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Let the muppets out

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Metabolic risk factors and long COVID: a cross-sectional study in Aotearoa New Zealand

Bailey Yee, Fiona McKenzie, Lis Ellison-Loschmann, Lynne Russell, Mona Jeffreys

This study looks at the metabolic risk factors for long COVID in Aotearoa New Zealand, using nationwide data from the *Ngā Kawekawe o Mate Korona | Impacts of COVID-19 in Aotearoa* study. It found that having a high body mass index (BMI of 25kg/m^2 or more) but no other metabolic illnesses such as high blood pressure or diabetes was associated with a higher likelihood of developing long COVID. This research suggests that long COVID should be considered as an immune-metabolic disease, which could lead to new possibilities for treatments in the future.

"Call-it-in": addressing older informal caregivers' support needs during the COVID-19 pandemic in Aotearoa New Zealand

Tyrone Barnard, Shinya Uekusa, Christine Stephens, Fiona Alpass

During the COVID-19 global pandemic, many older informal caregivers in Aotearoa New Zealand struggled to find and access the support they needed. Despite the availability of online resources, many participants preferred phone-based assistance, which provided emotional support and was easier to use. This study introduces the concept of "call-it-in", a centralised phone-based support system that would connect caregivers to essential, both existing and emerging, services and information. By offering a single, accessible point of contact, "call-it-in" could reduce stress, improve social connections and help informal caregivers navigate challenges more effectively during crises. Implementing such a system could ensure that informal caregivers receive timely and relevant support, reducing their reliance on personal resources and networks alone.

Common mental disorders and psychological distress among Pacific adults living in Aotearoa New Zealand

Joanna Ataera-Minster, Susanna Every-Palmer, Ruth Cunningham, Jesse Kokaua

This study combined data from multiple New Zealand Health Survey waves and analysed mental health outcomes in Pacific adults aged 15 years and over. Results showed mental health differs between specific Pacific ethnic groups, with doctor-diagnosed common mental disorders (depression and anxiety) being more common in Cook Island Māori and Niueans than other Pacific ethnic groups. The limitations of the measures used mean it is difficult to know whether these results indicate higher needs in these Pacific subpopulations and/or better access to services. Routine monitoring of mental health with structured diagnostic interviews is needed to better identify Pacific subpopulations with higher needs for targeted mental health promotion.

Capturing diversity in cancer incidence and outcomes among the New Zealand Pacific population using linked administrative data

Nicole Satherley, Andrew Sporle

Health outcomes such as cancer incidence and mortality are usually reported on for Pacific peoples as a single ethnic group. This study accounted for potential diversity in health outcomes among specific Pacific ethnicities, such as Samoan, Tongan and Niuean ethnicities. It showed variation in all-cancer and gastric cancer incidence between these specific ethnicities that would be hidden when examining incidence within the aggregated Pacific group.

Differences in systemic treatments for breast cancer between patients with and without diabetes

Chunhuan Lao, Jason Gurney, James Stanley, Andrea Teng, Marion Kuper-Hommel, Ian Campbell, Jeremy Krebs, Dianne Sika-Paotonu, Jonathan Koea, Jeannine Stairmand, Ross Lawrenson

Women with diabetes are less likely to be treated with chemotherapy for their breast cancer than women without diabetes. The difference in use of chemotherapy between the two groups was greatest in Pacific women and patients with stage II breast cancer. The lower usage of endocrine therapy and HER2-targeted therapy in patients with diabetes could be explained by the older age at diagnosis and more comorbidities in women with diabetes.

Ovarian torsion: determining the presenting features and where the delays occur

Karan Bedekar, Anna McInnes, Wendy Burgess

Our paper looks at the common presenting symptoms of ovarian torsion patients at a New Zealand tertiary hospital and the rates of ovarian conservation versus loss of ovary. Additionally, we looked at how long it took these patients to present to hospital, be seen by a gynaecologist, receive appropriate imaging and finally reach the operating theatre. This helped identify where the greatest delays are occurring, which can assist in expediting future management. The study's ultimate purpose is to facilitate the development of clinical guidelines, which will aid clinicians in better identifying ovarian torsion while promoting ovarian conservation.

Drug driving, sedation, reaction time and blood levels: a prescriber's approach to the Land Transport (Drug Driving) Amendment Act 2022

Marleen van Oeveren, Paul Glue, Charlotte Mentzel

The new *Land Transport (Drug Driving) Amendment Act* (*LTAA*) went into effect in 2023, and it will affect people driving on 25 prescription medications, particularly benzodiazepines, opioids and THC. Driving on these medications is about as dangerous as driving a black or green car.

If patients take medication according to prescription, they are exempt from charges. However, there is no guidance for prescribers on how to interact with the *LTAA* and there is currently no information on if they will face criticism by the Medical Council of New Zealand, the Health and Disability Commissioner or the Coroner if they prescribe these medications.

Cryogenic burns to the upper aerodigestive tract following recreational nitrous oxide inhalation

Matt McCall, Hayleigh Miller, Samuel JM Hale, Rebecca Field

Nitrous oxide (N_2O) is publicly available, and its recreational inhalation is becoming more common.

It is most often from pressurised canisters for cream whippers (commonly referred to as “nangs”). Breathing it in gives a sense of brief euphoria or dissociation. This can result in burns to the mouth and throat, which can lead to potentially life-threatening complications. There have been legislative changes regarding its use and distribution in Aotearoa New Zealand recently; however, it may be that improving awareness of such risks, rather than prohibition, may minimise potential harms.

A pain in the hip: the under-used potential of fascia iliaca compartment block in the prehospital setting

Sarah E Maessen, Jon Leach, Verity F Todd, Elena Garcia, Bridget Dicker

In late 2020, New Zealand ambulance guidelines introduced a technique called fascia iliaca compartment block (FICB) to treat hip fracture pain by blocking nerves with an anaesthetic injection. This study looked at almost 4,000 patients treated by Hato Hone St John ambulance personnel for hip fracture over a 1-year period and found that FICB was rarely used. The 3.6% of patients who did receive FICB had greater reductions in pain scores than those with similar injuries and no FICB. This study provides evidence that FICB is a safe and effective pain relief option in the pre-hospital setting but suggests that the restriction of this skill to a subset of paramedics with advanced training has resulted in low use of FICB.

Let the muppets out

Frank Frizelle, Roger Mulder

The present Minister of Health, Mr Simeon Brown expressed concern that public health officials were spending time on health advocacy when they should be responding to the concerns of the minister and ministry officials. This opinion was supported by the then Acting Prime Minister David Seymour, who hit back at concerns about Mr Brown's comments, saying he's "*cheering on Simeon* [Brown] *putting those muppets back in their box*."[1] Mr Seymour's comments show a lack of respect that borders on bullying for the medical profession as well as inferring that the opinion of the "muppets" was not valued.

Who are these muppets? We believe he was referring to public health physicians, who are considered by many in the medical profession to be the intelligentsia of medicine. As a group they spend much of their time teaching and advising the government and the community on how to avoid illness and thereby reduce healthcare costs.

Criticism of this section of the medical community is important, especially coming from highly ranked government officials. Their comments may be seen as a direct attack on the free speech and open discussion we value as a profession. It also suggests that political leaders do not value or respect public health doctors working in Health New Zealand – Te Whatu Ora. This would, however, be in direct contrast to the earlier views expressed by Mr Seymour in his statement *Strengthening Free Speech in Universities*, released on 19 December 2024. Here he stated, "*That's why the National/ACT coalition agreement committed to introduce protections for academic freedom and freedom of speech to ensure universities perform their role as the critic and conscience of society*."[2] Though he was talking about university staff at that time, his point appears to support open discussions about issues.

These restrictions on freedom of speech are not confined to New Zealand—similar but more severe restrictions are being propagated in the United States of America (USA). The Trump administration has launched an all-out attack on the health and medical research community in the post-COVID space. The USA Government's retreat from international health partnerships, combined with substantial budget reductions, poses real threats to USA and global health security.

The use of certain terms is banned on USA government websites (and in manuscripts submitted to scientific journals), including "gender", "transgender", "LGBT" and "non-binary", and a directive has paused the submission of new work for publication for all Centre for Disease Control (CDC) employees and contractors. These measures have been reported to have an effect on medical journal processes outside of the USA, with journals such as the *Lancet* reporting that the impact has already been felt, with reviewers declining and authors self-censoring.[3] This politicisation of science has resulted in some researchers' ability to work being limited or stopped altogether and is being interpreted that "free speech" is restricted.

Freedom of speech and expression, especially discussion about public issues such as health, is important in any democracy. A democracy depends upon a literate, knowledgeable population who access information that allows them to participate fully in the public life of society. This includes criticising unwise or tyrannical government officials or policies. Citizens and elected representatives should recognise that democracy depends upon the widest possible access to ideas, data and opinions. The right to freedom of expression is enshrined in the *New Zealand Bill of Rights Act 1990*, which states "*Everyone has the right to freedom of expression, including the freedom to seek, receive and impart information and opinions of any kind in any form*."[4] Perhaps government ministers should ponder the bill of rights before taking up their posts.

Those who work for the government as employees of Health New Zealand – Te Whatu Ora also have this enshrined in their employment contract:[5]

Section 40: Public Debate and Dialogue

40.1 *In recognition of the rights and interests of the public in the health service, the employer respects and recognises the right of its employees to comment publicly and engage in public debate on matters relevant to their professional expertise and experience.*

40.2 *In exercising this provision employees shall, prior to entering into such public debate and dia-*

logue, where this is relevant to the employer, have advised and/or discussed the issues to be raised with the employer.

Those who work in the university system within the health sector or in joint clinical contacts have similar rights. The University of Otago statement[6] is the clearest and—we suspect—speaking out is actually an obligation.

University of Otago Statement on Free Speech

Free speech is the lifeblood of a university. It enables the exploration of ideas, the challenging of assumptions, and the uncovering of truth through open exchange. It allows students, teachers and researchers to know better the variety of beliefs, theories and opinions in the world. Only through a preparedness to challenge, question, and criticise ideas can progress in understanding take place. Consistent with its motto Sapere Aude, 'dare to be wise' and the ikoa Māori Ōtākou Whakaihu Waka, the University of Otago is committed to the fearless pursuit of knowledge driven by māhirahira (curiosity) and bounded by pono (integrity). That commitment requires a wide range of views to be freely presented, discussed and debated.

The University affirms that it will not restrict debate or deliberation simply because the ideas put forth are thought by some to be offensive, unwise, immoral, or wrong-headed. It is for the members of the University community – its students and staff – to make those judgments for themselves. The University is not a place for safety from ideas – it is a place to engage in critical thought and debate in the pursuit of knowledge and understanding. Our students will not be prepared for a complex and challenging world unless they have experience negotiating conflict and disagreement.

The University therefore guarantees all members of its community, including invited visitors, the right to advance ideas in the spirit of free and open enquiry. Furthermore, in its role as critic and conscience of society, the University provides a space in which contrary and unpopular positions can be advanced free from political interference or suppression.

This commitment to free speech does not mean that any utterance is appropriate in a university setting. The University may properly restrict expression which violates the law. Moreover, the University accepts no duty to provide a space for those who are not members of its community to advance their ideas or theories in ways which fundamentally undermine the University's character as an institute of higher learning. The University may also reasonably regulate the time, place, and manner of expression to ensure that it does not disrupt the ordinary activities of the University.

The University's support for free speech carries with it corollary responsibilities. Although students, staff, and visitors are free to criticise, contest, and condemn the views expressed on campus, they should not obstruct, disrupt, or otherwise interfere with the freedom of others to express those views.

The University challenges members of its community and invited visitors to be tolerant of the diversity of identities and beliefs of others. We encourage debate in good faith guided by the principles of manaakitaka (care and respect for others).

This statement was ratified on 9 July 2024, to reiterate the University's solemn and long-standing responsibility not only to promote lively and fearless freedom of debate and deliberation, but also to protect that freedom when others attempt to restrict it. Portions of the statement were adapted from the University of Chicago's Report of the Committee on Freedom of Expression (January 2015) and the Model Code advanced as part of the Independent Review into Freedom of Speech in Australian Higher Education Providers (April 2019), the University of Oxford's Statement on Free Speech (August 2016), and the University of Cambridge's Statement on Free Speech (December 2020).

While The University of Auckland statement[7] is difficult to find and appears to be in draft form on their website, we suspect it will end up expressing similar ideas.

Restrictions on debate and discussion of health issues and advocacy should be called out for the damage they cause. It does leave one wondering, however, who the real muppets are.

COMPETING INTERESTS
Nil.

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Metabolic risk factors and long COVID: a cross-sectional study in Aotearoa New Zealand

Bailey Yee, Fiona McKenzie, Lis Ellison-Loschmann, Lynne Russell, Mona Jeffreys

ABSTRACT

AIM: To describe the association between metabolic risk factors and the risk of developing long COVID in Aotearoa New Zealand.

METHODS: Individuals aged 16 years and above who had confirmed or probable COVID-19 before December 2021 were eligible for inclusion. Metabolic risk factors were high body mass index (BMI, ≥25kg/m^2), high blood pressure, diabetes, heart disease and stroke. Logistic regression was used to estimate the association between metabolic risk factors and long COVID.

RESULTS: Of the 990 survey respondents, 21.9% met the definition of long COVID. After adjusting for socio-demographic factors, COVID-19 vaccination and hospitalisation, high BMI was strongly associated with long COVID (adjusted odds ratio [aOR] 2.35; 95% confidence interval [CI] 1.33–4.17, p=0.003). There was a suggestion of an association between heart disease and long COVID (aOR 4.31; 95% CI 0.80–23.3, p=0.090). No other metabolic factors were associated with long COVID. Among Māori, no associations were found between high BMI and long COVID compared with underweight/normal BMI.

CONCLUSION: High BMI as a risk factor adds to accumulating evidence on the aetiology of long COVID.

Many studies have reported participants experiencing prolonged, relapsing or exacerbated COVID-19 symptoms.[1] "Long COVID" is a term used to refer to ongoing or new symptoms 3 months after the initial onset of COVID-19, with symptoms that last for at least 2 months and cannot be explained by an alternative diagnosis.[2] Other labels that refer to long COVID have been used interchangeably, such as "post-COVID syndrome", "persistent COVID", "chronic COVID" and "long haul COVID".[3] The international incidence of long COVID has been estimated to be between 4 and 14% per infection,[4,5] with similar results demonstrated in Aotearoa New Zealand.[6] Long COVID can affect various organ systems, with manifestations involving the lungs, brain and cardiovascular systems.[3] Even those with mild presentations of COVID-19 can experience medium- to long-term impacts.[3]

Metabolic syndrome includes interrelated cardiometabolic abnormalities such as insulin resistance, abdominal obesity, elevated blood pressure, atherogenic dyslipidaemia (hypertriglyceridemia and/or low high-density lipoprotein cholesterol) and is a significant determinant of the global burden of cardiovascular disease.[7] These abnormalities can affect an individual's immune response,[8] and illnesses, including high blood pressure, diabetes, heart disease and obesity, have been reported to be associated with COVID-related hospitalisations and mortality.[9,10] These same illnesses have also been reported as determinants of long-term morbidity in other infectious diseases, suggesting a possible role as risk factors in long COVID.[8,11]

Previous international literature has reported a higher risk of long COVID in those with a high body mass index (BMI).[11–13] A systematic review and meta-analysis found that a BMI of ≥30kg/m^2 was associated with long COVID (odds ratio [OR] 1.15; 95% confidence interval [CI] 1.08–1.23) compared with a BMI of <30kg/m^2, with the associations present in both those hospitalised with COVID-19 and those not.[14] Similarly, a second meta-analysis identified obesity and diabetes as potential risk factors for long COVID.[15] Obesity, in particular, was associated with poorer health than that of non-obese participants due to a higher number of long COVID symptoms, longer symptom persistence and a higher prevalence of pathological pulmonary limitations and metabolic abnormalities.[15]

Despite these findings, our understanding of the pathogenesis of long COVID remains limited. The pattern of COVID-19 illness in Aotearoa New Zealand was different to overseas experiences, particularly the high rates of COVID-19 vaccination before infection. To date, no studies have examined metabolic risk factors associated

with long COVID in Aotearoa New Zealand. This presents an opportunity to gather national evidence specific to the population, including evidence specific to Māori.

Methods

The analysis used data from the nationwide *Ngā Kawekawe o Mate Korona | Impacts of COVID-19 in Aotearoa* study. Full details have been previously reported.[16] Briefly, those over the age of 16 years who had confirmed or suspected COVID-19 before December 2021 and were not living in a dementia unit were eligible for inclusion.[16] The Ministry of Health (MOH) sent letters of invitation to participate in the study, followed by two reminder SMS (text messages). A Tiriti o [Treaty of] Waitangi Relationship Framework underpinned the research, based on the assertion that co-governance between Māori and non-Māori is fundamental to achieving positive research outcomes.[16] Quantitative data were collected via four online survey modules between February and June 2022 through the study's website (www.covidaotearoa.com). The survey topics comprised: a) support and wellbeing, b) health and health services, c) cost of COVID-19, and d) long COVID (on which this analysis is based). All survey modules were available to complete concurrently, and participants were not required to complete every survey module. If participants preferred, surveys could be completed over the phone with a research team member.

Outcome and exploratory variables

The presence of metabolic risk factors was self-reported. Participants were asked if they had a pre-existing diagnosis of high blood pressure, heart disease, diabetes or stroke. A participant's BMI was calculated from self-reported height and weight and was categorised as "high" (≥25kg/m^2) or "underweight/normal" (<25kg/m^2). The primary outcome was the presence of COVID-19 and/or long COVID symptoms that persisted for 3 or more months from the time of acute illness ("long COVID"), as identified from a predefined list.[17,18]

We assessed the role of the following sociodemographic factors as possible confounders in multivariate regression models: age group, prioritised ethnicity (Māori, Pacific, Asian and Other),[19] gender, education and pre-existing disability before contracting COVID-19 ("pre-COVID disability").[20] Income struggle, defined as a self-reported struggle within the household to pay for essential living costs such as food, bills or accommodation in the first month of having COVID-19,[21] and household crowding, defined as more than two people per bedroom,[22] were used as markers of socioeconomic position. We also considered the potential confounding effect of having had at least one dose of the COVID-19 vaccine and hospital admission for COVID-19.

Missing data manipulation

For binary variables such as household crowding, pre-COVID disability, high blood pressure, diabetes, heart disease and stroke, any missing data were analysed with the unexposed group. For categorical variables, missing information was recoded into the largest existing group. As few participants did not specify their gender or identified as "Other gender" (<1%), these people were analysed with the "female" group. However, as nearly a third of participants (n=126/405, 31.1%) did not answer the question concerning income struggle, missing data for this variable were treated as a separate category.

A third of participants (n=137/405, 33.8%) did not report their height and/or weight for BMI calculations; we used three approaches to deal with missing data. In our *a priori* main analysis, those with missing height and/or weight were treated as a distinct category. Then, a sensitivity analysis was undertaken to account for possible misclassification in the reporting of BMI. In one scenario, all participants with missing data were analysed with the underweight/normal BMI group; in a second scenario, those with missing data were analysed with the high BMI group.

Statistical analyses

Percentages of each category of all variables were tabulated separately for people with and without long COVID. Logistic regression was used to estimate crude ORs and 95% CIs of the association between possible risk factors and long COVID. This was followed by a multivariable model adjusting for all potential confounders, for which we have the data simultaneously. Analyses were conducted using Stata (Version 16). Conventional levels of statistical significance were defined as P<0.05; however, due to the small numbers of people in some categories, focus was placed on the magnitude of effect sizes.

Ethics

Ethical approval was given by the Health and Disability Ethics Committee on 15 January 2022 (ref: 2021, EXP 11900). An amendment was

approved on 26 April 2022 (ref: 2022 AM 11900).

Results

Letters of invitation were estimated to have been delivered to 8,012 of the 8,735 participants whose positive COVID-19 test had been reported to the MOH before December 2021. Fourteen percent (n=1,227) of those who received an invitation began the survey, and 990 participants answered at least one of the four available survey modules, equating to a response rate of 12.4%.

Accounting for all people identified by the MOH who had a positive COVID-19 test before December 2021, 2.5% (n=217/8,737) reported long COVID. Of those who answered at least one survey, 21.9% (n=217/990) reported long COVID. This analysis is based on those participants who answered the long COVID survey module (n=405/990, 40.9%), of whom 53.6% (n=217/405) reported long COVID. A flow diagram of the study recruitment process is presented in Figure 1.

Characteristics of the participant cohort on which this analysis is based are shown in Table 1. Most participants with long COVID identified as female (71.9) and were mostly between 25 and 44 years (41.5%). Nearly half with long COVID (46.9%) were classified as having a BMI of ≥25kg/m^2. The risk of long COVID in Pacific peoples was lower than in the "Other" ethnic group, which primarily consisted of NZ Europeans. There was an apparent protective effect of having at least one COVID-19 vaccination and the risk of developing long COVID compared with those not vaccinated.

Associations between metabolic risk factors and long COVID are shown in Table 2.

Figure 1: Flow diagram of study participant recruitment process.

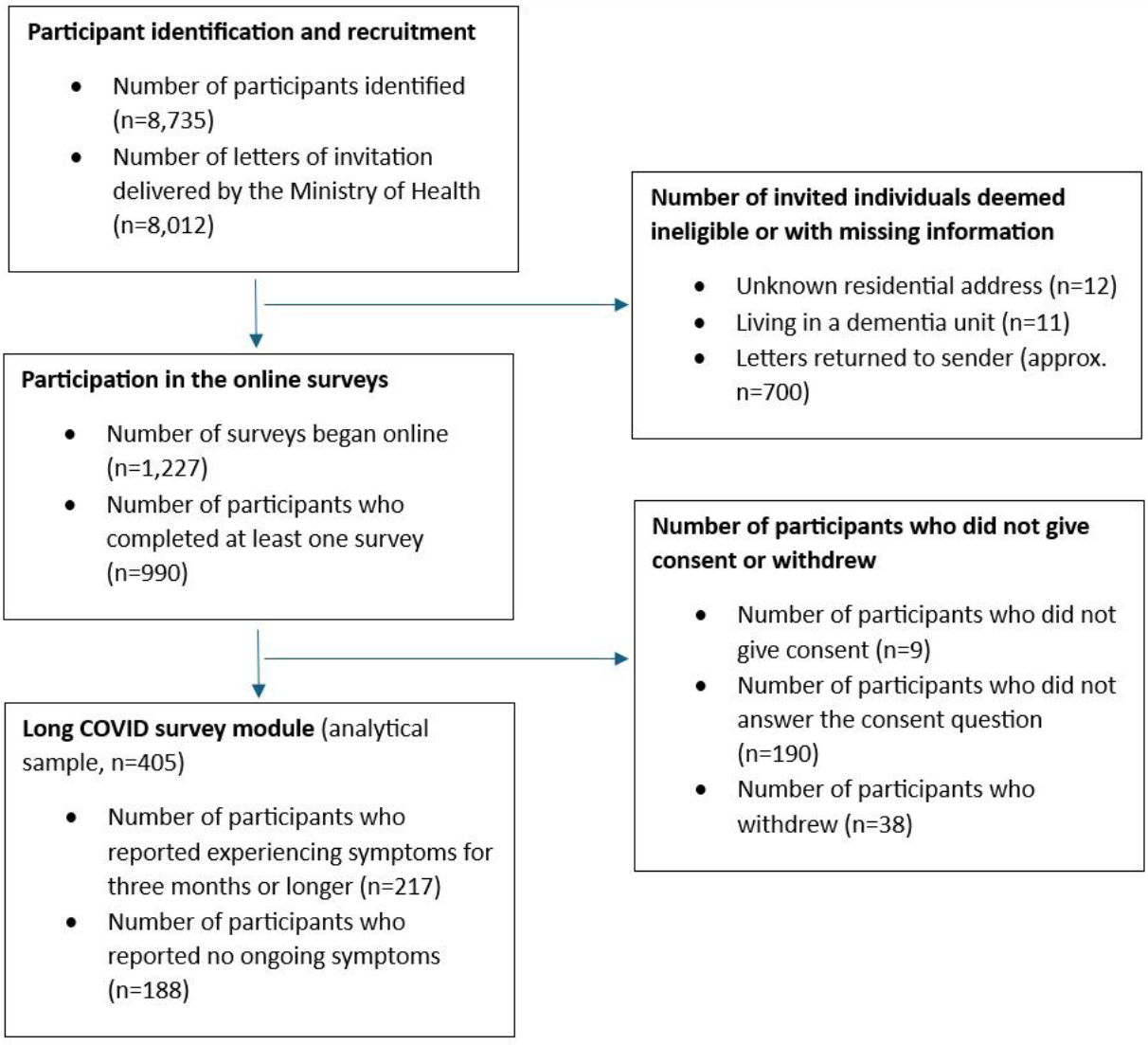

Table 1: Participant characteristics and the association between exposure variables and long COVID among 405 people in Aotearoa New Zealand.

Variable	Category	LC (%) n=217	No LC (%) n=188	OR (95% CI)	p-value
Age	15–24	21 (9.7)	21 (11.2)	0.89 (0.45–1.75)	0.73
	25–44	90 (41.5)	80 (42.6)	Reference	
	45–64	86 (39.6)	67 (35.6)	1.14 (0.74–1.77)	0.56
	65+	20 (9.2)	20 (10.6)	0.89 (0.45–1.77)	0.74
Gender	Male	61 (28.1)	60 (31.9)	Reference	
	Female	156 (71.9)	128 (68.1)	1.20 (0.78–1.84)	0.40
Ethnicity	Māori	33 (15.2)	28 (14.9)	0.90 (0.52–1.57)	0.72
	Pacific peoples	3 (1.4)	15 (8.0)	0.15 (0.04–0.54)	0.004
	Asian	9 (4.2)	13 (6.9)	0.53 (0.22–1.28)	0.16
	Other	172 (79.3)	132 (70.2)	Reference	
Education	School	46 (21.2)	38 (20.2)	1.02 (0.62–1.68)	0.94
	Post-school	44 (20.3)	43 (22.9)	0.86 (0.53–1.41)	0.56
	University	127 (58.5)	107 (56.9)	Reference	
Income struggle	Strongly agree/ agree	29 (13.4)	24 (12.8)	1.19 (0.65–2.21)	0.57
	Neither agree nor disagree	25 (11.5)	22 (11.7)	1.12 (0.59–2.14)	0.72
	Disagree/ strongly disagree	90 (41.5)	89 (47.3)	Reference	
	Missing	73 (33.6)	53 (28.2)	1.36 (0.86–2.16)	0.19
Household crowding	Overcrowding	10 (4.6)	12 (6.4)	0.71 (0.30–1.68)	0.43
	None	207 (95.4)	176 (93.6)	Reference	
Pre-COVID disability	Pre-COVID disabled	23 (10.6)	13 (6.9)	1.60 (0.78–3.25)	0.20
	Non-disabled[i]	194 (89.4)	175 (93.1)	Reference	
COVID Vaccination	Vaccinated	126 (58.1)	123 (65.4)	0.73 (0.49–1.10)	0.13
	Unvaccinated	91 (41.9)	65 (34.6)	Reference	
Hospitalisation with COVID	Hospitalised	23 (10.6)	19 (10.1)	1.05 (0.56–2.00)	0.87
	Not hospitalised	194 (89.4)	169 (89.9)	Reference	

LC = long COVID; OR = odds ratio; 95% CI = 95% confidence interval.
[i]Non-disabled cohort also includes participants who identified as having a disability that began after COVID-19 infection (n=30).

Table 2: Crude association between metabolic risk factors and long COVID among 405 people in Aotearoa New Zealand.

Variable	Category	LC (%) n=217	No LC (%) n=188	OR (95% CI)	p-value
BMI	High (≥25kg/m^2)	104 (47.9)	86 (45.7)	2.16 (1.25–3.72)	0.006
	Underweight/ normal (<25kg/ m^2)	28 (12.9)	50 (26.6)	Reference	
	Missing	85 (39.2)	52 (27.7)	2.92 (1.64–5.20)	<0.001
High blood pressure	Yes	11 (5.1)	9 (4.8)	1.06 (0.43–2.62)	0.90
	No	206 (94.9)	179 (95.2)	Reference	
Diabetes	Yes	6 (2.8)	5 (2.7)	1.04 (0.31–3.47)	0.95
	No	211 (97.2)	183 (97.3)	Reference	
Heart disease	Yes	7 (3.2)	2 (1.1)	3.10 (0.64–15.11)	0.16
	No	210 (96.8)	186 (98.9)	Reference	
Stroke	Yes	1 (0.5)	1 (0.5)	0.87 (0.05–13.94)	0.92
	No	216 (99.5)	187 (99.5)	Reference	

LC = long COVID; OR = odds ratio; 95% CI = 95% confidence interval; BMI = body mass index.

Study participants with a BMI of ≥25kg/m^2 had over twice the risk of developing long COVID than those with a BMI of <25kg/m^2. Participants who did not report their height and/or weight had nearly a three times higher risk of developing long COVID compared with those with a BMI of <25kg/m^2. It was suggested that having pre-existing heart disease was associated with a higher likelihood of developing long COVID. However, this did not reach conventional levels of statistical significance. No other associations between socio-demographic variables and long COVID were identified in our crude analyses.

After adjusting for all explanatory variables shown in Table 1, there remained a strong positive association between BMI and the risk of long COVID. Participants with missing BMI data also appeared to have a higher risk than those with underweight/normal BMI (see Table 3). It was suggested that participants with pre-existing heart disease were more likely to develop long COVID than those who did not have heart disease. However, this did not reach conventional levels of statistical significance (see Table 3). When comparing very high BMI (≥30kg/m^2) to underweight/normal BMI, the association showed similar effects to those observed when comparing high BMI (≥25kg/m^2 to 30kg/m^2) to underweight/normal BMI (for "very high BMI': adjusted odds ratio [aOR] 2.26; 95% CI 1.17–4.39, and for "high BMI": aOR 2.43; 95% CI 1.29–4.58).

There was a significant degree of missing data in the BMI variable. Nearly 40% (n=85/217, 39.2%) of participants with long COVID did not report their height and/or weight in the survey. Participants who did not report their height and/or weight had a higher risk of developing long COVID than those who did report their height and weight (Appendix Table 1).

Most measures of socio-economic position were not related to missing BMI data (Appendix Table 2). However, there was weak evidence suggesting that participants who reported income struggle were more likely not to report their height and/or weight compared with those who reported no income struggle. Furthermore, participants who did not indicate their level of income struggle were significantly more likely not to report their

Table 3: The association between metabolic risk factors and long COVID (multivariable logistic regression model).

Variable	Category	*aOR (95% CI)	p-value
BMI	High BMI (≥25kg/m^2)	2.35 (1.33–4.17)	0.003
	Underweight/normal (<25kg/m^2)	Reference	
	Missing	5.46 (1.74–17.2)	0.004
High blood pressure	Yes	1.34 (0.50–3.60)	0.56
	No	Reference	
Diabetes	Yes	1.52 (0.41–5.62)	0.53
	No	Reference	
Heart disease	Yes	4.31 (0.80–23.3)	0.090
	No	Reference	
Stroke	Yes	1.02 (0.05–18.69)	0.99
	No	Reference	

aOR = adjusted odds ratio; 95% CI = 95% confidence interval; BMI = body mass index.
*Adjusted for age, gender, prioritised ethnicity, education, income struggle, overcrowding, pre-COVID disability, COVID-19 vaccine and COVID-related hospitalisation.

Table 4: Sensitivity analysis for BMI and the risk of long COVID.

	Treatment of missing BMI data	OR (95% CI)	p-value	*aOR (95% CI)	p-value
Scenario 1	Underweight/ normal/missing BMI	Reference		Reference	
	High BMI	1.09 (0.74–1.61)	0.66	1.73 (1.02–2.93)	0.042
Scenario 2	Underweight/ normal BMI	Reference		Reference	
	High/missing	2.45 (1.47–4.08)	0.001	2.53 (1.44–4.45)	0.001

BMI = body mass index; OR = odds ratio; 95% CI = 95% confidence interval; aOR = adjusted odds ratio.
*Adjusted for age, gender, prioritised ethnicity, education, income struggle, overcrowding, pre-COVID disability, COVID-19 vaccine and COVID-related hospitalisation.

Table 5: BMI and the risk of long COVID stratified by Māori/non-Māori.

Variable	Category	Māori (%)	OR (95% CI)	p-value	Non-Māori (%)	OR (95% CI)	p-value
BMI	High BMI	32 (52.5)	1.70 (0.25–11.59)	0.59	158 (45.9)	2.22 (1.25–3.93)	0.006
	Under-weight/ normal BMI	5 (8.2)	Reference		73 (21.2)	Reference	
	Missing	24 (39.3)	2.10 (0.29–14.98)	0.66	113 (32.9)	3.06 (1.66–5.64)	<0.001

BMI = body mass index; OR = odds ratio; 95% CI = 95% confidence interval.

height and/or weight than those who reported no income struggle.

The primary analyses presented above estimated the association between BMI and long COVID with missing BMI data treated as a separate category. We conducted a sensitivity analysis in which missing BMI data were recategorised with the underweight/normal BMI (<25kg/m^2) group (scenario one) and with the high BMI (≥25kg/m^2) group (scenario two); see Table 4. In the fully adjusted multivariable model, the most conservative estimate of risk between BMI and long COVID (scenario 1) found that participants with a BMI of ≥25kg/m^2 remained more likely to develop long COVID than those with a BMI of <25kg/m^2. The least conservative estimate of risk between BMI and long COVID (scenario 2) showed that participants with a high BMI had a higher risk of developing long COVID than those with an underweight/normal BMI. From these estimations, it appears that the association between BMI and long COVID is not explained by the degree of missing data in our study.

Sub-group analysis by Māori/non-Māori

We repeated the analyses stratified by Māori/non-Māori ethnicity. Over half (52.5%) of Māori were identified as having a high BMI compared with 45.9% of non-Māori (p=0.058) (see Table 5). When treating missing BMI data as a distinct category, among Māori no association was found between BMI and long COVID. Similarly, there was no evidence of an association among Māori who did not report their height and/or weight and long COVID. Among non-Māori, participants with a high BMI had over a two times higher likelihood of developing long COVID than those with an underweight/normal BMI. Similarly, non-Māori participants who did not report their height and/or weight had over a three times higher risk of developing long COVID compared with non-Māori with an underweight/normal BMI.

Using a formal test of interaction, there was no evidence to reject the null hypothesis that the effect of high BMI on long COVID differs between Māori and non-Māori (p=0.73).

Discussion

Our analyses demonstrate a strong association between high BMI and long COVID, but found no evidence of an association between other metabolic risk factors and long COVID. This finding suggests that the association between obesity and long COVID may be mediated by a mechanism other than the metabolic pathways that we measured, although the small numbers of people with diagnosed metabolic illnesses may have precluded our ability to detect real results. As the pathophysiology of long COVID is still poorly understood, this study provides epidemiological evidence to strengthen the accumulating biomedical evidence of the immune-metabolic disruption evident in people with long COVID.

To our knowledge, this is the only study in Aotearoa New Zealand to investigate the association between metabolic risk factors and long COVID. Rates of metabolic disease and obesity are high in Aotearoa New Zealand, particularly among Māori and Pacific populations; being able to report associations among Māori is an important strength of this study.[7,23,24]

The definition of long COVID used within this research was based on self-reported ongoing symptoms for 3 months or longer. As recognised in other international literature, there is no unified consensus on the definition of long COVID, especially concerning the duration of persistent symptoms, which span from 4 to 12 weeks following COVID-19 infection.[2] This limitation impedes the ability to compare findings with other studies. Moreover, as reported in the qualitative analysis of *Ngā Kawekawe o Mate Korona*, many interviewees, disproportionately Māori and Pacific peoples, attributed their ongoing symptoms to underlying conditions.[6] This difficulty in understanding and interpreting health information may contribute to an under-counting of long COVID cases among Māori and Pacific peoples (i.e., the misclassification of outcome). Similarly, considering the well-described barriers to healthcare access and experiences of institutional racism within Aotearoa New Zealand's health system,[25] it is likely that self-reported diagnoses of metabolic illnesses are disproportionately under-reported by Māori and Pacific peoples. Both biases of under-reporting and under-counting may impede our ability to identify associations between metabolic factors and long COVID.

Moreover, the 12% response rate could have resulted in a biased estimate of long COVID prevalence. In particular, the prevalence of long COVID could be over-estimated as this was the first national research conducted in this area, and people with a "new" condition were more likely to take part. The estimated prevalence of long COVID in our study ranged from 2.5% (based on a denominator of all eligible people) to 21.9% (based on a denominator of all respondents). While there is no national estimate for long COVID prevalence, international studies suggest a range between 4 and 14%,[4,5] which is consistent with our findings.

While response bias likely over-inflates the prevalence estimate, it is unlikely to have biased the association between high BMI and long COVID. Since the survey was available to anyone who had COVID-19 prior to December 2021, differential participation by both BMI and long COVID status is unlikely to have occurred. Although ORs are an appropriate measure of relative risk when the outcome is rare, the high prevalence of long COVID in our sample means that the use of logistic regression to estimate ORs may have inflated the estimated association.

Although widely used in population-based studies, BMI is limited as an accurate measure of obesity, with differential associations with obesity across genders, ages and ethnicities.[26] This is due to BMI not discriminating between levels of muscle mass, bone density, overall body composition and metabolism, alongside other risk factors, such as social and environmental determinants.[26] In the context of Aotearoa New Zealand, Māori and Pacific peoples often have higher lean body mass and lower fat mass than non-Māori, non-Pacific individuals at the same value of BMI.[27] The relatively few cases of long COVID among Māori means that stratified analyses are underpowered to detect real effects between Māori and non-Māori. Although there were high levels of missing BMI data in the study, the observation that both scenarios of our sensitivity analysis found significant associations between high BMI and long COVID strengthens our confidence in the accuracy of our interpretation.

The findings of this study are consistent with previous evidence. A cross-sectional study of 1,056 general practices in Germany reported a linear association between BMI and the risk of developing long COVID.[11] That study also reported an association between BMIs of >25kg/m^2 and long COVID, with this association present for females and no clear evidence of an association for males.[11] A matched cohort study of non-hospitalised adults reported that a BMI between 25 and 30kg/m^2 was associated with a slight risk increase in developing long COVID compared with a BMI between 18 and 25kg/m^2 (adjusted hazard ratio 1.07; 95% CI 1.04–1.10).[13] More recently, a systematic review and meta-analysis of 16 studies investigating obesity found that a BMI of ≥30kg/m^2 was statistically associated with long COVID.[14] At the time of this study, the population in Aotearoa New Zealand had high vaccination rates compared with the rest of the world, highlighting the importance of adding our findings to the international literature. However, the magnitude of the associations between BMI and long COVID that we report are higher than the estimates in previous literature. The reasons for this remain unclear; we emphasise that our results should be interpreted with caution.

In Aotearoa New Zealand, over two-thirds of individuals were classified as overweight or obese, with a BMI of >25kg/m^2, in 2023–2024.[23] There are significant differences in the prevalence of obesity among ethnic groups, with Māori and Pacific peoples having disproportionately higher rates compared with individuals of European/Other and Asian ethnicities.[23] These inequities begin early in life and are intergenerational. It

has been suggested that prolonged exposure to obesity throughout one's life could be associated with a higher risk of developing long COVID.[28] These observations suggest a mechanism through which the ongoing impacts of COVID-19 could exacerbate existing health inequities.

Although causal inferences cannot be made from observational data such as our investigation, this study provides epidemiological evidence to support our understanding of long COVID as an immuno-metabolic disease. Associations between nutritional status and immune dysfunction have been widely documented.[29] Obesity is associated with chronic, low-grade systemic inflammation, which hinders an individual's effective and timely immune response to an infectious disease like COVID-19,[28,29] and, as suggested in our study and others, a higher likelihood of developing long COVID.[14] These findings encourage further investigation exploring the link between immune dysfunction, obesity and long COVID and open possibilities of avenues for therapeutic advances.

Conclusion

In summary, we found that high BMI, but not other metabolic illnesses or risk factors, was associated with a higher risk of long COVID. This highlights the need for long COVID to be considered as an immune-metabolic disease and opens possibilities of avenues for therapeutic advances. Future research should explore the associations between immune function, obesity and long COVID to expand on our findings.

COMPETING INTERESTS
Mona Jeffreys is a Board Member of ME Support.

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Appendix

Appendix Table 1: The risk of long COVID in participants with missing compared with non-missing BMI.

Variable	Category	LC (%) n=217	No LC (%) n=188	OR (95% CI)	p-value
Missingness of BMI	Missing variables	85 (39.2)	52 (27.7)	1.68 (1.12–2.56)	0.015
	Not missing	132 (60.8)	136 (72.3)	Reference	

LC = long COVID; OR = odds ratio; 95% CI = 95% confidence interval; BMI = body mass index.

Appendix Table 2: Determinants of missingness in BMI.

Variable	Category	OR (95% CI)	p-value
Age	15–24	1.27 (0.64–2.15)	0.50
	25–44	Reference	
	45–64	0.75 (0.47–1.20)	0.23
	65+	0.49 (0.22–1.10)	0.09
Gender	Male	Reference	
	Female	1.05 (0.67–1.65)	0.83
Ethnicity	Māori	1.38 (0.78–2.44)	0.26
	Pacific peoples	1.07 (0.39–2.93)	0.90
	Asian	1.78 (0.74–4.26)	0.20
	Other	Reference	
Education	School	0.99 (0.59–1.67)	0.98
	Post-school	0.64 (0.37–1.11)	0.11
	University	Reference	
Income struggle	Strongly agree/agree	2.41 (0.98–5.94)	0.06
	Neither agree nor disagree	0.80 (0.22–2.92)	0.74
	Disagree/strongly disagree	Reference	
	Missing	87.20 (40.50–187.81)	<0.001
Household crowding	Overcrowding	1.68 (0.71–3.99)	0.24
	None	Reference	
Pre-COVID disability	Pre-COVID disabled	1.45 (0.72–2.90)	0.30
	Non-disabled	Reference	

BMI = body mass index; OR = odds ratio; 95% CI = 95% confidence interval.

"Call-it-in": addressing older informal caregivers' support needs during the COVID-19 pandemic in Aotearoa New Zealand

Tyrone Barnard, Shinya Uekusa, Christine Stephens, Fiona Alpass

ABSTRACT

AIMS: This study aims to address the knowledge gap in older informal caregivers' support needs by exploring their caregiving experiences during the COVID-19 pandemic in Aotearoa New Zealand. It proposes a conceptual solution termed "call-it-in".

METHODS: An interpretative qualitative approach was employed, and we conducted in-depth interviews with 81 informal caregivers. Data were analysed using thematic analysis to identify key themes related to participants' support needs.

RESULTS: The study found that many participants were disconnected from formal health systems, peer support groups and community organisations, leading to uncertainty about available support systems. Despite the availability of resources and information online, many participants preferred more traditional forms of communication such as phone helplines due to greater familiarity and the emotional support provided by speaking with someone directly.

CONCLUSIONS: The findings highlight the critical need for a centralised support system, which we term "call-it-in", to provide a single point of contact for accessing information and connecting users to various forms of existing/emergent support. This system aims to enhance informal caregivers' social capital during crises like the pandemic. Implementing "call-it-in" could improve the accessibility and visibility of support services, meeting the actual needs of older informal caregivers and reducing their reliance on personal networks and resources.

Informal caregivers—family, whānau and aiga carers/caregivers providing unpaid care for friends, family/whānau members, partners and others requiring support due to ageing, health declines, injuries, cognitive declines or disability[1,2]—form the backbone of the health system. Their contributions were critical, particularly during the COVID-19 pandemic, which placed unprecedented strains on health and social care systems.[3] During the pandemic, informal caregivers, typically untrained older adults, were recognised as a vulnerable population.[3,4] The pandemic's lockdown measures and the shortages in health and human services led to increased stress and other mental health issues, increased workloads and heightened social isolation for these informal caregivers.[3–5] Despite their critical role, the literature on informal caregivers remains sparse, particularly in the context of the pandemic in Aotearoa New Zealand (hereafter New Zealand).[6] While the pandemic-exacerbated challenges they faced are generally highlighted in the literature, both international and New Zealand studies indicate that caregiving experiences during the pandemic were not uniformly negative.[5,7,8] These experiences were dynamic and complex, shaped by the interplay of individual, environmental and social circumstances including ethnicity/race, socio-economic status, gender, social capital, past experiences and health conditions of their own and care recipients, among others.[7–9] These complexities highlight the need for more nuanced research to understand the experiences of caregiving and the specific support systems needed to meet informal caregivers' needs effectively.

A critical aspect of studying informal caregivers is that they are often overlooked and often disconnected from the formal health systems and service providers.[3,5–7] Particularly, during the pandemic, this disconnection left informal caregivers navigating uncertainty about available resources, how to access them and where to find reliable information.[3–5] Limited exploration of barriers to accessing support services and resources during the pandemic in New Zealand[6] has resulted in a lack of practical recommendations. Addressing this gap is critical to ensuring that

informal caregivers, especially those from under-represented groups such as older, Māori, migrant and rural informal caregivers, are supported in their roles.[5,8,9] As part of our larger research project that explored the lived experiences of older informal caregivers in New Zealand during the pandemic, this paper focusses on a specific question: what specific support needs and resources did participants require during the pandemic? This short paper focusses on overarching findings, since detailed analyses for Māori and rural participants are beyond its scope and are currently under review and already published.[9] Using an inductive, interpretative qualitative approach, we aim to illuminate the key areas where support for older informal caregivers can be enhanced during future pandemics, disasters and national emergencies.

Methods

We employed an exploratory, interpretative qualitative approach with purposive sampling to conduct in-depth interviews with 81 informal caregivers, including Māori and rural informal caregivers who are under-represented in existing research (see Table 1 for participant demographics). A multidisciplinary team of Pākehā (NZ European), tauiwi (non-Māori) and Māori social scientists conducted the interviews between

Table 1: Study participant demographic information (n=81).

		Count	Percentage
Average age*	66.16		
Gender	Male	34	42.0%
	Female	47	58.0%
Māori/non-Māori	Māori	35	43.2%
	Non-Māori	46	56.8%
Race/ethnicity**	Pākehā/NZ European	49	
	Māori	34	
	Irish	2	
	Dutch	1	
	Fijian	1	
	Japanese	1	
	Samoan	1	
	Zimbabwean	1	
Rural	Rural	39	48.1%
	Urban	42	51.9%
Economic Living Standard Index Short Form (ELSI-SF)	Good	57	70.4%
	Comfortable	14	17.3%
	Hardships	10	12.3%
Location	Northland	4	4.9%
	Auckland	13	16.9%
	Waikato	13	16.9%

Table 1 (continued): Study participant demographic information (n=81).

Location (continued)	Bay of Plenty	11	14.3%
	Taranaki	3	3.9%
	Manawatū	6	7.8%
	Gisborne	2	2.6%
	Hawke's Bay	5	6.5%
	Wellington	7	9.1%
	Marlborough	2	2.6%
	Tasman	4	5.2%
	West Coast	1	1.3%
	Canterbury	8	10.4%
	Otago	1	1.3%
	Southland	1	1.3%
Care recipient***	Parent	20	
	Spouse/partner	29	
	Sibling	2	
	Child	7	
	Grandchild	3	
	Extended family	5	
	Friend	6	
	Neighbour	4	
	Flatmate	1	
	Other	10	

*Oldest: 88, youngest 57.
**Some participants selected more than one race/ethnicity.
***Some participants cared for more than one recipient.

May 2023 and February 2024, ensuring diverse regional representation and amplifying informal caregivers' voices across New Zealand. Interviews were primarily conducted face-to-face (with four via Zoom and three by phone) and focussed on caregiving experiences during the pandemic in New Zealand (between March 2020 and December 2022). Participants were recruited from the longitudinal New Zealand Heath, Work and Retirement (HWR) database—a study of individuals aged 55+ living in New Zealand—administered by Massey University's Health and Ageing Research Team (HART). Participants who self-identified as informal caregivers and indicated caregiving during the pandemic (n=407) were initially contacted by mail. Potential participants, who responded to the invitation letter by phone or email and expressed interest in a 1-hour in-depth interview, were invited to participate.

Audio-recorded interviews were transcribed,

Table 2: Health conditions of care recipients.

Care recipient main illness and health conditions	Count
Dementia/Alzheimer's disease	11
Cancer	7
Physical/intellectual disability	4
Autism	4
Primary progressive multiple sclerosis	1
Physical decline (semi-independent)	15
Heart problems	6
Stroke	5
Mastectomy	1
Depression	1
Drug and alcohol addiction	1
Mobility issues (e.g., fall risk)	9
Incontinence	1
Unspecified	16
Total	81

and thematic analysis[10] was used to identify key themes related to informal caregivers' experiences and support needs (see Table 2 for health conditions of care recipients). Ethics approval was obtained from the Health and Disability Ethics Committee (2022 EXP 13416), with all procedures classified as low risk. Pseudonyms are used to protect participants' privacy and identifying details have been removed.

Results

Our qualitative study explored participants' lived experiences and identified several key areas where support for informal caregivers can be improved during future pandemics and disasters: 1) awareness of existing support systems, 2) clear and reliable information, 3) accessible and user-friendly support channels, 4) improved communication from formal support systems, 5) emotional and psychological support, 6) dedicated support for informal caregivers, 7) streamlined access to support by reducing bureaucratic hurdles, and 8) context-specific strategies.

Awareness of existing support systems

While organisations such as Health New Zealand – Te Whatu Ora, Whaikaha – Ministry of Disabled People, Needs Assessment Services Co-ordination (NASCA), Stroke Clubs, Cancer Society NZ and Carers NZ, among many other service providers, offer valuable assistance, many participants were simply unaware of these services. For example, Isabella described her uncertainty during the lockdown: "*The 24/7-ness of lockdown was quite intense. Somehow, you've got to be aware that someone needs help.* [I knew] *there was help out there, but you just got to know who to call, and I didn't know who to call*" (female, 60s, caring for her husband). Similarly, Mary emphasised:

> *"What would have probably been a good thing would be even just a phone contact* [during the pandemic], *because I did not know there was any. There*

may have been helplines, but I did not know, I am still not aware of where to find support." (Female, 60s, caring for her ex-husband and mother)

Participants frequently expressed a lack of awareness about available support systems. As elaborated later, this left many balancing the time, risks and benefits of seeking external help—often resulting in reliance on personal and familial resources. This aligns with international research highlighting that informal caregivers tend to lack awareness of available services or choose not to engage with them, even when informed.[2,11]

Clear and reliable information

Building on the awareness gap, participants emphasised the critical need for clear, reliable information about available support and resources. A recurring challenge was the lack of visibility and awareness of where to seek help, particularly in navigating unfamiliar and often fragmented systems, as discussed above. For many, the absence of accessible guidance intensified feeling of frustration and isolation. Hannah shared her struggle in trying to provide adequate care without external advice and assistance. She described the emotional toll of relying on personal resources:

"I didn't know how to support [my flatmate] *at all. I was getting frustrated because I thought, 'Get a grip!' But it was a real thing, and there was no one to talk to except my 32-year-old daughter … It would have been nice to have had someone to call for advice, to say, 'Look, how can I help her? What can I do to reassure her?' All I did was get angry, and then I felt bad for getting angry at her." (Female, 60s, caring for her flatmate)*

Hannah's experience was echoed by other participants, who also expressed the pressing need for a clear contact point or a supportive person to consult. For many participants, simply knowing who to call and, as elaborated later, having someone to talk to during difficult moments would have alleviated caregiving-related challenges.

Accessible and user-friendly support channels

Participants also emphasised the importance of support channels that are more easily accessible, straightforward and user-friendly, especially for those without internet access. Mary reflected this sentiment, stating: "*If there had been a helpline, a call line, it would have been so helpful if someone has a need for something or has a query, because not everyone had internet*." Although many participants, including Mary, demonstrated digital competence during the recruitment and interviews, they expressed a clear preference for traditional methods of communication, such as telephone helplines. These channels were perceived as more familiar, reliable and emotionally supportive compared to digital platforms. This preference underscored the need to prioritise accessible and inclusive support options that would cater to the diverse technological capabilities and comfort levels of participants.

Improved communication from formal support systems

Effective communication from formal support systems, including healthcare providers and government agencies, was identified as crucial. Informal caregivers in our study needed timely and accurate updates, as well as proactive "checking-in" on them. For some participants, it was highly appreciated when general practitioners (GPs) and clinics proactively checked in on them and provided some support during the pandemic. Fred explained: "*The clinic did ring us a couple of times* [during the lockdowns]. *The clinic did ring us to see what was going on and if we were OK. That was great. They offered help if we needed*" (male, 70s, caring for his wife). Even if Fred and his wife did not need any support at that time, they appreciated someone reaching out and checking in on them, especially when, for many, social engagements were reduced. This reinforces the need for emotional support and reassurance, as highlighted in the following section.

Emotional and psychological support

The emotional toll of informal caregiving is significant due to the nature of caregiving and informal caregivers' personal circumstances,[12] which were exacerbated during the pandemic.[13] For participants and care recipients alike, "*calling*" and "*talking on the phone*" were emotionally and practically significant, despite the availability of information and resources available online. Indeed, the importance of having someone to talk to for emotional support was a general finding in our study. Emma reflected this need: "*I think just making it known who people can talk to is*

important. Probably we need someone who they can call to just talk and call for help for whatever" (female, 60s, caring for her husband and mother-in-law). Hannah expands on this and specifies the need for emotional support related to caregiving challenges:

> "[My daughter] *still rings me up and says, 'Hey mum, now what's the matter?' It'll be simple, like she dropped the milk and* [it] *went everywhere. But for her, I need to talk to someone about it. You know, I'm strong. I'm strong. My daughter says I'm a bitch, but I'm strong. But there comes a point where I'm just, that pandemic kind of brought that out. And that's what made me, it makes you feel like a bad person. And we're not; we just need someone to just talk to." (Female, 60s, caring for her flatmate)*

As Emma, Hannah and other participants noted, providing opportunities for emotional and psychological support, such as counselling services, peer support groups or even just "*having someone to talk to on the phone*" would help disconnected informal caregivers better manage stress and prevent burnout.

Dedicated support for informal caregivers

Despite the abundance of online information (see, e.g., Ministry of Social Development[14] for available information and resources for informal caregivers), participants consistently highlighted the need for dedicated, tailored support for informal caregivers. Mary expressed frustration at the lack of clear guidance and personalised assistance. Lily also recounted her challenges with the existing Healthline service:

> *"It would have been good to have had a health helpline exclusively for* [informal] *caregivers, because often when I rang up on the Healthline, they would ask to speak to the patient. And he was not exactly aware of what was going on. And then they'd have to hand it back to me or the phone back to me to talk about it and discuss." (Female, 70s, caring for her daughter and husband)*

While some support services were technically available during the pandemic, they were often underutilised due to, as discussed, barriers such as inaccessibility, lack of visibility and misalignment with caregivers' actual needs. The pandemic also altered the way support was delivered, with phone consultations and telehealth becoming more prevalent.[15] Chloe, who had prior positive experiences with Healthline, appreciated its potential in a caregiving context: "*I had used the Healthline once for me [*in relation to a different health issue]. *Yes, something like that might have been useful too* [during the pandemic], *particularly with the distance that we've got* [here in a rural area]" (female, 70s, caring for her mother and close friend). However, such services typically focus on medical concerns but cannot assist with other caregiving-related or everyday challenges such as grocery shopping, bills and housework—issues that became even more pressing for participants during lockdowns.

Streamlined access to support by reducing bureaucratic hurdles

Participants faced bureaucratic challenges when trying to access fragmented supports, such as lengthy processes and strict eligibility criteria. Simplifying these procedures and reducing "red tape" would have made it easier for participants to obtain necessary assistance. Aki described these issues:

> *"I need to find a care support person myself when I need care support for a day. I had to search and call them myself to schedule the best visit time. In the midst of all this caregiving busyness, I had to manage everything on my own. It would be helpful if someone could take charge of these arrangements, like organising support from a specific time to another time, especially for tasks like changing diapers. If there were a structured way for someone else to handle this, it would be a huge help. Just organising that would be a great relief. I often have to make multiple calls if there isn't a bed available, and sometimes I'm told there might be one later, even if there isn't one right now. There's a lot of paperwork to do before a visit, so I can't just leave the house. While managing all this, I also have to co-ordinate with the care support person who might only be available for one hour. It's quite overwhelming." (Female, 50s, caring for her husband)*

Aki underscores the need for a more streamlined and supportive system that can alleviate the administrative burdens on informal caregivers, allowing them to focus more on the actual care and less on logistics. The time and effort required to organise care support often exceed the benefits, as, for Aki, it took several hours to arrange a care person for just 1 hour of assistance (e.g., respite). Harper also described how navigating the system to access necessary support for her mother was fraught with administrative hurdles:

> *"Were we well supported? No, we just kind of* [managed on our own]*, I think it was less stressful to just do it* [ourselves]*. Do what you need to do yourself. Like I said, there's a lot of red tape for some of the things I've been trying to get for Mum." (Female, 60s, caring for her mother)*

The inefficiency may lead many informal caregivers, like Aki and Harper, to make the rational choice to forego available services and manage on their own despite the overwhelming nature of their responsibilities. This insight reveals why informal caregivers often prefer relying on personal networks over formal support systems, highlighting the need for a more streamlined and supportive system that can alleviate the administrative burdens on informal caregivers, allowing them to focus more on actual care and less on logistics.

Context-specific strategies

Participants' experiences also highlight that support needs to be more adaptable to specific contexts, particularly during crises. As discussed above, some participants expressed that, during the pandemic, they needed practical support such as help with grocery shopping or someone to talk to rather than financial aid and additional information. Furthermore, several participants highlighted barriers to use available support, often rooted in concerns about health and safety. Many pre-existing supports required physical contact, which participants reduced or stopped using to protect care recipients and themselves. Brad spoke about reduced access to in-home care:

> *"*[My mother] *was worried, and we didn't want her to get COVID-19. Even though she had all her vaccinations, I mean, she was as protected as anyone, but it could hit her badly. So, we stopped anyone* [including home helpers, cleaners, gardeners, etc.] *from seeing her. She was starting to get quite reclusive, which was probably not a good thing." (Male, 70s, caring for his mother)*

Like Brad, other participants mentioned the limitation of social engagements due to social distancing and other COVID-19 measures, which extended to professional in-home care support such as care agencies, respite services and home helpers—resources that were vital to their caregiving responsibilities. Despite their reliance on these services, no alternative strategies were introduced to address their absence during the pandemic, or simply these participants were unaware of alternatives.

Access difficulties were further exacerbated by systemic issues such as staff shortages caused by the COVID-19 measures.[16] Ted noted this increased challenge: "*The caregiving was harder* [during the pandemic]*, because pretty much all the help we had before stopped*" (male, 70s, caring for his wife). While study participants understood the pandemic situations, they preferred pre-existing traditional support that they were familiar with, highlighting the trust and reliability these services provided. The lack of alternative, or awareness of it, during the pandemic intensified the burden of caregiving, leaving participants to navigate their responsibilities with little/no external assistance.

Discussion: "call-it-in" as a conceptual solution

The findings reveal critical gaps in the support available to informal caregivers, particularly in navigating fragmented systems and accessing context-specific, emotional and logistical assistance. These challenges align with existing literature on the challenges of caregiving during crises and highlight opportunities for improvement in preparedness for future pandemics and disasters.[3,5,6,11,17,18] The interplay of health management, altered social support and emotional impacts has been well documented, emphasising the pressing need for robust, tailored, culturally responsive support systems for informal caregivers.[17,19,20] While some participants acknowledged the value of services such as Carer Support and Home and Community Support Services (HCSS),[21] many were unaware of these service providers and resources or found them impractical due to the need to independently seek and organise support within

their allocated funding. Although this approach allows for individualisation, it often places an extra burden on caregivers who may be already overwhelmed. While our intention is not to over-generalise, these findings were consistent across all participant groups, including Māori, non-Māori, rural and urban participants, highlighting the widespread nature of these challenges.

Existing frameworks like the New Zealand *Carers' Strategy Action Plan 2019–2023*[17] emphasise recognition and financial support for informal caregivers. However, our study highlights the need for a centralised, user-friendly system to streamline access and provide tailored assistance. Participants like Isabella and Mary expressed the importance of having a central point of contact—such as a dedicated helpline or call centre—that is designed to act as a bridge between informal caregivers and existing/emergent services. The "call-it-in" concept addresses these gaps by offering a single point of contact that integrates culturally and linguistically appropriate services, emotional support and practical assistance. This approach could be developed as an independent initiative or integrated into existing frameworks, with the primary goal of facilitating connections and addressing unmet needs. Drawing inspiration from screening tools designed for at-risk populations,[22] the "call-it-in" approach would serve as a central hub, connecting informal caregivers to both formal and informal resources. Our findings contribute to the field, highlighting an approach that, while requiring funding and training, addresses gaps in both accessibility and emotional support, particularly in the context of the pandemic.[23–25]

Key features of "call-it-in" include: 1) a national call centre, 2) the integration of existing and emergent support mechanisms, and 3) a triage system for prioritisation. Firstly, a dedicated national call centre would provide informal caregivers with timely assistance, reduce misinformation and streamline access to services. By offering a familiar and easily accessible platform, it would address informal caregivers' immediate needs while reducing the stress associated with navigating fragmented systems and information available online. Secondly, to enhance the effectiveness of "call-it-in", it should connect users to pre-existing and emergent community resources. For example, volunteer groups like the Student Volunteer Army (SVA), which played a critical role during the Canterbury earthquakes and the COVID-19 pandemic,[26,27] could provide practical support and resources such as grocery shopping or transportation services.[28] Lastly, implementing a triage system would ensure that support for informal caregivers is tailored to their unique circumstances and needs. By screening informal caregivers' backgrounds, needs, stress levels and specific challenges, resources such as SVA's grocery shopping services, local Māori health providers and peer support community groups can be allocated more effectively, ensuring timely and targeted assistance for those in (urgent) need.

While a "one-size-fits-all" approach is often not recommended, this centralised approach is particularly crucial, given that many informal caregivers in our study were simply disconnected from formal health systems and community groups and were unsure of available information and support options. Beyond practical support, "call-it-in" has the potential to enhance informal caregivers' social capital. By facilitating connection between informal caregivers and formal as well as informal resources, it extends beyond bonding social capital (connections within close-knit groups like family or neighbours) to include bridging and linking social capital.[29] These broader networks provide mutual support and connect informal caregivers to essential services. Importantly, this approach acknowledges the challenges of pre-planning social capital, as disaster studies have shown that its mobilisation is *ad hoc*, driven by immediate circumstances rather than planned efforts.[30] Emergent groups like SVA exemplify how support networks may form organically during crises to fill the gaps in social and human services as needed. The "call-it-in" system could complement these efforts by providing a structured yet flexible platform for mobilising resources during crises, disasters and national emergencies.

Conclusion

Further exploration is essential to provide effective and practical support to informal caregivers based on their unique needs and individual circumstances. By addressing gaps in awareness, accessibility and emotional support, "call-it-in" can empower informal caregivers to navigate crises more effectively. Although we acknowledge this study lacks broad generalisability and poses a clear limitation due to the under-representation of Asian, Pacific peoples and other social groups, this approach may not only mitigate the potential

negative impacts of informal caregiving during pandemics but also build informal caregivers' resilience and wellbeing for future adversities. Proactively and spontaneously fostering connections between informal caregivers and support systems affirms the importance of dedicated, culturally and linguistically responsive and innovative solutions for this vital yet often overlooked population. The proposed conceptual solution as an example offers a centralised point of contact and support, which should be further developed, implemented and properly evaluated, particularly in ways that reflect the cultural diversity and diverse realities of informal caregivers.

COMPETING INTERESTS
Nil.
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Appendix

Appendix 1: Support needs–related interview questions.

What support did you have available to you to deal with the challenges during the lockdowns and the pandemic you have just described?
What did/do you know about support available to caregivers before and during lockdown?
Do you think you were well supported by others as a caregiver in a lockdown situation (and during the pandemic in general)? (either family, government, community organisations).
What do you think could be done to better support caregivers in a pandemic?

Common mental disorders and psychological distress among Pacific adults living in Aotearoa New Zealand

Joanna Ataera-Minster, Susanna Every-Palmer, Ruth Cunningham, Jesse Kokaua

ABSTRACT

AIM: To examine common mental disorders and psychological distress in Pacific adults and between Pacific ethnic groups.

METHODS: Data were pooled from multiple New Zealand Health Survey waves from 2014/2015 to 2018/2019. Estimated period prevalence of common mental disorders (depression and/or anxiety) and psychological distress were calculated for Pacific adults aged 15 years and over, analysed by socio-demographic factors (age, sex and socio-economic deprivation), specific Pacific ethnic groups (Samoan, Tongan, Cook Islands and Other Pacific) and Realm country status. Log-binomial regression methods were used to calculate unadjusted and adjusted risk ratios (ARRs) for comparative analyses.

RESULTS: Doctor-diagnosed common mental disorders were more prevalent in Pacific women, adults aged 24–64 years and those living in the least deprived areas (compared with Pacific men, adults aged 15–24 years and those in the most deprived areas respectively). Psychological distress was more prevalent in Pacific females and Cook Islands Māori. Some within-Pacific mental health differences were evident, with higher rates of diagnosed common mental disorders in adults affiliated with Pacific Realm countries (Cook Islands Māori and Niueans) compared with those affiliated with non-Realm countries.

CONCLUSIONS: Higher rates of doctor-diagnosed common mental disorders in Pacific adults from the least deprived areas suggest either higher needs and/or better care access in these groups. Mental health varies among Pacific peoples, with Cook Islands Māori in particular experiencing poorer outcomes. Further research and interventions targeting specific Pacific subpopulations are warranted.

"Pacific peoples" is a heterogeneous population in Aotearoa New Zealand, composed of individuals whose homelands and ancestral connections stem from various Pacific nations, predominantly from Melanesia and Polynesia.[1] Pacific peoples make up almost 9% of the total New Zealand population and are predicted to increase to 11% of the total New Zealand population by 2043.[2] The Pacific population has a young age structure (median age: 23.4 years) and is highly concentrated in urban regions, with three-quarters of Pacific living in the Auckland and Wellington Regions in 2018.[3] Pacific peoples is an ethnically and culturally diverse population, which includes at least 17 different Pacific ethnic groups. The six largest groups are Samoan (48%), Tongan (22%), Cook Islands Māori (21%), Niuean (8%), Fijian (5%) and Tokelauan (2%).[3,4] The Cook Islands, Niue and Tokelau fall within the "Realm of New Zealand", as these Pacific nations were annexed by the British monarch in the early 1900s and colonised under the New Zealand flag. Individuals affiliated with these "Pacific Realm countries" are New Zealand citizens by birth,[5] and these Pacific subpopulations have greater proportions of people living in New Zealand than in their Pacific homelands, with over 90% of the Cook Islands and Niue populations currently living in New Zealand.[4] Importantly, the Pacific Realm countries share similar histories of colonisation and migration to New Zealand, both of which contribute to the shaping of mental health inequities in cultural populations through the intergenerational disruption of health-protective social structures and cultural resources.[6]

Pacific peoples experience high rates of mental health conditions and disproportionately low rates of mental health service use. *Te Rau Hinengaro* (The New Zealand Mental Health Survey) is the only population mental health survey to date that measured mental health with structured clinical interviews, based on assessment criteria for mental disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). This survey, conducted in 2003–2004, showed that Pacific adults aged 16 years and over had a higher prevalence of mental disorders than Others (non-Māori, non-Pacific) and low rates of mental health service use.[7] While elevated crude rates of mental

disorders were explained by the young population structure and higher levels of socio-economic deprivation,[7] Pacific peoples' underutilisation of mental health care was only partly explained by these, suggesting other factors, such as known care access barriers, underpin Pacific peoples' mental health service use.[8,9]

An analysis of *New Zealand Health Survey* (NZHS) data from 2014 to 2019 demonstrated that Pacific adults have lower rates of doctor-diagnosed mood and/or anxiety disorders despite higher rates of psychological distress than non-Māori, non-Pacific adults.[10] This same pattern was also demonstrated in the *New Zealand Attitudes and Values Study* (NZAVS),[11] although these surveys are limited by their use of self-report measures of diagnosed disorders, which contrasts with the systematic measurement of diagnosable mental disorders in *Te Rau Hinengaro*.

The predominant focus of national research and policy involves examining and addressing mental health inequities in the total Pacific population. This pan-Pacific approach is understandable given the way Pacific peoples formed collectively in New Zealand as a result of shared cultural customs, values and beliefs and similar migration histories within the country. However, there is growing evidence that mental health varies *within* Pacific peoples, particularly in various subpopulations defined by socio-demographic factors, such as Pacific born in New Zealand ("NZ-born"), or those who affiliate with more than one ethnic group ("multi-ethnic"). Nationally representative cross-sectional surveys,[10] including *Te Rau Hinengaro*,[12] have consistently demonstrated that mental disorders are more prevalent in Pacific peoples born in New Zealand ("NZ-born") compared with those born overseas ("Overseas-born"). Similarly, evidence from nationally representative cross-sectional surveys show that multi-ethnic Pacific adults have poorer mental health than sole-Pacific adults, with lower self-esteem[13] and higher rates of personal experiences with mental illness[14] and diagnosed mood and/or anxiety disorders.[10]

Examining mental health within Pacific peoples is challenging due to the relatively small size of the population and even smaller sizes of Pacific subpopulations of interest. Specialised statistical techniques, such as Bayesian modelling, are one way of examining subpopulation outcomes, and these methods have previously been used to compare both health and mental health outcomes between Pacific ethnic groups.[15,16] Pooling data across multiple waves of a routine national survey is another method for looking more closely at mental health in Pacific subpopulations. This paper aims to use pooled NZHS data to examine mental health within Pacific adults, by socio-demographic factors and the specific Pacific ethnic groups. Such within-Pacific analyses are useful for identifying subpopulations with higher needs and informing the development of targeted mental health promotion policy and initiatives.

Methods

Study design

This study used data from the NZHS—an annual cross-sectional survey administered by the Ministry of Health – Manatū Hauora. Anonymised NZHS data in the form of confidentialised unit record files (CURFs) were sourced from Stats NZ by standard microdata access application processes. Data were supplied in January 2023.

Participants

The NZHS uses a complex multi-stage sampling approach with a stratified probability-proportional-to-size design to obtain a nationally representative sample. Participants are selected, first by area-based selection of primary sampling units (PSUs) from Stats NZ's household survey frame, then by selecting households within the PSUs and randomly selecting eligible participants from those households. All survey waves in this study used PSUs for first-stage selection, except for the 2014/2015 wave, which used meshblocks—smaller areas (around 90 people), one or more of which fit into a single PSU. Eligible participants include adults aged 15 years and over, and children aged 0–14 years. Targeted sampling of PSUs where more Pacific people live is undertaken to increase Pacific sample sizes.[17] Only NZHS adult participants were included in this study (N=68,317; mean age=45.31 years, SD=19.03). Overall response rates ranged from 79–80% across survey waves.

Procedures

The NZHS uses trained interviewers to conduct face-to-face computer-assisted interviews from 1 July to 30 June of the following calendar year. Information is collected across various health domains, including long-term conditions, health status, health behaviours and risk factors and health service utilisation and barriers.[16]

Measures

Mental health outcomes

Two mental health outcome measures were included in this study: doctor-diagnosed common mental disorders and psychological distress. "Common mental disorders" is a term referring to depressive disorders and anxiety disorders, which are highly prevalent and considered "common" in global populations.[18] Doctor-diagnosed common mental disorders were measured with a composite binary (yes/no) variable, derived from two questions: "Have you ever been told by a doctor that you have depression?" and "Have you ever been told by a doctor that you have an anxiety disorder? This includes panic attacks, phobia, post-traumatic stress disorder and obsessive-compulsive disorder."

Psychological distress was measured with the Kessler Psychological Distress Scale (K10).[19] The K10 is an internationally validated 10-item scale that measures how often respondents experienced symptoms of psychological distress (e.g., hopelessness, nervousness, restlessness, depression or worthlessness) over the past 4 weeks. Responses are recorded on 5-point scales ("none of the time", "a little of the time", "some of the time", "most of the time" and "all of the time") and scored 0–4 for each item. Total scores range from 0 to 40, with higher total scores indicating higher psychological distress. In this study, "high psychological distress" was defined as a score total of 12 or more—the cut-off point used in NZHS reporting. K10 scores of 12 or more indicate a high probability of depression or anxiety disorder.[20] The K10 has good predictive validity and, for example, was shown to discriminate between cases and non-cases of anxiety and mood disorders in *Te Rau Hinengaro*.[21] However, the K10 is not a diagnostic tool, and is therefore not recommended as a measure of the prevalence of mental health conditions in New Zealand population groups.[22]

Ethnicity

The NZHS uses the standard ethnicity question from the New Zealand Census of Population and Dwellings. Respondents identify the ethnic group/s they belong to, with a number of options provided, including an "Other" category and accompanying free-text field for written responses. This study used total response ethnicity classification, meaning participants who identified with Pacific as any of their ethnic groups were included in the Pacific ethnic group for analysis.

Level 2 ethnicity data were supplied for the four largest Pacific ethnic groups (Samoan, Cook Islands Māori, Tongan and Niuean). These were used to group participants into four Pacific ethnic groups. Niuean data were not disaggregated because of small strata sizes within each mental health outcome (n=45, common mental disorders; n=36, psychological distress). A residual "Other Pacific" category included Niueans (n=381) and all remaining Pacific participants for whom Level 2 ethnicity data were not supplied (n=481). Participants who self-identified with two or more Pacific ethnic groups (n=225) were preferentially assigned to the smaller Pacific group, creating mutually exclusive Pacific ethnic groups.

Cook Islands Māori and Niueans were combined into a "Realm" Pacific group for analysis by Realm country status. Tokelauans are also part of the Realm of New Zealand, but were assigned to the "non-Realm" group because Tokelauan ethnicity data were not included in CURFs. "Tokelauan" is not a response option in the standard NZHS ethnicity question, and free-text responses associated with the "Other" ethnic group responses were not supplied upon request due to confidentiality concerns and limited NZHS team capacity to provide customised CURFs.

Other covariates

Other covariates entered into the models included age (15–24, 25–44, 45–64 and 65+ years) and sex (male/female). Socio-economic position (SEP) was measured with both an area-based measure (New Zealand Index of Deprivation 2013 [NZDep2013])[23] and an individual-level measure (highest educational qualification attained). NZDep2013 data were collapsed into three categories: low (NZDep deciles 1–3), medium (deciles 4–7) and high deprivation (deciles 8–10). Highest educational qualification data were used to create a binary variable (no secondary qualification vs secondary qualification or higher).

Analysis

Data from the 2014/2015 to 2018/2019 NZHS waves were pooled into a single dataset and analysed using Stata/SE version 18.0. The complex sampling method was accounted for by applying supplied calibrated survey weights, which adjust for inverse sampling weighting, stratification and clustering due to area-based sampling. Supplied jackknife replicate weights were used to estimate variance. All survey weights were adjusted by dividing by the total number of survey waves

included in the study. Unweighted frequencies and weighted prevalence estimates were calculated for mental health outcomes in Pacific adults, analysed by socio-demographic factors (age, sex, deprivation and education), Pacific ethnicity (Samoan, Tongan, Cook Islands Māori and Other Pacific) and Realm country status (Realm/non-Realm). Unadjusted and adjusted risk ratios for mental health outcomes and associated 95% confidence intervals (CIs) were calculated using generalised linear regression, specifying a binomial distribution with a log link. Outcomes and covariates were entered into models in a stepwise process: 1) unadjusted, 2) adjusted for age and sex, and 3) adjusted for SEP, entering NZDep and educational qualification sequentially.

Table 1: Demographic characteristics of the Pacific respondents, *New Zealand Health Survey*, 2014/2015–2018/2019.

Demographic characteristics		**n**[a]	**%**[b]
Pacific ethnicity	Samoan	1,843	43.7
	Cook Islands Māori	951	18.2
	Tongan	821	21.0
	Other Pacific	720	17.2
Realm country status[c]	Realm	1,246	25.3
	Non-Realm	3,089	74.7
Gender	Male	1,794	46.8
	Female	2,541	53.2
Age	15–24 years	973	29.4
	25–44 years	1,853	38.7
	45–64 years	1,098	24.7
	65+ years	411	7.2
Deprivation level (NZDep deciles)	Low (1–3)	270	8.9
	Medium (4–7)	947	25.7
	High (8–10)	3,112	65.4
	Missing	6	0.1
Education (highest qualification)	No secondary qualification	1,621	35.7
	Secondary qualification or higher	2,616	62.4
	Missing	98	1.9
Total Pacific		4,335	6.1

[a]Unweighted frequencies.
[b]Weighted percentages.
[c]The "Realm of New Zealand" is a convenient way of referring to the five countries or territories over which the Sovereign reigns in the right of New Zealand. The Realm of New Zealand includes New Zealand, the self-governing states of the Cook Islands and Niue, Tokelau and the Ross Dependency. Tokelauans were included in the non-Realm group because ethnicity data on Tokelauans were not supplied (see Methods section).
NZDep = New Zealand Index of Deprivation.

Table 2: Five-year weighted prevalence and adjusted risk ratios (ARRs) of common mental disorders and psychological distress in Pacific adults by demographic covariates, *New Zealand Health Survey*, 2014/2015–2018/2019.

Demographic covariates	Common mental disorder (diagnosed) n=497		Psychological distress (K10 ≥12) n=532	
	Prevalence % (95% CI)	ARR (95% CI)	Prevalence % (95% CI)	ARR (95% CI)
Gender				
Female	10.9 (9.4, 12.6)	1 (reference)	13.2 (11.5, 15.2)	1 (reference)
Male	6.2 (5.1, 7.5)	0.57* (0.46, 0.71)	9.3 (7.8, 10.9)	0.70* (0.57, 0.86)
Age				
15–24 years	5.5 (3.9, 7.7)	1 (reference)	10.8 (8.7, 13.4)	1 (reference)
25–44 years	10.3 (8.7, 12.2)	1.84* (1.26, 2.68)	11.1 (9.5, 13.0)	1.02 (0.78, 1.32)
45–64 years	10.4 (8.5, 12.7)	1.85* (1.25, 2.76)	12.6 (10.1, 15.8)	1.15 (0.85, 1.55)
65+ years	6.9 (4.5, 10.5)	1.18 (0.69, 2.04)	11.0 (7.9, 15.0)	0.98 (0.65, 1.48)
Deprivation level (NZDep deciles)				
Low (1–3)	12.3 (8.7, 17.1)	1.77* (1.19, 2.65)	7.9 (4.7, 12.9)	0.74 (0.44, 1.24)
Medium (4–7)	10.7 (8.5, 13.4)	1.43* (1.11, 1.83)	12.9 (10.3, 16.0)	1.14 (0.90, 1.45)
High (8–10)	7.4 (6.3, 8.7)	1 (reference)	11.2 (10.0, 12.7)	1 (reference)
Education				
No secondary qualification	7.2 (6.0, 8.7)	1 (reference)	12.1 (10.2, 14.2)	1 (reference)
Secondary qualification or higher	9.4 (8.1, 11.0)	1.21 (0.97, 1.50)	10.9 (9.5, 12.5)	0.91 (0.74, 1.13)

*p<.05.
CI = confidence interval; NZDep = New Zealand Index of Deprivation.

Post-estimation adjusted Wald tests were run to test for differences between the levels of socio-demographic factors and Pacific ethnic groups.

Results

There were 4,335 Pacific adults aged 15 years and over in the NZHS dataset, pooled from the 2014/2015 to 2018/2019 survey waves (mean age=31.17 years; SD=17.05). Table 1 summarises the socio-demographic characteristics of the participants.

Five-year period prevalence estimates of doctor-diagnosed common mental disorders and psychological distress during 2014–2019 are presented in Table 2. Results showed the prevalence of diagnosed common mental disorders was significantly lower in Pacific males (6.2%) than females (10.9%, ARR=0.57; 95% CI 0.46–0.71). The prevalence of diagnosed common mental disorders was significantly higher in Pacific adults from low- and medium-deprivation areas (ARR_{Low}=1.77; 95% CI 1.19–2.65; ARR_{Medium}=1.43; 95% CI 1.11–1.83) than in Pacific adults from high-deprivation areas and was significantly lower in Pacific youth aged 15–24 years (5.5%) than Pacific adults aged 25–64 years (25–44 years, p=0.002; 45–64 years, p=0.003). Table 2 also demonstrates the prevalence of psychological distress was lower in Pacific males (9.3%) than Pacific females (13.2%, ARR=0.70,

Table 3: Five-year weighted prevalence and adjusted risk ratios (ARRs) for diagnosed common mental disorders and psychological distress in Pacific adults aged 15 years by Pacific groups, New Zealand Health Survey, 2014/2015–2018/2019.

Pacific groups	Common mental disorder (diagnosed) n=497		Psychological distress (K10 ≥12) n=532	
	Prevalence % (95% CI)	ARR (95% CI)	Prevalence % (95% CI)	ARR (95% CI)
Pacific ethnicity				
Samoan n=1,843	7.2 (5.9, 8.7)	1 (reference)	10.4 (8.7, 12.2)	1 (reference)
Tongan n=821	6.5 (4.9, 8.7)	0.94 (0.68, 1.31)	11.8 (9.3, 14.9)	1.19 (0.87, 1.62)
Cook Islands Māori n=951	12.1 (9.8, 14.9)	1.67* (1.28, 2.17)	14.2 (11.2, 17.8)	1.41* (1.06, 1.89)
Other Pacific n=720	11.4 (9.0, 14.4)	1.43* (1.04, 1.98)	10.6 (8.1, 13.6)	1.03 (0.77, 1.39)
Total Pacific n=4,335	8.7 (7.6, 9.8)		11.4 (10.2, 12.7)	
Realm country status				
Realm n=1,246	12.2 (10.0, 14.7)	1.65* (1.30, 2.08)	12.5 (10.1, 15.4)	1.14 (0.89, 1.48)
Non-Realm n=3,089	7.5 (6.5, 8.7)	1 (reference)	11.0 (9.7, 12.6)	1 (reference)

*p<.05.
Adjusted for age, gender, New Zealand Index of Deprivation (NZDep) and education (Model 4, Appendix Table 2).
CI = confidence interval.

0.57–0.86). There were no other significant differences in psychological distress observed by sex, age, deprivation or education.

Table 3 shows that, among the Pacific ethnic groups, the prevalence of diagnosed common mental disorders was higher in Cook Islands Māori (12.1%) than both Samoans (7.2%, ARR=1.67; 95% CI 1.28–2.17) and Tongans (6.5%, p=0.0031; Appendix Table 1). Diagnosed common mental disorders were also significantly more common in Other Pacific (11.4%) than in Samoans (ARR=1.43; 95% CI 1.04–1.98) and Tongans (p=0.037; Appendix Table 1). When analysed by Realm country status, the risk of diagnosed common mental disorders was over 60% higher in Pacific adults affiliated with Realm countries compared with those affiliated with non-Realm countries (ARR=1.65, 95% CI 1.30–2.08). The prevalence of psychological distress was higher in Cook Islands Māori (14.2%) than Samoans (10.4%, ARR=1.41; 95% CI 1.06–1.89), but no other differences between Pacific ethnic groups reached statistical significance in post-tests (Appendix Table 1). Full regression model results are appended (Appendix Table 2).

Discussion

This study showed that mental health outcomes vary within Pacific peoples, with Cook Islands Māori adults reporting significantly higher rates of doctor-diagnosed common mental disorders

than Samoans and Tongans. This is consistent with results from *Te Rau Hinengaro*, which demonstrated a general patterning of a higher prevalence of mental disorders in Cook Islands Māori adults, although results did not reach statistical significance.[7] A related finding was that the risk of common mental disorders was collectively 65% higher in Pacific ethnic groups affiliated with nations that fall under the "Realm of New Zealand" (Cook Islands and Niue in this paper; see Methods section). To our knowledge, no other studies have demonstrated variability in diagnosed mental disorders by Realm country status, although one previous study of the NZHS demonstrated hazardous drinking was significantly higher in females from Pacific Realm countries,[24] and the cross-sectional national *Youth Insights Survey* showed past-month substance use was significantly higher in Pacific youth affiliated with the Realm countries.[25] More broadly, the *New Zealand Census Mortality Study* demonstrated that cardiovascular disease mortality rates were significantly higher in Cook Islands Māori and Niueans in 2001–2004.[15] This consistent patterning of poorer health and mental health outcomes in Cook Islands Māori and Niueans indicates an urgent need for research and policy interventions focussing on these Pacific subpopulations.

This study demonstrates the period prevalence of diagnosed common mental disorders was significantly higher in Pacific adults living in low- and medium-deprivation areas. This is consistent with national specialist mental health service use data that show anxiety and depression diagnoses decrease with deprivation in Pacific young people aged 10–24 years.[26] This patterning of Pacific mental health by deprivation that differs from what is seen in the total New Zealand population, whereby, for the latter, poorer outcomes are generally observed in more deprived areas.[27]

There are multiple possible interpretations for these within-Pacific differences in diagnosed mental disorders, and this highlights the limitations of self-report measures of diagnosed disorders. It is possible there are financial barriers for Pacific adults, preventing access to services in those with lower SEP; or, similarly, that the citizenship status of Realm-Pacific individuals affords them easier access to primary and specialist services. It is also possible that Realm Pacific adults or those from low deprivation areas have different concepts of mental health/illness and, therefore, have different help-seeking behaviours and care preferences. In short, the outcome measure conflates both diagnosable and diagnosed mental conditions and, therefore, is not as useful for directing policy as measures derived from structured clinical interviews (e.g., composite international diagnostic interview [CIDI] used in *Te Rau Hinengaro*), which are more indicative of need. This limitation underscores a need for more frequent routine monitoring of national mental health—with robust measures of diagnosable mental conditions—to enable policymakers to accurately differentiate between mental health needs and service access and track mental health outcomes over time.

We found the elevated risk of diagnosed common mental disorders in Realm Pacific ethnic groups was stable and did not change appreciably after adjusting for demographic or socio-economic factors (Appendix Table 2). This pattern was also demonstrated in an NZHS study on doctor-diagnosed mood and anxiety disorders among New Zealand-born and multi-ethnic Pacific adult subpopulations.[10] Together, these results suggest other causal factors unaccounted for in the models should be considered, highlighting a need to look more broadly at the structural causes of Pacific mental health. Structural causal factors are the underlying causes of mental health that occur in an individual's surrounding socio-cultural, geopolitical and physical environments.[28] These factors shape the circumstances we grow, work, live and age in—the "social determinants of mental health" (e.g., housing, education and employment/income)—which influence our mental health over the life course and across generations.[29] For global Indigenous populations, the legacy of colonisation is an important structural factor[28] because the impacts of dispossession of ancestral lands, institutional racism, forced assimilation and subjugation through legislative and social policies are intergenerational, and these continue to influence the multiple social determinants that contribute to present-day health inequities.[6] The higher rate of diagnosed common mental disorders in Realm Pacific observed in this paper, for example, could be explained by lower rates of cultural connectedness among Realm Pacific adults compared with non-Realm Pacific adults,[14] which itself is rooted in post-colonial social policies (e.g., English-only language schools) that led to the gradual decline of Indigenous Pacific health-protective cultural resources (e.g., Realm Pacific languages) over successive generations. While research is needed to understand how cultural connectedness is

associated with Pacific mental health, it is likely the within-Pacific mental health differences in this study are underpinned by these broader structural and social determinants of mental health. Indeed, a small body of national cross-sectional and longitudinal studies demonstrate that factors such as migration, acculturation and racism are all associated with Pacific mental health.[9] Similarly, nationally representative cross-sectional studies demonstrate that social determinants, such as ethnic identity[30] and Pacific language,[31] are associated with better mental health outcomes and, therefore, would be worth considering for clinical- and population-level intervention.

This study showed the prevalence of both psychological distress and doctor-diagnosed common mental disorders was higher in Pacific females than males. This latter result differs from *Te Rau Hinengaro*, which found no significant sex differences in Pacific adults who met diagnostic criteria for mental disorders or serious mental disorders experienced in the past 12 months.[7] Given *Te Rau Hinengaro* used a more comprehensive measure of mental disorders, our result could be reflecting a broad range of factors, such as unconscious gender biases, or male–female differences in self-reporting of mental disorders and/or presentation to services.

The results on psychological distress within Pacific adults are somewhat inconclusive. This is first because psychological distress did not vary significantly by age, deprivation or education, all of which are covariates with known associations with psychological distress.[27] Second, psychological distress did not differ by Realm country status. Third, the wide errors around the Pacific ethnic group estimates mean the finding of higher psychological distress in Cook Islands Māori than Samoans should be interpreted with caution, particularly since Cook Islands Māori did not differ from either Tongans or Other Pacific in post-testing (Appendix Table 1). Nevertheless, together these results raise two points about 1) the validity of the K10 and its suitability for measuring acute mental distress in Pacific peoples, and 2) the likelihood that Pacific mental health is driven largely by broader social determinants, which have a cumulative and pervasive impact on mental wellbeing, operating over years and generations, and, therefore, would show up more strongly in long-term measures than short-term measures of distress. These points are supported by a recent NZHS analysis,[10] which demonstrated psychological distress did not differ between New Zealand-born/Overseas-born and multi-ethnic/sole-Pacific subpopulations—results that are incongruous with cross-sectional national surveys that show New Zealand-born and multi-ethnic Pacific sub-groups have significantly higher rates of assessed mental disorders[7] and doctor-diagnosed mental health conditions.[14] Further research is needed to better understand within-Pacific mental health differences and to examine the validity of current measures used to monitor Pacific mental health.

Strengths and limitations

A key strength of this study is its acknowledgment that Pacific peoples is made up of several (sub) populations, each with different socio-cultural influences on mental health that contribute to diverse outcomes. The use of pooled data from multiple waves of a routine nationally representative survey enabled closer examination of the patterning of mental health in Pacific sub-groups with greater precision than is possible with single survey waves. To our knowledge, this is also one of the first studies to examine mental health outcomes by Pacific Realm country status—a Pacific subpopulation of emerging importance in Pacific health research.[14,24]

This study is limited first by the measures of mental health used in the NZHS, particularly self-reported doctor-diagnosed disorders, which may not reflect the true burden of Pacific mental health because it does not separate out mental health needs from access (i.e., it is difficult to ascertain whether apparent increased risk is due to increased burden or better care access). Second, by combining multiple survey waves, this study assumes mental health outcomes are stable across time. Third, not having access to Level 2 ethnicity data, and the consequent inability to identify Tokelauans for analysis, may have introduced misclassification bias, potentially producing underestimated effect sizes in the analysis by Realm country status. However, it is worth noting that Tokelau has a different governance structure within the Realm of New Zealand and was annexed by the Crown slightly later than the Cook Islands and Niue;[5] therefore, is it possible Tokelauan mental health differs from Cook Islanders' and Niueans'. Nevertheless, these ethnicity data constraints do signal a need for national survey administrators to consider either modifying the standard ethnicity question by including "Tokelauan" as a response option or ensuring

Level 2 ethnicity data are made available to researchers under conditions where data confidentiality is preserved in research outputs (e.g., aggregated Pacific sub-groups).

Conclusions

In conclusion, this study demonstrates that mental health outcomes vary significantly within Pacific peoples, which suggests mental health promotion should be targeted to Pacific ethnic groups with higher needs, particularly Cook Islands Māori and Niueans—both with Pacific homelands that fall under the Realm of New Zealand. To enable a more accurate understanding of Pacific mental health inequities, a routine national survey with a structured diagnostic interview schedule is needed, particularly since the previous survey of this nature—*Te Rau Hinengaro*—is 20 years old. Data access policies should allow researchers to use Pacific ethnicity data on Tokelauans under conditions where identifiability issues can be mitigated through aggregated research outputs. This study also shows that within-Pacific variability in mental health was not accounted for by socio-demographic or socio-economic differences between the Pacific ethnic groups. This supports a socio-ecological view of Pacific mental health and suggests researchers and policymakers should consider how broader structural and social determinants of mental health influence individual Pacific psychology, and how broad-based interventions could help reduce Pacific mental health inequities.

COMPETING INTERESTS

JK was part of a joint study in 2022 that investigated Pacific child and adult mental health using a pooled NZHS analysis. The study was led by research team members from the Ministry for Pacific Peoples and supported by the Better Start Big Data team.

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Appendix

Appendix Table 1: Wald test results for diagnosed common mental disorder and psychological distress in Pacific adults aged 15 years, by Pacific ethnicity, *New Zealand Health Survey*, 2014/2015–2018/2019.

Post-tests	Common mental disorder (diagnosed) n=497		Psychological distress (K10 ≥12) n=532	
	$F\text{-test}_{(1,99)}$	p-value	$F\text{-test}_{(1,99)}$	p-value
Tongan vs Cook Islands Māori	9.33	0.0029*	1.04	0.3103
Tongan vs Other Pacific	4.84	0.0302*	0.64	0.4261
Cook Islands Māori vs Other Pacific	1.02	0.3156	2.97	0.0878

*p<.05.
Model adjusted for age, gender, New Zealand Index of Deprivation (NZDep) and education.

Appendix Table 2: Relative risks of mental health outcomes in Pacific adults aged 15 years, by Pacific ethnicity and Realm country status, *New Zealand Health Survey*, 2014/2015–2018/2019.

		Mental health outcomes RR (95% CI)	
		Common mental disorder (diagnosed) n=502	Psychological distress (K10 ≥12) n=532
Model 1: unadjusted			
Pacific ethnicity	Samoan	1 (reference)	1 (reference)
	Tongan	0.91 (0.65, 1.26)	1.14 (0.85, 1.54)
	Cook Islands Māori	1.68* (1.31, 2.14)	1.37* (1.04, 1.81)
	Other Pacific	1.58* (1.15, 2.16)	1.02 (0.76, 1.37)
Realm country status	Non-Realm	1 (reference)	1 (reference)
	Realm	1.62* (1.29, 2.03)	1.13 (0.89, 1.45)
Model 2: adjusted for age and gender			
Pacific ethnicity	Samoan	1 (reference)	1 (reference)
	Tongan	0.92 (0.67, 1.28)	1.16 (0.86, 1.55)
	Cook Islands Māori	1.65* (1.29, 2.10)	1.36* (1.03, 1.80)
	Other Pacific	1.47* (1.08, 2.01)	1.00 (0.75, 1.34)
Realm country status	Non-Realm	1 (reference)	1 (reference)
	Realm	1.59* (1.27, 2.00)	1.12 (0.87, 1.43)

Appendix Table 2 (continued): Relative risks of mental health outcomes in Pacific adults aged 15 years, by Pacific ethnicity and Realm country status, *New Zealand Health Survey*, 2014/2015–2018/2019.

Model 3: adjusted for age, gender and NZDep			
Pacific ethnicity	Samoan	1 (reference)	1 (reference)
	Tongan	0.93 (0.68, 1.29)	1.16 (0.86, 1.56)
	Cook Islands Māori	1.63* (1.27, 2.08)	1.37* (1.04, 1.81)
	Other Pacific	1.40* (1.02, 1.92)	1.00 (0.75, 1.35)
Realm country status	Non-Realm	1 (reference)	1 (reference)
	Realm	1.60* (1.27, 2.00)	1.12 (0.88, 1.43)
Model 4: adjusted for age, gender, NZDep and education			
Pacific ethnicity	Samoan	1 (reference)	1 (reference)
	Tongan	0.94 (0.68, 1.31)	1.19 (0.87, 1.62)
	Cook Islands Māori	1.67* (1.28, 2.17)	1.41* (1.06, 1.89)
	Other Pacific	1.43* (1.04, 1.98)	1.03 (0.77, 1.39)
Realm country status	Non-Realm	1 (reference)	1 (reference)
	Realm	1.65* (1.30, 2.08)	1.14 (0.89, 1.48)

*p<.05.
RR = risk ratio; CI = confidence interval; NZDep = New Zealand Index of Deprivation.

Capturing diversity in cancer incidence and outcomes among the New Zealand Pacific population using linked administrative data

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ABSTRACT

AIM: The New Zealand population defined as "Pacific" is ethnically diverse, but this diversity is seldom examined in health research. This paper applies novel methods for describing health outcomes for specific Pacific populations in New Zealand using all-cancer and gastric cancer incidence and mortality as examples. Effects of loss to follow-up from leaving the country are also assessed.

METHOD: The New Zealand Cancer Registry was linked to administrative datasets and analysed within Stats NZ's Integrated Data Infrastructure (IDI). All-cancer and gastric cancer incidence was examined over the 1995–2022 period, as well as 1–5-year mortality among 1998–2017 diagnoses.

RESULTS: There was variability in age-standardised all-cancer incidence and gastric cancer incidence for different Pacific groups. Less variation in mortality was identified between groups, and these rates increased only modestly when adjusting for those who left the country. Lower all-cancer mortality was observed in 2008–2017 compared with 1998–2007.

CONCLUSION: Variation in health outcomes among specific Pacific ethnicities is masked when examining Pacific peoples as an aggregated ethnic group. However, small counts among small ethnicities create challenges for producing detailed, reliable data when using the IDI.

New Zealand's Pacific population count reached 442,632 in the 2023 Census, representing 8.9% of the total population and a 16% increase over the 2018 Census.[1] The Pacific population comprises several specific ethnicities, including Samoan, Cook Islands Māori, Tongan, Niuean, Tokelauan and Fijian ethnicities, which are categorised under "Level 2" of the Statistical Standard for Ethnicity.[2] Demographic differences between Level 2 Pacific ethnic groups include population size, migration histories and right of travel to and from New Zealand. Cook Islands Māori, Niueans and Tokelauans, for example, are much more likely to have been born in New Zealand and are also more likely to identify with multiple other ethnic groups.[3] This diversity drives explicit calls, such as from the Ministry for Pacific Peoples, for detailed analysis of outcomes for specific Pacific ethnic groups.[4]

This study addresses this need by examining cancer incidence and mortality by Level 2 Pacific ethnicity. Pacific peoples experience higher incidence rates of many cancers and lower survival than non-Pacific, non-Māori New Zealanders.[5–10] It is possible that cancer incidence and outcomes vary by specific Pacific ethnicity,[11] but there is currently little research into this. We also examine the impact of outward migration on mortality estimates.

Movement of Pacific peoples between New Zealand and the island nations has often been raised as potentially inflating cancer survival estimates among the population.[6,9,11–13] Specifically, it is possible that Pacific peoples are more likely to leave the country to return to a home nation following a cancer diagnosis, meaning survival outcomes (potential deaths) may not be captured by the New Zealand administrative system. Prior research has estimated that this form of return migration could result in a 4–12% underestimation of deaths among Pacific peoples, and that return migration could be greater among Samoan and Tongan people due to more recent migration among these groups.[11] New Zealand citizens from Realm countries (i.e., Cook Islands, Niue and Tokelau) also have freedom of travel to New Zealand and are entitled to publicly funded New Zealand healthcare.[14] This could make this issue more likely among Realm country ethnicities (Realm ethnicity is used here to

refer to those with Cook Islands Māori, Tokelauan or Niuean ethnicity) who may receive a cancer a diagnosis in New Zealand. However, there are currently no published data that quantify the scale of this type of migration and the impact on survival estimates.

We examined cancer incidence and mortality (all cancers and gastric cancer specifically) among the Level 2 Pacific ethnicities using Stats NZ's Integrated Data Infrastructure (IDI). The IDI is a collection of linked, de-identified administrative microdata about people and households from providers such as the Ministry of Health (MOH), Department of Internal Affairs (DIA) and Stats NZ (e.g., censuses).[15] We examined 1995–2022 cancer incidence (the full range of data available in the New Zealand Cancer Registry [NZCR] in the IDI) and 1–5-year mortality rates among those diagnosed during a 20-year period from 1998 to 2017. Gastric cancer incidence and mortality is relatively high among Pacific peoples compared with Europeans in New Zealand, and it was the sixth most diagnosed cancer from 2007 to 2019 among Pacific peoples.[6] Yet counts of gastric cancer cases are relatively low. It is, therefore, both an important cancer to monitor with the Pacific population and a useful demonstration on the potential possibilities and limitations of reporting on small counts of specific cancer diagnoses within smaller specific Pacific populations—particularly when using the IDI.

The key benefit of the IDI is the ability to link the NZCR to other administrative datasets to expand the scope of available information that is not routinely collected in the NZCR. In particular, here we link the NZCR to New Zealand border movement data to quantify the level of emigration from New Zealand following diagnoses. This enables us to examine mortality rates when adjusting for those departing the country without return. However, confidentiality requirements when using IDI data, including suppression of counts below 6 and random rounding of counts to base 3, pose challenges when examining small population outcomes, which we demonstrate here.

Overall, our aims were to: a) examine variation in age-standardised cancer incidence (all cancers and gastric cancer) for those of Level 2 Pacific ethnicity, b) capture diversity in all-cancer and gastric cancer mortality for Level 2 Pacific ethnicities, c) quantify the amount of outward migration from New Zealand among Pacific peoples diagnosed with gastric cancer, and the extent to which this impacts mortality estimates, and d) demonstrate the extent of error in Pacific mortality estimates created by the random rounding requirement for IDI data.

Method

Study population

We analysed Pacific peoples' first recorded diagnosis (any cancer, then gastric cancer specifically; International Classification of Disease [ICD] 10 codes C16-C169) in the cancer registry between 1995 and 2022 for incidence and 1998 and 2017 for mortality. Diagnosis date ranges for mortality estimates were based on data availability in the required collections in the IDI.

NZCR and IDI data sources

The NZCR contains information on all cancers first diagnosed in New Zealand, with diagnosis records from 1995 to 2022 available in the October 2023 refresh of the IDI. Ethnicity information is included to Level 2 in the NZCR, and ethnicity records are periodically updated. Up to three ethnicities are listed per person if they are present in at least 20% of a person's records across the National Health Index, mortality collection and hospital discharge data collections.[16]

For this study, we supplemented and expanded existing NZCR records with IDI data. For incidence rates among the population, we used the administrative population census (APC) as our source of denominator population counts. The APC is available within the IDI and is an estimated resident population based on activity in administrative records.[17] Ethnicity in the APC is coded using a source ranked approach, where the highest quality available source was used to code ethnicity. We used the 30 June 2008 APC for the denominator year for all years of gastric cancer diagnoses, as it is the midpoint of the study period. As such, incidence reflects both previous diagnoses made prior to the denominator date to 1995 and future diagnoses made following the denominator date to 2022. A total response approach to ethnicity coding was used, such that each individual was counted for each of their ethnic group affiliations. As such, ethnic groups in this study are not independent.

For mortality rates, Level 2 ethnicity information was updated using an "ever recorded" approach for records across the NZCR, 2013 and 2018 Census and DIA birth records (as either child or parents of a child). An individual was recorded as a given Level 2 Pacific ethnicity if that ethnicity was stated across any of these records. In this

way, we combined data across the three highest-quality data sources on Level 2 ethnicity in the IDI, rather than relying solely on MOH records (used in the NZCR), which tend to under-count Level 2 Pacific ethnicities relative to the 2013 Census.[19] A total response approach to ethnicity was taken for all analysis, such that each individual is included in each ethnic group they belong to. The NZCR was further joined to border movements data to determine whether a person had a record of leaving the country within 1–5 years of receiving their diagnosis, without a death record. Because border movement data are only available from 1997, we opted to examine diagnoses from 1998 for mortality rates to enable easier comparison of rates when adjusting for those who had left the country.

Mortality was determined based on linked mortality records (available for the full 2022 year). As death diagnosis information was only available up to 2018, examination of 1–5-year cancer-specific mortality was limited to the 1995–2013 diagnosis cohort.

Results

All-cancer and gastric cancer incidence among specific Pacific ethnicities

For 1995–2022 all-cancer incidence among the 2008 APC Pacific populations shown in Table 1, the overall rate of 201.7 per 100,000 among Pacific peoples in general masks some variability in all-cancer incidence among specific Pacific groups. In particular, annual average age-standardised incidence per 100,000 was highest among Cook Islands Māori (217.5) and Tongans (217.7), and lowest among Tokelauan (192.4) and Fijian people (160.7; see top half of Table 1). (Note, however, that the Fijian ethnic group is coded from administrative data records, which tend to produce a large over-count of the Fijian group relative to the 2013 and 2018 Census. This may be due to the coding of Fijian Indians as Fijian rather than or in addition to Indian, and results for the Fijian group should therefore be interpreted with caution.) It is important to note that as these are diagnoses between 1998 and 2017 among the 2008 estimated resident population, higher incidence here is a product of both higher rates of occurrence as well as greater survival among those diagnosed prior to the denominator reference date (June 2008).

There was also variability in gastric cancer incidence among Level 2 Pacific groups (see lower half of Table 1). The overall rate of 8.0 per 100,000 among Pacific peoples in general masks the higher rate observed among those of Niuean (10.7 per 100,000) and Samoan (9.5 per 100,000) ethnicity, and lower rates among Cook Islands Māori (6.1 per 100,000) and Tokelauans (6.4 per 100,000). Similar patterns can be observed among men and women. Sex rate ratios indicated that men generally had higher gastric cancer incidence than women, with the highest men–women ratio of 1.9 among Tongans. However, there were few cases of gastric cancer among some Level 2 Pacific ethnicities (particularly Tokelauans) from 1995 to 2022, thus confidence intervals indicate considerable uncertainty in some of these estimates.

All-cause mortality among 1998–2017 cancer cases

There was variation in all-cause mortality among the full cancer registry cohort (any diagnosis) across specific Pacific ethnicities. As shown in Table 2, 27% of Pacific peoples diagnosed with any cancer died within 1 year of their diagnosis, and 45% died within 5 years. However, 1-year mortality rates were higher for Tongan and Niuean people (31% and 32% respectively), and 5-year mortality rates were notably higher (48% and 51% respectively). Fijian people had clearly lower mortality across follow-up periods. Restricting cancer mortality analysis to only the aggregate Pacific group would mask this variation.

Examining all-cause mortality over time, rates had improved (decreased) for all Pacific ethnic groups in 2008–2017 compared with 1998–2007 (see Figure 1; underlying counts available in Appendix Table 1). For the overall Pacific group, mortality over a 1–5-year period was 6–7 percentage points lower in 2008–2017. Across Level 2 Pacific ethnic groups, there was some variability in mortality reduction. One-to-5-year mortality among Samoans decreased the most (by 8–9%). Reductions in mortality rates tended to be smaller for Cook Islands Māori (3–4%) and Tokelauans (0–4%).

Table 3 provides the counts of people diagnosed with any cancer from 1998 to 2017 and who left the country within 1–5 years of their first diagnosis (without subsequent records of return to the country or death records). The data indicate that rates of loss to follow-up through departures are generally low, but higher among Pacific peoples than the total cancer cohort. For Pacific peoples overall, 3.8% left the country within 5 years of their diagnosis (vs 1% of the total cases), but this was highest for Fijians (4.2%) and Samoans

Table 1: Average annual rates (all-cancer and gastric cancer) per 100,000 among Level 2 Pacific ethnicities in the 2008 APC diagnosed 1995–2022.

				Age-standardised rates			
	2008 APC population	**Total cancer cases**	**Annual crude rate per 100,000**	**Total annual age-standardised rate per 100,000**	**Men (95% CI)**	**Women (95% CI)**	**Rate ratio (Men: Women; 95% CI)**
Full cancer registry							
Pacific	340,560	13,134	137.7	201.7 (198.2–205.1)	198.7 (193.6–203.8)	208.8 (204.0–213.6)	1.0 (0.9–1.0)
Samoan	161,031	5,991	132.9	201.2 (196.1–206.3)	198.2 (190.7–205.7)	207.8 (200.7–214.8)	1.0 (0.9–1.0)
Cook Islands Māori	67,836	2,511	132.2	217.5 (209.0–226.0)	215.6 (202.8–228.3)	222.9 (211.3–234.5)	1.0 (0.9–1.0)
Tongan	64,554	2,349	130.0	217.7 (208.9–226.5)	205.4 (193.0–217.9)	235.0 (222.3–247.8)	0.9 (0.8–0.9)
Niuean	25,476	879	122.8	195.5 (182.5–208.4)	204.6 (184.4–224.8)	194.4 (177.0–211.7)	1.1 (0.9–1.2)
Tokelauan	6,963	240	123.0	192.4 (168.1–216.8)	178.4 (142.7–214.1)	207.3 (173.8–240.8)	0.9 (0.7–1.1)
Fijian	27,195	987	129.6	160.7 (150.7–170.8)	165.9 (250.4–181.4)	161.4 (147.9–174.9)	1.0 (0.9–1.2)
Other Pacific	13,140	516	140.2	206.1 (188.3–223.9)	187.2 (162.1–212.4)	224.8 (199.5–250.1)	0.8 (0.7–1.0)
Gastric cancer cases							
Pacific	-	492	5.2	8.0 (7.3–8.7)	9.5 (8.3–10.6)	6.7 (5.8–7.6)	1.4 (1.2–1.7)
Samoan	-	261	5.8	9.5 (8.4–10.7)	11.2 (9.4–13.0)	8.2 (6.7–9.7)	1.4 (1.1–1.7)
Cook Islands Māori	-	72	3.8	6.1 (4.7–7.5)	7.4 (5.1–9.8)	5.0 (3.3–6.7)	1.5 (0.9–2.4)
Tongan	-	84	4.6	7.8 (6.2–9.5)	10.5 (7.8–13.2)	5.4 (3.4–7.4)	1.9 (1.2–3.1)
Niuean	-	45	6.3	10.7 (7.6–13.9)	12.1 (7.3–16.9)	9.3 (5.3–13.3)	1.3 (0.7–2.3)

Table 1 (continued): Average annual rates (all-cancer and gastric cancer) per 100,000 among Level 2 Pacific ethnicities in the 2008 APC diagnosed 1995–2022.

				Age-standardised rates			
	2008 APC population	**Total cancer cases**	**Annual crude rate per 100,000**	**Total annual age-standardised rate per 100,000**	**Men (95% CI)**	**Women (95% CI)**	**Rate ratio (Men: Women; 95% CI)**
Tokelauan	-	6	4.6	6.4 (1.3–11.5)	4.0 (S–S)	7.8 (S–S)	0.5 (S–S)
Fijian	-	18	2.4	2.8 (1.5–4.1)	3.4 (1.2–5.6)	2.3 (0.8–3.8)	1.5 (0.6–3.7)
Other Pacific	-	15	4.1	6.1 (3.0–9.1)	6.1 (2.1–10.0)	5.9 (1.2–10.5)	1.0 (0.4–2.9)

APC = administrative population census; 95% CI = confidence interval; S = suppressed data due to counts under 6, in accordance with Stats NZ confidentiality requirements.
Age-standardised to World Health Organization (2000–2025) Standard Population. Counts have been random rounded to base 3 according to Stats NZ Integrated Data Infrastructure confidentiality requirements. Crude rate based on rounded values. Age-standardised rates are based on unrounded values.

Table 2: One-to-5-year all-cause mortality rates for any diagnosed cancer from 1998 to 2017.

Ethnicity	Individuals diagnosed	1-year mortality (95% CI)	2-year mortality (95% CI)	3-year mortality (95% CI)	5-year mortality (95% CI)
All	370,518	25.3 (25.1–25.4)	33.0 (32.8–33.1)	37.6 (37.5–37.8)	44.1 (43.9–44.3)
Pacific	15,030	27.4 (26.7–28.1)	35.3 (34.6–36.1)	39.6 (38.8–40.4)	44.7 (43.9–45.5)
Samoan	6,654	27.6 (26.6–28.7)	35.4 (34.2–36.5)	39.6 (38.5–40.8)	44.7 (43.5–45.9)
Cook Islands Māori	2,937	27.7 (26.1–29.3)	35.4 (33.7–37.2)	39.7 (38.0–41.5)	45.5 (43.7–47.3)
Tongan	2,751	31.1 (29.4–32.8)	39.4 (37.5–41.2)	44.0 (42.1–45.9)	48.1 (46.2–50.0)
Niuean	1,017	31.6 (28.7–34.4)	40.1 (37.1–43.1)	45.7 (42.7–48.8)	50.7 (47.7–53.8)
Tokelauan	309	25.2 (20.4–30.1)	37.5 (32.1–42.9)	42.7 (37.2–48.2)	50.5 (44.9–56.1)
Fijian	1,497	17.5 (15.5–19.4)	24.0 (21.9–26.2)	27.3 (25.0–29.5)	32.5 (30.1–34.8)
Other Pacific	1,266	26.3 (23.9–28.7)	32.7 (30.1–35.3)	37.0 (34.3–39.6)	42.2 (39.5–44.9)
Realm	4,203	28.6 (27.2–29.9)	37.0 (35.5–38.4)	41.6 (40.1–43.1)	47.3 (45.8–48.8)

95% CI = 95% confidence interval.
Realm includes New Zealand Realm country ethnicities (Cook Islands Māori, Niuean, Tokelauan).

Figure 1: All-cause mortality among all cancer cases diagnosed from 1998 to 2007 and 2008 to 2017. Error bars represent 95% confidence intervals.

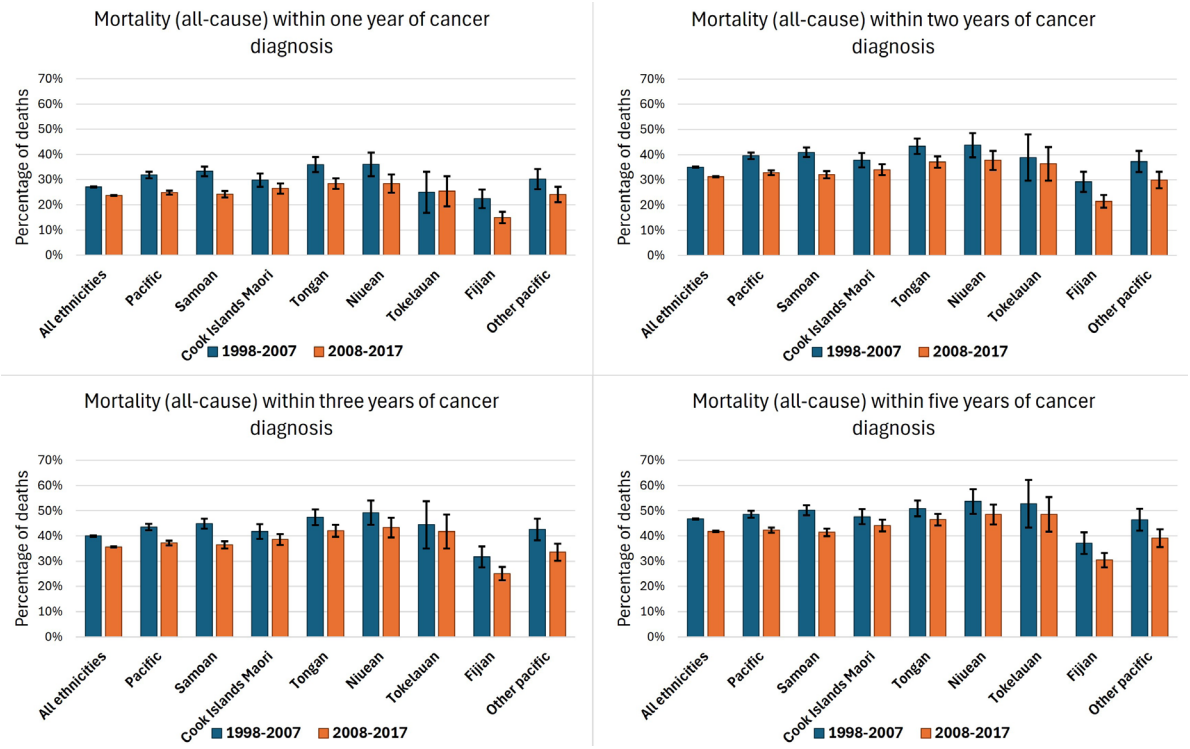


Table 3: Counts of individuals diagnosed with any cancer between 1998 and 2017 and who left New Zealand within the specified follow-up period without ever returning or having a death record.

Ethnicity	Total diagnosed	Total who left without record of return or death within specified period			
		1-year post-diagnosis	2-year post-diagnosis	3-year post-diagnosis	5-year post-diagnosis
All	370,518	1,845 (0.5%)	2,499 (0.7%)	3,021 (0.8%)	3,810 (1.0%)
Pacific	15,030	321 (2.1%)	417 (2.8%)	486 (3.2%)	573 (3.8%)
Samoan	6,654	159 (2.4%)	198 (3.0%)	222 (3.3%)	267 (4.0%)
Cook Islands Māori	2,937	57 (1.9%)	78 (2.7%)	90 (3.1%)	108 (3.7%)
Tongan	2,751	57 (2.1%)	69 (2.5%)	84 (3.1%)	96 (3.5%)
Niuean	1,017	9 (0.9%)	9 (0.9%)	12 (1.2%)	18 (1.8%)
Tokelauan	309	6 (1.9%)	9 (2.9%)	9 (2.9%)	12 (3.9%)
Fijian	1,497	27 (1.8%)	42 (2.8%)	54 (3.6%)	63 (4.2%)
Other Pacific	1,266	21 (1.7%)	27 (2.1%)	33 (2.6%)	39 (3.1%)
Realm	4,203	72 (1.7%)	99 (2.4%)	114 (2.7%)	135 (3.2%)

Table 4: Difference (increase) in 1–5-year all-cause mortality rates for any diagnosed cancer from 1998 to 2017 after removing individuals who left the country during the follow-up period without record of return or death.

Ethnicity	1-year mortality rate difference	2-year mortality rate difference	3-year mortality rate difference	5-year mortality rate difference
All	0.1%	0.2%	0.3%	0.5%
Pacific	0.6%	1.0%	1.3%	1.8%
Samoan	0.7%	1.1%	1.4%	1.9%
Cook Islands Māori	0.5%	1.0%	1.2%	1.7%
Tongan	0.7%	1.0%	1.4%	1.7%
Niuean	0.3%	0.5%	0.7%	0.8%
Tokelauan	0.8%	0.9%	1.3%	1.6%
Fijian	0.3%	0.7%	1.0%	1.5%
Other Pacific	0.4%	0.7%	1.0%	1.3%
Realm	0.5%	0.9%	1.2%	1.6%

Percentage change reflects percentage point increase in mortality. Adjusted mortality rates with 95% confidence intervals are available in Appendix Table 2.

(4.0%), and lowest for Niueans (1.8%). As shown in Table 4, adjusting for those who left the country (who may be lost to follow-up and were therefore removed from the calculations) increased mortality percentages by only 0.1–1.9 points, with a larger difference over longer follow-up periods (as people are more likely to leave over longer periods of time).

All-cause and cancer-specific mortality among gastric cancer cases

All-cause mortality among the gastric cancer cohort was reasonably consistent across specific Pacific ethnicities (see Table 5). All-cause mortality was slightly lower among Niueans and Fijians, although confidence intervals were larger among the cohort due to the smaller number of cases.

Mortality for the 1998–2007 and 2008–2017 diagnosis periods are displayed in Figure 2. For some ethnicities (e.g., Tokelauan), mortality cannot be presented due to low case counts that cannot be outputted from the IDI. For other smaller Pacific ethnic groups including Fijian, other Pacific and Niuean, low mortality counts over the different follow-up periods also mean the random rounding requirement for outputting of counts has a larger effect on uncertainty in the mortality rate. To demonstrate this, plotted error bars represent random rounding uncertainty intervals based on the range of numerator and denominator values for each rate that could have been rounded to the outputted count. For example, there were 12 deaths among the Fijian gastric cohort over a 5-year follow-up period during 2008–2017 (see Appendix Table 3 for counts), but random rounding means this could reflect an unrounded value of anywhere between 10 and 14. Due to this effect of random rounding, caution should be taken when interpreting or comparing results for these smaller Pacific groups.

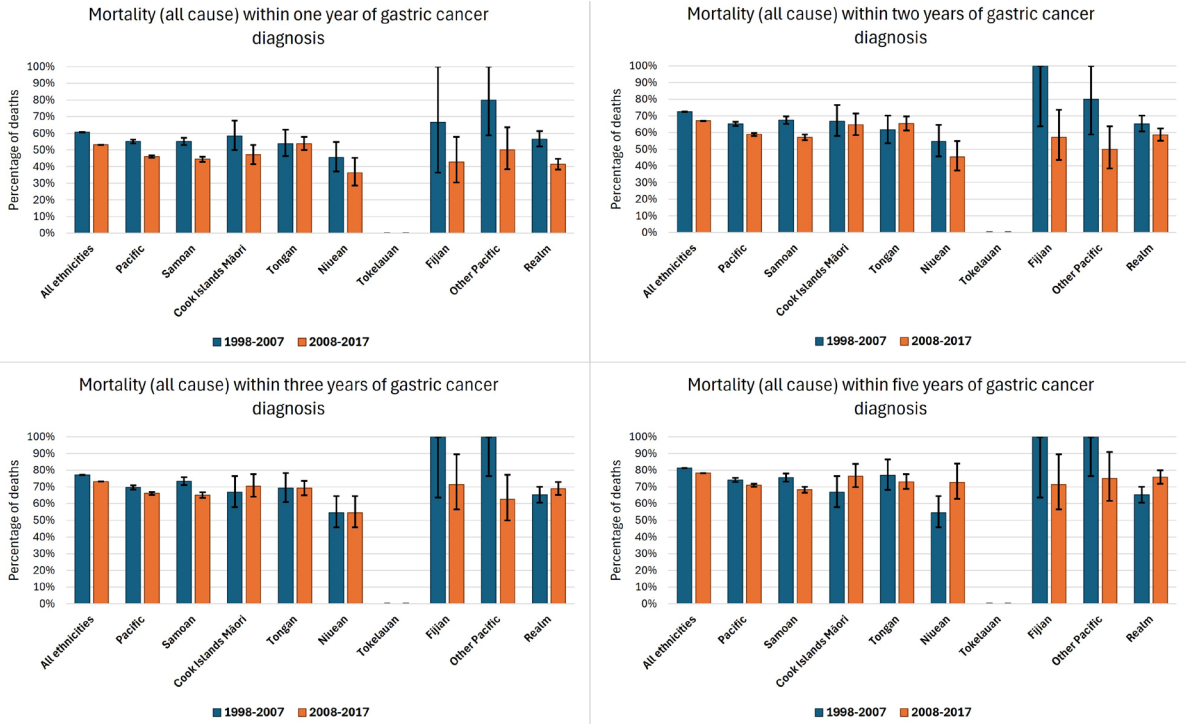
For the overall Pacific ethnic group, 1–5-year all-cause mortality decreased by approximately 3–9 percentage points in 2008–2017, relative to 1998–2007. Changes over time among Level 2 Pacific ethnicities, however, were mixed. For the largest Pacific ethnicities, all-cause mortality was consistently lower in 2008–2017 among Samoans. However, mortality was very similar, and often higher in 2008–2017 for Cook Islands Māori and Tongans, relative to 1998–2007 (but note the large overlap in confidence intervals). For Realm country ethnic groups combined (Cook Islands Māori,

Table 5: One-to 5-year all-cause mortality rates for gastric cancer diagnoses from 1998 to 2017.

Ethnicity	Individuals diagnosed	1-year mortality (95% CI)	2-year mortality (95% CI)	3-year mortality (95% CI)	5-year mortality (95% CI)
All	7,686	56.8 (55.7–57.9)	69.6 (68.6–70.7)	75.1 (74.2–76.1)	79.8 (78.9–80.7)
Pacific	645	49.3 (45.4–53.2)	61.4 (57.6–65.2)	67.0 (63.3–70.6)	72.1 (68.6–75.6)
Samoan	336	49.1 (43.8–54.5)	62.2 (57.0–67.3)	67.9 (62.9–72.9)	72.3 (67.5–77.1)
Cook Islands Māori	90	48.3 (38.0–58.6)	62.1 (52.0–72.1)	69.0 (59.4–78.5)	73.3 (64.2–82.5)
Tongan	120	51.2 (42.3–60.2)	62.5 (53.8–71.2)	67.5 (59.1–75.9)	72.5 (64.5–80.5)
Niuean	63	45.5 (33.2–57.8)	54.5 (42.2–66.8)	61.9 (49.9–73.9)	66.7 (55.0–78.3)
Tokelauan	9	S	S	S	S
Fijian	36	45.5 (29.2–61.7)	54.5 (38.3–70.8)	58.3 (42.2–74.4)	66.7 (51.3–82.1)
Other Pacific	42	57.1 (42.2–72.1)	60.0 (45.2–74.8)	71.4 (57.8–85.1)	78.6 (66.2–91.0)
Realm	156	47.1 (39.2–54.9)	59.6 (51.9–67.3)	66.0 (58.6–73.5)	73.1 (66.1–80.0)

95% CI = 95% confidence interval; S = suppressed data due to counts under 6, in accordance with Stats NZ confidentiality requirements.

Figure 2: All-cause mortality among gastric cancer cases diagnosed from 1998 to 2007 and 2008 to 2017. Note the error bars represent uncertainty intervals of estimates based on random rounding to base 3 for underlying counts—a requirement of Integrated Data Infrastructure data outputting.



Niuean, Tokelauan), 3- and 5-year mortality was also higher in 2008–2017, compared with 1998–2007.

Mortality rates where gastric cancer was the underlying cause of death are presented in Table 6. Although the data correspond to a different diagnosis period (1995–2013, due to the limited availability of cause of death data), these rates were generally similar to the all-cause mortality rates. The largest difference between all-cause and gastric cancer–specific mortality is for the Fijian group, who had among the lowest all-cause mortality rates but highest gastric cancer–caused mortality.

Discussion

Using linked NZCR and administrative data, we identified variation in cancer incidence and outcomes among specific Pacific groups in New Zealand. Among the 2008 Pacific population, the overall 1995–2022 annual age-standardised cancer incidence of 202 per 100,000 masked higher incidence for Cook Islands Māori and Tongan ethnicities (218/100,00) in particular. The overall age-standardised gastric cancer rate of 8 per 100,000 also masked higher rates for Samoan (10 per 100,000) and Niuean ethnicities (11 per 100,000). All-cause mortality was also higher for Tongan and Niuean ethnicities for all cancers diagnosed between 1998 and 2017 across 1–5-year follow-up periods. However, there tended to be less variation in all-cause mortality among the gastric cancer cohort, with slightly lower mortality for Niuean ethnicity.

All-cause mortality for all cancers was around 6 percentage points lower in 2008–2017 compared with 1998–2007, regardless of follow-up period, with the largest decrease occurring for Samoan ethnicity (approximately 9 percentage point reduction). Changes in all-cause mortality among gastric cancer cases was more variable, but with the largest reductions again seen for Samoan ethnicity. In some cases, all-cause mortality increased in the 2008–2017 period; however, this may be due to smaller sample sizes when examining rates among a narrower diagnosis period. Random rounding required for outputting IDI data has much larger effects on small counts, creating a significant challenge in providing reliable, detailed health data for small populations. It will be important to find solutions to this challenge, such as case-by-case exceptions by

Table 6: One-to-5-year mortality rates for gastric cancer diagnoses from 1995 to 2013 where gastric cancer was the underlying cause of death.

Ethnicity	Individuals diagnosed	1-year mortality (95% CI)	2-year mortality (95% CI)	3-year mortality (95% CI)	5-year mortality (95% CI)
All	7,251	59.7 (58.6–60.9)	71.9 (70.9–72.9)	77.2 (76.3–78.2)	81.5 (80.7–82.4)
Pacific	552	51.6 (47.5–55.8)	63.9 (59.9–67.9)	69.2 (65.3–73.0)	73.4 (69.7–77.1)
Samoan	288	49.5 (43.7–55.3)	62.5 (56.9–67.9)	69.1 (63.7–74.4)	71.9 (66.7–77.1)
Cook Islands Māori	78	53.8 (42.8–64.9)	65.4 (54.8–75.9)	69.2 (59.0–79.5)	73.1 (63.2–82.9)
Tongan	99	51.5 (41.7 –61.4)	64.7 (55.3–74.1)	69.7 (60.6–78.7)	75.8 (67.3–84.2)
Niuean	57	52.6 (39.7–65.6)	63.2 (50.6–75.7)	68.4 (56.4–80.5)	73.7 (62.3–85.1)
Tokelauan	S	S	S	S	S
Fijian	27	60.0 (41.5–78.5)	66.7 (48.9–84.4)	75.0 (58.7–91.3)	77.8 (62.1–93.5)
Other Pacific	42	53.3 (38.2–68.4)	57.1 (42.2–72.1)	66.7 (52.4–80.9)	78.6 (66.2–91.0)
Realm	135	54.5 (46.1–62.9)	66.7 (58.7–74.6)	70.5 (62.8–78.2)	75.6 (68.3–82.8)

95% CI = 95% confidence interval; S = suppressed data due to counts under 6, in accordance with Stats NZ confidentiality requirements.

Stats NZ, to investigate questions that can only be addressed using linked administrative data.

It is important to note that we counted all cases in each specific Pacific group reported. Thus, although differences can be observed between ethnic groups, they cannot be formally statistically compared as individuals reporting multiple Pacific ethnicities mean the groups are not mutually exclusive. There are also issues of data quality of ethnicity collection to bear in mind. In particular, age-standardised gastric cancer incidence and gastric cancer mortality appeared lower among the Fijian group compared to other Pacific ethnicities. However, previous work has identified an over-count of the Fijian group in administrative data sources compared with the Census.[18,19] The standard classification of ethnicity requires Fijian Indian ethnicity to be coded as Indian (under the Level 1 Asian ethnic category) rather than Fijian (under the Level 1 Pacific category), but evidence suggests Fijian Indian ethnicity is often recorded as Fijian or both Fijian and Indian, leading to an over-count of the Fijian group.[19,20] This makes it difficult to determine whether our results reflect true differences or are influenced by coding errors in the administrative data sources and reinforces the importance of adhering to data collection and ethnicity classification standards when coding data. Despite this, we used consistent sources of information on ethnicity for numerator and denominator data where necessary, removing the impact of numerator–denominator bias.

By analysing linked data in the IDI we were able to quantify the extent of emigration from New Zealand following cancer diagnosis and what effect this might have on mortality estimates. Although we identified higher rates of loss to follow-up emigration (i.e., records of an individual leaving the country without return or record of death) among Pacific peoples (3.8% of those diagnosed from 1998 to 2017 within a 5-year period, compared with 1.0% of diagnosed individuals in general), these generally had only small effects on basic mortality rates of within 0–2 percentage points (with a larger impact on longer follow-up periods). Nonetheless, we demonstrate methods developed for detailed analyses for the Pacific population that consider these important factors.[18]

Samoan and Tongan people have been previously theorised to be most likely to exhibit return migration than other Level 2 Pacific ethnic groups as they are less likely to be born in New Zealand and Tonga and Samoa have more substantial health service infrastructure than other island nations.[11] It is also possible that ethnicities associated with Realm countries (Cook Islands, Niue, Tokelau) may have higher rates of loss to follow-up if they travelled to New Zealand specifically for healthcare. Our results show loss to follow-up was most common for Samoans and Fijians (4.0% and 4.2% respectively over a 5-year post-diagnosis period). Cook Islands Māori and Tokelauans (i.e., those associated with Realm nations) also had relatively high rates of return migration, but Niueans had the lowest (1.8%). Relatedly, travel by Realm-nation citizens to New Zealand could also inflate the incidence for those ethnicities in New Zealand. However, there is currently no information in the NZCR or IDI that indicates for whom that is the case.

Overall, the findings emphasise the importance of generating key cancer statistics for specific Pacific ethnic groups in New Zealand. The IDI provides a useful tool for examining issues that are beyond the scope of the NZCR alone. However, data confidentiality rules for outputting data from the IDI (suppression of counts under 5, and random rounding of all counts) mean basic incidence and mortality rates would be best produced routinely from the NZCR outside the IDI, especially where differences in incidence and outcomes are identified for specific Pacific ethnicities. Analysis of the NZCR outside the IDI would also enable more timely production of data, as data in the IDI is updated roughly four times a year, with collections such as the NZCR being updated less frequently (e.g., data to the end of 2022 were available in the October 2023 IDI refresh). Future research should continue to examine where cancer incidence and outcomes differ between specific Pacific (and other ethnic) groups to enable improved surveillance for these groups. This may be particularly beneficial for more common cancers, such as breast, prostate or lung, for example.[6]

COMPETING INTERESTS
Nil.
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DISCLAIMERS
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).

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Appendix Table 1: All cancer diagnoses and deaths (all-cause) by 10-year diagnosis period.

	1998–2007 cancer diagnoses					2008–2017 cancer diagnoses				
Ethnicity	**N Diagnosed**	**5-year deaths**	**3-year deaths**	**2-year deaths**	**1-year deaths**	**N Diagnosed**	**5-year deaths**	**3-year deaths**	**2-year deaths**	**1-year deaths**
All	170,625	79,776	68,190	59,793	46,254	199,893	83,634	71,292	62,391	47,448
Pacific	5,571	2,709	2,427	2,208	1,773	9,453	4,002	3,522	3,108	2,349
Samoan	2,499	1,254	1,122	1,023	831	4,155	1,722	1,515	1,332	1,008
Cook Islands Māori	1,095	522	459	414	327	1,842	813	711	627	489
Tongan	996	507	471	432	357	1,755	816	738	651	498
Niuean	408	219	201	177	147	612	297	264	231	174
Tokelauan	108	57	48	42	27	204	99	84	72	51
Fijian	492	183	156	144	111	1,005	306	252	216	150
Other Pacific	504	234	216	189	153	759	297	255	228	183
Realm	1,593	795	702	630	498	2,613	1,194	1,047	924	705

Random rounding to base 3 has been applied to all values in this table.

Appendix Table 2: One-to-5-year all-cause mortality rates for any diagnosed cancer from 1998 to 2017 after removing individuals who left the country during the follow-up period without record of return or death.

Ethnicity	Individuals diagnosed	5-year mortality rate (proportion, 95% CI)	3-year mortality rate (proportion, 95% CI)	2-year mortality rate (proportion, 95% CI)	1-year mortality rate (proportion, 95% CI)
All	366,708	44.6 (44.4–44.7)	38.0 (37.8–38.1)	33.2 (33.0–33.4)	25.4 (25.3–25.6)
Pacific	14,454	46.4 (45.6–47.2)	40.9 (40.1–41.7)	36.4 (35.6–37.1)	28.0 (27.3–28.8)
Samoan	6,387	46.6 (45.4–47.8)	41.0 (39.8–42.2)	36.5 (35.3–37.7)	28.3 (27.2–29.4)
Cook Islands Māori	2,826	47.1 (45.3–49.0)	40.9 (39.1–42.8)	36.4 (34.6–38.2)	28.2 (26.5–29.9)
Tongan	2,652	49.8 (47.9–51.7)	45.4 (43.5–47.3)	40.4 (38.5–42.2)	31.8 (30.0–33.5)
Niuean	1,002	51.5 (48.4–54.6)	46.4 (43.3–49.5)	40.6 (37.6–43.6)	31.8 (29.0–34.7)
Tokelauan	294	52.0 (46.3–57.8)	44.0 (38.3–49.7)	38.4 (32.8–43.9)	26.0 (21.0–31.0)
Fijian	1,431	34.0 (31.5–36.4)	28.3 (25.9–30.6)	24.8 (22.6–27.0)	17.8 (15.8–19.7)
Other Pacific	1,227	43.5 (40.7–46.3)	38.0 (35.2–40.7)	33.4 (30.8–36.1)	26.7 (24.3–29.2)
Realm	4,068	48.9 (47.4–50.4)	42.8 (41.3–44.3)	37.8 (36.3–39.3)	29.1 (27.7–30.5)

Random rounding to base 3 has been applied to all values in this table.
95% CI = 95% confidence interval.

Appendix Table 3: Stomach cancer diagnoses and deaths (all-cause) by 10-year diagnosis period.

	1998–2007 gastric cancer diagnoses					2008–2017 gastric cancer diagnoses				
Ethnicity	**N Diagnosed**	**5-year deaths**	**3-year deaths**	**2-year deaths**	**1-year deaths**	**N Diagnosed**	**5-year deaths**	**3-year deaths**	**2-year deaths**	**1-year deaths**
All	3,816	3,105	2,943	2,763	2,313	3,867	3,027	2,832	2,589	2,052
Pacific	267	198	186	174	147	372	264	246	219	171
Samoan	147	111	108	99	81	189	129	123	108	84
Cook Islands Māori	36	24	24	24	21	51	39	36	33	24
Tongan	39	30	27	24	21	78	57	54	51	42
Niuean	33	18	18	18	15	33	24	18	15	12
Tokelauan	S	S	S	S	S	9	S	S	S	S
Fijian	9	9	9	9	6	21	15	15	12	9
Other Pacific	15	15	15	12	12	24	18	15	12	12
Realm	69	45	45	45	39	87	66	60	51	36

Random rounding to base 3 has been applied to all values in this table.
S = suppressed data due to counts under 6, in accordance with Stats NZ confidentiality requirements.

Differences in systemic treatments for breast cancer between patients with and without diabetes

Chunhuan Lao, Jason Gurney, James Stanley, Andrea Teng, Marion Kuper-Hommel, Ian Campbell, Jeremy Krebs, Dianne Sika-Paotonu, Jonathan Koea, Jeannine Stairmand, Ross Lawrenson

ABSTRACT

AIM: The objectives of this study are to investigate whether diabetes affects the systemic treatment of breast cancer.

METHODS: Patients diagnosed with invasive breast cancer between 2005 and 2020 were identified from the Te Rēhita Mate Ūtaetae – Breast Cancer Foundation National Register. Logistic regression modelling was used to estimate odds ratios (ORs) with 95% confidence intervals (95% CIs) for the outcomes of endocrine therapy for estrogen receptor+/progesterone receptor+ cancer, targeted therapy for human epidermal growth factor receptor 2+ (HER2) cancer and chemotherapy in patients with breast cancer, comparing those with and without diabetes.

RESULTS: Compared with patients without diabetes, patients with diabetes had lower probabilities of receiving endocrine therapy (64.2% vs 60.4%, p-value <0.001), HER2-targeted therapy (65.6% vs 54.8%, p-value <0.001) and chemotherapy (32.1% vs 20.4%, p-value <0.001). Most of the differences in receipt of endocrine therapy and HER2-targeted therapy between these two groups could be explained by adjustment for differences in age at diagnosis and comorbidity. The difference in usage of chemotherapy by diabetes status remained apparent after adjustment for other factors (OR 0.85, 95% CI 0.75–0.97), with a stronger difference in women with stage II breast cancer (OR 0.71, 95% CI 0.59–0.86) and in Pacific women (OR 0.70, 95% CI 0.51–0.94).

CONCLUSIONS: Women with diabetes are less likely to be treated with chemotherapy, and the difference is greatest in Pacific women and patients with stage II breast cancer. The lower usage of endocrine therapy and HER2-targeted therapy in patients with diabetes could be explained by the older age at diagnosis and more comorbidities.

Most patients with breast cancer are initially treated with surgery.[1] However, additional systemic treatment such as endocrine therapy, chemotherapy and targeted therapy can improve patient outcomes. The decision of whether to start these systemic treatments is based on patient and tumour factors. Patient factors include demographic factors such as age and access to care, comorbidities and patient choice. Tumour factors include stage of disease, grade and the presence of biomarkers. The common biomarkers for breast cancer are the estrogen receptor (ER), progesterone receptor (PR) and human epidermal growth factor receptor 2 (HER2). The main systemic treatments for breast cancer include endocrine therapy, chemotherapy and HER2-targeted therapy. Invasive tumours that are ER and/or PR positive (+) are sensitive to endocrine therapy,[2–4] which can then be prescribed for 5–10 years depending on the risk of cancer recurrence. Patients who have HER2+ breast cancer would benefit from HER2-targeted therapy.[5,6] HER2-targeted therapy including trastuzumab (funded since 2002), pertuzumab (funded since January 2017), trastuzumab emtansine (funded since December 2019) and lapatinib (funded since 2012 as a first-line treatment only) are funded for patients with HER2+ breast cancer in Aotearoa New Zealand.[3,6–8]

Treatment toxicity is an important factor for decision making by patients and clinicians on initiating or ceasing a particular treatment, especially for patients who are frail and have multiple comorbidities.[9] Diabetes is a significant and common comorbidity,[10] and may itself cause complications including heart disease, chronic kidney disease, peripheral vascular disease and neuropathy, and other problems with oral health, vision, hearing and mental health. The prevalence of diabetes and its secondary complications may therefore influence the use of these systemic treatments. Several factors may explain these phenomena, including clinical concern for treatment toxicity and ability to complete the

treatment. A systematic review showed that chemotherapy toxicity was greater in patients with comorbidity and was associated with a greater likelihood of hospitalisation during treatment.[11]

Twelve percent of women with breast cancer in Aotearoa New Zealand also have diabetes at cancer diagnosis, and the prevalence of diabetes in Māori, Pacific and Asian women in the general population is greater than for European women.[12–13] Breast cancer characteristics, treatments and outcomes were also reported to be different between these ethnic groups.[6,14–16] How diabetes affects the systemic treatment of breast cancer may also differ by ethnic group, especially for Māori and Pacific peoples. The objectives of this study are to investigate whether diabetes affects the systemic treatment of breast cancer in Aotearoa New Zealand, and whether these effects vary by ethnicity.

Methods

Women diagnosed with invasive breast cancer between 2005 and 2020 were identified from the Te Rēhita Mate Ūtaetae – Breast Cancer Foundation National Register (NBCR). Men were not included. The NBCR combines the Auckland, Waikato, Wellington and Christchurch registers, which includes 98% of prevalent breast cancer patients in these regions.[17] This study used data on age at diagnosis, menopausal status, ethnicity, diagnosis date, mode of detection (screen detected or symptomatic), tumour, node and metastasis (TNM) cancer stage (I, II, III and IV, 8th edition of American Joint Committee on Cancer [AJCC] Cancer Staging),[18] grade (1, 2 and 3), biomarkers (ER, PR and HER2) and systemic treatments. These NBCR data were linked by National Health Index (NHI) number to national-level health data to determine diabetes status (from the Virtual Diabetes Register [VDR]) at the time of cancer diagnosis, comorbidities (from the National Minimum Dataset [NMDS]) and systemic treatments (from Pharmaceutical Collection [PHARMS]). The NHI number is a unique identifier for people receiving healthcare services in Aotearoa New Zealand. The VDR is used to determine official diagnosed diabetes prevalence, but does not differentiate between type 1 and type 2 diabetes.[19] The PHARMS contains claim and payment information from pharmacists for subsidised dispensings. The VDR and PHARMS cover the whole population in Aotearoa New Zealand.

Patients were classified into Māori, Pacific, Asian and European/Other ethnic groups. Ethnicity is self-identified in Aotearoa New Zealand and recorded in the NBCR. Socio-economic deprivation was defined using the New Zealand Index of Deprivation 2018 (NZDep2018) and was analysed by quintile, from 1 (least deprived) to 5 (most deprived).[20] The study period was separated into three groups: 2005–2009, 2010–2014 and 2015–2020. Breast cancer subtypes were categorised into five groups according to biomarker status:[4,6,15,21] 1) luminal A: ER+, PR+ and HER2-; 2) luminal B HER2-: ER or PR+ (but not both +), HER2-; 3) luminal B HER2+: ER+ and/or PR+, HER2+; 4) HER2 non-Luminal: ER-, PR-, HER2+; and 5) triple negative: ER-, PR-, HER2-. Patients ever recorded as having diabetes before cancer diagnosis based on the VDR were considered to have diabetes at the time of cancer diagnosis. Comorbid conditions recorded on the NMDS for hospitalisations in the 5 years up to the index hospitalisation date were identified to calculate a C3 Index score for each patient.[22] The C3 Index is a cancer-specific index of comorbidity, with scores categorised into "0" (⩽0), "1" (⩽1.00), "2" (⩽2.00) and "3" (>2.00).[22] Diabetes was excluded as a comorbidity from the C3 Index score calculation.

We compared the proportion of patients with ER+/PR+ cancer who received endocrine therapy, the proportion of patients with HER2+ cancer who had targeted therapy and the proportion of women who had chemotherapy between the diabetes group and the non-diabetes group. Logistic regression modelling was used to estimate odds ratios (ORs) with 95% confidence intervals (95% CIs) for the outcomes of endocrine therapy for ER+/PR+ cancer, targeted therapy for HER2+ cancer, and chemotherapy in patients with breast cancer comparing those with and without diabetes. Analyses are reported stratified by cancer stage, after adjustment for period of diagnosis, age, menopausal status, ethnicity, deprivation quintile, mode of detection, comorbidities (C3 Index score), grade and biomarker subtype. The ORs were estimated from an overall model that included all patients, and then in separate analyses stratified by cancer stage and ethnic group.

All data analyses were performed in R 4.0 (R Institute, Vienna, Austria). Ethics approval for the study was granted through the University of Waikato Human Research Ethics Committee (reference: HREC(Health)2021#89).

Results

During the study period, 26,968 women were diagnosed with breast cancer, of whom 3,137

(11.6%) had a diabetes classification on the VDR at the time of cancer diagnosis. The characteristics of these patients have been reported in our earlier publication.[12] Of the 22,964 patients with ER+/PR+ cancer, 14,632 (63.7%) had endocrine therapy (Table 1). Among the 3,995 patients with HER2+ disease, 2,577 (64.5%) had HER2-targeted therapy. Only 31% (8,291/26,968) of all patients had chemotherapy. The proportions of patients receiving endocrine therapy, HER2-targeted therapy and chemotherapy were all lower in the patients with diabetes than in the patients without diabetes, and the relative gaps were wider in the stage I and II cancers than in the stage III and IV cancers. For example, for stage I and II cancers the percentages of patients having chemotherapy in the diabetes group were approximately half of those in the non-diabetes group, and for the stage III and IV cancers the percentages in the diabetes group were around 30% lower than in the non-diabetes group.

The unadjusted OR of having endocrine therapy for ER+/PR+ cancers for patients with diabetes versus patients without diabetes was 0.85 (95% CI 0.78–0.92, Table 2). Most of this difference could be explained by adjustment for differences in age at diagnosis and comorbidity between these two groups. Women with diabetes were older and had more comorbidities.[12] When stratifying the patients by cancer stage, the fully adjusted OR for endocrine therapy for ER+/PR+ disease by diabetes status was ameliorated for stage I, II and IV cancers. In contrast, the difference between the two groups for stage III cancer magnified after adjustment for other factors, with endocrine therapy being more common for women with diabetes than women without diabetes (OR 1.66, 95% CI 1.16–2.36).

For HER2-targeted therapy, the ORs between patients with diabetes and patients without diabetes followed a similar pattern as for endocrine therapy, before and after adjustment for other factors (Table 3). The unadjusted OR of having HER2-targeted therapy for patients with diabetes compared with patients without diabetes was 0.64 (95% CI 0.51–0.79), and the fully adjusted OR was 1.04 (95% CI 0.80–1.35). Age, ethnicity and comorbidity accounted for most of the difference in receipt of HER2-targeted therapy by diabetes status. When stratifying the cancer patients by stage, the adjusted OR increased with cancer stage, from 0.85 (95% CI 0.51–1.42) for stage I cancer to 1.75 (95% CI 0.78–4.50) for stage IV cancer. Results for stage III and IV suggested more use of targeted therapy in the group for those with diabetes.

The OR of having chemotherapy for patients with diabetes compared with patients without diabetes was 0.54 (95% CI 0.50–0.59) before adjustment, and 0.85 (95% CI 0.75–0.97) after adjustment for other factors (Table 4). Age and comorbidity still had the most substantial impact on the estimated ORs. When stratifying patients by cancer stage, there was strong evidence in stage II disease for reduced treatment for those with diabetes (adjusted OR 0.71, 95% CI 0.59–0.86), but there was no strong evidence for the other cancer stages.

Finally, we considered the differences in treatment receipt by diabetes status separately for ethnic group (Table 5). The unadjusted OR of having endocrine therapy for women with diabetes compared with women without diabetes varied by ethnic group. The unadjusted ORs for Māori and Pacific peoples were higher than the ORs for Asian and European women (0.95 and 0.85 vs 0.68 and 0.79). After adjustment for other factors, the ORs of having endocrine therapy in different ethnic groups were all close to 1. When stratified by ethnicity, the unadjusted and adjusted OR of having HER2-targeted therapy for those with diabetes compared with those without diabetes followed a similar pattern. The unadjusted OR of having chemotherapy was approximately 0.5 for all ethnic groups, except for Pacific women (unadjusted OR 0.42, 95% CI 0.33–0.53). After adjustment for other factors, the OR of receiving chemotherapy for Pacific peoples with diabetes compared with Pacific peoples without diabetes was 0.70 (95% CI 0.51–0.94). Similar results were found in Asian people (adjusted OR 0.74, 95% CI 0.52–1.05).

Discussion

Of those women eligible for endocrine therapy, two-thirds received treatment. Overall, only 60.4% of women with diabetes received endocrine therapy compared with 64.2% without diabetes. However, the difference in the use of endocrine therapy between the two groups can be explained by age, ethnicity and the presence of comorbidities, except for people with stage III breast cancer where diabetes was associated with an increased likelihood of having endocrine therapy after adjustment. The increased use of endocrine therapy for diabetic patients with stage III cancer is consistent with a Dutch study that showed women aged 35–64 years with diabetes were more likely to have endocrine therapy for breast cancer

than women without diabetes.[23] This is probably because of the high-risk nature of their breast cancer, combined with the fact that these women were often deemed unsuitable for adjuvant chemotherapy. Therefore, endocrine therapy may have been considered the most viable option to reduce the risk of cancer recurrence. In summary, while diabetes may have influenced treatment patterns, the clinical circumstances (high-risk cancer) played a more important role in deciding the treatment plan.

After adjustment for relevant factors, patients with diabetes were less likely to receive chemotherapy than patients without diabetes. A 2007 study from the Netherlands[23] also showed that patients with diabetes were less likely to have chemotherapy for breast cancer than patients without diabetes, and the difference was found to be greater in women aged 35–64 years than women aged 65 years or older.[23] The patterning of chemotherapy use by diabetes status for breast cancer is consistent with a systematic review showing that early breast cancer patients with comorbidities receive less chemotherapy than their counterparts without comorbidity.[11] Our dataset lacked information to determine whether the decision to forgo chemotherapy resulted from an active, informed discussion with the patient or if it was a passive choice made without patient involvement. The difference in chemotherapy between the two groups was most pronounced in patients with stage II breast cancer. In contrast, for those with stage III and IV disease after adjustment for other factors we could not show a difference, suggesting that for those with diabetes and a poorer breast cancer prognosis, the benefits of chemotherapy outweigh the harm; therefore, chemotherapy was offered to patients in both groups.

Treatment toxicity is a key reason for the reduced use of chemotherapy for breast cancer for patients with diabetes. A United States (US) based study showed that women with diabetes and breast cancer are at increased risk of chemotherapy-related toxicities compared with patients without diabetes, including higher odds of hospitalisation for toxicity.[24] Reduced treatment benefits of chemotherapy for patients with diabetes is another contributor to reduced use of chemotherapy. This US study also found that patients with and without diabetes who did not receive chemotherapy had similar breast cancer–specific mortality, but patients with diabetes who received chemotherapy had higher breast cancer–specific mortality than patients without diabetes receiving chemotherapy after adjustment for comorbidities and other confounding factors.[24] This is similar to what was found in chemotherapy for colon cancer,[25] where the benefit of chemotherapy in improving survival for colon cancer was greater for patients without diabetes than patients with diabetes.[25] However, patients with comorbidities can still benefit from chemotherapy if offered.[26]

Diabetes is strongly patterned by ethnicity in Aotearoa New Zealand. While the unadjusted results suggested differences in the use of chemotherapy, the adjusted results differed by ethnic groups. These suggested that there were ethnic differences in the likelihood of receiving chemotherapy depending on diabetes status. For example, it looked like the proportions of European women receiving chemotherapy were similar in both groups regardless of diabetes status. However, Pacific women with diabetes were 30% less likely to receive chemotherapy than their counterparts without diabetes. Our previous study found no survival differences between women with and without diabetes based on treatment variations.[27] Differences in survival between the two groups are attributed to age and the tendency for women with diabetes to present with more advanced disease at diagnosis.[12,27] This suggests that the differences in use of chemotherapy in women with diabetes do not have a major impact on survival.

This study found differences in systemic treatments between breast cancer patients with and without diabetes, but our other study[27] demonstrated that these treatment variations did not impact survival outcomes. Our future studies will examine whether breast cancer patients with substantial comorbidities can benefit from systemic treatment. Do the harms of adjuvant systemic therapy outweigh the benefits? Which patients with existing comorbidities are most likely to benefit from systemic anti-cancer treatment? These investigations will provide clearer guidance for optimising treatment strategies for patients with comorbidities.

The strengths of this study include: 1) utilisation of the comprehensive NBCR recording detailed data on patient demographics and tumour characteristics as well as treatment, 2) the use of a national prevalent diabetes database to establish diabetes status, and 3) the linkage of data to the comprehensive PHARMS dataset. This enabled us to examine the association of diabetes with breast cancer treatments. These data were linked to other health data to allow

for adjustment for the impact of comorbidities when estimating the impact of diabetes on breast cancer treatment. This study also has limitations. The VDR dataset does not differentiate between type 1 and type 2 diabetes; therefore, we could not examine the differences between type 1 diabetes and type 2 diabetes on the impact of cancer treatment. This is a retrospective study, and there may still be uncontrolled confounding factors that could impact the results. These confounding variables, which were not accounted for in the analysis, may introduce biases that affect the validity of the findings. Additionally, the treatment patterns have changed over the 16-year period covered by the study, which could further complicate the interpretation of the results.

Conclusions

Most of the difference in probability of women with breast cancer having any form of systemic treatment between those with diabetes and those without diabetes can be explained by age, comorbidity and ethnicity. However, even after adjustment for these factors, women with diabetes are less likely to receive chemotherapy for their breast cancer than women without diabetes, and the difference is greatest in Pacific women. The lower usage of endocrine therapy and HER2-targeted therapy in patients with diabetes could also be explained by the older age at diagnosis and more comorbidities. We are not able to determine from these data whether these differences are active decisions made by the women and their healthcare providers, taking into account the risks of treatment in the context of diabetes and comorbidity. These differences need to be taken into account when considering factors that may impact on overall outcomes for women with breast cancer and diabetes. This is especially true for those ethnic groups who have a high prevalence of diabetes and who have poorer outcomes from breast cancer, such as Māori and Pacific women.

Table 1: Proportion of patients receiving systemic treatment between the diabetes and non-diabetes groups.

Cancer stage	Endocrine therapy (For ER+ or PR+ cancers)			HER2-targeted therapy (For HER2+ cancers)			Chemotherapy (For all cancers)		
	No diabetes	Diabetes	Total	No diabetes	Diabetes	Total	No diabetes	Diabetes	Total
Number of patients eligible for treatment									
I	9,901	1,144	11,045	1,309	89	1,398	11,241	1,259	12,500
II	7,151	1,069	8,220	1,349	181	1,530	8,554	1,253	9,807
III	2,330	332	2,662	650	73	723	2,856	394	3,250
IV	865	172	1,037	298	46	344	1,180	231	1,411
Total	20,247	2,717	22,964	3,606	389	3,995	23,831	3,137	26,968
Number of patients receiving treatments									
I	5,274	573	5,847	739	37	776	1,602	89	1,691
II	5,410	724	6,134	943	100	1,043	3,683	292	3,975
III	1,824	250	2,074	455	44	499	1,768	177	1,945
IV	483	94	577	227	32	259	597	83	680
Total	12,991	1,641	14,632	2,364	213	2,577	7,650	641	8,291
Proportion of patients receiving treatments									
I	53.3%	50.1%	52.9%	56.5%	41.6%	55.5%	14.3%	7.1%	13.5%
II	75.7%	67.7%	74.6%	69.9%	55.2%	68.2%	43.1%	23.3%	40.5%
III	78.3%	75.3%	77.9%	70.0%	60.3%	69.0%	61.9%	44.9%	59.8%
IV	55.8%	54.7%	55.6%	76.2%	69.6%	75.3%	50.6%	35.9%	48.2%
Total	64.2%	60.4%	63.7%	65.6%	54.8%	64.5%	32.1%	20.4%	30.7%

HER2 = human epidermal growth factor receptor 2; ER = estrogen receptor; PR = progesterone receptor.

Table 2: The odds ratio of having endocrine therapy for ER+ or PR+ cancers for patients with diabetes versus patients without diabetes.

Adjusted factors	Stage I	Stage II	Stage III	Stage IV	All stages
Unadjusted	0.88 (0.78–1.00)*	0.68 (0.59–0.78)***	0.85 (0.65–1.11)	0.95 (0.69–1.32)	0.85 (0.78–0.92)***
Period of diagnosis	0.88 (0.78–1.00)*	0.72 (0.62–0.83)***	0.91 (0.69–1.19)	0.92 (0.66–1.28)	0.87 (0.80–0.95)***
Age	0.98 (0.86–1.10)	0.89 (0.76–1.03)	1.05 (0.79–1.40)	0.73 (0.52–1.04)	1.00 (0.92–1.09)
Menopausal status	0.98 (0.86–1.11)	0.89 (0.76–1.04)	1.06 (0.80–1.41)	0.73 (0.52–1.04)	1.00 (0.92–1.09)
Ethnicity	0.94 (0.83–1.07)	0.93 (0.80–1.09)	1.24 (0.92–1.65)	0.70 (0.49–1.00)*	0.99 (0.91–1.08)
Deprivation quintile	0.92 (0.81–1.04)	0.94 (0.80–1.10)	1.25 (0.93–1.68)	0.70 (0.49–1.00)	0.98 (0.89–1.07)
Mode of detection	0.92 (0.81–1.05)	0.93 (0.80–1.09)	1.24 (0.92–1.66)	0.71 (0.50–1.02)	0.97 (0.89–1.07)
Comorbidities	0.99 (0.87–1.13)	1.06 (0.90–1.25)	1.38 (1.02–1.88)*	0.69 (0.47–1.00)*	1.06 (0.97–1.17)
Stage, grade, subtype	0.97 (0.85–1.12)	1.14 (0.95–1.37)	1.66 (1.16–2.36)**	0.67 (0.44–1.01)	1.05 (0.96–1.16)

ER = estrogen receptor; PR = progesterone receptor.
*<0.05
**<0.01
***<0.001

Table 3: The odds ratio of having targeted therapy for HER2+ cancers for patients with diabetes versus patients without diabetes.

Adjusted factors	Stage I	Stage II	Stage III	Stage IV	All stages
Unadjusted	0.55 (0.36–0.85)**	0.53 (0.39–0.73)***	0.65 (0.40–1.07)	0.72 (0.36–1.41)	0.64 (0.51–0.79)***
Period of diagnosis	0.53 (0.34–0.82)**	0.48 (0.35–0.66)***	0.59 (0.35–0.99)*	0.72 (0.36–1.43)	0.60 (0.49–0.75)***
Age	0.70 (0.44–1.10)	0.66 (0.47–0.94)*	0.90 (0.52–1.55)	0.96 (0.47–1.99)	0.84 (0.67–1.06)
Menopausal status	0.68 (0.43–1.08)	0.65 (0.46–0.93)*	0.86 (0.50–1.49)	0.98 (0.47–2.06)	0.83 (0.66–1.05)
Ethnicity	0.70 (0.44–1.12)	0.82 (0.57–1.18)	1.11 (0.63–1.97)	1.23 (0.56–2.67)	0.95 (0.75–1.21)
Deprivation quintile	0.73 (0.46–1.18)	0.86 (0.59–1.19)	1.13 (0.63–2.01)	1.20 (0.55–2.64)	0.98 (0.77–1.24)
Mode of detection	0.74 (0.46–1.18)	0.86 (0.59–1.26)	1.10 (0.62–1.98)	1.21 (0.55–2.67)	0.98 (0.77–1.24)
Comorbidities	0.84 (0.52–1.37)	1.02 (0.68–1.51)	1.61 (0.85–3.06)	2.04 (0.83–5.03)	1.15 (0.89–1.48)
Stage, grade, subtype	0.85 (0.52–1.41)	0.99 (0.66–1.51)	1.56 (0.80–3.07)	1.89 (0.75–4.77)	1.04 (0.80–1.35)

HER2 = human epidermal growth factor receptor 2.
*<0.05
**<0.01
***<0.001

Table 4: The odds ratio of having chemotherapy for patients with diabetes versus patients without diabetes.

Adjusted factors	Stage I	Stage II	Stage III	Stage IV	All stages
Unadjusted	0.46 (0.37–0.57)***	0.40 (0.35–0.46)***	0.50 (0.41–0.62)***	0.55 (0.41–0.73)***	0.54 (0.50–0.59)***
Period of diagnosis	0.46 (0.37–0.57)***	0.41 (0.36–0.47)***	0.51 (0.41–0.63)***	0.55 (0.41–0.73)***	0.55 (0.51–0.61)***
Age	0.66 (0.53–0.83)***	0.68 (0.58–0.79)***	0.83 (0.65–1.05)	0.79 (0.58–1.09)	0.86 (0.78–0.95)**
Menopausal status	0.65 (0.52–0.82)***	0.66 (0.57–0.77)***	0.78 (0.61–1.00)*	0.77 (0.56–1.06)	0.85 (0.77–0.94)**
Ethnicity	0.70 (0.56–0.88)**	0.72 (0.61–0.84)***	0.97 (0.76–1.25)	0.84 (0.60–1.17)	0.87 (0.78–0.96)**
Deprivation quintile	0.71 (0.56–0.89)**	0.72 (0.61–0.85)***	0.99 (0.77–1.28)	0.79 (0.56–1.10)	0.87 (0.78–0.96)**
Mode of detection	0.72 (0.57–0.90)**	0.72 (0.62–0.85)***	0.96 (0.75–1.25)	0.84 (0.60–1.17)	0.87 (0.79–0.96)**
Comorbidities	0.76 (0.60–0.96)*	0.81 (0.69–0.96)*	1.19 (0.91–1.57)	1.12 (0.79–1.60)	0.96 (0.87–1.07)
Stage, grade, subtype	0.83 (0.63–1.11)	0.71 (0.59–0.86)***	1.15 (0.87–1.52)	1.09 (0.76–1.57)	0.85 (0.75–0.97)*

*<0.05
**<0.01
***<0.001

Table 5: The odds ratios of having systemic treatments for patients with diabetes versus patients without diabetes by ethnic group.

Systemic treatment	Odds ratio	Māori	Pacific	Asian	European/Others
Endocrine therapy	Unadjusted	0.95 (0.77–1.17)	0.85 (0.68–1.07)	0.68 (0.53–0.86)**	0.79 (0.71–0.88)***
	Adjusted†	1.10 (0.87–1.42)	1.02 (0.77–1.35)	0.90 (0.67–1.22)	1.04 (0.91–1.18)
HER2-targeted therapy	Unadjusted	0.98 (0.59–1.63)	0.71 (0.43–1.15)	0.58 (0.32–1.05)	0.53 (0.39–0.73)***
	Adjusted†	1.44 (0.74–2.79)	0.95 (0.49–1.85)	0.86 (0.40–1.84)	1.02 (0.69–1.49)
Chemotherapy	Unadjusted	0.52 (0.42–0.65)***	0.42 (0.33–0.53)***	0.47 (0.37–0.61)***	0.51 (0.44–0.58)***
	Adjusted†	0.87 (0.65–1.16)	0.70 (0.51–0.94)*	0.74 (0.52–1.05)	0.90 (0.76–1.08)

HER2 = human epidermal growth factor receptor 2.
*<0.05
**<0.01
***<0.001
†Adjusted for period of diagnosis, age, menopausal status, deprivation quintile, mode of detection, comorbidities, cancer stage, grade and biomarker subtype.

COMPETING INTERESTS
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DATA AVAILABILITY
The data used for this study are not publicly available because of the ethics for patient information. They can be accessed through the National Breast Cancer Register and the Ministry of Health with appropriate ethics approval.

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Ovarian torsion: determining the presenting features and where the delays occur

Karan Bedekar, Anna McInnes, Wendy Burgess

ABSTRACT

BACKGROUND: Delayed ovarian torsion management can lead to unnecessary oophorectomy and halving of a woman's fertility potential.

AIMS: To improve recognition and efficiency of ovarian torsion management at Waitematā by examining common presenting symptoms/signs and identifying areas of treatment delay.

METHODS: An audit of all ovarian torsion cases at Waitematā over 24 months (01/05/2022–30/04/2024).

RESULTS: Forty-five women had ovarian torsion, and 42 were premenopausal. Common presenting features included abdominal pain (100%), ovarian cysts (97.8%), nausea (82%) and vomiting (51%). Peritonism was rare (13.3%). Oophorectomy was performed in 26 cases (57.8%). Rates of oophorectomy increased with increasing time from symptom onset until presentation. The time from presentation until surgery (average of 28 hours) was longer than other tertiary centres, with delays largely arising from triage to ultrasound, and then while awaiting access to theatre.

CONCLUSIONS: Improving torsion recognition and expediting both imaging and surgery should reduce oophorectomy rates. Women presenting with symptomatic ovarian cysts without torsion should be adequately counselled on the risk of future torsion events and encouraged to seek acute medical attention for changes in symptoms. Additionally, more study is needed to determine if relying on computed tomography (CT) findings alone (e.g., presence of ovarian cysts more than 5cm) in the presence of symptoms suggestive of ovarian torsion can reduce time to diagnosis and improve surgical outcomes, compared to the commonly used CT followed by ultrasound.

Ovarian torsion is a gynaecological emergency caused by the twisting of the ovary around its supporting ligaments, causing ischaemia and, ultimately, necrosis.[1,2] Ovarian torsion management is time-critical, as delays are more likely to result in necrosis and a subsequent decrease in fertility. Patients with ovarian torsion are most commonly of reproductive age and present with acute unilateral pelvic pain, nausea, vomiting and ovarian masses greater than 5cm.[3,4] Symptoms of fever and peritonism are less common according to the literature; however, it remains a widespread misconception that ovarian torsion must present with severe signs.[3–5] This can result in misdiagnosis and delays in treatment in those with mild pain or intermittent symptoms.[5] Clinically diagnosing ovarian torsion is challenging, as its common features align similarly with numerous surgical diagnoses like appendicitis, pelvic inflammatory disease (PID), ectopic pregnancies and nephrolithiasis,[6] delaying management. Treatment of ovarian torsion is surgical and includes ovary-sparing procedures, including detorsion, +/- cystectomy or oophorectomy.[1] The choice of procedure is largely influenced by the presumed viability of the ovary determined by direct visualisation.[1]

Waitematā District Health Board (DHB) includes North Shore Hospital (663 beds) and Waitākere Hospital (283 beds). North Shore Hospital is a secondary hospital and the only hospital of the two that provides acute gynaecological services. An audit of complications by the North Shore gynaecology department reports 1–2 cases of delayed management of adnexal torsion monthly, potentially resulting in unnecessary oophorectomy. This study's purpose is to assist in formulating hospital guidelines that enable clinicians to better recognise and manage ovarian torsion cases. The study shall firstly present the most common symptoms, signs and findings seen among ovarian torsion cases. Secondly, it shall examine the time frames in the clinical management of ovarian torsion to identify where delays in management occur, potentially resulting in avoidable oophorectomy.

Methods

A retrospective audit of 2-year electronic records was performed for all patients coded for ovarian torsion between 01/05/2022 and 01/05/2024 at one New Zealand hospital. Eighty-four patients met this criterion. Thirty-nine of these patients were excluded from this analysis as they were incorrectly coded as having ovarian torsion, but rather had isolated fallopian tube torsion, symptomatic ovarian cysts, no evidence of torsion intraoperatively or were incorrectly coded for ovarian torsion. One patient was initially seen in a clinic environment for an already known necrotic ovarian torsion and was treated with elective surgery. As this was not an acute case like the others, it was also excluded. Patients included in the final analyses were given a diagnosis in their discharge summary, confirmed by intraoperative visualisation, leaving a sample population of 45.

The following variables were extracted from the electronic records: age, ethnicity, menopausal status, presenting symptoms, physical examination findings, surgical procedure and the time periods between different time points of clinical management. These time points were symptom onset, initial healthcare presentation (either to their general practitioner [GP] or the emergency department [ED]), initial hospital clinician review, diagnostic imaging, gynaecology diagnosis and finally theatre. These variables were collected from admission notes, discharge summaries, operation notes, radiology reports, clinic letters and clinical notes. These data were approved for use within the scope of this research by The University of Auckland Human Participants Ethics Committee (reference number 021825). The sample population was further subdivided into two groups. The control group were those that

Table 1: Characteristics of the sample population.

	Number of cases (n)	Percentage of sample population	Percentage receiving detorsion +/- cystectomy	Percentage receiving oophorectomy
Sample population size	45	100%	42.2% (19)	57.8% (26)
Age groups				
10–30	21	46.7%	57.1% (12)	42.9% (9)
30–50	20	44.4%	35.0% (7)	65.0% (13)
50+	4	8.9%	0% (0)	100% (4)
Menopausal status				
Premenopausal	42	93.3%	45.2% (19)	54.8% (23)
Postmenopausal	3	6.7%	0% (0)	100% (3)
Ethnicity				
European	22	48.9%	36.4% (8)	63.6% (14)
Asian	11	24.4%	45.5% (5)	54.5% (6)
Māori	6	13.3%	50.0% (3)	50.0% (3)
Pacific	4	8.9%	75.0% (3)	25.0% (1)
Middle Eastern	1	2.2%	0% (0)	100% (1)
Other	1	2.2%	0% (0)	100% (1)

Table 2: Presenting signs and symptoms among the sample population.

	Percentage of total cases	Prevalence within the detorsion +/- cystectomy group	Prevalence within the oophorectomy group
Abdominal pain	100% (45)	100% (19)	100% (26)
Side of abdominal pain			
Right	55.5% (25)	42.1% (8)	65.4% (17)
Left	35.6% (16)	47.4% (9)	26.9% (7)
Nausea	82.2% (37)	78.9% (15)	84.6% (22)
Vomiting	51.1% (23)	47.4% (9)	53.8% (14)
Fever	13.3% (6)	5.3% (1)	19.2% (5)
Presence of ovarian cyst	97.7% (44)	100% (19)	96.2% (25)
Recent diagnosis of symptomatic cysts <1 year	22.2% (10)	15.8% (3)	26.9% (7)
Tenderness on palpation	97.8% (44)	100% (19)	96.2% (25)
Guarding	22.2% (10)	26.3% (5)	19.2% (5)
Percussion/rebound tenderness	26.7% (12)	31.6% (6)	23.1% (6)
Peritonism	13.3% (6)	10.5% (2)	15.4% (4)

underwent laparoscopic or open ovarian detorsion +/- cystectomy, while the case group underwent laparoscopic or open oophorectomy.

Results

Presenting symptoms and signs

During the 24 months, there were 45 cases of ovarian torsion at a New Zealand tertiary hospital. The patient characteristics and the prevalence of the main symptoms reported among the case and control groups are shown in Table 1 and 2. For patients presenting with torsion, the most common procedure performed was an oophorectomy, at 57.8%. This is similar to audits at other hospitals, with oophorectomy rates between 47–64.3%.[7–9] The most common presentation was a premenopausal woman experiencing acute unilateral lower abdominal pain with nausea, vomiting and concurrent ovarian cysts. This is consistent with the standard "textbook" presentation of ovarian torsion.

Right-sided pain was more common than left, likely due to the proximity of the right ovary to the more mobile ileum and caecum as opposed to the relatively fixed sigmoid colon on the left.[6] Women who presented with right-sided pain were more likely to need an oophorectomy. This was likely due to the propensity to assume acute right lower quadrant pain is of surgical cause, such as appendicitis. This is supported by the fact that 44% of women with right lower quadrant pain were initially reviewed by general surgery, delaying gynaecology review.

Another pertinent finding was that 22.2% of the sample population had presented to a hospital or a clinic within a year before their torsion for symptomatic ovarian cysts. These women were much more likely to require oophorectomy. Peritonism with signs of guarding, percussion tenderness and rebound tenderness were rare findings.

Management of ovarian torsion

The clinical journey of ovarian torsion management, as defined in the methods, was consistent across patients. Tracking each patient's progress through a systematic set of time points allowed for easier comparison and identification of delays. Obtaining electronic clinical records ensured accuracy in these time frames. The major source of inaccuracies came from admission notes that subjectively document the time from symptom onset to initial presentation, particularly when symptoms started more than 12 hours before presentation. Generally, if symptoms started fewer than 12 hours before presentation, the exact time of symptom onset was well documented. Beyond 12 hours, the time of symptom onset was rarely accurately documented, and generalisations were used, such as "for 1 day", or "yesterday evening". Thus, if the exact duration was not clearly defined, then this was rounded to the nearest 12 hours, e.g., if the patient had symptoms for 1 day, then the time used was 24 hours.

The stage titled "initial presentation" is significant, as it shows when patients first sought medical attention from symptom onset. For 76.6% of patients, their first presentation was to the ED, while the remainder presented to their GP. Exceptions included cases referred directly to the surgical acute diagnostic unit (ADU) from the GP and thus were reviewed by a general surgical clinician.

Pelvic ultrasound is the preferred diagnostic imaging modality due to its lack of radiation, non-invasiveness and cost-effectiveness compared with computed tomography (CT) or magnetic resonance imaging (MRI).[10] However, ultrasound findings can be variable, and though doppler increases sensitivity, the absence of blood flow is a relatively late sign.[11] Therefore, the presence of arterial perfusion should not rule out torsion.[12] Due to the non-specific presentation, CT scans are often the initial imaging performed. CT findings can be utilised to make a diagnosis, with retrospective studies showing twisted pedicles and ovarian masses are strong positive predictive findings for ovarian torsion.[13] However, 66.6% of patients who initially received a CT scan underwent a further pelvic ultrasound to necessitate diagnosis anyway. Recent radiographic literature identifies a need for better familiarisation of the CT signs of ovarian torsion, such that a diagnosis may be made upon the first scan.[14]

Time frames

The average time from symptom onset to theatre was 72.8 hours. Patients who received an oophorectomy had an average time of 83.6 hours compared with 57.9 hours for those who underwent ovarian detorsion +/- cystectomy, a difference of 25.7 hours. This reflects the time-critical nature of the condition and is consistent with literature showing detorsion and revascularisation is possible up to 72 hours. Interestingly, however, there was no significant difference in time from initial healthcare presentation to theatre between the two groups, with an average time of 28.7 hours

Figure 1: Rates of detorsion +/- cystectomy vs oophorectomy and average time from initial presentation to theatre for different durations of symptom onset.

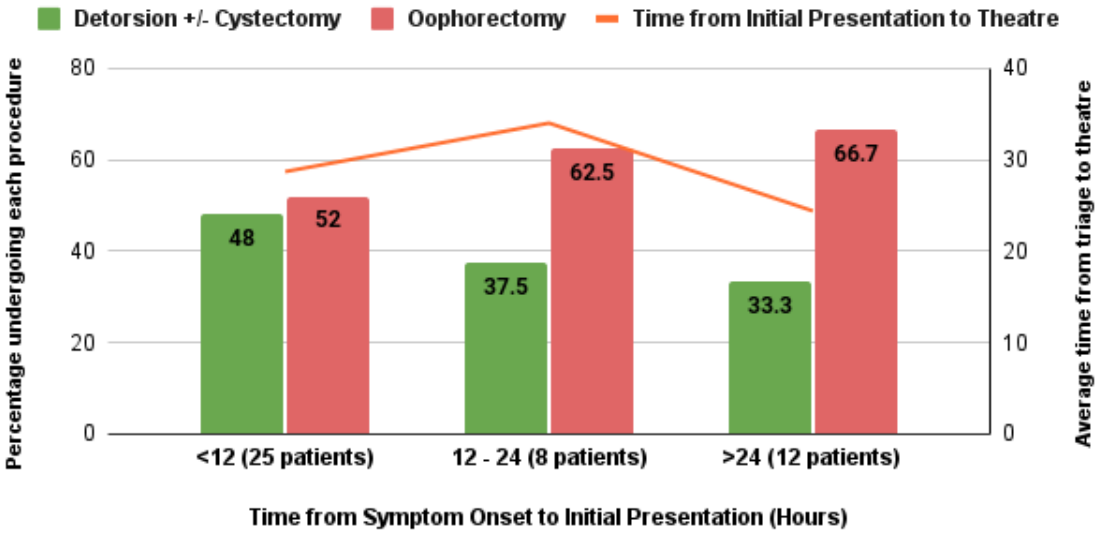
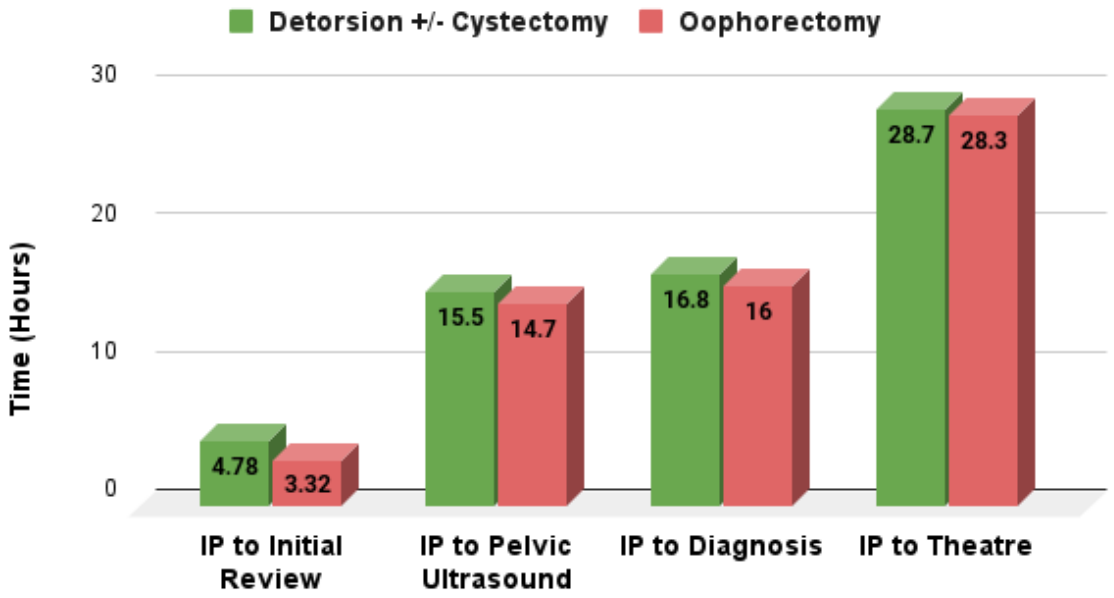


Figure 2: Time from initial presentation (IP) to different clinical stages.



for detorsion and 28.3 hours for oophorectomy. Similar audits in Australia, New Zealand and the United States of America (USA) showed average times from initial presentation to theatre were much lower than the studied hospital, ranging from 6 to 22 hours,[7–9,15] suggesting more delays are occurring irrespective of the procedure. If these global delays were to be decreased, even to a comparable level to these other tertiary centres, perhaps the overall oophorectomy rate would also decrease.

To observe the effect of symptom acuteness on outcomes, the sample population was divided into three groups based on the time from symptom onset to initial presentation: fewer than 12 hours, 12–24 hours and greater than 24 hours. Oophorectomy rates increased as the time from symptom onset to first presentation increased (Figure 1). Interestingly, however, those with symptoms for greater than 24 hours experienced the shortest time from initial presentation to theatre, at 24.4 hours. For those with symptoms for 12–24 hours and fewer than 12 hours, this time was 34 and 28.7 hours respectively. This raises the question of why management was more efficient for those with prolonged symptoms. A total of 22.2% of the sample population had a healthcare presentation for symptomatic ovarian cysts in the year before their torsion. For those presenting beyond 24 hours, the prevalence of recent symptomatic cysts was 58.3%. This suggests that a recent presentation for ovarian cysts is more likely to delay a future presentation for ovarian torsion, when it should in fact prompt the diagnosis of torsion. This delay explains the greater prevalence of oophorectomy among this group.

The final analysis aimed to identify where in the clinical management the greatest delays occurred (Figure 2). The goal was to find areas where improvements would yield substantial reductions in delay to theatre. Figure 2 shows that the greatest delays were from the initial hospital clinician review to pelvic ultrasound and from gynaecology diagnosis to theatre. A similar USA study showed a reduced time from initial triage to initial clinician assessment (1.9 hours), initial review to ultrasound (5.5 hours) and overall time from initial presentation to operating theatre (OT) (22.4 hours).[15]

Discussion

The ideal outcome for women experiencing ovarian torsion is to have a detorsion +/- cystectomy, especially in the case of premenopausal women who have not completed their family, as losing an ovary can significantly affect future fertility. While the results of this audit show that patients who present more acutely (<24 hours of symptoms) are more likely to avoid an oophorectomy, their management in hospital is still delayed, resulting in average oophorectomy rates above 50%.

This delay is evident when compared to data from other tertiary centres. The solution to reduce delays and rates of oophorectomy is three-fold: better identification of ovarian torsion, more efficient treatment and a greater emphasis on trialling ovarian conservation.

Identification of torsion is challenging due to its non-specific presentation. Future guidelines should increase awareness and serve to educate clinicians about the severity of ovarian torsion. Twenty-eight hours from triage to theatre is far too long of a delay to treat a condition where the loss of an organ is involved and thus should take priority over other acute cases. Furthermore, the risk of torsion in women with pre-existing ovarian cysts must be emphasised to all clinicians, as they are at greater risk of developing torsion while more likely to experience delays. Perhaps new clinical guidelines targeted at gynaecology and emergency clinicians can be of benefit. Clinical guidelines should reflect the variability in clinical features to prevent tunnel vision in diagnosis and to dismantle stringent expectations of how torsion should present. The prime example is peritonism, which is a relatively rare finding, and pain of acute onset, which may not always be the case due to intermittent torsion. Another suggestion could be to upskill junior gynaecology trainees to use bedside ultrasound in the diagnostic process, which could identify large ovarian cysts sooner than a formal pelvic ultrasound or CT. A proven example of successful education was from the American Pediatric Surgical Association (APSA), which implemented one such programme that over a 10-year period more than halved the rate of salpingo-oophorectomy and encouraged ovarian conservation procedures.[16]

Alongside improving recognition of torsion, reducing delays to OT and detorsion is critical. Key areas to target are reducing time to pelvic ultrasound and time from gynaecology diagnosis to theatre. Reducing the time to imaging requires multiple considerations. As previously mentioned, most patients receive CT initially, which can add delays to ultrasound. Perhaps one option to nullify this is to develop clear CT diagnostic criteria that can be used to diagnose ovarian torsion, bypassing the need for an additional pelvic ultrasound if there is high clinical suspicion of ovarian torsion. Key features include large ovarian cysts, signs of peri-ovarian inflammation and torted pedicles.[13] The inability of CT to demonstrate blood flow does put it at a disadvantage to ultrasound. However, the absence of arterial flow is usually only a late sign,[11] and there is a lack of consistency among studies on how common this is, with some showing it is absent in a majority of patients while in others only a minority show an absence of ovarian perfusion.[17,18] Further research that looks at patients who were transferred to theatre solely after a CT scan could be insightful in showing the sensitivities of CT findings and to see if this expedites management and improves outcomes. Another suggestion is that the general surgery/ED team could consider ordering a pre-emptive ultrasound alongside referring to gynaecology. This would remove the need for gynaecology to re-see the patient before booking the ultrasound.

Research is now showing returning function of necrotic appearing ovaries, suggesting oophorectomy may not always be necessary. Within the operation notes examined in this study, there were instances where the ovaries disintegrated on contact due to necrosis; however, frequently a decision for oophorectomy was made due to a failure to show signs of revascularisation upon detorsion of necrotic appearing ovaries. However, evidence shows necrotic appearing ovaries that do not appear to immediately revascularise still have the potential to return to function. A study among 12 children who all received detorsion found improved vascularity and follicular development in follow-up sonography, despite 76% of the ovaries showing moderate to severe signs of necrosis on intraoperative visualisation, with some having no return of colour on detorsion.[19] There were also no major complications in these children.[19] There are relatively few studies addressing the preservation of necrotic-appearing ovaries and monitoring for return of function in adult women. Perhaps our ability to judge necrosis on simple visualisation is also flawed, as a 2021 retrospective study found that of 31 ovaries that were visually judged as necrotic, only five of them (16%) had histopathologically confirmed necrosis.[20] Twenty (64.5%) had haemorrhage or venous congestion and six (19%) had normal ovarian tissue.[20] Evidence supports conserving necrotic-appearing ovaries as opposed to removing them.

Conclusion

For patients presenting with ovarian torsion, the most common symptoms were unilateral abdominal pain, nausea and vomiting, as well as the presence of ovarian cysts on imaging, either CT or ultrasound scan (USS). The majority of the patients presenting to a tertiary New Zealand

hospital in this 2-year period underwent an oophorectomy, with delays in management largely arising from initial presentation to diagnostic pelvic ultrasound and diagnosis to theatre. These delays resulted in an average time from initial presentation to theatre that was comparably longer than other tertiary centres in New Zealand, Australia and the USA.[7–9,15]

CT imaging is often the first-line scan for acute abdomen presentations. Thus, more study is needed to determine if relying solely on CT findings, alongside a clinical picture suggestive of ovarian torsion, can reduce time to diagnosis and improve outcomes, compared to the commonly used CT followed by ultrasound.

Although the study is limited in its sample size and would likely benefit from further longitudinal analysis that captures more patients, we recommend the development and implementation of guidelines that improve torsion recognition and expedite both imaging and surgery to reduce oophorectomy rates. Furthermore, women presenting with symptomatic ovarian cysts should be counselled on the risk of future torsion and advised to seek urgent acute medical attention for changes in symptoms to reduce the risk of oophorectomy.

COMPETING INTERESTS
Nil.

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Drug driving, sedation, reaction time and blood levels: a prescriber's approach to the Land Transport (Drug Driving) Amendment Act 2022

Marleen van Oeveren, Paul Glue, Charlotte Mentzel

ABSTRACT

AIM: To update prescribers about the revised *Land Transport (Drug Driving) Amendment Act 2022* (*LTAA*) and implications for prescribing.

METHODS: We reviewed the legislation of the *LTAA* and the specific drugs identified in it, mainly benzodiazepines and opioids. We also briefly reviewed published evidence on the impact of benzodiazepines and opioids on driving.

RESULTS: Both opioids and benzodiazepines are associated with increased accidents in observational (non-controlled) studies, though the odds ratios are small (at most 1.65 for benzodiazepines and around 2.0 for opioids), and accident events are rare. Prescribers are not advised to do blood levels or bedside testing to assess driving fitness. They should consult a peer group or mental health pharmacist when advising patients taking multiple psychoactive medications if they are safe to drive.

CONCLUSION: There are no current jurisprudence or guidelines for prescribers on how to interact with the *LTAA*. Nor is it clear if or how investigations or complaints relating to the *LTAA* would be handled by the Health and Disability Commissioner (HDC), the Medical Council of New Zealand and/or the Coroner. Until more detailed instructions are published, this article should provide some guidance for professionals who prescribe benzodiazepines or opioids.

On 11 March 2023 the *Land Transport (Drug Driving) Amendment Act 2022* (*LTAA*) came into effect.[1] This amendment introduced changes to the *Land Transport Act 1998* and is part of the *Road to Zero* strategy, a plan from Waka Kotahi, the New Zealand transport agency, to reduce the number of deaths on roads in Aotearoa.[2] Data from the New Zealand Police indicate in 2021 there were 93 people killed in motor vehicle accidents where a driver was found to have drugs in their system—nearly a third of all fatalities that year.[3] It can be surmised that the *Act* was in large part revised in order to address drug driving, in which those intended to be captured under the *Act* were using non-prescribed or illicit drugs. However, the broadening of scope to capture prescribed medications does not appear to have been thought through by legislators. No prescribers were consulted in the development of this legislation, resulting in the implications for prescribers currently being poorly defined. It is also important to view the risks in a larger context, particularly as the odds of causing an accident on only a benzodiazepine or an opioid is similar to the risk of driving a black car (univariate odds 1.2, multivariate odds 2.0).[4]

While it has long been illegal to drive impaired, the new *Act* identifies 25 drugs (21 that can be reasonably prescribed), defined in the *Act* as Schedule 5 drugs, and the blood levels of these drugs that are thought to impair driving (Table 1). Drivers that are found to be impaired can be charged. The *Act* also has additional penalties when drivers are found to have taken Schedule 5 drugs with alcohol or have multiple Schedule 5 drugs in their system.[1]

As part of the amended *Act*, drivers will have the option of a "medical defence" if they are found to have blood levels of Schedule 5 drugs above the pre-defined blood levels. This is defined as *"... a way for you to dispute your drug driving infringement by providing evidence that you have taken your prescription medication in accordance with your current prescription, and any instructions from a health practitioner or manufacturer."*[5] Patients will have to provide both a copy of the current and valid prescription for identified drug(s) and a copy of the label from the container in which the qualifying drug(s) were dispensed in, alongside any other relevant information they wish to have considered.

This shift in legislation requires patients to

Table 1: *LTAA* prescribable Schedule 5 drugs and prohibited blood concentrations.

Drug	NZ threshold concentration (ng/mL)	NZ criminal concentration (ng/mL)
Benzodiazepines		
Alprazolam*	20	50
Clonazepam	20	50
Diazepam	100	200
Lorazepam	10	30
Midazolam	10	30
Nitrazepam*	20	50
Oxazepam*	200	800
Temazepam	200	800
Triazolam*	4	4
Zopiclone	20	50
Opioids		
Buprenorphine	1	1
Codeine	50	200
Dihydrocodeine	50	200
Fentanyl	0.5	0.5
Methadone	50	200
Morphine	10	20
Oxycodone	20	50
Tramadol	100	250
Others		
Amphetamine	20	100
Ketamine	10	50
THC	1	3

*Not available in Aotearoa.
LTAA = Land Transport (Drug Driving) Amendment Act 2022; NZ = New Zealand; THC = tetrahydrocannabinol.

provide proof they have taken their Schedule 5 medications as prescribed. Therefore, prescribers have a duty to provide evidence-based advice on the possible effect of medication on driving, as well as any potential interactions/effects if prescribed medication is taken with other drugs (including over-the-counter medications) and/or alcohol. Should patients cause a motor vehicle accident while under the influence of medication, they will be exempt from prosecution under the *LTAA*, irrespective of the tested blood levels, if they can provide evidence that they have taken the medicine as prescribed. Practitioners cannot be charged under the same *Act* but could become subject to investigation and/or face liability by the Health and Disability Commissioner (HDC), the Medical Council of New Zealand (Medical Council) and/or the Coroner (in the event of a fatality).

As yet, there have been no cases in which this has happened. Nor has there been any guidance from governing bodies on how prescribers should incorporate the *LTAA* mandates into their practice. Thus, general good prescribing rules apply. The Medical Council describes good prescribing as being *"… in accordance with accepted practice and any relevant best practice guidelines"* in their 2020 statement on prescribing practice.[6] Therefore, for practitioners to avoid being found liable by the above regulating bodies, they would have to know what accepted practice is. Currently, to the authors' knowledge, there do not appear to be best practice guidelines for the prescribing and use of specific drugs or drug categories in relation to driving, other than Medsafe data sheets and the *LTAA*.

This article aims to clarify the available evidence for the effect of the prescribable Schedule 5 drugs on driving and provide guidance for prescribers on how to prescribe these medicines in context of the updated *LTAA*. This article specifically covers benzodiazepines, benzodiazepine-like sedatives (i.e., "z-drugs") and opioids, as these are the Schedule 5 medicines commonly prescribed in Aotearoa.

Effects of benzodiazepines on driving

Benzodiazepines and benzodiazepine-like medications (hereafter referred to as just benzodiazepines) are extensively used internationally. In Aotearoa zopiclone was the 14th most frequently dispensed medicine in 2020. The most commonly dispensed benzodiazepines in 2020 were lorazepam and diazepam. Benzodiazepine use is prevalent in older adults, with approximately 10% of people aged 75 years and older regularly dispensed these medicines. As Papoutsis et al.[7] pointed out, due to the high frequency of benzodiazepine use within the population, a zero-tolerance policy seems impractical despite the lack of experimental data indicating a safe cutoff for driving.

Benzodiazepines, like alcohol, work by modulating the effects of gamma-aminobutyric acid (GABA) by binding to the $GABA_A$ receptor. By increasing GABA activity, benzodiazepines and alcohol have sedative, anxiolytic and anti-epileptic effects, and impact cognitive functioning. In this way, benzodiazepines and alcohol share similar pharmacodynamic properties, and it might therefore be useful for practitioners to conceptualise the effect of benzodiazepines as like the effects of alcohol on driving.

Several small (around 10 to 20 participants per arm) randomised controlled trials have shown that acutely, benzodiazepines have a significant negative impact on performance on various psychometric assessments.[7–11] These psychometric assessments are designed to identify individual brain functions affected, some of which are relevant to driving a vehicle such as psychomotor reactions or processing speed.[7–9,12] There are also many observational studies that have linked benzodiazepine use to crash risk in real-life settings both at therapeutic and supratherapeutic levels.[7,13,14] However, a meta-analysis that considered both prescribed and illicit benzodiazepine use was able to factor out multidrug use. For those using only benzodiazepines it showed a significant publication bias; when this was corrected for the odds ratio for involvement in traffic accidents, this was reduced from 1.65 (self-reported use risk) to 1.17, a much more modest risk.[14]

Studies regarding the effect of long-term (stable) benzodiazepine use on driving is scant. Van der Sluizen et al.[11] examined the effects of regular benzodiazepine use on both driving and neurocognitive tests outcomes in 100 volunteers and found driving test results were indistinguishable from controls after 3 years of consecutive use, but not before. Cognitive tests did, however, remain abnormal after driving tests normalised.[11] The subjective sedative effects of benzodiazepines often wear off well before the 1–3-year mark, thus indicating that a lack of subjective drowsiness is not a good indicator of safety for driving.

Effect of opioids on driving

Opioid pain relief is indicated for acute pain associated with injury or surgery, or pain secondary to malignancy. Use of opioids for chronic non-cancer pain is discouraged in most international pain management guidelines due to lack of benefit relative to the associated risks of long-term opioid use. However, there will be exceptions to this rule for various reasons, including opioid substitution therapy. The rate of opioid prescribing in Aotearoa has been relatively stable since 2013, with a slight downwards trend observed year-on-year, although an increased rate was seen in 2020–2021, which may reflect delayed surgeries during the COVID-19 pandemic lockdowns. Rates of opioid prescribing are higher for weak opioids than strong opioids; for example, in 2020 the rate of ≥1 codeine dispensing was 60 per 1,000 population, and the rate for morphine dispensing was 9 per 1,000 population. Overall, there is a general trend of higher opioid use in older adults, who may be particularly susceptible to adverse effects due to pharmacokinetic changes, polypharmacy and comorbidities, and in the context of driving typically have slower reaction times.[15]

Opioid medications bind to endogenous opioid receptors in the brain and spinal cord, blocking pain pathways and thus altering the perception of pain. In addition to a pain-relieving effect, agonism of the opioid receptor is also linked to the regulation of processing, mood, motivation, learning and memory and gastrointestinal function.[16] Side effects of exogenous opioids include sedation, constipation, nausea, euphoria with acute use and depressed mood with more chronic use, respiratory depression and physical dependence.[17]

To what extent opioids affect a person's ability to safely drive a vehicle was assessed in a recent review by Cameron-Burr et al.[17] There was significant heterogeneity in the results, likely secondary to heterogeneity in study design, small sample sizes, publication bias and whether the opioid was prescribed or illicitly obtained. Overall, the majority (69%) of included experimental studies (i.e., prescribed rather than illicitly obtained opioids) found that opioids impair psychomotor function. There was evidence of a dose–response relationship between opioid therapy and impairment. While the authors of the review study did not do any statistical analysis, the summary of results suggests in many of the included studies there was approximately a twofold increased risk of impairment or motor vehicle accident compared to non-opioid using controls. This is consistent with the older meta-analysis from Elvik et al., who put the odds ratio of a fatal accident at 2.30, and 1.94 for accidents causing injury.[14] Unfortunately, it was not possible to distinguish between prescribed and illicit use in the odds ratio because of a lack of data. However, data were for people who were identified to have had only opioids, which will have reduced the interference from the more extreme illicit use.

The impact of chronic use of opioids on driving is not well understood, and studies in this area have been small. Overall, the available results suggest that chronic use of prescription opioids does not impair driving; however, the tight inclusion criteria for these studies and small sample sizes make it difficult to generalise these results to the general population.[17]

Practical questions

Question 1: Can and should a practitioner assess for sedation and reaction time in a medical appointment?

In Aotearoa it is already common for general practitioners (GPs) or secondary care providers to make decisions around driving capacity for those with dementia or minimal cognitive impairment. It would be sensible to assume similar practice in driving capacity that may be affected by medications. The 2014 *Dementia and Driving Safety* clinical guideline[18] states "*Clinical bedside testing is a poor guide to deciding on a person's driving safety*", and advises when there is doubt a patient should undertake an occupational therapist (OT) driving assessment through one of the local agencies. This advice is equally valid for those with impairment secondary to medication. If a clinician is uncertain about whether or not driving is impaired, such a test would be useful. The authors are aware some opioid substitution clinics have had their patients assessed with the aid of a Work and Income disability grant.

Question 2: Should practitioners be taking benzodiazepine or opioid blood levels to determine whether a patient should drive?

Measuring blood concentrations of Schedule 5 drugs to try to quantify risk is impractical, as most of the drugs listed in Schedule 5 do not have

validated assays at community laboratories. As drug concentrations change over a dosing interval based on a drug's pharmacokinetic characteristics, a single sample taken at a random time point is of little value in advising patients about suitability of driving, even when the question of active metabolites is not considered. Drug interactions, whether pharmacokinetic (e.g., inhibiting the clearance of a Schedule 5 drug) or pharmacodynamic (e.g., combining another sedative drug with a Schedule 5 drug) further complicate advice to patients. Drug concentrations for people who have yet to reach a steady state are particularly unhelpful in predicting future drug levels, thus making testing unhelpful in those who use *pro re nata* (*PRN*) doses.

Question 3: What should prescribers advise regarding interactions between two or more Schedule 5 drugs, or between Schedule 5 drugs and unlisted qualifying drugs?

The *LTAA* specifies an NZ$4,500 fine or up to 3 months imprisonment for driving under the influence of a single Schedule 5 drug, and NZ$6,000 or 6 months imprisonment for driving with two Schedule 5 drugs or combining alcohol with a Schedule 5 drug. It does not specify how many Schedule 5 drugs can be given before a medical defence can no longer be provided. In many cases the prescriber would not only need to think about the cumulative sedative effect of the prescribed medications, but also other pharmacokinetic and pharmacodynamic interactions.

Some medications can affect the blood levels of opioids and benzodiazepines by affecting their metabolism and clearance. For example, fluoxetine inhibits the conversion of codeine to morphine—meaning the plasma level of codeine may be elevated and the elimination half-life prolonged. Other substances, e.g., gabapentinoids, not only have effects on alertness and cognition, but have also been shown to reverse the tolerance to opioids when initiated in someone taking opioid medication.

Given the complexity of these factors alongside individual variation in drug response and metabolism, it is not possible to predict how use of two or more interacting substances will affect an individual. As such, it may be best to advise patients taking two or more medications that may increase the risk of impaired driving that they should not drive while taking these medicines.

Question 4: How long should patients not drive after taking *PRN* benzodiazepines or opioids?

Unfortunately, there is no published evidence that has looked at how many hours after taking a Schedule 5 medication effects on driving impairment are no longer present. Based on known medication principles it is likely that certain factors such as age and dose affect the duration of impairment. However, there have been no specific research studies into this.

Based on the paucity of specific evidence regarding this question, the most logical approach may be to use drugs' half-lives to determine how long a person should not drive after taking medication. The debate on how many drug half-lives to use as a cutoff has no direct evidence to make a decision. The art of medicine is interpreting the available information and then applying it to a practical situation with a patient rather than waiting for such research to be published. It can be assumed that after 4–5 half-lives all medication will be cleared from the body. However, given how commonly Schedule 5 medications are prescribed in Aotearoa, using 4 half-lives as a rule would be very disruptive for many patients. In an attempt to balance clinical practicalities and pharmacokinetic principles, the authors suggest a more reasonable approach may be to use 2 half-lives (75% of medication cleared) as a general rule. However, for patients with renal or liver impairment, those who take doses higher than those recommended by Medsafe, the elderly, people using multiple medications, people who still feel sedated, people who are using *PRN* medication more than incidentally (i.e., more than 2–3 times a week) or for any other reason identified by the prescriber, it would be wise to recommend waiting 4 half-lives after taking the medicine before driving.

Practical recommendations for prescribers

1. Patients prescribed Schedule 5 medications should be advised to avoid any alcohol if driving, both due to possible additive effects and interactions and because using non-prescribed substances while driving might make their medical defence invalid.
2. Currently there is no recommendation for medical practitioners to do "bedside" reaction time testing or any other driving suitability tests. Patients could be referred

for driving assessment.
3. There is little to no value in ordering blood levels of Schedule 5 medicines in a clinical setting.
4. It is useful that any instructions re driving are both put in the patient notes and on the prescription so the pharmacist can reiterate the advice to the patient.
5. If patients do feel sedated or tired, they should, of course, be advised to avoid driving. The general statement that it is safe to resume driving once a patient no longer feels sedated is common among medical practitioners and is taught during medical training. However, there is no empirical evidence to substantiate this for Schedule 5 medications, and there is some evidence that driving impairment on medication lasts longer than the subjective feeling of sedation.
6. For patients on stable doses of one Schedule 5 medication with no psychoactive polypharmacy, it would be good practice to inform the patients of the new *LTAA* rules. The authors would argue that a reasonable practitioner would not tell these patients that they could not drive.
7. For patients on multiple Schedule 5 drugs or one Schedule 5 drug and other psychoactive substances likely to qualify as unlisted qualifying drugs, and whose polypharmacy cannot be limited, it would be wise to get advice from a (mental health) pharmacist or a peer group around suitability to drive and document this carefully in the patients' notes. In these cases, a driving assessment might be warranted, or it may be necessary to advise the patient that they should not be driving, and document as such.
8. For short-term or *PRN* prescriptions, it is likely most appropriate the patient is counselled not to drive until 2 half-lives have passed in simple cases and 4 half-lives in the exceptions mentioned in point 4, as no habituation to the impairing effects is likely to occur.
9. The authors suggest that until other guidelines have been developed, this article provides an outline of what a reasonable prescriber might do. If adhered to, this advice should provide a defensible position should a prescriber become the subject of an investigation or complaint related to the *LTAA*.

Discussion

The aim of this article is to aid prescribers in familiarising themselves with the updated *LTAA* and the implications for prescribing benzodiazepines and opioids. It also aims to identify what a "reasonable prescriber" might do when prescribing Schedule 5 medication. Currently, Medsafe data sheets do not have sufficient detail to support prescribers in offering advice to drivers about the potential for impairment, or when they should or should not drive. The common advice of not to drive if one feels drowsy is of little practical use, nor does it appear to be supported by research.

Individual clinicians will have to determine their own risk appetite when it comes to prescribing and advising patients on these medicines. Cautious prescribers who take a conservative approach to prescribing may be reluctant to offer medical exemptions to any patient taking opioids or benzodiazepines, especially given the current lack of clarity on how the HDC complaints committee, the Medical Council of New Zealand and coronial bodies will respond to incidents where someone with a medical exception causes a traffic accident. This lack of clarity is likely to cause high regional variability in approaches and thus negatively impact patients' access to either the ability to drive or their ability to access required medical care, despite this being explicitly stated as something the new *LTAA* was intending to avoid.[5] The recommendation for OT driving assessments may induce issues with equity relating to the inability to pay or physical access to testing and resources and potential for wait lists. It is recommended that improved tests or guidelines are developed to support practitioners (though the authors are aware of instances where Work and Income has supported people on opioid substitution to financially access an OT driving assessment).

For practical purposes, this article groups the Schedule 5 medications into three categories: benzodiazepines, opioids and others (THC amphetamine and ketamine). The latter are less frequently prescribed and therefore not covered in this article. It is notable that several other medications that may affect driving are sometimes used as sleep aids; anxiolytics or pain medication are not mentioned in the legislation. Examples of these include sedating antihistamines (e.g., promethazine), sedating antipsychotics (e.g., quetiapine and olanzapine) and gabapentinoid drugs (gabapentin and pregabalin). The *LTAA* makes reference to "*unlisted qualifying drugs*",

defined as "*… a qualifying drug not listed in Schedule 5*" and assigns similar penalties for both Schedule 5 substances or unlisted qualifying drugs.[1] However, there are no plasma concentrations provided for unlisted qualifying drugs. It is possible that there will be an increase in the use of these medications listed above or other unlisted qualifying drugs over Schedule 5 medications due to a (mis)understanding that drugs not on the Schedule 5 list will not result in legislative consequences for drivers. How the use of these drugs will be interpreted by judges and police is currently unclear. It is also possible that prescribers, in the light of this article, start viewing *PRN* prescribing as more risky than regular prescribing, which would be against guideline recommendations and might negatively affect patients.

It appears clear to the authors that prescribers cannot be held responsible for patients who continue to drive when the prescriber documents they are not supporting a medical exemption. However, how the HDC, Medical Council of New Zealand and coronial bodies will interpret prescribers giving medical exemptions to patients who they know, or could reasonably suspect, use recreational substances including alcohol also remains unclear, especially if these patients cause an accident on a combination of prescribed and recreational substances. It would be helpful if these regulatory bodies provided a position statement addressing these issues to help clarify prescriber expectations and responsibilities when prescribing these medicines.

How the legislation will be applied to specific situations remains unknown and will likely become clear over time, especially as the Government is in the process of developing the legislation to allow for roadside saliva testing. Guidance for clinicians and patients, including specific patient information leaflets that include application of the *LTAA* legislation in practice, would be useful, and would aid in the safe use of these medicines.

COMPETING INTERESTS
CM has received James Hume bequest, Lottery Health Research Grant.
PG is named on a patent for an extended-release ketamine tablet, developed with Douglas Pharmaceuticals.

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Appendix

Appendix Table 1: Drug half-lives of commonly used Schedule 5 medication. Medication with an asterisk likely safe for driving the next day.

Benzodiazepines	1 half-life	2 half-life threshold	4 half-life threshold	Opioids	1 half-life	2 half-life threshold	4 half-life threshold
Alprazolam	12–15 hours (16 in old age)	1.5 days	2.5 days	Buprenorphine	38 hours	3 days	6 days
Clonazepam	30–40 hours	3 days	6.7 days	Codeine*	3–4 hours	8 hours	16 hours
Diazepam	Metabolites up to 100 hours	8–9 days	16.7 days	Dihydrocodeine*	4 hours	8 hours	16 hours
Lorazepam	12–16 hours	24 hours	2.5 days	Fentanyl	Depending on tablet between 7–22 hours	14 hours to 2 days	3.5 days
Midazolam	1.5–2.5 hours (oral)	4 hours	8 hours	Methadone	19–55 hours	Variable	9 days
Nitrazepam	30 hours (40 in old age)	2.5 days	5 days	Morphine*	2–3 hours	6 hours	12 hours
Oxazepam	4–15 hours	20 hours	1.5 days	Oxycodone	Delayed release 4–8 hours	16 hours	1.3 days
Temazepam	7–11 hours	14–22 hours	1.7 days				
Triazolam*	2–5 hours	10 hours	20 hours	Tramadol*	6 hours	12 hours	24 hours
Zopiclone*	5 hours (7 in old age)	10 hours	20 hours				

Cryogenic burns to the upper aerodigestive tract following recreational nitrous oxide inhalation

Matt McCall, Hayleigh Miller, Samuel JM Hale, Rebecca Field

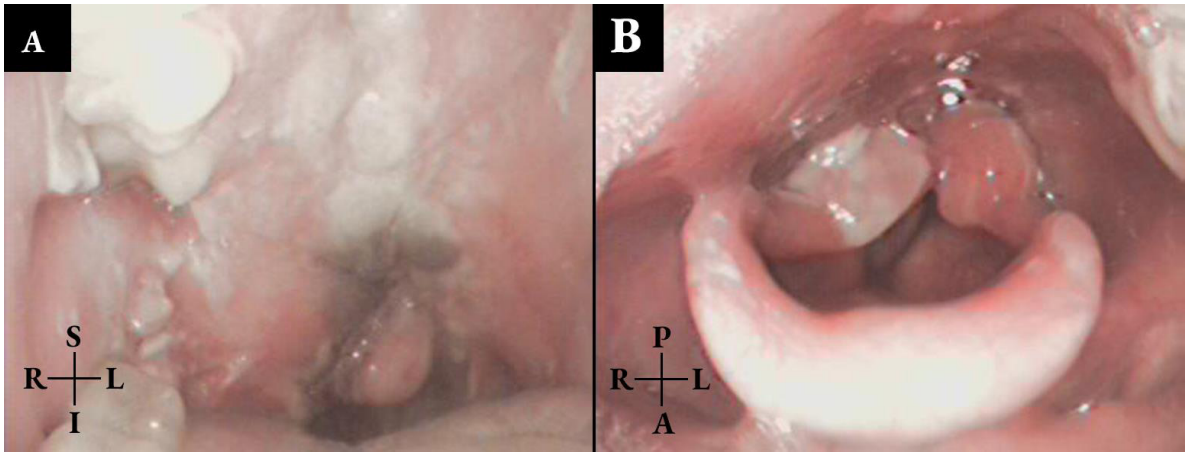
Nitrous oxide (N_2O) is a tasteless, colourless gas used commonly as a short-acting inhalational anaesthetic. It is also publicly available for use in food preparation. N_2O is inhaled recreationally, most often from pressurised canisters for cream whippers (commonly referred to as "nangs"), giving brief euphoria or dissociation.[1,2] The gas rapidly depressurises when it is expelled from the canister, causing its temperature to drop to as low as −55 degrees Celsius in accordance with Boyle's law.[3,4] Balloons are often filled with N_2O for inhalation, though some users inhale directly from the canister.[1] Recreational N_2O use is becoming increasingly common.[1,2] While short-term adverse effects, including transient cognitive impairment and hypoxia are uncommon, inhalation of rapidly depressurising N_2O can cause cryogenic airway burns with resulting oedema and airway compromise.[1,5–8]

Case report

A 30-year-old female presented to the Christchurch Hospital Emergency Department 8 hours after inhaling N_2O directly from a canister, with immediate pain followed by dysphonia and a sensation of throat swelling. She had also used cocaine. Oropharyngeal and flexible nasendoscopic examination showed oedema of the uvula, right soft palate, right aryepiglottic fold and both arytenoids, with a patent airway.

She was admitted to the otolaryngology ward and commenced on intravenous dexamethasone 8mg twice daily. The mucosa in the region of injury developed clearly demarcated necrotic areas over the following 48 hours, with improvement in oedema (Figure 1). The area of necrosis extended onto the right side of the hard palate, beyond the zone of injury identified on hospital admission. Her dysphonia resolved and she was discharged with analgesia when she could tolerate oral intake. Improvement in the appearance of the mucosa was observed at a planned review in the outpatient clinic 48 hours after discharge, with only mild palatal oedema and slough over the previously necrotic areas.

Figure 1: Endoscopic photos of the oral cavity and oropharynx (A) and larynx (B). Mucosal pallor indicating superficial necrosis affecting the palate, uvula and right arytenoid was observed on day 2 post-injury.



R = right; L = left; S = superior; I = inferior; P = posterior; A = anterior.

Discussion

Recreational N_2O use is common, but only four previous reports of associated cryogenic injuries were identified in the literature. Such injuries may therefore be under-reported. In the majority of cases, burns were caused by direct inhalation of pressurised gas. A further case report describes burns sustained from non-intentional inhalation from an exploding anaesthetic cylinder. Three of these five patients required intubation, with one subsequently requiring tracheostomy.[4–8]

N_2O is stored at approximately 30psi in whipped cream canisters and between 900 and 1,000psi in medical or automotive cylinders. While higher pressures are associated with more severe injuries, inhalation from lower-pressure canisters may still lead to clinically significant cryogenic burns.[6,7] Burns to the skin are more commonly reported, usually of the inner thigh or hands caused by direct contact with the canister.[9]

Neurological complications of N_2O abuse are more widely recognised. A recent local case series described 12 patients with N_2O-associated myelopathy over a 7-year period, with a median consumption of 100 canisters per day. Three patients required ongoing assistance for activities of daily living at 1–3 months following hospital admission.[2] While N_2O is generally considered safe in limited doses, a significant minority of recreational users experience serious harm.[1–8]

Until September 2024, the sale of non-medical N_2O canisters in Aotearoa New Zealand was unrestricted, but the New Zealand Government recently advised that the *Psychoactive Substances Act 2013* would now apply to its sale for recreational inhalation. Those found to sell, offer to sell or possess to sell N_2O are now liable on conviction to imprisonment or large fines.[10] This clarification of how legislation would be applied was intended to reduce the potential harms associated with N_2O abuse, despite clear evidence that prohibition's harms to individuals and society are greater than its benefits.[11–13] Interventions focussed on harm minimisation, such as decriminalising the sale and recreational use of N_2O, regulating its distribution, educating users on how to handle N_2O safely and distribution of delivery adjuncts like balloons are strategies that may reduce the harms of N_2O use more effectively than prohibition.[1,12,13]

Conclusion

Cryogenic injuries to the upper aerodigestive tract are a potentially life-threatening but preventable complication of recreational N_2O inhalation. Improved awareness of such injuries may inform further legislative changes and public health interventions to minimise potential harms.

Table 1: Summary of cases reported in the literature of nitrous oxide–related airway burns.

Report	Source of N_2O	Recreational use	Additional substance use	Location of burn	Method of inhalation	Airway management
Rowson et al. 2023	Whipped cream canister	Yes	Amphetamines	Oropharynx	Direct	IV dexamethasone, IV antibiotics
Bagerman et al. 2020	Not stated	Yes	None	Arytenoids, vocal cords, arm	Indirect via balloon	Intubation (not stated if AFOI), IV dexamethasone
Chan et al. 2018	Automotive canister	Yes	LSD	Oral cavity, nasopharynx, oropharynx, supraglottis, mid-face, hand	Direct	AFOI, IV dexamethasone

Table 1 (continued): Summary of cases reported in the literature of nitrous oxide–related airway burns.

Report	Source of N_2O	Recreational use	Additional substance use	Location of burn	Method of inhalation	Airway management
Svartling et al. 1996	Anaesthetic cylinder	No	None	Pharynx, supraglottis, lower face	Direct	AFOI, tracheostomy, IV methylprednisolone, IV antibiotics
Rowbottom 1988	Anaesthetic cylinder	Yes	None	Lips, oral cavity, tongue, palate	Direct	Unknown

N_2O = nitrous oxide; IV = intravenous; AFOI = awake fibreoptic intubation; LSD = lysergic acid diethylamide.

COMPETING INTERESTS
Nil.

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Notes from a small island: Ireland passes Tobacco 21 (T21) legislation

Frank Houghton, John Lombard

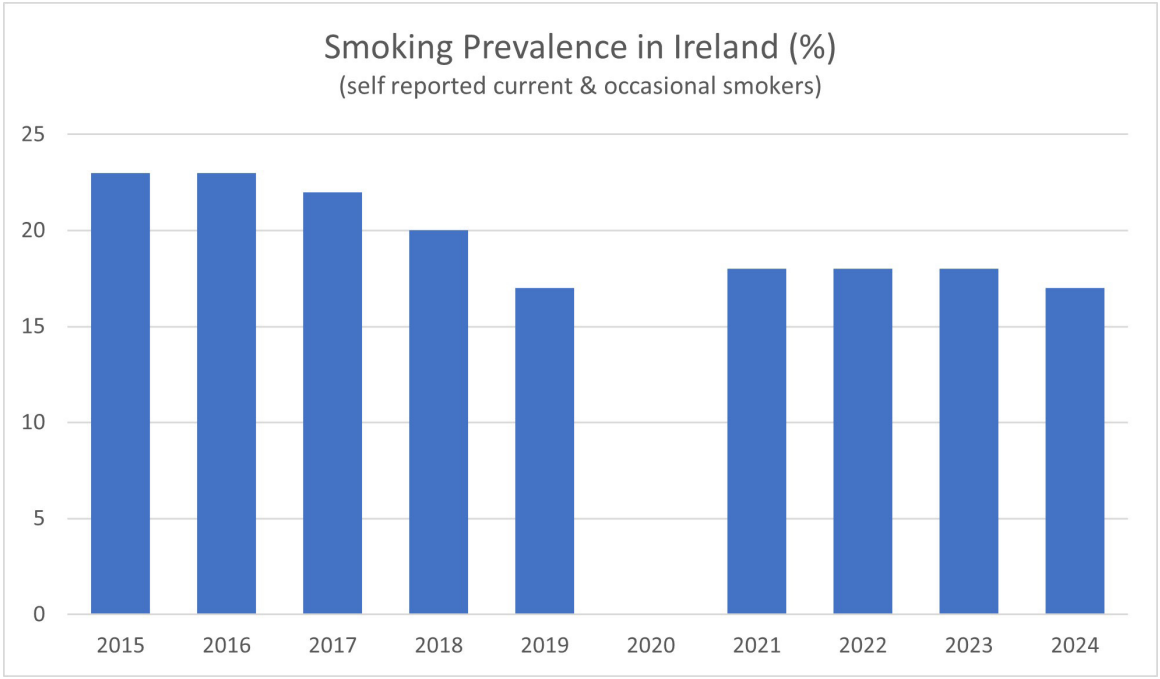
Tobacco-related disease continues to be a major burden of disease globally.[1] Smoking is particularly prevalent in low socio-economic and minority ethnic groups and is a significant factor in health inequalities.[2,3] Ireland set a target in 2013 of becoming *Smokefree*, which is having a smoking prevalence of <5%, by 2025.[4] However, as shown in Figure 1, Ireland has had a relatively stable smoking rate in recent years and has not achieved this target by a considerable margin.[5,6]

Critics have suggested that the World Health Organization's (WHO's) MPOWER strategy (monitoring tobacco use; protecting people from tobacco smoke; offering help to people quitting tobacco; warning about the dangers of tobacco; enforcing tobacco advertising, promotion and sponsorship bans; raising taxes on tobacco) is not enough to achieve *Smokefree* status in Ireland.[5] Recent projections suggest that Ireland might at best have been on a trajectory to meet its *Smokefree* target by 2037,[8] although even this projection may be overly optimistic.[9]

However, in a bold move that once again has Ireland "punching above its weight" in tobacco control,[10] Ireland has just passed the *Public Health (Tobacco) (Amendment) Act 2024*. Section 11(3) of the *Act* provides that the *Act* will come into operation on 1 February 2028.[11] From then on, the minimum purchase age for tobacco products in Ireland will be 21. Ireland is the only country within the European Union to have taken such a step, although Latvia has enacted legislation to raise the minimum age of sale to 20 in 2025. On the global stage, Ireland now follows in the footsteps of countries such as Ethiopia, Honduras, Kazakhstan, Mongolia, the Philippines, Singapore and the United States (US) in raising the minimum purchase age for tobacco to 21.[12,13]

Unlike the US, it must be acknowledged that the Irish legislation does not include vapes.[13] However, moving the minimum tobacco purchasing

Figure 1: Smoking prevalence in Ireland 2015–2024 among those aged 15+.[7,8]



age to 21 is a significant step forwards. The benefits of such a move are manifold. Such policies have been proven to reduce smoking rates elsewhere in the 18–20 age group.[14] This is particularly important, as few people start smoking after their teenage years.[14] In addition, as most underage smokers access their cigarettes via informal social avenues, evidence has found that such routes are also impacted. Evidence suggests, therefore, that such laws also reduce smoking prevalence among 16- and 17-year-olds.[15]

Delays in smoking initiation may be crucial, as it has been argued that adolescent brains are highly susceptible to rapid nicotine addiction.[16] The US Department of Health and Human Services suggests that early initial onset of smoking leads to addictive behaviours, making it extremely challenging for an individual to quit and, hence, is more likely to result in heavy tobacco use as an adult.[17] Concerns have also been raised about the possible long-term cognitive impacts of nicotine exposure during adolescent brain development.[18] Further protections for children and young persons are required, given Big Tobacco's ongoing marketing to this vulnerable demographic.[19,20]

The Tobacco 21 (T21) legislation in Ireland, although important, is only a partial amelioration strategy compared with Aotearoa New Zealand's game-changing legislation that would have effectively eliminated smoking within a generation.[21] That legislation appeared to herald a template to end the tobacco scourge, a factor particularly important for Māori.[22] The abrupt U-turn by the newly elected Government in Aotearoa New Zealand in early 2024 on this important legislation is best described by Edwards et al. as "*public health vandalism*."[23,24] Although Ireland has also just had a general election in late November 2024, such a U-turn here seems highly unlikely. The now-leader of the largest party in Ireland was formerly the minister for health who introduced ground-breaking smokefree workplace legislation in 2004.[9,25]

Ireland's legislation will not herald the tobacco endgame in the short term. However, to quote Otto von Bismarck, it may reflect the reality that "*Politics is the art of the possible, the attainable—the art of the next best.*"[26] Public health, tobacco control and community advocates in Aotearoa New Zealand must now decide whether to hold firm and try once again to launch their revolutionary tobacco endgame legislation or, alternatively, campaign for a Tobacco 21 approach, at least as an intermediate step.

COMPETING INTERESTS
JL is a committee member of the Pharmaceutical Society of Ireland. He has received funding from the Health Service Executive for development of a national DNACPR Policy.

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A pain in the hip: the under-used potential of fascia iliaca compartment block in the prehospital setting

Sarah E Maessen, Jon Leach, Verity F Todd, Elena Garcia, Bridget Dicker

Fascia iliaca compartment block (FICB) is a technique for flooding the fascia iliaca compartment with local anaesthetic, resulting in blockade of the femoral and lateral cutaneous nerves. It has demonstrated potential as an alternative to opiates for perioperative pain management in shaft or neck of femur (NOF) fracture patients, with a favourable side effect profile.[1]

Evidence for FICB in the prehospital setting is comparatively scarce, but it appears to be feasible, safe and effective when administered by emergency medical services (EMS) for proximal femoral fractures, with the majority of evidence from studies of emergency physicians, anaesthetists or nurses.[2] FICB was adopted into Aotearoa New Zealand EMS clinical practice guidelines for suspected femoral neck or proximal shaft fractures in late 2020.[3]

Despite promising results from a small trial (n=24) in Australia and a feasibility study in Wales,[4] we are not aware of any previous studies looking at real-life implementation of paramedic-administered FICB in the prehospital setting. This study aims to describe characteristics and pain outcomes for patients who were administered FICB by Hato Hone St John (HHStJ) in a 1-year period and compare them with a cohort with similar injuries and no FICB.

Methods

HHStJ is New Zealand's largest EMS provider, covering 90% of the country's population. In September 2020, FICB training began for specialist (postgraduate-qualified) paramedics. The anatomically guided, tactile loss of resistance technique may be used to administer 30ml (patients weighing <60kg) or 40ml (patients weighing >60kg) of 0.375% ropivacaine to patients with severe pain associated with clinically obvious fractured NOF or proximal shaft of femur in line with clinical practice guidelines.[3] Guidelines recommend FICB for patients whose pain is not adequately controlled by an opiate, offering FICB as an alternative to additional medicinal analgesia for this patient group.[3]

The Aotearoa New Zealand Paramedic Care Collection (ANZPaCC) combines EMS patient and incident data and is linked to Ministry of Health – Manatū Hauora data using National Health Index numbers.[5] Within ANZPaCC, we identified emergency call incidents attended by HHStJ between 1 May 2022 and 30 April 2023 with International Classification of Diseases 10th revision (ICD-10) codes indicating a fracture of the neck or proximal shaft of the femur (Appendix Table 1). Patients with Glasgow Coma Scale scores ≤12, a clinical impression of major trauma, clinical status indicating immediate threat to life, aged under 12 years, not transported by HHStJ or who declined assessment by ambulance staff were excluded. The patient's home address was used to estimate relative socio-economic deprivation using the New Zealand Index of Deprivation 2018 (NZDep2018),[6] and incident location was categorised by rurality using the Geographic Classification for Health.[7] Pain was patient-reported on a numerical scale from 0 to 10. Transport time was in minutes from scene of the injury to arrival at a hospital. Ministry of Health – Manatū Hauora gender, ethnicity and ICD-10 diagnosis codes were used (Appendix Table 1). FICB cases were checked against a list of reportable events to identify auditor or crew-reported adverse events, and were manually screened to identify further possible safety concerns.

Statistical analysis used SPSS v29. Patient characteristics were compared between FICB and comparison groups using Independent Samples median tests (continuous variables) or Chi-squared tests (categorical variables). The primary outcome was pain score reduction, calculated as the difference between the first and last score recorded for the incident and compared between FICB and comparison groups using linear regression, with the final model adjusted for initial pain score, gender and rurality. This study was approved by the Northern B Health and Disability Ethics

Committee (Aotearoa New Zealand, Paramedic Care Collection [ANZPaCC], 2022 FULL 13415).

Results

There were 3,860 incidents in the time frame potentially eligible for FICB. Patients receiving FICB were predominantly female (73%), aged 80 years or older (71%), of European ethnicity (92%) and injured in urban areas (79%) (Table 1). A higher proportion of FICB patients were female, but they did not differ from other patients on ethnicity, age or neighbourhood deprivation score (Table 1). Distribution across urban/rural locations differed between FICB and comparison patients, with higher proportions of the FICB group injured in Urban 2 and Rural 1 areas (Table 2). FICB patients more often had initial pain scores in the severe range (74% vs 46%).

No adverse events were reported by crews or auditors for any FICB patient. Based on manual screening of patient records, one patient was identified as experiencing a tonic-clonic seizure shortly after ropivacaine administration. The patient recovered without intervention and is not known to have experienced any further adverse outcomes.

Final pain scores were missing for 1,105 patients. For those with complete pain scores, final scores were 3.2 points lower than initial pain scores, on average. FICB patients had a greater mean reduction in pain score than comparison patients (Table 2), which remained significant after adjustment for initial pain scores, gender and rurality (R^2=.38, F[1,11]=150.2, p<.001).

Discussion

This study adds to evidence for safe and effective prehospital use of FICB to manage pain for NOF fracture. Patients who received FICB reported a greater reduction in pain between ambulance arrival and handover to hospital care, even when controlling for higher initial pain scores. Most FICB patients (94%) experienced a reduction in pain, with 21% reporting no pain on handover. These results are consistent with previous literature on FICB indicating favourable outcomes in emergency department, perioperative and prehospital settings.[1,4,8] Different proportions of patients by rurality likely reflect less need in urban areas close to hospitals and less availability in the most rural areas.

One patient experienced a seizure, a documented symptom of ropivacaine toxicity,[9] within minutes of FICB administration. Two similar incidences were reported in a recent meta-analysis including 257 FICB in the prehospital setting.[4] Though rare, training should prepare clinicians for such events.

Only 3.6% of patients with eligible fractures were treated with FICB in this study. Though we were unable to confirm that the comparison patients were suitable candidates for FICB with this dataset, the proportion has reduced from 5.6% in the first year after the skill was introduced in clinical practice guidelines.[10] Changes to the way paramedics trained to perform FICB are deployed has likely resulted in fewer trained clinicians in ambulances tasked to patients with isolated femoral fractures. EMS often must choose between requesting and waiting for trained personnel to perform the procedure or prioritising prompt transport to definitive treatment. Welsh paramedics also noted that painful repositioning of the patient was sometimes needed to prepare for FICB, whereas intravenous medications could more easily be administered prior to extrication, after which obvious benefits of FICB were less apparent.[11]

Perioperatively, FICB has been associated with improved pain management for up to 48 hours.[1] With increasing lengths of time in emergency departments for hip fracture patients in New Zealand,[12] the value of prompt arrival at hospital should be considered against the potential of prehospital interventions to have longer-term benefits for patients while they are awaiting further assessment or treatment after handover from ambulance care.

This study was limited by the use of administrative data, with a high rate of missing pain scores, particularly in the comparison group. Initial pain score was captured at arrival on scene and may not reflect the pain level used for treatment decisions, which takes into account the need for patient mobilisation or transfer. Other potential benefits of FICB compared with other pain management that we were not able to examine in this study include lower morphine consumption and related nausea,[1,8,13] fewer complications,[8] lower cost[4] and better control of dynamic pain in particular.[13] Further research with frontline staff and longer patient follow-up is needed to understand whether FICB use should be promoted in the prehospital environment.

Table 1: Participant and incident characteristics and comparison between hip fracture patients with and without FICB.

	All participants	**FICB**	**Comparison**	**p-value**
	n=3,860	**n=139 (3.6%)**	**n=3,721 (96.4%)**	
Age (years) Median, IQR	84.0, 14	84.0, 12	83.0, 14	.222
Sex (female)	2,531 (65.6%)	102 (73.4%)	2,429 (65.3%)	**.028**
Ethnicity				.557
Māori	205 (5.3%)	8 (5.8%)	197 (5.3%)	
European/other	3,479 (90.1%)	128 (92.1%)	3,352 (90.1%)	
Pacific people	42 (1.1%)	0	42 (1.1%)	
Asian	133 (3.4%)	3 (2.2%)	130 (3.5%)	
Incident rurality				**.001**
Urban 1	2,087 (54.1%)	63 (45.3%)	2,024 (54.4%)	
Urban 2	924 (23.9%)	47 (33.8%)	877 (23.6%)	
Rural 1	522 (13.5%)	27 (19.4%)	495 (13.2%)	
Rural 2	264 (6.8%)	2 (1.4%)	262 (7.0%)	
Rural 3	34 (0.9%)	0	34 (1.0%)	
NZDep quintile				.841
1	549 (14.5%)	21 (15.9%)	528 (14.5%)	
2	809 (21.4%)	30 (22.7%)	779 (21.4%)	
3	874 (23.1%)	25 (18.9%)	849 (23.3%)	
4	873 (23.1%)	32 (24.2%)	841 (23.1%)	
5	673 (17.8%)	24 (18.2%)	649 (17.8%)	
Transport time (minutes)				
Median, IQR	19.1, 20.9	21.9, 27.8	18.9, 20.8	.052
Pain score initial[a]				**<.001**
0	229 (636%)	2 (1.5%)	227 (6.8%)	
Mild	633 (18.3%)	8 (5.9%)	625 (18.8%)	
Moderate	958 (27.7%)	25 (18.5%)	933 (28.1%)	
Severe	1,638 (47.4%)	100 (74.1%)	1,538 (46.3%)	

FICB = fascia iliaca compartment block; IQR = interquartile range; NZDep = NZDep2018 index of deprivation.[6]
[a]Pain scores of 0 were interpreted as no pain, 1–3 as mild pain, 4–6 as moderate pain and 7-10 as severe pain; n=3,323 due to missing pain score data.
Values are presented as n (%) unless otherwise specified.

Table 2: Pain outcomes for hip fracture patients with and without FICB.

	FICB	Comparison	p-value
	n=126*	n=2,629*	
Final pain score[a]			**<.020**
0	26 (20.6%)	346 (12.5%)	
Mild	64 (50.8%)	1,392 (50.5%)	
Moderate	26 (20.6%)	792 (28.7%)	
Severe	10 (7.9%)	225 (8.2%)	
Pain reduction M(SD)	4.8 (2.9)	3.1 (2.8)	**<.001**
Pain reduction adjusted[b] (EMM, 95% CI)	4.6 (3.7–5.4)	3.2 (2.9–3.4)	**<.001**

FICB = fascia iliaca compartment block; M(SD) = mean (standard deviation); EMM = estimated marginal mean; CI = confidence interval.
*Excludes 13 FICB and 1,092 comparison patients with incomplete pain records.
[a]Pain scores of 0 were interpreted as no pain, 1–3 as mild pain, 4–6 as moderate pain and 7–10 as severe pain.
[b]Adjusted for initial pain score, gender and rurality.

COMPETING INTERESTS

SM, JL, EG and BD are employed by Hato Hone St John. The authors have no further conflicts of interest to declare.

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Appendix

Appendix Table 1: ICD-10 codes used to select patients diagnosed with a fracture of the neck or proximal shaft of femur.

ICD-10 code	Definition
S7200	Fracture of neck of femur, part unspecified
S7201	Fracture of intracapsular section of femur, unspecified
S7202	Fracture of upper epiphysis (separation) of femur
S7203	Fracture of subcapital section of femur
S7204	Fracture of midcervical section of femur
S7205	Fracture of base of neck of femur
S7208	Fracture of other parts of neck of femur
S7210	Fracture of trochanteric section of femur
S7211	Fracture of intertrochanteric section of femur
S722	Subtrochanteric fracture
S723	Fracture of shaft of femur
S727	Multiple fractures of femur
S728	Fractures of other parts of femur
S729	Fracture of femur, part unspecified

ICD-10 = International Classification of Diseases 10th revision.

Notes on Eight Cases of Neuro-Syphilis.

NZMJ, 1925

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The following are notes on the salient features of eight cases of neuro-syphilis, from the records of this hospital for the last six months. They exhibit no particular feature in common, but illustrate rather the variety of the clinical picture due to a syphilitic infection of the brain, cord, or meninges. Special reference is made to the laboratory findings as regards both blood and cerebrospinal fluid. The notes on treatment, so far as they are available to date, indicate that certain of these cases are strikingly amenable to anti-syphilitic measures; this applies to the meningeal and meningo-vascular lesions, but not to the cases of frank tabes or general paresis, in which degeneration of nervous tissue has occurred.

The classification adopted is as follows:—

(1) *Syphilitic meningitis*.—A chronic inflammation of the meninges, having its origin in the small blood vessels. It is usually basal, and the symptoms are entirely referable to the meningitis.

(2) *Meningo-vascular syphilis*.—A syphilitic meningitis together with a syphilitic arteritis affecting the larger vessels, and causing vascular paralysis (thrombosis, etc.). (3) *The so-called "parasyphilitic infections"*: (a) Tabes dorsalis; (b) general paralysis.—In these the syphilitic virus has chiefly attacked the nerve tissue itself, in the cord or the brain; an associated meningitis is often present.

CASE I.—J. W. S., male, æt. 38. Admitted 21st January, 1924, complaining of headache and difficulty of speech. Primary infection denied. Tertiary symptoms began eight to ten months ago with occipital headache, difficulty in articulation, intermittent motor aphasia and some tremor of hands. These were all attributed to an injury—a blow on the back of the head.

Present condition.—He shows considerable mental vagueness, his memory is imperfect and he says that "sometimes he can think and sometimes he can't." His speech is slow, laboured and indistinct, and often wandering and irrelevant. He has delusions of poisoning and castration, and also to the effect that someone is interfering with his wife. He resists the attendants violently. Physical examination shows tremor of lips and tongue, with slow, hesitant, indistinct speech; dullness of hearing on the left side. Eyes react to light and his fundi are normal. Deep reflexes are normal.

Laboratory findings.—Wasserman reaction blood serum, strongly positive. Spinal fluid, pleocytosis—28 cells per cm.m. Pandy's test for globulin strongly positive. Wassermann reaction, strongly positive.

Diagnosis.—Early general paresis.

The diagnosis of this case, from a purely clinical point of view, was complicated by the history of injury, for which compensation had been claimed, and by the fact that the knee jerks and eye reflexes were normal. It was not established until the serological and cytological findings were obtained. The case was one of early G.P.I., and since his transfer to the mental hospital, the typical physical and mental signs have developed.

CASE II.—J. R., male, æt. 49.—Admitted 12th May, 1924, for observation of his mental condition. Primary infection in Egypt in 1915. Treatment was commenced one week afterwards and since then he has had several courses of N.A.B. and mercury. During the two years he has been under treatment at the Outpatients' Department here, his Wassermann reaction has remained persistently positive. He had been told that he had valvular disease of the heart, and has suffered from occasional attacks of giddiness, shortness of breath and precordial pain. Since December his friends had noticed marked mental changes in the patient. He was profane in the presence of women, forgot things, readily, and called at the Post Office for £200 which was not there.

Present condition.—The patient is garrulous and in a state of exaltation, he talks freely about financial arrangements and says he is going for a motor tour of the South Island. He is careless in his physical habits and exposes himself in the ward. On physical examination there is no Rombergism, his pupils are equal and react to light. Knee jerks present and not exaggerated. Tongue shows no tremor. The other systems appear normal.

Laboratory findings.—Wassermann reaction

in the blood serum is strongly positive, (44444) on Kolmer's quantitative scale. Spinal fluid shows a marked pleocytosis—53 cells per cm.m. Pandy's test for globulin double plus. W.R. double plus.

Diagnosis.—Early general paresis.

The mental change in this patient is typical of early G.P.I., but as yet no physical signs have appeared. It is interesting to note that he received intensive anti-syphilitic treatment one week after the primary lesion appeared, and since then has received several further courses of arsenic and mercury. Cases in which in spite of treatment, the Wassermann reaction remains consistently and strongly positive, appear to be more likely than others to develop neuro-syphilis.

CASE III.—R. D., male, æt. 32. Admitted 8th April 1924, complaining of sleeplessness and absent-mindedness. Primary infection is denied. For the past six months the patient's friends have noticed some mental change in him. He is "queer" and inattentive at times. He forgets with undue readiness, and has fits of absent-mindedness.

Present condition.—The patient has a somewhat helpless, vacant look, and in manner and speech appears rather simple, but shows no very definite abnormal symptom. On physical examination no signs can be elicited which suggest any lesion of the nervous system.

Laboratory findings.—Wassermann reaction in the blood serum is strongly positive. Spinal fluid shows a considerable pleocytosis, 58 cells per cm.m. Pandy double plus. Wassermann reaction strongly positive.

Diagnosis.—Cerebral syphilis.

This patient shows only a slight degree of mental change, in the direction of forgetfulness and absent-mindedness, and no physical signs whatever. The discovery on routine examination of a positive blood Wassermann, suggested a serological examination of his spinal fluid, with the above findings. These mark the case as one of cerebral syphilis, a meningitis is undoubtedly present, but it is uncertain as yet how far the nerve cells are involved.

CASE IV.—R. H., male, æt. 40. Admitted 18th September, 1923, complaining of giddiness, nausea and vomiting, and headache. Primary infection unknown. The above symptoms appeared suddenly six months ago and have recurred fortnightly or more frequently since. He has also noticed failing eyesight and drowsiness.

Present condition.—While in hospital patient had an attack of mental derangement with hallucinations of sight and hearing, and a strongly expressed desire to commit suicide. Physical examination shows impaired function of V. and VII. on the left side. There is slight nystagmus on looking to the left. He is slightly deaf on the left side. His pupils react normally. Deep reflexes are exaggerated. Jaw jerk marked. Plantar response markedly extensor on both sides. Rombergism present.

Laboratory findings.—Wassermann reaction blood strongly positive. Spinal fluid, Wassermann reaction strongly positive.

Diagnosis.—Syphilitic meningitis.