefore, the findings of this study may not be as relevant for high-risk patients. Six-weeks post-operative is still relatively early in the recovery period, particularly for knee arthroplasty. It remains unclear whether the differences between low fit and high fit participants persist later in recovery; nevertheless, fitness appears to be associated with superior early post-operative functional and subjective recovery. All participants were provided in-hospital rehabilitation and standardised advice to continue their rehabilitation at home. Participation in rehabilitation is influential in the recovery from surgery, and as rehabilitation compliance was not assessed in this study, this must be considered when interpreting the study findings.[34] CPETs were performed on two different modalities, and based on previous work this will have influenced some CPET variables.[14] It is possible that the presence of comorbidities (i.e., higher rates of obesity, hypertension and chronic kidney disease) or the severity of osteoarthritis contributed to lower fitness and/or the poor post-operative recovery. However, controlling for WOMAC score and BMI in the statistical analysis will have limited confounding or bias. Moreover, the presence of good fitness can offset the deleterious effects of comorbidities,[35] underscoring the importance of prehabilitation to minimise the potential impact of pre-operative comorbidities. The potential for overfitting and multicollinearity in regression modelling is acknowledged, particularly the high correlation between pre-operative and post-operative daily step count.

Conclusions

These findings suggest that participants with low pre-operative fitness have poorer functional and perceived outcomes following hip or knee arthroplasty. Directly measured pre-operative peak VO_2 had similar associations with post-operative functional outcomes as pre-operative daily step count and estimated peak VO_2 via the DASI questionnaire; these have potential as a post-arthroplasty quality of recovery prediction tool when pre-operative CPET is not indicated or available. Pre-operative fitness, either measured or estimated, and daily step count are useful clinical tools for predicting functional and subjective recovery following hip or knee arthroplasty. Future work should investigate whether pre-operative optimisation of these predictors translates to improved post-operative functional and subjective recovery, and clinical outcomes.

COMPETING INTERESTS
No conflicts of interest, financial or otherwise, are declared by the authors.
This study was funded by the Health Research Council of New Zealand (grant number: 18/636 [KT]) and a Health Research South Start-Up Award, University of Otago (KT). BR was supported by a University of Otago Doctoral Scholarship. The funders had no role in any part of the study or any decision related to publication.

ACKNOWLEDGEMENTS
The authors would like to thank all participants for the time and effort associated with study participation. Additionally, Prof Andre van Rij for his expertise and advice related to this study and Ruth Ford for her assistance with participant recruitment.

AUTHOR INFORMATION
Brendon H Roxburgh: Lecturer, School of Physical Education, Sport and Exercise Sciences, University of Otago, Dunedin, New Zealand; Department of Surgical Sciences, University of Otago, Dunedin, New Zealand; HeartOtago, University of Otago, Dunedin, New Zealand.
Holly A Campbell: Assistant Research Fellow, Department of Surgical Sciences, University of Otago, Dunedin, New Zealand; HeartOtago, University of Otago, Dunedin, New Zealand.
James D Cotter: Professor, School of Physical Education, Sport and Exercise Sciences, University of Otago, Dunedin, New Zealand; HeartOtago, University of Otago, Dunedin, New Zealand.
Ulla Reymann: Anaesthetist, Department of Surgical Sciences, University of Otago, Dunedin, New Zealand.
Michael JA Williams: Cardiologist, HeartOtago, University of Otago, Dunedin, New Zealand; Department of Medicine, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand.
David P Gwynne-Jones: Professor and Orthopaedic Surgeon, Department of Surgical Sciences, University of Otago, Dunedin, New Zealand; Te Whatu Ora – Southern, Dunedin, New Zealand.
Kate N Thomas: Senior Lecturer, Department of Surgical Sciences, University of Otago, Dunedin, New Zealand; HeartOtago, University of Otago, Dunedin, New Zealand.

CORRESPONDING AUTHOR
Dr Brendon H Roxburgh: School of Physical Education, Sport and Exercise Sciences, University of Otago, 55 Union Street West, Dunedin, New Zealand.
E: brendon.roxburgh@otago.ac.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/the-effect-of-pre-operative-cardiorespiratory-fitness-on-functional-and-subjective-outcomes-following-total-hip-and-knee-arthrop

REFERENCES
1. Wang W, Morrison TA, Geller JA, et al. Predicting short-term outcome of primary total hip arthroplasty: a prospective multivariate regression analysis of 12 independent factors. J Arthroplasty. 2010;25(6):858-64. doi: 10.1016/j.arth.2009.06.011.
2. Lingard EA, Katz JN, Wright EA, et al. Predicting the outcome of total knee arthroplasty. J Bone Joint Surg Am. 2004;86(10):2179-86. doi: 10.2106/00004623-200410000-00008.
3. Moran J, Wilson F, Guinan E, et al. Role of cardiopulmonary exercise testing as a risk-assessment method in patients undergoing intra-abdominal surgery: a systematic review. Br J Anaesth. 2016;116(2):177-91. doi: 10.1093/bja/aev454.
4. Roxburgh BH, Cotter JD, Campbell HA, et al. Physiological relationship between cardiorespiratory fitness and fitness for surgery: a narrative review. Br J Anaesth. 2023;130(2):122-32. doi: 10.1016/j.bja.2022.10.039.
5. Levett DZH, Jack S, Swart M, et al. Perioperative cardiopulmonary exercise testing (CPET): consensus clinical guidelines on indications, organization, conduct, and physiological interpretation. Br J Anaesth. 2018;120(3):484-500. doi: 10.1016/j.bja.2017.10.020.
6. Fletcher GF, Ades PA, Kligfield P, et al. Exercise standards for testing and training: a scientific statement from the American Heart Association. Circulation. 2013;128(8):873-934. doi: 10.1161/CIR.0b013e31829b5b44.
7. Richards SJ, Jerram PM, Brett C, et al. The association between low pre-operative step count and adverse post-operative outcomes in older patients undergoing colorectal cancer surgery. Perioper Med. 2020;9:1-10.
8. Nakajima H, Yokoyama Y, Inoue T, et al. How many steps per day are necessary to prevent postoperative complications following hepato-pancreato-biliary surgeries for malignancy? Ann Surg Oncol. 2020;27(5):1387-97. doi: 10.1245/s10434-020-08218-x.
9. Sobejana M, van den Hoek J, Metsios GS, et al. Cardiorespiratory fitness and physical activity in people who have rheumatoid arthritis at an increased risk of cardiovascular disease: a cross-sectional study. Rheumatol Int. 2021;41(12):2177-

83. doi: 10.1007/s00296-021-04903-6.
10. Wijeysundera DN, Pearse RM, Shulman MA, et al. Assessment of functional capacity before major non-cardiac surgery: an international, prospective cohort study. Lancet. 2018;391(10140):2631-40. doi: 10.1016/S0140-6736(18)31131-0.
11. Roxburgh BH, Campbell HA, Cotter JD, et al. Upper-limb high-intensity interval training or passive heat therapy to optimise cardiorespiratory fitness prior to total hip or knee arthroplasty: a randomised controlled trial. Arthritis Care Res. 2024;76(3):393-402. doi: 10.1002/acr.25238.
12. Paddison JS, Sammour T, Kahokehr A, et al. Development and Validation of the Surgical Recovery Scale (SRS). J Surg Res. 2011;167(2):85-91. doi: 10.1016/j.jss.2010.12.043.
13. Myers J, Forman DE, Balady GJ, et al. Supervision of exercise testing by nonphysicians: a scientific statement from the American Heart Association. Circulation. 2014;130(12):1014-27. doi: 10.1161/CIR.0000000000000101.
14. Roxburgh BH, Campbell HA, Cotter JD, et al. Cardiopulmonary exercise testing in severe osteoarthritis: a crossover comparison of four exercise modalities. Anaesthesia. 2021;76(1):72-81. doi: 10.1111/anae.15162.
15. Rikli RE, Jones CJ. Senior Fitness Test Manual. US: Human Kinetics; 2013.
16. Norkin CC, White DJ. Measurement of joint motion: a guide to goniometry. US: FA Davis Company; 2016.
17. Gandhi SK, Salmon JW, Zhao SZ, et al. Psychometric evaluation of the 12-item short-form health survey (SF-12) in osteoarthritis and rheumatoid arthritis clinical trials. Clin Ther. 2001;23(7):1080-98. doi: 10.1016/s0149-2918(01)80093-x.
18. Fransen M, Edmonds J. Reliability and validity of the EuroQol in patients with osteoarthritis of the knee. Rheumatology. 1999;38(9):807-13. doi: 10.1093/rheumatology/38.9.807.
19. Hlatky MA, Boineau RE, Higginbotham MB, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index). Am J Cardiol. 1989;64(10):651-4. doi: 10.1016/0002-9149(89)90496-7.
20. Bellamy N, Buchanan WW, Goldsmith CH, et al. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. J Rheumatol. 1988;15(12):1833-40.
21. Mukaka MM. Statistics corner: A guide to appropriate use of correlation coefficient in medical research. Malawi Med J. 2012;24(3):69-71.
22. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. National Institutes of Health. Obes Res. 1998;6 Suppl 2:51S-209S. Erratum in: Obes Res 1998;6(6):464.
23. Rose GA, Davies RG, Davison GW, et al. The cardiopulmonary exercise test grey zone; optimising fitness stratification by application of critical difference. Br J Anaesth. 2018;120(6):1187-94. doi: 10.1016/j.bja.2018.02.062.
24. Wright AA, Cook CE, Baxter GD, et al. A comparison of 3 methodological approaches to defining major clinically important improvement of 4 performance measures in patients with hip osteoarthritis. J Orthop Sports Phys Ther. 2011;41(5):319-27. doi: 10.2519/jospt.2011.3515.
25. Yuksel E, Kalkan S, Cekmece S, et al. Assessing Minimal Detectable Changes and Test-Retest Reliability of the Timed Up and Go Test and the 2-Minute Walk Test in Patients With Total Knee Arthroplasty. J Arthroplasty. 2017;32(2):426-30. doi: 10.1016/j.arth.2016.07.031.
26. Mathijssen NM, Verburg H, van Leeuwen CC, et al. Factors influencing length of hospital stay after primary total knee arthroplasty in a fast-track setting. Knee Surg Sports Traumatol Arthrosc. 2016;24(8):2692-6. doi: 10.1007/s00167-015-3932-x.
27. Harding P, Holland AE, Delany C, Hinman RS. Do activity levels increase after total hip and knee arthroplasty? Clin Orthop Relat Res. 2014;472(5):1502-11. doi: 10.1007/s11999-013-3427-3.
28. Arnold JB, Walters JL, Ferrar KE. Does Physical Activity Increase After Total Hip or Knee Arthroplasty for Osteoarthritis? A Systematic Review. J Orthop Sports Phys Ther. 2016;46(6):431-42. doi: 10.2519/jospt.2016.6449.
29. Moellenbeck B, Horst F, Gosheger G, et al. Sedentary Behavior in Older Patients before and after Total Hip Arthroplasty: A Prospective Cohort Study. Healthcare (Basel). 2020;8(3):346-58. doi: 10.3390/healthcare8030346.
30. Gwam CU, Mohamed NS, Castrodad IMD, et al. Accelerometer–measured physical activity levels before and after total hip arthroplasty: A cohort study. Hong Kong J Orthop Res. 2019;2(3):17-20.
31. Boersma AR, Brouwer S, Koolhaas W, et al. No association between preoperative physical activity level and time to return to work in patients after total hip or knee arthroplasty: A prospective cohort study. PLoS ONE. 2019;14(9):e0221932. doi: 10.1371/journal.pone.0221932.
32. Poortinga S, van den Akker-Scheek I, Bulstra SK, et al. Preoperative physical activity level has no relationship to the degree of recovery one year after

primary total hip or knee arthroplasty: a cohort study. PLoS ONE. 2014;9(12):e115559. doi: 10.1371/journal.pone.0115559.
33. Pozzobon D, Ferreira PH, Blyth FM, et al. Can obesity and physical activity predict outcomes of elective knee or hip surgery due to osteoarthritis? A meta-analysis of cohort studies. BMJ Open. 2018;8(2):e017689. doi: 10.1136/bmjopen-2017-017689.
34. Minns Lowe CJ, Barker KL, Dewey M, Sackley CM. Effectiveness of physiotherapy exercise after knee arthroplasty for osteoarthritis: systematic review and meta-analysis of randomised controlled trials. BMJ. 2007;335(7624):812-5. doi: 10.1136/bmj.39311.460093.BE.
35. Blair SN, Kampert JB, Kohl HW 3rd, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA. 1996;276(3):205-10.

Appendix: Supplementary results

Six-week post-operative physical function

Daily step count was not different before and 6-weeks after surgery (p=0.468; Appendix Table 11). Post-operatively, participants spent approximately 10 hours per day in sitting activities. This included more than 6 hours per day in seated bouts greater than 30 minutes and almost 4 hours per day in seated bouts greater than 60 minutes, with no obvious between-group differences. Timed up and go time improved by 16% (~2 seconds decrease; p=0.015; Appendix Table 12); this decrease was a 25% improvement for hip arthroplasty participants (p=0.047). On the arthroplasty side, knee extensor strength increased by 12% from pre-operative to 6-weeks post-operative (p=0.030; Appendix Table 13).

Six-week post-operative perceived subjective health and surgical recovery

The subjective impact of osteoarthritis improved overall (p <0.001), with the largest improvements in hip arthroplasty participants (p <0.001; Appendix Table 14). Perceived quality of life (EQ-5D) improved overall (p ≤0.007), but the improvement was larger in hip arthroplasty participants (p=0.001). SRS score increased across time (p <0.001) and similarly for hip and knee arthroplasty participants (p=0.206).

Appendix Table 1: Six-week post-operative functional, subjective and other measures in low fit (CPET derived pre-operative peak VO_2 <14.2mL·min^{-1}·kg^{-1}) and high fit (>18.2mL·min^{-1}·kg^{-1}) participants. Data are presented as mean (SD) and analysed with an ANCOVA test (covariates—age and pre-operative WOMAC).

Variable	Low fit (*n*=11) <14.2mL·min^{-1}·kg^{-1}	High fit (*n*=25) >18.2mL·min^{-1}·kg^{-1}	Statistical significance
Functional			
Sit to stand (reps)	7 (4)	12 (4)	<0.001
Timed up and go (s)	10.4 (2.8)	7.7 (1.7)	<0.001
Daily steps (n)‡	3,780 (1,555)	7,997 (4,531)	0.003
Knee ROM TJA side (°)	109 (12)	113 (18)	0.501
Knee ROM non-TJA side (°)	124 (13)	128 (11)	0.310
Subjective			
WOMAC	27 (17)	19 (11)	0.034
DASI—estimated peak VO_2 (mL·min^{-1}·kg^{-1})	18.6 (6.3)	22.7 (4.8)	0.026
SRS—7-days post-operative	46 (18)	61 (12)	0.007
SRS—6-weeks post-operative	66 (14)	76 (13)	0.012
Other			
Length of hospital stay (days)	4.4 (2.7)	3.6 (1.4)	0.226

DASI = Duke Activity Status Index (0 worst–58 best); peak VO_2 = peak oxygen consumption; ROM = range of motion; SRS = Surgical Recovery Score—higher score is best; TJA = total joint arthroplasty; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index (best 0–94 worst).
‡ = log transformed for statistical analysis.

Appendix Table 2: Six-week post-operative function, subjective and other measures in participants with low anaerobic threshold (<11mL·min^{-1}·kg^{-1}) and high anaerobic threshold (>11mL·min^{-1}·kg^{-1}). Data are presented as mean (SD) and analysed with an ANCOVA test (covariates—age and pre-operative WOMAC).

Variable	Low fit (*n*=23) <11mL·min^{-1}·kg^{-1}	High fit (*n*=28) >11mL·min^{-1}·kg^{-1}	Statistical significance
Functional			
Sit to stand (reps)	8 (5)	12 (4)	0.002
Timed up and go (s)	9.7 (2.4)	7.8 (1.7)	0.003
Daily steps (n)‡	4,867 (2,752)	7,622 (4,567)	0.017
Knee ROM TJA side (°)	113 (11)	113 (11)	0.999
Knee ROM non-TJA side (°)	121 (11)	128 (11)	0.049
Subjective			
WOMAC	23 (15)	19 (11)	0.350
DASI—estimated peak VO_2 (mL·min^{-1}·kg^{-1})	21.2 (9.5)	22.3 (5.0)	0.575
SRS—Day 7 post-operative	50 (17)	61 (11)	0.016
SRS—6-weeks post-operative	68 (15)	77 (11)	0.016
Other			
Length of hospital stay (days)	4.2 (2.1)	3.5 (1.4)	0.154

DASI = Duke Activity Status Index (0 worst–58 best); peak VO_2 = peak oxygen consumption; ROM = range of motion; SRS = Surgical Recovery Score—higher score is best; TJA = total joint arthroplasty; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index (best 0–94 worst).
‡ = log transformed for statistical analysis.

Appendix Table 3: Multiple linear regression analysis of pre-operative peak VO_2 and 6-weeks post-operative daily step count. n=51.

Outcome	Slope	SE	t	R^2	Adjusted-R^2	*F*	P-value
				0.53	0.48	11.9	<0.001
Intercept	17,408	7,034	2.5				0.017
Relative peak VO_2	209	88	2.4				0.022
Age	-107	64	-1.7				0.103
Sex (male)	742	1,040	0.7				0.480
Body mass index	-262	90	-2.9				0.006

F = F-statistic; peak VO_2 = peak oxygen consumption; SE = standard error of the estimate; t = t-statistic.

Appendix Table 4: Multiple linear regression analysis of pre-operative peak VO_2 and 6-weeks post-operative timed up and go test. n=51.

Outcome	Slope	SE	t	R^2	Adjusted-R^2	*F*	P-value
				0.42	0.36	7.7	<0.001
Intercept	10.6	4.2	2.5				0.016
Relative peak VO_2	-0.2	0.1	-3.3				0.002
Age	0.0	0.0	-0.6				0.545
Sex (male)	1.0	0.6	1.6				0.113
Body mass index	0.1	0.1	1.4				0.163

F = F-statistic; peak VO_2 = peak oxygen consumption; SE = standard error of the estimate; t = t-statistic.

Appendix Table 5: Simple linear regression analysis of pre-operative peak VO_2 with 6-weeks post-operative assessments for hip arthroplasty participants. n=23.

Outcome	Slope	Standard error	R^2	*F*	P-value
Functional					
Daily step count	490	102	0.53	23.3	<0.001
Timed up and go test	-0.2	0.06	0.33	9.2	0.007
Subjective					
WOMAC	-0.6	0.3	0.16	4.10	0.056
SRS—7-days post-operative	0.2	0.5	0.01	0.18	0.680
SRS—6-weeks post-operative	0.5	0.5	0.05	1.2	0.282
Other					
Length of hospital stay	-0.1	0.1	0.05	0.9	0.350

DASI = Duke Activity Status Index; peak VO_2 = peak oxygen consumption; SRS = Surgical Recovery Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index.

Appendix Table 6: Simple linear regression analysis of pre-operative peak VO_2 with 6-weeks post-operative assessments for knee arthroplasty participants. n=28.

Outcome	Slope	Standard error	R^2	*F*	P-value
Functional					
Daily step count	267	75	0.35	12.6	0.002
Timed up and go test	-0.2	0.0	0.31	11.0	0.003
Subjective					
WOMAC	-0.5	0.3	0.08	2.0	0.165
SRS—7-days post-operative	1.2	0.4	0.36	10.6	0.004
SRS—6-weeks post-operative	0.6	0.3	0.15	4.3	0.048
Other					
Length of hospital stay	0.0	0.0	0.02	0.6	0.434

DASI = Duke Activity Status Index; peak VO_2 = peak oxygen consumption; SRS = Surgical Recovery Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index.

Appendix Table 7: Multiple linear regression analysis of pre-operative peak VO_2 and 6-weeks post-operative subjective surgical recovery. n=51.

Outcome	Slope	SE	t	R^2	*F*	P-value
				0.21	3.0	0.028
Intercept	54.9	31.2	1.8			0.085
Relative peak VO_2	0.9	0.4	2.3			0.024
Age	0.2	0.3	0.6			0.554
Sex (male)	-7.0	4.7	-1.5			0.142
Body mass index	-0.2	0.4	-0.6			0.548

F = F-statistic; SE = standard error of the estimate; t = t-statistic.

Appendix Table 8: Multiple linear regression analysis of pre-operative peak VO_2 and 6-weeks post-operative WOMAC score. n=51.

Outcome	Slope	SE	t	R^2	*F*	P-value
				0.15	2.0	0.111
Intercept	20.6	28.7	0.7			0.477
Relative peak VO_2	-0.5	0.3	-1.4			0.154
Age	-0.1	0.3	-0.3			0.804
Sex (male)	4.4	4.3	1.0			0.313
Body mass index	0.4	0.4	1.0			0.300

F = F-statistic; SE = standard error of the estimate; t = t-statistic.

Appendix Table 9: Simple linear regression analysis of estimated peak VO_2 (DASI questionnaire) with 6-weeks post-operative assessments. n=51.

Outcome	Slope	SE	R^2	P-value
Functional				
Daily step count	497	123	0.26	<0.001
Timed up and go test	-0.3	0.1	0.26	<0.001
Subjective				
WOMAC	-0.9	0.4	0.08	0.051
SRS—7-days post-operative	1.3	0.5	0.13	0.018
SRS—6-weeks post-operative	1.1	0.5	0.10	0.029
Other				
Length of hospital stay	-0.1	0.0	0.04	0.162

DASI = Duke Activity Status Index; peak VO_2 = peak oxygen consumption; SE = standard error of the estimate; SRS = Surgical Recovery Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index.

Appendix Table 10: Pearson correlation coefficient and simple linear regression analysis of pre-operative daily step count with 6-weeks post-operative assessments. n=51.

Outcome	Slope	SE	R^2	P-value
Functional				
Daily step count	1.06	0.11	0.68	<0.001
Timed up and go test	0.001	<0.001	0.19	0.003
Subjective				
WOMAC	-0.001	<0.001	0.14	0.007
SRS—7-days post-operative	0.002	<0.001	0.17	0.006
SRS—6-weeks post-operative	0.001	<0.001	0.09	0.036
Other				
Length of hospital stay	0.0	0.0	0.02	0.414

DASI = Duke Activity Status Index; peak VO_2 = peak oxygen consumption; SE = standard error of the estimate; SRS = Surgical Recovery Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index.

Appendix Table 11: Pre-operative and 6-week post-operative daily physical activity overall and in total hip and knee arthroplasty participants. Data collected over a 7-day period and presented as daily mean (SD) and analysed with a paired samples *t*-test or repeated-measures ANOVA test.

	Overall (n=51)		**Hip arthroplasty (n=23)**		**Knee arthroplasty (n=28)**		**Hip vs knee**		
Variable	**PRE-OP**	**POST-OP**	**PRE-OP**	**POST-OP**	**PRE-OP**	**POST-OP**	**Group**	**Time**	**Group×Time Interaction**
Total steps (n)	5,835 (3,161)	6,359 (4,050)	5,619 (3,239)	6,571 (4,684)	6,034 (3,142)	6,165 (3,453)	0.996	0.106	0.217
Time spent upright (min)	327 (136)	320 (135)	347 (146)	347 (127)	309 (127)	295 (140)	0.204	0.696	0.673
Time spent stepping (min)	80 (36)	85 (46)	77 (38)	87 (53)	83 (35)	83 (39)	0.937	0.202	0.211
Time spent sitting (min)	576 (137)	610 (184)	562 (159)	563 (145)	589 (115)	654 (208)	0.138	0.192	0.201
Sit to stand transitions (reps)	36 (11)	36 (12)	32 (12)	33 (11)	39 (9)	39 (12)	0.029	0.747	0.969
Time spent in sitting bouts >30 min (min)	326 (134)	371 (187)	334 (145)	336 (142)	319 (125)	404 (219)	0.514	0.081	0.090
Time spent in sitting bouts >60 min (min)	187 (124)	225 (172)	191 (128)	209 (136)	183 (123)	239 (369)	0.768	0.114	0.403

PRE-OP = pre-operative; POST-OP = 6-weeks post-operative.
* p <0.05 vs PRE-OP (Overall only). Statistical significance for hip arthroplasty vs knee arthroplasty.

Appendix Table 12: Pre-operative and 6-week post-operative functional measures overall and in hip and knee arthroplasty participants. Data are presented as daily mean (SD) and analysed with a paired samples *t*-test or repeated-measures ANOVA test.

	Overall (n=51)		Hip arthroplasty (n=23)		Knee arthroplasty (n=28)		Statistical significance^		
Variable	**PRE-OP**	**POST-OP**	**PRE-OP**	**POST-OP**	**PRE-OP**	**POST-OP**	**Group**	**Time**	**Group×Time Interaction**
Functional measures									
30-second sit to stand test (*n*)	11 (6)	10 (5)	9 (5)	9 (4)	12 (5)	11 (4)	0.045	0.830	0.088
Timed up and go test (s)	10.4 (4.3)	8.7 (2.2)*	11.5 (4.3)	8.7 (2.1)^	9.6 (4.1)	8.7 (2.4)	0.295	<0.001	0.047
30-second arm curl test (*n*)	23 (6)	24 (5)	24 (5)	24 (4)	23 (7)	24 (6)	0.666	0.364	0.893

PRE-OP = pre-operative; POST-OP = 6-weeks post-operative.
* p <0.05 vs PRE-OP (Overall only). ^ = p <0.05 for hip arthroplasty vs knee arthroplasty.

Appendix Table 13: Isometric muscle strength on scheduled arthroplasty and non-scheduled arthroplasty side overall and in hip and knee arthroplasty participants. Data are presented as daily mean (SD) and analysed with a paired samples *t*-test or repeated-measures ANOVA test.

Variable	Overall (n=51)		Hip arthroplasty (n=23)		Knee arthroplasty (n=28)		Statistical significance^		
	PRE-OP	POST-OP	PRE-OP	POST-OP	PRE-OP	POST-OP	Group	Time	Group×Time Interaction
TJA side									
Knee Extension (N)	127 (55)	142 (44)	133 (56)	147 (50)	123 (54)	138 (39)	0.443	0.030	0.984
Knee Flexion (N)	87 (35)	85 (30)	85 (34)	97 (34)	90 (37)	74 (22)^	0.227	0.751	0.013
Hip Abduction (N)	78 (39)	91 (28)*	67 (26)	83 (24)	87 (46)	98 (29)	0.036	0.004	0.553
Hip Extension (N)	87 (38)	83 (35)	78 (33)	82 (29)	95 (42)	83 (40)	0.329	0.444	0.131
Non-TJA side									
Knee Extension (N)	157 (61)	156 (45)	162 (56)	153 (43)	152 (65)	159 (47)	0.852	0.881	0.223
Knee Flexion (N)	100 (39)	97 (35)	95 (34)	92 (32)	104 (43)	102 (37)	0.310	0.688	0.934
Hip Abduction (N)	87 (35)	94 (25)	86 (29)	94 (23)	89 (41)	94 (28)	0.833	0.193	0.804
Hip Extension (N)	106 (43)	94 (40)	91 (30)	87 (32)	116 (52)	99 (46)	0.105	0.019	0.155
Knee range of motion									
TJA side (°)	114 (17)	113 (15)	121 (15)	123 (12)	108 (16)	105 (11)	<0.001	0.871	0.099
Non-TJA side (°)	124 (13)	125 (11)	128 (9)	127 (10)	121 (14)	123 (13)	0.100	0.727	0.090

PRE-OP = pre-operative; POST-OP = 6-weeks post-operative; TJA = total joint arthroplasty.
* p <0.05 vs PRE-OP (Overall only). ^ = p <0.05 for hip arthroplasty vs knee arthroplasty.

Appendix Table 14: Pre-operative and 6-week post-operative subjective responses overall and in hip and knee arthroplasty participants. Data are presented as daily mean (SD) and analysed with a paired samples *t*-test or repeated-measures ANOVA test.

Variable	Overall (n=51)		Hip arthroplasty (n=23)		Knee arthroplasty (n=28)		Statistical significance		
	PRE-OP	POST-OP	PRE-OP	POST-OP	PRE-OP	POST-OP	Group	Time	Group×Time Interaction
WOMAC									
Overall score‡	59 (18)	21 (13)*	64 (17)	16 (11)	55 (18)	24 (14)^	0.936	<0.001	<0.001
Pain	12 (4)	4 (3)*	13 (4)	2 (2)	12 (4)	5 (3)^	0.303	<0.001	<0.001
Stiffness	5 (2)	2 (2)*	5 (2)	2 (1)	5 (2)	3 (1)^	0.970	<0.001	0.007
Physical function‡	43 (13)	15 (10)*	46 (12)	12 (8)	39 (13)	18 (10)^	0.568	<0.001	0.003
EQ-5D									
Health today‡	59 (23)	81 (15)*	54 (25)	83 (15)	64 (20)	79 (15)^	0.472	<0.001	0.038
Quality of life	15 (4)	8 (2)*	16 (3)	8 (2)	14 (3)	9 (2)^	0.1.29	<0.001	0.001
SF12									
Physical component score‡	27 (5)	39 (8)*	26 (5)	39 (8)	29 (5)	39 (9)	0.447	<0.001	0.121
Mental component score‡	45 (12)	53 (12)*	43 (11)	55 (9)	48 (12)	52 (14)	0.722	<0.001	0.022
DASI									
Score‡	17 (10)	26 (12)*	16 (10)	29 (13)	19 (9)	24 (11)	0.698	<0.001	0.013
Estimated peak $\dot{V}O_2$ ($mL \cdot min^{-1} \cdot kg^{-1}$)‡	17.1 (4.1)	21.8 (7.3)*	16.4 (4.3)	23.7 (9.0)	17.7 (3.9)	20.3 (5.3)	0.415	<0.001	0.030
	Day 7	**Week 6**	**Day 7**	**Week 6**	**Day 7**	**Week 6**			
Surgical recovery score	56 (15)	73 (14)*	53 (15)	74 (16)	58 (15)	73 (13)	0.565	<0.001	0.206

DASI = Duke Activity Status Index (0 worst–58 best); EQ-5D = European Quality of Life Five Dimension, EQ-5D Health today (0 worst–100 best), EQ-5D Quality of life (5 best–25 worst); OHS/OKS = Oxford hip score/Oxford knee score (0 worst–48 best); peak $\dot{V}O_2$ = peak oxygen consumption; PRE-OP = pre-operative; POST-OP = 6-weeks post-operative; SF-12 = 12-item short form survey; SRS = Surgical Recovery Score (higher score is best); WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index (0 best–94 worst).

* p <0.05 vs PRE-OP (Overall only). ^ = p <0.05 for hip arthroplasty vs knee arthroplasty. ‡ = log transformed for statistical analysis.

Delay to diagnosis in childhood bone and joint infection

Sarah Hunter, Elsie Brown, Haemish Crawford, Vanessa Selak, Cameron Grant

ABSTRACT

AIM: To determine the proportion of delayed diagnosis among cases of acute childhood bone and joint infection (BJI) and examine the impact of delayed diagnosis on illness trajectory.

METHODS: A retrospective review was undertaken of patients <16 years with acute haematogenous osteomyelitis (AHO) or septic arthritis (SA) treated in the Auckland region from 2018–2023. Electronic case information was used to identify any alternative diagnosis given prior to identification of BJI (delayed disease recognition). Cases were grouped into the following subtypes: multifocal sepsis or shock, "isolated" AHO or SA, or contiguous local infection such as pyomyositis and subperiosteal abscess. Primary outcomes included length of stay (LOS) and hospitalisation cost.

RESULTS: A total of 563 cases of childhood BJI were identified, of whom 512 had clearly documented presenting complaint. A high proportion received an alternative initial diagnosis (43%). Alternatively diagnosed children were more likely to have attended primary or urgent care (82% vs 38%, p=0.00001) and have a recent viral illness (46% vs 34%, p=0.008). Receiving alternative diagnosis was associated with greater delay to treatment (7.8 vs 4 days, p=<0.00001). Contiguous local infection was more likely in children with >1 week of symptoms (34% vs 17%, p=0.002). Contiguous infection required more surgical intervention, longer LOS and higher hospitalisation cost when compared to isolated AHO.

CONCLUSION: Delayed recognition of childhood AHO and SA is common and is associated with delayed treatment. Symptoms present for >1 week are associated with contiguous infection, which, compared with isolated AHO, requires more surgery with increased hospitalisation costs.

Prompt diagnosis and treatment for childhood bone and joint infection (BJI) is necessary to avoid long-term disease consequences.[1] Chronic infections often require multiple surgeries and can have devastating outcomes.[2] The exact time frame for irreversible damage is unknown, although *in vitro* studies report articular cartilage destruction within as little as 8 hours of bacterial inoculation.[3]

The clinical consequences of chronic and subacute disease are well characterised in the literature, but less attention has been given to the delayed management of acute disease.[3] Recent studies focus on diagnostic success in the emergency care setting.[4,5] Epidemiologic studies on BJI in the New Zealand population only report tertiary management.[6,7] There is a lack of information available about prehospital disease characteristics and prehospital medical care. These aspects of childhood BJI are important, as duration of symptoms can predict disease complexity and treatment success.[8–10]

Scoring systems to recognise complicated illness incorporate features of hospital presentation, but none include consideration of prehospital delays that contribute to more severe disease.[11,12] For instance, the A-score by Alhinai et al. defines "delayed source control" as 3 days between admission and surgery.[13] However, it is arguable that a child unwell in the community for a week has also experienced "delayed source control". The full impact of symptom duration is not measurable unless we account for prehospital disease course.

In addition to this, acute haematogenous osteomyelitis (AHO) and septic arthritis (SA) can be exceptionally difficult to diagnose.[14] For many children, the diagnostic process leads to apparently unavoidable treatment delay. Diagnostic algorithms developed in the 1990s and early 2000s have not been successfully validated in the current clinical environment.[9] Traditional teaching emphasises fever and limp as indicative of BJI, despite evolving disease aetiology that has altered clinical presentation.[15] In a recent case series only 15% of BJI infections secondary to *Kingella kingae* (*K. Kingae*) presented with fever.[16,17] Overlapping clinical presentation with transient synovitis leads to misdiagnosis rates of up to 60% in the emergency care setting.[14] These misconceptions surrounding disease presentation require review and refinement. If they are not revised, misdiagnosis will result in delayed treatment and management

for some children, with the consequences being more severe acute disease and increased rates of subsequent disability.

There is likely a temporal relationship between contiguous local infection and duration of symptoms. This relationship has not yet been defined in the literature. Greater utilisation of magnetic resonance imaging (MRI) over recent decades has increased clinical awareness of these contiguous infections.[18] We do not know the exact time frame in which "isolated" osteomyelitis disseminates locally into the surrounding tissues, creating focal collections such as subperiosteal abscess or pyomyositis, which may require more surgery. Children with contiguous local infection experience greater morbidity and incur more direct medical cost.[8] Determining the temporal nature of disease progression will help us understand the impact of delayed diagnosis.

In summary, the aim of this research is to determine the proportion of delayed diagnosis in childhood BJI and the impact of pre-diagnosis symptom duration on disease trajectory, with focus on length of stay (LOS) and hospitalisation cost.

Methods

This study is a retrospective analysis of all cases of suspected osteomyelitis and SA managed in multiple institutions across Auckland region from 2018–2023. Children from birth to age <16 years were included. Health and Disability Ethics Committee (HDEC) approval was obtained for this study together with institutional review board approval (reference: 19/NTA/46 & 18NTA219).

Comprehensive review of electronic clinical records was conducted. Data were collected on patient demographics, illness presentation and disease type. Household socio-economic deprivation was measured using the New Zealand Index of Deprivation (NZDep), based on patient address at time of treatment.[19] NZDep is an area-based surrogate measure of socio-economic hardship. AHO was defined based on radiographic investigation via MRI or computed tomography (CT) and/or positive intraoperative culture or bone biopsy. SA was defined based upon intraoperative culture results, culture results from aspirate or positive radiographic investigation in the setting of a positive blood culture. Cases of chronic BJI, post-viral or reactive arthritis, post-operative infection, cases associated with significant malignancy or patients with insufficient clinical data for analysis were excluded.

Children were considered to have multifocal sepsis if they had multiple concurrent infections; for example, AHO with pneumonia. Locally contiguous infection was defined as AHO with contiguous pyomyositis, subperiosteal abscess or adjacent joint SA.

Electronic hospital data contains primary and urgent care referrals, community laboratory results and community pharmacy dispensing records. These were reviewed to describe medical attention prior to hospital admission. In New Zealand, all medications dispensed to children from community pharmacies are listed electronically.[20]

Cases were assessed for any alternative diagnosis prior to identification of BJI. For example, if they presented to primary care or the emergency department (ED) and were discharged with a diagnosis of "transient synovitis" but subsequently confirmed to have AHO or SA. For the purposes of this study, diagnostic delay is defined as those children with symptoms for >3 days before treatment initiation.

LOS, number of surgeries and duration of combined intravenous and oral antibiotic therapy were recorded. The cost of hospitalisations was determined using a weighted discharge value (i.e., Weighted Inlier Equivalent Separations [WIES]) for all National Minimum Dataset events as calculated by the New Zealand Ministry of Health – Manatū Hauora.[21] This paediatric WIES cost-weight encompasses medical costs, ward stays, medications, laboratory investigations, operations and nursing and other ward staff. It is based on diagnostic-related groups (DRGs), with additional costs for interventions such as mechanical ventilation.

Results

An initial 994 encounters for BJI were identified by clinical codes for pyogenic arthritis and osteomyelitis. Of these, 563 met criteria for first presentation with acute BJI. The majority of exclusions were readmissions for the same condition or chronic disease (n=192), incorrect diagnosis (n=74), or infections secondary to operative complication or penetrating injury (n=56). Eligible cases were primarily AHO (39%); multifocal sepsis was less frequent (16%) (Table 1).

A presenting complaint was clearly outlined in 512 cases. A total of 218 children (43%) received an alternative diagnosis before identification of BJI (Table 2). A "classical" presentation with fever and pain at the site of infection was only seen in 45%. History of a recent viral illness was common (39%), as was history of trauma (23%).

An alternative diagnosis was less common in children of Māori or Pacific ethnic groups (Table 2). There was a lower average NZDep in children who received another diagnosis first (NZDep 6 vs 7, p=0.0003). Of note, within our cohort, the proportion of NZ European patients attending community medical centres was slightly higher (n=116, 66%) than Māori (n=40, 48%) or Pacific (n=69, 45%) (p=<0.01).

Children who were alternatively diagnosed were more likely to have presented to a primary or urgent care centre (82% vs 38%, p=<0.0001). They were equally likely to have reported fever and pain at infection site (47% vs 44%, p=0.62). However, they were more likely to have a recent viral illness (46% vs 34%, p=0.008).

Delay between symptom onset and treatment for BJI was greater if children received another diagnosis first (7.8 vs 4 days, p=<0.0001). Once hospitalised, children with initial misdiagnosis were equally likely to require surgery (50% vs 53%, p=0.56). Pathogen type in those with an alternative first diagnosis was more likely culture-negative or *K. kingae*–mediated BJI (58% vs 47%, p=0.004).

Considering diagnostic delay, Figure 1 shows the duration of symptoms prehospital for children with and without an alternative first diagnosis. Those without alternative first diagnoses had shorter duration of symptoms before treatment. Diagnostic delay of >3 days was more common in those with an alternative first diagnosis (75% vs 33%, p=<0.001).

The relationship between duration of symptoms and disease subtype is demonstrated in Figure 2. This Kaplan–Meier survival analysis compares days of symptoms before diagnosis across different subtypes of disease. It shows high proportion of SA and multifocal sepsis diagnosed after a short duration of symptoms. AHO and locally contiguous infections remain undiagnosed for longer.

Examining for statistical significance, children with multifocal sepsis typically presented within 48 hours, or "early" (Table 3). Conversely, locally contiguous infections were more often diagnosed "late" (>1 week of symptoms) (p=0.02). For the entire cohort, surgical intervention rates appear higher for those who present during the first 48 hours of symptoms (50–60%) or after 10 days of symptoms (64%) (p=0.09) (Figure 3).

Surgical intervention, LOS and cost differed by disease subtype. In Table 4, these outcomes for children with "isolated" AHO have been compared to locally contiguous infections. This is for two reasons. Firstly, it is already established that patients with SA and multifocal sepsis generally are more likely to require surgery, which raises hospitalisation cost.[22] Operative drainage of SA is deemed gold standard in the majority of cases.[23,24] Secondly, it is possible that earlier recognition and treatment of AHO could prevent progression to locally contiguous infection. Any additional costs and morbidity associated with contiguous infection may be avoidable.

Our results showed the surgical intervention rate for contiguous disease was higher than for isolated AHO (70% vs 14%, p=0.0001) (Table 4). Children with locally contiguous infection spend longer in hospital (8.42 vs 5.14 days, p=<0.0001). Hospitalisation cost appeared higher but did not reach statistical significance (NZ$14,102 vs NZ$8,587, p=0.26). A large standard deviation in cost averages is due to high additional costs associated with paediatric intensive care admission.

Discussion

Overall, in this study almost half of children with BJI were given an alternative diagnosis first. Alternatively diagnosed children were more likely to have attended primary or urgent care centres and report a recent viral illness. An alternative diagnosis delayed treatment by an average of 3.8 days.

Greater duration of symptoms is associated with locally contiguous forms of BJI. The consequences of this can be seen clinically as increased need for surgical intervention and longer hospital stay. Hospitalisation for locally contiguous infections was on average 3 days longer, with direct hospitalisation costs almost twice as high as for "isolated" AHO. Although this difference in hospitalisation cost did not reach statistical significance, it is likely clinically meaningful; longer hospitalisation also confers additional treatment expense to patients and families.

Delay in making a diagnosis may reflect outdated understanding of disease aetiology. Transient synovitis, unlike bacterial infection, is commonly associated with a pre-existing respiratory illness.[14,25] Traditional teaching emphasises the relationship between viral illnesses and transient synovitis, without acknowledging a potential relationship between viral infections and bacterial BJI.[26] A viral infection may be concomitant with *Staphylococcus aureus* (*S. aureus*) or *S. pyogenes* (*Streptococcus pyogenes*) mediated AHO or SA, whereby initial viral infection lowers immunity to

allow bacterial invasion.[26] This is supported by the lower rates of bacterial bone infections during the COVID-19 pandemic, as well as the high incidence of pre-existing viral infection in our cohort.[27] For clinicians, recognising the association between viral infections and childhood BJI may improve diagnostic rate. A recent virus may in fact be associated with BJI, rather than pointing to a diagnosis of transient synovitis.

This study suggests less clinical awareness for disease presentation associated with low virulence organisms and, in particular, culture-negative and *K. kingae*–mediated infections. Our results confirmed the proportion of culture-negative and *K. kingae*–mediated disease is higher in our region compared to previous years.[6,28] Emergence of *K. kingae* has been attributed to widespread vaccination for *Haemophilus influenzae* since the 1980s.[17] In addition, daycare use has increased, which is associated with spread of *K. kingae*.[29,30] These children often appear clinically well, increasing reliance on laboratory and radiographic information for diagnosis. Those with culture-negative or *K. kingae* infections were more likely to experience diagnostic delay in our cohort, perhaps in association with subtle disease presentation.

In contrast, *S. aureus*–mediated BJI is classically associated with febrile illness, has increased representation in lower socio-economic groups and is over-represented in Māori and Pacific children.[31] In our study delayed diagnosis was less common for these ethnic groups; although, it is important to note that Māori and Pacific children in our region experience reduced primary healthcare access.[32] Māori and Pacific children with BJI are also more likely to have severe illness and receive treatment in the paediatric intensive care unit (PICU).[33] To improve diagnostic success across a range of disease presentations, contemporary teaching for identification and management of BJI needs to reflect changes in clinical epidemiology.

The consequence of diagnostic delay in childhood BJI is delayed treatment initiation, specifically intravenous antibiotics and surgical source control. Importantly, diagnostic delay was equally likely among children who presented with fever and pain at site of infection in this cohort. Furthermore, children who received an alternative diagnosis first went on to receive equivalent rates of surgical intervention, suggesting delays were not limited to mild forms of disease. Although management guidelines vary between institutions, evidence supports urgent antibiotic administration in cases of musculoskeletal sepsis.[34] If osteomyelitis or SA are part of the disease differential, timely referral to a tertiary centre with radiographic and laboratory analysis may support earlier treatment initiation.

A limitation of this research is the quality of documentation. In electronic case records, a recent viral illness or trauma could be deemed irrelevant by clinicians and not documented. As a consequence, prevalence of these may be under-reported in this retrospective case series. However, we have sought to reduce the potential for this bias by careful analysis of primary care referral documentation and community laboratory and dispensing records where available. It is possible that this does not capture all consultation data, particularly where a prescription is not filled.

There are several findings from this study that can inform ongoing clinical practice. Firstly, models of teaching and diagnosis for BJI should reflect current clinical and molecular epidemiology of disease. This means that ongoing collection of data around presentation and causative pathogens is essential to inform surgeons and the wider medical community. Knowledge of how disease presents in the current environment is crucial to minimise delays to diagnosis and misdiagnosis.

Secondly, without accessible imaging and laboratory testing it is very difficult to diagnose childhood BJI. This is reflected in the greater delay to treatment and diagnosis after presentation to primary care. With any clinical suspicion of AHO or SA, referral to a facility with imaging and laboratory capability is essential. Healthcare professionals need to understand that the goal is to prevent contiguous infection, which develops over time and is associated with greater surgical intervention and morbidity. For patients and families, the accumulation of direct medical costs and productivity loss is greater with delayed diagnosis.

Conclusion

Childhood AHO and SA are frequently given an alternative diagnosis at presentation, leading to delayed treatment initiation. Delays of >1 week are associated with contiguous infection, which more frequently requires surgery, with prolonged morbidity and cost.

Figure 1: Prehospital duration of symptoms for children with and without alternative first diagnosis.

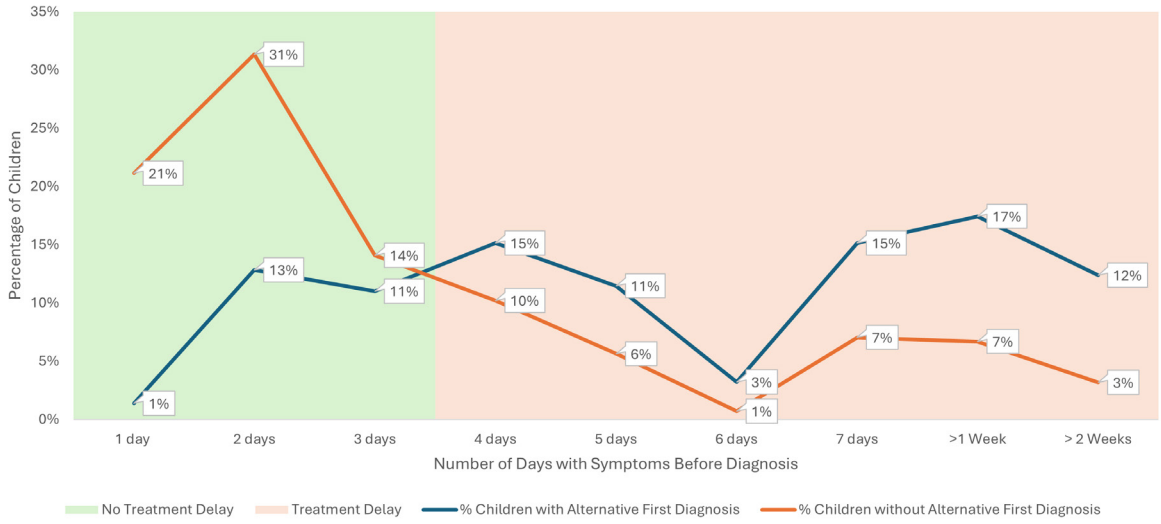
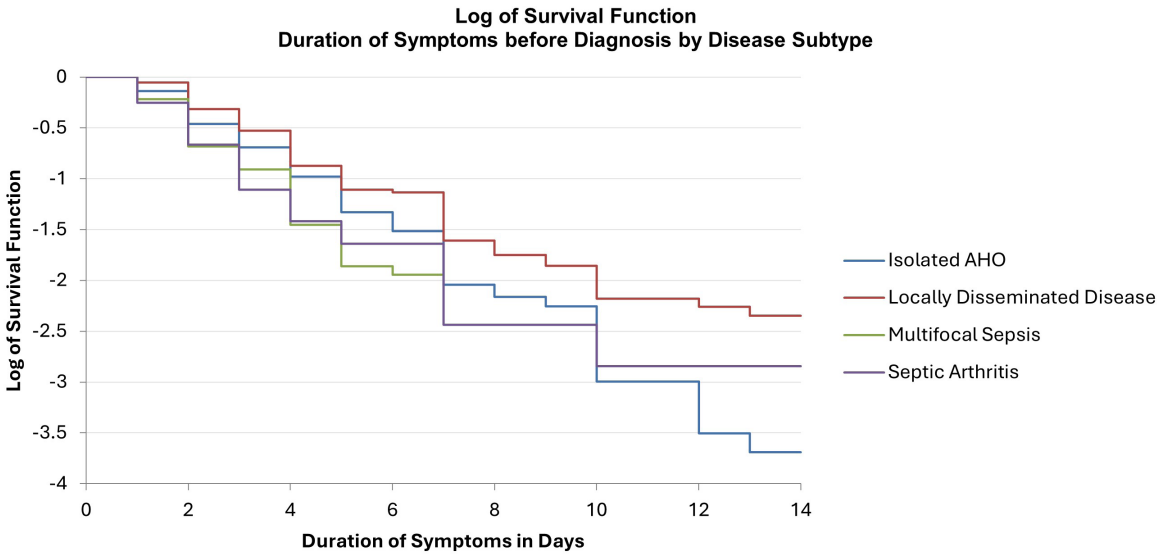


Figure 2: Kaplan–Meier analysis of time to diagnosis by disease subtype.



Mean duration of symptoms in days

Groups	Mean	Std dev	LCL (95%)	UCL (95%)
All	4.28	0.15	3.99	4.57
Status = isolated AHO	4.37	0.22	3.93	4.8
Status = locally disseminated disease	5.3	0.35	4.6	5.99
Status = multifocal sepsis	3.25	0.23	2.8	3.69
Status = septic arthritis	3.75	0.33	3.1	4.39

Figure legend: Log of survival function denotes the probability of a patient remaining in the community at each specified time point.
Std dev = standard deviation; LCL = lower confidence limit; UCL = upper confidence limit; AHO = acute haematogenous osteomyelitis.

Figure 3: Proportion of children receiving surgical intervention by duration of symptoms prior to diagnosis.

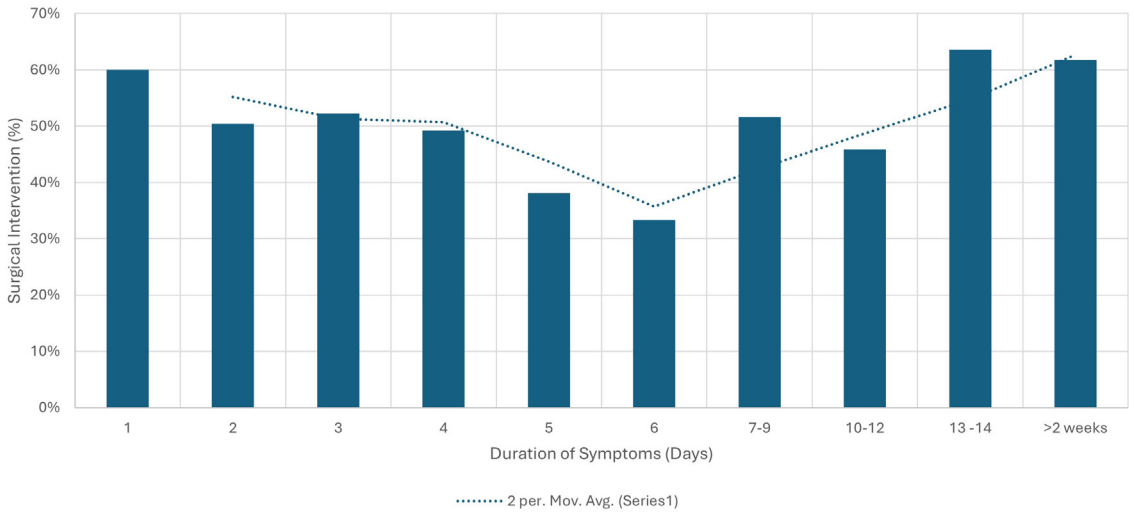


Table 1: Demographic information.

Auckland cases of childhood BJI 2018–2023 (n=563)		
Basic demographics		
	N	**%/Standard deviation**
Male	351	62%
Female	212	38%
Mean age (interquartile range)	6 (9.7)	4.6
NZ European	175	32%
Pacific	152	28%
Māori	106	19%
Indian	32	6%
Asian	57	10%
Other	39	5%
Average NZDep (interquartile range)	7 (5)	3
Disease type		
Isolated AHO	222	39%
AHO with contiguous local disease	139	25%
Multifocal sepsis	90	16%
SA	112	20%

Table 1 (continued): Demographic information.

Severe or complicated disease course (N=328)		
Intensive care admission	39	7%
>1 surgery to control infection	85	15%
Multifocal sepsis	90	16%
Chronic infection	30	5%
Readmission for treatment failure or further surgery	106	19%
Causative pathogen (from 503 cases with microbiological samples)		
Staphylococcus aureus	236	42%
Streptococcus pyogenes	30	5%
Kingella kingae	23	4%
Culture-negative	214	38%

BJI = bone and joint infection; NZ = New Zealand; NZDep = New Zealand Index of Deprivation; AHO = acute haematogenous osteomyelitis; SA = septic arthritis.

Table 2: Characteristics of children with and without alternative first diagnosis.

	Full cohort		**Children who receive alternative diagnosis first**		**Children who do not receive alternative diagnosis first**		**P value**
	N	%/SD[IQR]	N	%/SD[IQR]	N	%/SD[IQR]	
Number of cases	512		218	43%	294	57%	
Demographics							
Median age (IQR)	5	4.5 [9]	4	4.4 [8]	6	4.4 [9]	0.0003
Māori	94	18%	40	43%	54	57%	0.04
Pacific	130	25%	42	28%	88	68%	<0.0001
NZ European	169	33%	83	47%	86	54%	0.04
Other ethnic groups	114	22%	52	46%	62	54%	0.2
Median NZDep (IQR)	7	3 [5]	6	3 [6]	7	3[6]	0.0003
Illness presentation							
Attended primary care	290	57%	178	82%	112	38%	<0.0001
Recent viral illness	202	39%	101	46%	101	34%	0.008
History of trauma	116	23%	55	25%	61	21%	0.23
Febrile	336	66%	147	67%	189	64%	0.51

Table 2 (continued): Characteristics of children with and without alternative first diagnosis.

Fever + pain at site of infection	232	45%	102	47%	130	44%	0.62
Duration of symptoms in days	4	5.9 [5]	7.8	6.7 [6]	4	4.2 [2]	<0.0001
Disease characteristics							
AHO	392	77%	172	83%	220	75%	0.81
SA	120	23%	45	21%	74	25%	0.81
Multifocal sepsis	78	15%	22	10%	56	19%	0.005
AHO with locally disseminated infection	139	25%	80	37%	59	20%	0.00002
Spine	23	4%	14	6%	9	3%	0.51
Pelvis	53	10%	25	11%	28	10%	0.47
Surgical intervention	267	52%	110	50%	157	53%	0.56
>1 surgery	111	22%	42	19%	69	23%	0.36
Culture positive *S. aureus* or *S. pyogenes*	229	45%	84	39%	145	49%	0.005
Culture-negative or *K. kingae*	263	51%	126	58%	137	47%	0.004

SD = standard deviation; IQR = interquartile range; NZ = New Zealand; NZDep = New Zealand Index of Deprivation; AHO = acute haematogenous osteomyelitis; SA = septic arthritis; *S. aureus* = *Staphylococcus aureus*; *S. pyogenes* = *Streptococcus pyogenes*; *K. kingae* = *Kingella kingae*.
*512 cases with prehospital information available were included in this analysis out of 563 children with BJI.

Table 3: Disease subtypes by duration of symptoms prior to diagnosis.

Duration of symptoms	**Multifocal sepsis**		**Septic arthritis**		**Acute hae-matogenous osteomyelitis**		**Contiguous disease**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Symptoms for </= 48 hours (“early” presentation)	38	20%	50	26%	74	38%	34	17%
Symptoms >48hrs <1 week	28	15%	33	18%	82	44%	50	25%
>1 week symptoms (“late” presentation)	14	10%	22	15%	63	43%	85	34%
P value: early vs late	<0.00001		0.03		0.34		0.002	

Table 4: Rates of surgical intervention, length of stay and hospitalisation cost by subtype of disease.

	Surgical intervention	%	Median LOS (days)	Mean ± SD	IQR	Median hospitalisation cost (NZ$)	Mean ± SD	IQR
Isolated SA	100	89%	5.93	6.95±8.52	4.56	14,230.50	17,535.56±20,278.26	5,940.1
Isolated AHO	32	14%	5.14	6±4.8	4.2	8,587.9	27,402.16±229,346.62	4,217.2
Multifocal sepsis	71	89%	19.64	27±22.1	22.5	27,505.85	190,350 ± 566,998	71,502
Contiguous disease	97	70%	8.42	10.3±7.21	6.2	14,102.4	48,452.8±229,365.83	10,488.28
P value (isolated AHO vs contiguous disease)	0.0001*		<0.0001†			0.26†		

LOS = length of stay; SD = standard deviation; IQR = interquartile range; NZ = New Zealand; SA = septic arthritis; AHO = acute haematogenous osteomyelitis.
* Chi-squared test.
† Mann–Whitney U test.

COMPETING INTERESTS
This manuscript received funding from the Health and Research Council of New Zealand, Starship Foundation and the Wishbone Orthopaedic Trust.
VS has received funding from the Health Research Council of New Zealand, Healthier Lives – He Oranga Hauora – National Science Challenge and the Heart Foundation of New Zealand via a programme grant to The University of Auckland. VS is a Board Member of EQUIT3 (vaping cessation trial) DSMB, Cess@Tion (smoking cessation trial) DSMB, and Auckland Medical Research Foundation. VS is Deputy Chair for the Auckland Medical Research Foundation Medical Committee.

AUTHOR INFORMATION
Dr Sarah Hunter, MBChB, MHSc: Faculty of Medical and Health Sciences, The University of Auckland; Paediatric Orthopaedic Department, Starship Children's Hospital.
Dr Elsie Brown, MBChB: Paediatric Orthopaedic Department, Starship Children's Hospital.
Dr Haemish Crawford, MBChB, FRACS: Faculty of Medical and Health Sciences, The University of Auckland; Paediatric Orthopaedic Department, Starship Children's Hospital.
Dr Vanessa Selak, MBChB, MPH, PhD: Faculty of Medical and Health Sciences, The University of Auckland; School of Population Health, The University of Auckland.
Professor Cameron Grant, MBChB, FRACP, PhD: Faculty of Medical and Health Sciences, The University of Auckland; Department of Child and Youth Health, Starship Children's Hospital.

CORRESPONDING AUTHOR
Sarah Hunter: Starship Hospital, Grafton Road, New Zealand. E: shun472@aucklanduni.ac.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/delay-to-diagnosis-in-childhood-bone-and-joint-infection

REFERENCES
1. Fabry G, Meire E. Septic Arthritis of the Hip in Children: poor results after late and inadequate treatment. J Pediatr Orthop. 1983;3(4):461-6. doi: 10.1097/01241398-198309000-00008.
2. Matzkin EG, Dabbs DN, Fillman RR, et al. Chronic osteomyelitis in children: Shriners Hospital Honolulu experience. J Pediatr Orthop B. 2005;14(5):362-6. doi: 10.1097/01202412-200509000-00009.
3. Erkilinc M, Gilmore A, Weber M, Mistovich RJ. Current Concepts in Pediatric Septic Arthritis. J Am Acad Orthop Surg. 2021;29(5):196-206. doi: 10.5435/JAAOS-D-20-00835.
4. Otani Y, Aizawa Y, Hataya H, Horikoshi Y. Diagnostic errors in pediatric bacterial osteomyelitis. Pediatri Int. 2019;61(10):988-93. doi: 10.1111/ped.13979.
5. Vardiabasis NV, Schlechter JA. Definitive Diagnosis of Children Presenting to A Pediatric Emergency Department With Fever and Extremity Pain. J Emerg Med. 2017;53(3):306-312. doi: 10.1016/j.jemermed.2017.05.030.
6. Hunter S, Baker JF. Ten-year retrospective review of paediatric septic arthritis in a New Zealand centre. Int Orthop. 2021;45(1):147-54. doi: 10.1007/s00264-020-04611-z.
7. Hunter S, Crawford H, Baker JF. Ten-year Review of Acute Pediatric Hematogenous Osteomyelitis at a New Zealand Tertiary Referral Center. J Pediatr Orthop. 2023;43(5):e396-e401. doi: 10.1097/BPO.0000000000002385.
8. Hunter S, Baker JF. Early CRP Trends in Childhood Osteomyelitis Predict Complicated Disease. J Pediatr Orthop. 2023;43(1):e74-9. doi: 10.1097/BPO.0000000000002275.
9. Hunter S, Kioa G, Baker JF. Predictive Algorithms in the Diagnosis and Management of Pediatric Hip and Periarticular Infection. J Bone Joint Surg Am. 2022;104(7):649-58. doi: 10.2106/JBJS.21.01040.
10. Hunter S, Kennedy J, Baker JF. External validation of an algorithm to predict adjacent musculoskeletal infection in pediatric patients with septic arthritis. J Pediatr Orthop. 2020;40(10):e999-e1004. doi: 10.1097/BPO.0000000000001618.
11. Tuason DA, Gheen T, Sun D, et al. Clinical and laboratory parameters associated with multiple surgeries in children with acute hematogenous osteomyelitis. J Pediatr Orthop. 2014;34(5):565-70. doi: 10.1097/BPO.0000000000000136.
12. Athey AG, Mignemi ME, Gheen WT, et al. Validation and Modification of a Severity of Illness Score for Children with Acute Hematogenous Osteomyelitis. J Pediatr Orthop. 2019;39(2):90-7. doi: 10.1097/BPO.0000000000000879.
13. Alhinai Z, Elahi M, Park S, et al. Prediction of Adverse Outcomes in Pediatric Acute Hematogenous Osteomyelitis. Clin Infect Dis. 2020;71(9):e454-64. doi: 10.1093/cid/ciaa211. Erratum in: Clin Infect Dis. 2020;71(7):1805. doi: 10.1093/cid/ciaa862. Erratum in: Clin Infect Dis. 2022;74(3):565. doi: 10.1093/cid/ciaa1010.
14. Lipshaw MJ, Walsh PS. Transient synovitis of the hip: Current practice and risk of misdiagnosis. Am J Emerg Med. 2022;61:1-6. doi: 10.1016/j.ajem.2022.08.022.

15. Nguyen A, Kan JH, Bisset G, Rosenfeld S. Kocher Criteria Revisited in the Era of MRI: How Often Does the Kocher Criteria Identify Underlying Osteomyelitis? J Pediatr Orthop. 2017;37(2):e114-19. doi: 10.1097/BPO.0000000000000602.
16. Basmaci R, Bidet P, Bonacorsi S. *Kingella kingae* and viral infections. Microorganisms. 2022;10(2):230. doi: 10.3390/microorganisms10020230.
17. Samara E, Spyropoulou V, Tabard-Fougère A, et al. *Kingella kingae* and osteoarticular infections. Pediatrics. 2019;144(6):e20191509. doi: 10.1542/peds.2019-1509.
18. Griswold BG, Sheppard E, Pitts C, et al. The Introduction of a Preoperative MRI Protocol Significantly Reduces Unplanned Return to the Operating Room in the Treatment of Pediatric Osteoarticular Infections. J Pediatr Orthop. 2020;40(2):97-102. doi: 10.1097/BPO.0000000000001113.
19. Atkinson J, Salmond C, Crampton P. NZDep2018 Index of Deprivation. Wellington (NZ): Department of Public Health, University of Otago; 2020 [cited 2024 Mar 1]. Available from: https://www.otago.ac.nz/__data/assets/pdf_file/0020/326711/nzdep2018-index-of-deprivation-research-report-final-dec-2020-823833.pdf
20. Health New Zealand – Te Whatu Ora. Pharmaceutical Collection. National Collections Data and Statistics [Internet]. Wellington (NZ): Health New Zealand – Te Whatu Ora; 2024 [cited 2024 Jun 5]. Available from: https://www.tewhatuora.govt.nz/for-health-professionals/data-and-statistics/nz-health-statistics/national-collections-and-surveys/collections/pharmaceutical-collection
21. Health New Zealand – Te Whatu Ora. Data references - Cost Weights Definition 2022 [Internet]. Wellington (NZ): Health New Zealand – Te Whatu Ora; 2025 [cited 2024 Mar 1]. Available from: https://www.tewhatuora.govt.nz/for-health-professionals/data-and-statistics/nz-health-statistics/data-references
22. Racimo AR, Talathi NS, Zelenski NA, et al. How Much Will My Child's Operation Cost? Availability of Consumer Prices from US Hospitals for a Common Pediatric Orthopaedic Surgical Procedure. J Pediatr Orthop. 2018;38(7):e411-16. doi: 10.1097/BPO.0000000000001194.
23. Mathews CJ, Kingsley G, Field M, et al. Management of septic arthritis: A systematic review. Ann Rheum Dis. 2007;66(4):440-5. doi: 10.1136/ard.2006.058909.
24. Livingston KS, Kalish LA, Bae DS, et al. Wash, Rinse, Repeat: Which Patients Undergo Serial Joint Irrigation in Pediatric Septic Hip Arthritis? J Pediatr Orthop. 2019;39(7):e494-e499. doi: 10.1097/BPO.0000000000001323.
25. Caird MS, Flynn JM, Leung YL, et al. Factors distinguishing septic arthritis from transient synovitis of the hip in children: A prospective study. J Bone Joint Surg Am. 2006;88(6):1251-7. doi: 10.2106/JBJS.E.00216.
26. Principi N, Autore G, Ramundo G, Esposito S. Epidemiology of Respiratory Infections during the COVID-19 Pandemic. Viruses. 2023;15(5):1160. doi: 10.3390/v15051160.
27. Mason B, Stott S, Beamish R. Reduced presentations with fractures or orthopaedic infections to a major children's hospital during a national COVID-19 elimination strategy. ANZ J Surg. 2022;92(1-2):206-11. doi: 10.1111/ans.17354.
28. Street M, Puna R, Huang M, Crawford H. Pediatric acute hematogenous osteomyelitis. J Pediatr Orthop. 2015;35(6):634-9. doi: 10.1097/BPO.0000000000000332.
29. Yagupsky P. *Kingella kingae*: Carriage, Transmission, and Disease. Clin Microbiol Rev. 2015;28(1):54-79. doi: 10.1128/CMR.00028-14.
30. Yagupsky P. Outbreaks of *Kingella kingae* infections in daycare facilities. Emerg Infect Dis. 2014;20(5):746-53. doi: 10.3201/eid2005.131633.
31. Hunter S, Chan H, Baker JF. Global epidemiology of childhood bone and joint infection: a systematic review. Infection. 2022;50(2):329-41. doi: 10.1007/s15010-021-01741-3.
32. Jeffreys M, Ellison-Loschmann L, Irurzun-Lopez M, et al. Financial barriers to primary health care in Aotearoa New Zealand. Fam Pract. 2024;41(6):995-1001. doi: 10.1093/fampra/cmad096.
33. Hunter S, Brown E, Crawford H, et al. Musculoskeletal Sepsis in the Pediatric Intensive Care Unit. Pediatr Infect Dis J. 2024;44(3):189-194. doi: 10.1097/INF.0000000000004604.
34. Benvenuti MA, An TJ, Mignemi ME, et al. Effects of Antibiotic Timing on Culture Results and Clinical Outcomes in Pediatric Musculoskeletal Infection. J Pediatr Orthop. 2019;39(3):158-62. doi: 10.1097/BPO.0000000000000884.

Anticoagulation management and poor clinical outcomes in tamariki and rangatahi with rheumatic heart disease following mechanical valve replacement surgery in Counties Manukau

Prathyusha Tangirala, Bridget Farrant, Rachel Webb

ABSTRACT

AIM: Rheumatic heart disease (RHD) causes significant cardiovascular morbidity and mortality, with persisting inequitably high rates in Māori and Pacific tamariki and rangatahi. Mechanical valve replacement surgery is required for people with severe RHD and requires lifetime anticoagulation. Contemporary data regarding anticoagulation management and outcomes for tamariki and rangatahi following mechanical valve replacement surgery for RHD are lacking. We aimed to describe patient characteristics, anticoagulation management practices and complications in a cohort of tamariki and rangatahi ≤25 years of age.

METHODS: A retrospective observational study of patients aged ≤25 years with RHD and mechanical valves, living in Counties Manukau, South Auckland, 2016–2021, was conducted.

RESULTS: A total of 53 patients were identified. The median age at time of first mechanical valve surgery was 15 years (range 4–23 years). Nineteen percent of the cohort were Māori and 81% were Pacific peoples. The median duration of anticoagulation was 4 years (range 0.5–18 years). The most common method of monitoring was via the community laboratory service and general practitioner. There were 38 individuals who had ≥1 anticoagulation-related hospitalisation. There were 80 anticoagulation-related hospitalisations: 52% were due to a subtherapeutic international normalised ratio (INR) without clinical complication; 15% had a supratherapeutic INR without clinical complication; 14% haemorrhage; 9% stroke; 6% other thromboembolic events; and 4% prosthetic valve thrombosis. Five deaths occurred between 2016 and 2021.

CONCLUSION: The majority of the cohort had serious anticoagulation-related hospitalisation events, and 10% died. Urgent efforts are required to improve services for anticoagulation monitoring and management and clinical outcomes in young adults following mechanical valve surgery for RHD.

Rheumatic heart disease (RHD) is the chronic sequela of acute rheumatic fever (ARF), a multisystem inflammatory condition that develops in a susceptible host due to Group A *Streptococcus* pharyngeal infection.[1] In 2019, the global burden of RHD was estimated to be 40.5 million cases, mostly in low- and middle-income countries.[2,3] Despite Aotearoa New Zealand being a developed country, RHD is a significant cause of cardiovascular morbidity and premature mortality among Māori and Pacific peoples.[4,5] Serious consequences of RHD include infective endocarditis, atrial fibrillation, thromboembolism, stroke and premature death.[6]

In Aotearoa, RHD prevalence is estimated to be around 2% among Pacific tamariki and rangatahi, and 1% among Māori.[7–10] The New Zealand Rheumatic Heart Disease Registry estimates there are around 3,600 individuals with moderate or severe RHD.[5] ARF and RHD hospitalisation costs in Aotearoa peak between the ages of 5 and 19 years.[11] The most recent analysis of direct hospitalisation costs for ARF/RHD in young people was published in 2012 and found an average annual hospitalisation cost of NZ$12 million, with heart valve surgery accounting for 71% of hospitalisation costs for those between 5 and 14 years of age.[12] Around 160 deaths in Aotearoa annually can be attributed to RHD.[12]

A proportion of tamariki and rangatahi with severe RHD require heart valve surgery. In those <16 years, surgery is performed at Starship

Hospital, whereas older patients usually undergo surgery in adult cardiosurgical centres. Where possible, valve repair procedures are favoured over mechanical valve replacement in order to avoid the need for lifelong anticoagulation.[13,14] Valve repair surgery in Aotearoa has excellent outcomes with 90% survival and 75% freedom from reoperation at 10 years.[14] However, in some situations mechanical valve replacement surgery is required due to the unrepairable state of the native valve.

Mechanical valve surgery necessitates lifelong oral anticoagulation therapy with the vitamin K antagonist warfarin to prevent thromboembolic events.[15] Tamariki and rangatahi face significant challenges with long-term warfarin therapy due to the complexity of international normalised ratio (INR) monitoring, the need for frequent warfarin dose adjustments and the effect of diet, medication interactions, illness and missed doses on INR levels.[16] Furthermore, for women in childbearing years, warfarin is teratogenic and can lead to bleeding complications during pregnancy, along with increased risk of major thromboembolic events.[17,18] The challenges are particularly profound for rangatahi during the transition from paediatric to adult health services. In the Auckland Region, patients are typically transitioned from the Starship service to primary care providers around the age of 16 years. Coagu-Chek® machines and test strips are not routinely available or funded for adult patients in Aotearoa. Currently there are two main methods for primary care INR management: firstly, via general practitioner (GP) and community laboratory, and secondly, via the community pharmacy anticoagulation monitoring service (CPAMS).[19] Further challenges in the journey towards independence and self-management can include frequently changing addresses and busy work and study schedules. There are little published data about anticoagulation-related outcomes in young people with RHD. To date, there have been no Aotearoa studies on this subject.

Aims

The primary objective of this study was to describe the patient characteristics of all tamariki and rangatahi with RHD on anticoagulation after mechanical valve surgery in Counties Manukau and to review anticoagulation management and complications. Secondary objectives included describing ARF recurrences in this population, and describing deaths in those ≤25 years following RHD mechanical valve surgery occurring between January 2016 and January 2021.

Methods

This study was undertaken in Counties Manukau, the district with the highest prevalence of RHD in Aotearoa.

We undertook a retrospective review of all tamariki and rangatahi aged ≤25 years on anticoagulation after metal mechanical valve replacement for RHD, domiciled in Counties Manukau between 1 January 2016 and 1 January 2021. Patients with RHD and on anticoagulation for other reasons, such as heart failure or atrial fibrillation, and patients with valve repairs were excluded. Those with only bioprosthetic valves were excluded. Data census was conducted on 1 January 2021, or at the time of last documentation in clinical records for those who had died.

The following variables were investigated: age as of 31 January 2021, gender, ethnicity and age at RHD diagnosis. Clinical variables included mechanical valve surgery details, anticoagulation monitoring and hospitalisations due to subtherapeutic INR without clinical complication, supratherapeutic INR without clinical complication, haemorrhage, stroke, other thromboembolic events and mechanical valve thrombosis. Cardiology and rheumatic fever clinic attendance, medication dispensing records, transition process, age of transfer to adult services and clinical details of deaths were also examined. Hospitalisation events were defined as events where the patient was admitted to an inpatient service and stayed overnight. Emergency department visits were excluded.

Multiple data sources were interrogated to optimise case ascertainment, including the New Zealand Rheumatic Heart Disease Registry Project,[5] Counties Manukau rheumatic fever secondary prophylaxis database, Starship Hospital anticoagulation database and clinician records. Electronic records and primary care prescribing records were evaluated.

Continuous variables are presented as median and range. Categorical data are presented as frequencies and percentages. Statistical analyses were performed using Microsoft Excel.

Ethics approval was granted by the Auckland Health Research Ethics Committee (AH22290). Locality approval was obtained from the Counties Manukau Health Research Office.

Results

There were 53 patients in Counties Manukau with a mechanical metal valve on anticoagulation secondary to RHD between 1 January 2016 and 1 January 2021. Review of clinical records identified that five of these individuals died sometime between 1 January 2016 and 1 January 2021.

Patient demographics are presented in Table 1. Of the 48 patients alive in 2021, the majority were aged ≥16 years (42/48, 88%). There was a male predominance (58%). The highest represented ethnicities were Samoan (39%), Cook Island Māori (21%), Māori (19%) and Tongan (17%).

The median age at time of first mechanical valve surgery was 15 years (range 4–23).

A description of the valves in surgical management of affected valves is presented in Table 2. The aortic valve was the most commonly operated on valve (35%), and the sole mitral valve was the least common (10%). The majority of the cohort had one or two valves operated on (44% and 41%, respectively). On-X was the most frequently chosen type of mechanical valve (62%).

There were 43 patients under the care of adult services and 10 patients under the care of paediatric services. The median age of transition from paediatric to adult services was 16 years (range 15–19). Of the 43 patients under adult services, 33 patients (62%) had been transferred to adult services via formal referral from paediatrics, and seven patients (13%) underwent surgery and commenced warfarin under adult services. Five patients in the cohort had become lost to follow-up during or after transition and were re-referred to adult cardiology services either following an acute hospital presentation or by primary care.

The median duration of anticoagulation was 4 years (range 0.5–18 years). The most common mode of anticoagulation monitoring in the past year was via the community laboratory service and GP (28, 53%), followed by CPAMS point-of-care testing (11, 21%), self-testing with CoaguChek® machines and paediatric cardiac nurse specialist input (8, 14%)

Table 1: Cohort demographics (n=53).

Variable	n	%
Sex		
Male	31	58
Female	22	42
Ethnicity		
Samoan	21	39
NZ Māori	10	19
Cook Island Māori	11	21
Tongan	9	17
Niuean	1	2
Other Pacific	1	2
Age group (as of January 2021)*		
6–10	1	2
11–15	5	10
16–20	20	42
21–25	22	46

*Deaths (n=5) excluded from age range calculations.

Table 2: Summary of surgical management of the cohort.

Total RHD surgeries per person (includes repairs and replacements)	**n**	**%**
1	28	53
2	21	40
3	4	7
Total valves operated on including repairs and replacements		
1	24	44
2	21	41
3	8	15
Valve operated on		
AV	18	35
MV and AV	14	25
MV, AV and TV	8	16
MV and TV	7	14
MV	6	10
Mechanical valve type		
On-X	33	62
St. Jude	13	25
ATS	7	13

RHD = rheumatic heart disease; AV = aortic valve; MV = mitral valve; TV = tricuspid valve.

Table 3: Anticoagulation-related hospitalisation events.

Type of anticoagulation-related complication leading to hospitalisation event	**Hospitalisation events, N=80 (%)**
Subtherapeutic INR without clinical complication	42 (52)
Supratherapeutic INR without clinical complication	12 (15)
Haemorrhage	11 (14)
Stroke	7 (9)
Other thromboembolic events	5 (6)
Prosthetic valve thrombosis	3 (4)

INR = international normalised ratio.

and monitoring at Middlemore Hospital (5, 10%). One patient had point-of-care testing at a GP practice. Of the five patients who monitored INR at the hospital, two were on renal dialysis, two had prolonged hospitalisations and one infrequently monitored INR intermittently during hospital admissions.

During the 5-year study period, there were 80 anticoagulation-related hospitalisations, occurring in 38 individuals (72% of the cohort).

Causes for hospitalisation included subtherapeutic INR without clinical complication (42,52%), INR without clinical complication (12, 15%), haemorrhage (11, 14%), stroke (7, 9%), other thromboembolic events (5, 6%) and prosthetic valve thrombosis (3, 4%). One pregnant patient experienced foetal demise. Among the 53 individuals, 38 (72%) had ≥1 hospitalisation, and 20 individuals had two or more hospitalisations. Thirty-three percent of hospitalisations were due to significant complications such as haemorrhage, stroke, other thromboembolic events and prosthetic valve thrombosis. Of the 26 hospitalisations for a major clinical complication, 15 individuals (58%) had a prior admission with supratherapeutic or subtherapeutic INR.

Eleven hospitalisations secondary to haemorrhage included three patients with severe menorrhagia, four patients with epistaxis and one patient each with adnexal bleeding, per rectal bleeding and knee haemarthrosis. Other thromboembolic events were limb ischaemia, splenic infarction, renal pole infarction, cardioembolic myocardial infarction and multiple small emboli. All three cases of prosthetic valve thrombosis required prolonged hospital admissions. All the subtherapeutic and supratherapeutic INR admissions without clinical complications required bridging anticoagulation or administering vitamin K with observation.

Treatment adherence was challenging to determine from the available records. Medical dispensing records were used as a surrogate marker. Around half the cohort (21/45 individuals where dispensing records were available) had medication dispensed <5 times in the preceding 12 months.

The majority of patients (37/53 [70%]) presented with ARF, and the median age at ARF diagnosis was 10 years (range 3–21 years). The remaining 16 patients presented with established RHD. Six patients (11%) had a documented ARF recurrence during the 5-year study period. All six cases with recurrent ARF had a history of benzathine penicillin non-adherence. Three ARF recurrences occurred post-mechanical valve replacement.

Five deaths occurred between January 2016 and January 2021: two in the community and three in hospital. The age at death for both community cases was 17 years. The cause of death was reported as “direct complications of RHD”, and warfarin non-adherence was mentioned in clinic letters. One had a sudden collapse, severe aortic regurgitation and long QT interval; the other had an enlarged heart. Of note, post-mortem computed tomography scans of both brains did not reveal intracranial bleeding. Three deaths occurred in hospital. All three patients who died in hospital had prolonged intensive care unit admissions, difficult anticoagulation control and poor cardiac function.

Discussion

To our knowledge, this is the first published report to describe anticoagulation management practices and anticoagulation-related morbidity and mortality in tamariki and rangatahi following mechanical valve surgery for RHD in Aotearoa. Our study highlights the high rates of serious thromboembolic complications such as stroke, bleeding and valve thrombosis and premature deaths in this cohort.

Among the 53 tamariki and rangatahi in this study, almost three-quarters had at least one hospitalisation. Over half of those who experienced significant complications had a prior hospitalisation for subtherapeutic or supratherapeutic INR, indicating that current services are failing to optimise management and avert future complications. These findings are likely to be a conservative assessment of overall morbidity, as a small number of complications may have been managed in the emergency department without hospitalisation, particularly among older patients in the cohort. There were five RHD-related deaths in rangatahi between 2016 and 2021 in Counties Manukau. These deaths may well have been prevented if accessible, culturally responsive clinical services and enhanced patient support were available.

We found that RHD patients used a variety of anticoagulation services. The most common method of anticoagulation monitoring was via the GP and community laboratory service, followed by CPAMS. While CPAMS has been previously found to provide a high standard of anticoagulation management in the general

population on warfarin,[19] only 21% of this RHD cohort utilised CPAMS. This may be due stringent referral criteria, which require the person to have three in-range INR recordings from the GP, prior to referral to the CPAMS service. A small number of younger patients were under the care of the national paediatric and congenital cardiac service (PCCS) at Starship Children's Hospital. Under PCCS, patients are provided with a free CoaguChek® machine and test strips. They are closely supported by nurse specialists to conduct home-based INR testing, with dosing advice provided by the nurse specialist team. Previous systematic reviews demonstrate that self-monitoring in this way is safe and results in reduced thromboembolic events and more time in the therapeutic range of INR compared to other monitoring modes.[20,21]

It is notable that none of the available services are integrated with the Counties Manukau rheumatic fever benzathine penicillin secondary prophylaxis service. In future, flexible referral pathways and widened access to point-of-care INR testing devices for GP clinics and community nurses who deliver benzathine penicillin may improve anticoagulation management options for rangatahi.

It is well recognised that patient experience of ARF and RHD clinical services in many parts of Aotearoa is poor, with service access and both interpersonal and systemic racism contributing to their poor experience.[22] Future anticoagulation services need to be designed taking rangatahi perspectives into consideration, and there is a need for qualitative research and quality of life measures to inform service design. Further work is needed to determine what tamariki and rangatahi would find useful to help support them with anticoagulation management. Culturally responsive frameworks, such as those developed in the recently completed Ministry of Health co-design initiative, should be used when developing anticoagulation services for this group of patients.[23] Tamariki and rangatahi with RHD have numerous contacts with healthcare services and there are opportunities for future models to better integrate with other aspects of primary healthcare and ARF/RHD healthcare.[24]

The high rate of complications in this cohort also highlights the importance of careful preoperative planning for RHD valve surgery. Wherever technically possible, valve repair techniques should be used. This is particularly important for young women of childbearing potential and may also be important for young people who play high-level contact sports. Ideally, a multidisciplinary team experience in RHD management, and where possible patient preference, should inform surgical planning.[13,14]

This was a single-centre study, and as such findings may not be widely generalisable beyond South Auckland. However, poor outcomes in this cohort highlight the need to understand anticoagulation service models and outcomes around the country for other RHD patients following valve replacement surgery. A national stocktake of ARF/RHF tertiary services would identify regional variation in services, and it is likely that patients living in rural areas and under-served urban centres experience the greatest barriers to accessing anticoagulation services.

COMPETING INTERESTS
None to declare.

ACKNOWLEDGEMENTS
The authors would like to thank Mrs Belinda Paku from the Counties Manukau district nursing service and Ms Elizabeth Tilton, nurse researcher at Starship Hospital, for their assistance with case ascertainment.

AUTHOR INFORMATION
Prathyusha Tangirala: Paediatrician, Health New Zealand – Te Whatu Ora Te Tai Tokerau, Aotearoa New Zealand.
Bridget Farrant: Adolescent Physician, Centre for Youth Health, Health New Zealand – Te Whatu Ora Counties Manukau, Aotearoa New Zealand.
Rachel Webb: Paediatric Infectious Diseases Specialist, The University of Auckland, Department of Paediatrics, Aotearoa New Zealand; Child and Youth Health, Health New Zealand – Te Whatu Ora Counties Manukau, Starship Children's Hospital, Aotearoa New Zealand.

CORRESPONDING AUTHOR
A/Prof Rachel Webb: Department of Paediatrics, Child and Youth Health, The University of Auckland, C/o KidzFirst Children's Health, Middlemore Hospital, Hospital Road, Ōtāhuhu.
E: Rachel.web@middlemore.co.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/anticoagulation-management-and-poor-clinical-outcomes-in-tamariki-and-rangatahi-with-rheumatic-heart-disease-following-mechanica

REFERENCES
1. Carapetis JR, Beaton A, Cunningham MW, et al. Acute rheumatic fever and rheumatic heart disease. Nat Rev Dis Primers. 2016;2:15084. doi: 10.1038/nrdp.2015.84.
2. Watkins DA, Johnson CO, Colquhoun SM, et al. Global, Regional, and National Burden of Rheumatic Heart Disease, 1990-2015. N Engl J Med. 2017;377(8):713-22. doi: 10.1056/NEJMoa1603693.
3. Roth GA, Mensah GA, Johnson CO, et al. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update From the GBD 2019 Study. J Am Coll Cardiol. 2020;76(25):2982-3021. doi: 10.1016/j.jacc.2020.11.010. Erratum in: J Am Coll Cardiol. 2021 Apr 20;77(15):1958-1959. doi: 10.1016/j.jacc.2021.02.039.
4. 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;27(1):36-46. doi: 10.3201/eid2701.191791.
5. Tilton E, Mitchelson B, Anderson A, et al. Cohort profile: methodology and cohort characteristics of the Aotearoa New Zealand Rheumatic Heart Disease Registry. BMJ Open. 2022;12(12):e066232. doi: 10.1136/bmjopen-2022-066232.
6. Zühlke L, Engel ME, Karthikeyan G, et al. Characteristics, complications, and gaps in evidence-based interventions in rheumatic heart disease: the Global Rheumatic Heart Disease Registry (the REMEDY study). Eur Heart J. 2015 May 7;36(18):1115-22a. doi: 10.1093/eurheartj/ehu449.
7. Webb RH, Wilson NJ, Lennon DR, et al. Optimising echocardiographic screening for rheumatic heart disease in New Zealand: not all valve disease is rheumatic. Cardiol Young. 2011;21(4):436-43. doi: 10.1017/S1047951111000266.
8. Culliford-Semmens N, Nicholson R, Tilton E, et al. The World Heart Federation criteria raise the threshold of diagnosis for mild rheumatic heart disease: Three reviewers are better than one. Int J Cardiol. 2019;291:112-118. doi: 10.1016/j.ijcard.2019.02.058.
9. Culliford-Semmens N, Tilton E, Wilson N, et al. Echocardiography for latent rheumatic heart disease in first degree relatives of children with acute rheumatic fever: Implications for active case finding in family members. EClinicalMedicine. 2021;37:100935. doi: 10.1016/j.eclinm.2021.100935.
10. Webb R, Culliford-Semmens N, ChanMow A, et al. High burden of rheumatic heart disease confirmed by echocardiography among Pacific adults living in New Zealand. Open Heart. 2023;10(1):e002253. doi: 10.1136/openhrt-2023-002253.
11. Cannon JW, Zhung J, Bennett J, et al. The economic and health burdens of diseases caused by group A Streptococcus in New Zealand. Int J Infect Dis. 2021 Feb;103:176-181. doi: 10.1016/j.ijid.2020.11.193.
12. Milne RJ, Lennon D, Stewart JM, et al. Mortality and hospitalisation costs of rheumatic fever and rheumatic heart disease in New Zealand. J Paediatr Child Health. 2012;48(8):692-7. doi: 10.1111/j.1440-1754.2012.02446.x.
13. Liao YB, Wang TKM, Wang MTM, et al. Meta-Analysis of Mitral Valve Repair Versus Replacement for Rheumatic Mitral Valve Disease. Heart Lung Circ. 2022;31(5):705-10. doi: 10.1016/j.hlc.2021.11.011.
14. Remenyi B, Webb R, Gentles T, et al. Improved long-term survival for rheumatic mitral valve repair compared to replacement in the young. World J Pediatr Congenit Heart Surg. 2013;4(2):155-64. doi: 10.1177/2150135112474024. Erratum in: World J

Pediatr Congenit Heart Surg. 2013 Jul;4(3):330.
15. Otto CM, Nishimura RA, Bonow RO, et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021;143(5):e35-e71. doi: 10.1161/CIR.0000000000000932.
16. Bauman ME, Kuhle S, Bruce AA, et al. The journey for adolescents and young adults with chronic conditions transitioning to adult care with successful warfarin management. Thromb Res. 2016;141:183-8. doi: 10.1016/j.thromres.2016.03.019.
17. North RA, Sadler L, Stewart AW, et al. Long-term survival and valve-related complications in young women with cardiac valve replacements. Circulation. 1999;99(20):2669-76. doi: 10.1161/01.cir.99.20.2669.
18. McLintock C. Anticoagulant choices in pregnant women with mechanical heart valves: balancing maternal and fetal risks--the difference the dose makes. Thromb Res. 2013;131 Suppl 1:S8-10. doi: 10.1016/S0049-3848(13)70010-0.
19. Harper P, McMichael I, Griffiths D, et al. The community pharmacy-based anticoagulation management service achieves a consistently high standard of anticoagulant care. N Z Med J. 2015;128(1422):31-41.
20. Heneghan C, Ward A, Perera R, et al. Self-monitoring of oral anticoagulation: systematic review and meta-analysis of individual patient data. Lancet. 2012;379(9813):322-34. doi: 10.1016/S0140-6736(11)61294-4.
21. Ansell JE, Patel N, Ostrovsky D, et al. Long-term patient self-management of oral anticoagulation. Arch Intern Med. 1995;155(20):2185-9.
22. Anderson A, Peat B, Ryland J, et al. Mismatches between health service delivery and community expectations in the provision of secondary prophylaxis for rheumatic fever in New Zealand. Aust N Z J Public Health. 2019;43(3):294-299. doi: 10.1111/1753-6405.12890.
23. Health New Zealand – Te Whatu Ora. Rheumatic Fever Co-Design Initiative [Internet]. 2024 [cited 2024 Oct]. Available from: https://www.tewhatuora.govt.nz/publications/rheumatic-fever-co-design-initiative
24. Vervoort D, Yilgwan CS, Ansong A, et al. Tertiary prevention and treatment of rheumatic heart disease: a National Heart, Lung, and Blood Institute working group summary. BMJ Glob Health. 2023;8(Suppl 9):e012355. doi: 10.1136/bmjgh-2023-012355.

Electric scooter–related orthopaedic injuries in Wellington

Wing Yung Agnes Chu, Michael T Lee, Ilia Elkinson

ABSTRACT

AIM: Commercially operated electric scooters (e-scooters) were introduced to Wellington City in June 2019. This study aims to compare e-scooter–related orthopaedic injuries presenting to Wellington Hospital from June 2017 to June 2021, with data from other regions of New Zealand.

METHODS: The Wellington Hospital Accident Compensation Corporation Department provided a list of e-scooter–related claims presenting to Wellington Hospital over the 48-month period. A retrospective review was performed. Data were collected for patient demographics, circumstances of injury, management and follow-up. A single investigator reviewed all relevant imaging.

RESULTS: Between June 2017 and 2019, there were 14 e-scooter–related presentations with a total of two orthopaedic fractures in a single patient. Over the 2 years following June 2019, there were 295 e-scooter–related presentations. One hundred and seventeen patients sustained a total of 145 orthopaedic fractures. Twenty-nine percent of orthopaedic fractures required operative management. Intoxicated riders had a higher rate of open long bone fractures (p=0.003).

CONCLUSION: Orthopaedic injuries are common with e-scooter use. Higher rates of orthopaedic fractures in Wellington City require operative management compared with Auckland. The establishment of regulations and education around use of personal protective equipment, use while intoxicated and speed limits would also be beneficial.

Electric scooters (e-scooters) have emerged as a novel and accessible mode of transport. Worldwide, patterns of e-scooter–associated injuries are emerging, where minor contusions, abrasions and lacerations are the most common. This is followed by fractures or other orthopaedic injuries, particularly among young male riders.[1–4] Drug and alcohol use, in conjunction with lack of helmet use, has also been shown to increase injury severity, hospital length of stay and need for surgical intervention.[5]

In New Zealand, e-scooters are classified as low-powered vehicles.[6] E-scooter riders do not require a driver's licence and helmets are not legally required.[6] Commercially operated e-scooters can travel up to 25 kilometres per hour.[7] Since their introduction in New Zealand, Brownson et al. investigated the epidemiology of these injuries in the Auckland Region, with 41.7% of their cohort sustaining fractures.[1] Of those requiring orthopaedic operative management, the total economic cost was estimated to be NZ$19,282 per person.[8]

On 18 June 2019, 800 commercial e-scooters were introduced to Wellington City as part of an 18-month trial.[9] Not only known for its wind, Wellington City also has a unique geography. Being surrounded by hills, this densely populated city is filled with narrow and tortuous roads. By design of its small wheels, e-scooters may be susceptible to instability in such an environment.[8,10] The primary aim of this study is to compare e-scooter–related orthopaedic injuries presenting to Wellington Regional Hospital with known data from other regions of New Zealand. Secondary aims are to compare presentations in the 2 years preceding and following the introduction of commercial e-scooters, and to assess resources required for investigation and management.

Method

A retrospective chart review of all e-scooter–related injuries presenting to the Wellington Emergency Department over a 48-month period (June 2017 to June 2021) was performed. This period included 24 months prior to the introduction of commercial e-scooters (June 2017 to June 2019) and the 24 months after its introduction. Ethics approval was granted by the New Zealand Health and Disability Ethics Committees (Reference 2024 EXP 20944).

The Wellington Hospital Accident Compensation Corporation (ACC) Department provided a list of presentations to the Wellington Emergency Department with claims related to "scooter". The list was then screened manually to confirm relevance to e-scooters. E-scooters were defined

as scooters with a foot board, steering handle, two to three wheels and an electric motor. Mopeds or non-motorised scooters were excluded.

Using the screened list, we generated a list of National Health Indexes (NHIs) appropriate for review. These NHIs were then searched on the local electronic patient record system, known as the Medical App Portal (MAP). MAP allows access to patient data across the Wellington Region hospitals, which includes the Wellington Regional, Hutt and Wairarapa hospitals. Information was collected on patient demographics, including age, gender and ethnicity. Data on time and method of presentation, as well as circumstances of injury, considering mechanism, presence of personal protective equipment and intoxication were also collected. Alcohol involvement is a compulsory question prior to discharge from the electronic system of the Wellington Emergency Department. In instances where details were not documented, due to acuity or other reasons, these were noted as "no record".

Injuries were classified as non-orthopaedic fractures, orthopaedic fractures and orthopaedic soft tissue injuries. Fractures involving the skull, facial bones, ribs and teeth were not considered orthopaedic fractures for the purpose of this review. To note, a fracture diagnosis could only be made following physical examination and imaging. Relevant imaging of all modalities was retrospectively reviewed by a single investigator. Data on acute admission, type of operative management, clinic appointments, readmission and complications were also collected.

Statistical analysis was performed using frequency and percentage for categorical variables and mean with standard deviation for continuous variables.

Results

Between 17 June 2017 and 17 June 2019, 14 e-scooter–related presentations were identified. There was a total of two orthopaedic fractures in a single patient. These were managed non-operatively and the patient was discharged from fracture clinic with no complications.

Between 18 June 2019 and 18 June 2021, there were 295 e-scooter related presentations. The monthly number of presentations is presented in Figure 1. As shown in Table 1, 177 of these patients identified as male. The most common age group was 20–29 years of age. The first quarter of the day had a significantly lower number of presentations compared with the remaining quarters (Chi-squared test, p=0.006). Alcohol was involved in 30.5% of male presentations and 15.3% of female presentations. Falling from the e-scooter was the most common mechanism of injury. No deaths were observed at 90 days post-presentation.

Figure 1: Trend of electric scooter presentations to Wellington Emergency Department 18 June 2017–2021.

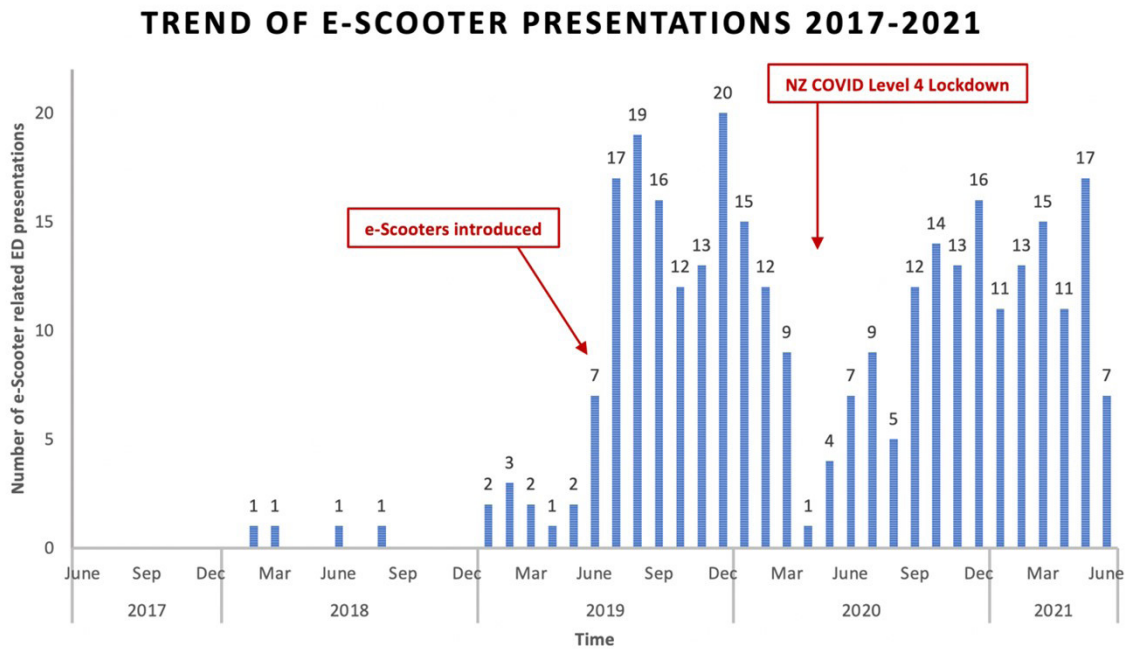


Table 1: Characteristics of the study cohort June 2019–2021.

	Entire cohort (n=295)	Male (n=177)	Female (n=118)
Age (years)	33.00±14.60	33.68±14.99	31.98±13.92
<19	40 (13.6%)	23 (13.0%)	17 (14.4%)
20–29	107 (36.3%)	64 (36.2%)	43 (36.4%)
30–39	68 (23.1%)	39 (22.0%)	29 (24.6%)
40–49	38 (12.9%)	24 (13.6%)	14 (11.9%)
50–59	23 (7.8%)	15 (8.5%)	8 (6.8%)
>60	19 (6.4%)	12 (6.8%)	7 (5.9%)
Ethnicity			
NZ European	164 (55.6%)		
NZ Māori	23 (7.8%)		
Pacific peoples	7 (2.4%)		
Other European	55 (18.6%)		
Asian	37 (12.5%)		
Other	9 (3.1%)		
Presentation			
Ambulance	79 (26.8%)	49 (27.7%)	30 (25.4%)
Self/other non-ambulance	216 (73.2%)	128 (72.3%)	88 (74.6%)
Triage time			
0000–0559	41 (13.9%)	29 (16.4%)	12 (10.2%)
0600–1159	75 (25.4%)	39 (22.0%)	36 (30.5%)
1200–1759	91 (30.8%)	57 (32.2%)	34 (28.8%)
1800–2359	88 (29.8%)	52 (29.4%)	36 (30.5%)
Personal protective equipment			
Yes	24 (8.1%)	14 (7.9%)	10 (8.5%)
No	16 (5.4%)	8 (4.5%)	8 (6.8%)
No record	255 (86.4%)	155 (87.6%)	100 (84.7%)
Alcohol			
Yes	72 (24.4%)	54 (30.5%)	18 (15.3%)
No	194 (65.8%)	108 (61.0%)	86 (72.9%)
No record	29 (9.8%)	15 (8.5%)	14 (11.9%)

Table 1 (continued): Characteristics of the study cohort June 2019–2021.

	Entire cohort (n=295)	Male (n=177)	Female (n=118)
Mechanism			
Rider			
Fall from e-scooter	238 (80.7%)	140 (79.1%)	98 (83.1%)
Stop/start	13 (4.4%)	8 (4.5%)	5 (4.2%)
Hit object	19 (6.4%)	16 (9.0%)	3 (2.5%)
Hit by moving object	11 (3.7%)	7 (4.0%)	4 (3.4%)
Non-rider			
Hit by e-scooter	8 (2.7%)	3 (1.7%)	5 (4.2%)
Tripped over e-scooter	2 (0.7%)	1 (0.6%)	1 (0.8%)
Other	4 (1.4%)	2 (1.1%)	2 (1.7%)

Table 2: Orthopaedic fractures and management.

	Number of fractures	Non-operative‡	Operative‡
Upper limb	n=107	n=88	n=19
Clavicle	8	8 (100%)	0
Scapula	2	2 (100%)	0
Humerus	12	8 (66.7%)	4 (33.3%)
Proximal radius/ulna	33	31 (93.9%)	2 (6.1%)
Both bone forearm‖	4 (2)†	2 (50%)	2 (50%)
Distal radius/ulna	23	16 (69.6%)	7 (30.4%)
Scaphoid	7	7 (100%)	0
Other carpal bones	5	5 (100%)	0
Metacarpals	11	8 (72.7%)	3 (27.3%)
Phalanges	2	1 (50%)	1 (50%)
Lower limb	n=36	n=15	n=23
Femur	2	0	2 (100%)
Patella	1	1 (100%)	0
Tibia§	9 (2)†	0	11 (100%)
Fibula	3 (2)†	3 (100%)	0
Malleolar	10	4 (40%)	6 (60%)

Table 2 (continued): Orthopaedic fractures and management.

	Number of fractures	Non-operative‡	Operative‡
Tarsal bones	2	1 (50%)	1 (50%)
Metatarsals	7	4 (57.1%)	3 (42.9%)
Phalanges	2	2 (100%)	0
Spine			
Transverse/spinous process	2	2 (100%)	0

‡ Percentage of particular fracture included in parentheses.
||Two cases of both bone forearm fractures.
†Number of open fractures included in parentheses.
§External fixators prior to open reduction internal fixation in two cases.

Of the 295 presentations since 18 June 2019, 54 patients sustained minor contusions, abrasions and lacerations. Forty-seven patients were diagnosed with a head injury and 28 patients sustained dental trauma. There were 11 patients with fractures of the skull or facial bones, and seven patients with rib fractures.

Regarding orthopaedic injuries, 13 patients sustained soft tissue injuries, such as patella tendon rupture. Two of these patients required surgery. A total of 145 orthopaedic fractures were diagnosed in 117 patients. Nine patients had pure joint dislocations without fracture.

Of the 107 upper limb fractures, fractures of the proximal radius/ulna were the most common (30.8%), followed by distal radius/ulna fractures (21.5%). Fifty-four point eight percent of radial head/neck fractures and 58.8% of distal radius fractures were intra-articular. Eighty-two point two percent of upper limb fractures were managed non-operatively, as demonstrated in Table 2. One patient had planned removal of metalware, and three patients had surgical complications. One readmitted for pain, one readmitted for wound infection and one referred to private orthopaedic surgeons with avascular necrosis. Further details on patients requiring readmission are included in the Appendix.

Of the 36 lower limb fractures, malleolar fractures were the most common (27.8%), followed by fractures of the tibia (25%). Fifty-eight point three percent of lower limb fractures required operative management. All femur and tibia fractures resulted in operative management, with two tibia fractures requiring an external fixator prior to open reduction internal fixation. Two patients had planned removal of metalware in the public hospital and two patients were referred to private orthopaedic surgeons for the same. One patient developed septic arthritis following open reduction internal fixation of their tibial plateau fracture, resulting in two reoperations followed by a stitch abscess.

Assessing the effect of alcohol on the severity of long bone fractures, there were 14 long bone fractures sustained while intoxicated and 81 fractures sustained with no/unknown alcohol intake. Of the fractures sustained while intoxicated, 50% of these were intra-articular and 28.5% were open fractures. In the no/unknown alcohol group, 55.6% of fractures were intra-articular and 2.5% were open. The difference in open fractures between the two groups were statistically significant (Fisher's exact test, p=0.003).

Of the 61 advanced imagings performed acutely within the Wellington Region hospitals, 38 were performed to evaluate orthopaedic injuries. Of these, 37 were computerised tomography (CT) scans and one was an magnetic resonance imaging (MRI) scan to evaluate for soft tissue knee injury. In the follow-up period, five scans were performed to assess for complications, e.g., ultrasound for deep vein thrombosis to CT to assess for union.

There were 40 admissions to Wellington Regional Hospital, with one to neurosurgery, one to general surgery and two to urology. The remaining 36 presentations resulted in acute

(72.2%) or planned-acute (27.8%) admissions under the orthopaedic service. For this group, range of stay was 0–27 days, with a median of 2.5 days. Eight patients required readmission. The total acute surgical time at Wellington Hospital was 3,393 minutes from surgical incision to closure, with a further 126 surgical minutes for planned removal of metalware or further surgeries related to complications. Ninety-three patients were seen at the Wellington Fracture Clinic with a total of 253 clinic appointments.

Discussion

E-scooters are an accessible and convenient mode of transport. However, as illustrated by our study, orthopaedic injuries are common. In our cohort, 39.7% were diagnosed with orthopaedic fractures. While this is similar to the 37.2% described in the 2019 Auckland study by Brownson et al., 29.0% of orthopaedic fractures in our study required operative management, compared to the 15.0% found in the Auckland study.[1] Our study, as well as identifying 4.1% of total fractures as open fractures, also assessed further markers of high-energy trauma, such as fracture–dislocations and intra-articular involvement.

While blood alcohol level is not routinely measured, the Wellington Hospital Emergency Department electronic documentation system requires the discharging clinician to comment on alcohol involvement for every patient, whether trauma-related or not. In our cohort, alcohol was involved in approximately one quarter of presentations. This is again comparable to Brownson's study in Auckland.[1] Across Australasia, intoxication rates as high as 46% have also been observed.[11] With our study showing a higher rate of open long bone fractures in intoxicated riders, this calls for further regulations or education to prevent e-scooter use while intoxicated.

Our study observed radial head/neck fractures to be the most common upper limb fracture. A fall onto an outstretched hand can produce such an injury, and this is in keeping with our cohort's young mean age and most common mechanism of injury of rider fall. Only 8.1% of our cohort was wearing personal protective equipment at the time of injury. The low numbers are similar to that observed in other trauma centres, and are unsurprising given helmet use is not legally enforced in New Zealand.[12]

Our study found a stark difference in the number of e-scooter–related injuries since the introduction of commercially operated e-scooters in Wellington City. For fractures alone, there were two prior to their introduction, compared with the 145 noted in the 2 years following their introduction. This difference is likely dulled by the social effects of COVID-19 between 2020 and 2021, as supported by the low number of presentations around the lockdown periods. In our study, riders presenting in the post-introduction period were not classified into private and commercial e-scooter groups. While this is a limitation due the study's retrospective nature, it is known that private and commercial e-scooter riders have different user profiles, with an Australian study demonstrating a higher prevalence of illegal riding in commercial e-scooter users.[13] The large increase in the number of injuries seen is therefore likely from users of commercial e-scooters.

In the 2018 Census, 74.1% of the Wellington City population identified as European, 8.6% as Māori, 5.1% as Pacific peoples and 18.3% as Asian.[14] Our study reflects a similar proportion of European and Māori patients (74.2% and 7.8% respectively), but found a lower percentage of patients identifying as Pacific peoples or Asian. While this may represent a true difference in the number of injuries, this may also indicate differences in injury severity or preference for accessing primary care. A 2020 study in Auckland found 68.1% of patients with e-scooter injuries presented to primary care.[15] This indicates that the total number of e-scooter injuries and associated socio-economic cost are much higher than shown by our study.

Our study has redemonstrated the resource-intensive nature of e-scooter injury management. In our cohort, 36 patients required hospital admission and 42 fractures were managed operatively. With the average study participant being 33 years of age, such injuries often result in time off work or reduced duties, both of which have financial and productivity implications as demonstrated by Campbell et al.[8] While our study focussed on orthopaedic injuries alone, it is important to acknowledge that head and facial injuries, as well as minor contusions, abrasions and lacerations are also common in e-scooter–related presentations, and can be a source of morbidity.[2,16–18]

This study has a number of limitations. It is a retrospective review, whereby accuracy of our data is dependent upon the quality of prior documentation. In addition, data collection was carried out by a single unblinded investigator. While this promotes consistent data interpretation, it is also a source for potential bias. Furthermore,

ACC data on mechanism of injury are reliant on details recorded by the patient and clinician. This, together with the high number of e-scooter injuries presenting to primary care, suggests the true burden of these injuries is much higher than that found in our study.

E-scooters are an increasingly popular transport option. Their unique user profile and accessibility create a new mechanism and pattern of orthopaedic injuries. The mountainous landscape of Wellington City poses new challenges in maintaining rider safety, where orthopaedic fractures in Wellington are almost twice as likely to require operative management as those in Auckland. Orthopaedic soft tissue injuries further add to the burden. Assessment and management of these injuries are also resource intensive. Future studies can further assess the economic cost of e-scooter injuries in Wellington, as well as the relationship between e-scooter models and risk of fall. The establishment of regulations and education around use of personal protective equipment, use while intoxicated and speed limits would also be beneficial.

COMPETING INTERESTS
Nil.

ACKNOWLEDGEMENTS
We would like to acknowledge Associate Professor Peter Larsen for his assistance with our statistical analysis.

AUTHOR INFORMATION
Wing Yung Agnes Chu, MBChB: Orthopaedic Registrar, Wellington Hospital, New Zealand.
Michael T Lee, MBBS: Orthopaedic SET Trainee, Wellington Hospital, New Zealand.
Ilia Elkinson, BHB, MBChB: Orthopaedic Surgeon, Wellington Hospital, New Zealand.

CORRESPONDING AUTHOR
Wing Yung Agnes Chu, MBChB: Department of Orthopaedics, Wellington Regional Hospital, Private Bag 7902, Wellington 6242, New Zealand.
E: agneschuwy@gmail.com

URL
https://nzmj.org.nz/journal/vol-138-no-1614/electric-scooter-related-orthopaedic-injuries-in-wellington

REFERENCES
1. Brownson AB, Fagan PV, Dickson S, Civil ID. Electric scooter injuries at Auckland City Hospital. N Z Med J. 2019;132(1505):62-72.
2. Trivedi TK, Liu C, Antonio ALM, et al. Injuries Associated With Standing Electric Scooter Use. JAMA Netw Open. 2019;2(1):e187381. doi: 10.1001/jamanetworkopen.2018.7381.
3. Mitchell G, Tsao H, Randell T, et al. Impact of electric scooters to a tertiary emergency department: 8-week review after implementation of a scooter share scheme. Emerg Med Australas. 2019;31(6):930-934. doi: 10.1111/1742-6723.13356.
4. Fisher ND, Nwakoby E, Hernandez H, McLaurin TM. Electric scooter injuries: Incidence and injury patterns at a level I trauma center. Chin J Traumatol. 2023;26(6):334-338. doi: 10.1016/j.cjtee.2023.02.003.
5. Mitra B, Heald E, Sri-Ganeshan M, et al. Electric scooter-related trauma, alcohol and other drugs. Emerg Med Australas. 2023;35(2):353-356. doi: 10.1111/1742-6723.14170.
6. NZ Transport Agency Waka Kotahi. Low-powered vehicles [Internet]. [cited 2023 Sep 3]. Available from: https://www.nzta.govt.nz/vehicles/vehicle-types/low-powered-vehicles/
7. Flamingo. Flamingo Scooters [Internet]. [cited 2023 Sep 3]. Available from: https://flamingoscooters.com/how-to/scooter
8. Campbell A, Wong N, Monk P, et al. The cost of electric-scooter related orthopaedic surgery. N Z Med J. 2019;132(1501):57-63.
9. Wellington City Council. E-scooter share scheme launches in Wellington [Internet]. 2019 [cited 2023 Sept 3]. Available from: https://wellington.govt.nz/news-and-events/news-and-information/our-wellington/2019/06/e-scooter-launch
10. Barker R. Electric scooters. Emerg Med Australas. 2019;31(6):914-915. https://doi.org/10.1111/1742-6723.13424.
11. Moran J, Buck A, Williams J, et al. Impact on the health service in the Top End, Northern Territory following the introduction of an electric scooter sharing service. Emerg Med Australas. 2023;35(2):194-199. doi: 10.1111/1742-6723.14090.
12. Lavoie-Gagne O, Siow M, Harkin W, et al. Characterization of electric scooter injuries over 27 months at an urban level 1 trauma center. Am J Emerg Med. 2021;45:129-136. doi: 10.1016/j.ajem.2021.02.019.
13. Haworth N, Schramm A, Twisk D. Comparing the risky behaviours of shared and private e-scooter and bicycle riders in downtown Brisbane, Australia. Accid Anal Prev. 2021;152:105981. doi: 10.1016/j.aap.2021.105981.
14. Stats NZ. 2018 Census place summaries: Wellington City [Internet]. [cited 2024 Jun 30]. Available from: https://www.stats.govt.nz/tools/2018-census-place-summaries/wellington-city#ethnicity-culture-and-identity
15. Bekhit MNZ, Le Fevre J, Bergin CJ. Regional healthcare costs and burden of injury associated with electric scooters. Injury. 2020;51(2):271-277. doi: 10.1016/j.injury.2019.10.026.
16. Hennocq Q, Schouman T, Khonsari RH, et al. Evaluation of Electric Scooter Head and Neck Injuries in Paris, 2017-2019. JAMA Netw Open. 2020;3(11):e2026698. doi: 10.1001/jamanetworkopen.2020.26698.
17. Akalin BE, Alten A, Gündeş E. Electric Scooter-related Craniofacial Injuries. J Craniofac Surg. 2023;34(8):2328-2331. doi: 10.1097/SCS.0000000000009660.
18. Shichman I, Shaked O, Factor S, et al. Epidemiology of Fractures Sustained During Electric Scooter Accidents: A Retrospective Review of 563 Cases. J Bone Joint Surg Am. 2021;103(12):1125-1131. doi: 10.2106/JBJS.20.01746.

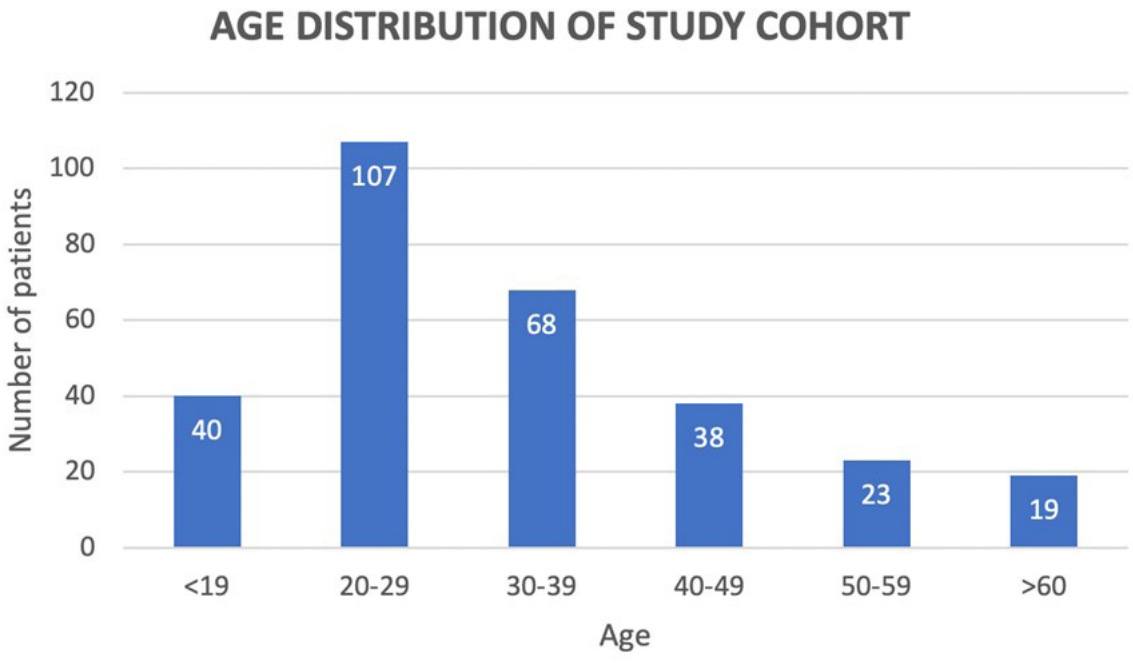
Appendix

Appendix Table 1: Table of patients requiring readmission.

Patient	No. admissions	LOS	LOS total	Readmission reason
A	2	**0**+3	3	Post-operative wound infection
B	2	2+**1**	3	Nil
C	3	2+**2**+1	5	Post-operative pain
D	2	**4**+12	16	Post-operative knee septic arthritis (closed initial injury)
E	2	1+**2**	3	Nil
F	3	1+**1**+0	2	Removal of metalware
G	2	**2**+0	2	Removal of k-wires
H^	2	**1**+0	1	Removal of de-functioning wire

NO. = number; LOS = length of stay.
Admission with operation highlighted in bold.
^Soft tissue knee injury.

Appendix Figure 1: Age of patients presenting with electric scooter injuries 18 June 2019–2021.



Proton pump inhibitors in cirrhosis: a retrospective five-year analysis of increased risks of hepatic decompensation and infections

Abhimati Ravikulan, Natalie Russell, Christin Coomarasamy, Ashok Raj

ABSTRACT

AIM: Proton pump inhibitors (PPIs) are widely used in cirrhotic patients, often without a clear indication. Evidence links PPI use to adverse outcomes such as hepatic encephalopathy and spontaneous bacterial peritonitis. This study analyses outcomes associated with PPI use in cirrhosis over 5 years at a New Zealand tertiary centre.

METHODS: This retrospective study included all patients diagnosed with liver cirrhosis at Counties Manukau Health in 2014. Patients were divided into two groups: those taking PPIs and those not taking PPIs. Demographic data, relevant blood tests and cumulative PPI doses were recorded. Outcomes such as mortality, liver-related events (hepatic encephalopathy, spontaneous bacterial peritonitis, variceal bleeding and ascites) and infections were monitored over 5 years. Logistic regression analyses calculated odds ratios (ORs) for the association of PPI usage with outcomes, adjusting for age, comorbidities, medications, aetiology of cirrhosis and liver disease severity.

RESULTS: Of 392 patients, 304 (77%) received PPIs and 88 (23%) did not. Only 31% had a clear indication for PPI use. PPI users had higher comorbidity and liver disease severity. Adjusted analysis showed no significant difference in all-cause mortality (29.9% vs 19.3%, p=0.67) or liver-related mortality (35.2% vs 17.6%, p=0.37). However, there were higher liver-related events (30.9% vs 10%, OR 2.9, p=0.046) and all-cause infections (30.9% vs 11.2%, OR 2.4, p=0.024).

CONCLUSION: PPI use in cirrhosis is linked to hepatic decompensation and higher infection risk. Judicious PPI use with clear indications is essential.

Proton pump inhibitors (PPIs) are among the most commonly prescribed medications worldwide, valued for their efficacy in managing symptoms from acid-related diseases. They are particularly effective in controlling conditions such as gastro-oesophageal reflux disease, peptic ulcer disease and for the treatment of *Helicobacter pylori* infections when used in combination with antibiotics.[1]

In patients with chronic liver disease, PPIs are often prescribed, albeit frequently without a definitive indication. A review conducted in 2017 suggests that up to 60% of chronic liver disease patients receiving PPIs do so without clear clinical justification.[1] The commonly approved indications for PPI use in this demographic include peptic ulcer disease, variceal bleeding and reflux disease.[1,2]

Emerging evidence has highlighted potential adverse outcomes associated with PPI use in patients with cirrhosis, a severe form of chronic liver disease. Notably, studies have indicated an elevated risk of hepatic encephalopathy and spontaneous bacterial peritonitis in this patient group.[3,4] These findings suggest that while PPIs are effective for their intended uses, their application in cirrhotic patients may necessitate cautious consideration due to these potential risks.

Additionally, there is a broader concern among clinicians regarding the association of PPI use with an increased risk of all-cause infections.[2] This is particularly relevant in the context of chronic liver disease, where patients are already at heightened risk of infections due to compromised liver function and other comorbidities.

Despite the global insights into the risks of PPI use in chronic liver disease, there is a notable paucity of data specific to New Zealand. This lack of localised data is significant given the high prevalence of chronic liver disease within the New Zealand population. Understanding the implications of PPI use in this setting is critical for optimising patient outcomes and guiding clinical practice.

This study aims to fill this gap by analysing the outcomes associated with PPI use in patients with cirrhosis over a 5-year period at a major tertiary centre in New Zealand. By examining mortality rates, the incidence of liver-related events and infection rates, this research seeks to provide valuable insights into the safe and effective use of PPIs in the management of chronic liver disease within the New Zealand healthcare context.

Methods

This retrospective observational study analysed data from patients presenting with chronic liver disease at Middlemore Hospital in Auckland, either as inpatients or outpatients, in the year 2014. Ethical approval was obtained from the Health and Disability Ethics Committee.

Patients diagnosed with liver cirrhosis were identified using the International Classification of Diseases 10th Revision (ICD-10) coding for a diagnosis of "cirrhosis" or "chronic liver disease" from electronic patient records for the year 2014. The cohort was divided into two groups: those who received PPI therapy and those who did not. Both groups were followed over a 5-year period to monitor primary and secondary outcomes. The primary outcome was mortality, while secondary outcomes included incidences of hospitalisations due to hepatic decompensation (such as hepatic encephalopathy, ascites, variceal bleeds) and all-cause infections (including spontaneous bacterial peritonitis).

Inclusion criteria required patients to have a clear ICD-defined diagnosis of cirrhosis. Exclusion criteria were not specifically outlined; however, only patients meeting the diagnostic criteria were included. Data already collected as part of patient management at Middlemore Hospital were utilised.

Baseline data extracted for both groups included demographics (age, gender, ethnicity), causes of cirrhosis, comorbidities (including ischemic heart disease, heart failure, renal disease, chronic obstructive pulmonary disease, asthma, hepatocellular carcinoma [HCC] and non-HCC malignancy), relevant blood test results (liver function tests, full blood count, renal function) and concurrent medications. The severity of liver disease in both groups was assessed using the Child–Pugh and MELD scores. This was used in analysis to correct for liver disease severity. The Charlson Comorbidity Index was employed to evaluate the systemic health of patients with cirrhosis. Indications for PPIs prescribed were also collected.

Patients in the PPI group had their PPI therapy classified by dose, duration and type of PPI. In New Zealand, only three types of PPIs are available (omeprazole, pantoprazole and lansoprazole). Medication exposure was quantified using the defined daily dose (DDD) recommended by the World Health Organization, which represents the assumed average maintenance dose per day for a drug used for its main indication in adults. For the purpose of this study, the maintenance dose was defined as omeprazole 20mg. Consequently, 20mg of omeprazole taken once daily for 3 months equated to a cumulative defined daily dose (cDDD) of 3 months. Potency equivalence among the three PPIs was standardised as omeprazole 20mg = pantoprazole 40mg = lansoprazole 30mg.

Primary outcomes recorded for this study were mortality in the PPI group vs non-PPI group. This was split into all-cause mortality and liver-related mortality (as identified by the primary cause of death recorded on patient file pertaining to their chronic liver disease).

Secondary outcomes recorded included incidence of liver-related events such as spontaneous bacterial peritonitis, hepatic encephalopathy, variceal bleeding and ascites that required hospital admission in the PPI group vs non-PPI group.

Furthermore, primary and secondary outcomes were analysed in the PPI group with respect to cDDD and whether increased cumulative exposure to PPIs increased risk of mortality of liver-related events.

Statistical analysis

Demographic and clinical characteristics were presented as counts and proportions for categorical variables and as means with standard deviations or medians with interquartile ranges for continuous variables. For comparisons between groups, analysis of variance (ANOVA) or Kruskal–Wallis tests were utilised for continuous variables, and Chi-squared or Fisher's exact tests were employed for categorical variables, as deemed appropriate. Logistic regression analyses were conducted to calculate the odds ratio (OR) with a 95% confidence interval (CI) of the association between PPI use and the outcomes. The model was adjusted for demographic characteristics, causes of liver disease, comorbidities, liver function tests and medications to determine if the difference in PPI

use and any risk factors remained significantly associated with the outcomes. Model selection was performed using forward and backward selection methods, retaining those risk factors with a p-value of less than 0.15 in the model. A two-tailed p-value of less than 0.05 was considered statistically significant.

Association between PPI dose and mortality

A multivariable logistic regression model was employed to assess the relationship between cumulative PPI dose (cDDD) and liver-related mortality. The dependent variable was mortality (binary: 1 = death, 0 = survival), while the independent variables included cDDD and covariates such as age, Charlson Comorbidity Index, aetiology of cirrhosis, cirrhosis severity, antiviral use and beta-blocker use. A constant term was added to the model, and ORs with 95% CIs were calculated for each covariate. Statistical significance was determined using a p-value threshold of <0.05.

Association between PPI dose and liver-related events

To evaluate the association between cDDD and liver-related events (ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, variceal bleeding and other infections) Poisson regression models were used. Each event was treated as a count outcome (e.g., number of occurrences per patient). The predictor variables included cDDD, age, Charlson Comorbidity Index, cirrhosis severity, aetiology of cirrhosis, antiviral use and beta-blocker use. A log link function was used to model the relationships, and the coefficients were exponentiated to produce incidence rate ratios. The statistical significance of the association between cDDD and each outcome was evaluated using a p-value threshold of <0.05.

All statistical analyses were carried out using the Statistical Analysis Software version 9.4 (SAS Institute, Cary, North Carolina).

Results

Overall results

A total of 392 patients diagnosed with cirrhosis were included in this study. Of these, 304 patients (78%) were administered PPIs, while 88 patients (22%) were not. The mean duration of PPI usage was approximately 71 months (standard deviation 88), encompassing use for up to a year prior to the commencement of the study.

Within the PPI group, only 32% (n=60; peptic ulcer disease n=23; variceal bleeding n=8; gastro-oesophageal reflux disease n=29) of patients were prescribed PPIs for an evidence-based indication. Seventy percent of patients (n=216) were prescribed PPI therapy for unspecified indications. The remaining patients were prescribed PPI therapy for non-evidence-based indications, including non-ulcer-related gastrointestinal bleeding, dyspepsia and oesophagitis unrelated to reflux disease (Figure 1).

Figure 1: Indications for proton pump inhibitor (PPI) use.

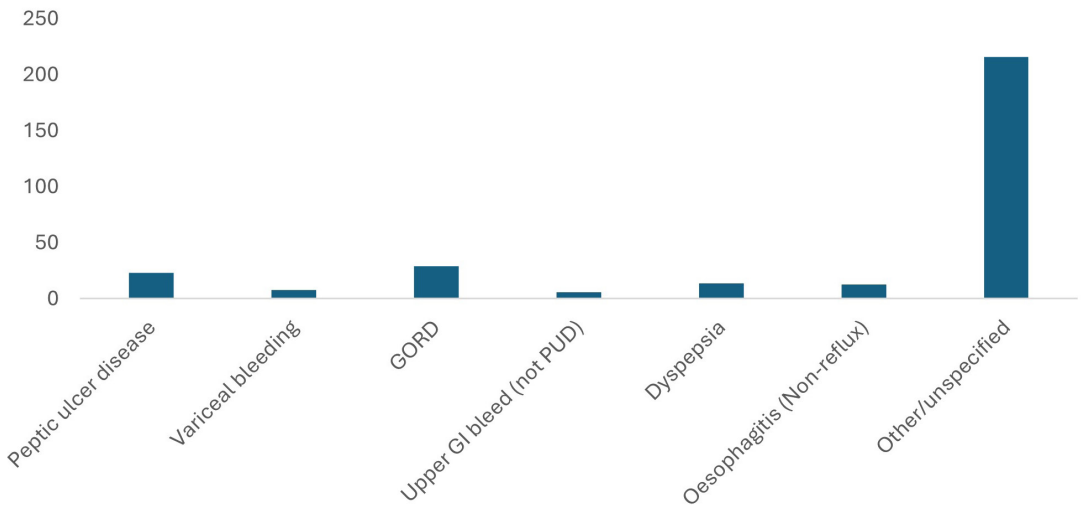


Table 1: Baseline demographics.

	PPI (N=304)	Non-PPI (N=88)	p-value
Age (*mean*)	60	58	0.06
Sex (%)	65 (M); 35 (F)	68 (M); 32 (F)	0.635
Biochemistry (*Median [range]*):			
ALT (U/L)	34 (8–555)	30 (10–165)	**0.049**
Bilirubin (µmol/L)	14 (4–401)	11 (4–70)	**0.015**
Albumin (g/L)	40 (14–51)	42 (27–48)	**0.013**
Prothrombin ratio	1 (0.8–3.7	1 (0.9–2.3)	**0.001**
Platelet count (x10^9)	156 (23–472)	178 (77–468)	0.007
MELD score (*Median [range]*)	7 (6–37)	6 (6–24)	0.000
Child–Pugh Score (*N [%]*):			
A	229 (75.3)	79 (89.8)	**0.010**
B	64 (21.1)	9 (10.2)	
C	11 (3.0)	0 (0)	
Aetiology (*N [%]*):			
Hepatitis B	101 (33.2)	43 (48.9)	**0.007**
Hepatitis C	60 (19.7)	24 (27.3)	0.129
Alcohol	77 (25.3)	11 (12.5)	**0.011**
NASH	58 (19.1)	9 (10.2)	0.052
Autoimmune	12 (4)	1 (1.1)	0.313
Other	30 (10)	6 (6.8)	0.383
Comorbidities (*N [%]*):			
IHD	20 (6.6)	1 (1.1)	0.057
CCF	14 (4.6)	3 (3.4)	0.773
COPD	16 (5.2)	2 (2.3)	0.385
Renal	40 (13.2)	6 (6.8)	0.104
Malignancy	29 (9.5)	3 (3.4)	0.064
Charlson Comorbidity Index (*Median [IQR]*)	5 (3.5–7)	4 (3–5.5)	**0.002**
HCC	28 (9.2)	10 (11.36)	0.548

Table 1 (continued): Baseline demographics.

Medications (*N [%]*):			
H2 receptor blocker	1 (0.3)	0 (0)	0.950
Hepatitis B antiviral	84 (27.6)	39 (44.3)	**0.003**
Hepatitis C antiviral	2 (0.7)	0 (0)	0.950
Metformin	52 (17.1)	11 (12.5)	0.300
Statins	82 (27.0)	17 (19.3)	0.146
Antiplatelets	58 (19.1)	14 (15.9)	0.499
Non-selective beta-blocker	38 (12.5)	4 (4.6)	**0.034**
Antihypertensive	113 (37.2)	31 (35.2)	0.739

PPI = proton pump inhibitors; ALT = alanine transaminase; NASH = non-alcoholic steatohepatitis; IHD = ischaemic heart disease; CCF = congestive cardiac failure; COPD = chronic obstructive pulmonary disease; IQR = interquartile range; HCC = hepatocellular carcinoma.

Baseline characteristics

The average age of patients across both cohorts was 59 years and was comparable between the groups. At baseline, patients in the PPI group had more severe liver disease as seen by the liver function markers, MELD score (p=0.000) and Child–Pugh score (p=0.010) for the patients in this group.

Hepatitis B was the most common cause of cirrhosis in both groups, followed by alcoholic liver disease. There was a marginally higher prevalence of hepatitis B in patients not exposed to PPIs (p=0.007), while alcoholic liver disease was more prevalent in those exposed to PPIs (p=0.011).

More patients in the PPI group were also on a non-selective beta blocker agent (p=0.034) compared to the non-PPI group. This is likely to reflect the more severe nature of liver disease in these patients with complications such as portal hypertension and variceal bleeding.

Regarding comorbidities, the overall Charlson Comorbidity Index score indicated a higher comorbidity burden in the PPI group (p=0.002) compared to the non-PPI group (Table 1).

Outcomes

The primary outcome assessed was mortality. Ninety-one patients (30%) in the PPI group died during the 5-year follow-up period, compared with 17 patients (19%) in the non-PPI group. After adjusting for confounding factors significantly different at baseline and factors significant on univariate analysis (age, aetiology of cirrhosis, Charlson Comorbidity Index, medications), the final multivariate adjusted ORs indicated no significant difference in mortality between the two groups (OR 0.85, p=0.668) (Figure 2).

The primary outcome was further analysed looking specifically at liver-related deaths. Of the patients who had died during observation period, 32 deaths (35.2%) were attributed to liver-related causes in the PPI group and three deaths (17.6%) were attributed to liver-related causes in the non-PPI group. After adjusting again for confounding variables, the final OR and p-value indicated no statistical difference in liver-related mortality between both groups (OR 1.79; p=0.365) (Figure 3).

The secondary outcome evaluated was the number of hospitalisations due to hepatic decompensation and all-cause infections over the 5-year follow-up period. There were 94 (31%) hospital admissions in the PPI group and nine (10%) admissions in the non-PPI group. The multivariate adjusted analysis revealed a significant difference in hospital admissions between the groups (OR 2.95, p=0.046). Furthermore, the incidence of infections was statistically significant between the groups, with 94 (30%) hospital admissions for infections in the PPI group and 10 (11%) admissions in the non-PPI group (OR 2.42, p=0.025) (Figure 4, Figure 5).

Outcomes with cDDD

Primary and secondary outcomes were further analysed in the PPI group with respect to cumulative dose exposure to PPIs using the cDDD.

When mortality in the PPI group was analysed

Figure 2: All-cause mortality PPI vs non-PPI.

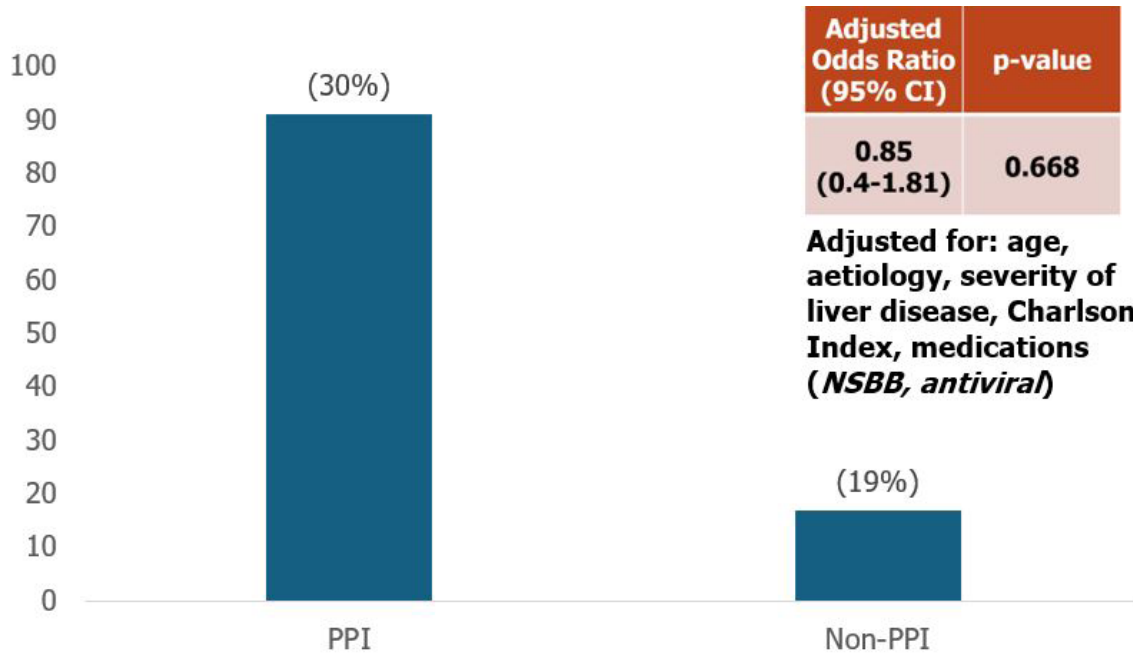


Figure 3: Liver-related mortality PPI vs non-PPI.

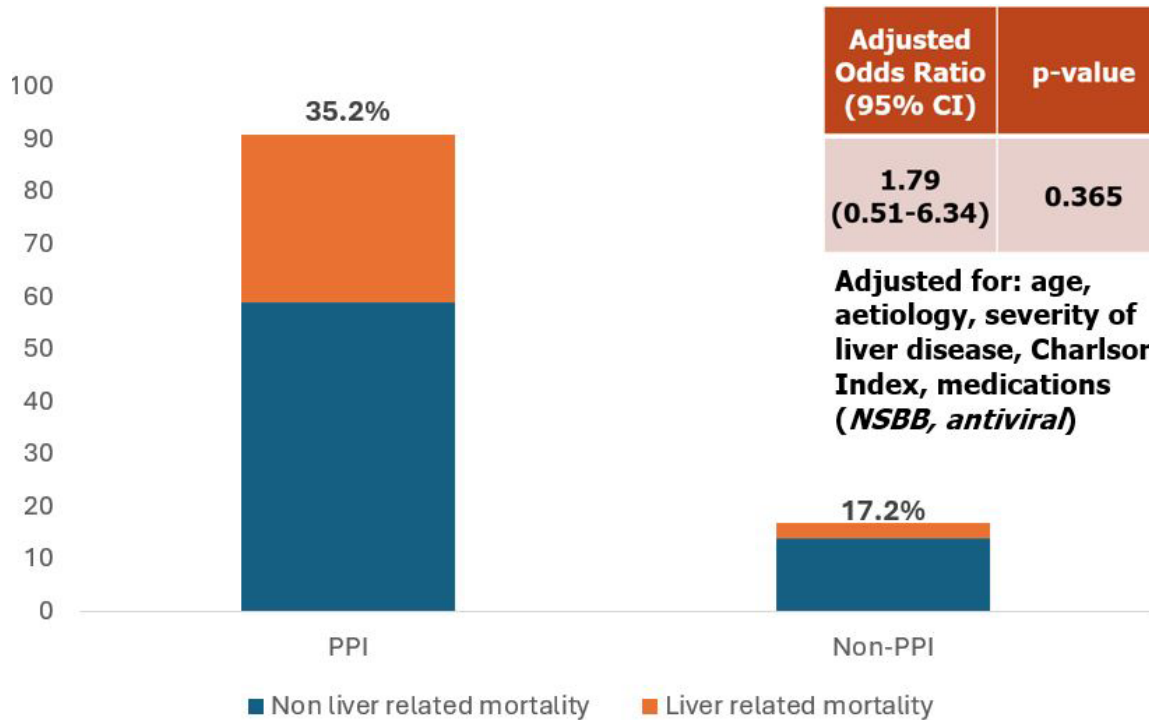


Figure 4: Hepatic decompensation events PPI vs non-PPI.

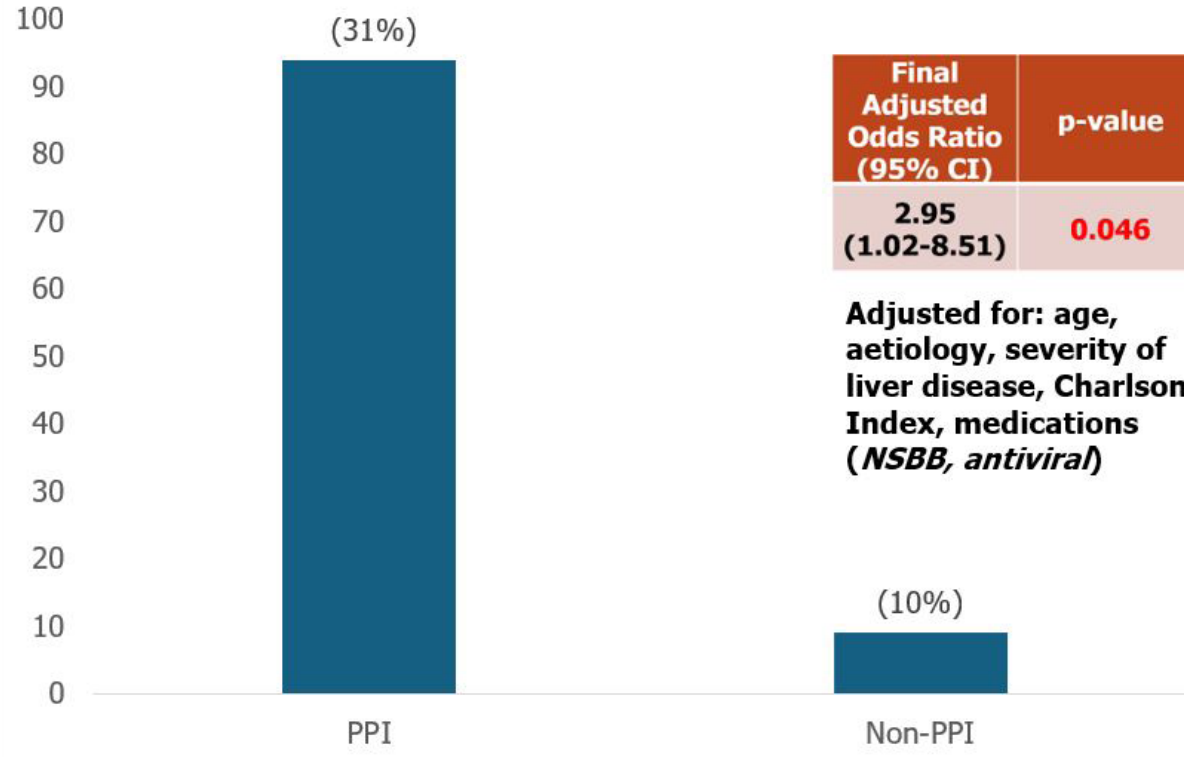


Figure 5: All-cause infections PPI vs non-PPI.

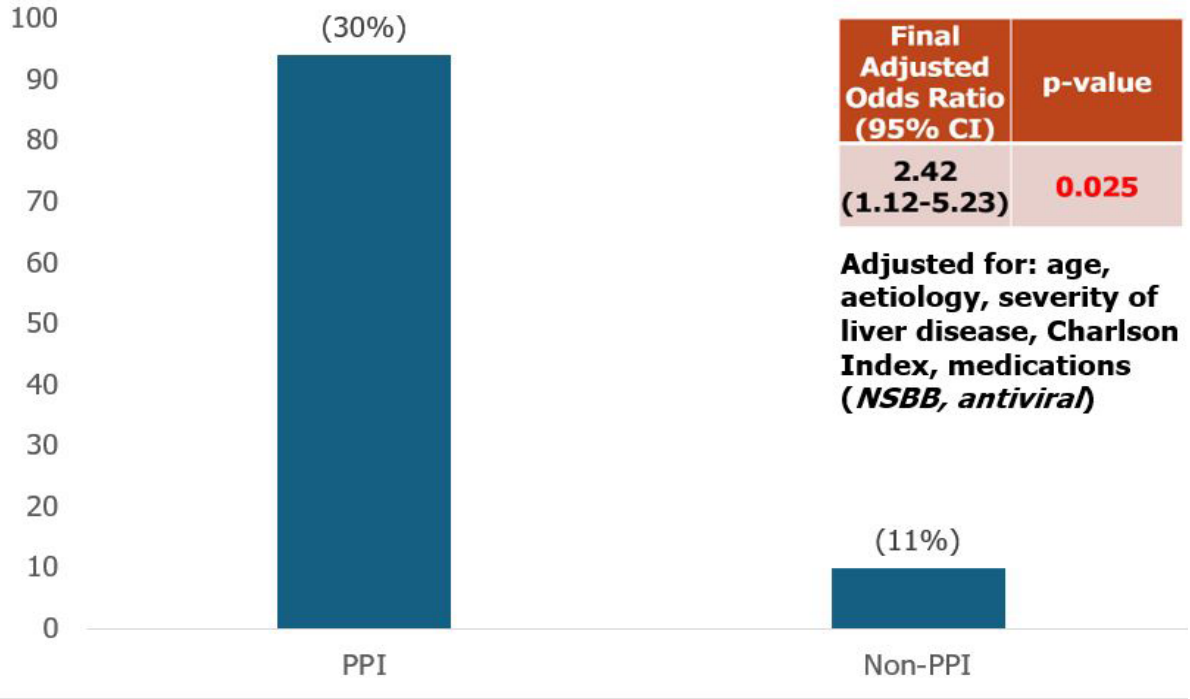


Table 2: Cumulative dose association with liver-related outcomes in the proton pump inhibitor group.

Outcome	Cumulative defined daily dose coefficient	Odds ratio	p-value
Ascites	0.0046	1.005	<0.001
Hepatic encephalopathy	0.0053	1.005	**<0.001**
Spontaneous bacterial peritonitis	0.0024	1.002	**0.029**
Variceal bleeding	0.0042	1.004	0.008
Other infections	0.0027	1.003	0.020

using logistic regression, with other factors corrected for, there was a small statistically significant decrease in mortality in the PPI group (OR 0.0044, p=0.037).

When liver-related events were analysed using Poisson regression in the PPI group with other factors corrected for, it was found that cumulative PPI use is significantly associated with increased risk of all liver-related events and other infections (Table 2).

Discussion

In this study conducted in a tertiary centre, we observed a significant association between the use of PPIs and the incidence of infections in patients with liver cirrhosis. Specifically, the literature supports a link between PPI use and spontaneous bacterial peritonitis, which may be attributed to bacterial dysbiosis caused by alterations in the gut microbiome.[5–8] PPIs disrupt the normal gut flora by altering gastric pH, leading to bacterial overgrowth and changes in the microbiome composition.[5–8] These changes can compromise the intestinal barrier, facilitating bacterial translocation into the bloodstream.[5–8] This mechanism might explain the higher incidence of infections, such as spontaneous bacterial peritonitis, and an increased risk of hepatic encephalopathy observed in PPI users with cirrhosis.[8,9] It is worth noting that our study demonstrates an increased risk of infections and liver-related events with cumulative increase in PPI dose, further supporting this hypothesis. One study carried out in Singapore also looked at cDDD of PPI in cirrhosis and found increased events of hepatic decompensation.[10] Our study is in accordance with these findings.

Our study does not reflect a significant increase in mortality associated with PPI use, and in fact our study suggests a slight decrease in mortality associated with increasing cumulative dose of PPIs. Previous studies have suggested that PPI use may be associated with increased mortality in patients with cirrhosis. Dultz et al.[11] reported PPI use to be an independent predictor of mortality in cirrhosis. The study carried out in Singapore also showed an increase in mortality with increased cumulative dose exposure to PPI.[10] However, this was within an observation period of 1 year. Another retrospective study performed in Scotland showed no difference in mortality between PPI users and non-PPI users.[12] A further retrospective study carried out in Taiwan looked specifically at mortality in patients admitted with hepatic encephalopathy on PPIs and reported an increased short-term and long-term mortality risk.[13] It is difficult to make confident conclusions on the association between PPI use and mortality in chronic liver disease patients. Some studies have demonstrated an increased risk while others have not. A vast majority of these studies are retrospective in nature and as such correcting for confounding factors will be challenging to establish clear association. This was explored by China et al.[14] in a *post hoc* analysis of a large multicentre randomised trial (the ATTIRE trial) of patients with cirrhosis. The outcomes of this analysis were that there was no significant increase in infections, renal disease or mortality in patients taking PPI therapy, but the study acknowledged the large real-world difference in baseline

characteristics between cirrhosis patients that contributed significantly to confounding factors in analysis, and this should be considered in any further recommendations made with regards to PPI use in these patients.[14] Given these conflicting findings in the literature, this topic remains debated and it is difficult to say with certainty whether PPIs increase mortality in patients with cirrhosis. This will ideally need to be further explored with prospective studies in future.

Our study stands out due to its reasonable sample size of 394 patients with liver cirrhosis and an extensive follow-up period of 5 years. This allows for a robust assessment of the relationship between PPI use and clinical outcomes in liver disease. Both groups were comparable at baseline in terms of demographics such as age, ethnicity and aetiology of cirrhosis, thereby minimising selection bias. However, it is noteworthy that the severity of liver disease was significantly greater in the PPI group compared with the non-PPI group, which could have contributed to the poorer morbidity outcomes observed in that cohort.

Additionally, our study revealed a high prevalence of hepatitis B among the participants, which contrasts with other studies carried out internationally.[1–4,10–15] This discrepancy highlights the unique demographic and clinical characteristics of our study population, emphasising the need for region-specific research to understand the diverse impacts of PPI use in different populations.

Despite these strengths, our study is not without limitations. As a retrospective cohort study, it inherently possesses certain constraints that may impact the validity and generalisability of its findings. Retrospective designs are particularly susceptible to confounding variables, and although efforts were made to adjust for known confounders, it is acknowledged that not all confounding factors can be accounted for in such a design. In this study, while we were able to establish exposure to PPIs using cDDD, we acknowledge that we were unable to accurately explore the effect of the different PPIs available in New Zealand on outcomes individually. Additionally, retrospective studies rely on the accuracy and completeness of existing records. In our study, we did not have access to community/general practitioner (GP) records for patients and as such were unable to accurately ascertain the indication for initiating PPIs and indication for dose changes that occurred in the community. We also were unable to capture any decompensation events that may have been managed in the community not requiring hospital admission. Furthermore, the retrospective nature of the study limits our ability to establish causality. While associations between PPI use and adverse outcomes can be identified, definitive clinical conclusions regarding the causative effects of PPIs cannot be drawn from this type of study. Prospective, randomised controlled trials are necessary to further elucidate these relationships and provide more conclusive evidence on the impact of PPI therapy in patients with liver cirrhosis.

PPIs are commonly prescribed for cirrhotic patients to manage complications such as gastro-oesophageal reflux disease and peptic ulcers, and to prevent gastrointestinal bleeding. However, in our study, a significant number of patients were prescribed PPIs for unspecified indications. This raises questions about the appropriateness of PPI use in this population and suggests a potential over-prescription issue. It is important to note that the majority of PPIs were initiated in the community by the patient's GP rather than in a hospital setting. This finding underscores the need for education and awareness among all practitioners and careful evaluation of PPI indications in cirrhotic patients to avoid unnecessary exposure to potential adverse effects.

Moreover, while we stated that patients who were prescribed PPIs had more severe liver disease, this aspect was not explored in depth. The greater severity of liver disease in the PPI group could be a confounding factor contributing to the higher incidence of adverse outcomes observed. Patients with advanced liver disease are more susceptible to infections and other complications, which might explain the increased risks associated with PPI use in this cohort. Future studies should aim to stratify patients based on the severity of liver disease to better understand the impact of PPIs in different sub-groups of cirrhotic patients.

Conclusions

In conclusion, our study showed that over 5 years in a large tertiary centre, PPI use in cirrhosis was associated with a significantly higher risk of hepatic decompensation and an increased risk of infection without a significant increase in mortality.

Given that only 32% of the PPI prescriptions met the recommended indications, there is a

strong case for more judicious use of PPIs in patients with cirrhosis. Emphasising the indication, duration of therapy and close monitoring for adverse effects are crucial steps. Further studies should focus on establishing clearer guidelines and investigating the pathological processes by which PPIs influence disease progression and infection risks in liver cirrhosis. These studies should also aim to delineate the safe use of PPIs in this vulnerable patient population.

COMPETING INTERESTS
Nil.

ACKNOWLEDGEMENTS
Christin Coomarasamy (biostatistician).

AUTHOR INFORMATION
Dr Abhimati Ravikulan: Registrar, Gastroenterology Department, Counties Manukau District Health Board, Christchurch, New Zealand.
Dr Natalie Russell: Registrar, Department of Medicine, Counties Manukau District Health Board, Auckland, New Zealand.
Christin Coomarasamy: Biostatistician, Counties Manukau District Health Board, Auckland, New Zealand.
Dr Ashok Raj: Gastroenterology Consultant, Gastroenterology Department, The Royal Melbourne Hospital, Melbourne, Australia.

CORRESPONDING AUTHOR
Dr Abhimati Ravikulan: Registrar, Gastroenterology Department, Counties Manukau District Health Board. E: arav175@aucklanduni.ac.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/proton-pump-inhibitors-in-cirrhosis-a-retrospective-five-year-analysis-of-increased-risks-of-hepatic-decompensation-and-infectio

REFERENCES
1. Li DK, Chung RT. Use of proton pump inhibitors in chronic liver diseases. Clin Liver Dis (Hoboken). 2018;10(6):148-151. doi: 10.1002/cld.678.
2. Zhu J, Yu H, Mancuso A, Qi X. Proton pump inhibitors in liver cirrhosis: a review of benefits and harms. AME Med J. 2017;2:36-36. doi: 10.21037/amj.2017.03.04
3. Dam G, Vilstrup H, Watson H, Jepsen P. Proton pump inhibitors as a risk factor for hepatic encephalopathy and spontaneous bacterial peritonitis in patients with cirrhosis with ascites. Hepatology. 2016;64(6):1265-72. doi: 10.1002/hep.28737.
4. Zhu J, Qi X, Yu H, et al. Association of proton pump inhibitors with the risk of hepatic encephalopathy during hospitalization for liver cirrhosis. United European Gastroenterol J. 2018;6(8):1179-1187. doi: 10.1177/2050640618773564.
5. Imhann F, Bonder MJ, Vich Vila A, et al. Proton pump inhibitors affect the gut microbiome. Gut. 2016;65(5):740-8. doi: 10.1136/gutjnl-2015-310376.
6. Freedberg DE, Toussaint NC, Chen SP, et al. Proton Pump Inhibitors Alter Specific Taxa in the Human Gastrointestinal Microbiome: A Crossover Trial. Gastroenterology. 2015;149(4):883-5.e9. doi: 10.1053/j.gastro.2015.06.043.
7. Wellhöner F, Döscher N, Tergast TL, et al. The impact of proton pump inhibitors on the intestinal microbiota in chronic hepatitis C patients. Scand J Gastroenterol. 2019;54(8):1033-1041. doi: 10.1080/00365521.2019.1647280.
8. Yamamoto K, Ishigami M, Honda T, et al. Influence of proton pump inhibitors on microbiota in chronic liver disease patients. Hepatol Int. 2019;13(2):234-244. doi: 10.1007/s12072-019-09932-9.
9. Khan MA, Kamal S, Khan S, et al. Systematic review and meta-analysis of the possible association between pharmacological gastric acid suppression and spontaneous bacterial peritonitis. Eur J Gastroenterol Hepatol. 2015;27(11):1327-36. doi: 10.1097/MEG.0000000000000448.
10. De Roza MA, Kai L, Kam JW, et al. Proton pump inhibitor use increases mortality and hepatic decompensation in liver cirrhosis. World J Gastroenterol. 2019;25(33):4933-4944. doi: 10.3748/wjg.v25.i33.4933.
11. Dultz G, Piiper A, Zeuzem S, et al. Proton pump inhibitor treatment is associated with the severity of liver disease and increased mortality in patients with cirrhosis. Aliment Pharmacol Ther. 2015;41(5):459-66. doi: 10.1111/apt.13061.
12. Cole HL, Pennycook S, Hayes PC. The impact of proton pump inhibitor therapy on patients with liver disease. Aliment Pharmacol Ther. 2016;44(11-12):1213-1223. doi: 10.1111/apt.13827.
13. Hung TH, Lee HF, Tseng CW, et al. Effect of proton pump inhibitors in hospitalization on mortality of patients with hepatic encephalopathy and cirrhosis but no active gastrointestinal bleeding. Clin Res Hepatol Gastroenterol. 2018;42(4):353-359. doi: 10.1016/j.clinre.2017.11.011.
14. China L, Tittanegro T, Crocombe D, et al. Investigating potential confounding by indication when considering the association between proton pump inhibitor use, infection, hepatic encephalopathy and mortality in hospitalised decompensated cirrhosis: a post-hoc analysis of the ATTIRE trial. EClinicalMedicine. 2023;58:101924. doi: 10.1016/j.eclinm.2023.101924.
15. Mahmud N, Serper M, Taddei TH, Kaplan DE. The Association Between Proton Pump Inhibitor Exposure and Key Liver-Related Outcomes in Patients With Cirrhosis: A Veterans Affairs Cohort Study. Gastroenterology. 2022;163(1):257-269.e6. doi: 10.1053/j.gastro.2022.03.052.

Applying Indigenous identity definitions in official health statistics: a case study using linked cancer registry data on stomach cancer

Nicole Satherley, Brandon de Graaf, Gabrielle Davie, Sheree Gibb, Andrea Teng, Andrew Sporle

ABSTRACT

AIM: Ethnicity and descent are two different officially recognised identity definitions for the Indigenous Māori population of New Zealand. Official health statistics are usually reported by Māori ethnicity but not descent, as health collections such as the New Zealand Cancer Registry (NZCR) do not record Māori descent information. We explored the potential of linked administrative data to describe health outcomes by Māori descent using gastric (stomach) cancer as an example.

METHODS: The Integrated Data Infrastructure (IDI) was used to source information on Māori descent from the 2013 and 2018 censuses as well as birth and death records linked to the NZCR for gastric cancer registrations for the years 1995–2021 (*N*=10,575).

RESULTS: Māori descent information could be sourced for 81.8% of gastric cancer registrations. Descent information was available for 65.2% of gastric cancer registrations in death records, 39.5% in the 2013 or 2018 census, 6.1% from a child's birth record and ≤0.3% from personal birth records. Of the registrations for whom Māori descent information could be obtained, 18.6% were identified as being of Māori descent vs 17.3% identified as Māori by ethnicity. Missing Māori descent data was lower (around 5%) in more recent gastric cancer registrations (2012 onwards).

CONCLUSION: Based on our case study, classifying cancer registrations by Māori descent for health outcome reporting, in addition to Māori ethnicity, may be feasible for recent years of data. Use of death records for Māori descent information should be carefully considered, as this may introduce bias to analyses such as survival analysis.

There are two conceptual approaches to defining Indigeneity in the New Zealand official statistics system—ethnic affiliation and Māori descent. Ethnicity and descent are two different concepts, with different theoretical foundations and legislative frameworks, as well as different applications by researchers, policy-makers and Māori organisations. The concepts define closely related yet distinct populations in New Zealand, who may experience different health outcomes.

Ethnicity is a measure of self-reported cultural affiliation with an ethnic group and is widely used in research to define populations. In New Zealand, the official ethnicity classification standard defines ethnic groups as consisting of people with some or all of a) a proper name, b) one or more elements of common culture, c) unique community interests, feelings and actions, d) a shared sense of common origins or ancestry, and e) a common geographic origin.[1]

Māori descent is based on biological ancestry and genealogy using definitions that are defined in legislation, including those that attribute specific constitutional or legal relationships. Numerous legislative acts, including the *Electoral Act 1993* and the *Māori Land Act 1993* (*Te Ture Whenua Māori Act 1993*), state *"'Māori' means a person of the Māori race of New Zealand; and includes any descendant of such a person."*[2,3] Māori descent is the concept that the Māori population use to construct identity, with the cultural concept of whakapapa (genealogy or descent) being foundational to individual and collective identities.[4] Providing information by descent in addition to ethnicity is also consistent with the United Nations' *Declaration on the Rights of Indigenous Peoples*, which outlines Indigenous peoples' right to determine their identity and that their cultures, traditions, histories and aspirations *"shall be appropriately reflected in education and public information."*[5] In New Zealand, the census is a key data source for Māori descent information, and census questions on iwi (tribal nation)

affiliation follow for those who indicate they are of Māori descent. Methods enabling analysis of outcomes by Māori descent may therefore also provide a precursor to examining outcomes by iwi affiliation.

The Māori descent population (19.6% of the total 2023 census usually resident New Zealand population) is larger than the Māori ethnicity population (17.8% of the total 2023 census usually resident population) in New Zealand, as not everyone of Māori descent identifies as Māori ethnically and vice versa. In the 2018 census (used as a data source alongside the 2013 census in the present study), 96% of the Māori population by ethnicity (775,836) also identified as being of Māori descent. However, a smaller percentage (86%) of the Māori descent population (869,850) also identified as being of Māori ethnicity.[6] In other words, about 14% of the total Māori descent population who do not identify as Māori ethnically is excluded when analysing outcomes by Māori ethnicity.

Although Māori ethnicity is very widely collected in New Zealand's official statistics, Māori descent is only collected in a few official statistical sources, namely the census, birth and death records and the Electoral Commission.[7] For this reason, social and health outcomes for Māori are routinely reported using the Māori ethnicity variable, and it is largely unknown how they might differ if reported on using Māori descent. However, there has been increasing use of Māori descent to define populations, especially in surveys focussed on or designed by Māori such as *Te Kupenga* and the Māori Disability Survey.[8,9] Defining the Māori population by *descent*, which is based on ancestry, is also relevant when studying health outcomes associated with genetic causes, as the concept of descent is more aligned (but still imperfectly) with genotype than the social affiliation-based concept of ethnicity.

This paper looks at how health outcomes can be reported by Māori descent using existing information within the official statistics system in New Zealand. We examine the feasibility of reporting on cancer by Māori descent, using gastric cancer as an example. In New Zealand approximately 400 people are diagnosed with gastric cancer per year.[10] There are large and persistent ethnic disparities in gastric cancer statistics in New Zealand,[11] with gastric cancer incidence and mortality rates being more than three times higher in Māori compared to non-Māori.[10] *Helicobacter pylori* (*H. pylori*) infection is the largest contributor of high gastric cancer prevalence among Māori, and is estimated to contribute to up to 61% of the excess cases among Māori compared to NZ European men.[12] However, *CDH1* gene mutation is also a significant contributor to advanced disease in the Māori population, being associated with an estimated 6% of advanced gastric cancers, and 13% of advanced diffuse-type gastric cancers among Māori.[13] There is also evidence of *H. pylori* infection being strongly associated with the expression of *CDH1* mutations in diffuse gastric cancer.[14]

We construct a Māori descent variable for gastric cancer registrations using linked administrative records in the Stats NZ Integrated Data Infrastructure (IDI). While Māori descent information is not recorded in the New Zealand Cancer Registry (NZCR), it is recorded in several linked administrative datasets available in the IDI; namely, the 2013 and 2018 census and Department of Internal Affairs (DIA) birth and death records. We document the percentage of the 1995–2021 gastric cancer registrations that can be linked to Māori descent information in the IDI, and where that information is located. We then compare the number of Māori descent gastric cancer registrations and the degree of overlap with the Māori ethnicity cancer registrations and consider the quality of the Māori descent information for potential future reporting on gastric cancer outcomes.

Method

Stats NZ's IDI

Stats NZ's IDI is a large database containing linked then de-identified individual-level microdata about people and households in New Zealand.[14] The IDI consists of a range of government and non-government administrative and survey data sources connected to a central "spine", including people who have ever been a resident in New Zealand. This enables analysis of populations of interest across government sectors (e.g., health, social services, housing, justice, education and training).[14] Data in the IDI is periodically updated throughout the year in distinct "refreshes". This study analysed data available in the June 2023 IDI refresh, using SAS Enterprise Guide version 7.1. Data was output from the IDI according to Stats NZ's confidentiality rules, which means random rounding to base 3 has been applied to all count data in this study and counts of less than 6 have been suppressed.

Gastric cancer registrations and Māori ethnicity in the NZCR

The NZCR is a population-based register of all primary malignant diseases diagnosed. The register collates details about patients and their cancer diagnosis, including sex, ethnicity, address, date of diagnosis and type of cancer, as well as information about cancer staging and grading.[15]

For this study, records on all gastric cancer registrations (i.e., ICD-10 codes C160 to C169) from 1 January 1995 to 31 December 2021 were available.

Māori ethnicity was sourced from the NZCR, which records up to three ethnicities per person. An ethnic identification is allocated by an algorithm process if it is present on at least 20% of a person's records across the National Health Index ([NHI] person-level health identifier) database, the Mortality Collection and the National Minimum Dataset of hospital discharges. The algorithm is periodically re-run to account for any new records across these sources.[16]

Māori descent in the IDI

Information on Māori descent in the IDI can be obtained from the 2013 and 2018 censuses and in New Zealand birth and death records (see **Table 1**).[7] Māori descent data held by the Electoral Commission and iwi organisations are not available within the IDI.[7] Māori descent information is taken from September 1995 onwards (with much improved coverage from 1998 when the collection of birth and death data was digitised) due to significant definitional issues of Māori descent and resulting missing records prior to 1995.[17]

Information about Māori descent for the individual, as well as their parents, was present on birth records. Where the descent information was missing for the individual's birth registration but was present for either one of the parents, the parental information was used. Descent information could also be sourced from the birth registrations of children of those with a cancer registration, based on an individual's record as parent 1 or parent 2 on the child's birth record.

Information on Māori descent from the different data sources was combined into one Māori descent variable. If a person had identified as being of Māori descent in at least one of the 2013 census, the 2018 census, New Zealand birth records or death records, they were classified as being of Māori descent.

Results

A total of 10,575 people were diagnosed with gastric cancer between 1995 and 2021 in New Zealand. Of these, 1,833 (17.3%) had Māori recorded as one of their ethnic group affiliations in the NZCR.

Figure 1 summarises the data obtained on Māori descent from IDI sources for the gastric

Table 1: Data sources in the IDI with Māori descent information, the specific questions and response categories.

Data source	Māori descent question	Response categories
New Zealand Census of Population and Dwellings (2013, 2018)	"Are you descended from a Māori (that is, did you have a Māori birth parent, grandparent or great-grandparent, etc.)?" *"He tūpuna Māori ōu?"*	Yes, No, Don't know Ae, Kaore, Aua
New Zealand birth registrations (from September 1995)	"Is this child/person the descendant of a New Zealand Māori?" "Is the mother/father the descendant of a New Zealand Māori?"	Yes, No, Not sure Yes, No, Not sure
NZ death registrations (from September 1995)	"Was the deceased descended from a New Zealand Māori?"	Yes, No, Don't know

For the 2018 census, 8.3% of Māori descent data was obtained from 2013 census responses and 2.2% from DIA birth records through admin enumeration, and a further 6.2% through imputation.[18]

Figure 1: Summary of data obtained on Māori descent information in the IDI for the gastric cancer registrations to construct a Māori descent variable. Note that S = suppressed count below 6.

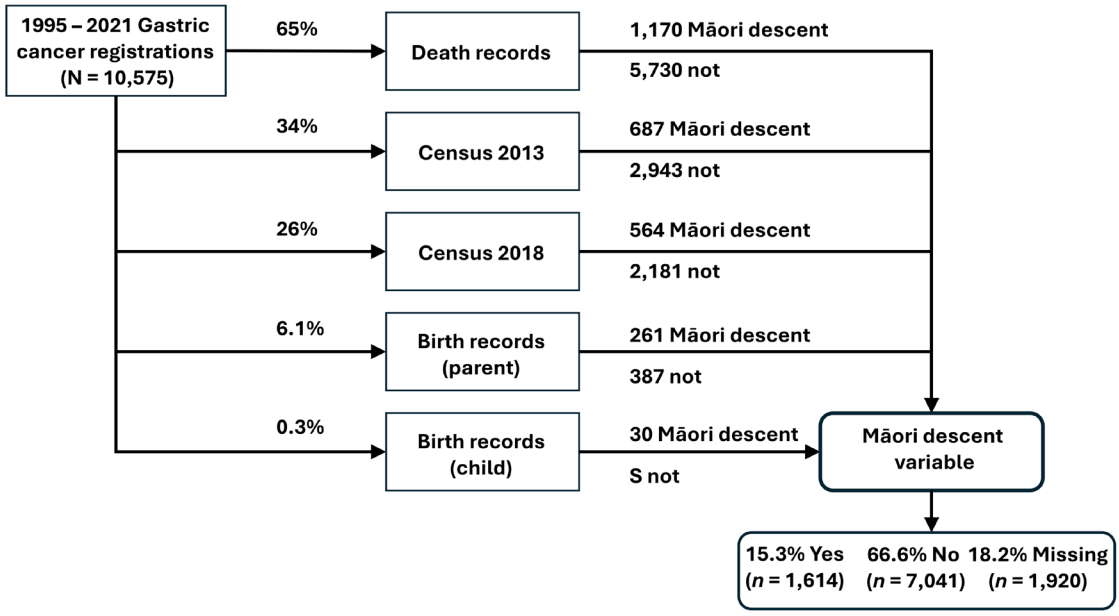


Table 2: Consistency in Māori descent information recorded in the 2013/2018 census and New Zealand death and birth records for gastric cancer registrations.

	New Zealand death records			New Zealand birth records		
Census 2013 or 2018	**No Māori descent**	**Māori descent**	**Total**	**No Māori descent**	**Māori descent**	**Total**
No Māori descent	1,983	24	2,007	261	S	264
Māori descent	60	405	465 (87.1%)	9	180	189 (95.2%)
Total	2,043	429 (94.4%)	2,472	267	186 (96.8%)	453

Row/column total percentages indicate the percent of row/column Māori descent gastric cases recorded as Māori descent in the comparison source.

cancer registrations. New Zealand death records contained information on Māori descent for 65.2% (6,900) of the gastric cancer registrations, representing the highest coverage among IDI sources. The 2013 census and 2018 census contained Māori descent information for 34.3% (3,630) and 26.0% (2,745) of the gastric cancer registrations respectively (and 4,179 or 39.5% of registrations combined). Only 0.3% (30) of the registrations had Māori descent information from their own birth record. However, 6.1% (648) had descent information from their child/children's birth registration (i.e., they were listed as parent 1 or parent 2 to a child). In total, combining information across these sources provided Māori descent information for 8,655 (81.8%) of gastric cancer registrations, of which 1,614 (18.6%) were recorded as being of Māori descent in at least one of New Zealand death records, 2013 or 2018 census, or New Zealand birth records.

Although the extent to which information on Māori descent for gastric cancer registrations was recorded in multiple sources was low, when it was recorded in two sources the level of

Table 3: Consistency in Māori descent information recorded in New Zealand birth and death records for gastric cancer registrations.

	New Zealand death records		
New Zealand birth records	**No Māori descent**	**Māori descent**	**Total**
No Māori descent	210	9	219
Māori descent	18	144	162 (88.9%)
Total	228	153 (94.1%)	381

Row/column total percentages indicate the percent of row/column Māori descent gastric cases recorded as Māori descent in the comparison source.

Table 4: Consistency in Māori descent information recorded in the 2013 census and 2018 census (with and without 2013 census enumerations included) for gastric cancer registrations.

	2018 census			2018 census (without 2013 census enumerations)		
2013 census	**No Māori descent**	**Māori descent**	**Total**	**No Māori descent**	**Māori descent**	**Total**
No Māori descent	1,743	9	1,752	1,557	9	1,566
Māori descent	15	426	441 (96.6%)	15	345	360 (95.8%)
Total	1,761	435 (97.9%)	2,196	1,572	354 (97.5%)	1,926

Row/column total percentages indicate the percent of row/column Māori descent gastric cases recorded as Māori descent in the comparison source.

Table 5: Cross tabulation of Māori ethnicity and Māori descent in the 1995–2021 gastric cancer registrations.

	No Māori descent	**Māori descent**	**Unknown/missing descent**	**Ethnicity Total**
Non-Māori ethnicity	6,915 (65.4%)	138 (1.3%)	1,617 (15.3%)	8,670 (82.1%)
Māori ethnicity	96 (1.0%)	1,476 (14.0%)	264 (2.5%)	1,833 (17.3%)
Unknown/missing ethnicity	30 (0.3%)	S (S%)	39 (0.4%)	69 (0.7%)
Descent Total	7,041 (66.6%)	1,614 (15.3%)	1,920 (18.2%)	10,566

Cell percentages calculated from the total number of gastric cancer registrations.

Figure 2: Percentage of total gastric cancer cases and those of Māori and non-Māori ethnicity missing Māori descent information for different diagnosis periods.

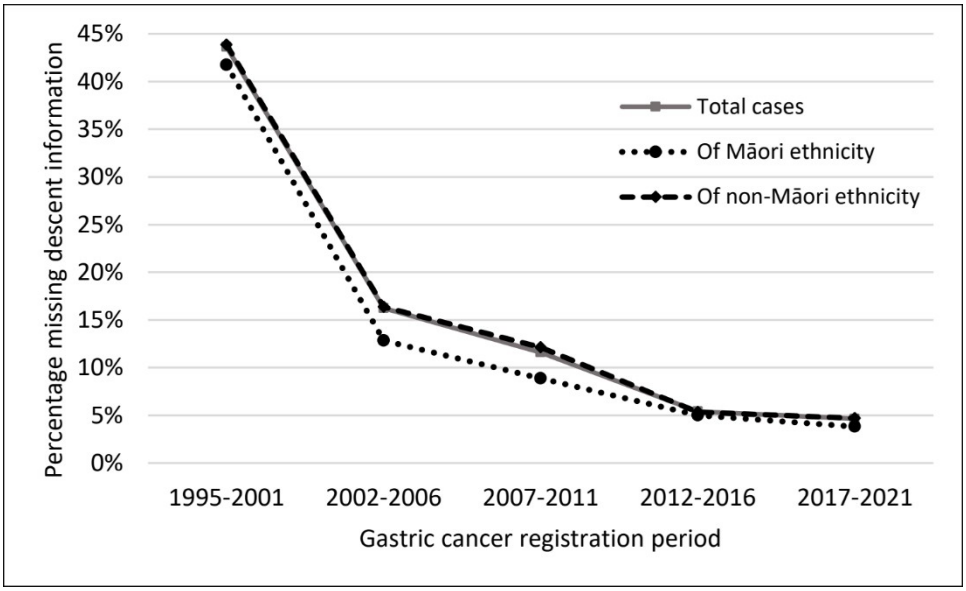
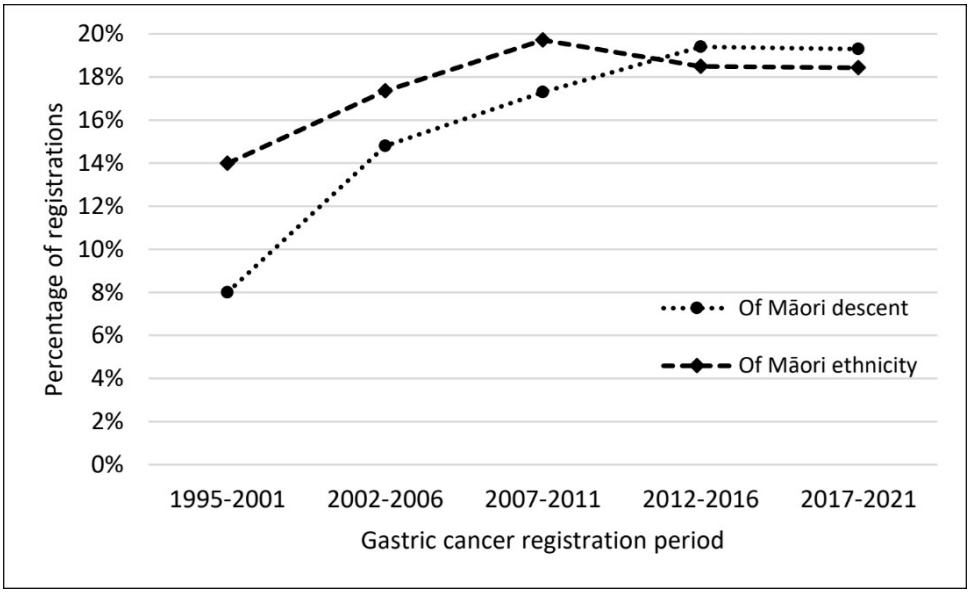


Figure 3: Percentage of total gastric cancer cases with recorded Māori ethnicity or Māori descent for different diagnosis periods.



agreement of the information recorded was high. As shown in Table 2 and Table 3, the percentage of gastric cancer registrations identified as being of Māori descent in a given source (New Zealand death records, New Zealand birth records, or the 2013/2018 census) who were also recorded as being of Māori descent in a corresponding source was between 87–98%. Consistency was generally highest with and between the 2013 and 2018 censuses, and lowest for New Zealand death records. Specifically, a lower percentage of individuals with gastric cancer registrations recorded as being of Māori descent in the 2013/2018 census (87%; Table 2) or New Zealand birth records (89%; Table 3) were also recorded as being of Māori descent in New Zealand death records.

For the 2018 census, missing descent records were obtained where necessary and possible from the 2013 census, which may increase the degree of alignment between these sources. We therefore also looked at alignment between the 2013 and 2018 censuses when excluding administrative data–sourced enumerations from the 2013 census for Māori descent in the 2018 census (but retained enumerations from other administrative data sources). As shown in Table 4, this made little difference to the consistency in descent information between the 2013 and 2018 censuses.

Comparing Māori descent and Māori ethnicity among those with complete data for both measures (see Table 5), a high proportion of cases (93.9%) of those with Māori ethnicity recorded in the cancer registry were also of Māori descent. A slightly lower percentage (91.4%) of the Māori descent gastric cancer cases were also of Māori ethnicity.

As shown in Figure 2, the percentage of missing Māori descent information among gastric cancer cases decreased in more recent diagnosis periods. Missing descent information was highest among those diagnosed from 1995–2001 (43.6%), but lower among those of Māori ethnicity, and noticeably lower among those diagnosed during 2012–2016 (5.4% of total cases) or 2017–2021 (4.6% of total cases). During these years, the number of cancer registrations of Māori descent was larger than the number of registrations by Māori ethnicity (see Figure 3).

Discussion

In total, information on Māori descent was found for 81.8% of the gastric cancer registrations (*N*=8,655). For registrations with available Māori descent information, 18.6% were recorded as being of Māori descent in one or more of New Zealand death records, 2013 or 2018 census, or New Zealand birth records. This is in line with what was found for the general population in the 2013 census (i.e., 17.5%) and the 2018 census (i.e., 18.5%).[6,19] However, because of missing descent data, only 15.3% of the total population gastric cancer registrations were identified as being of Māori descent, less than the percentage of Māori ethnicity (17.3%), and in contrast to the relatively smaller size of the Māori ethnic population compared with the Māori descent population.

Most of the information on Māori descent was retrieved from New Zealand death records, whereas comparatively less information about Māori descent was found in the 2013 or 2018 census and New Zealand birth records. There are several explanations for this finding. Gastric cancer is predominantly a disease affecting older ages,[10] meaning the vast majority of individuals with cancer registrations were born well before 1995 (when information on Māori descent was not routinely collected in a standardised format in New Zealand birth records). A major factor is the poor survival rate of gastric cancer in New Zealand,[20] meaning many cancer cases died prior to the 2013 and/or 2018 census. Even for those for whom census records were found, Māori descent was "not stated" or answered with "don't know" for 9.2% of the cases in the 2013 census and 22.9% of cases in the 2018 census. Furthermore, the 2013 and 2018 censuses both undercounted the Māori population (with a 4.2% net undercount of the descent population in 2018).[21,22] This means some individuals with cancer registrations may have simply been missed by the censuses.

The results indicate it would be problematic to explore gastric cancer rates and outcomes by Māori descent for all cancer registrations due to the amount of missing Māori descent data (18.2%). However, examining outcomes by descent is feasible among cases diagnosed from around 2012 onwards due to significant reductions in missing data from this point in time to around 5%. This is likely due to a combination of a) a higher proportion of the population being born after 1995 meaning greater coverage of descent information on birth records, and b) more cancer cases having census records (i.e., the 2013 and 2018 censuses). The proportion of the gastric cancer cases that could be identified as of Māori descent also exceeds the proportion identified as of Māori ethnicity from 2012, matching the relative sizes of these Indigenous groups in the total New Zealand population. However, a reliance on death records for Māori descent information poses issues. It may introduce bias in survival outcomes among cases of Māori descent, as surviving cases of Māori descent would be more likely to have missing Māori descent information, potentially resulting in inflated mortality rates. Death records could be removed as a source of Māori descent information for analyses where this would be an issue, although this will reduce the number of cases with Māori descent information.

There are other limitations with the methods used here to source Māori descent information. For example, Māori descent identification can vary between sources and over time based on factors such as knowledge of ancestry and

willingness to report one's descent in a particular context. However, information was combined here across sources regardless of the time the data was collected, relative to diagnosis date. Descent information can also be self-reported in some sources (e.g., censuses) but not others (e.g., birth and death records). Finally, it is also important to note that changes in methods for collecting descent information in some sources could lead to changes in incidence rates reported by Māori descent. For example, the 2023 census uses additional sources of data for administrative enumerations of Māori descent information (including electoral roll data),[23] which could lead to increases in the size of the Māori descent due to a combination of both improved population coverage and population growth.

This research demonstrates the utility of the IDI for applying different Indigenous identity definitions in official health statistics. As we made use of all available Māori descent data sources in the IDI at the June 2023 refresh, the methods could be applied to other administrative datasets to examine other outcomes by Māori descent as an additional method of reporting alongside ethnicity. Routinely collected Māori descent information in healthcare data itself, such as through NHI numbers, could reduce potential issues and sources of bias when joining descent information across different data collections (e.g., mortality records). The issues identified here may apply to other outcomes and should be considered when designing or assessing any research focussed on health or social outcomes for the Māori descent population.

COMPETING INTERESTS
This project was approved by the University of Otago Human Ethics Committee (HD18/064).
This research was funded by Health Research Council (HRC) programme grant 17-610 led by Professor Parry Guilford, University of Otago.
AT is funded by the Health Research Council, Symbiotic Programme, to co-lead a survey of *H. pylori* in Aotearoa New Zealand, with the aim of supporting gastric cancer prevention.
SG has received grants from the University of Otago, University of Otago Research Grant, and MBIE, Healthier Lives National Science Challenge, as Principal Investigator.
Disclaimer: Access to the data used in this article was provided by Stats NZ under conditions designed to give effect to the security and confidentiality provisions of the *Data and Statistics Act 2022*. The results presented in this report are the work of the authors, not Stats NZ or individual data suppliers. These results are not official statistics. They have been created for research purposes from the Integrated Data Infrastructure (IDI) which is carefully managed by Stats NZ. For more information about the IDI please visit https://www.stats.govt.nz/integrated-data/
Data in this article have been reported in accordance with Stats NZ's confidentiality rules for microdata use, and as such random rounding to the base 3 has been applied to all count data and counts of 5 or less have been suppressed (S).

ACKNOWLEDGEMENTS
The authors would like to thank Frederieke S Petrović-van der Deen for her data exploration and analysis work in the early stages of the project.

AUTHOR INFORMATION
Nicole Satherley: iNZight Analytics Ltd, Department of Statistics, The University of Auckland.
Brandon de Graaf: Department of Preventive and Social Medicine, University of Otago.
Gabrielle Davie: Department of Preventive and Social Medicine, University of Otago.
Sheree Gibb: Department of Public Health, University of Otago.
Andrea Teng: Department of Public Health, University of Otago.
Andrew Sporle: iNZight Analytics Ltd, Department of Statistics, The University of Auckland.

CORRESPONDING AUTHOR
Andrew Sporle: iNZight Analytics, Auckland, New Zealand. E: andrew@inzight.co.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/applying-indigenous-identity-definitions-in-official-health-statistics-a-case-study-using-linked-cancer-registry-data-on-stomach

REFERENCES
1. Stats NZ. Ethnicity [Internet]. Wellington (NZ): Stats NZ; [date unknown] [cited 2024 May 3]. Available from: https://www.stats.govt.nz/topics/ethnicity
2. *Electoral Act 1993* (NZ) s 3.
3. *Te Ture Whenua Māori Act 1993* (NZ) s 4.
4. Kukutai T. The problem of defining an ethnic group for public policy: Who is Māori and why does it matter. Soc Policy J N Zeal. 2004;23(23):86-108.
5. United Nations. United Nations Declaration On The Rights of Indigenous Peoples [Internet]. Geneva (CH): United Nations; 2007 [cited 2024 Apr 20]. Available from: https://social.desa.un.org/issues/indigenous-peoples/united-nations-declaration-on-the-rights-of-indigenous-peoples
6. Stats NZ. Māori descent indicator, Māori ethnicity indicator, age, and gender for the census usually resident population count, (RC, TALB, SA2, Health), 2013, 2018, and 2023 Censuses [Internet]. Wellington (NZ): Stats NZ; [date unknown] [cited 2025 Apr 1]. Available from: https://www.stats.govt.nz/tools/aotearoa-data-explorer/
7. Bycroft C, Reid G, McNally J, Gleisner F. Identifying Māori populations using administrative data: A comparison with the census. Wellington (NZ): Stats NZ; 2016 [cited 2024 Jun 1]. Available from: https://www.stats.govt.nz/assets/Research/Identifying-Maori-populations-using-administrative-data-A-comparison-with-the-census/Identifying-Maori-populations-using-administrative-data-a-comparison-with-the-census.pdf
8. Kukutai T, Sporle A, Roskruge M. Subjective whānau wellbeing in Te Kupenga [Internet]. Wellington (NZ): Social Policy Research and Evaluation Unit; 2017 [cited 2024 Mar 20]. Available from: https://thehub.sia.govt.nz/assets/Uploads/Subjective-whanau-wellbeing-report.pdf
9. Ingham TR, Jones B, Perry M, et al, Te Ao Mārama Study Group. Measuring Māori Health, Wellbeing, and Disability in Aotearoa Using a Web-Based Survey Methodology. Int J Environ Res Public Health. 2023;20(18):6797. doi: 10.3390/ijerph20186797.
10. Ministry of Health – Manatū Hauora. Cancer: New registrations and deaths 2013 [Internet]. Wellington (NZ): Ministry of Health – Manatū Hauora; 2016 [cited 2023 Dec 10]. Available

from: https://www.health.govt.nz/publications/cancer-new-registrations-and-deaths-2013
11. Teng AM, Atkinson J, Disney G, et al. Ethnic inequalities in cancer incidence and mortality: census-linked cohort studies with 87 million years of person-time follow-up. BMC Cancer. 2016;16(1):755. doi: 10.1186/s12885-016-2781-4.
12. Teng AM, Blakely T, Baker MG, Sarfati D. The contribution of *Helicobacter pylori* to excess gastric cancer in Indigenous and Pacific men: a birth cohort estimate. Gastric Cancer. 2017;20(4):752-55. doi: 10.1007/s10120-016-0671-8.
13. Hakkaart C, Ellison-Loschmann L, Day R, et al. Germline *CDH1* mutations are a significant contributor to the high frequency of early-onset diffuse gastric cancer cases in New Zealand Māori. Fam Cancer. 2019;18(1):83-90. doi: 10.1007/s10689-018-0080-8.
14. Milne BJ, Atkinson J, Blakely T, et al. Data Resource Profile: The New Zealand Integrated Data Infrastructure (IDI). Int J Epidemiol. 2019;48(3):677-677e. doi: 10.1093/ije/dyz014. Erratum in: Int J Epidemiol. 2019;48(3):1027. doi: 10.1093/ije/dyz054.
15. Health New Zealand – Te Whatu Ora. Table of available data [Internet]. Wellington (NZ): Health New Zealand – Te Whatu Ora; 2024 [cited 2025 Apr 1]. Available from https://www.tewhatuora.govt.nz/for-health-professionals/clinical-guidance/diseases-and-conditions/cancer/new-zealand-cancer-registry-nzcr/new-zealand-cancer-registry-table-of-available-data
16. Health New Zealand – Te Whatu Ora. Technical details [Internet]. Wellington (NZ): Health New Zealand – Te Whatu Ora; 2024 [cited 2024 May 15]. Available from: https://www.tewhatuora.govt.nz/for-health-professionals/clinical-guidance/diseases-and-conditions/cancer/new-zealand-cancer-registry-nzcr/new-zealand-cancer-registry-technical-details
17. Stats NZ. IDI Data Dictionary: Life event data (July 2015 edition). Wellington (NZ): Stats NZ; 2015.
18. Stats NZ. Data sources, editing, and imputation in the 2018 Census [Internet]. Wellington (NZ): Stats NZ; 2019 [cited 2024 Jun 5]. Available from https://www.stats.govt.nz/methods/data-sources-editing-and-imputation-in-the-2018-census/
19. Stats NZ. 2013 Census QuickStats about Māori [Internet]. Wellington (NZ): Stats NZ; 2013 [cited 2024 Jun 20]. Available from: https://www.stats.govt.nz/reports/2013-census-quickstats-about-maori/
20. Sarfati D, Gurney J, Stanley J, Koea J. A retrospective cohort study of patients with stomach and liver cancers: the impact of comorbidity and ethnicity on cancer care and outcomes. BMC Cancer. 2014;14(1):821.
21. Stats NZ. Māori population under-estimation in 2013: Analysis and findings [Internet]. Wellington (NZ): Stats NZ; 2022 [cited 2024 May 3]. Available from: https://www.stats.govt.nz/reports/maori-population-under-estimation-in-2013-analysis-and-findings/
22. Stats NZ. Post-enumeration survey: 2018 [Internet]. Wellington (NZ): Stats NZ; 2020 [cited 2024 May 3]. Available from: https://www.stats.govt.nz/information-releases/post-enumeration-survey-2018
23. Stats NZ. Data sources and imputation for Māori descent in the 2023 Census [Internet]. Wellington (NZ): Stats NZ; 2024 [cited 2025 Jan 29]. Available from: https://www.stats.govt.nz/methods/data-sources-and-imputation-for-maori-descent-in-the-2023-census/

Cancers potentially attributable to excess body weight in Aotearoa New Zealand from 2019 to 2023

Michael Walsh, Jennifer Brenton-Peters, Olivia Perelini, Karen Bartholomew

ABSTRACT

AIM: This study quantifies the incidence of cancers attributable to excess body weight (EBW) in Aotearoa New Zealand adults aged 30+ from 2019 to 2023 and assesses public health implications.

METHODS: Relative risk estimates from an existing review and EBW prevalence from the New Zealand Health Survey were used to calculate population attributable fractions (PAFs) for 12 cancer types. PAFs were applied to Cancer Registry data to estimate EBW-attributable cases. Confidence intervals were calculated using bootstrap techniques. Two scenarios explored the potential impact of reducing EBW prevalence.

RESULTS: An estimated 6,962 cancers (5.1% of all cases) were potentially attributable to EBW, averaging 1,390 cases annually. The impact was greater for females (PAF 6.3%) than males (PAF 4.1%). Among Māori, 6.9% of cancers (221 per year) were attributed to EBW, while Pacific peoples had a higher PAF of 11.8% (145 cases per year). PAFs were highest for Pacific females (16.1%, 110 per year). Modelling suggests halving EBW prevalence could potentially prevent 600 cases annually.

CONCLUSION: EBW contributes to a large number of cancers in New Zealand, compounding health inequities, particularly for Māori and Pacific peoples. These inequities highlight the urgent need for multisectoral, collaborative interventions that address the complex, inequitable drivers of EBW. Public health must strengthen its pro-equity, anti-stigmatising approach to prevention, management and treatment. However, sustained reductions in EBW-related cancers will ultimately depend on preventing EBW rather than relying on treatment-based interventions.

People can be healthy across a spectrum of diverse body sizes, emphasising the complex and multifaceted nature of body size beyond Western-defined norms.[1,2] The language, framing and terminology around body weight are important due to the known impacts of weight bias and stigma on healthcare access and quality, and professional interactions.[3,4] Recognising that excess body weight (EBW) is a risk factor for several diseases, and that weight stigma influences health outcomes, is an important consideration for public health practitioners that underscores the need for a nuanced understanding of the interplay between body size and health.[5]

EBW is defined using body mass index (BMI) categories. While widely employed, BMI has well-documented limitations, as it is based solely on height and weight and does not account for individual variations in health risk. Its applicability across different ethnic groups has also been critiqued.[6] Despite these limitations, BMI is a useful population-level measure for assessing health risk attribution.[7] BMI is calculated as weight in kilograms divided by the square of height in meters (kg/m^2), with the World Health Organization classifying adult BMI as follows: normal weight (18.50–24.99kg/m^2), overweight (25.00–29.99kg/m^2) and obese (≥30kg/m^2).[7] In this paper, we use the World Health Organization BMI classifications to define EBW.

When attributing health risks to EBW, it is essential to consider the whole person, including their broader wellbeing, lifestyle, whānau, social and cultural context and disabilities. As BMI does not capture these complexities, acknowledging the multifactorial drivers of EBW is crucial. Nevertheless, BMI remains a practical and cost-effective tool for tracking population-level weight trends, facilitating comparisons across groups and time periods, and informing public health monitoring and policy development.[8]

For this paper, we have adopted the term "excess body weight" over "obesity", informed by critical public health perspectives, equity-focussed frameworks, Indigenous lived experiences and fat studies literature, which challenge the medicalisation and stigma attached to body weight.[9–11] This approach reflects a commitment

to destigmatisation and equity, recognising intersecting biases and health inequities experienced by people with EBW related to gender, ethnicity, socio-economic status, geography and other social determinants.[11–13] Our approach is also aligned to Indigenous and collective perspectives recognising that numerical or clinical criteria do not solely define health—rather, health is deeply rooted in cultural, historical and social contexts. We draw on multiple perspectives to support a balance between achieving public health objectives and mitigating weight-related stigma, thereby promoting a more inclusive and respectful dialogue around health outcomes. Striking this balance remains a challenging yet necessary consideration.

Aotearoa New Zealand has one of the highest rates of people living with EBW in the Organisation for Economic Co-operation and Development (OECD),[14] disproportionately impacting Māori and Pacific peoples, with 48% of Māori and 65% of Pacific adults living with EBW.[15] New Zealand has high rates of children living with EBW, at one point ranking as the third highest among OECD countries.[16] This trend raises concerns about subsequent cancer risk, as high childhood EBW rates would likely contribute to increased EBW prevalence in adulthood.[17]

Numerous complex and interacting factors are related to body weight, such as a person's genetics, biology and socio-ecological factors.[7] Other key drivers include health-disrupting environments that provide easy availability of cheap, energy-dense foods, combined with persuasive and pervasive food marketing and reduced opportunities for physical activity.[18,19] This has led to what is often termed an "obesogenic" environment, whereby making a healthy choice has become increasingly difficult and expensive, especially for vulnerable populations living in lower socio-economic areas.[20] These factors contribute to a socio-economic gradient, with those in lower socio-economic groups being more likely to experience EBW.[12]

From a public health perspective, EBW is categorised as both a potentially modifiable and socially mediated risk factor and is associated with various non-communicable diseases, including type 2 diabetes and cardiovascular disease. There is sufficient evidence for a causal association with at least 12 types of cancer.[21–23] These cancers include breast (postmenopausal), colorectal, uterine, ovary, pancreas, kidney, gallbladder, stomach (cardia), liver, oesophagus (adenocarcinoma), thyroid and multiple myeloma. More recently, probable evidence of a causal association has emerged for advanced prostate cancer.[24] The causal link of EBW to cancer risk is supported by evidence from numerous epidemiological studies demonstrating a robust dose–response relationship in the association, as well as experimental studies proposing multiple biological mechanisms.[21]

Systemic challenges that contribute to ethnic-specific inequities in cancer outcomes in New Zealand are well reported.[25–27] Factors such as barriers to primary care, diagnostic delay and the lack of culturally responsive care, alongside the need for systemic improvements in healthcare delivery, have been recognised.[28] Māori face excess mortality rates, influenced by comorbidities and systemic barriers, including inequities within the health system.[29–31] The recent rise in endometrial cancer incidence, with a 59% increase in cases over the past decade, calls for focussed attention, especially as Pacific communities experience substantial impacts. This trend also affects other ethnic groups, with an increase in younger age groups, underscoring the importance of comprehensive strategies to address these health challenges effectively.[32]

In this paper we firstly aim to assess the cancer burden associated with EBW in New Zealand adults aged 30 years and older from 2019 to 2023. Second, we seek to draw attention to language and approach in public health with respect to EBW and how health organisations should be prioritising the creation of safe, accessible healthcare and supportive environments. This includes enhancing healthcare accessibility and quality for all, particularly for minoritised communities, and ensuring that interventions are inclusive and respectful of all body types.

Method

Ethnicity

Ethnicity data used were as recorded in the New Zealand Cancer Registry, following the Ethnicity Data Protocols set out in Health Information Standards Organisation 10001:2017.[33] These protocols ensure standardised collection and reporting of ethnicity across the health system, with prioritised ethnicity applied—where individuals with multiple ethnic affiliations are assigned to a single group based on a set hierarchy, typically prioritising Māori and Pacific peoples. While this method aids in focussed analysis of specific ethnic groups, it carries certain limitations, including the potential for ethnicity misclassification and undercounting.[34] Misclassification can occur

when individuals' self-identified ethnicities are inaccurately recorded, leading to potential biases in health outcome analyses. Additionally, the use of prioritised ethnicity may mask the presence of smaller ethnic groups and undercount individuals with multiple ethnic affiliations. These limitations can affect the accuracy of ethnic health equity assessments, particularly when examining outcomes for Māori and Pacific populations.

Sex

Sex classification was based on data recorded in the New Zealand Cancer Registry, which follows the sex recorded in the patient's National Health Index at the time of assignment or registration. This method ensures consistency across health datasets and facilitates comparison with other health outcomes. However, the reliance on sex assigned at birth may not capture gender identity or the experiences of individuals whose gender does not align with their assigned sex. While this approach is necessary for ensuring data comparability, it presents limitations when considering broader aspects of gender diversity in health outcomes.

Age

The analysis was limited to cancers among those aged 30 years or older as the evidence for cancers associated with EBW in those aged younger than 30 years is limited.[21] An exception was postmenopausal breast cancer, where an age cutoff of 50 was used.

Relative risk estimates

The estimates of relative risk associated with EBW relative to normal weight for the 12 cancers (Table 1 and Table 2) were obtained from Brown et al.,[35] a systematic analysis of modifiable cancer risk factors using meta-analyses and cohort studies. While the study provides estimates for the United Kingdom, the fundamental biological mechanisms linking EBW to cancer are well established and likely applicable across populations. Given New Zealand's high prevalence of EBW and the lack of large-scale New Zealand-specific relative risk estimates, these findings offer a robust evidence base for assessing EBW-related cancers in New Zealand. Where available, sex specific estimates by cancer type were used.

Table 1: Cancer types associated with EBW and corresponding relative risk (male).

		Relative risk	
Cancer type	**ICD-10 codes**	**25.00–29.99kg/m²**	**≥30kg/m²**
Colorectal	C18–C20, C26.0	1.17	1.38
Oesophageal	C15 (adenocarcinoma only)[a]	1.87	2.73
Stomach	C16.0 (cardia only)	1.22	1.61
Liver	C22.0, C22.2–C22.4, C22.7, C22.9	1.18	1.83
Gallbladder	C23	1.00	1.54
Pancreas	C25	1.15	1.20
Kidney and renal	C64–C65	1.22	1.63
Thyroid	C73	1.10	1.27
Multiple myeloma	C90.0, C90.2[b]	1.17	1.23

[a] Morphology codes for oesophageal adenocarcinoma includes codes 8140–8147, 8160–8162, 8180–8221, 8250–8507, 8514, 8520–8551, 8560, 8570–8574, 8576 and 8940–8941.
[b] Morphology codes for multiple myeloma are 9731–9732 and 9734.
EBW = excess body weight.

Table 2: Cancer types associated with EBW and corresponding relative risk (female).

Cancer type	ICD-10 codes	Relative risk	
		25.00–29.99kg/m²	≥30kg/m²
Colorectal	C18–C20, C26.0	1.07	1.17
Oesophageal	C15 (adenocarcinoma only)[a]	1.87	2.73
Stomach	C16.0 (cardia only)	1.22	1.61
Liver	C22.0, C22.2–C22.4, C22.7, C22.9	1.18	1.83
Gallbladder	C23	1.22	1.75
Pancreas	C25	1.12	1.15
Breast (female postmenopausal)	C50 (postmenopausal only)[b]	1.13	1.20
Uterine	C54–C55	1.34	2.54
Ovarian	C56	1.08	1.11
Kidney and renal	C64–C65	1.38	1.95
Thyroid	C73	1.10	1.27
Multiple myeloma	C90.0, C90.2[c]	1.12	1.15

[a] Morphology codes for oesophageal adenocarcinoma includes codes 8140–8147, 8160–8162, 8180–8221, 8250–8507, 8514, 8520–8551, 8560, 8570–8574, 8576 and 8940–8941.
[b] Fifty years and over was used as a menopausal cutoff.
[c] Morphology codes for multiple myeloma are 9731–9732 and 9734.
EBW = excess body weight.

Exposure prevalence estimates

Annual sex- and ethnic-specific crude prevalence data from the New Zealand Health Survey from 2006/2007 were used as our estimate of EBW.[36] This provided a greater-than-10-year lag period between risk exposure and cancer incidence. Two classifications of EBW were used that aligned with different estimates of risk: a BMI of 25.00–29.99kg/m² and a BMI of ≥30kg/m².[7]

Cancer data

We used anonymised data from the New Zealand Cancer Registry for the period 2019–2023. It is a population-based register of all primary malignant diseases diagnosed in New Zealand, excluding squamous and basal cell skin cancers.

Statistical analysis

The population attributable fraction (PAF) for each sex- and ethnic-specific group utilising two levels of EBW was calculated for each cancer using the standard formula:

$$PAF = \frac{Po(Ro - 1) + Pw(Rw - 1)}{1 + Po(Ro - 1) + Pw(Rw - 1)}$$

PAF is the attributable proportion for each cancer attributable to EBW.

Pw is the proportion of the population who have a BMI between 25.00 and 29.99kg/m².

Po is the proportion of the population who

have a BMI ≥30kg/m^2.

Rw is the cancer-specific relative risk for a BMI between 25.00 and 29.99kg/m^2.

Ro is the cancer-specific relative risk for a BMI ≥30kg/m^2.

The sex- and ethnic-specific PAF for each cancer were multiplied by the number of primary cancer registrations in the population from each respective cancer to obtain cancer-specific EBW attributable estimates. For each cancer, EBW-attributable estimates were calculated separately for males and females and for each of the Māori, Pacific, Asian and European/Other ethnic groups. The proportion of EBW-attributable cancers was summed to generate the total number of cancers attributable to EBW.

Recognising the uncertainties in both relative risk and EBW prevalence estimates, we applied a parametric bootstrap technique, incorporating the 95% confidence intervals (CIs) of these parameters, to generate robust CIs for each cancer-specific PAF as well as the aggregated PAFs. We executed 10,000 bootstrap iterations, in which each iteration involved random selection of relative risk and EBW prevalence estimates within their respective CIs. This selection informed the recalculation of PAFs for each cancer type. The PAFs were then weighted by the incidence of each cancer to estimate the total number of cases attributable to EBW for that iteration. The 95% CI for the overall EBW-related cancer burden was derived from the 2.5th and 97.5th percentiles of the aggregated PAFs across all bootstrap samples.

Estimating cancers with a reduction in EBW

Two scenarios are modelled to assess the potential impact of reducing the prevalence of EBW on EBW-attributable cancers. This modelling can be thought of as the number of cancers that could potentially be prevented in a single year at the given prevalence of EBW. Scenario one proposes a reduction in the population prevalence with a BMI of 30kg/m^2 or more by 50%, with the assumption that individuals from this category move into the BMI range of 25.00–29.99kg/m^2. Scenario two envisions a more comprehensive reduction, halving the prevalence of EBW across both BMI categories (BMI ≥30kg/m^2 and BMI 25.00–29.99kg/m^2).

Ethical approval

The study was considered low risk as it only used deidentified data and received locality authorisation approval from the Health New Zealand – Waitematā District, Research & Knowledge Centre (Approval Code: WAI20249).

Results

Total number of cancers attributable to EBW

Between 2019 and 2023, 5.1% (95% CI 4.7–5.5) of all cancers in New Zealand among those aged 30 and above were attributed to EBW, amounting to 6,962 cancers (Table 3) or around 1,390 per year on average. The proportion of EBW-related cancers was higher in females at 6.3% (95% CI 5.7–6.9) with 4,010 cancers (802 per year) compared with males at 4.1% (95% CI 3.6–4.6) with 2,952 cancers (590 per year). The cancer types with the highest totals attributable to EBW (Table 5) were colorectal, with 1,801 cases (360 per year) and a PAF of 10.5% (95% CI 8.0–13.1), uterine cancer with 1,331 cases and a PAF of 36.7% (95% CI 34.3–39.2) and breast (in females 50+ only) with 1,231 cases (266 per year) and a PAF of 8.8% (95% CI 6.8–10.7). Oesophageal adenocarcinoma, although less frequent, had the highest PAF (Table 4) at 44.2% (95% CI 41.6–46.9), leading to 454 cases (91 per year).

Among Māori, the overall PAF was 6.9% (95% CI 6.3–7.5), translating to 1,103 cancers (221 per year) attributable to EBW. The PAF for Māori females was 8.2% (95% CI 7.2–9.2), accounting for 705 cancers (141 per year), while Māori males had a PAF of 5.4% (95% CI 4.8–6.0), translating to 398 cancers (79 per year). Among specific cancers, the highest PAF was observed in oesophageal adenocarcinoma, which had a PAF of 50.0% (95% CI 46.7–53.2), followed by uterine cancer with a PAF of 42.7% (95% CI 38.5–47.0). High PAFs were also observed for kidney and renal pelvis cancer (28.3%, 95% CI 25.1–31.6) and liver cancer (28.6%, 95% CI 24.9–32.4).

In the Pacific population, the total PAF was 11.8% (95% CI 10.9–12.7), accounting for 723 cancers (145 per year) attributable to EBW. Females showed a higher PAF of 16.1% (95% CI 14.6–17.6), translating to 550 cancers (110 per year), compared with males at 6.4% (95% CI 5.7–7.1), with 172 cancers (34 per year). Uterine cancer had the most substantial attributable burden with a PAF of 51.5% (95% CI 47.0–56.1) and 329 cancers (66 per year), accounting for more than half of the overall EBW cancer burden in Pacific females. Breast cancer in Pacific females over 50 had a PAF of 13.6% (95% CI 8.6–18.6), leading

to 103 cancers, while colorectal cancer had a PAF of 17.0% (95% CI 12.9–21.3), with 89 cancers.

Among the Asian population, the total PAF was 3.7% (95% CI 3.2–4.1), accounting for 277 cancers (55 per year) attributable to EBW. Female cancers were more prevalent with a PAF of 4.2% (95% CI 3.6–4.7), accounting for 177 cancers (35 per year), compared with males at 3.0% (95% CI 2.4–3.7), with 100 cancers (20 per year). Uterine cancer had the highest number of EBW-attributable cancers with 67 (13 per year) and a PAF of 21.4% (95% CI 17.6–25.3).

For the European and Other populations, the total PAF was 4.5% (95% CI 4.0–5.0), corresponding to 4,859 cancers (972 per year) attributable to EBW. Females had a PAF of 5.4% (95% CI 4.6–6.2), leading to 2,577 cancers (515 per year), while males had a lower PAF of 3.8% (95% CI 3.2–4.4), contributing to 2,282 cases (456 per year). Colorectal cancer had the highest number of EBW-attributable cancers with 1,464 (293 per year) and a PAF of 10.3% (95% CI 7.2–13.3), followed by breast cancer in females over 50 with 865 cancers (173 per year) and a PAF of 8.3% (95%

Table 3: Proportion and count of cancers attributable to EBW by cancer type and ethnic group (2019–2023).

Ethnic group and sex	PAF % (95% CI)	Number of cancers attributable to EBW—5-year (95% CI)	Average per year (95% CI)
Māori			
Male	5.4 (4.8–6.0)	398 (355–442)	80 (75–84)
Female	8.2 (7.2–9.2)	705 (623–789)	141 (131–151)
Total	6.9 (6.3–7.5)	1,103 (1,010–1,199)	221 (207–234)
Pacific			
Male	6.4 (5.7–7.1)	172 (153–191)	34 (32–37)
Female	16.1 (14.6–17.6)	550 (500–601)	110 (100–12)
Total	11.8 (10.9–12.7)	723 (669–777)	145 (132–157)
European/Other			
Male	3.8 (3.2–4.4)	2,282 (1,926–2,643)	456 (446–467)
Female	5.4 (4.6–6.3)	2,577 (2,184–2,974)	515 (503–528)
Total	4.5 (4.0–5.0)	4,859 (4,326–5,390)	972 (957–986)
Asian			
Male	3.0 (2.4–3.7)	100 (79–122)	20 (18–22)
Female	4.2 (3.6–4.7)	177 (153–202)	35 (30–41)
Total	3.7 (3.2–4.1)	277 (245–310)	55 (49–62)
Overall			
Male	4.1 (3.6–4.6)	2,952 (2,591–3,318)	590 (573–608)
Female	6.3 (5.6–6.9)	4,010 (3,601–4,419)	802 (770–834)
Total	5.1 (4.1–5.5)	6,962 (6,422–7,508)	1,392 (1351–1433)

EBW = excess body weight; PAF = population attributable fraction; 95% CI = 95% confidence interval.

Table 4: Fraction of cancers attributable to EBW between 2019 and 2023 by cancer type and ethnic group.

Cancer type	PAF % (95% CI)				
	Māori	Pacific	Asian	European and Other	Total
Breast[a]	10.9 (7.3–14.4)	13.6 (8.6–18.4)	5.3 (3.6–6.9)	8.3 (5.8–10.9)	8.8 (6.8–10.7)
Colorectal	13.3 (9.9–16.8)	17.0 (12.7–21.3)	6.5 (4.0–9.0)	10.3 (7.3–13.3)	10.5 (8.0–13.1)
Oesophageal[b]	50.0 (46.8–53.2)	57.0 (53.5–60.5)	32.9 (28.3–37.5)	43.7 (40.8–46.7)	44.2 (41.7–46.9)
Gallbladder	24.7 (21.4–28.2)	31.2 (27.3–35.2)	9.8 (7.7–11.9)	17.6 (14.8–20.4)	20.2 (18.3–22.1)
Kidney and renal pelvis	28.3 (25.0–31.6)	35.0 (31.3–38.7)	14.1 (11.2–17.0)	21.8 (18.6–25)	22.8 (20.4–25.3)
Liver	28.6 (25.0–32.3)	36.4 (32.4–40.4)	12.7 (9.6–15.9)	21.4 (18.0–24.8)	23.6 (21.5–25.8)
Multiple myeloma	11.2 (7.7–14.6)	13.6 (9.3–18.0)	6.4 (3.9–9.0)	9.7 (6.4–12.8)	9.9 (7.4–12.5)
Ovary	6.5 (1.7–11.3)	8.1 (1.7–14.5)	3.2 (0.3–6.0)	5.0 (0.8–9)	5.3 (2.2–8.2)
Pancreas	10.3 (7.2–13.5)	12.6 (8.5–16.7)	5.8 (3.5–8.1)	8.7 (5.9–11.4)	8.9 (6.7–11.0)
Stomach[c]	24.4 (20.9–27.9)	30.7 (26.8–34.4)	12.1 (8.8–15.3)	19.1 (15.7–22.5)	19.6 (16.7–22.5)
Thyroid	12.5 (8.8–16.2)	16.4 (11.8–20.9)	5.5 (3.2–7.9)	9.2 (6.2–12.3)	9.8 (7.9–11.7)
Uterine	42.7 (38.5–47.0)	51.5 (47.0–56.1)	21.4 (17.6–25.3)	32.8 (29.0–36.6)	36.7 (34.2–39.2)

[a] Female 50+ only.
[b] Adenocarcinoma only.
[c] Cardia only.
EBW = excess body weight; PAF = population attributable fraction; 95% CI = 95% confidence interval.

CI 5.9–10.9) and uterine cancer with a PAF of 32.8% (95% CI 28.9–36.7), resulting in 684 cancers (137 per year).

Impact of reduction in EBW on cancer

The estimated annual reduction in various types of cancers attributable to EBW under each EBW reduction scenario is presented in Table 6.

In scenario one, focussing on halving the prevalence of individuals with a BMI of ≥30kg/m^2, just over 210 cancers could be prevented annually with the largest decrease in cancer cases seen in uterine cancer, with an annual reduction of 59 cases. This is followed by colorectal cancer, which shows a decrease of 55 cases, and kidney and renal cancers with a reduction of 26 cases.

Examining the impact by ethnicity under scenario one, the European/Other group exhibits the most significant decrease in these cancer types, with a total reduction of 136 cases. For Māori there is a reduction of 39 cases, for Pacific 28 cases, and for Asian eight cases.

By sex, females show a total reduction of 129 cases, largely attributed to decreases in breast and uterine cancers, while males, predominantly affected by colorectal cancer, see a reduction of 82 cases.

Under scenario two, where there is a halving of the EBW prevalence across both BMI categories, the reductions are more substantial, with a reduction of just over 610 cancers annually. Colorectal cancer shows the largest decrease with 168 cases, followed by breast cancer in females over 50 with a reduction of 117 cases and uterine cancer, which sees a decrease of 101 cases.

In terms of the ethnic-specific impact under scenario two, European/Other again leads with the largest reduction, totalling 435 cases. This is

Table 5: Number of cancers attributable to EBW by cancer type and ethnic group.

Cancer type	Number of cancers attributable to EBW—average per year (95% CI)					Number of cancers attributable to EBW—5-year (95% CI)				
	Māori	Pacific	Asian	European and Other	Total	Māori	Pacific	Asian	European and Other	Total
Breast[a]	43 (29–57)	21 (13–28)	10 (7–13)	173 (122–224)	246 (192–300)	215 (145–286)	103 (65–140)	49 (34–64)	865 (604–1,120)	1,231 (961–1,500)
Colorectal	38 (28–47)	18 (13–22)	12 (7–17)	293 (206–379)	360 (272–447)	188 (139–236)	89 (67–112)	60 (37–83)	1,464 (1,038–1,897)	1,801 (1,367–2,231)
Oesophageal[b]	8 (8–9)	2 (2–2)	1 (1–1)	80 (75–86)	91 (85–96)	41 (38–44)	9 (8–9)	4 (4–5)	401 (374–428)	454 (428–482)
Gallbladder	4 (3–4)	3 (3–4)	1 (0–1)	9 (7–10)	16 (15–18)	18 (15–20)	17 (15–19)	3 (2–3)	44 (37–51)	81 (73–89)
Kidney and renal pelvis	28 (24–31)	10 (9–11)	6 (4–7)	114 (98–131)	158 (141–175)	138 (122–154)	50 (45–55)	28 (22–34)	572 (490–655)	789 (704–873)
Liver	20 (18–23)	10 (9–11)	4 (3–5)	33 (28–39)	67 (61–73)	101 (88–114)	48 (43–53)	21 (15–26)	166 (140–192)	336 (306–366)
Multiple myeloma	6 (4–8)	4 (3–5)	1 (1–2)	33 (22–45)	44 (33–56)	30 (20–39)	18 (12–24)	7 (4–10)	167 (111–222)	222 (165–278)
Ovary	2 (1–4)	2 (0–3)	1 (0–1)	9 (2–16)	13 (6–21)	11 (2–20)	8 (2–14)	4 (0–7)	44 (8–79)	67 (30–103)
Pancreas	11 (7–14)	4 (3–5)	3 (2–4)	47 (32–62)	64 (48–79)	53 (37–69)	19 (13–26)	13 (8–18)	234 (158–308)	319 (242–395)
Stomach[c]	4 (3–4)	1 (1–2)	1 (1–1)	26 (22–31)	32 (28–37)	18 (15–21)	7 (6–8)	4 (3–5)	131 (107–154)	161 (137–184)
Thyroid	8 (5–10)	5 (4–6)	4 (2–5)	18 (12–23)	34 (27–40)	39 (28–51)	25 (18–32)	18 (10–25)	88 (59–117)	170 (137–203)

Table 5 (continued): Number of cancers attributable to EBW by cancer type and ethnic group.

Cancer type	Number of cancers attributable to EBW—average per year (95% CI)					Number of cancers attributable to EBW—5-year (95% CI)				
	Māori	Pacific	Asian	European and Other	Total	Māori	Pacific	Asian	European and Other	Total
Uterine	50 (45–55)	66 (60–71)	13 (11–16)	137 (121–153)	266 (248–284)	251 (226–276)	329 (300–358)	67 (55–79)	684 (603–765)	1,331 (1,242–1,421)

[a] Female 50+ only.
[b] Adenocarcinoma only.
[c] Cardia only.
EBW = excess body weight; 95% CI = 95% confidence interval.

Table 6: Number of cancers potentially prevented each year under different EBW scenarios.

Cancer type, ethnic group and sex	Scenario 1* (95% CI)	Scenario 2** (95% CI)
Cancer type		
Breast (female 50+ only)	23 (11–36)	117 (108–127)
Colorectal	55 (40–70)	168 156–180)
Oesophageal (adenocarcinoma only)	7 (1–14)	32 (27–38)
Gallbladder	4 (2–7)	7 (5–10)
Kidney and renal pelvis	26 (17–35)	68 (60–76)
Liver	17 (11–22)	29 (24–34)
Multiple myeloma	2 (0–8)	21 (17–25)
Ovary	1 (0–4)	6 (4–9)
Pancreas	3 (0–10)	30 (25–35)
Stomach (cardia only)	6 (1–10)	14 (11–18)
Thyroid	7 (2 to 11)	16 (12–20)
Uterine	59 (49–70)	101 (91–111)
Ethnic group		
Māori	39 (28–51)	94 (84–104)
Pacific	28 (19–37)	56 (48–64)
Asian	8 (2–14)	26 (21–31)
European/Other	136 (111–161)	435 (415–455)
Sex		
Male	82 (63–102)	261 (245–276)
Female	129 (107–151)	350 (331–368)
Total	211 (182–241)	611 (586–634)

* Scenario 1: a reduction in the population prevalence with a BMI of 30kg/m^2 or more by 50%, with the assumption that individuals from this category move into the BMI range of 25.00–29.99kg/m^2.
** Scenario 2: halving the prevalence of EBW across both BMI categories (BMI ≥30kg/m^2 and BMI 25.00–29.99kg/m^2).
EBW = excess body weight; 95% CI = 95% confidence interval.

followed by Māori with a decrease of 94 cases, Pacific with 56 cases and Asian with 26 cases.

In scenario two, the sex-specific impact shows a more significant reduction in cancer cases among females, with a total decrease of 350 cases. This substantial reduction is largely attributed to decreases in breast and uterine cancers. Conversely, males exhibit a reduction of 261 cases, predominantly in colorectal cancer.

Discussion

We estimate that between 2019 and 2023 there were 6,962 cancer cases among New Zealand adults aged over 30 years that could be potentially attributed to EBW (5.1% of all cancers in this group). The overall PAF was higher in females (6.3%) than in males (4.1%), largely reflecting the associations of EBW with female-specific cancer risk. The overall PAF was highest for oesophageal cancer (44.2%) and uterine cancer (36.7%). In absolute terms, the greatest number of cancers attributable to EBW were colorectal (1,801), followed by uterine (1,331) and breast among postmenopausal females (1,231). Inequities are evident with higher proportions of EBW-attributable cancers within the Māori and Pacific populations. Pacific peoples had the highest PAF (11.8%), and this was highest among Pacific females (16.1%). Māori also had a higher PAF (6.9%) than European/Other (4.5%).

The estimates of PAF of the New Zealand population can be compared with other published reports. However, there are differences in PAF estimates across studies, reflecting population differences in the prevalence of EBW and the choice of relative risk estimates. Our estimates of PAF are slightly higher than those previously published for New Zealand.[37] Blakely et al. reported a PAF for all cancers of 5.0% for males and 4.0% for females. For Māori, the PAF was a slightly less at 4.0% for males and slightly higher for Māori females at 5.0%. This difference is primarily a result of the different source used for estimates of relative risk and different estimates of EBW.

In Australia, estimates of PAF are lower at 3.4% overall and for both males (2.5%) and females (4.6%). In the United States of America, 4.7% of cancers in males and 9.6% of cancers in females have been shown to be attributable to EBW.[38] Our estimate for the PAF for colorectal cancer was slightly higher than that published by Richardson et al. (9%); however, this difference could be accounted for as our study looked at only those in the 30+ age group.[39]

Our PAF modelling shows there is potential to impact cancer incidence by addressing the prevalence of EBW. In our first modelled scenario, reducing the BMI ≥30kg/m^2 prevalence by half could prevent over 200 cancer cases annually, particularly in types such as uterine, colorectal and breast cancers. Differences were observed by ethnicity and sex, with the largest decreases seen in the European/Other group and in females. Our second scenario, which targets reductions in EBW across all BMI categories, suggests even greater potential reductions in colorectal and breast cancer incidences.

The potential reductions in cancer burden under different scenarios are based on observed associations between EBW and cancer incidence at a population level. While these associations are well documented, our approach does not establish direct causality and does not account for potential mediating factors that may influence the relationship between weight loss and cancer risk. Furthermore, the timeframe over which weight reduction might lead to measurable changes in cancer incidence remains uncertain. As such, these projections should be interpreted as exploratory estimates rather than definitive predictions of causal impact.

EBW increases cancer risk through a series of biological changes that promote tumour growth. In people with EBW, higher levels of insulin, insulin-like growth factor 1 (IGF-1) and leptin, combined with lower levels of protective adiponectin, create a pro-tumour environment. Leptin stimulates cancer cell growth and survival, while reduced adiponectin removes a natural defence against tumours.[40] Specifically, gastrointestinal cancers like gastric and colorectal cancer are linked to EBW through altered lipid metabolism and chronic inflammation. Visceral adipose tissue secretes pro-inflammatory cytokines and promotes insulin resistance, fuelling tumour progression in the gastrointestinal tract.[41] In postmenopausal women, EBW raises oestrogen levels, increasing the risk of hormone-sensitive cancers such as breast and endometrial cancers.[40] Additionally, EBW-related increases in insulin and IGF-1 not only drive cell growth but also inhibit apoptosis, thereby contributing to cancer development in multiple organs, particularly gastrointestinal sites.[41,42] Type 2 diabetes mellitus also independently increases the risk of endometrial cancer, even when EBW is accounted for, and

the combination of both conditions further elevates the risk. This heightened risk is driven by chronic hyperinsulinaemia, insulin resistance and systemic inflammation, which together promote cellular proliferation and inhibit apoptosis. In women with EBW and diabetes, these processes are compounded, significantly contributing to tumour development.[43]

New Zealand's recent funding of advanced cancer medications represents an important step forward in the treatment of EBW-related cancers.[44] Immunotherapies—like pembrolizumab, now funded for advanced breast and colorectal cancer, nivolumab for kidney cancer and cetuximab for specific colorectal cancers—offer targeted treatment approaches. These treatments are especially relevant as EBW is a known risk factor for cancers such as breast, kidney and colorectal. By making these advanced therapies accessible, New Zealand has strengthened its cancer control strategy, addressing both the burden of EBW and the pressing need for effective treatment options in populations at higher risk due to EBW.

While evidence on intentional weight loss reducing cancer risk remains limited,[45] evidence supports bariatric surgery as an effective intervention for lowering the risk of at least seven EBW-associated cancers, with the largest risk reductions seen in gynaecological cancers.[46] Specifically for endometrial cancer, post-surgery reductions in key inflammatory markers, such as interleukin-6 and tumour necrosis factor alpha, contribute to reversing cancer-promoting conditions in the endometrium. Additionally, for women with precursor lesions like atypical endometrial hyperplasia, bariatric surgery has been effective in resolving these abnormalities, highlighting its potential in both cancer prevention and risk reduction for endometrial cancer in individuals with EBW.[47] Furthermore, recent advances in pharmacotherapy are transforming the landscape for the treatment and management of EBW with clinical trials indicating marked weight reductions and health improvements.[48] However, while bariatric surgery and pharmacotherapy show effectiveness in treatment, ensuring equitable access to these treatments is essential to achieving the benefits across populations.[49] Integrated treatment pathways, linked with non-medical, community-centred and compassionate ongoing care, are vital to optimising long-term health outcomes, particularly for those with higher health needs.[50]

Sustained reductions in the burden of EBW-related cancers will largely depend on preventing EBW rather than relying solely on treatment-based interventions. At a broader population health level, New Zealand has implemented multifactorial, multilevel interventions aimed at reducing the burden of disease associated with EBW. These initiatives include the Healthy Eating: Healthy Action Plan, the Food and Beverage Classification System and the Childhood Obesity Plan. While these plans demonstrate a commitment to addressing EBW, progress has been uneven, particularly among vulnerable populations, and addressing the high prevalence of EBW remains challenging, indicating that continued innovative action is required. The obesogenic/health-disrupting environment, heavily influenced by commercial determinants, continues to play a significant role in shaping health behaviours. It has been argued that stronger, mandatory regulations, such as stricter advertising controls, taxation on unhealthy food components and improved food labelling, should be considered to complement existing voluntary measures.[18,51] However, refining approaches and definitions is essential to enabling more equitable, non-stigmatising interventions that improve health outcomes related to EBW and enhance access to care for the most at-risk populations in New Zealand.[5,52,53]

Traditionally, the treatment, management and prevention of EBW focus on lifestyle. While personal choices in diet and exercise are essential, there is often an overemphasis on these factors, which tends to overlook the significant role that socioeconomic disadvantage and systemic barriers play in shaping health outcomes. To effectively combat the root causes of EBW, it is essential to confront the underlying societal systems that contribute to weight gain. These systems include political, commercial, economic and socio-cultural dimensions. Action to support people, whānau and communities living with or at risk of EBW needs to be multidimensional, including focussing on tackling the obesogenic environment through policy and implementation at a system level, alongside community-driven programmes.[54,55]

Individuals with EBW report experiencing bias and stigma when seeking healthcare, including differential treatment and weight-related stigma from healthcare providers.[56–58] Large-bodied females have been shown to have lower screening participation due to experiences of negative weight bias from their healthcare providers.[58] In addition, treatments such as bariatric surgery demonstrate significant inequities in access.[49] Pacific individuals have been shown to receive these interventions at half the rate of Europeans

and Māori.[59] This underscores the necessity of integrating strategies to eliminate bias and stigma in the development and implementation of programmes, services and policies aimed at preventing and treating EBW. Services looking to eliminate bias and stigma and to enhance equity might include incorporating Kaupapa Māori principles throughout their care pathway and introducing mentors who provide support and guidance to individuals undergoing the procedures.[50] Such measures are essential to ensure equitable healthcare access for all population groups.

While this paper focusses on EBW and cancer, the considerations around language and destigmatisation extend to broader discussions of body size and health. Stigmatisation affects individuals across the weight spectrum, including those with very low body weight, who may face assumptions related to disordered eating or aesthetic motivations. Acknowledging these dynamics reinforces the need for nuanced, respectful language in public health discourse to avoid reinforcing harmful stereotypes and to support equitable, person-centred health approaches. Language and compassionate messaging are critical components in shaping public perceptions and stigma around body weight in healthcare settings.[1] The concept of an "ideal body weight", frequently highlighted by the media and emphasised in the medical community as both achievable and necessary, may not only misrepresent the complexities of individual health but also contribute to undue pressure on individuals to attain an ideal weight standard. Such ideals often reinforce stigmatising attitudes and overlook the diversity of healthy body types. It is vital for healthcare providers, patients and the public recognise that even slowing weight gain and moderate or small sustained weight loss (5–10%) can lead to significant health improvements, especially in managing comorbidities associated with EBW like hypertension, elevated blood glucose and abnormal plasma lipid levels.[52,60] Thus, the focus of weight management strategies should shift from striving for an "ideal weight" to supporting realistic and health-centric goals, advocating for body diversity and challenging the stigma associated with varying body sizes.[1,5]

Moving forward, applying learnings from the destigmatising efforts in tobacco,[61,62] infectious diseases,[63] fat studies[11] and Indigenous lived experiences,[10] health practitioners need to recognise the value of adopting a more pro-equity, non-deficit approach to not only addressing EBW but the barriers individuals face in accessing and navigating healthcare services. The discipline of fat studies offers insight into approaches that destigmatise and aim to dismantle the societal stigma tied to weight, which will go some way to breaking down service access barriers and stigma associated with weight.[11] However, these approaches must ensure to continue to be carefully aligned with broader public health objectives and messaging.

The findings of this study highlight the importance of investing in programmes, services and policy initiatives that prevent and manage EBW in New Zealand, particularly with respect to improving cancer outcomes. Improved access to healthcare can be achieved by ensuring services adopt a non-stigmatising and more holistic approach to care, which will assist in breaking down barriers to access and ensure we are in a better position to respond to the needs of patients with EBW and prevent associated cancers.

Strengths

A strength of this current study was the use of cancer registry data, which is considered a full list of all primary cancers registered in New Zealand. In addition, we used relative risk estimates from meta-analysis of high-quality cohort studies of cancers where a causal link of EBW to cancer risk has been shown to have a robust dose–response relationship and a biological mechanism has been described. Our estimates of BMI came from a national representative survey and were not self-reported measures of BMI.

Limitations

The reliance on relative risk estimates from overseas studies necessitates caution, as their applicability to the New Zealand context may be limited. This limitation is particularly relevant given the potential for differences in associations of EBW in certain ethnic groups within New Zealand, such as Māori or Pacific peoples, due to unique genetic, lifestyle or environmental factors.[64]

An important limitation of this study is the broad categorisation of participants as "Asian", which may mask considerable heterogeneity within this group. Differences in dietary practices and disease profiles between New Zealand-born Asians and recent immigrants may affect EBW-related outcomes. However, due to limitations of the data relating to EBW, we were unable to further subdivide the Asian category. Future

studies with more detailed subgroup data are needed to address these potential differences.

A further limitation is using BMI in calculating the PAF for EBW-related cancers. A critique of BMI is its inability to differentiate between fat mass and lean muscle, potentially leading to misclassification of an individuals' EBW.[65] While BMI is commonly employed as the primary indicator for assessing trends in body weight at the population level, it is important to recognise that it does not directly measure body fat but rather estimates it based on height and weight.[21]

The medicalisation of EBW, particularly through the use of BMI, has also been critiqued for framing body weight as a medical condition requiring intervention, often neglecting the broader social, economic and behavioural factors that contribute to it. BMI, as a simplified measure, tends to focus on weight as the primary indicator of health, which can reduce complex health issues to a single metric without considering the full context of an individual's health.[66]

The issues relating to BMI underscore the potential usefulness of other measures, such as waist circumference or waist-to-hip ratio, which may provide more specific insights into fat distribution. These measures are especially relevant in assessing health risks associated with EBW, such as cancers where central or visceral fatness plays a crucial role.[67,68] However, the scarcity of relative risk estimates based on alternate measures, coupled with the lack of population-level data using them, poses a challenge. Nonetheless, studies of disease burden indicate that the excess risk of disease associated with EBW is evident, whether defined via BMI or waist circumference.[69,70] It is also important to note the possibility of overestimation of EBW in some ethnic groups, particularly Pacific peoples, when using current cutoffs, given the previous findings of higher lean mass compared with Europeans.[71] The absence of ethnic-specific cutoffs for EBW highlights an area for future research and development of more culturally sensitive and accurate measures to address EBW's health implications.

When assessing the impact of changes in EBW prevalence on the PAF for cancer, several limitations must be acknowledged. First, the relationship between EBW and cancer is complex and influenced by additional factors such as physical activity, diet and genetic predisposition, which may act as confounders. Our estimates assume that the effect of EBW is independent of these factors, but in reality, these influences overlap and collectively shape cancer risk at a population level. Second, EBW prevalence is not static and can change over time due to shifts in population health trends, lifestyle behaviours and environmental influences. These broader changes not only affect the burden of EBW-related cancers but also contribute to fluctuations in overall cancer incidence across the population. Third, the impact of population-level changes in EBW prevalence on cancer incidence is not immediate or uniform across all cancer types. While some cancers may respond to long-term shifts in EBW prevalence, others may be less sensitive to these changes, making it difficult to estimate the exact timeline and magnitude of cancer reductions. Finally, population heterogeneity means that cancer risk varies across different demographic and socio-economic groups, making it difficult to generalise a single PAF estimate across the entire population.

While our estimates of cancer burden attributable to EBW are based on well-documented associations, they do not demonstrate direct causality. The PAF methodology relies on an assumed causal relationship between EBW and cancer incidence, grounded in observational data that may be subject to residual confounding and unmeasured variables. Nonetheless, strong biological mechanisms support this association. Despite the inherent challenges of establishing causality in cancer development, our findings highlight the considerable contribution of EBW as a modifiable risk factor and reinforce the importance of prevention strategies to reduce the future burden of EBW-related cancers. The PAF remains a valuable tool for estimating the relative significance of EBW and the potential impact of primary prevention efforts, and for contextualising global epidemiological evidence within the New Zealand population.

Conclusion

Between 2019 and 2023, we estimate that EBW contributed to over 6,900 cancers in New Zealand, representing a significant and preventable burden on individuals, communities and the healthcare system. This study reveals clear inequities, particularly among Pacific women, whose PAF is nearly double that of the next closest group, Māori women. These differences underscore the need for tailored interventions that address the distinct socio-cultural, economic and environmental factors faced by these populations.

Addressing the cancer burden associated with EBW requires a sustained and coordinated approach that prioritises the integration of EBW management within cancer control strategies. This includes focussing on prevention through culturally appropriate and community-driven programmes, particularly for Māori and Pacific populations. Equally important is reducing stigma and bias in healthcare settings to ensure equitable access to care, particularly for large-bodied individuals who may face barriers to receiving appropriate treatment. By adopting holistic, non-stigmatising approaches and improving access to services like bariatric surgery and weight management, we can significantly improve cancer outcomes for high-risk groups.

In conclusion, reducing the incidence of cancers associated with excess EBW and improving health equity in New Zealand will require a long-term, sustained commitment. This includes investing in culturally appropriate prevention programmes, ensuring equitable access to clinical services, such as bariatric surgery and weight management, and implementing initiatives that address stigma and bias in healthcare. These efforts must be integrated into accessible health pathways across communities, and within both primary and secondary care. By prioritising equity, eliminating weight-related stigma and addressing the broader determinants of EBW, we can reduce cancer rates and improve overall health outcomes, especially for those disproportionately affected, such as Māori and Pacific communities.

COMPETING INTERESTS
JBP is currently employed with AIA Vitality New Zealand. The data for this study were collected and analysed and the original paper written while JBP was employed as programme manager with Health New Zealand – Te Whatu Ora.

AUTHOR INFORMATION
Michael Walsh: Epidemiologist, Planning, Funding and Outcomes, Health New Zealand – Te Whatu Ora, Auckland, Aotearoa New Zealand.
Jennifer Brenton-Peters: Programme Manager, Planning, Funding and Outcomes, Health New Zealand – Te Whatu Ora, Auckland, Aotearoa New Zealand.
Olivia Perelini: Registrar, Planning, Funding and Outcomes, Health New Zealand – Te Whatu Ora, Auckland, Aotearoa New Zealand.
Karen Bartholomew: Director Health Gain Development, Health New Zealand – Te Whatu Ora, Auckland, Aotearoa New Zealand.
Disclaimer: The views and opinions expressed in this paper are those of the authors and do not necessarily reflect those of Health New Zealand – Te Whatu Ora.

CORRESPONDING AUTHOR
Michael Walsh: Epidemiologist, Planning, Funding and Outcomes, Health New Zealand – Te Whatu Ora, Level 2, Q4 Building, Smales Farm, 74 Taharoto Road, Takapuna, Auckland 0622 | Private Bag 93-503, Takapuna 0740, Aotearoa New Zealand.
E: michael.walsh@tewhatuora.govt.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/cancers-potentially-attributable-to-excess-body-weight-in-aotearoa-new-zealand-from-2019-to-2023

REFERENCES
1. Wharton S, Lau DCW, Vallis M, et al. Obesity in adults: a clinical practice guideline. CMAJ. 2020;192(31):E875-E891. doi: 10.1503/cmaj.191707.
2. Gaspar MCMP, Sato PM, Scagliusi FB. Under the 'weight' of norms: Social representations of overweight and obesity among Brazilian, French and Spanish dietitians and laywomen. Soc Sci Med. 2022;298:114861. doi: 10.1016/j.socscimed.2022.114861.
3. Black CK, Vartanian LR, Faasse K. Lay beliefs about the perceived harmfulness of excess weight: Influence of weight status and the cause of weight. Appl Psychol Health Well Being. 2024;16(2):653-671. doi: 10.1111/aphw.12509.
4. Phelan SM, Burgess DJ, Yeazel MW, et al. Impact of weight bias and stigma on quality of care and outcomes for patients with obesity. Obes Rev. 2015;16(4):319-26. doi: 10.1111/obr.12266.
5. English S, Vallis M. Moving beyond eat less, move more using willpower: Reframing obesity as a chronic disease impact of the 2020 Canadian obesity guidelines reframed narrative on perceptions of self and the patient-provider relationship. Clin Obes. 2023;13(6):e12615. doi: 10.1111/cob.12615.
6. Butler ÉM, Pillai A, Morton SMB, et al. A prediction model for childhood obesity in New Zealand. Sci Rep. 2021;11(1):6380. doi: 10.1038/s41598-021-85557-z.
7. WHO Consultation on Obesity, World Health Organization. Obesity: preventing and managing the global epidemic: report of a WHO consultation. Geneva, Switzerland: World Health Organization; 1999. https://iris.who.int/handle/10665/42330
8. Daniels L, Cutfield WS, Taylor RW, Taylor BJ. Why BMI should still be on the table. N Z Med J. 2020;133(1513):97-100.
9. Puhl RM, Heuer CA. Obesity stigma: important considerations for public health. Am J Public Health. 2010;100(6):1019-28. doi: 10.2105/AJPH.2009.159491.
10. Gillon A, Pausé C. Kōrero Mōmona, Kōrero ā-Hauora: a Kaupapa Māori and fat studies discussion of fatness, health and healthism. Fat Studies. 2022;11(1):8-21. doi: 10.1080/21604851.2021.1906525.
11. Lee JA, Pausé CJ. Stigma in Practice: Barriers to Health for Fat Women. Front Psychol. 2016;7:2063. doi: 10.3389/fpsyg.2016.02063.
12. Anekwe CV, Jarrell AR, Townsend MJ, et al. Socioeconomics of Obesity. Curr Obes Rep. 2020;9(3):272-279. doi: 10.1007/s13679-020-00398-7.
13. Arena R, Pronk NP, Woodard C. Physical Inactivity and Obesity in the United States: At the Intersection of Politics, Socioeconomics, Race, and Culture. Curr Probl Cardiol. 2023;48(12):102007. doi: 10.1016/j.cpcardiol.2023.102007.
14. Organisation for Economic Co-operation and Development. The Heavy Burden of Obesity: The Economics of Prevention. Paris, France: OECD Publishing; 2019 [cited 2025 Mar 31]. Available from: https://doi.org/10.1787/67450d67-en
15. Ministry of Health – Manatū Hauora. New Zealand Health Survey 2023/24 Annual Data Explorer [Internet]. Wellington, New Zealand; 2024 [cited 2025 Mar 31]. Available from: https://minhealthnz.shinyapps.io/nz-health-survey-2023-24-annual-data-explorer/
16. Organisation for Economic Cooperation and

Development. Health at a Glance 2019: OECD Indicators. 2019:1-243. doi: 10.1787/4dd50c09-en.
17. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. Obes Rev. 2016;17(2):95-107. doi: 10.1111/obr.12334.
18. Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. Lancet. 2011;378(9793):804-14. doi: 10.1016/S0140-6736(11)60813-1.
19. Pineda E, Stockton J, Scholes S, et al. Food environment and obesity: a systematic review and meta-analysis. BMJ Nutr Prev Health. 2024;7(1):204-211. doi: 10.1136/bmjnph-2023-000663.
20. Swinburn B, Egger G, Raza F. Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity. Prev Med. 1999;29(6 Pt 1):563-70. doi: 10.1006/pmed.1999.0585.
21. Lauby-Secretan B, Scoccianti C, Loomis D, et al. Body Fatness and Cancer--Viewpoint of the IARC Working Group. N Engl J Med. 2016;375(8):794-8. doi: 10.1056/NEJMsr1606602.
22. Guh DP, Zhang W, Bansback N, et al. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health. 2009;9:88. doi: 10.1186/1471-2458-9-88.
23. Larsson SC, Spyrou N, Mantzoros CS. Body fatness associations with cancer: evidence from recent epidemiological studies and future directions. Metabolism. 2022;137:155326. doi: 10.1016/j.metabol.2022.155326.
24. Bandini M, Gandaglia G, Briganti A. Obesity and prostate cancer. Curr Opin Urol. 2017;27(5):415-421. doi: 10.1097/MOU.0000000000000424.
25. Teng AM, Atkinson J, Disney G, et al. Ethnic inequalities in cancer incidence and mortality: census-linked cohort studies with 87 million years of person-time follow-up. BMC Cancer. 2016;16(1):755. doi: 10.1186/s12885-016-2781-4.
26. Cleverley T, Meredith I, Sika-Paotonu D, Gurney J. Cancer incidence, mortality and survival for Pacific Peoples in Aotearoa New Zealand. N Z Med J. 2023;136(1586):12-31. doi: 10.26635/6965.6299.
27. Gurney J, Stanley J, McLeod M, et al. Disparities in Cancer-Specific Survival Between Māori and Non-Māori New Zealanders, 2007-2016. JCO Glob Oncol 2020;6:766-774. doi: 10.1200/GO.20.00028.
28. Curtis E, Jones R, Tipene-Leach D, et al. Why cultural safety rather than cultural competency is required to achieve health equity: a literature review and recommended definition. Int J Equity Health. 2019;18(1):174. doi: 10.1186/s12939-019-1082-3.
29. Soeberg M, Blakely T, Sarfati D. Trends in ethnic and socioeconomic inequalities in cancer survival, New Zealand, 1991-2004. Cancer Epidemiol. 2015;39(6):860-2. doi: 10.1016/j.canep.2015.10.018.
30. Talamaivao N, Harris R, Cormack D, et al. Racism and health in Aotearoa New Zealand: a systematic review of quantitative studies. N Z Med J. 2020;133(1521):55-68.
31. Gurney J, Stanley J, Sarfati D. The inequity of morbidity: Disparities in the prevalence of morbidity between ethnic groups in New Zealand. J Comorb. 2020;10:2235042X20971168. doi: 10.1177/2235042X20971168.
32. Williams L, Henry C, Simcock B, Filoche S. Increasing incidence of endometrial cancer in Aotearoa New Zealand: Health professionals' perspective. Aust N Z J Obstet Gynaecol. 2024 Apr;64(2):114-119. doi: 10.1111/ajo.13751.
33. Ministry of Health – Manatū Hauora. HISO 10001:2017 Ethnicity Data Protocols [Internet]. Wellington, New Zealand: 2017 [cited 2025 Mar 31]. Available from: https://www.tewhatuora.govt.nz/assets/Our-health-system/Digital-health/Health-information-standards/HISO-10001-2017-Ethnicity-Data-Protocols.pdf
34. Harris R, Paine SJ, Atkinson J, et al. We still don't count: the under-counting and under-representation of Māori in health and disability sector data. N Z Med J. 2022;135(1567):54-78. doi: 10.26635/6965.5849.
35. Brown KF, Rumgay H, Dunlop C, et al. The fraction of cancer attributable to modifiable risk factors in England, Wales, Scotland, Northern Ireland, and the United Kingdom in 2015. Br J Cancer. 2018;118(8):1130-1141. doi: 10.1038/s41416-018-0029-6.
36. Ministry of Health – Manatū Hauora. A Portrait of Health. Key Results of the 2006/07 New Zealand Health Survey [Internet]. Wellington, New Zealand: Ministry of Health; 2008 [cited 2024 Dec 14]. Available from: https://thehub.sia.govt.nz/resources/a-portrait-of-health-key-results-of-the-200607-new-zealand-health-survey
37. Blakely T, Sarfati D, Shaw C. What proportion of cancer is due to obesity? N Z Med J. 2009;122(1290):9-13.
38. Islami F, Goding Sauer A, Gapstur SM, Jemal A. Proportion of Cancer Cases Attributable to Excess Body Weight by US State, 2011-2015. JAMA Oncol. 2019;5(3):384-392. doi: 10.1001/jamaoncol.2018.5639.
39. Richardson A, Hayes J, Frampton C, Potter J. Modifiable lifestyle factors that could reduce the

incidence of colorectal cancer in New Zealand. N Z Med J. 2016;129(1447):13-20.
40. Hopkins BD, Goncalves MD, Cantley LC. Obesity and Cancer Mechanisms: Cancer Metabolism. J Clin Oncol 2016;34(35):4277-4283. doi: 10.1200/JCO.2016.67.9712.
41. Mitchelson KAJ, O'Connell F, O'Sullivan J, Roche HM. Obesity, Dietary Fats, and Gastrointestinal Cancer Risk-Potential Mechanisms Relating to Lipid Metabolism and Inflammation. Metabolites. 2024;14(1):42. doi: 10.3390/metabo14010042.
42. Lee-Rueckert M, Canyelles M, Tondo M, et al. Obesity-induced changes in cancer cells and their microenvironment: Mechanisms and therapeutic perspectives to manage dysregulated lipid metabolism. Semin Cancer Biol. 2023;93:36-51. doi: 10.1016/j.semcancer.2023.05.002.
43. Njoku K, Agnew HJ, Crosbie EJ. Impact of Type 2 Diabetes Mellitus on Endometrial Cancer Survival: A Prospective Database Analysis. Front Oncol 2022;12:899262. doi: 10.3389/fonc.2022.899262.
44. Pharmac – Te Pātaka Whaioranga. First cancer medicine decision following Pharmac funding boost [Internet]. 2024 Sep 9 [cited 2024 Nov 4]. Available from: https://pharmac.govt.nz/news-and-resources/news/first-cancer-medicine-decision-following-pharmac-funding-boost
45. Ma C, Avenell A, Bolland M, et al. Effects of weight loss interventions for adults who are obese on mortality, cardiovascular disease, and cancer: systematic review and meta-analysis. BMJ. 2017;359:j4849. doi: 10.1136/bmj.j4849.
46. Wilson RB, Lathigara D, Kaushal D. Systematic Review and Meta-Analysis of the Impact of Bariatric Surgery on Future Cancer Risk. Int J Mol Sci. 2023;24(7):6192. doi: 10.3390/ijms24076192.
47. Naqvi A, MacKintosh ML, Derbyshire AE, et al. The impact of obesity and bariatric surgery on the immune microenvironment of the endometrium. Int J Obes (Lond). 2022;46(3):605-612. doi: 10.1038/s41366-021-01027-6.
48. Shi Q, Wang Y, Hao Q, et al. Pharmacotherapy for adults with overweight and obesity: a systematic review and network meta-analysis of randomised controlled trials. Lancet. 2024;403(10434):e21-e31. doi: 10.1016/S0140-6736(24)00351-9.
49. Rahiri JL, Lauti M, Harwood M, MacCormick AD, Hill AG. Ethnic disparities in rates of publicly funded bariatric surgery in New Zealand (2009-2014). ANZ J Surg. 2018;88(5):E366-E369. doi: 10.1111/ans.14220.
50. Rahiri JL, Tuhoe J, Gillon A, et al. Enhancing responsiveness to Māori in a publicly funded bariatric service in Aotearoa New Zealand. ANZ J Surg. 2020;90(4):508-513. doi: 10.1111/ans.15610.
51. Swinburn BA. Obesity prevention: the role of policies, laws and regulations. Aust New Zealand Health Policy. 2008;5:12. doi: 10.1186/1743-8462-5-12.
52. Mack M, Savila F, Bagg W, et al. Interventions to prevent or manage obesity in Māori and Pacific adults: a systematic review and narrative synthesis. Ethn Health 2023;28(4):562-585. doi: 10.1080/13557858.2022.2078482.
53. Rubino F, Cummings DE, Eckel RH, et al. Definition and diagnostic criteria of clinical obesity. Lancet Diabetes Endocrinol. 2025;13(1):221-262. doi: 10.1016/S2213-8587(24)00316-4. Erratum in: Lancet Diabetes Endocrinol. 2025 Mar;13(3):e6. doi: 10.1016/S2213-8587(25)00006-3.
54. Masters-Awatere B, Cassim S, et al. He Pikinga Waiora Kimi Ora lifestyle programme: case study of a successful community-based Indigenous diabetes intervention. N Z Med J. 2021;134(1545):68-78.
55. Savila F, Leakehe P, Bagg W, et al. Understanding engagement with Brown Buttabean Motivation, an Auckland grassroots, Pacific-led holistic health programme: a qualitative study. BMJ Open. 2022;12(4):e059854. doi: 10.1136/bmjopen-2021-059854.
56. Norman K, Chepulis L, Burrows L, Lawrenson R. Barriers to obesity health care from GP and client perspectives in New Zealand general practice: A meta-ethnography review. Obes Rev. 2022;23(10):e13495. doi: 10.1111/obr.13495.
57. Kirk SF, Price SL, Penney TL, et al. Blame, Shame, and Lack of Support: A Multilevel Study on Obesity Management. Qual Health Res. 2014;24(6):790-800. doi: 10.1177/1049732314529667.
58. Russell N, Carryer J. Living large: the experiences of large-bodied women when accessing general practice services. J Prim Health Care. 2013;5(3):199-205.
59. 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;30(6):2285-2293. doi: 10.1007/s11695-020-04463-x.
60. Magkos F, Fraterrigo G, Yoshino J, et al. Effects of Moderate and Subsequent Progressive Weight Loss on Metabolic Function and Adipose Tissue Biology in Humans with Obesity. Cell Metab. 2016;23(4):591-601. doi: 10.1016/j.cmet.2016.02.005.
61. Hamann HA, Williamson TJ, Studts JL, Ostroff JS. Lung Cancer Stigma Then and Now: Continued Challenges Amid a Landscape of Progress. J Thorac Oncol. 2021;16(1):17-20. doi: 10.1016/j.jtho.2020.10.017.

62. Ostroff JS, Banerjee SC, Lynch K, et al. Reducing stigma triggered by assessing smoking status among patients diagnosed with lung cancer: De-stigmatizing do and don't lessons learned from qualitative interviews. PEC Innov. 2022;1:100025. doi: 10.1016/j.pecinn.2022.100025.
63. Kagan D, Seear K, Lenton E, et al. The trouble with normalisation: Transformations to hepatitis C health care and stigma in an era of viral elimination. Sociol Health Illn. 2023;45(7):1421-1440. doi: 10.1111/1467-9566.13638.
64. Rush EC, Freitas I, Plank LD. Body size, body composition and fat distribution: comparative analysis of European, Maori, Pacific Island and Asian Indian adults. Br J Nutr. 2009;102(4):632-41. doi: 10.1017/S0007114508207221.
65. Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes (Lond). 2008;32 Suppl 3:S56-9. doi: 10.1038/ijo.2008.87.
66. Campos P, Saguy A, Ernsberger P, et al. The epidemiology of overweight and obesity: public health crisis or moral panic? Int J Epidemiol. 2006;35(1):55-60. doi: 10.1093/ije/dyi254.
67. Venniyoor A. The most important questions in cancer research and clinical oncology-Question 2-5. Obesity-related cancers: more questions than answers. Chin J Cancer. 2017;36(1):18. doi: 10.1186/s40880-017-0185-8.
68. Shuster A, Patlas M, Pinthus JH, Mourtzakis M. The clinical importance of visceral adiposity: a critical review of methods for visceral adipose tissue analysis. Br J Radiol. 2012;85(1009):1-10. doi: 10.1259/bjr/38447238.
69. Turley M, Tobias M, Paul S. Non-fatal disease burden associated with excess body mass index and waist circumference in New Zealand adults. Aust N Z J Public Health. 2006;30(3):231-7. doi: 10.1111/j.1467-842x.2006.tb00863.x.
70. Taylor RW, Brooking L, Williams SM, et al. Body mass index and waist circumference cutoffs to define obesity in indigenous New Zealanders. Am J Clin Nutr. 2010;92(2):390-7. doi: 10.3945/ajcn.2010.29317.
71. Duncan JS, Duncan EK, Schofield G. Ethnic-specific body mass index cut-off points for overweight and obesity in girls. N Z Med J 2010;123(1311):22-9.

Asian health trends in New Zealand from 2002 to 2021, and the case for dedicated research funding

Robert Scragg, Zhenqiang Wu, Sally F Wong

ABSTRACT

The proportion of the Aotearoa New Zealand population with Asian ancestry is growing, from 17% in the 2023 census to an expected 26% by 2043. Thus, the health of the Asian community in New Zealand is increasing in importance. We have recently completed a major report on the health status of Asian people living in New Zealand since 2002 using data from the New Zealand Health Survey. While there have been some improvements, levels of most risk factors—such as fruit and vegetable intake, physical activity, alcohol intake and obesity—have worsened or not improved over the last 20 years. These have resulted in elevated risk of cardiometabolic disease, particularly among South Asians, at levels similar to those for Māori and Pacific. We have reviewed the funding of Asian health research by the Health Research Council of New Zealand since 2010 by searching the lay summaries of grants. We have found a mismatch between the number of funded grants and the size of the Asian population in New Zealand (respectively, 2.3% and 17% in 2023). The Health Research Council needs to ring-fence funding for Asian researchers so that Asian researchers have increased resources to research the major health issues that are adversely affecting their communities.

Aotearoa New Zealand is currently in the middle of a profound change in the demographic composition of its people. The main change over the last 20 years has been in its ethnic composition. Data from the New Zealand census (Table 1) show that while the total population increased from 2006 by 24% to nearly 5 million in 2023, larger increases have occurred in non-European ethnic groups. For Māori, the increase over this period has been 57%, and for Pacific 66%. However, the largest increase has been in the Asian community, which has increased from being 9% of the total population in 2006 to 17% in 2023—a relative increase of 143% from 2006. This increase is expected to continue so that 26% of the New Zealand population by 2043 will be Asian.[1]

Because of its increasing size, the health of the Asian community is of great importance for New Zealand, not only for individual Asian people but also for funding health services at the population level. Evidence has emerged over the last decade showing that some diseases remain elevated in the Asian community.[4] Failure to address the causes of these diseases will impact adversely on the New Zealand health budget in the long term. In this paper, we describe key findings from a major report we have co-authored on the health of the Asian community in New Zealand, and the trends in health research funding by ethnicity. Together, these document a mismatch between the level of Asian health research funding and the disease burden affecting the Asian community.

Table 1: New Zealand population census: trends by ethnicity since 2006.[2,3]

Ethnicity	2006 N (% of total)	2013 N (% of total)	2018 N (% of total)	2023	
				N (% of total)	Increase from 2006
Asian	354,552 (9%)	471,708 (11%)	707,598 (15%)	861,576 (17%)	143%
Māori	565,329 (14%)	598,602 (14%)	775,836 (17%)	887,493 (18%)	57%
Pacific	265,974 (7%)	295,941 (7%)	381,642 (8%)	442,632 (9%)	66%
Total	4,027,947	4,242,048	4,699,755	4,993,923	24%

1. Asian health report

We have recently prepared a major report on the health of the Asian community in New Zealand.[5] It is based on data collected in the New Zealand Health Survey from 2002/2003 up to the recent data release for 2020/2021. Briefly, the method used in the New Zealand Health Survey is as follows.

Methods

All New Zealand Health Surveys have used a three-stage, stratified complex sampling method to over-sample Māori, Pacific and Asian participants to allow for ethnic-specific analyses of all three ethnicities, along with European and Other participants.[6,7] Face-to-face interviews were carried out in the homes of participants. Information was collected on the following topics: demographic status, health behaviours, health conditions and health service utilisation.[8] This was done separately for children aged 0–14 years and adults aged ≥15 years, although only results for adults are reported in this paper.

Ethnicity was self-defined, and participants were allowed to choose their affiliation with more than one ethnic group, in which case the following priority for categorising ethnicity was applied: Asian, followed by Māori, then Pacific, and lastly European and Other. The Asian sample was further categorised, based on the coding available from the Ministry of Health – Manatū Hauora, into three groupings, with the following order of priority: South Asian (Indian, Fiji-Indian, Pakistani, Sri Lankan, Bengali, Nepali and Afghani), Chinese, and Other Asian.[4] The ethnic-specific sample sizes for the two recent survey periods (2019–2021) for adults were: South Asian 805, Chinese 573, Other Asian 708, Māori 3,790, Pacific 1,000 and European and Other 12,532.

Participants are weighted by the inverse of their sampling probability so that collectively those surveyed represent the total resident New Zealand population.[5] For the difference across all ethnic groupings, the combined data from the 2019 to 2020 and 2020 to 2021 surveys were analysed to compare: All Asian (as reference, combined South Asian, Chinese, Other Asian), Māori, Pacific and European and Other, after controlling for age and gender (as appropriate). Mantel–Haenszel common relative risks (RRs) for binary disease and prevention service variables were also calculated for selected two-ethnic grouping comparisons (European and Other as reference), adjusting for age and gender (as appropriate).

For trend analyses, comparisons among Asian were made using available data between the 2002 to 2003 and 2019 to 2021 (as reference) surveys for adults aged 15 years and older. All p-values have been adjusted for age and gender as appropriate. Data were analysed using SUDAAN (version 11.0.4, Research Triangle Park, North Carolina, United States of America) and SAS 9.4 (SAS Institute Inc, Cary, North Carolina, United States of America).

Results

Table 2 summarises selected adult data from the main report. It compares the four main ethnic groups (Asian, Māori, Pacific and European and Other) in the 2019–2020 and 2020–2021 surveys combined, using Asian as the reference, and changes over time among Asian only since their first inclusion in the 2002–2003 New Zealand Health Survey.

Demographic variables

The Asian community in New Zealand has aged over the last two decades, with the proportion of Asian people aged ≥65 years increasing between 2002 to 2003 and 2019 to 2021 ($p=0.01$), and is now similar to that for Pacific ($p=0.13$), but below that for Māori and European and Other ($p<0.01$). The length of time lived in New Zealand by Asian people has increased, with the proportion who have lived in New Zealand for ≥10 years, or who were born here, nearly doubling between 2002 to 2003 and 2019 to 2021. The Asian community is very highly educated, with the proportion of Asian people with a university degree increasing between 2002 to 2003 and 2019 to 2021, and it is currently much higher than in the other main ethnic groups. Over the same period, the proportion of Asian people on government support (e.g., unemployment benefit) has decreased such that it is a much lower proportion than the other main ethnic groups, indicating that Asian people overall are net contributors to the government purse. Of concern, the proportion of Asian people who report being a victim of an ethnically motivated verbal or physical attack has increased during the period from 2011 to 2012 (the first year this was measured in the New Zealand Health Survey) to 2020 to 2021, and is now similar to that for Māori (but higher than that for Pacific and European and Other).

Health behaviours

The trends in health behaviours since 2002–2003 are mixed, with some improving and others worsening. Pleasingly, the proportion of current smokers among Asian people has decreased during this period and currently is the lowest of the main ethnic groups. The pattern for physical activity itself is mixed, with no significant change between 2002 to 2003 and 2019 to 2021 in the proportion of Asian people who are active, although the proportion who are sedentary declined over this period.

Of concern, the proportion of Asian people consuming the recommended five+ serves of fruit and vegetables each day has decreased between 2002 to 2003 and 2019 to 2021, and currently is much lower than for Māori and European and Other groups. Alcohol consumption has also increased among the Asian community between 2002 to 2003 and 2019 to 2021, based both on the proportion who drank alcohol in the last 12 months and also the proportion reporting hazardous drinking (Alcohol Use Disorders Identification Test [AUDIT] score ≥8[9]): however, their current percents for these are both lower than those of other ethnic groups. In addition, the proportion of Asian people with obesity (based on internationally accepted ethnic-specific definitions[10,11]) has doubled between 2002 to 2003 and 2019 to 2021, so that it is only slightly lower currently than for Pacific but higher than for Māori (and European and Other).

Diseases

The above adverse behaviour profiles among Asian people for fruit and vegetable intake, physical activity and obesity are being manifested by increased risk of some chronic diseases. Table 3 shows RR of disease separately for three Asian groupings (South Asian, Chinese and Other Asian) because of their different patterns, and for Māori and Pacific, in comparison with European and Other, adjusted for age and gender to remove confounding effects from these variables. The adjusted RRs of hypertension and high blood cholesterol are elevated by a similar amount in South Asian, Māori and Pacific, as is diabetes in South Asian and Other Asian (RR >3.0). In contrast, the risks of all of the above diseases are similar in Chinese to those for European and Other. In addition, the risk of cardiovascular disease is also lower in Chinese, as are asthma, arthritis and depression in all three Asian groupings.

Prevention services

The level of prevention services provided in the last 12 months is a measure of the degree to which clinical services are responding to the adverse cardiometabolic disease profile of Asian people. Table 3 shows that South Asian people, along with Māori and Pacific, were more likely to be offered weight/height measurements, cholesterol testing and advice regarding exercise and physical health, compared with European and Other. Appropriately, increased testing for diabetes was also offered to South Asian, Other Asian, Māori and Pacific. Of concern, South Asian people were not more likely to be offered a blood pressure test in the last 12 months than European and Other, despite having a higher risk of hypertension. Also, all three Asian groupings were no more likely to be offered green prescriptions (advice to be physically active and eat healthily) or advice on weight than European and Other, in contrast to the situation for Māori and Pacific, who were, despite all three Asian groupings having elevated levels of obesity.[5] These latter findings point to gaps in the prevention services offered to Asian people.

In summary, the data in Table 2–3 on trends in health behaviours among Asian adults since 2002–2003—with a worsening in the situation for fruit and vegetable intake, obesity and alcohol and no improvement in activity levels, along with the current elevated risk of cardiometabolic disease, particularly for South Asian, at levels similar to those for Māori and Pacific—indicate a societal and health system failure to improve the health of the Asian community, a need that was identified nearly 20 years ago.[12] These findings are consistent with the report on Asian health in New Zealand in 2011–2013,[4] and many are supported by additional New Zealand[13] and international studies.[14,15]

2. Health research funding

One important area that has the potential to identify possible factors contributing to worse health outcomes or preventing improvements is research—particularly government-funded health research. The Ministry of Health – Manatū Hauora eventually recognised the need to collect separate data from Asian people in the 2002 New Zealand Health Survey, 10 years after the first survey conducted in collaboration with Stats NZ in 1992–1993. It is the monitoring of the health status of Asian people in the New Zealand Health Surveys that has provided the data for our report[5]

Table 2: Summary of results from the New Zealand Health Surveys: trends in Asian people over time, current comparison with other ethnicities (adults only)—weighted percents.

Variable	2002–2003 survey, weighted % (p value #)	2019–2020 and 2020–2021 surveys combined, weighted % (p value #)			
	Asian, n=1,217 (6.2%)	Asian (reference), n=2,086 (15.0%)	Māori, n=3,790 (14.1%)	Pacific, n=1,000 (5.7%)	European and Other, n=12,532 (65.3%)
Demographic					
Aged ≥65 years	5.3 (0.01)	7.2	9.5 (0.005)	9.3 (0.13)	25.0 (<0.001)
Lived in New Zealand ≥10 years or New Zealand–born	29.5 (<0.001)	57.1	99.7 (<0.001)	87.0 (<0.001)	93.1 (<0.001)
Have a university degree	35.6 (<0.001)	51.0	13.4 (<0.001)	10.1 (<0.001)	28.3 (<0.001)
Receive government support	24.9 (<0.001)	13.3	39.1 (<0.001)	29.2 (<0.001)	34.3 (<0.001)
Victim of ethnically motivated attack	24.2 (0.12)**	29.1*	29.6 (0.65)*	16.7 (<0.001)*	13.9 (<0.001)*
Health behaviours					
Fruit and vegetables: 5+ serves/day	40.8 (<0.001)	28.3	37.1 (<0.001)	33.3 (0.19)	45.2 (<0.001)
Physical activity					
Active	40.1 (0.14)	45.2	54.9 (<0.001)	45.9 (0.39)	55.7 (<0.001)

Table 2 (continued): Summary of results from the New Zealand Health Surveys: trends in Asian people over time, current comparison with other ethnicities (adults only)—weighted percents.

Variable	2002–2003 survey, weighted % (p value #)	2019–2020 and 2020–2021 surveys combined, weighted % (p value #)			
	Asian, n=1,217 (6.2%)	Asian (reference), n=2,086 (15.0%)	Māori, n=3,790 (14.1%)	Pacific, n=1,000 (5.7%)	European and Other, n=12,532 (65.3%)
Sedentary	21.4 (0.004)	14.9	13.5 (0.13)	18.2 (0.13)	11.5 (<0.001)
Current smokers	11.9 (0.001)	7.9	29.5 (<0.001)	20.6 (<0.001)	9.6 (<0.001)
Alcohol					
In the last 12 months	49.8 (0.001)	60.7	82.4 (<0.001)	60.6 (0.27)	85.6 (<0.001)
Hazardous drinking	4.5 (0.03)	9.2	42.6 (<0.001)	39.8 (<0.001)	24.1 (<0.001)
Obesity	25.7 (<0.001)	52.9	38.7 (<0.001)	58.0 (0.02)	29.5 (<0.001)

#Comparing 2019–2021 data for Asian.
*2020–2021 data.
**2011–2012 data.

Table 3: Age- and gender-adjusted relative risks (RR) (95% confidence interval [CI]) compared with European and Other, among adults aged ≥15 years in the 2019–2021 surveys.

Variable	2019–2020 and 2020–2021 surveys combined, RR (95% CI)					
	South Asian	Chinese	Other Asian	Māori	Pacific	European and Other (reference)
Diseases						
Hypertension	1.37 (1.01–1.85)	0.68 (0.49–0.96)	0.93 (0.67–1.30)	1.37 (1.26–1.50)	1.45 (1.20–1.75)	1.00
High cholesterol	1.71 (1.28–2.30)	1.07 (0.74–1.55)	1.25 (0.88–1.78)	1.31 (1.13–1.52)	1.56 (1.31–1.86)	1.00
Cardiovascular disease	1.00 (0.67–1.49)	0.49 (0.31–0.77)	0.68 (0.42–1.11)	1.16 (1.02–1.32)	0.98 (0.76–1.27)	1.00
Diabetes	3.26 (2.22–4.78)	1.20 (0.62–2.31)	3.05 (2.20–4.21)	2.28 (1.92–2.72)	4.19 (3.30–5.32)	1.00
Asthma	0.37 (0.25–0.53)	0.34 (0.21–0.55)	0.45 (0.30–0.69)	1.55 (1.36–1.76)	1.20 (0.94–1.53)	1.00
Arthritis	0.57 (0.39–0.82)	0.57 (0.41–0.80)	0.58 (0.42–0.81)	1.23 (1.12–1.36)	0.99 (0.81–1.21)	1.00
Depression	0.36 (0.21–0.62)	0.33 (0.16–0.70)	0.20 (0.11–0.36)	0.92 (0.77–1.08)	0.41 (0.27–0.62)	1.00
Prevention services provided in the last 12 months						
Weight/height measure	1.29 (1.15–1.43)	0.97 (0.81–1.15)	1.02 (0.88–1.19)	1.26 (1.18–1.34)	1.34 (1.23–1.45)	1.00
Blood pressure test	1.06 (0.99–1.14)	0.86 (0.78–0.95)	0.94 (0.86–1.03)	1.08 (1.04–1.12)	1.09 (1.02–1.15)	1.00
Cholesterol test	1.42 (1.26–1.60)	1.04 (0.90–1.21)	1.02 (0.87–1.19)	1.19 (1.12–1.27)	1.26 (1.15–1.38)	1.00
Diabetes test	1.58 (1.34–1.86)	1.18 (0.97–1.44)	1.21 (1.02–1.43)	1.39 (1.28–1.50)	1.59 (1.43–1.77)	1.00
Green prescription	0.88 (0.34–2.30)	1.13 (0.33–3.94)	0.70 (0.30–1.61)	2.35 (1.65–3.35)	1.41 (0.81–2.48)	1.00
Health food/nutrition	1.34 (1.05–1.72)	0.92 (0.64–1.32)	1.24 (0.96–1.59)	1.87 (1.65–2.12)	2.45 (2.08–2.88)	1.00
Weight	1.11 (0.88–1.40)	0.71 (0.48–1.04)	0.95 (0.68–1.35)	1.81 (1.60–2.05)	2.31 (2.00–2.66)	1.00
Exercise/physical health	1.36 (1.11–1.68)	0.87 (0.58–1.32)	1.14 (0.87–1.49)	1.59 (1.41–1.80)	1.77 (1.50–2.09)	1.00

and this article.

However, the main government funder of health research in New Zealand is the Health Research Council of New Zealand (HRC). What have they done over the last 10 years or so, during a period when the Asian community has become a major part of the New Zealand population, and after the publication of our previous report in 2016,[4] which found very similar findings to our current report?

Methods

In order to explore the allocation of health research funding by ethnicity and track their trends, we extracted information on funded health research projects from the HRC Research Repository[16]—the main government funder of health research—for the period between 2010 and 2023. The extracted data included the study title, year, approved budget, lead investigator, host organisation, proposal type and lay summary.

We then searched the study titles and lay summaries, and categorised all funded studies into Asian-, Māori- and Pacific-focussed studies using the following keywords: Asian = Asian, Asia, Chinese, India; Māori = Maori, Māori, Mäori, Rongoā, Hauora, Whakatōhea, Wahine, Tangata Whenua, Tāne, Ngāti Rangitihi; and Pacific = Pacific, Pasifika, Tonga, Samoa, Cook Island, Fiji, Niue, Niuean, Tokelau, Ni-Vanuatu. Trends in the number of funded studies, and approved budget, by ethnicity and year were analysed. If a study mentioned more than one ethnic group, it was allocated to each one.

Results

Over the 14-year period from 2010 to 2023, a total of 2,685 studies were funded with an approved budget of NZ$1.55 billion. Around 1.2% (n=32) of these studies were Asian-focussed (had Asian-related keywords in the title or abstract), accounting for 1.1% (NZ$16.9 million) of the total approved budget.

Figure 1 and Appendix Table 1 show the annual number of HRC-funded studies during 2010–2023, by ethnicity of the study participants, based on key words in the titles or lay summaries of these studies. Over this period the total number of studies increased by over 60% (from 183 to 300 per year), as did the number of studies where Māori or Pacific ethnicity was mentioned in the title or lay summary. In contrast, there has been no discernible increase in the number of studies with "Asian" in the title or lay summary. Further, the number of such studies is very low—far lower than the proportion that

Figure 1: Annual number of funded studies by ethnicity.

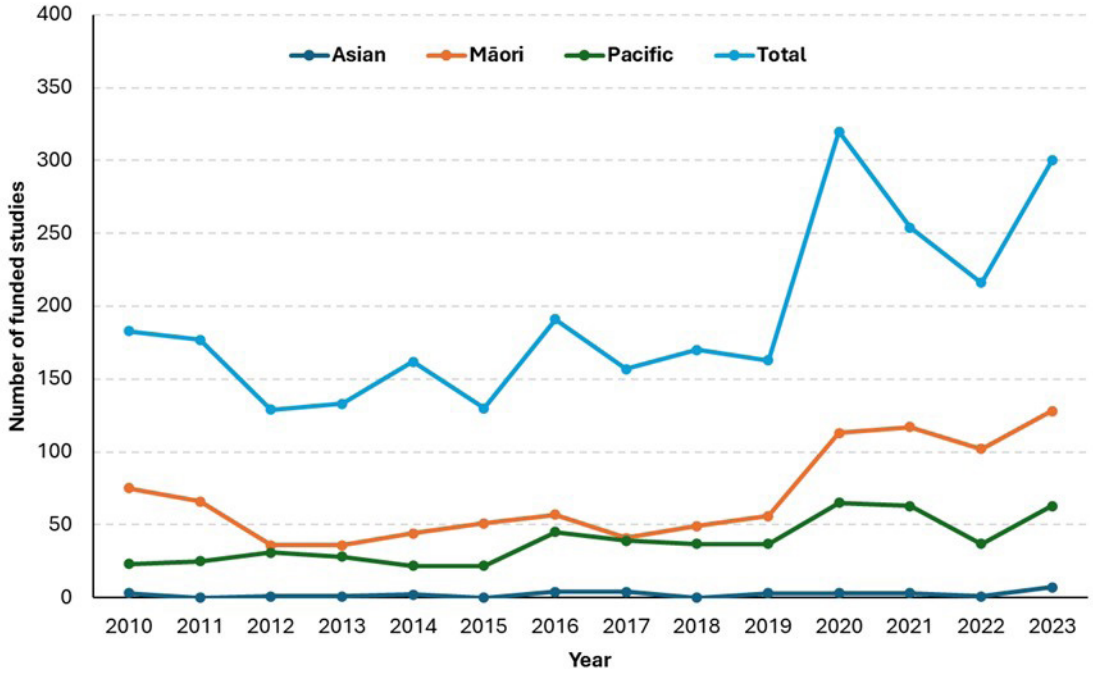
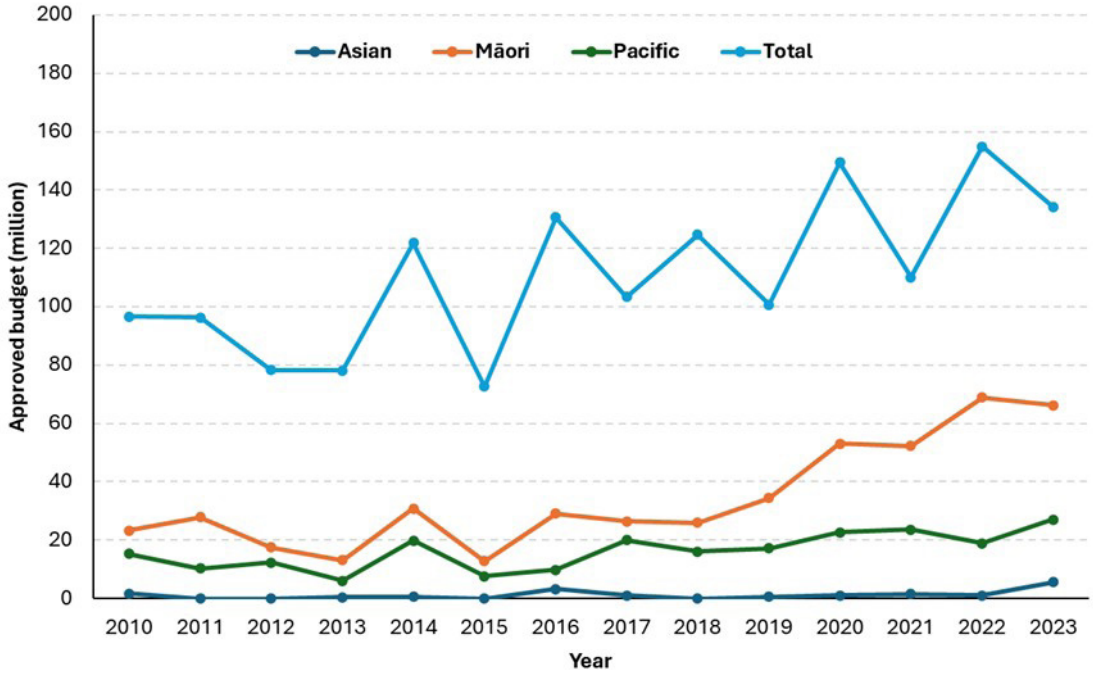


Figure 2: Annual approved budget by ethnicity.



Asian people contribute to the total New Zealand population (Table 1). A similar pattern is seen for the approved budgets of funded studies (Figure 2, Appendix Table 2). We acknowledge there are limitations with the method we used to identify studies with Asian participants. By relying on titles and lay summaries, we may have missed some studies that did have an Asian focus. The HRC has much more extensive data about funded studies that could be used to confirm or refute our findings.

3. Discussion

Given the mismatch between health needs, population size and health research funding, in our view the time has come for the main government funder of health research (i.e., HRC) to ring-fence funding for Asian researchers who submit grant applications focussing on Asian health. The HRC has twice in its past ring-fenced ethnic-specific funding. The first was for Māori, with the creation by the *Health Research Council Act 1990* of the Māori Research Committee, which was provided with money ring-fenced for Māori researchers. This resulted in a flowering of research by and for Māori, which up until then had been blocked by the relative inexperience of Māori researchers operating in an openly competitive environment. This experience was repeated in 2017 when the HRC finally implemented a policy to ring-fence project funding (up to NZ$1.2 million) for research by and for Pacific.[17] Since then, the number and funding of Pacific research has increased (Figure 1–2). The ring-fencing of funds for Māori and Pacific researchers are both excellent examples of the benefits from such a strategy.

The same arrangement is now needed for Asian researchers, e.g., starting with summer studentships, master's scholarships, PhD scholarships and postdoctoral fellowships, as a recent review has found that the evidence base on Asian health in New Zealand is weak.[18] Increasing the number of and funding for research by and for Asians is more likely to identify factors that are preventing improvements in health outcomes than the current situation that has remained or worsened over the last 20 years. This is also highlighted in the recently submitted petition for a National Health and Well-Being Policy/Strategy for Asian and MELAA Communities.[19] Over time, the continuing increase in the size of the Asian population will require research on other subpopulations, such as Filipinos and those who are gender diverse. The earlier decision by the Ministry of Health – Manatū Hauora to recruit sufficient

Asian participants in the 2002–2003 New Zealand Health Survey is an example of the benefits that flow from a policy that allows for the carrying out of ethnic-specific analyses.

4. Conclusion

The health of the Asian community has not improved over the last 20 years. Given its rapidly increasing size in New Zealand, and the limited funding currently awarded for Asian health research, funding agencies such as the HRC need to ring-fence and increase funding for Asian researchers so that they can identify solutions within their communities to rectify this situation.

COMPETING INTERESTS
All authors received funding from The Asian Network Inc (TANI) to write the recent report on Asian health in New Zealand. ZW is supported by a Health Research Council Health Delivery Research Career Development Award (22/796).

AUTHOR INFORMATION
Robert Scragg: Professor of Epidemiology, School of Population Health, The University of Auckland, Auckland, New Zealand.
Zhenqiang Wu: Biostatistician, School of Population Health and Department of Medicine, The University of Auckland, Auckland, New Zealand.
Sally F Wong: Independent Researcher, Auckland, New Zealand.

CORRESPONDING AUTHOR
Prof Robert Scragg: School of Population Health, The University of Auckland, Private Bag 92019, Auckland Mail Centre, Auckland 1142, New Zealand.
E: r.scragg@auckland.ac.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/asian-health-trends-in-new-zealand-from-2002-to-2021-and-the-case-for-dedicated-research-funding

REFERENCES
1. Stats NZ. Subnational ethnic population projections: 2018(base)–2043 [Internet]. 2022 [cited 2024 Jul 24]. Available from: https://www.stats.govt.nz/information-releases/subnational-ethnic-population-projections-2018base2043/
2. Stats NZ. 2018 Census ethnic group summaries [Internet]. [cited 2024 Jul 24]. Available from: https://www.stats.govt.nz/tools/2018-census-ethnic-group-summaries/
3. Stats NZ. 2023 Census population counts (by ethnic group, age, and Māori descent) and dwelling counts [Internet]. 2024 May 29 [cited 2024 Jul 24]. Available from: https://www.stats.govt.nz/information-releases/2023-census-population-counts-by-ethnic-group-age-and-maori-descent-and-dwelling-counts/
4. Scragg R. Asian Health in Aotearoa in 2011 - 2013: trends since 2002-2003 and 2006-2007. Auckland, New Zealand: Northern Regional Alliance Ltd; 2016.
5. Wu Z, Wong S, Scragg R. Asian Health in Aotearoa: Findings from the New Zealand Health Survey 2002–2021 [Internet]. The Asian Network Inc; 2024 [cited 2024 Oct 7]. Available from: https://d2057z2iq79qyw.cloudfront.net/uploads/1/page/asian-health-in-aotearoa-august-2024/Asian_Health_in_Aotearoa_Full_Report_Aug_2024_compressed.pdf
6. Clark RG, Templeton R, McNicholas A. Developing the design of a continuous national health survey for New Zealand. Popul Health Metr. 2013;11(1):25. doi: 10.1186/1478-7954-11-25.
7. Ministry of Health – Manatū Hauora. Sample Design from 2015/16: New Zealand Health Survey [Internet]. Wellington, New Zealand: Ministry of Health – Manatū Hauora; 2016 [cited 2024 Oct 7]. Available from: https://www.health.govt.nz/publications/sample-design-from-201516-new-zealand-health-survey
8. Ministry of Health – Manatū Hauora. Content Guide 2020/21: New Zealand Health Survey [Internet]. Wellington: Ministry of Health – Manatū Hauora; 2021 [cited 2024 Oct 7]. Available from: https://www.health.govt.nz/publications/questionnaires-and-content-guide-202021-new-zealand-health-survey
9. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG. AUDIT: The Alcohol Use Disorders Identification Test: Guidelines for Use in Primary Care [Internet]. World Health Organization, 2001 [cited 2024 Oct 7]. Available from: https://www.who.int/publications/i/item/WHO-MSD-MSB-01.6a
10. World Health Organization. The Asia-Pacific perspective: redefining obesity and its treatment [Internet]. Sydney, Australia; 2000 [cited 2024 Oct 7]. Available from: https://iris.who.int/handle/10665/206936
11. Sundborn G, Metcalf PA, Gentles D, et al. Overweight and obesity prevalence among adult Pacific peoples and Europeans in the Diabetes Heart and Health Study (DHAHS) 2002-2003, Auckland New Zealand. N Z Med J. 2010;123(1311):30-42.
12. Rasanathan K, Ameratunga S, Tse S. Asian health in New Zealand--progress and challenges. N Z Med J. 2006;119(1244):U2277.
13. Atlantis E, Joshy G, Williams M, Simmons D. Diabetes Among Māori and Other Ethnic Groups in New Zealand. In: Dagogo-Jack S, ed. Diabetes Mellitus in Developing Countries and Underserved Communities. Springer International Publishing; 2017. p. 165-90.
14. Vicks WS, Lo JC, Guo L, et al. Prevalence of prediabetes and diabetes vary by ethnicity among U.S. Asian adults at healthy weight, overweight, and obesity ranges: an electronic health record study. BMC Public Health. 2022;22(1):1954. doi: 10.1186/s12889-022-14362-8.
15. National Health Service England. Health Survey England Additional Analyses, Ethnicity and Health, 2011-2019 Experimental statistics [Internet].

2022 [cited 2025 Feb 17]. Available from: https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-england-additional-analyses/ethnicity-and-health-2011-2019-experimental-statistics
16. Health Research Council of New Zealand. HRC Research Repository [Internet]. 2024 [cited 2024 Jul 24]. Available from: https://www.hrc.govt.nz/resources/research-repository
17. Health Research Council of New Zealand. Report of the Health Research Council of New Zealand for the year ended 30 June 2016 [Internet]. 2017 [cited 2024 Oct 7]. Available from: https://www.hrc.govt.nz/sites/default/files/2019-06/Resource%20Library%20PDF%20-%20Annual%20Report%202016.pdf
18. Chiang A, Simon-Kumar R, Peiris-John R. A decade of Asian and ethnic minority health research in New Zealand: findings from a scoping review. N Z Med J. 2021;134(1542):67-83.
19. Public Health Association of New Zealand. National Health and Well-Being Policy/Strategy for Asian and MELAA Communities [Internet]. 2024 Apr 15 [cited 2024 Oct 7].

Appendix

Appendix Table 1: Annual number of funded studies by ethnicity.

Year	Number of funded studies	Māori		Pacific		Asian	
		n	Percent	n	Percent	n	Percent
2010	183	75	41.0%	23	12.6%	3	1.6%
2011	177	66	37.3%	25	14.1%	0	0.0%
2012	129	36	27.9%	31	24.0%	1	0.8%
2013	133	36	27.1%	28	21.1%	1	0.8%
2014	162	44	27.2%	22	13.6%	2	1.2%
2015	130	51	39.2%	22	16.9%	0	0.0%
2016	191	57	29.8%	45	23.6%	4	2.1%
2017	157	41	26.1%	39	24.8%	4	2.5%
2018	170	49	28.8%	37	21.8%	0	0.0%
2019	163	56	34.4%	37	22.7%	3	1.8%
2020	320	113	35.3%	65	20.3%	3	0.9%
2021	254	117	46.1%	63	24.8%	3	1.2%
2022	216	102	47.2%	37	17.1%	1	0.5%
2023	300	128	42.7%	63	21.0%	7	2.3%

One study can be categorised into more than one ethnic-focussed category.

Appendix Table 2: Annual approved budget by ethnicity.

Year	Total approved budget, NZD (million)	Māori		Pacific		Asian	
		Budget	Percent	Budget	Percent	Budget	Percent
2010	96.6M	23.3M	24.2%	15.4M	15.9%	1.7M	1.7%
2011	96.4M	27.9M	29.0%	10.4M	10.8%	0.0M	0.0%
2012	78.2M	17.5M	22.4%	12.3M	15.7%	<0.01M	0.01%
2013	78.2M	13.1M	16.8%	6.0M	7.7%	0.4M	0.5%
2014	121.9M	30.8M	25.2%	19.9M	16.3%	0.7M	0.5%
2015	72.7M	12.8M	17.6%	7.7M	10.6%	0.0M	0.0%
2016	130.7M	29.1M	22.2%	9.8M	7.5%	3.2M	2.5%
2017	103.3M	26.4M	25.6%	20.0M	19.4%	1.1M	1.1%
2018	124.7M	26.0M	20.8%	16.1M	12.9%	0.0M	0.0%
2019	100.8M	34.4M	34.2%	17.1M	17.0%	0.6M	0.6%
2020	149.5M	53.0M	35.5%	22.7M	15.2%	1.0M	0.7%
2021	110.0M	52.3M	47.6%	23.6M	21.4%	1.5M	1.4%
2022	154.9M	69.0M	44.5%	18.9M	12.2%	1.1M	0.7%
2023	134.3M	66.3M	49.4%	27.0M	20.1%	5.6M	4.1%

One study can be categorised into more than one ethnic-focussed category.

Scurvy in the modern era: a case of vitamin C deficiency with unexplained bruising and anaemia

Akram Shmendi

Scurvy,[1] caused by a deficiency in vitamin C (ascorbic acid), is a rare but still relevant diagnosis, particularly in individuals with poor dietary habits or certain social vulnerabilities. The condition results from insufficient vitamin C, which plays a key role in collagen synthesis, wound healing and maintaining the integrity of connective tissues. Although scurvy was once common among sailors and soldiers who lacked access to fresh fruit and vegetables,[2] it remains an occasional diagnosis in contemporary clinical practice, especially among those who have an unbalanced diet, social vulnerability, alcoholism and severe psychiatric disorders.[3] We report the case of a 62-year-old male who presented with right leg swelling, bruising and anaemia, initially suspected of having deep vein thrombosis (DVT) or a coagulation disorder, but was later diagnosed with scurvy.

Case report

Patient history and presentation

A 62-year-old NZ European male presented to the emergency department (ED) with a 1-week history of right leg swelling and bruising, without any history of trauma. His past medical history included previous treatment for stage T4a N2a squamous cell carcinoma of the right tonsil in 2007, treated with chemoradiotherapy. He also had chronic thoracic back pain from a prior trauma. He was an ex-smoker, having quit 14 years ago, and denied alcohol consumption, over-the-counter medications, supplements or herbal remedies.

Upon presentation, the patient reported initially noticing small red spots on his right knee a week earlier, which had progressed to significant bruising and swelling of the right leg. He also described similar lesions on his left leg. Over the following days, he developed shortness of breath on exertion and dizziness upon standing. However, he denied any gastrointestinal bleeding, including melena, haematuria or oral bleeding. He also denied any recent infections or prior bleeding episodes, and there was no family history of bleeding disorders.

Clinical examination

The patient appeared alert and comfortable, with a slim build. His blood pressure was 140/90mmHg in the supine position, dropping to 87/67mmHg upon standing, with a heart rate of 100 beats per minute. Examination revealed non-palpable, non-tender petechiae on the left leg, alongside extensive ecchymoses and swelling of the right leg. The oral mucosa was normal, and there were no signs of bleeding from the gums. Otherwise, his examination was unremarkable.

Investigations

Initial blood tests revealed normocytic normochromic anaemia with a high reticulocyte count and normal iron studies, B12 and folate levels. Peripheral blood smear showed normocytic red cells with increased polychromatic cells and target cells, but no fragments or spherocytosis. Liver function tests were normal apart from mildly raised total bilirubin (34µmol/L) and conjugated bilirubin (15µmol/L), with a slight increase in haptoglobin levels. Lactate dehydrogenase (LDH) was normal, and renal function was intact. Coombs test was negative. Given the patient's shortness of breath and dizziness, a computed tomography pulmonary angiogram (CTPA) was performed by the ED doctor, which ruled out pulmonary embolism. Ultrasound of the right leg excluded deep vein thrombosis (DVT). A dermatology consult was requested for probable cutaneous vasculitis causing extensive bruises and swelling. The patient exhibited perifollicular haemorrhages and corkscrew hairs on the right leg, which were strongly suggestive of scurvy. The absence of bleeding in the oral mucosa, gums or other mucosal surfaces and the patient's poor dietary intake led to a diagnosis of scurvy. The

lack of typical findings for vasculitis, such as oral ulceration or systemic symptoms, further supported this diagnosis.

Diagnosis and management

The diagnosis of scurvy was confirmed after the patient's vitamin C levels were found to be severely low (<1µmol/L; normal range: 26–85µmol/L). Given the clinical features, including the characteristic dermatological signs and the patient's inadequate intake of fruit and vegetables, treatment with high-dose vitamin C (250mg twice daily) was initiated. Over the following days, the patient's bruising and leg swelling improved significantly, and he reported a reduction in his symptoms of shortness of breath and dizziness.

Discussion

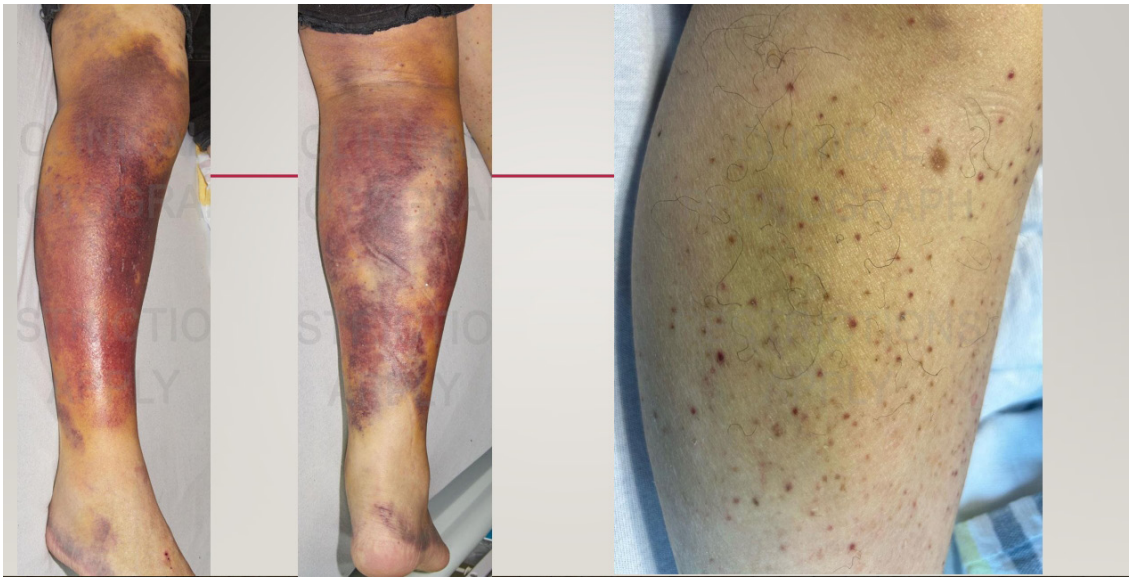
Scurvy is a disease that results from a deficiency of vitamin C, which is essential for the hydroxylation of proline and lysine residues in collagen. This deficiency leads to impaired collagen synthesis, resulting in weakened blood vessels, which can cause bruising, petechiae and poor wound healing.[3] In severe cases, scurvy can lead to spontaneous bleeding, bone pain and anaemia. It is most commonly seen in individuals with limited access to fresh fruit and vegetables, or those with psychiatric disorders, alcoholism or eating disorders.[3]

In this case, the patient's clinical presentation —marked by easy bruising, leg swelling and anaemia—initially raised concerns of a coagulopathy, DVT or vasculitis. However, the absence of systemic inflammatory signs, the patient's lack of trauma and the characteristic dermatological signs led to the diagnosis of scurvy. Notably, scurvy can mimic vasculitis, especially in patients presenting with purpura or ecchymosis, making it important to consider in the differential diagnosis.

Conclusion

Scurvy remains an important diagnosis in patients with unexplained bruising, anaemia and impaired wound healing, especially those with poor nutritional intake or risk factors for vitamin C deficiency. Early recognition and treatment with vitamin C supplementation can result in rapid improvement and prevent complications. This case highlights the importance of obtaining a detailed dietary history and considering nutritional deficiencies in the differential diagnosis of bleeding disorders.

Figure 1: Lower limb ecchymosis with perifollicular haemorrhage and corkscrew hairs.



COMPETING INTERESTS
None declared.

ACKNOWLEDGEMENTS
The authors would like to acknowledge the Department of Dermatology for their valuable contribution and help in management of this patient.

CORRESPONDING AUTHOR INFORMATION
Akram Shmendi: Consultant Physician, General Medicine Department, Middlemore Hospital, Auckland.
E: akram.shmendi@middlemore.co.nz

URL
https://nzmj.org.nz/journal/vol-138-no-1614/scurvy-in-the-modern-era-a-case-of-vitamin-c-deficiency-with-unexplained-bruising-and-anaemia

REFERENCES

1. Hodges RE, Hood J, Canham JE, et al. Clinical manifestations of ascorbic acid deficiency in man. Am J Clin Nutr. 1971;24(4):432-43. doi: 10.1093/ajcn/24.4.432.
2. Magiorkinis E, Beloukas A, Diamantis A. Scurvy: past, present and future. Eur J Intern Med. 2011;22(2):147-52. doi: 10.1016/j.ejim.2010.10.006.
3. Léger D. Scurvy: reemergence of nutritional deficiencies. Can Fam Physician. 2008;54(10):1403-6.

Splanchnic Analgesia.

NZMJ, 1925

By J.W. COSTELLO, M.B., Ch.B., *Napier Hospital*.

In the following series of eight cases of upper abdominal operations, the general condition of the patients was such that everything that could be done to diminish shock was of primary importance, and it was thought that in splanchnic analgesia this to a large extent would be eliminated in addition to conferring other advantages on the patient.

With the permission and helpful assistance of Dr. J. Allan Berry I was enabled to use this method of anæsthesia for some of his cases in an attempt to reduce the shock, heart strain and post-operative distress to a minimum, with results that would suggest its suitability for use over a much wider range of cases.

As with every form of local anæsthesia the physical condition of the patient must be considered, as well as the fact that it is impossible in most cases to prevent certain sensations caused by traction on the peritoneum or mesentery which although not described as painful by the patient are such as to cause apprehension. The fear of pain together with the somewhat awesome sights and sounds of the operating theatre, would appear often to more than counter-balance the advantages of the local nerve-blocking in the prevention of shock, even when the operation is preceded by liberal injections of morphine and hyoscine, and for this reason most of the cases were given a light ether anæsthesia throughout—from one to one-and-a-half ounces per hour was all that was required to keep them in a quiet sleep, from which they would occasionally waken sufficiently to talk to the anæsthetist. Complete relaxation of the abdominal muscles and the absence of any straining or forcible breathing were maintained for periods varying between 1¼ and 1¾ hours, after which any further anæsthesia required had to be obtained by increasing the amount of ether used. On return to the ward consciousness was recovered in from 5 to 20 minutes, the pain in all cases seemed to be somewhat less than after general anæsthetics, and there was a remarkable freedom from vomiting or nausea. In most cases the chief difficulty was to persuade the patients that they were unable to go back to a full diet on the first or second day after operation.

TECHNIQUE.—The patients are prepared by giving liberal quantities of glucose and fluids and sufficient sod. bicarb. to render the urine alkaline, for 24 to 48 hours before operation. At least an hour before leaving the ward they are given 1–6 to 1–8 gr. Morphine and 1–150 to 1–250 gr. hyoscine, according to their condition, and this dose is repeated, if the first is well tolerated, just before being taken to the theatre. Blood-pressure and pulse record are made before starting the splanchnic injections which consist of 25 to 30 c.c. 1 per cent. novocain with 5 to 10 minims adrenaline on either side of the body of the twelfth thoracic and first lumbar vertebra, according to the method described by Labat. With the patient on his side and a pillow to keep the vertebral column straight, a perpendicular line is dropped to meet the last rib at a distance of 7 c.m. from the mid-line and a lumbar puncture needle inserted at an angle of 45 degrees with the sagittal plane of body till it comes in contact with the body of the first lumbar vertebra—the point of the needle is then withdrawn and raised till it can be felt passing just tangentially to the anterior surface of the vertebral body. It is then pushed in 1 c.m. further, and 15 to 20 c.c. of the solution injected slowly. The syringe should be aspirated before and at intervals during the injection to be sure the needle is not in a blood vessel. If the point of the needle is in correct position very slight pressure is needed, the weight of the piston being almost sufficient to diffuse the solution through the loose tissues surrounding the coeliac plexus. The needle is then withdrawn till the point is in the subcutaneous tissues and reintroduced in a similar way till it just grazes the anterior surface of the second lumbar vertebra, it is then pushed in 1 c.m. further and 10 to 15 c.c of the solution injected—the process is then repeated on the other side. The danger of penetrating some of the large vessels is not great if care is taken to keep the point of the needle in contact with the body of the vertebra and to pass it not more than 1 c.m. further once it is felt to graze the anterior surface—with a little practice on the cadaver the necessary landmarks can easily be acquired.

After these injections there occurred in all

cases a pronounced fall in blood-pressure and a degree of collapse that at times seemed rather alarming, but when turned on their backs and kept quiet for 10 to 15 minutes, during which time the abdominal wall was injected, all the cases made a rapid recovery and the operation was not started till the blood-pressure was rising again.

The initial shock of the splanchnic injections was minimised by slow injection and keeping the temperature of the solution at 100 degrees F. In some cases the use of more adrenalin in the splanchnic injection and less in the abdominal wall seemed to lessen the disturbance also. Never more than a total of 15 to 20 minims adrenalin was used for all the injections. The abdominal wall is then anæthetised with from 60 to 80 c.c., ½ per cent. novocain on either side—the injections being made in two layers—into the muscles and subcutaneously. The former can be made either into the lateral border of the rectus or along the costal margin and down to the iliac crests on both sides beneath the deep fascia covering the oblique muscles; of the two the latter is no more difficult and gives a wider area of relaxation. By the time the injections are completed, 20 to 30 minutes from starting, the patient is recovering from the first shock of the injection and full anæsthesia of the abdominal wall and abdominal contents is present. Whilst making the splanchnic injections a light third degree anæsthesia is preferable, as even with the minimum of scratching of the bone of the vertebra it is a painful process, described as the most painful part of the whole operation by two patients who had no general anæsthesia at all. As soon as the posterior injections are completed the anæsthetic can be stopped till the abdomen is opened.

The following is a record of the cases:—

(1) Male, 60, shepherd. Abdomen tensely distended with hydatids. History suggestive of heart failure for several months past—marked signs of pressure on thoracic organs—dullness and many coarse creps. both bases—liver at upper margin of fourth rib on inspiration—heart lying almost transversely with apex 2 inches outside nipple in fourth space. X-ray not suggestive of any hyatids in thorax.

Throughout the operation the patient insisted on having his head raised sufficiently to see what was going on and his interest in the number of cysts of all sizes being washed out of his abdomen was only diverted by an occasional mouthful of brandy. At the end of 1½ hours after starting the operation, just before closing the abdomen, a firm pull on the liver with the hand between it and the diaphragm caused no comment, except that it made him feel a little sick.

He returned to the ward with a strong steady pulse of 99 which had fallen to 85 by the evening and the general condition of the patient was very good till about midnight, when he collapsed while attempting to sit up in bed. The pulse recovered for a few hours and then failed rapidly, death occurring about 18 hours after the operation. The condition of the patient was so good for 12 hours after the operation that the sudden collapse seems to be more due to the effect of the patient's efforts to sit up, on a heart already labouring under some extreme changes of pressure, rather than to the shock of the operation.

(2) Male, 47, railwayman. Extensive carcinoma of pylorus with marked cachexia and general debility. He had a complete anæsthesia and relaxation for 1½ hours, during which time a partial gastrectomy and wide dissection of the glands in mesentery was done. The first ether was given whilst closing the peritoneum which was a difficult one to approximate in the upper part of the wound. He was fully conscious a few minutes after return to the ward, with a strong pulse, between 70 and 80, there was no vomiting, restlessness or abdominal tension, and the patient was up and eating well a week later. He was discharged 15 days after the operation in rapidly improving condition; ten months later he reported himself to be fit and working harder than at any time during the past 8 years.

(3) Female, 60. Diagnosed as extensive carcinoma of the stomach—marked emaciation, and heart sounds weak—tic-tic rhythm and frequent extra systoles, response to effort poor, three million reds, 45 per cent. Hb. Kept under observation for several months and decided to be inoperable, till test-meals and a slight improvement in her general condition threw some doubt as to whether the condition was carcinoma. Operation showed two chronic ulcers with extensive infiltration of the stomach wall. Ulcers were excised and gastro-enterostomy performed. The patient was kept under light ether most of the time, but was allowed to become conscious one hour after the operation started; she had no complaint to make except to want a drink. Fully conscious and feeling well 10 minutes after return to the ward, she never vomited, nor had any nausea—there was moderate distension of the abdomen for several days, beyond which very little discomfort was complained of. She was discharged feeling very well three weeks after the operation.

Proceedings of the Waikato Clinical Campus Research Seminar, Wednesday 12 March 2025

DEVELOPING A "LIVE" MAP OF SPATIAL ACCESS TO HEALTH SERVICES IN AOTEAROA NEW ZEALAND

Mitchell Pincham,[1] Marcus Blake,[2] Sam Quinsey,[2] Jesse Whitehead*[1]

[1]*Te Ngira: Institute for Population Research, University of Waikato, New Zealand*
[2]*Centre of Australian Research into Accessibility, Deakin Rural Health, Deakin University, Australia*

AIMS

To develop a real-time model that incorporates current road conditions to estimate spatial access to health services daily, at the address level.

METHODS

National highway road-closure data were collected from the New Zealand Transport Agency Application Programming Interface. Data about local road closures were scraped from local council websites. A road network from Open Street Maps was modified by removing any closed highways or local roads. The distance from each address in the Manawataki Health Region, through this new road network, to the nearest hospital was calculated. The programme was automated to run each day in January, using current road conditions for that day to estimate hospital accessibility.

RESULTS

Daily estimates of hospital accessibility were successfully automated, with variations in spatial accessibility over time noted. However, the importance of data quality for the accuracy of this model is paramount. Reporting structures and formats meant that data obtained from some local councils were found to be imprecise or unreliable at times.

CONCLUSIONS

This approach shows potential for quickly estimating access to health services under changing road conditions, such as during and after extreme weather events. NZTA and local councils should be encouraged to work together to improve the reporting of road closures.

"LOOK BELOW THE CROTCH": PROVIDER-PERCEIVED SYSTEMIC BARRIERS TO PODIATRY CARE IN THE WAIKATO REGION, AOTEAROA NEW ZEALAND

Lynley Uerata,[1] Nina Scott,[1] Amy Jones,[1] Jade Tamatea,[1,2] Polly Atatoa-Carr,[1,3] Lynne Chepulis,[4] Ryan Paul,[1,4] Haylee Simon,[1] Tia Dargaville,[1] Ross Lawrenson[4]

[1]*Hauora Māori Services, Te Whatu Ora Waikato, Hamilton, Aotearoa New Zealand*
[2]*Faculty of Medical and Health Sciences, Te Kupenga Hauora Māori, The University of Auckland, Aotearoa New Zealand*
[3]*Te Ngira: Institute for Population Research, University of Waikato, Hamilton, Aotearoa New Zealand*
[4]*Waikato Medical Research Centre, University of Waikato, Hamilton, Aotearoa New Zealand*

BACKGROUND

Foot and leg problems are more prevalent for people living with chronic disease. Yet, access to podiatry care rarely arises as a significant determinant of health. While barriers to podiatry care at individual, whānau (family) and healthcare provider levels have been described, the core systemic barriers are rarely identified and discussed. Proposed solutions to poor access tend to ask providers to operate differently and/or for health pathways to be simplified, rather than address the main drivers of poor access to podiatry care.

METHOD

Kaupapa Māori and mixed-method approaches were weaved to undertake recorded focus groups with 21 Māori living with chronic disease and semi-structured engagements with 127 healthcare providers between 2021 and 2023. These conversations explored participants' perspectives on the barriers to podiatry care and how to improve the way it is provided.

FINDINGS

While podiatry care was easy to access for some Māori patients, it was the most difficult form of healthcare to access for others. Healthcare providers described a diversity of interconnected barriers that are overarching and across the referral pathways that hindered and delayed access. These barriers all emerge from the way podiatry care is structured

and funded in the Waikato Region, how primary and secondary care operate, and workforce shortages both regionally and nationally.

CONCLUSION

Barriers to podiatry care continue to be significant for Māori patients and can lead to poor outcomes including amputation. The current approach to podiatry care is equity negative and helps to increase inequities between Māori and non-Māori New Zealanders. Podiatry care must be re-designed to make it more accessible for Māori patients and whānau/families and must include stronger connections between secondary and community podiatrists.

IMPLEMENTATION OF STEREOTACTIC ABLATIVE RADIOTHERAPY (SABR) FOR LIVER LESIONS AT WAIKATO REGIONAL CANCER CENTRE (WRCC)

Jordan Mckenzie, Marina Izman, Omer Ali, John Chin, Komeela Naidoo

Department of Radiation Oncology, Te Whatu Ora Waikato, Hamilton, New Zealand

The Waikato Regional Cancer Centre (WRCC) has recently implemented stereotactic ablative radiotherapy (SABR) as a treatment option for metastatic liver lesions. SABR offers a non-invasive alternative to surgery, delivering high-dose radiation with minimal toxicity. While surgical resection remains the preferred treatment, approximately 25% of patients with liver lesions are eligible. This abstract outlines our implementation process for liver SABR.

A multidisciplinary approach was essential for implementation, involving the development of comprehensive patient management guidelines, including strict inclusion and exclusion criteria to ensure patients could tolerate treatment. Eligible patients underwent a pre-simulation quality assurance (QA) assessment, followed by treatment planning and delivery. Treatment involved up to five sessions over 1 to 2 weeks. Treatment planning was conducted using Varian Eclipse V16, with delivery on the Varian TrueBeam system. Each patient's treatment underwent rigorous quality control (QC) assessments performed by the medical physics team.

To date, four patients have been treated. The average gamma pass rate for quality control tests was 98% (2%, 2mm criteria). Full target coverage (100% of the prescribed dose) was often compromised due to the proximity of the liver to organs at risk (OAR), such as the bowel, heart and remaining liver parenchyma. Accurate targeting was further complicated by respiratory motion. However, advancements in imaging technology, including cone-beam CT (CBCT) and motion management strategies, have helped mitigate these challenges.

The liver SABR protocol is continuously reviewed and refined to optimise patient outcomes, with ongoing monitoring of efficacy and toxicity.

ADDISON'S DISEASE AND ADRENAL CRISIS: A PHENOMENOLOGICAL STUDY OF THE PATIENT EXPERIENCE

Sarah L Fostekew, MN,[1] Patrea R Andersen, PhD,[1,2,3] Isaac Amankwaa, PhD[4,5]

[1]Centre for Health and Social Practice, Waikato Institute of Technology, Hamilton, New Zealand
[2]School of Nursing, Midwifery and Social Science, Central Queensland University, Australia
[3]School of Health, University of the Sunshine Coast, Australia
[4]Health Sciences, Auckland University of Technology, Auckland, New Zealand
[5]Faculty of Health, University of Canberra, Australia

This study aimed to investigate the lived experiences and issues central to patients with Addison's disease when they are hospitalised due to an adrenal crisis. Interpretive phenomenology was used to explore and understand the experiences of six participants with a diagnosis of Addison's disease who had experienced one or more adrenal crises.

Their shared experiences were analysed using Braun and Clarke's thematic analysis.

Three key themes arose from the analysis that explained participants' experiences: responding, adjusting and learning. The "Addison's and adrenal crisis patient experience model" was developed to illustrate how responding, adjusting and learning impact the patient experience.

To support future patients with Addison's disease, this inquiry recommends education for healthcare practitioners in primary care and secondary services, particularly medical practitioners, nurse practitioners and nurses who are likely to aid treatment of adrenal crises.

The development of a support group for people living with Addison's disease to be able to connect and share experiences was suggested by the participants. This in conjunction with education programmes may prevent adrenal crises and hospitalisations.

The development of a clinical guideline is recommended to best support and instil confidence for healthcare practitioners, their patients and families.

AGITG SPAR: A RANDOMISED PLACEBO-CONTROLLED PHASE II TRIAL OF SIMVASTATIN IN ADDITION TO NEOADJUVANT CHEMOTHERAPY AND RADIATION FOR RECTAL CANCER

Michael B Jameson,[1,2] Kirsten Gormly,[3] David Espinoza,[4] Michael Arendse,[2] Derrick HW Siu,[4] Ailsa Langford,[4] Mark Jeffery,[5] Christos Stelios Karapetis,[6] Swetha Sridharan,[7] Matthew E Burge,[8] Anne O'Donnell,[9] Andrew Oar,[10] Angela Mweempwa,[11] James Armstrong,[12] Timothy Jay Price,[13] Samuel Y Ngan,[14] Frank Frizelle,[5] Andrew Stevenson,[8] Katrin Marie Sjoquist,[4] Stephen P Ackland[15]

[1]*Waikato Clinical Campus, The University of Auckland, Hamilton, New Zealand*
[2]*Waikato Hospital, Hamilton, New Zealand*
[3]*Dr Jones & Partners, Stepney, Australia*
[4]*NHMRC Clinical Trials Centre, University of Sydney, Camperdown, Australia*
[5]*Christchurch Hospital, Christchurch, New Zealand*
[6]*Flinders Medical Centre and Flinders University, Adelaide, Australia*
[7]*Calvary Mater Hospital, Newcastle, Australia*
[8]*Royal Brisbane & Women's Hospital, Brisbane, Australia*
[9]*Wellington Hospital, Wellington, New Zealand*
[10]*Icon Cancer Centre, Gold Coast University Hospital, Southport, Australia*
[11]*Te Whatu Ora Te Toka Tumai, Auckland, New Zealand*
[12]*Consumer Advisory Panel, AGITG, Sydney, Australia*
[13]*Queen Elizabeth Hospital, University of Adelaide, Woodville, Australia*
[14]*Peter MacCallum Cancer Centre, Melbourne, Australia*
[15]*University of Newcastle, Callaghan, Australia*

BACKGROUND

Retrospective clinical studies and preclinical studies demonstrated that statin use during preoperative (chemo)radiation (pCRT) for rectal cancer is associated with improved survival, response and toxicity. Tumour regression following pCRT has strong prognostic significance and can be assessed using MRI-based tumour regression grading (mrTRG), including with non-operative management. SPAR was designed to prospectively evaluate the benefits of adding simvastatin (SIM) to pCRT on tumour regression and gastrointestinal (GI) adverse events (AE).

METHODS

SPAR is a double-blind randomised phase 2 trial investigating SIM/placebo (PBO) in addition to long-course fluoropyrimidine-based pCRT for rectal adenocarcinoma. Stratification included trial site, clinical T stage (< 4 vs 4), clinical N stage (< 2 vs 2), either mesorectal fascia involvement (MRFI) or extramural venous invasion (EMVI) on MRI (yes vs no) and total neoadjuvant therapy (TNT): induction vs consolidation chemotherapy vs none. Study treatment was SIM 40mg/PBO daily for 90 days, starting 1 week prior to pCRT; recent statin use was excluded. Pelvic MRI was repeated 6–8 weeks after pCRT to determine mrTRG. An amendment in January 2022 allowed TNT with either induction or consolidation chemotherapy; the timing of postpCRT MRI remained unchanged. The design was amended to open-label due to PBO supply issues. Primary objective: rate of centrally assessed grade 1–2 mrTRG. Secondary objectives include centrally assessed favourable pathologic TRG (pathTRG), safety and cancer outcomes. Analysis was by intention-to-treat.

RESULTS

Between April 2018 and November 2023, 135 of 222 planned participants from 17 sites in Australia and New Zealand were randomised (68 SIM; 67 no SIM). Recruitment was hampered by the COVID-19 pandemic and adoption of TNT as standard care before the protocol amendment. Participant characteristics: median age 59 years; 85 (63%) males; 118 (87%) T2–3 disease, 80 (59%) N0–1 disease; 55 (41%) MRFI or EMVI; 25 (19%) had TNT. Rates of grades 1–2 mrTRG with SIM vs no SIM were 38.5% and 29.7% (X2=1.09, p=0.30), respectively. There was no significant difference in rates of grade 2 GI and non-GI AE. Four serious AE were recorded, none related to study treatment. Median follow-up was 3.2 years. Three-year local recurrence rates (LRR) were low: 1 (2.2%) and 3 (5.5%) with SIM vs no SIM, respectively (HR: 0.29, 95% Cl: 0.03–2.67, p=0.26). Three-year disease-free survival (DFS) with SIM vs no SIM was 84% and 72%, respectively (HR: 0.48, 95% Cl: 0.21–1.08, p=0.07).

CONCLUSIONS

The rates of favourable mrTRG, 3-year LRR and DFS were numerically better with the addition of SIM to pCRT, though the differences were not statistically significant. SIM was well tolerated. Interpretation is limited by reduced sample size and statistical power. PathTRG and other key outcomes will be presented at the meeting.

"DO YOU EAT BOIL UPS?": MĀORI PATIENT EXPERIENCES OF DIETETIC CARE AND NUTRITION SUPPORT AT WAIKATO HOSPITAL

Lynley Uerata,[1] Nina Scott,[1] Amy Jones,[1] Jade Tamatea,[1,2] Polly Atatoa-Carr,[1,3] Lynne

Chepulis,[4] Ryan Paul,[1,4] Haylee Simon,[1] Tia Dargaville,[1] Ross Lawrenson[4]

[1]Hauora Māori Services, Te Whatu Ora Waikato, Hamilton, Aotearoa New Zealand
[2]Faculty of Medical and Health Sciences, Te Kupenga Hauora Māori, The University of Auckland, Aotearoa New Zealand
[3]Te Ngira: Institute for Population Research, University of Waikato, Hamilton, Aotearoa New Zealand
[4]Waikato Medical Research Centre, University of Waikato, Hamilton, Aotearoa New Zealand

BACKGROUND

People living with chronic disease can experience many challenges around nutrition and malnutrition, which can be complicated by the cost of living and the way food systems operate. Dietitians have a role in supporting patients and whānau/family through these challenges and by providing evidence-based nutrition support about nutrition and food. This presentation describes the experiences of Māori patients in relation to kai/food and when engaging with dietitians at Waikato Hospital.

METHOD

Kaupapa Māori and mixed method approaches were used to undertake 1) recorded focus groups with 21 Māori patients living with chronic disease and 2) semi-structured interviews with four additional Māori patients about their experience of speaking to a dietitian at Waikato Hospital. The former patients were asked about the wider determinants of health affecting their lives, including the experiences of food, nutrition and speaking to a dietitian. The latter patients were asked: How was your experience of speaking to a dietitian? How did they help you?

RESULTS

Participants valued dietetic care and nutrition support that was specific to their health condition and when healthcare providers teamed up to talk through related issues. Some advice was difficult for patients and whānau to connect to for economic, whānau and cultural reasons. Some patients felt their dietitian did not know how to approach kai Māori (Māori food practices) and some kai were targeted as "bad" from a scientific perspective. While support was patient-centred, whānau and/or household members were largely excluded. Economic constraints and high living costs also posed significant challenges for some whānau. These factors made it difficult to learn to cook and eat to a different diet.

CONCLUSION

Many individual, cultural, whānau, economic and societal factors shape everyday experiences of food and nutrition for Māori living with chronic disease. Understanding how these affect nutrition and everyday disease management is important to providing useful dietetic care and nutrition support. Whānau experiences show that dietetic care and nutrition support must be re-designed to make it more accessible and useful to patients and whānau. Increasing the number of Māori dietitians and integrating cultural perspectives at the training levels will help improve patient and whānau experiences.