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Does screening for myopia in New Zealand meet screening programme criteria?

Ben Wilkinson, Graham Wilson

There is a worldwide myopia (short-sighted epidemic). Our paper aimed to assess whether a myopia (short-sighted) screening programme could be useful in New Zealand to provide early diagnosis, treatment and prevention of secondary cause of blindness associated with myopia. Seven out of 10 of the criteria were met for a screening programme being worthwhile. Once robust clinical treatment guidelines are established, a pilot screening programme could be run in New Zealand to test real-world viability of a screening programme for myopia in New Zealand.

Quality assessment of a large primary GP skin cancer service in Auckland, New Zealand

Daniel Wen, Katherine Gale, Richard CW Martin

A group of general practitioners were recruited in 2014 by Waitematā District Health Board to be further trained in the management of skin cancer by experts in the field. This study aimed to evaluate the performance of this group of doctors and identify the strengths and weaknesses of the programme. The results of our research showed that the upskilled general practitioners' performance was as good as, if not better than, other skin cancer services and was acceptable under international skin cancer standards. Treatment by these general practitioners was also less expensive for the health sector when compared to in-hospital treatment and also associated with reduced wait times.

Ethnic inequities in life expectancy attributable to smoking

Michael Walsh, Karen Wright

Our study provides an updated assessment of the burden of smoking-related mortality in New Zealand for deaths registered from 2013 to 2015 and examines the contribution of these deaths to the life expectancy gap in the Māori and Pacific populations compared with the non-Māori/non-Pacific population. Over this time, 13.4% of deaths were potentially attributable to smoking. Among Māori, 22.6% of deaths were potentially attributable to smoking, compared with 13.8% of deaths among Pacific peoples and 12.3% among non-Māori/non-Pacific people. We estimate that smoking attributable deaths contributed 2.1 years to the life expectancy gap in Māori men, 2.3 years in Māori women (nearly one third), 1.4 years of Pacific men (one quarter) and 0.3 years in Pacific women. Smoking remains an important preventable risk factor contributing to ethnic inequities in life expectancy for Māori men and women, and Pacific men. Addressing this inequity needs to remain a top health priority. In order to achieve health equity and the Smokefree 2025 goal, a Tiriti o Waitangi compliant response inclusive of targeted investment and expansion of Māori and Pacific tobacco control programmes is required.

Good care close to home: local health professional perspectives on how a rural hospital can contribute to the healthcare of its community

Katharina Blattner, Tim Stokes, Marara Rogers-Koroheke, Garry Nixon, Susan M Dovey

This study was set at Hauora Hokianga, an established rural health service with a small rural hospital serving a largely Māori community in New Zealand's far north. The aim of the study was to understand how Hauora Hokianga's staff (both medical and non-medical) viewed the role of the Hokianga hospital. The study provides a perspective of how a rural hospital can contribute to the healthcare of its community. Findings highlight the importance of geographically as well as culturally appropriate health services. Further research into the function of New Zealand rural hospitals and their contribution to the health system is needed.

Epidemiology of traumatic spinal cord injury in New Zealand (2007–2016)

John Mitchell, Joanne Nunnerley, Chris Frampton, Tracey Croot, Alpesh Patel, Rowan Schouten

The causes and population affected by traumatic spinal cord injuries (TSCI) is changing in New Zealand. Falls are now the most common cause of TSCI, which is reflected by an increased rate in elderly patients. Māori patients are 1.8 times more likely to have a TSCI compared to New Zealand Europeans, a disparity that is shown to be increasing over time. Sporting-related TSCI remains high in New Zealand compared to the rest of the world with team ball sports (eg, rugby) the biggest cause.

Healthcare-associated *Staphylococcus aureus* bacteraemia: time to reduce the harm caused by a largely preventable event

Sally A Roberts, Nikki Grae, Sharmini Muttaiyah, Arthur J Morris

Bacteria that live on our skin such as *Staphylococcus aureus* usually do not cause us any harm. However, if you are admitted to hospital and require surgery or treatment for a medical condition then the risk of infection is increased if you have a surgical wound or your skin is punctured by a needle or other medical device. Each time this happens *S. aureus* to is able to get beyond the barrier created by your healthy skin and cause infection. There are a number of ways that healthcare workers can reduce this from happening; cleaning your skin before breaching it, performing hand hygiene at appropriate times, removing medical devices when they are no longer needed and looking after wounds. Interventions to support adherence to these and other activities can significantly reduce the risk for acquiring infection in hospital.

Nutrition guidelines for dental care vs the evidence: is there a disconnect?

Sarah Hancock, Caryn Zinn, Grant Schofield, Simon Thornley

Eating refined, high carbohydrate foods is the principal cause of tooth decay, and consumption of these foods are also implicated in the increased prevalence of both malnutrition and obesity in children and young people. Furthermore, eating full-fat dairy produce is associated with reduced risks of tooth decay and obesity in children and young people. Although government-endorsed dietary guidelines for young people correctly provide recommendations to decrease intake of high-sugar foods, recommendations are also provided to increase the amount, and frequency of consumption of high carbohydrate foods as children age, and to choose low-fat dairy produce. Given that the epidemics of dental caries and obesity are a significant and ongoing public health challenge in New Zealand, it is imperative that the guidelines for healthy eating for young New Zealanders incorporate the best dietary advice to improve their health. It is time to update the guidelines to limit the intake of ultra-processed foods and encourage intake of full-fat dairy products to prevent the dental caries and obesity, which share a common cause.

A disclosure form for work submitted to medical journals: a proposal from the International Committee of Medical Journal Editors

Darren B Taichman, Joyce Backus, Christopher Baethge, Howard Bauchner, Annette Flanagin, Fernando Florenzano, Frank A Frizelle, Fiona Godlee, Laragh Gollogly, Abraham Haileamlak, Sung-Tae Hong, Richard Horton, Astrid James, Christine Laine, Pamela W Miller, Anja Pinborg, Eric J Rubin, Peush Sahni

Many factors, including professional and personal relationships and activities, can influence the design, conduct and reporting of the clinical science that informs healthcare decision. The potential for conflict of interest exists when these relationships and activities may bias judgement.[1] Many stakeholders—editors, peer reviewers, clinicians, educators, policymakers, patients and the public—rely on the disclosure of authors' relationships and activities to inform their assessments. Trust in the transparency, consistency and completeness of these disclosures is essential.

Ten years ago, the International Committee of Medical Journal Editors (ICMJE) adopted the "ICMJE Form for the Disclosure of Potential Conflicts of Interest" as a uniform mechanism for collecting and reporting authors' relationships and activities that readers might consider relevant to a published work.[2] The goal was to avoid the confusion (and often ensuing controversy) created when journals vary in how they collect and report this information. We believe a uniform disclosure form has been helpful, but problems remain. First, the software supporting the current form is increasingly problematic, making its use difficult or impossible for an increasing number of authors. More important, however, is that many authors and readers misunderstand, misapply or misinterpret the disclosures.

Although some individuals violate the public trust by purposefully hiding relevant relationships and activities, we believe most authors are committed to transparent reporting and consider it as vital to the advancement of clinical science. Nonetheless, disagreement, confusion and controversy regarding authors' disclosures arise when opinions differ over which relationships and activities to report. An author might not report an item that others deem important because of a difference in opinion regarding what is "relevant", confusion over definitions or a simple oversight. Some authors may be concerned that readers will interpret the listing of any item as a "potential conflict of interest" as indicative of problematic influence and wrongdoing, a concern often raised regarding the requirement to report publicly funded grants. For their part, some readers fail to recognise that their own relationships and activities influence how they assess the work of others and what they deem to be a "conflict" for others or themselves.

We propose several changes to the ICMJE disclosure form to help address these issues. First, words matter. Despite including the word "potential", a form entitled "…for the Disclosure of Potential Conflicts of Interest" may imply that any relationship or activity listed represents a problematic influence or wrongdoing. The proposed new title, "The ICMJE Disclosure Form", aims to dispel

that interpretation and potential stigma. Second, we no longer ask authors to decide what might be interpreted as a potential conflict of interest. Authors disclose their relationships and activities so that readers can decide whether these relationships or activities should influence their assessments of the work. Further, to avoid omissions—inadvertent or purposeful—we now provide a checklist of relationships and activities for authors to complete.

We welcome feedback about the proposed new form, which is available with a link to provide comments, at www.icmje.org. We will consider comments received by 30 April 2020, before finalising and adopting a revised version. In the interim, the extant "ICMJE Form for the Disclosure of Potential Conflicts of Interest" will remain in use and available as a downloadable PDF at our website.

In a further step to avoid inconsistencies and omissions, and to help ease the disclosure process for authors, some journals will change the mechanism by which disclosures are collected. Authors are required to provide disclosures to multiple entities (eg, to academic institutions, continuing education providers, guideline and other committees as well as medical journals). Disclosing information repeatedly, with varying reporting requirements, formats and definitions, is frustrating for authors and contributes to problematic and controversial discrepancies across disclosures. The ICMJE will therefore accept disclosures from web-based repositories. These enable authors to maintain an inventory of their relationships and activities and create electronic disclosures tailored to the requirements of entities such as ICMJE, without having to reenter information repeatedly.

ICMJE will accept disclosures from repositories that meet the following criteria: collection and reporting of relationships and activities consistent with ICMJE requirements; no fees for individuals to enter, store or export their data; provision of disclosures to journals electronically as well as an option for journals without a digital interface; and compliant with the General Data Protection Regulation (GDPR).

One currently available repository that is consistent with these criteria is Convey (www.convey.org), but we encourage the development of other repositories as necessary to meet regional, linguistic and regulatory needs. A template that enables authors to create disclosures that emulate the extant "ICMJE Form for the Disclosure of Potential Conflicts of Interest" is already available at the Convey platform, and some of our journals have begun to collect author disclosures electronically in this way. This template will be updated to conform to the new ICMJE Disclosure Form when it is finalised, and all ICMJE journals can begin accepting disclosures in this manner. Ultimately, the currently employed PDF-based ICMJE form will be unavailable.

While no approach to disclosure will be perfect or foolproof, we hope the changes we propose will help promote transparency and trust. We look forward to your feedback.

Note: This article is being published simultaneously in Annals of Internal Medicine, BMJ (British Medical Journal), Bulletin of the World Health Organization, Deutsches Ärzteblatt (German Medical Journal), Ethiopian Journal of Health Sciences, JAMA (Journal of the American Medical Association), Journal of Korean Medical Science, The Lancet, New England Journal of Medicine, New Zealand Medical Journal, Revista Medica de Chile (Medical Journal of Chile) and Ugeskrift for Laeger (Danish Medical Journal).

Disclaimer: Dr Sahni's affiliation as representative and past president of the World Association of Medical Editors (WAME) does not imply endorsement by WAME member journals that are not part of the ICMJE.

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Dr Taichman is an employee of the Annals of Internal Medicine and the American College of Physicians; Dr Baethge is an editor of a medical journal (Deutsches Arzteblatt) and employed by a publishing house (Deutscher Arzteverlag); Dr Haileamlak is the Editor of Ethiopian Journal of Health Sciences and member of ICMJE; Ms Flanagin is a non-paid board member of STM: International Association of Scientific, Technical, and Medical Publishers; Dr Laine is a full time employee of the American College of Physicians and serves as Senior Vice President of the organisation, and the Editor in Chief of Annals of Internal Medicine. The journal is owned and published by the ACP.

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REFERENCES:

1. Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Work in Medical Journals. Accessed at www.icmje.org/icmje-recommendations.pdf on 17 December 2019.
2. Drazen JM, Van Der Weyden MB, Sahni P, et al. Uniform format for disclosure of competing interests in ICMJE journals [Editorial]. Accessed at www.icmje.org/news-and-editorials/format_disclosure_coi_oct2009.pdf on 17 December 2019.

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Does screening for myopia in New Zealand meet screening programme criteria?

Ben Wilkinson, Graham Wilson

ABSTRACT

AIM: The purpose of this paper is to assess whether screening for myopia in New Zealand is valid under scrutiny of the Wilson and Jungner criteria. There is a worldwide myopia epidemic which requires urgent attention to reduce vision impairment, blindness and costs to wider society. The risks associated with myopia are under-appreciated in New Zealand, and treatments need to be refocused from correcting refractive error to preventing axial length elongation.

METHODS: The Wilson and Jungner criteria was used to assess the validity of screening for myopia in New Zealand through review of the latest evidence relevant to each point within the criteria.

RESULTS: We found that the screening for myopia in New Zealand met 7 out of 10 of the Wilson and Jungner criteria.

CONCLUSIONS: The concept of a screening programme for myopia in New Zealand performed relatively well, and should be considered further. Further randomised clinical trials, which clearly identify the appropriate treatment modalities and timing, would allow the establishment of robust New Zealand specific myopia management guidelines. We would then suggest a trial of a screening programme in New Zealand setting to assess real-world feasibility and cost-effectiveness to identify early myopia and provide treatment to slow progression. Adjustments could be made to the already available screening programme, consisting of suitable reduction of screening age, and introduction of autorefractors.

Myopia is the most common ocular problem internationally and prevalence is increasing. Research suggests myopia currently affects 23% of the world population, with estimates of 49% affected by 2050.[1] In certain East Asian countries myopia affects 90% of young adults.[2] Nearly a third (30%) of 17-year-olds in Sydney are myopic, which represents a doubling of prevalence from a decade earlier.[3] The only published New Zealand data come from a survey (1984) with a prevalence of myopia of 4.2% in Dunedin 11-year-olds.[4] Unpublished data from the same cohort at age 45 shows prevalence of myopia of 34.1%.[5] The increasing prevalence of myopia is linked primarily to environmental factors including near work activities and decreased outdoor time.[6] High myopia increases the risk of irreversible vision loss through predisposition to ocular changes, including cataract, glaucoma, retinal detachment and myopic macular degeneration.[1] Even a low myope (one to three dioptres) is twice as likely to develop myopic maculopathy, glaucoma or posterior subcapsular cataract, and at least three times as likely to develop retinal detachment.[7–9]

Petty et al suggested that the risks associated with myopia in New Zealand are under-appreciated by the medical, educational and public health community.[10] Current treatment is aimed at correcting refractive error rather than preventing axial length elongation. Attempting to prevent myopia progression offers an opportunity to decrease the burden of myopia on individuals and wider society.[10] Current New Zealand national vision screening guidelines published in 2014

state two purposes: to identify children with amblyopia at an age when treatment might be effective; and to identify and refer children with reduced visual acuity for further assessment. Screening consists of a 'B4 School Check' (<age 4) and Year 7 (age 11) vision screening. Visual acuity testing is measured and referrals are made to optometrists or ophthalmologists if screening requirements are not met.[11] The 2010 Eye Health Workforce Service Review suggested improvements are needed in vision screening. Health Workforce New Zealand supports the rationalisation and standardisation of child vision screening services in New Zealand.[12]

The aim of a screening programme is to identify disease in a community early, to enable earlier intervention to avoid suffering from the disease.[13] Proposed screening programmes must be carefully evaluated to avoid potential adverse effects of screening.[14] The aim of this paper is to evaluate the validity of screening for childhood myopia in New Zealand utilising the Wilson and Jungner criteria. The Wilson and Jungner criteria published in 1968 is a method for considering the utility of screening programmes for disease control.[15] Although newer policy tools are available, the validity of the criteria remains undisputed today.[13] Ideally all 10 of the Wilson and Jungner criteria should be met before a screening programme is adopted. If there is uncertainty or failure to meet criteria, then further research or pilot screening programmes should be conducted. The criteria have different weights in different settings; for example, in a wealthier jurisdiction there may be less emphasis on cost-effectiveness. Furthermore, issues such as "service availability" will be more important in rural settings compared with highly urbanised settings.[13] Below, the criteria are used to examine the validity of screening for childhood myopia in New Zealand.

The condition sought should be an important health problem

Myopia is the most common cause of distance vision impairment internationally and prevalence is increasing.[1] Individuals not treated endure lifelong visual impairment and increased risk of blindness.[7–9] Financial cost to individuals and society are significant.[16,17] Refractive error and low vision are ranked number five on the World Health Organization priority eye disease list.[18] Myopia ranks as the third most common long-term health condition in Australian children.[19]

There should be an accepted treatment for patients with recognised disease

Researched methods to reduce the rate of myopia progression include behavioural, pharmacological and optical approaches.[20] The International Myopia Institute concluded that modalities within all three approaches are worthy of further exploration, and variability in treatment efficacy exists at an individual level.[21] Increased near work and lack of outdoor activity are risk factors for myopia progression, and therefore are the targets of behavioural intervention.[6] A 2017 meta-analysis showed increased outdoor time reduced myopia onset and subsequent progression.[22]

Pharmacologically, multiple antimuscarinic drugs have been studied. Low-dose atropine has proven most promising with clinical efficacy, and tolerability with minimal adverse effects.[23–25] Chia et al concluded that low-dose atropine (0.01%) for periods up to five years is a clinically viable treatment.[26] Their results showed mean myopia progression at five years (1.38 D) in children receiving atropine 0.01% was similar to placebo eyes at 2.5 years (1.40 D), suggesting a 50% reduction in progression of spherical equivalence.[26] However, it is important to note that the effect of low-dose atropine (0.01%) on slowing axial elongation has not been convincingly established.[27] Issues also exist around possible myopic rebound with treatment cessation, and requirement for long-term adherence.[23–25] The recent one-year results of the LAMP study conclude that 0.05%, 0.025% and 0.01% atropine eye drops were well tolerated, and reduced myopia progression with a concentration-dependent response.[28] 0.05% atropine was the most effective in controlling spherical equivalence progression and axial length elongation over a period of one year.[28]

Results showed a larger axial length change at one year in the placebo group (0.41 +/- 0.22mm) than in the 0.05% (0.20+/-0.25mm), 0.025% (0.29+/-0.20mm) and 0.01% (0.36+/-0.29mm) atropine groups (P<0.001). Pairwise comparison of axial length change between the 0.01% atropine and placebo groups was not statistically significant (P<0.18).[28]

Various optical treatments have been investigated for myopia treatment. The majority have shown some efficacy in small studies, although some systematic reviews question viability. The International Myopia Institute suggests that orthokeratology lenses slow myopia by approximately 30–60%.[21] A 2015 systematic review also suggested orthokeratology is viable for myopia treatment.[29] A further meta-analysis in 2018 concluded that adoption of orthokeratology for myopia control in children requires careful thought, given the risk-benefit ratio combined with the low compliance of the patients has not yet delivered unidirectional results.[25] Benefits of orthokeratology include not having to wear a vision correction during the day.[21] Issues include risk-benefit ratio with increased risk of keratitis, possible myopic rebound on cessation, and compliance.[24] The International Myopia Institute suggested multifocal soft contact lenses are expected to slow myopia progression by about 30–50%.[21]

Available guidelines from the International Myopia institute and the Brien Holden Institute advise clinicians on implementing methods of intervention for myopia control using available evidence.[30,31] Given existing uncertainties around optical and pharmacological treatment, further randomised clinical trials which clearly define appropriate myopia treatment modality and timing will be integral in ensuring clinical guidelines are robust. New Zealand Myopia Action Group (NZMAG) is a panel of experts with an aim to reduce the impact of myopia in New Zealand. They aim to create New Zealand-specific guidelines adapted from the aforementioned international guidelines.

Facilities for diagnosis and treatment should be available

Vision screening programmes already exist through New Zealand Vision Hearing Technicians, ie, the B4 School check (age <4) and year 7 (age 11) vision screening.[12] The B4 School Check is performed in conjunction with hearing testing. Once an individual is diagnosed with myopia, they would be referred to publicly funded optometry or ophthalmology clinics for ongoing management. The New Zealand Myopia Action Group (NZMAG) is advocating for Pharmac to fund low-dose atropine. Current monthly cost of low-dose atropine (0.01%) in New Zealand is approximately $50/month per individual.[32]

There should be a recognisable latent or early symptomatic stage

Myopia can be easily identified at an early stage through photorefraction combined with an accepted definition of myopia.[33–35]

There should be a suitable test or examination

A variety of reliable and user friendly vision screening devices exist including PlusoptiX, Retinomax, Welch Allyn Spot Vision Screener, Topcon KR-8900, Nidek ARK-510A and Huvitz HRK-7000A. Photorefraction is advantageous over visual acuity given it obtains an estimation of severity of myopia.[33] PlusoptiX photorefraction vision screening device aims to empower primary healthcare providers to detect prevalent vision disorders in children as early as possible. Screening with PlusoptiX is possible from the age of five months and meets the guidelines of the American Academy of Pediatrics.[33] PlusoptiX A12 has been shown to yield a good estimation of the spherical and cylindrical component of refractive errors compared to cycloplegic examination, with greater accuracy in the myopic and astigmatic subgroups compared with hyperopic subgroups.[36] The sensitivity, specificity, positive and negative predictive values for myopia were, respectively, 86%, 93%, 82% and 94%, with an average overestimation of myopia by 0.05 D.[36]

The test should be acceptable to the population

Photorefraction vision screening devices offer a reliable, fast, non-invasive, user friendly screening method that is acceptable to the population.[33–36]

The natural history of the condition, including development from latent to declared disease, should be adequately understood

Our understanding of most medical conditions is often in a state constant evolution. It could be argued that we have an adequate understanding of myopia. Myopia is variably defined as a refractive error equal to or worse than −0.25 to −1.00 D, and is often graded according to severity and age of onset.[37] Severity of myopia is generally categorised as low (−0.50 to −2.99 D), moderate (−3.00 to −5.99 D) or severe (worse than or equal to −6.00).[38]

Myopia is understood as a multifactorial disease influenced by interplay between genetic and environmental factors.[24] In 2016 approximately 70 genetic loci had been linked to primary myopias.[39] Animal models have shown response of axial eye growth in order to compensate for imposed defocus.[40] Various bio-molecular pathways have been investigated; for example, dopamine plays an important role in the development of experimental myopia.[41] Myopia usually first occurs in school-age children. This is defined as youth onset myopia with onset prior to age 20. Because the eye continues to grow during childhood and adolescence, myopia typically progresses until about the age of 20 years.[39]

There should be an agreed policy on whom to treat as patients

A 2018 survey of paediatric ophthalmologists internationally concluded "there is no consensus of the best treatment method in order to prevent myopia progression, when to begin treatment, and in whom treatment should be tailored according to one's genetic background".[20] In August 2018, the Brien Holden Vision Institute released "Guidelines for Myopia Management" (Figure 1). Described as an "evidence-based, free, easy-to-use, practical tool developed to assist the busy eye-care professional manage patients with myopia". The guidelines inform practitioners of appropriate diagnostic tests, risk assessment, myopia management options and scheduling of follow-up visits and tests.[30] In February 2019, the International Myopia Institute released a "Clinical Management Guidelines Report". The report suggests effective guidelines for myopia control require understanding of the epidemiology of myopia, risk factors, interventions, as well as an appropriate communication strategy. The report detailed an evidence-based best practice approach to myopia control, including risk factor identification, examination, selection of treatment strategies and guidelines for ongoing management.[31] They highlighted that there is currently no research investigating the appropriate point of intervention based on age or refractive status. They suggested that appropriate treatment should be selected based on patient specific factors, with multiple risk factors requiring more strategic management and closer monitoring. Their guidelines suggest reassessment every six months to monitor efficacy and safety, with more frequent visits within the first six months.[41] New Zealand Myopia Action Group is a panel of experts with an aim to reduce the impact of myopia in New Zealand. They are refining New Zealand-specific guidelines adapted from aforementioned international guidelines.

The cost of case finding (including diagnosis) should be economically balanced in relation to possible expenditure on medical care as a whole

Myopia imposes considerable economic burden on patients, public health systems and wider society. Analyses performed in Australia and the US have shown the cost of refractive correction is the highest among ocular diseases.[42] Estimates suggests annual global loss of $202 billion of gross domestic product due to uncorrected refractive error.[17] A study of Singaporean adults with myopia estimated a cost of US$709 per person per year. This equates to lifetime cost to an individual with disease for 80 years of US$17,020.[43] Costs of an effective screening programme include staffing, administration, screening facilities and vision screening equipment. Facilities to provide ongoing monitoring of efficacy and safety would be required.[41] Efficacy would be monitored by axial length measurement and refraction. Safety monitoring of atropine would require intraocular pressure testing and pupil function, and orthokeratology would require corneal topography.[41] Treatment costs of identified cases with low-dose atropine are likely to be low.[30] Nevertheless, there is still a need for real-world cost-effectiveness studies of using antimuscarinic drugs for slowing progression of myopia.

Figure 1: Guidelines for Myopia Management, Brien Holden Institute.[40]

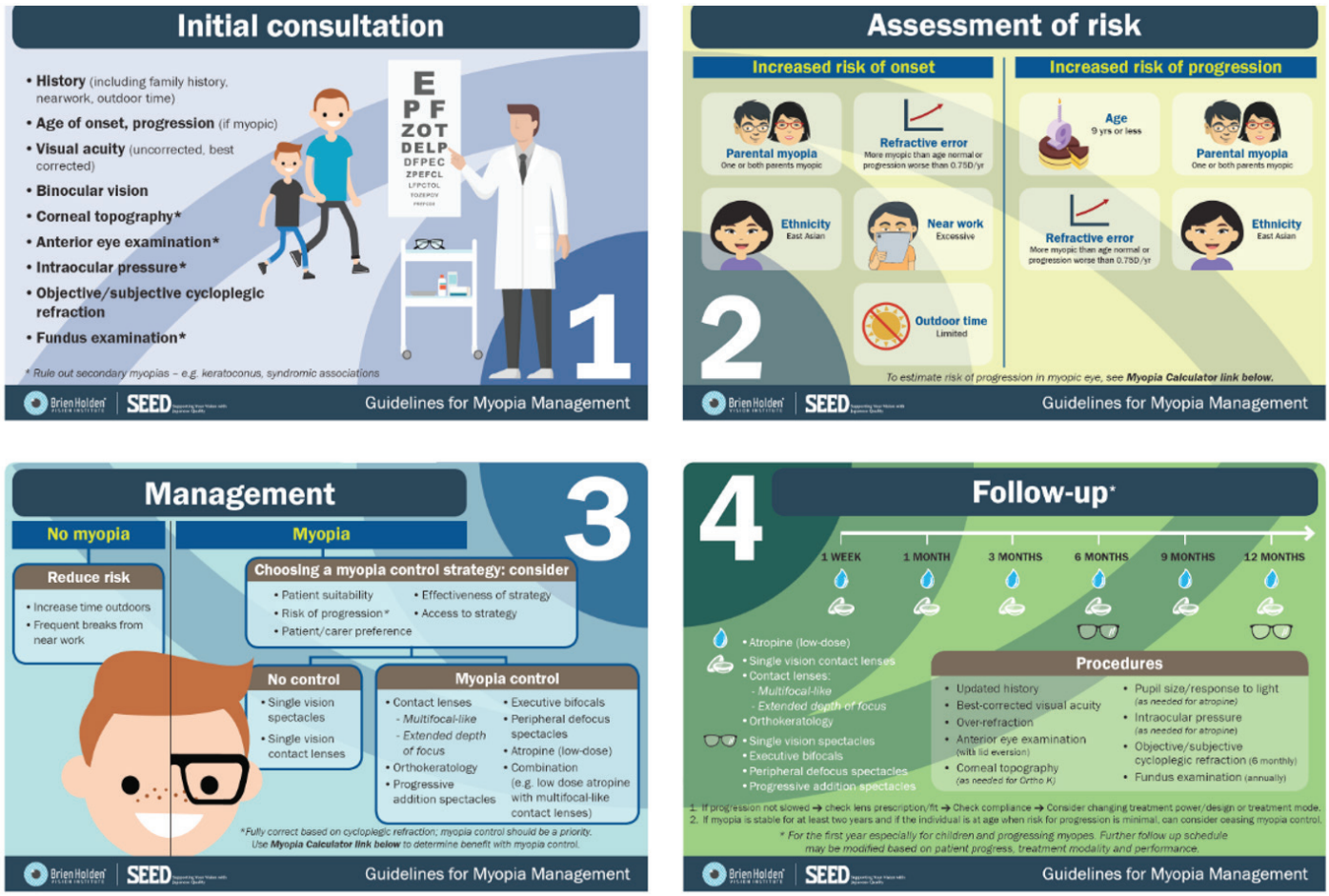

Table 1: Validity of Screening for Childhood Myopia in New Zealand—Wilson and Jungner Criteria.[15]

The condition sought should be an important health problem	✓
There should be an accepted treatment for patients with recognised disease	X
Facilities for diagnosis and treatment should be available	✓
There should be a recognisable latent or early symptomatic stage	✓
There should be a suitable test or examination	✓
The test should be acceptable to the population	✓
The natural history of the condition, including development from latent to declared disease, should be adequately understood.	✓
There should be an agreed policy on whom to treat as patients	X
The cost of case finding (including diagnosis) should be economically balanced in relation to possible expenditure on medical care as a whole	✓
Case finding should be a continuing process and not a “once and for all” project	X

Case finding should be a continuing process and not a "once and for all" project

We propose adjusting the year 7 (age 11) vision screening program to include a one-off myopia check. We therefore accept failure of our proposal to meet this requirement.

Conclusion

There is a worldwide myopia epidemic which requires urgent attention to reduce vision impairment, blindness and costs to wider society.[1] The risks associated with myopia are under-appreciated in New Zealand, and treatments need to be refocused from correcting refractive error to preventing axial length elongation.[10] We've shown that under scrutiny of the Wilson and Jungner criteria, the concept of a screening programme for myopia in New Zealand performed relatively well, and so should be considered further. What is needed is further results from randomised clinical trials which clearly establish the appropriate myopia treatment modalities and timing. The New Zealand Myopia Action Group will then work to establish New Zealand specific myopia management guidelines. Following this, a trial should be conducted in the New Zealand setting to assess real-world feasibility and cost-effectiveness of a screening programme to identify early myopia and provide treatment to slow progression. Adjustments could be made to the already available year 7 (age 11) screening programme, consisting of suitable reduction of screening age, and introduction of autorefractors. This would also serve to help accurately document the prevalence of myopia in New Zealand. Addressing the myopia epidemic is a hot topic internationally, with a high volume of ongoing discussion and research. We need to act now, remain active in international discussion, and have an evidence-based approach which is adaptable to new findings.

Competing interests:
Nil.

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https://www.nzma.org.nz/journal-articles/does-screening-for-myopia-in-new-zealand-meet-screening-programme-criteria

REFERENCES:

1. Holden BA, Fricke TR, Wilson DA, et al. Global Prevalence of Myopia and High Myopia and Temporal Trends from 2000 through 2050. Ophthalmology. 2016 May; 123(5):1036–42.
2. Pan C-W, Dirani M, Cheng C-Y, et al. The age-specific prevalence of myopia in Asia: a meta-analysis. Optom Vis Sci. 2015 Mar; 92(3):258–66.
3. French AN, Morgan IG, Burlutsky G, et al. Prevalence and 5- to 6-year incidence and progression of myopia and hyperopia in Australian schoolchildren. Ophthalmology. 2013 Jul; 120(7):1482–91.
4. Beauchamp G. Refractive error, IQ and reading ability: A longitudinal study from age seven to 11: by SM Williams, GF Sanderson, DL Share, and PA Silva. Dev Med Child Neurol 30: 735–742, 1988. Surv Ophthalmol. 1989; 34(3):234–5.

5. Wilson G. (2019). Dunedin multidisciplinary health and development study p45. Unpublished raw data.
6. Lin Z, Gao TY, Vasudevan B, et al. Near work, outdoor activity, and myopia in children in rural China: the Handan offspring myopia study. BMC Ophthalmol. 2017 Nov 17; 17(1):203.
7. Flitcroft DI. The complex interactions of retinal, optical and environmental factors in myopia aetiology. Prog Retin Eye Res. 2012 Nov; 31(6):622–60.
8. Mitchell P, Hourihan F, Sandbach J, Wang JJ. The relationship between glaucoma and myopia: the Blue Mountains Eye Study. Ophthalmology. 1999 Oct; 106(10):2010–5.
9. Vongphanit J, Mitchell P, Wang JJ. Prevalence and progression of myopic retinopathy in an older population. Ophthalmology. 2002 Apr; 109(4):704–11.
10. Petty AD, Wilson G. Reducing the impact of the impending myopia epidemic in New Zealand. N Z Med J. 2018 Dec 14; 131(1487):80–5.
11. Ministry of Health. National Vision and Hearing Screening Protocols. MOH. New Zealand. Revised 2014. 34.
12. Health Workforce New Zealand. Eye Health Workforce Service Review. HWNZ. New Zealand: August 2010. 52.1.
13. Andermann A, Blancquaert I, Beauchamp S, Déry V. Revisiting Wilson and Jungner in the genomic age: a review of screening criteria over the past 40 years. Bull World Health Organ. 2008 Apr; 86(4):317–9.
14. O'Sullivan et al. Overtesting and undertesting in primary care: a systematic review and meta-analysis. BMJ Open. 2018 Feb 11; 8(2):e018557.
15. Wilson J, Jungner Y. Principles and Practice of Screening for Disease. Ann Intern Med. 1968 Nov 1; 69(5):1085.
16. Keeffe JE, Chou S-L, Lamoureux EL. The cost of care for people with impaired vision in Australia. Arch Ophthalmol. 2009 Oct; 127(10):1377–81.
17. Fricke T, Holden B, Wilson D, et al. Global cost of correcting vision impairment from uncorrected refractive error. Bull World Health Organ. 2012 Oct 1; 90(10):728–38.
18. WHO | Priority eye diseases. 2018 Apr 18 [cited 2019 Sep 2]; Available from: http://www.who.int/blindness/causes/priority/en/index4.html
19. Australia's health 2014 (full publication; 21 Jul 2014 edition) (AIHW). Available from: http://www.aihw.gov.au/getmedia/d2946c3e-9b94-413c-898c-aa5219903b8c/16507.pdf.aspx?inline=true
20. Zloto et al. Current trends among pediatric ophthalmologists to decrease myopia progression—an international perspective. Graefes Arch Clin Exp Ophthalmol. 2018 Dec 1; 256(12):2457–66.
21. Wildsoet CF, Chia A, Cho P, et al. IMI - Interventions Myopia Institute: Interventions for Controlling Myopia Onset and Progression Report. Invest Ophthalmol Vis Sci. 2019 Feb 28; 60(3):M106–31.
22. Xiong S, Sankaridurg P, Naduvilath T, et al. Time spent in outdoor activities in relation to myopia prevention and control: a meta-analysis and systematic review. Acta Ophthalmol. 2017 Sep; 95(6):551–66.
23. Tran HDM, Tran YH, Tran TD, et al. A Review of Myopia Control with Atropine. J Ocul Pharmacol Ther. 2018 Jun; 34(5):374–9.
24. Vagge A, Ferro Desideri L, Nucci P, et al. Prevention of Progression in Myopia: A Systematic Review. Diseases. 2018 Sep 30; 6(4).
25. Gong Q, Janowski M, Luo M, et al. Efficacy and Adverse Effects of Atropine in Childhood Myopia: A Meta-analysis. JAMA Ophthalmol. 2017 Jun 1; 135(6):624–30.
26. Chia A, Lu Q-S, Tan D. Five-Year Clinical Trial on Atropine for the Treatment of Myopia 2: Myopia Control with Atropine 0.01% Eyedrops. Ophthalmology. 2016 Feb; 123(2):391–9.
27. Bullimore MA, Berntsen DA. Low-Dose Atropine for Myopia Control: Considering All the Data. JAMA Ophthalmol. 2018 Mar 1; 136(3):303.
28. Yam JC, Jiang Y, Tang SM, et al. Low-Concentration Atropine for Myopia Progression (LAMP) Study: A Randomized, Double-Blinded, Placebo-Controlled Trial of 0.05%, 0.025%, and 0.01% Atropine Eye Drops in Myopia Control. Ophthalmology. 2019 Jan; 126(1):113–24.
29. Wen D, Huang J, Chen H, Bao F, et al. Efficacy and Acceptability of Orthokeratology for Slowing Myopic Progression in Children: A Systematic Review and Meta-Analysis. J Ophthalmol. 2015 Jun 11; 2015:360806.

30. Myopia guidelines [Internet]. Brien Holden Vision Institute. [cited 2019 Sep 8]. Available from: www.brienholdenvision.org/translational-research/myopia/myopia-guidelines.html

31. Gifford KL, Richdale K, Kang P, et al. IMI - Clinical management guidelines report. Invest Ophthalmol Vis Sci. 2019 Feb 28; 60(3):M184–203.

32. Pharmaceutical Management Agency Section H Update for Hospital Pharmaceuticals Effective 1 August 2018. Pharmaceutical Management Agency; August 2018: 18.

33. Payerols A, Eliaou C, Trezeguet V, et al. Accuracy of PlusOptix A09 distance refraction in pediatric myopia and hyperopia. BMC Ophthalmol. 2016 Jun 1; 16:72.

34. Yu H, Shao Y, Yuan H, Yan B. Age-determined referral criteria of myopia for large-scale vision screening. Eye Sci. 2015 Dec; 30(4):151–5.

35. Ma Y, He X, Zou H, et al. Myopia screening: combining visual acuity and noncycloplegic autorefraction. Optom Vis Sci. 2013 Dec; 90(12):1479–85.

36. Fogel-Levin M, Doron R, Wygnanski-Jaffe T, et al. A comparison of plusoptiX A12 measurements with cycloplegic refraction. J AAPOS. 2016 Aug; 20(4):310–4.

37. Cumberland PM, Bountziouka V, Rahi JS. Impact of varying the definition of myopia on estimates of prevalence and associations with risk factors: time for an approach that serves research, practice and policy. Br J Ophthalmol. 2018; 102(10):1407–12.

38. Dirani M, Shekar SN, Baird PN. Evidence of Shared Genes in Refraction and Axial Length: The Genes in Myopia (GEM) Twin Study [Internet]. Vol. 49, Investigative Opthalmology & Visual Science. 2008. p. 4336. Available from: http://dx.doi.org/10.1167/iovs.07-1516

39. Yu L, Li Z-K, Gao J-R, et al. Epidemiology, genetics and treatments for myopia. Int J Ophthalmol. 2011 Dec 18;4(6):658–69.

40. Bock GR, Widdows K. Myopia and the control of eye growth. John Wiley & Sons; 2008. 266 p.

41. Zhang S, Yang J, Reinach PS, et al. Dopamine receptor subtypes mediate opposing effects on form deprivation myopia in pigmented guinea pigs. Invest Ophthalmol Vis Sci. 2018 Sep 4; 59(11):4441–8.

42. Frick KD, Gower EW, Kempen JH, Wolff JL. Economic impact of visual impairment and blindness in the United States. Arch Ophthalmol. 2007 Apr; 125(4):544–50.

43. Zheng Y-F, Pan C-W, Chay J, et al. The economic cost of myopia in adults aged over 40 years in Singapore. Invest Ophthalmol Vis Sci. 2013 Nov 13; 54(12):7532–7.

Quality assessment of a large primary GP skin cancer service in Auckland, New Zealand

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ABSTRACT

AIM: Waitemata District Health Board has implemented a new approach to the management of skin cancers by triaging lesions to specialist-trained general practitioners (GPSI) with the aim of reducing patient wait times and treatment costs. The primary outcome was to determine positive margin rates for the GP surgeons, with secondary outcome being infection rates.

METHOD: A retrospective audit was conducted on all excisions (n=2,705) performed between 1 January 2016 and 31 December 2016 by the 13 WDHB GPSIs. Electronic patient records were accessed to review data. Each lesion was classified into benign, in-situ (pre-malignant) and malignant categories. Surgical margins were analysed for non-melanotic skin cancers (NMSC) and determined as positive, close or negative. Infection rates determined by microbiology results and prescribing information and time to treat analyses were conducted.

RESULTS: WDHB GPSIs performed 2,705 excisions, 1,887 (69.8%) of which were malignant lesions. Among the 1,486 NMSC excised, a positive surgical margin was observed in 51 (3.4%). There were 294 (10.9%) cases of infection in 2,705 excisions. Median time to treat was 31 days across all lesions. New Zealand papers from the last two decades estimate the NMSC positive margin rate among primary care physicians varies between 16–31%; most recent papers have published rates 6.8–9.5%.European publications describe positive margin rates ranging between 13.9–33.5%.

CONCLUSION: This study validates the use of surgically trained GP surgeons and shows their integral role in managing the high volume of skin cancer in New Zealand.

Non-melanoma skin cancers (NMSC) are the most commonly diagnosed malignancies worldwide. To date, there is very limited data on NMSC incidence (not recorded by the New Zealand Cancer Registry) and treatment costs in New Zealand. Late last century, New Zealand papers from 1982 and 1998 reported an incidence of 231 and 781 per 100,000 people for BCC, and 124 and 377 per 100,000 people for SCC.[1,2] In 2018, it is estimated that 229,867 keratinocytic cancers will be diagnosed in New Zealand; with an age standardised rate of 1,385 basal cell carcinomas and 522 squamous cell carcinomas per 100,000 people.[3,4] In light of this data, it is clear that NMSC is a significant and increasing burden in New Zealand. Estimates of New Zealand's overall NMSC incidence range from 1,749 to 1,906.5 per 100,000. In comparison, UK data reports NMSC incidence of 15 to 154 per 100,000. It is clear that New Zealand leads the global skin cancer epidemic, and increased resources and novel ways are required to address this.[4–6]

In an Australian retrospective audit, the total cost of NMSC treatment was projected to increase by 22% between 2010 and 2015.[7] Considering New Zealand's similarly high NMSC incidence and population characteristics, it is expected that the New Zealand healthcare sector must also increase their

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spending beyond the NZD$51.4 million spent in 2007/2008 on treating NMSC alone.[5] Therefore, it is imperative that the New Zealand health system develops treatment strategies and increase resource provision to deal with the rising numbers of NMSC diagnoses.

Likewise, melanoma is a significant source of morbidity and mortality in New Zealand. This is reflected in the incidence rate of 41.2 per 100,000 per year (Liang et al 2010), compared to the melanoma incidence rates in US, UK, Sweden and Norway ranging from 19.8 to 31.0 per 100,000 per year (Whiteman et al 2016).[8,9] This equates to an increased burden on many areas of the health sector with associated increased financial and workforce implications, especially considering the extensive burden of melanoma on New Zealand's population.

The Waitemata District Health Board (WDHB) Skin Service has implemented an innovative approach to the management of skin cancer by triaging appropriate excisions to specialist-trained general practitioners (GPSI). Overall, GPSIs possess the ability to reduce patient waiting times, workload and financial burden on secondary/tertiary care and assist in earlier detection and treatment of invasive skin lesions.

The aim of this study was to conduct a retrospective audit of the performance of WDHB GPSIs. The primary outcome was to determine their positive margin rate, with secondary outcome measures being infection rates and time to definitive treatment. We then compared these results with international standards from previously published data on general practitioners performing simple surgery.

Method

Sample

A retrospective audit was conducted on all excisions (n=2,705) performed between 1 January 2016 to 31 December 2016 by surgically trained GPs (13 general practitioners, GPSIs) under the WDHB GPSI programme. Electronic patient records were accessed via Clinical Portal to review histology reports, microbiology reports, prescribing information and clinic letters where appropriate and available.

All lesions excised during the one-year period were included in this analysis, while biopsies (incisional, punch) were excluded. If a lesion had a diagnostic excision biopsy first then re-excised at a later date, it was recorded once in our analysis for the definitive procedure (wide local excision). If a lesion was biopsied and not excised for any reason within our analysis period, it was excluded.

Benign and malignant classification

Lesions were classified as malignant or benign based on the histology report. Our criteria for a lesion to be malignant was: 1) all basal cell carcinoma (BCC), 2) invasive squamous cell carcinoma (SCC), 3) melanoma in situ (MIS) or invasive melanoma (MM), 4) scars from a previously excised malignant lesion which met WLE criteria for the index lesion, 5) other clinically malignant lesions, eg, Merkel cell carcinoma (MCC), rare cutaneous malignancies (RCM) etc. A separate category of 'in situ' lesions was recorded for SCC in situ (Bowen's disease). All other lesions were classified as benign (lesions that are indolent, pre-malignant and/or confined to the epidermis, not including MIS) or non-invasive such as: Benign naevi, actinic keratoses, seborrheic keratoses, solar lentigo, keratoacanthoma, haemangioma, dermatofibroma and neurofibroma. A breakdown for this is available in Table 1.

Margins

Non-melanoma skin cancers (n=1,486), not including SCC in situ, were evaluated for completeness of excision in our analysis. Margins were not assessed for invasive melanoma and melanoma in situ as excisions would either be an excisional biopsy, for which a close or positive margin should not be a negative outcome, or a wide local excision, where acceptable margins differ to NMSC. Margins were not assessed for benign lesions.

Margins for NMSCs were assessed according to the following criteria: 'incomplete excision' as containing a positive margin along any border of the excision; and 'complete excision' containing all margins of healthy tissue around the lesion. 'Complete excisions' were further quantified into 'closely excised lesions' which contained <1.0mm of healthy tissue on either deep or radial margin(s), and 'definitively excised lesions' which contained >1.0mm of healthy

tissue on all margins around the excised lesion. This is the margin criteria reported by McLaughlin et al (2017).[10]

Infection rate

Post-operative infections were a secondary outcome measure in this analysis. Patient clinic follow-up notes were not available on the hospital database, so proxy measures were used to estimate post-operative infection rate: 1) if an antibiotic was prescribed between 3–31 days after the lesion was excised, and/or 2) if wound swab showed pathologic bacterial growth with concurrent antibiotics being prescribed; a post-operative infection was diagnosed. The prescribed antibiotics considered consistent with a skin infection were: flucloxacillin, amoxicillin, amoxicillin with potassium clavulanate, ciprofloxacin, co-trimoxazole, cephalexin monohydrate, cefaclor monohydrate and doxycycline. Finally, if a swab grew no bacteria but the practitioner prescribed antibiotics, this was not considered an infection as we believe that a significant postoperative wound infection would have a positive wound swab result.

Time to treat

Time to treat was defined as the number of days from when the lesion was registered with the WDHB Skin Cancer Service to the date of surgery.

Results

During the period 1 January 2016 to 31 December 2016, the 13 WDHB GPSIs excised a total of 2,705 lesions on 1,643 patients.

Types of lesions removed

Basal cell carcinomas (all types) were the most frequently excised lesion, making up a total of 1,173 (42.3%) lesions. Please refer to Table 1 for a full breakdown.

Total number of histological lesions exceeds number of excisions performed by GP surgeons because 63 excisions contained two histologically discrete lesions and one excision contained three histologically discrete lesions; margins were analysed for each discrete lesion by the same criteria as other excisions.

The four 'other malignant lesion' category included: one small lymphocytic lymphoma secondary to chronic lymphocytic leukaemia, one follicle centre lymphoma, one Merkel cell carcinoma and one pleiomorphic dermal sarcoma. The 216 'other benign lesions' were primarily: haemangioma, dermatofibromas, lichenoid keratoses and epidermal cysts.

Location of excisions

Lesions on the head and neck were the most commonly excised location with 936 (34.6%) excisions.

Figure 1: Total number of excisions by clinician.

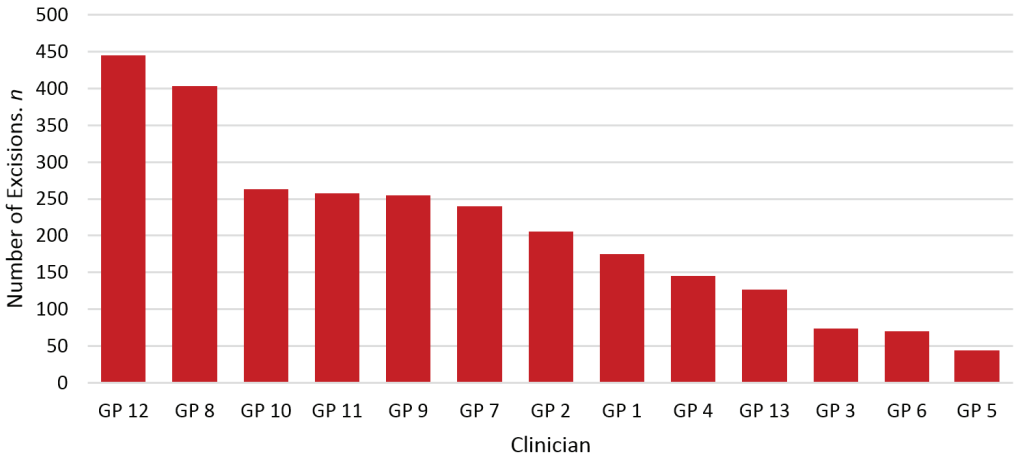

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Table 1:

Type of lesion	Count (*n*)	Percentage (%)
Malignant lesions	**1,891**	**68.3**
BCC	1,173	42.3
SCC	316	11.4
Melanoma in situ	244	8.8
Melanoma	154	5.6
Other malignant lesion	4	0.1
In-situ lesions	**178**	**6.4**
SCC in situ	178	6.4
Benign lesions	**701**	**25.3**
Actinic keratosis	125	4.5
Seborrheic keratosis	134	4.8
Keratoacanthoma	61	2.2
Naevus	165	6.0
Other benign lesion	216	7.8
Total	**2,770**	**100**

Positive margin rate of malignant lesions

During the one-year period, WDHB GPSIs removed 1,887 malignant lesions (69.8% of all excisions), of which 1,486 lesions were NMSCs. Among NMSCs, an incomplete surgical margin was observed in 51 (3.4%) excisions and 84 cases (5.7%) had margins that were clear but closely excised (<1.0mm margin). If we accept 'closely excised' margins as complete, the clear margin rate is 96.6%. If we do not accept 'closely excised' margins as complete, then the clear margin rate becomes 90.9%.

The effect of lesion location was analysed; showing the head/neck region to have the highest positive margin rate of 5.3% across the excision of 1,486 lesions.

Figure 2: Location of lesions removed.

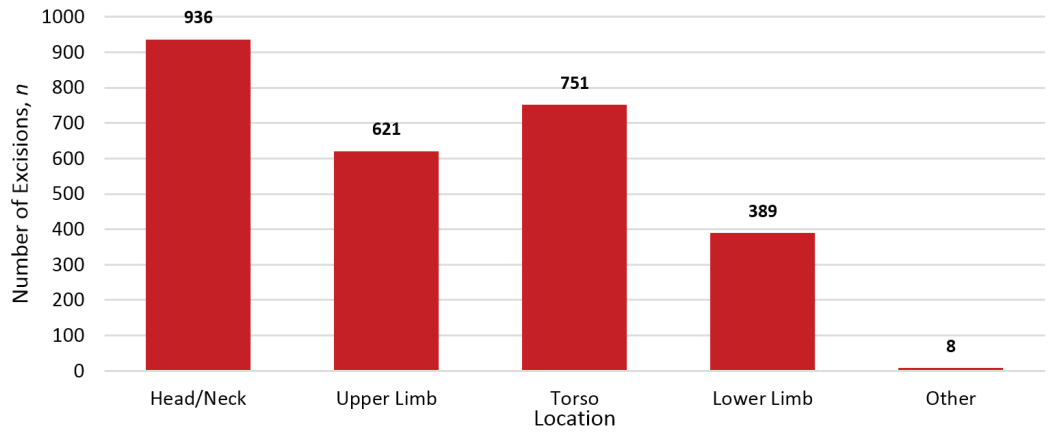


Figure 3: Positive margin rate of NMSC.

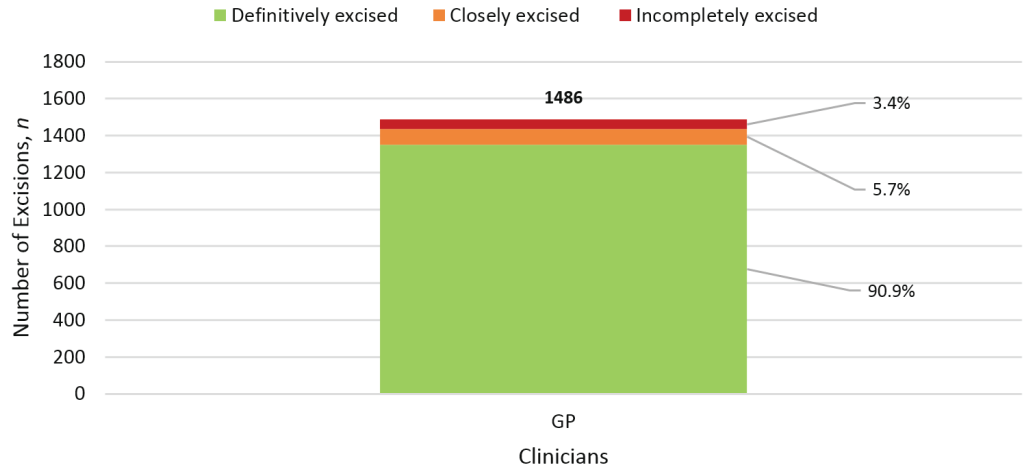


Figure 4: Positive margin rate of NMSC by location.

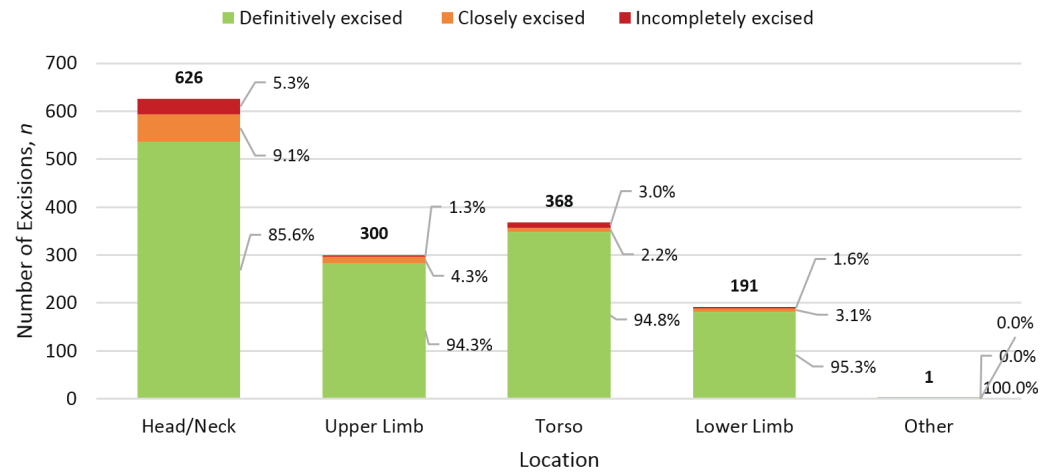
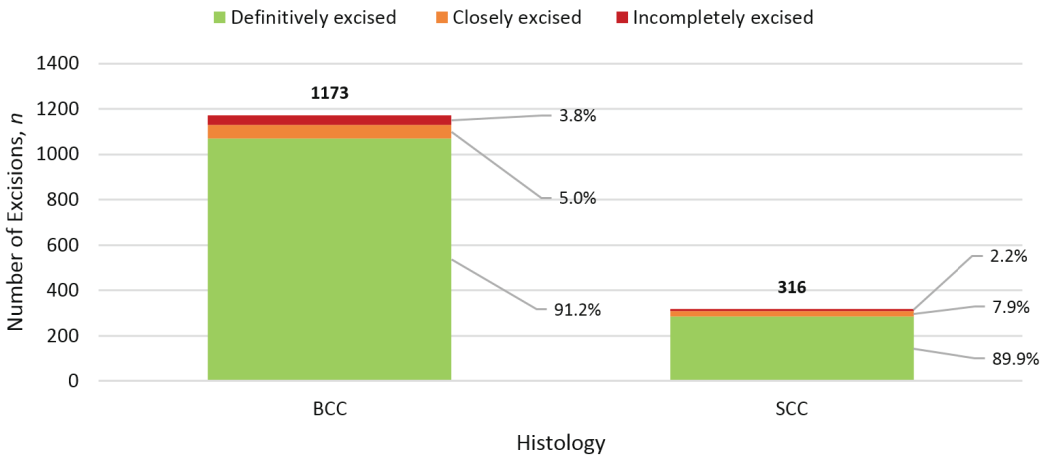


Figure 5: Positive margin rate by histology.



Re-excision rates

There were a recorded 46 lesions which required re-excision due to inadequate margins. This equates to a re-excision rate of 1.7%.

Infection rates

There were a total of 294 (10.9%) cases of infection among all 13 GPSIs within the one-year period. The average and median time to evidence of an infection (ie, prescription of antibiotics) were 11.7 days and 10 days respectively. There are no clinic reports or follow up letters available for the WDHB GPSI clinics and therefore we cannot determine if there were other complications but WDHB is unaware of any serious complications or complaints.

Time to treat

Across all excisions, median time to treat was 31 days and the average time to treat was 36 days. For Priority 1 (P1) lesions, median and average time to treat were 22 and 27 days respectively. For Priority 2 (P2) lesions, median and average time to treat were 34 and 39 days respectively. A further breakdown of time to treat is available in Table 2.

Discussion

In 2016, there were a total of 2,705 excisions performed by 13 GPSIs under the WDHB Skin Service. Of these lesions, 1,887 were malignant lesions making up 70% of all excisions. Across the excision of 1,486 NMSCs, the WDHB GPSIs achieved a clearance rate of 96.6%. With regards to infection, the GPSIs had 294 (10.9%) cases of infection over all 2,705 excisions within our study period, with an average time to antibiotic prescription of 11.7 days. Median time to treat was 31 days for all lesions and median time to treat was 22 days for Priority 1 (P1) lesions.

This study is one of the largest retrospective audits to date observing the performance of GPs in the management of cutaneous malignancies. This review is, to our knowledge, the first that investigates the performance of primary care doctors following specialist surgical training and will assist in validating the efficacy of such practitioners in the management of skin cancer.

Historically, the performance of GPs in New Zealand excising cutaneous malignancies has been poorly documented. A study 20 years ago determined the NMSC excision positive margin rate among GPs in New Zealand to be 31%, while another study 14 years ago from the Bay of Plenty observed a GP-positive margin rate of 16%.[11,12]

Consequently, we believe the best New Zealand standard to compare the WDHB GPSIs performance against is Counties Manukau District Health Board, Auckland, New Zealand's plastic surgical unit data (McLaughlin et al 2017); their paper described consultant surgeons and surgical registrars achieving a positive margin rate of 6.8–9.5% over 275 local anaesthetic procedures—for benign and malignant lesions (BCC, SCC, Bowen's disease, keratoacanthoma, melanoma, melanoma in situ, Kaposi sarcoma, actinic keratosis and other benign lesions).[10] Additionally, Elliott et al (2018) observed a 9.5% positive margin rate among GPs in Northland, New Zealand for the treatment of cutaneous squamous cell carcinomas.[13] Other published data originating from the UK and Netherlands describes variable GP NMSC excision rates, ranging from a 13.9% to 33.5% positive

Table 2:

Type of lesion	Median time to treat (days)	Average time to treat (days)
Priority 1 (P1) Lesions	**22 (1–97)**	**27**
Melanoma	20 (2–97)	24
Melanoma in situ	23 (1–97)	28
Priority 2 (P2) Lesions	**34 (2–119)**	**39**
BCC	35 (2–119)	40
SCC	30 (2–108)	35
Overall	**31 (1–119)**	**36**

margin rate.[14–16] Considering this data and the case mix of the WDHB GPSI, it is clear the WDHB GPSI's 96.6% negative margin rate is an impressive feat; additionally, these studies provide a baseline context to the performance of New Zealand GPs excising malignant skin lesions in the primary care setting over the past two decades. Our study demonstrates the WDHB GPSI scheme has achieved a significantly improved negative margin rate for malignant skin excisions in comparison both locally and internationally. Please refer to the Appendix for a detailed breakdown and comparison of the aforementioned publications.

A review of literature around existing primary care skin cancer protocols in New Zealand reveals a programme in CDHB that is a relevant comparator to the WDHB GPSI scheme.[17] The CDHB approach is a plastic surgeon-led see and treat clinic-based programme with the aim of increasing overall cutaneous surgical skills among all GPs in the region through six half-day sessions, compared to the WDHB scheme, which focuses on upskilling a focused group of GPs over a six-month training period in skin cancer surgery. At the time of the publication, the CDHB program had been active for six years and their complete excision rate was 92% and 94% across malignant and all lesions respectively, however they did not include a definition for 'complete excision' and a definition for 'malignant lesion'. The WDHB GPSI achieved a complete excision rate/clear margin rate of 96.6% across all NMSC excisions. McGeoch et al also displayed their benign to malignant ratio in the form of a graph, however no data table was available to reference absolute figures; based on our interpretation of their graph, it appeared that roughly 54% of all excised lesions were malignant (estimated 1,300 malignant and total 2,400 lesions excised in 2013–2014). The data from WDHB GPSI scheme identified that 74.7% of lesions excised were malignant when including SCC in situ, and 68.3% of lesions excised were malignant if excluding SCC in situ.

When analysing by location, we identified that the head and neck (n=626) was the most poorly excised region with a positive margin rate of 5.3% and close margins observed in 9.1% of excisions; in comparison the positive margin rate for the torso and limbs ranged from 1.3–3.0%. This increased positive margin rate likely reflects the difficulty of excising lesions on the head and neck and the proximity of neighbouring structures, which may limit adequate margins during excisions.

The calculated re-excision rate is 1.7% (n=46). There is a small difference in re-excision rate between the five highest volume GPSI and five lowest volume GPSI; with a re-excision rate of 1.6% (26 of 1,598 lesions) and 2.7% (12 of 448 lesions) respectively. However, it should be noted that this re-excision rate reflects the number of cases re-excised by a GPSI and does not include cases that were subsequently referred to hospital for surgery due to inadequate margins. Based on protocol, all incomplete excisions and close margins were re-evaluated and a decision was made whether or not to re-excise by a specialist surgeon. Therefore, the closest estimate of our true re-excision rate is the positive margin rate of 3.4%. It should be noted that in select cases, patients may refuse surgery or a decision was made to observe/withhold surgery; therefore, the positive margin rate may overestimate our re-excision rate.

Literature review of post-operative infection among GPs reveals limited data, with one Australian study finding an 8.7% infection rate for minor skin excisions at all body sites (including melanoma, BCC, SCC and other benign lesions).[18] We identified a greater infection rate (11.9%) among WDHB GPSI, however our measures are likely to be an overestimation considering the criteria for an infection may include non-excision related infections and variable antibiotic prescription practices. A proxy measure for infection rate was used as no clinical information on the wounds were available to review in conjunction with antibiotic prescribing information. Aside from returning to theatre for positive surgical margins, we were unable to determine if any excisions required a revision due to post-operative infection and/or other complications due to lack of clinical follow-up information in the GP rooms.

Patient selection is an important consideration for GP management versus specialist management of skin cancers. For the WDHB Skin Service, lesions are triaged by consultant surgeons at WDHB through an e-referral system with attached photos of lesions from general practitioners.

The consultant surgeon will decide if dermatoscopy imaging is required for the pigmented lesions, if the lesion needs to be excised at hospital by a surgeon, or if the lesion can be excised by a GPSI. If the latter applies, an e-referral is sent to one of the GPSI doctors and the patient receives a letter notifying them of this.

The outstanding key performance indicators (KPIs) of our skin cancer program indicated by this study are the result of GP training and close mentor supervision. WDHB GPSIs have completed College-accredited postgraduate skin cancer surgery and dermatoscopy courses and spend six months of in-house training with two specialist cutaneous oncology surgeons. GPSI operated in a variety of settings including hospital operating theatres as well as procedural rooms in their own practices; upon completing their training, GPSI operate nearly full-time in their own facilities. All GP facilities are visited, logbooks are audited every six months and signed off as acceptable best practice (including sterilisation, lighting, electrocautery, facility, AED etc) to monitor KPIs. GPSIs are credentialed for simple excisions and more complex grafts and flaps depending on their skill level.

In 2018 alone, 229,867 keratinocytic cancers were estimated to be diagnosed in New Zealand and, considering New Zealand's rapidly growing and ageing population, it is reasonable to assume the number of NMSCs treated will increase annually in the next decade.[3,19] Most recent data from the New Zealand Cancer Registry recorded an age-standardised melanoma registration rate of 35.1 per 100,000 in 2017.[20] In addition, although the invasive melanoma incidence in New Zealand appears to be plateauing for the last two decades, WDHB specific age-standardised melanoma rates increased by 14% from 44.2 per 100,000 in 1995–1999 to 50.2 per 100,000 in 2000–2004.[8,21] In spite of this data, these melanoma incidence rates are in fact likely to be underestimations of the true incidence of melanoma within at-risk populations, as Māori were estimated to have a melanoma incidence of 2.3 per 100,000 in 2000–2004.[22] Compounded with changes to private healthcare policies and veteran affairs limiting skin cancer claims, more patients are being driven into the public sector inflating the problem of managing enormous skin cancer volumes. Consequently, the funding bodies in New Zealand must ensure adequate financial support and planning for the management of skin cancer. This requires a multi-disciplinary collaborative approach to skin cancer management, including dermatologists, surgeons and general practitioners.

WDHB GP surgeons achieved a negative margin rate of 96.6% in 2016 for the excision of NMSC. This is in accordance with international guidelines that expect a 95% clearance rate for NMSC excision.[23–25] GP surgeons reduce the length of time patients wait before having their lesions assessed and/or excised; median time to treat for Priority 1 lesions is 22 days and overall median time to treat is 31 days. FY2017 WDHB Skin Service data demonstrates workload and cost per case figures that showed an 88% cost reduction per case to WDHB over a large caseload and therefore supports the involvement of GPSI in skin cancer management. It is important to consider that the cases managed at the secondary/tertiary centres are more complex and resource intensive; however, this does not discredit the cost-effectiveness of GPSI at managing large volumes of simple cutaneous malignancies. Overall, the WDHB GPSIs improve the efficiency of our skin cancer service and assist in the treatment of cutaneous malignancies in a timely and cost effective manner.

Our data supports the implementation of GPSIs as part of New Zealand's skin cancer workforce, where they will be an integral part of the multi-disciplinary team managing cutaneous malignancies. The WDHB GPSI scheme has become a reliable, efficient and safe resource for the management of malignant skin lesions in our community with a negative margin rate well below acceptable international guideline standards.

Conclusion

This study validates the safe use of GP surgeons and shows their integral role in managing the enormous volume of skin cancer in New Zealand. This data would suggest that all district health boards in New Zealand should allocate resources to and utilise GPs in the management of skin cancer.

Appendix

Reference	Author, year of publication, country	Number of cases, single or multi-center	Prospective or retrospective	Specialities involved	Types of lesions excised	Excision type	Positive margin rate (%)
	WDHB general practice surgeon 2016	1,828 malignant lesions 877 benign lesions, single-centre	Retrospective (01 January 2016–31 December 2016)	13 primary care physicians with special interest in skin cancer	BCC, SCC, SCC in situ, melanoma in situ, melanoma invasive, actinic keratosis, seborrheic keratosis etc.	Primary excision, residual/ recurrent tumour excision	For malignant lesions (excluding SCC in situ) only: 66 (3.5%) true positive margin 99 (5.2%) narrow margin (<1mm free margin)
10	McLaughlin et al, 2017, New Zealand	275 lesions, single-centre	Retrospective and prospective cohorts	Consultant surgeon, senior registrar, junior registrar	BCC, SCC, Bowen's disease, keratoacanthoma, melanoma, melanoma in situ, Kaposi sarcoma, actinic keratosis, other benign lesions	Local anaesthetic outpatient theatre operations (direct closure, split skin grafts, full thickness skin grafts, flaps)	Retrospective Cohort: 13 (6.8%) true positive margin; 21 (11.0%) narrow margin <1.0mm free margin Prospective Cohort: 8 (9.5%) true positive margin; 11 (13.1%) narrow margin <1.0mm free margin
11	Corwin et al, 1997, New Zealand	303 lesions, single-centre	Retrospective	28 general practitioners	BCC, SCC, melanoma, cutaneous lymphoma	-	Overall: 19 (31.1%)
12	Talbot and Hitchcock, 2004, New Zealand	1,833 lesions, single-centre	Retrospective (01 January 2001–30 June 2001)	General practitioners, specialists (general surgeons, otorhinolaryngologists, plastic surgeons, dermatologists), registrars	BCC, SCC, basosquamous carcinomas	Primary excision	GP: 163 (16.3%) Specialist: 84 (12.2%) Registrar: 10 (8.1%)
13	Elliott et al, 2018, New Zealand	819 lesions, single-centre	Retrospective (01 January 2015–31 December 2015)	Primary care (52.9%), secondary care (38.2%) and private specialist (8.9%) *22% lesions excised in secondary care were performed by GPwSI	cSCC	Elliptical excision, skin flap, partial and full thickness skin graft	Overall: 78 (9.5%)
14	Ramdas et al, 2018, Netherlands	2,986 lesions, multi-centre	Retrospective (2008–2014)	231 general practitioners 22 dermatologists 22 plastic surgeons	Primary BCC	Conventional excision of primary BCC	GP: 282 (30%) Dermatologists: 69 (6.8%) Plastic surgeons: 173 (16.6%)
15	Macbeth et al, 2009, UK	1,419 lesions, multi-centre	Retrospective (2005–2008)	Dermatologists, primary care physicians, other secondary care	BCC	-	Primary Care: 85 (33.5%) Dermatologists: 91 (9.5%) Other secondary care: 18 (8.6%)
16	Delaney et al, 2012, UK	1,184 lesions, single-centre	Retrospective (01 January 2005–31 December 2005)	General practitioner, dermatologist, plastic surgeon, other hospital specialist	SCC	Excisional, incisional and punch biopsy. Margin analysis included excisional biopsies only	GP: 30 (13.9%) Dermatologist: 10 (13.5%) Plastic surgeon: 57 (12.7%) Other: 46 (28.8%)

Competing interests:
Dr Wen reports grants from Waitemata District Health Board during the conduct of the study.

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REFERENCES:

1. Freeman NR, Fairbrother GE, Rose RJ. Survey of skin cancer incidence in the Hamilton area. The New Zealand Medical Journal, 1982; 95(713):529–533.
2. O'Dea D. A Report to the Cancer Society. The Costs of Skin Cancer to New Zealand: Wellington School of Medicine, University of Otago. 2000.
3. Sneyd MJ, Gray A. Expected non melanoma skin (Keratinocytic) cancer incidence in New Zealand for 2018. Wellington: Health Promotion Agency. 2018
4. Pondicherry A, Martin R, Meredith I, et al. The burden of non-melanoma skin cancers in Auckland, New Zealand. Australasian Journal Of Dermatology, 2018; 59(3):210–213.
5. O'Dea D. The costs of skin cancer to New Zealand. The Cancer Society of New Zealand. 2009.
6. Lomas A, Leonardi-Bee J, Bath-Hextall F. A systematic review of worldwide incidence of nonmelanoma skin cancer. British Journal Of Dermatology, 2012; 166(5):1069–1080.
7. Fransen M, Karahalios A, Sharma N, et al. Non-melanoma skin cancer in Australia. The Medical Journal Of Australia, 2012; 197(10):565–568.
8. Liang J, Robinson E, Martin R. Cutaneous melanoma in New Zealand: 2000-2004. ANZ Journal Of Surgery, 2010; 80(5):312–316.
9. Whiteman D, Green A, Olsen C. The Growing Burden of Invasive Melanoma: Projections of Incidence Rates and Numbers of New Cases in Six Susceptible Populations through 2031. Journal of Investigative Dermatology, 2016; 136(6):1161–1171.
10. McLaughlin S, Kenealy J, Locke M. Effect of a See and Treat clinic on skin cancer treatment time. ANZ Journal of Surgery, 2017; 88(5):474–479.
11. Corwin P, Munn E, Nicholls D. A study of general practitioners' skin surgery in Canterbury. The New Zealand Medical Journal, 1997; 110(1047):253–255.
12. Talbot S, Hitchcock B. Incomplete primary excision of cutaneous basal and squamous cell carcinoma in the Bay of Plenty. The New Zealand Medical Journal, 2004; 117(1192):U848.
13. Elliott BM, Douglass BR, McConnell D, et al. Incidence, demographics and surgical outcomes of cutaneous squamous cell carcinoma diagnosed in Northland, New Zealand. The New Zealand Medical Journal, 2018; 131(1475):61–68.
14. Ramdas K, van Lee C, Beck S, et al. Differences in Rate of Complete Excision of Basal Cell Carcinoma by Dermatologists, Plastic Surgeons and General Practitioners: A Large Cross-Sectional Study. Dermatology, 2018; 234(3–4):86–91.
15. Macbeth A, Torley D, Casie Chetty N, et al. Audit of incomplete excision rates of basal cell carcinoma: analysis of 1972 cases from four U.K. regions. British Journal Of Dermatology, 2009; 161(3):710–712.
16. Delaney E, Duckworth L, Thompson W, et al. Excising squamous cell

carcinomas: comparing the performance of GPs, hospital skin specialists and other hospital specialists. Family Practice, 2012; 29(5):541–546.

17. McGeoch G, Sycamore M, Shand B, Simcock J. A regional programme to improve skin cancer management. Journal of Primary Health Care, 2015; 7(4):339.

18. Heal C, Buettner P, Drobetz H. Risk factors for surgical site infection after dermatological surgery. International Journal of Dermatology, 2012; 51(7):796–803.

19. MacPherson L. National Population Projections: 2016(base) – 2068. Statistics New Zealand. ISSN 1178-0584. 2016.

20. Selected cancers 2015, 2016, 2017. New Zealand Cancer Registry, Ministry of Health, New Zealand. 2019.

21. Martin R, Robinson E. Cutaneous melanoma in Caucasian New Zealanders: 1995-1999. ANZ Journal of Surgery, 2004; 74(4):233–237.

22. Hore T, Robinson E, Martin R. Malignant Melanoma Amongst Maori and New Zealand Europeans, 2000–2004. World Journal Of Surgery, 2010; 34(8):1788–1792.

23. Telfer N, Colver G, Morton C. Guidelines for the management of basal cell carcinoma. British Journal Of Dermatology, 2008; 159(1):35–48.

24. Motley R, Kersey P, Lawrence C. Multiprofessional guidelines for the management of the patient with primary cutaneous squamous cell carcinoma. British Journal Of Dermatology, 2002; 146(1):18–25.

25. Basal cell carcinoma, squamous cell carcinoma (and related lesions) - a guide to clinical management in Australia. Cancer Council Australia and Australian Cancer Network, Sydney. 2008.

Ethnic inequities in life expectancy attributable to smoking

Michael Walsh, Karen Wright

ABSTRACT

AIM: To determine the contribution of smoking-related deaths to the life expectancy gap in both Māori and Pacific people compared with non-Māori/non-Pacific people in New Zealand.

METHODS: Death registration and population data between 2013 and 2015 were used to calculate life expectancy. To determine the contribution of smoking to the life expectancy gap, population attributable fractions for all causes of death where smoking is a casual risk factor were calculated using age- and ethnic-specific smoking data from the 2013 New Zealand Census and relative risk estimates from the American Cancer Society Cancer Prevention Study II. Population attributable fractions were applied to all deaths registered in New Zealand for the 2013–15 period to estimate the number of deaths attributable to tobacco smoking. The life expectancy gap was decomposed using the Arriaga method. The gap was decomposed both overall and by specific smoking attributable causes of death.

RESULTS: Between 2013 and 2015 an estimated 12,421 (13.4% of all deaths) were attributable to smoking. Nearly one in four (22.6%) deaths among Māori were attributable to smoking (2,199 out of 9,717 deaths) and nearly one in seven (13.8%) among Pacific people (512 out of 3,720 deaths). Among non-Māori/non-Pacific people, one in eight (12.3%) deaths were attributable to smoking (9,710 out of 78,759 deaths). Higher rates of smoking attributable mortality were responsible for 2.1 years of the life expectancy gap in Māori men, 2.3 years in Māori women, 1.4 years in Pacific men and 0.3 years among Pacific women. Cancers of the trachea, bronchus and lung, chronic obstructive pulmonary disease (COPD) and ischaemic heart disease were the leading smoking attributable causes of death contributing to the gap.

CONCLUSION: Smoking is an important preventable risk factor contributing to ethnic inequities in life expectancy for Māori men and women, and Pacific men. Dramatic declines in smoking-attributable deaths can be achieved by reducing smoking prevalence rates. Preventing smoking initiation and increasing cessation rates must remain a top priority for the Ministry of Health and District Health Boards. Smokefree initiatives should be reoriented to be Tiriti o Waitangi (Treaty of Waitangi) compliant and better meet the needs of Māori and Pacific people who smoke. Addressing the residual risk in ex-smokers through equitable early diagnosis and treatment of smoking-related conditions will further assist a more rapid closing of life expectancy gaps for Māori men and women and Pacific men. The next five years provide the opportunity to demonstrate commitment to achieving a smokefree Aotearoa for all: an aspiration, based on the current trajectory, which is most probably out of reach.

There are large and persistent inequities in mortality and life expectancy among Māori and Pacific when compared with non-Māori/non-Pacific people, in New Zealand. Life expectancy at birth, an easily understandable summary measure of population health, is reflective of current mortality across different age groups and allows comparison of groups with different population structures. In 2013–15, life expectancy differentials were approximately seven years for Māori (6.8 years in women, 7.3 years in men) and more than five years for Pacific peoples (5.2 years in women, 5.8 years in men) when compared to the non-Māori/non-Pacific population.[1]

Smoking is recognised as one of the most significant risk factors contributing to non-communicable diseases.[2] It is strongly linked to lung cancer and confers increased risk of death from other cancers, heart

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disease, stroke, chronic respiratory disease and a number of other conditions.[3] Tobacco smoking in New Zealand has previously been estimated to result in the deaths of between 4,500 and 5,000 individuals per year.[4]

Although the daily smoking prevalence in New Zealand adults has steadily declined,[5] an ethnic gradient exists, with the prevalence of smoking nearly three times higher among Māori and one and a half times higher among Pacific compared with non-Māori/non-Pacific people. Despite a declining prevalence of the past 10 years, the prevalence for Māori women remains persistently higher than Māori men (35% vs 27%). Trends for Pacific men and women have not changed significantly between 2006/07 and 2017/18 and are higher in men compared to women (25% vs 15%). More recently, some of the largest total population declines have occurred among the 15–34 year age group, particularly among those aged 15–17 years. This suggests that declining initiation rates in youth are contributing towards a reduction in smoking prevalence.

Consistent with persistent disparities in smoking prevalence, Māori and Pacific peoples are disproportionately burdened by smoking-related morbidity and mortality.[6] There is a large body of evidence indicating that tobacco is likely the leading contributor to health inequities among Māori and Pacific populations,[7–9] reflecting the unfair and unjust distribution of social, environmental and economic determinants of health. Both Māori and non-Māori current-smokers have been shown to have a lower life expectancy compared with non-smokers. The gap between current- and never-smokers, however, is smaller among Māori than among non-Māori (4.3 and 3.9 years among Māori men and women respectively, compared to 7.4 and 6.2 years among non-Māori) and has been attributed to the much higher background mortality among Māori never-smokers compared to non-Māori never-smokers.[10]

Understanding the impact of tobacco smoking and the contribution to ethnic inequities is necessary to inform policies and strategies for fair and just tobacco control as well as health service planning. In this study we use recent mortality and smoking prevalence data to provide updated estimates of its contribution to all-cause mortality, and of smoking-related mortality to the life expectancy gap in both Māori and Pacific ethnic groups compared with non-Māori/non-Pacific ethnic groups in New Zealand. We also decompose the life expectancy gap by specific smoking attributable causes of death.

Method

Smoking prevalence

Age-, ethnic- and sex-specific smoking prevalence was calculated using total response data from the 2013 Census.[11] Ethnicity in New Zealand is self-identified. Multiple ethnicities were prioritised to Māori, Pacific, Asian and European/Other as per standard protocols for health research.[12]

Estimating the number of smoking attributable deaths

We applied population-attributable fraction (PAF) methods that combine summary measures of prevalence, relative risk and mortality.[13] Population-attributable fraction estimates the proportion of disease that can be attributed to a particular risk factor. PAF is derived mathematically by combining the prevalence of the risk factor (Pc—current smokers and Pf—ex-smokers) in the target population with a measure of association between the risk factor and burden (Rc—current smokers, Rf—ex-smokers), obtained from epidemiological studies.

Consistent with previous international studies of smoking related mortality, we used the American Cancer Society Cancer Prevention Study II (CPS-II) relative risk estimates for smoking related diseases (Table 1).[14] The CPS-II is an ongoing prospective study of 1,185,106 residents in the US, aged 30 years or over, for those who, in 1982, had never smoked regularly, and for those who were then current cigarette smokers. The use of relative risk estimates from international studies has previously been used to describe the burden of smoking-related disease in New Zealand.[15–17] In many ways the CPS-II estimates have unofficially taken the role of being the "gold standard" for the effect measure in estimating smoking attributable mortality.

Using the applicable ICD10 codes for identified smoking-related diseases, smoking attributable fractions for each disease

casually linked to smoking were calculated using the following formula for multiple exposure groups:

$$PAF = \frac{Pc(Rc - 1) + Pf(Rf - 1)}{1 + Pc(Rc - 1) + Pf(Rf - 1)}$$

- *PAF* is the attributable proportion for each disease attributable to smoking
- *P*c is the proportion of the population who are current smokers
- *P*f is the proportion of the population who are ex-smokers
- *R*c is the disease specific relative risk of death for current smokers
- *R*f is the disease specific relative risk of death for ex-smokers

The age-, sex- and ethnic-specific PAF for each cause were multiplied by the number of deaths in the population from each respective cause to obtain cause-specific smoking-attributable mortality estimates. For each cause of death group, smoking-attributable mortality estimates were calculated for men and women, for each ethnic group and either for all age groups or by the four age groups 35–54, 55–64, 65–74 and 75+ years if age-specific relative risk estimates were available. The proportion of smoking attributable deaths was summed to generate the total number of deaths attributable to smoking. Our estimates of smoking attributable mortality do not take into account deaths attributable to second-hand smoke or occupational exposure.

Decomposition of life expectancy

Life expectancy at birth using data for 2013–15 (three year aggregated) was calculated using standard abridged life table techniques, with 90 and older being the final age group. The impact of smoking on the differentials in life expectancy was estimated by decomposing the difference in life expectancy using the method developed by Arriaga.[18] This method allows differentials in life expectancy to be decomposed into age group and cause-of-death-specific contributions. Each sums to the total differential in life expectancy between groups. Both the total sum of smoking attributable mortality and cause specific smoking attributable mortality were decomposed.

Life tables for Māori men, Māori women, Pacific men, Pacific women, non-Māori/non-Pacific men and non-Māori/non-Pacific women were developed. The sex-specific non-Māori/non-Pacific tables served as the comparator group for the Māori and Pacific sex-specific tables. The contribution of smoking attributable mortality on life expectancy differentials in both Māori and Pacific compared with non-Māori/non-Pacific was analysed. The gap in life expectancy was decomposed both by the sum of all smoking attributable deaths and by the sum of smoking attributable deaths by specific cause.

Results

Total smoking attributable deaths

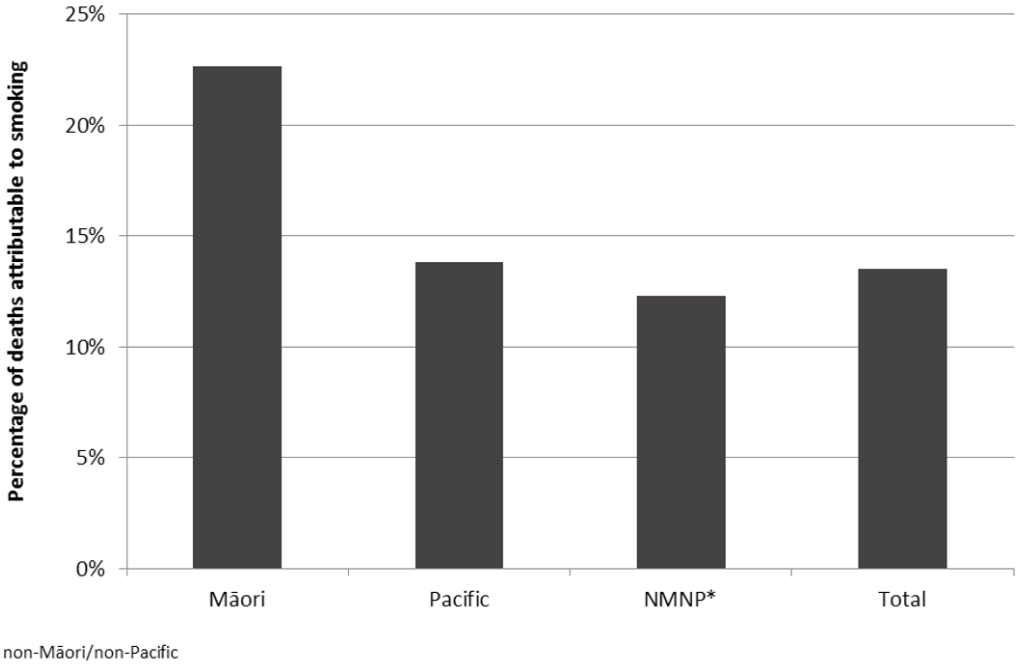
Between 2013 and 2015 there were 92,196 deaths registered in New Zealand. Of these deaths we estimated that 12,421 (13.4% of all deaths) or nearly one in seven were attributable to smoking. The proportion of smoking attributable deaths varied by ethnicity (Figure 1) with nearly one in four (22.6%) deaths among Māori attributable to smoking (2,199 out of 9,717 deaths) and nearly one in seven (13.8%) among Pacific People (512 out of 3,720 deaths). Among non-Māori/non-Pacific people one in eight (12.3%) deaths were attributable to smoking (9,710 out of 78,759).

Overall, 30.7% of smoking attributable deaths were from trachea, bronchus and lung cancers followed by COPD (26.0%) and ischaemic heart disease (13.2%). Among Māori 37.5% of smoking attributable deaths were from trachea, bronchus and lung cancers compared with 29.5% in Pacific.

Life expectancy at birth

For all deaths registered in 2013–15, estimates of life expectancy were: 73.5 years in Māori men, 77.3 years in Māori women, 75.0 years in Pacific men and 78.3 years in Pacific women. In comparison, life expectancy was 80.9 years in non-Māori/non-Pacific men and 84.3 years in non-Māori/non-Pacific women. These results equated to differentials in life expectancy of 7.4 years in Māori men, 7.0 years in Māori women, 5.9 years in Pacific men, and 6.0 years in Pacific women.

Figure 1: Percentage of deaths attributable to smoking by ethnicity, 2013–2015.



Contribution of smoking to the life expectancy gap—Māori

Among Māori men, 2.1 years (28.4%) of the 7.4 year gap in life expectancy was attributable to the higher mortality rates from smoking attributable deaths. Among Māori women, the contribution from smoking attributable deaths was 2.3 years (32.9%) of the 7.0 year gap (Table 1).

The leading smoking attributable causes of death contributing to the life expectancy gap were cancers of the trachea, bronchus and lung, contributing nearly one full year to the gap among Māori women (Figure 2) and 0.8 years among Māori men (Figure 3). This was followed in Māori men by smoking attributable ischaemic heart disease (0.5 years of the gap) and COPD (0.3 years of the gap). Among Māori women COPD was the second leading smoking attributable cause of death (0.5 years of the gap) followed by smoking attributable ischaemic heart disease (0.3 years of the gap).

Table 1: Contribution of smoking attributable mortality to the life expectancy gap, 2013–15.

	Men				**Women**			
	Smoking attributable		**Non-smoking attributable**		**Smoking attributable**		**Non-smoking attributable**	
	Māori	**Pacific**	**Māori**	**Pacific**	**Māori**	**Pacific**	**Māori**	**Pacific**
0–29 years	0.0	0.0	0.8	0.5	0.0	0.0	0.5	0.6
30–49 years	0.3	0.3	1.0	0.6	0.2	0.1	0.7	0.6
50–74 years	1.6	0.9	2.6	2.2	1.6	0.2	2.4	3.0
75+ years	0.3	0.2	0.9	1.1	0.5	-0.1	1.1	1.5
Overall	2.1	1.4	5.2	4.5	2.3	0.3	4.7	5.7

Figure 2: Decomposition of the life expectancy gap by smoking attributable cause—Māori women.

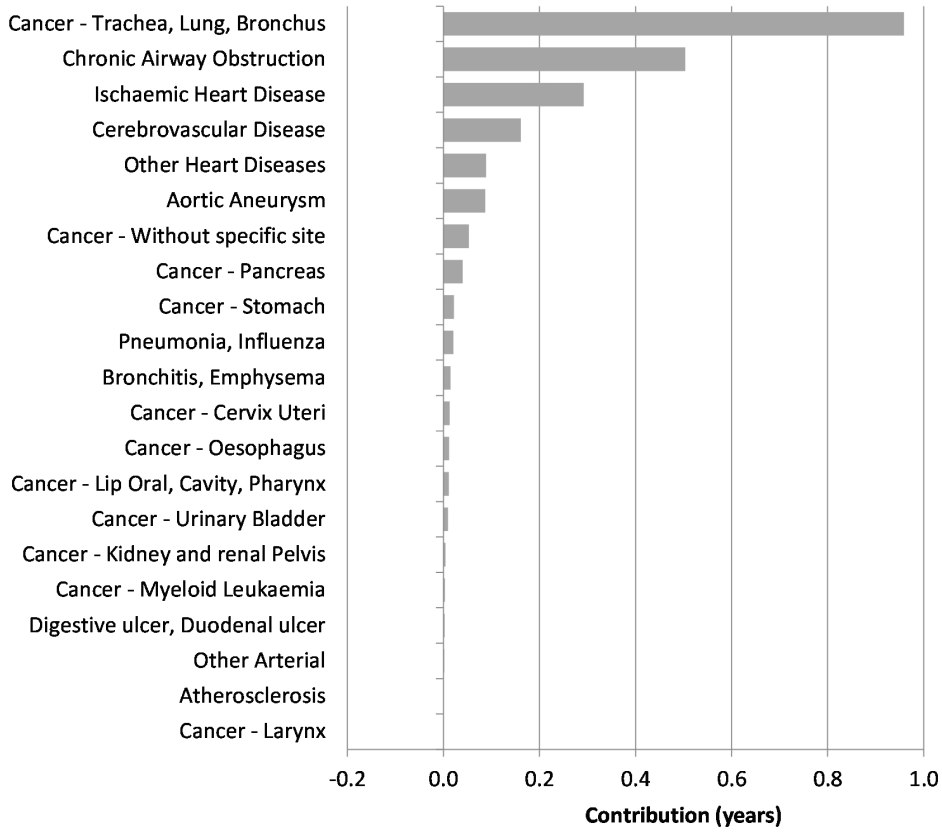


Figure 3: Decomposition of the life expectancy gap by smoking attributable cause—Māori men.

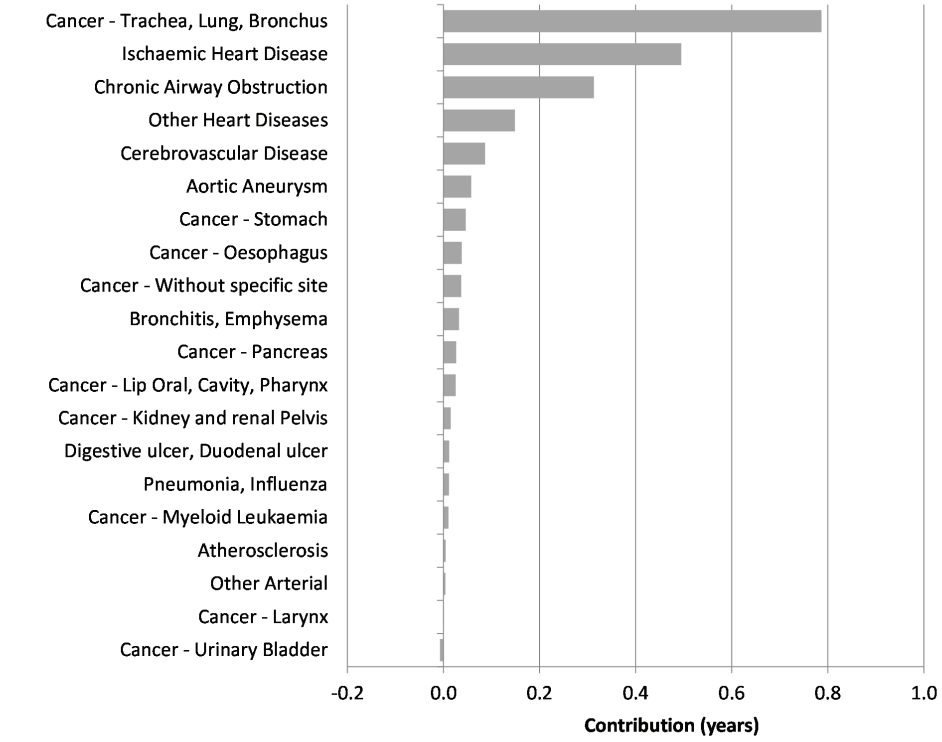


Figure 4: Decomposition of the life expectancy gap by smoking attributable cause—Pacific men.

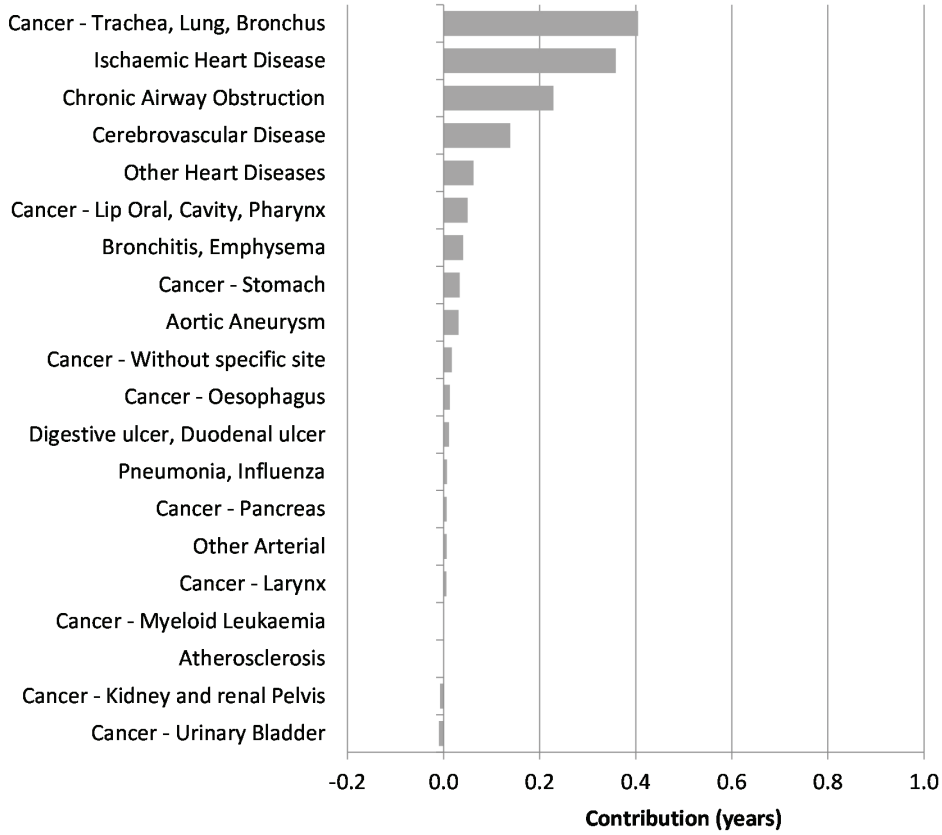
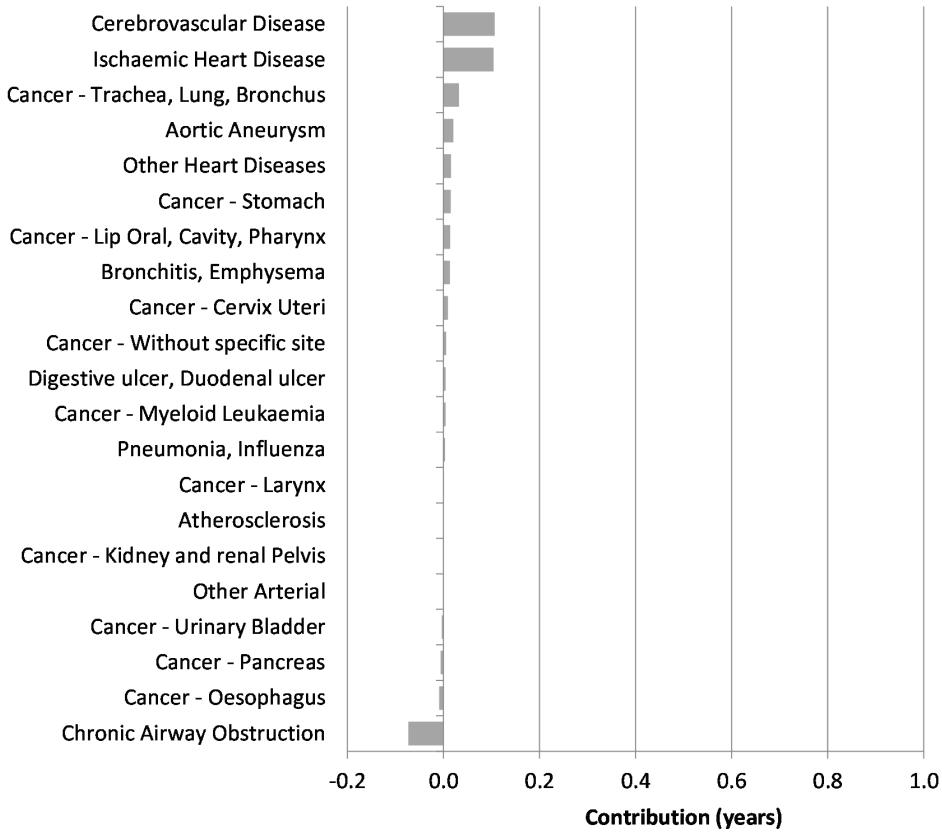


Figure 5: Decomposition of the life expectancy gap by smoking attributable cause—Pacific women.



Contribution of smoking to the life expectancy gap—Pacific

Among Pacific men 1.4 years (23.7%) of the 5.9 year gap in life expectancy was attributable to the higher mortality rates from smoking attributable causes of death. Among Pacific women, the contribution from smoking attributable causes of death was 0.3 years (5.0%) of the 6.0 year gap (Table 1).

The leading smoking attributable causes of death among Pacific men contributing to the life expectancy gap were cancers of the trachea, bronchus and lung and ischaemic heart disease, each contributing 0.4 years to the life expectancy gap (Figure 4), followed by smoking attributable COPD (0.2 years of the gap). In Pacific women cerebrovascular disease and ischaemic heart disease were the leading smoking attributable causes of death, each contributing 0.1 years to the gap (Figure 5).

Discussion

Our study provides an updated assessment of the burden of smoking-related mortality in New Zealand for deaths registered from 2013 to 2015 and examines the contribution of these deaths to the life expectancy gap in the Māori and Pacific populations compared with the non-Māori/non-Pacific population. Over this time, 13.4% of deaths were potentially attributable to smoking. Among Māori, 22.6% of deaths were potentially attributable to smoking, compared with 13.8% of deaths among Pacific peoples and 12.3% among non-Māori/non-Pacific people. We estimate that smoking attributable deaths contributed 2.1 years to the life expectancy gap in Māori men, 2.3 years in Māori women, 1.4 years of Pacific men and 0.3 years in Pacific women.

Smoking attributable deaths and the contribution to inequities in life expectancy have been previously described, albeit using differing methods. For the period 1996–1999, Blakely et al found that current smokers had an estimated 3.9–7.4 years less of life expectancy relative to never-smokers.[10] The smoking attributable difference in life expectancy was smaller among Māori than among non-Māori. The Ministry of Health has also estimated the burden of smoking using similar methods to this study to estimate the number of smoking attributable deaths and the contribution of smoking to ethnic inequities in life expectancy.[15] Using the life expectancy of the Europeans in the lowest level of deprivation (most socio-economically advantaged) with smoking deaths removed as a comparator, the report showed that 3.5 years of the life expectancy gap in Māori men and 2.4 years in Māori women was attributable to tobacco. Among Pacific peoples this gap was 3.6 years in men and 0.9 years in women. The contributions of tobacco in both studies were larger than those we present. This difference is primarily due to the reference group used. In the prior studies, the reference group was either life expectancy with the smoking attributable deaths removed or the life expectancy of never smokers. The reference group used in our study was the non-Māori/non-Pacific population without smoking deaths removed. As such, our estimates account for the smoking attributable deaths in the non-Māori/non-Pacific population and can be interpreted as the gap in life expectancy due to the difference in smoking attributable deaths in the Māori and Pacific populations compared with the non-Māori/non-Pacific population.

We found a smaller contribution of smoking to the life expectancy gap in Pacific men (1.4 years) and women (0.3 years) compared to Māori. This likely reflects the lower prevalence of smoking overall, lower prevalence in older age groups and that Pacific people are less likely to be daily smokers when compared with Māori.[5] For Māori the additional burden of smoking attributable mortality could be a result of a greater intensity of exposure to second-hand smoke when compared to other ethnicities. This likely comes about as a result of the higher density of Māori who smoke both at home and in the workplace.[19] Another explanation is that other risk factors, such as excess body weight, are potentially greater contributors to mortality and the life expectancy gap among the Pacific population. It has been previously shown that diseases related to excess body weight, such as diabetes and uterine cancers, are among the leading contributors to the life expectancy gap among the Pacific population.[1]

Drivers of inequity

Factors contributing to the pervasive and persisting ethnic health inequities are multifaceted and complex. Three main pathways have been identified: (i) differential access to the determinants of health or exposures leading to differences in disease incidence, (ii) differential access to healthcare and (iii) differences in quality of care received.[20] These pathways are driven by different levels of racism, particularly institutionalised and personally mediated or interpersonal racism.[21]

Similar to other indigenous populations, the colonisation of New Zealand severely impacted Māori and resulted in social marginalisation and poor health outcomes through the redistribution of Māori power and resources.[22] Historical and ongoing colonisation processes have led to the unequal distribution of resources, such as education, employment and income, which shape exposure to risk factors, such as smoking, for potentially avoidable diseases and mortality. In addition, tobacco was a common trade commodity between Māori and non-Māori in the 1800s, contributing towards widespread uptake among Māori.[23]

Persisting and pervasive health inequities are a breach of Te Tiriti o Waitangi. The recent Waitangi Tribunal Report on Stage One of the Health Service and Outcomes Kaupapa Inquiry identified Tiriti non-compliance across multiple areas of primary healthcare and systems that are relevant to smoking and smoking-related burden of disease in Māori.[24] Tobacco is also a focus area in stage two of the Waitangi Tribunal Health Services and Outcomes Inquiry. The inquiry will hear claims concerning grievances relating to health services and outcomes of national significance.[25]

Smokefree 2025

In 2011 the Government adopted the Smokefree Aotearoa 2025 goal in response to the landmark Parliamentary inquiry by the Māori Affairs Select Committee.[26] The goal aspires to achieve a daily smoking prevalence of less than five percent by 2025. Although wide-ranging interventions have been introduced since 2011 (including tobacco taxation, cessation support, mass media campaigns, smoke free environment legislation, and plain packaging and health warnings), a national strategy for achieving Smokefree Aotearoa is absent.

A business-as-usual approach to tobacco control is insufficient to meet the needs of Māori and Pacific peoples. Current trends, along with modelling studies, suggest that New Zealand is unlikely to meet its Smokefree 2025 goal and will likely be substantially missed for Māori.[27] Wilson et al projected smoking prevalence rates in 2025 to be 17.4% for Māori and 7.2% for non-Māori. The authors suggest that to achieve the New Zealand Government's Smokefree 2025 Goal, there would need to be an additional 8,400 Māori long-term quitters per year—more than five times the current annual level. Even for non-Māori, quit rates would need to double. Wilson et al state that reaching the 2025 goal would require significantly enhanced investment in established smoking cessation services and mass media campaigns or the addition of substantive novel interventions.

Long-term residual risk in ex-smokers

Following cessation, ex-smokers carry a disease-specific residual risk of between 10 and 40 years.[28] Subsequently, the life expectancy gap as a result of smoking attributable diseases will likely persist for some time. For the leading smoking attributable condition, lung cancer, the downturn in risk occurs faster in women than men. The risk in men returns to that of never-smokers at around 40 years following cessation, whereas in women this occurs between 25 to 30 years following cessation. COPD has the slowest decline in risk in the first 25 years after cessation, whereas cardiovascular disease has the most rapid reduction. After about 10 years of smoking cessation, the risk of dying from cardiovascular disease in ex-smokers is approximately equal to that of never-smokers.[28]

With the residual risk that ex-smokers continue to carry for many years following cessation, an important component of reducing smoking attributable mortality, and thus the life expectancy gap attributable to smoking, will be managing this risk in ex-smokers. Given that the prevalence of smoking still remains high for Māori men and women and Pacific men, and that patterns of disease incidence

largely reflect historical smoking patterns,[29] it is likely that differences in smoking attributable mortality and thus the life expectancy gap attributable to smoking will persist for some time.

Strengths and limitations

Our study has several strengths. First we used national mortality data that achieves nearly complete coverage of the New Zealand population. With a well-established mortality data collection system we had access to precise numbers of ethnic, gender and cause-specific death information for PAF estimation. Second, the relative risk estimates in our study were derived from a very large-scale population-based cohort study with over 1.1 million subjects, giving reliable relative risk estimates. Third, the smoking prevalence was obtained from national census data. The Census has the advantage of reaching between 93–95% of the New Zealand adult population (aged 15+), and, as it is a census of the total population, is not subject to sampling error. However, post-enumeration surveys show that there is likely to be an undercounting of people who smoke, since Māori and Pacific peoples are over-represented in the groups most likely to be missed by the Census.

PAF methods are a useful way to theoretically quantify the burden of a particular risk factor. However, they are based on assumptions that include some level of uncertainty and likely resulted in underestimating the smoking attributable fractions and thus the number of smoking attributable deaths. Many of the issues and limitations of PAF methods stem from using and combining ecological, summary measures of exposure, outcome and relative risk, across different sources of data. Firstly, exposure mismatch can occur where the definition of exposure categories (eg, current and former smoking) across data-sources for prevalence and relative risk differ. In addition, temporal changes in smoking prevalence and in accumulation/reversibility of disease-specific risks are not accounted for, along with deaths from second-hand smoke exposure. Second, external generalisability and heterogeneity of the selected relative risk estimates due to exposure definitions and distributions between the study source and usage in this study. It is plausible that the strength of the association varies in New Zealand and may also vary by ethnic group due to different baseline risks. To the best of our knowledge no single study has been able to provide disease-specific smoking-related risk estimates for New Zealand; as such the numbers from CPS-II should only be considered an approximation.[30]

Finally, the distribution of potential confounders in the population of interest has not been accounted for as individual-level adjustment is not possible. We have also not explicitly allowed for passive/second-hand smoking. This most probably affects more Māori than non-Māori.[31] As a result, we have likely further underestimated the full impact of smoking on ethnic gaps in life expectancy attributable to smoking. Despite these limitations, the estimates provide policymakers and the public with a general understanding of the magnitude of the burden imposed on the nation, and in particular Māori and Pacific peoples, by smoking.

Conclusion

A significant proportion of the life expectancy gap for Māori men and women and Pacific men is a result of smoking attributable deaths. Addressing this inequity needs to remain a top health priority. In order to achieve health equity and the Smokefree 2025 goal, a Tiriti o Waitangi compliant response inclusive of targeted investment and expansion of Māori and Pacific tobacco control programmes is required. Should New Zealand be successful in achieving its smokefree goal, addressing the residual risk in ex-smokers through equitable early diagnosis and treatment of smoking-related morbidity, will reduce the number of smoking attributable deaths and more rapidly close the life expectancy gap, particularly for Māori. History demonstrates that without deliberate attention and commitment to achieving equitable health outcomes for Māori and Pacific peoples they will not be achieved. The next five years provide an opportunity to demonstrate commitment to achieving a smoke-free Aotearoa for all, an aspiration, based on the current trajectory, which is most probably out of reach.

Competing interests:
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REFERENCES:

1. Walsh M, Grey C. The contribution of avoidable mortality to the life expectancy gap in Māori and Pacific populations in New Zealand-a decomposition analysis. N Z Med J. 2019; 132:46–60.
2. Kontis V, Mathers CD, Rehm J, et al. Contribution of six risk factors to achieving the 25x25 non-communicable disease mortality reduction target: a modelling study. Lancet. 2014; 384:427–37.
3. U.S. Department of Health and Human Services. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2014.
4. Ministry of Health. Tobacco Trends 2008: A brief update of tobacco use in New Zealand. Wellington: Ministry of Health; 2009.
5. Ministry of Health. Annual Data Explorer 2017/18: New Zealand Health Survey [Data File]. [cited]. Available from: http://minhealthnz.shinyapps.io/nz-health-survey-2017-18-annual-data-explorer
6. Ministry of Health. The burden of disease and injury in New Zealand. Public Health Intelligence Occasional Bulletin No.1. Wellington: Ministry of Health; 2001.
7. Blakely T, Fawcett J, Hunt D, et al. What is the contribution of smoking and socioeconomic position to ethnic inequalities in mortality in New Zealand? Lancet. 2006; 368:44–52.
8. Blakely T, Disney G, Valeri L, et al. Socioeconomic and Tobacco Mediation of Ethnic Inequalities in Mortality over Time: Repeated Census-mortality Cohort Studies, 1981–2011. Epidemiology. 2018; 29:506–16.
9. Wilson N, Blakely T, Tobias M. What potential has tobacco control for reducing health inequalities? The New Zealand situation. Int J Equity Health. 2006; 5:14.
10. Blakely T, Carter K, Wilson N, et al. If nobody smoked tobacco in New Zealand from 2020 onwards, what effect would this have on ethnic inequalities in life expectancy? N Z Med J. 2010; 123:26–36.
11. Statistics New Zealand. Census 2013, cigarette smoking behaviour and ethnic group (DHB) [Customised data request]. 2013.
12. Ministry of Health. HISO 10001:2017 Ethnicity Data Protocols. [cited 1/08/2019]. Available from: http://www.health.govt.nz/system/files/documents/publications/hiso-10001-2017-ethnicity-data-protocols.pdf
13. Hanley JA. A heuristic approach to the formulas for population attributable fraction. J Epidemiol Community Health. 2001; 55:508–14.
14. The Health Consequences of Smoking-50 Years of Progress: A Report of the Surgeon General. Atlanta (GA), 2014.
15. Ministry of Health. Inhaling inequality. Tobacco's contribution to health inequality in New Zealand. Wellington: Ministry of Health; 2001.
16. Blakely T, Cobiac LJ, Cleghorn CL, et al. Health, Health Inequality, and Cost Impacts of Annual Increases in Tobacco Tax: Multistate Life Table Model-

ing in New Zealand. PLoS Med. 2015; 12:e1001856.

17. Ministry of Health. Health Loss in New Zealand 1990–2013: A report from the New Zealand Burden of Diseases, Injuries and Risk Factors Study. Wellington: Ministry of Health, 2016.
18. Arriaga EE. Measuring and explaining the change in life expectancies. Demography. 1984; 21:83–96.
19. Gillespie J, Milne K, Wilson N. Secondhand smoke in New Zealand homes and cars: exposure, attitudes, and behaviours in 2004. N Z Med J. 2005; 118:U1782.
20. Reid P, Robson B. Understanding Health Inequities. Hauora: Māori Standards of Health IV A study of the years 2000–2005. Wellington: Te Rōpū Rangahau Hauora a Eru Pōmare, 2007.
21. Jones CP. Invited commentary: "race," racism, and the practice of epidemiology. Am J Epidemiol. 2001; 154:299–304; discussion 5–6.
22. Anderson I, Crengle S, Kamaka ML, et al. Indigenous health in Australia, New Zealand, and the Pacific. Lancet. 2006; 367:1775–85.
23. Reid R, Pouwhare R. Te Taonga Mai Tawhiti: The gift from a distant place. Auckland: Niho Taniwha, 1991.
24. Hauora - Report on Stage One of the Health Services and Outcomes Kaupapa Inquiry (WAI2575). Wellington, New Zealand: The Waitangi Tribunal, 2019.
25. Health Services and Outcomes Inquiry. [cited 19 July 2019]. Available from: http://www.waitangitribunal.govt.nz/inquiries/kaupapa-inquiries/health-services-and-outcomes-inquiry/
26. New Zealand Parliament. Inquiry into the tobacco industry in Aotearoa and the consequences of tobacco use for Māori, Report of the Māori Affairs Committee. Wellington: New Zealand Parliament, 2010.
27. Wilson N, Petrovic-van der Deen FS, Edwards R, et al. Modelling the number of quitters needed to achieve New Zealand's Smokefree 2025 goal for Māori and non- Māori. N Z Med J. 2018; 131:30–7.
28. Oza S, Thun MJ, Henley SJ, et al. How many deaths are attributable to smoking in the United States? Comparison of methods for estimating smoking-attributable mortality when smoking prevalence changes. Prev Med. 2011; 52:428–33.
29. Shaw C, Blakely T, Sarfati D, et al. Varying evolution of the New Zealand lung cancer epidemic by ethnicity and socioeconomic position (1981–1999). N Z Med J. 2005; 118:U1411.
30. Hunt D, Blakely T, Woodward A, et al. The smoking-mortality association varies over time and by ethnicity in New Zealand. Int J Epidemiol. 2005; 34:1020–8.
31. Mason K. Burden of disease from second-hand smoke exposure in New Zealand. N Z Med J. 2016; 129:16–25.

Good care close to home: local health professional perspectives on how a rural hospital can contribute to the healthcare of its community

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ABSTRACT

AIM: Hokianga Health in New Zealand's far north is an established health service with a small rural hospital, serving a largely Māori community. The aim of this study was to gain insights into the wider roles of one rural hospital from the perspective of its staff.

METHOD: Eleven face-to-face semi-structured interviews were conducted with employees of Hokianga Health, eight with past and current medical practitioners, three with senior non-medical staff. Interviews were recorded and transcribed. Thematic analysis of the interviews was undertaken using the Framework Method.

RESULTS: Four main themes were identified: 'Our Context', emphasising geographical isolation; 'Continuity of Care', illustrating the role of the hospital across the primary-secondary interface; 'Navigation' of health services within and beyond Hokianga; and the concept of hospital as 'Home'.

CONCLUSION: Findings highlight the importance of geographically appropriate, as well as culturally appropriate, health services. A hospital as part of a rural health service can enhance comprehensive and continuous care for a rural community. Study findings suggest rural hospitals should be viewed and valued as their own distinct entity rather than small-scale versions of larger urban hospitals.

Geographic isolation from urban centres where specialist and diagnostic resources are concentrated has been identified as the starting point for understanding rural remote health with access to healthcare widely recognised as the major rural health issue.[1,2] With increasing distance from cities and towns, boundaries between primary and secondary healthcare necessarily become more blurred.[3–5]

Rural community hospitals, situated at the interface between primary, secondary and community care services, can contribute to enhanced access and integration of service delivery and benefit the health of rural remote populations.[6,7]

In New Zealand, rural hospitals have come a long way since the health reforms of the 1990s when the withdrawal of specialist services and closure and downsizing of rural hospitals led to an erosion of access to secondary care services for rural communities.[8] A number of rural hospitals, notably in the Southern region, that were threatened with closure during this time were taken over by local community health trusts.[10]

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There had been a tendency to think of rural health as being limited to primary care (as it is defined in an urban context).[11–13]

More recently the role of rural hospitals in the wider New Zealand health system has been acknowledged [14,15] and, with the establishment of the Rural Hospital Medicine scope of practice in 2008, rural hospitals are beginning to be recognised as a distinct entity.[15–17]

The qualitative analysis presented is part of a case study undertaken to explore how the new Rural Hospital Medicine scope of practice had affected medical practitioners and the health service at one New Zealand rural hospital. The larger study findings emphasise the critical importance of targeted rural postgraduate medical training and professional development pathways and the role of a rural hospital in the provision of acute and emergency care for its community. These findings are reported elsewhere.[18]

The aim of the research reported in this paper was to gain insights into the wider roles of a rural hospital from the perspectives of its staff.

Methods

Study setting

Hokianga, an area of 1,520km^2 on the west coast of the far north of New Zealand, has a population of around 6,500 people, 70% identifying as Māori. Hokianga, though historically and culturally rich, is today socioeconomically poor. Transport networks are well behind the standard seen elsewhere in New Zealand.[19–21]

Hokianga hospital was built on grounds gifted by various hapu o Hokianga.[22] An integrated community health service, including the hospital, has been operating there since 1941.[23] Maintaining this service has required continued adaption to policy, funding and regulatory changes and nomenclature, (elements of the Hokianga Health care model have been variously described by others over the years as "a model of socialised medicine, comprehensive care, integrated care, whanau ora, community development and integrated family health centre").[19]

Today, Hokianga Health Enterprise Trust operates as an independent community-owned organisation. Funding for primary healthcare services is provided in association with the local Primary Health Organisation while funding for the hospital is provided in association with the Northland District Health Board. All services are provided at no cost at the point of care. The hospital provides in-patient, emergency and after-hours care 24/7. There are 10 acute, 10 long stay and four maternity beds. Base hospital in Whangarei is two hours away by road and Auckland, the closest tertiary centre, four hours away by road. There are about 750 acute admissions to Hokianga hospital each year: around 80% are managed to the point of discharge and around 20% result in transfer to Whangarei or Auckland. Medical staff are employed by Hokianga Health and provide all local medical services.[21]

Sampling, data collection

Individual semi-structured interviews were conducted between October 2016 and February 2017. All participants were employees of Hokianga Health between 2006 and 2016 for a minimum of six months. With the wider research study's focus on medical scopes of practice, the majority of interviewees were purposively selected (ie, medical practitioners). The interview schedule included questions about the role of the hospital within the integrated health service (reported here) and about clinical scope and safety (reported elsewhere).[18] The topic guide varied slightly for non-medical participants. Interviews lasted 40–60 minutes and were recorded and then transcribed for analysis. Transcripts were sent to participants to check their accuracy.

Data analysis

Thematic analysis was undertaken using the framework method.[25] Analysis used an iterative process in which, after initial coding (KB, TS) an analytical framework was devised by KB and TS to facilitate the development of categories and themes. N-Vivo (version 10) was used to manage the analysis. The consolidated criteria for reporting qualitative research (COREQ) were used to structure reporting of findings.[26]

Researcher positionality

The research subject and setting was embedded in the real-life experience of the lead researcher (KB). While 'insider status' can be a research strength, measures must be taken to ensure rigor in this context.[27] In this study these measures included: explicitly acknowledging the insider status of the lead researcher in participant information and consent forms; regular team review of data collection; regular team discussion during the analysis phase; and use of a reflective diary.

Ethics approval

Ethical approval for the research was obtained from the University of Otago Human Ethics Committee (16/085).

Results

Eleven face-to-face semi-structured interviews were undertaken. Eight participants were medical practitioners. Characteristics of medical participants are shown in Table 1.

Three other participants, all holding leadership roles at Hokianga for over 10 years, were a senior manager, a senior nurse and a member of the Taumata (Māori cultural advisor). Participants were designated a number (1–11) and were referred to throughout the study by this coding (eg, P1).

Participants' accounts of the role of the hospital covered four themes: our context; continuity of care; navigation; and the concept of home.

Our context

There was discussion by all participants around the wider context of healthcare and how geographical isolation, socioeconomic deprivation and local cultural factors influenced access to and provision of healthcare. All participants argued that high-quality hospital care for people 'in their own place' was integral to the service at Hokianga:

"The effect of place as well, in terms of in-patient beds—not the really sick people that you transfer off but the ones that actually stay in the hospital which is probably, in the outside world, the part that's least understood. So, emergency transfers et cetera, there's been a good understanding of that, but in terms of having a service that's high quality, that's in Hokianga, people can stay in their own place for things that don't need to go out." (P2)

Table 1: Key characteristics of medical participants (n=8).

Characteristic	No of medical practitioners
Year of graduation	
Before 1975	2
Before 1995	3
After 2000	3
Time employed at Hokianga	
>20y	3
>10y	2
<1y	3
Capacity at Hokianga	
Permanent	5
Locum	1
Registrar	2
Place of current employment	
Hokianga	3
Retired	2
Other rural NZ	3

Although emergency care was an important aspect of the hospital's role, participants emphasised the hospital's wider scope. The presence of an in-patient facility meant the ability to manage patients locally:

"Yes—observation. The medical intervention is quite a small part of medical treatment, isn't it, often? It's about being able to observe, have a place of safety, and have a place of recovery as well." (P11)

The hospital reduced anxiety for clinicians by providing a safe and appropriate place for further clinical assessment and management with a wider collegial team:

"Really, without the backup of the hospital, with our distances, it would be too stressful to work here for anyone with any aspiration to quality. As you know, in medicine there are just too many unknowns. Just for example, you get a kid with a fever from across the harbour—what are you going to do? You just don't know how it's going to develop." (P2)

What was generally normal practice in or closer to town, for example sending a patient on to the base hospital emergency department, in many cases would not make sense in Hokianga:

"A lot of clinical scenarios need time, don't they? They need half a day or two to see which direction it's going in. So… all those people [patients referred to base hospital] might be going somewhere for nothing, for lots of things that could be managed here. Like sometimes someone just needs a couple of days of intravenous antibiotics or … pneumonia … and then they can go home again." (P10)

"There is this knife edge that people are on, and a vital role of the hospital is to enable best management of that… so the COPD people, for example—ones that are on oxygen at home; they can have an exacerbation and they get sick." (P4)

Continuity of care

Participants described the patient journey for patients and family from home to clinic to hospital and back within Hokianga:

"We know the ones we need to keep—the ones we've safely sent home because of who they're living with, and they've got support. If you have to go to XX [another hospital], they're likely to get kicked out at midnight, with no way of getting home. They don't have their family—they [the other health service] don't know where they live." (P10)

Hokianga staff responsibility did not stop when someone was discharged home from the hospital ward: *"it is still our problem"* (P10). The hospital also facilitated safe discharge home of people transitioning back from city hospitals. For instance, after a surgical intervention or time in intensive care, local knowledge ensured that the right care was wrapped around them:

"The number of times we see people discharged home from [Base hospital]… and it falls apart within 24 hours, and they're back on our doorstep because they haven't been able—it's not through any lack of competence: it's just that you're too far away to make a plan that makes any sense. So you have to get people a bit closer to home and then plan the step home, because the family haven't been involved." (P11)

From disposition decisions (admit, discharge or transfer) to end-of-life care, participants discussed the continuity of care that the hospital as part of an integrated service enabled:

"…you can travel the whole journey with your patient in your rural area." (P6)

"That's what you see, even in the patients who are less acute and less sick: they really don't want to go out. It's a huge upheaval when you send them two or three hours away from their family, from their support, to a place that they don't feel comfortable, to people that they can't communicate well with. You can see a lot of practical problems—communication—just the physical distance is a problem, sending people out of the way, and then the cultural aspect…You break that continuity of care that we do have here." (P7)

The breadth of scope included cultural aspects, integral to the care:

"Yeah, remember our hospital has its own little Marae…that's your end of life journey, in however way you want it. One old lady, her whole whanau, all her grandchildren were in there with her, and they would sing hymns in the morning, sing hymns at night with her. They were all around. She was happy with that, and she did pass in time…that's what whanau is. So you have the outpatients, you have the in-patients, and from us we have that extra bit, and it's all part of the care." (P3)

Participants also highlighted roles of Hokianga hospital that extended beyond the roles that would generally be considered health services in a city:

"If you're in a town, there are a range of different services as well where … from a mental health point of view,… there isn't a refuge here, so if you're actually now running away from a violent situation, we provide one of the only—other than sort of social family and friends which are not always available—places that you can come and be safe. Whereas, in a city that wouldn't be health services that do that necessarily. There isn't anywhere else here." (P11)

Navigation

The approachability of the local health service was seen by participants as having wider positive health effects:

"I think that's fundamental to the importance of the hospital in the health service in Hokianga; the fact that even if they don't use it, people know that if anything goes wrong—if they've got any problems, they know where to go—they know how to use it… so, it contributes to the wellness of the whole population." (P2)

Assisting people to navigate the wider health system, across the primary-secondary-tertiary interface, was a part of the hospital's role:

"That Hokianga health is connected somehow to the system, and know everything about all the tests and investigations, and that's just something we have to work with a bit, and try and use the support systems…I don't think people here in this context have a problem with that, because they understand that the service does everything from primary care and rural hospital." (P11)

All participants saw as interwoven rather than add-on, the role of the hospital within the Hokianga health service:

"It's one service, and that's how people would see it. They'd go to the same place basically, in the same service whether it's in the middle of the night with acute pain, or they've cut off a limb or whatever, or if they just want to go and follow-up their results; that's the same thing." (P3)

Home

The concept of the hospital as 'home' was mentioned by all participants:

"In one way the hospital is like a home away from home. A place where the person is recognisably the individual that they are and where they have a sense of belonging and the ability to communicate their feelings about what is right for them… allows for patients to remain within their own home territory so to speak." (P9)

Participants commented on the different way the word 'hospital' was used in Hokianga, as part of, not separate from, community care:

"They don't describe our hospital in the same way they describe the DHB's hospitals. To them it's an extension of their homes… coming back here is like a step way back into what they're used to; the environment, the people that they know." (P5)

"They're the saying the same thing, nearly all of them were saying the same thing: alien…and homely …aye … the difference." (P3)

Participants attributed this to the wider Hokianga context, the people and the place, its history and the principles of the service including its governance:

"They own it. The community owns this place, so they have a say from a governance perspective. It makes a big difference when you can change and influence the whole of the operation of a place." (P10)

Discussion

The study findings demonstrate how spatial isolation can impact on health needs and service responses, influencing the breadth of care that a health service provides. The findings facilitate an understanding of rural health in its own context rather than looking at it through an urban lens, which is the usual view. This study found evidence that concurs with previous studies that rural health is *"much more than merely the practice of health in another location"*.[1] The Hokianga health service was understood by participants as strong and innovative, providing integrated, cooperative and holistic care. A rural hospital as part of a rural health service can fill multiple roles across both primary and secondary healthcare services and also wider health and social services that are not otherwise available or accessible in rural locations, for example refuge or hospice.

This concurs with the literature that rural hospitals can contribute to improved access and strengthened integration of service delivery for rural remote populations.[6,28]

The study also supports previous findings that for many rural communities: the local hospital is not just a provider of medical services but *"part of the economic and social fabric of the community"*.[3] The study findings introduce the concept of hospital as home, the hospital embedded in the community it serves and sharing its cultural values. In Hokianga Health's case, the hospital is not an entity standing alone but is part of the total whanau of the hau kainga, (whanau of the whole Hokianga community). It provides employment for hau kainga as well as wellbeing. The gift of land binds the hau kainga and the hospital service delivery and highlights the importance of culturally appropriate (in addition to geographically appropriate) health services for rural communities. This extends the findings of previous studies that local context is a key environmental enabler of sustainable rural health services.[29,30]

Study limitations

The study focused on a single rural health service with a particular model of care, geography and population. This needs to be taken into account when translating findings. The perspective of this study was that of healthcare providers and mainly of medical practitioners. Further research should consider non-medical staff and community perspectives as stakeholders of interest in further exploring the role of rural hospitals. At Hokianga Health a Kaupapa Māori collaborative and community-led research approach would be appropriate.

Implications for practice and policy

Rural hospitals and their communities depend on high-quality specialist medical services provided 'downstream' at secondary and tertiary hospitals. Study findings caution against viewing rural hospitals simply as small scale (and thus implied lesser value) versions of these larger urban hospitals. The different foundation, purpose and roles of rural hospitals demand a re-think in how they are valued, funded and judged within the wider New Zealand health system.

The recently released Health and Disability System review[31] reaffirmed the findings of earlier reports, that large gaps exist in our understanding of rural health outcomes and rural health services in New Zealand.[32,33] The interim report makes specific reference to the need for further research into the function of New Zealand rural hospitals and their contribution to the health system.[31] Alignment of the funding model for rural hospitals should follow clearer articulation of their value.

Conclusion

This study has provided a perspective of how one rural hospital contributes to the healthcare of its community. The study demonstrates that an integrated model of care can incorporate quality hospital-based care and that this in turn can enhance comprehensive and continuous care for a rural community.

Competing interests:
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https://www.nzma.org.nz/journal-articles/good-care-close-to-home-local-health-professional-perspectives-on-how-a-rural-hospital-can-contribute-to-the-healthcare-of-its-community

REFERENCES:

1. Bourke L, Humphreys JS, Wakerman J, Taylor J. Understanding rural and remote health: a framework for analysis in Australia. Health Place. 2012 May; 18(3):496–503. doi: 10.1016/j.healthplace.2012.02.009. Epub 2012 Mar 2.
2. Strasser R. Rural health around the world: challenges and solutions. Fam Pract. 2003 Aug; 20(4):457–63. PubMed PMID: 12876121.
3. Strasser R, Harvey D, Burley M. The health service needs of small rural communities. Aust J Rural Health, 1994. 2(2):7–13.
4. Humphreys JS, Jones JA, Jones MP, Mildenhall D, et al. The influence of geographical location on the complexity of rural general practice activities. Med J Aust, 2003; 179(8):416–421.
5. Smith JD, Margolis SA, Ayton J, Ross V, Chalmers E, Giddings P, Baker L, Kelly M, Love C. Defining remote medical practice. A consensus viewpoint of medical practitioners working and teaching in remote practice. Med J Aust. 2008 Feb 4; 188(3):159–61.
6. Nolte E, Corbett J, Fattore G, Kaunonen M, et al. Understanding the role of community hospitals: an analysis of experiences in five countries: Ellen Nolte. Eur J Public Health, 2016. 26(suppl_1). http://doi.org/10.1093/eurpub/ckw164.069
7. Winpenny EM, Corbett J, Miani C, King S, et al. Community Hospitals in Selected High Income Countries: A Scoping Review of Approaches and Models. Int Journal Integr Care, 2016; 16(4):13. doi: 10.5334/ijic.2463
8. Gauld, R. Beyond New Zealand's dual health reforms. Soc Policy Adm, 1999; 33(5):567–585.
9. Parliament New Zealand. Parliamentary support research papers: New Zealand Health System Reforms, 2009. Available from: http://www.parliament.nz/en-nz/parl-support/research-papers/00PLSocRP09031/new-zealand-health-system-reforms
10. Barnett P, Barnett JR. Community ventures in rural health: the establishment of community health trusts in southern New Zealand. Aust J Rural Health. 2001; 9(5):229–34.
11. Burton J. Rural Health Care in New Zealand. Occasional Paper 1999, Royal New Zealand College of General Practitioners: Wellington, NZ.

12. Janes R. Benign neglect of rural health: is positive change on its way. NZ Fam Physician, 1999; 26(1):20–2.
13. Lawrenson R, Nixon G, Steed R. The rural hospital doctors workforce in New Zealand. Rural Remote Health, 2011; 11(2):1588.
14. Williamson M, Gormley A, Farry P. Otago Rural Hospitals Study: What do utilisation rates tell us about the performance of New Zealand rural hospitals? N Z Med J, 2006; 119(1236).
15. Williamson M, Gormley A, Dovey S, Farry P. Rural hospitals in New Zealand: results from a survey. N Z Med J 2010; 123(1315).
16. Nixon G, Blattner K. Rural hospital medicine in New Zealand: vocational registration and the recognition of a new scope of practice. N Z Med J, 2007; 120(1259).
17. Students and faculty of Rural Postgraduate Programme University of Otago. The Qualities of a good rural hospital: A NZ 2017 perspective. [cited 2019 May 5]; Available from: http://blogs.otago.ac.nz/rural/2019/06/13/good-rural-hospital-2017/
18. Blattner K, Stokes T, Nixon G. A scope of practice that works “out here”: exploring the effects of a changing medical regulatory environment on a rural New Zealand health service. Rural Remote Health 2019; 19(4):5442. http://doi.org/10.22605/RRH5442 https://www.rrh.org.au/journal/article/5442
19. Hokianga Health Enterprise Trust, Hokianga Health Enterprise Trust. Annual Report 2016 2016. Hokianga Health Enterprise Trust Rawene, NZ.
20. Atkinson J, Salmond C, Crampton P. NZDep2013 Index of Deprivation. 2014. Dunedin: University of Otago, New Zealand. Available from http://www.health.govt.nz/publication/nzdep2013-index-deprivation
21. Hokianga Health Enterprise Trust. Hokianga Health. 2018 [cited 2019 March 2]; available from: http://www.hokianga-health.org.nz/HHET2017/base2.php?page=home
22. Hokianga Health Enterprise Trust, Tikanga Whakatau. 2013, Hokianga Health Enterprises Trust Hokianga Health.
23. Kearns RA. The place of health in the health of place: the case of the Hokianga special medical area. Soc Sci Med, 1991; 33(4):519–530.
24. Williams C. Hokianga Health. The First Hundred Years-Te Rautau Tuatahi. 2010, Rawene, NZ: Hokianga Health Enterprise Trust.
25. Gale NK, Heath G, Cameron E, Rashid S, et al. Using the framework method for the analysis of qualitative data in multi-disciplinary health research. BMC Med Res Methodol. 2013 Sep 18; 13:117. doi: 10.1186/1471-2288-13-117.
26. Tong A, Sainsbury P, Craig J. Consolidated criteria for reporting qualitative research (COREQ): a 32-item checklist for interviews and focus groups. Int J Qual Health Care, 2007; 19(6):349–357.
27. Heslop C, Burns S, Lobo R. Managing qualitative research as insider-research in small rural communities. Rural Remote Health, 2018; 18(3):4576–4576.
28. Pitchforth E, Nolte E, Corbett J, Miani C et al. Community hospitals and their services in the NHS: identifying transferable learning from international developments – scoping review, systematic review, country reports and case studies. Health Services and Delivery Research (HS&DR) 2017 5 (19) doi:10.3310/hsdr05190
29. Weinhold I, Gurtner S. Understanding shortages of sufficient health care in rural areas. Health Policy, 2014; 118(2):201–214.
30. Lyle D, Saurman E, Kirby S, Jones D, et al. What do evaluations tell us about implementing new models in rural and remote primary health care? Findings from a narrative analysis of seven service evaluations conducted by an Australian Centre of Research Excellence. Rural Remote Health, 2017. 17(3).
31. Health and Disability System Review. 2019. Health and Disability System Review - Interim Report. Hauora Manaaki ki Aotearoa Whānui – Pūrongo mō Tēnei Wā. Wellington: HDSR.
32. Fraser J. Rural Health: A literature review for the National Health Committee. Wellington, New Zealand Health Services Research Centre 2006.
33. Fearnley D, Lawrenson R, Nixon G. ‘Poorly defined’: unknown unknowns in New Zealand Rural Health. N Z Med J. 2016; 129(1439):77.

Epidemiology of traumatic spinal cord injury in New Zealand (2007–2016)

John Mitchell, Joanne Nunnerley, Chris Frampton, Tracey Croot, Alpesh Patel, Rowan Schouten

ABSTRACT

AIM: To investigate the epidemiology of traumatic spinal cord injury (TSCI) in New Zealand over a 10-year period.

METHODS: Ambispective data of all new patients admitted to New Zealand's two spinal rehabilitation units between January 2007 and December 2016 (n=929) were collated. Variables assessed included age at injury, gender, ethnicity, date of injury, aetiology, length of hospital stay, injury level, neurological status on discharge and discharge destination.

RESULTS: The incidence of TSCI averaged 22 (95% CI 21–24) per million, increasing 6% a year. The average incidence for Māori (29 per million people (95% CI 25–34)) was 1.8 times higher than New Zealand European (16 per million people (95% CI 15–18)), and show an increase of 14% a year. The median age of TSCI increased from 43 to 48 years. Overall, falls (32%), transport (32%) and sports (22%) were the most common causes of TSCI. Cervical TSCI (54%) were most common, particularly in older adults (70% over 75 years) and Māori (61%) and Pacific Island (72%) patients. Surgical rates remained stable (77%) but length of stay in hospital decreased over the study period.

CONCLUSIONS: The demographic of TSCI is changing in New Zealand. The median age of patients is increasing, as is the incidence, particularly for women, older adults and Māori patients.

A traumatic spinal cord injury (TSCI) is a life-changing event for an individual and their family/whānau. Vast costs are also associated with the extensive treatment, rehabilitation and lost productivity incurred.[1] The incidence for TSCI varies worldwide.[2,3] Recent multinational studies also suggest demographics are changing over time, with an increasing median age of TSCI and higher incidence in older adults.[3,4]

Previously published data on the epidemiology of TSCI in New Zealand is limited but suggests it may have one of the highest rates of TSCI in the western world, particularly among Māori and Pacific Islanders.[5,6] Understanding the demographics of this specific cohort will support healthcare planning, shape clinical research priorities and assist patient care to achieve optimal quality of life, social and economic outcomes.

The aim of this study is to utilise the newly established New Zealand Spinal Cord Injury Registry (NZSCIR) to better understand the epidemiology and demographic trends of patients admitted for spinal rehabilitation following TSCI in New Zealand over a 10-year period, from January 2007 to December 2016.

Methods

Patient population and the NZSCIR

Currently two centres provide spinal injury rehabilitation care in New Zealand, the Auckland Spinal Rehabilitation Unit (ASRU) and the Burwood Spinal Unit (BSU, Christchurch). The units were established with the aim that each location will serve half of the total New Zealand population (currently 4.69 million, 2018 census); however, due to the skewed population distribution in New Zealand the geographical catchment areas of each centre varies. The ASRU covers injuries sustained in the upper North Island, while the Burwood Spinal Unit (BSU) covers the lower North Island and the whole of the South Island.

In 2016, the NZSCIR was established in partnership with the Rick Hansen Institute (Canada), to collect relevant data on all patients sustaining a traumatic or non-traumatic SCI (NTSCI) in New Zealand. The NZSCIR prospectively records a minimal data set (MDS) (Table 1), comparable to the Rick Hansen Spinal Cord Injury Registry, for all patients admitted to both New Zealand spinal units and full prospective data on patients who give consent.[8] The NZSCIR defines SCI as impairment of the spinal cord or cauda equina function resulting in either a motor or sensory deficit or both.[9] To support the aims of this study, retrospective admission data from both rehabilitation units was used to identify all patients admitted from 1 January 2007 to 31 July 2016. MDS data (Table 1) were collected on these patients from electronic and hard copy medical notes and entered into the NZSCIR by three research assistants under the guidance of an NZSCIR Coordinator. Where data was unavailable, the respective fields were left 'unknown'.

These data were combined with five months of prospective MDS data from the NZSCIR, providing a combined 10-year ambispective cohort from January 2007 to December 2016. The inclusion criteria involved adult patients (age >16 years) admitted to either spinal unit for rehabilitation with a new TSCI. Patients with NTSCI were excluded from this study.

Aetiology was coded using the adapted International Classification of External Causes of Injuries (ICECI), as per the International SCI Core Data Set.[10] Sports, transport and falls were further classified, including a free text description. 'Other Traumatic Cause' includes SCI following medical procedures which directly injured or caused vascular compromise (eg, ischaemia or infarction) to the spinal cord or cauda equina.

Length of hospital stay was recorded in days, from acute admission at any New Zealand hospital to discharge from the spinal rehabilitation unit. International Standards for the Neurological Classification of Spinal Cord Injury (ISNCSCI) on rehabilitation discharge was collected, giving a single neurological level (SNL) and an American Spinal Injury Association Impairment Scale (AIS) grade. Neurological level was categorised anatomically as C1-4, C5-8, Thoracic, Lumbosacral. The AIS is a standardised grading system used to classify the severity (completeness) of neurological injury in individuals with SCI.[11] It is based on the testing of power in key muscle (motor) groups (graded as 0 (no movement) to 5 (full strength)), a dermatomal based sensory function examination combined with an anorectal assessment. The 5-point ordinal AIS classifies individuals from "A" (complete SCI) to "E" (normal sensory and motor function) based on this assessment of motor, sensory and anorectal function below

Table 1: NZSCIR minimal data set.

Age at injury
Gender
Ethnicity
NZ residency (Yes/No)
Date of injury
Aetiology (sports, assault, transport, fall, other traumatic cause, non-traumatic spinal cord dysfunction, unspecified/unknown)
Length of hospital stay (acute and rehabilitation phases)
Discharge destination
ISNCSCI* (SNL§ and AIS^) at discharge

*International Standards for Neurological Classification of Spinal Cord Injury; §Single Neurological Level; ^American Spinal Injury Association (ASIA) Impairment Scale.

the injured neurological level.[11] Patients with AIS Grade B injuries have some sensory function preserved but no motor function below the injured neurological level, AIS C injuries have motor function preserved with half the muscles below the injured level having a muscle grade less than 3 (full active movement against gravity but without resistance), while AIS D injuries have a motor grade of 3 or more in at least half of key muscles below the neurological level.[11]

Analysis

The data were imported from NZSCIR into SPSS V25.0 to produce descriptive demographic and aetiologic summaries for the incident TSCI cases. The incidence rates were calculated using the New Zealand Census data from 2006 and 2013, and 95% confidence intervals calculated using a Poisson approximation.

Ethics approval

The study was approved by the University of Otago Ethics Committee (Health) HD19/029.

Results

Data from 929 patients were collected over the 10-year interval from January 2007 to December 2016.

The absolute number of cases of admissions per year ranged from 72 to 126, with a mean of 93. Data showed a trend of increasing numbers per year (Figure 1). The proportion of male to female injuries decreased over the study period, with males accounting for 79% of admissions in 2007 and 72% in 2016. Comparisons of the first and last years of the 10-year period show female admissions had more than doubled (15 in 2007 to 32 in 2016), while males only increased 1.4 times (57 in 2006 to 83 in 2016).

The mean national annual incidence of TSCI was 22 per million people (95% CI 21–24). The mean incidence of TSCI in men was 34 per million (95% CI 32–37) and 11 per million people (95% CI 10–12) in women. Over the 10-year study period, the incidence of TSCI increased on average 6% per year (Figure 2).

Age

The mean age of patients increased from 43 years to 48 years over the 10-year period. In every age group, there was an increased annual incidence of TSCI, with the greatest increases in people over the age of 55. In the study's final year (2016) the age bracket of 55–74yr had the highest incidence of all age ranges (Figure 3).

Ethnicity

The highest incidence of TSCI was in the Māori population with mean rates of 29 per million people (95% CI 25–34), compared to New Zealand Europeans 16 per million

Figure 1: Traumatic spinal cord injury (TSCI) admissions 2007–2016.

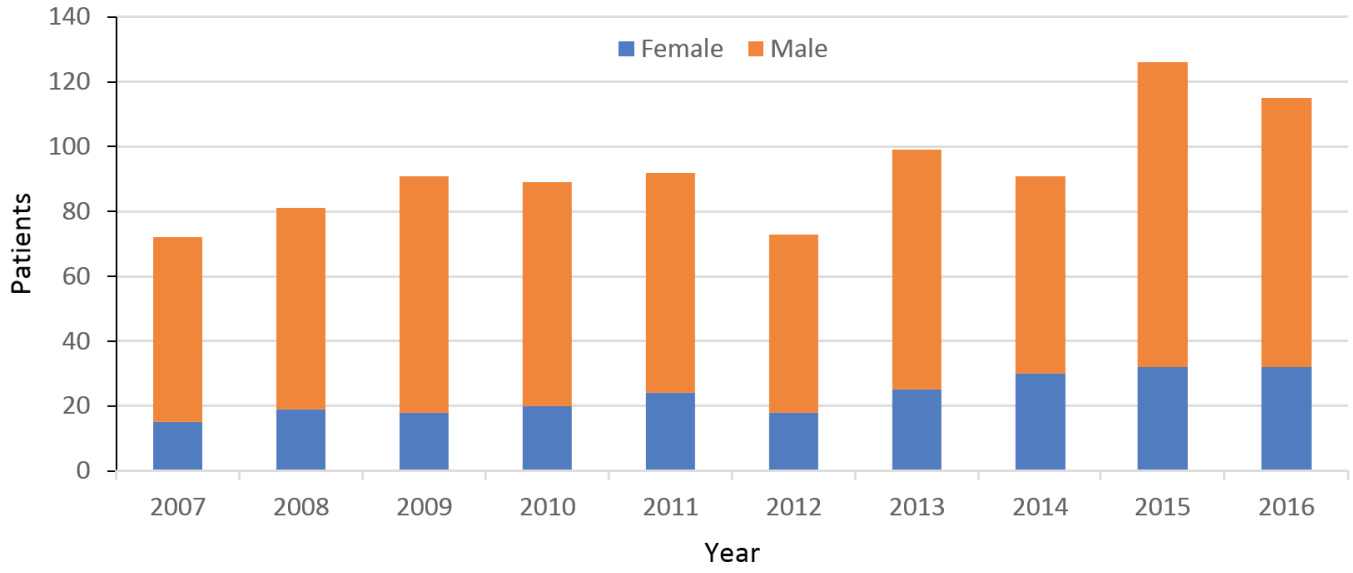


Figure 2: Traumatic spinal cord injury (TSCI) incidence rates 2007–2016.

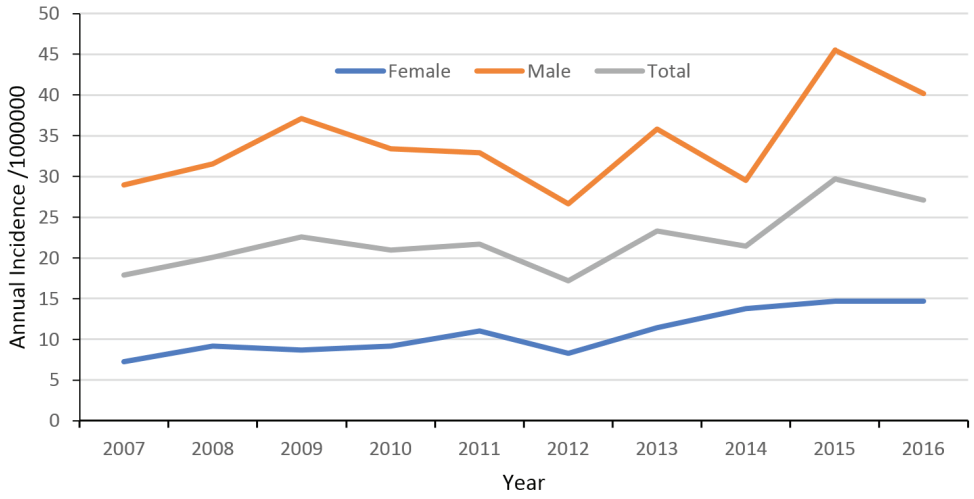


Figure 3: Age-specific traumatic spinal cord injury (TSCI) incidence rates 2007–2016.

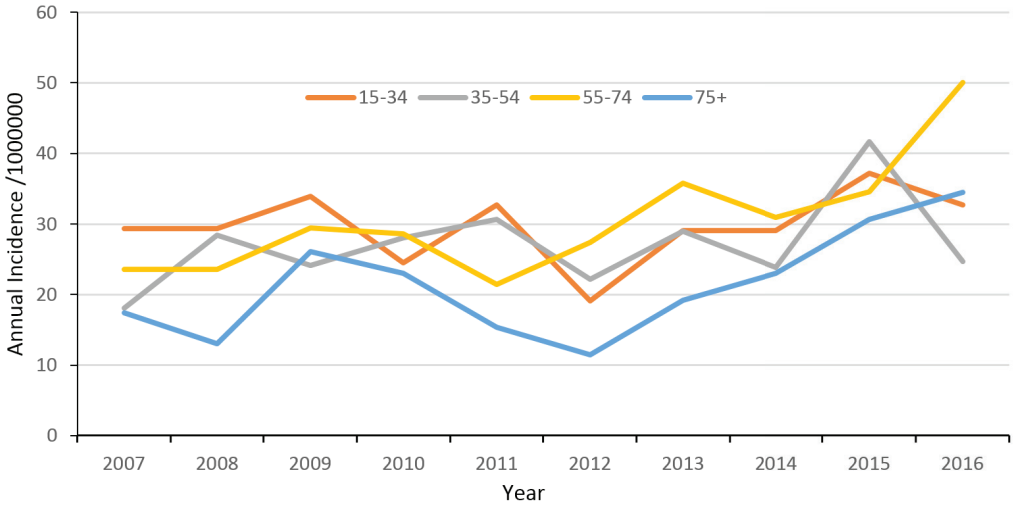
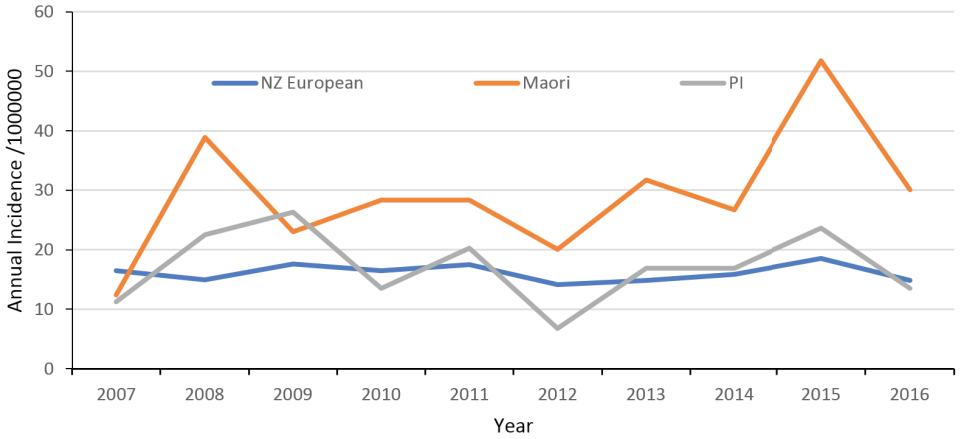
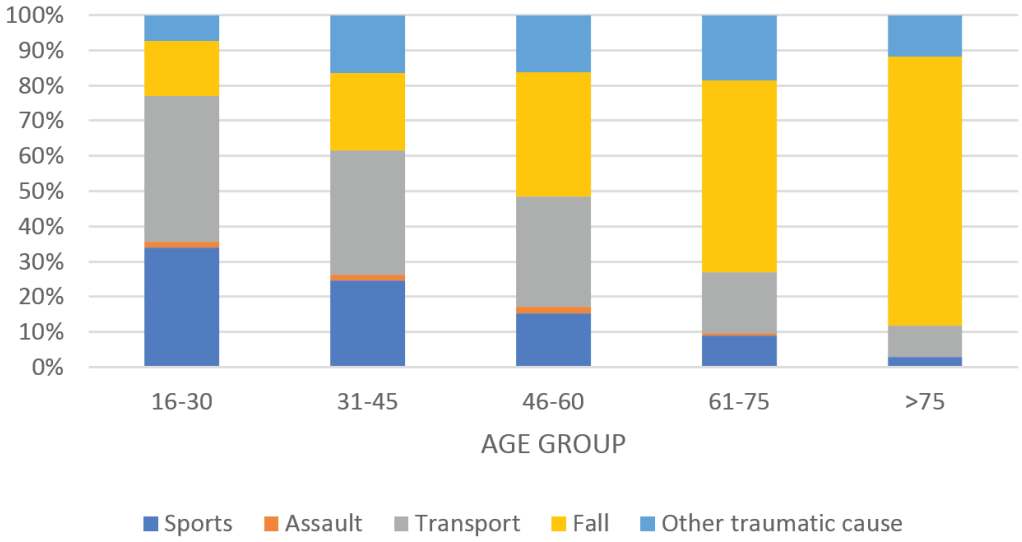


Figure 4: Traumatic spinal cord injury (TSCI) rates by ethnicity.



people (95% CI 15–18) and Pacific Islanders 17 per million people (95% CI 13–23) (Figure 4). TSCI in Māori was 1.8 times more frequent than in New Zealand Europeans. The rate of increase in incidence of TSCI in the Māori population was approximately 14% throughout the study period while the incidence among the New Zealand European and Pacific Islander groups remained largely static.

Figure 5: Aetiology of traumatic spinal cord injury (TSCI) by age group.



Aetiology

Over the 10-year study period the most common aetiology of TSCI was transport (32%), followed by falls (31%) and sports (21%). Aetiology varied significantly according to age. Motor vehicle crash and sports were the most common mechanisms of injury in younger cohorts and falls was the most prevalent cause in patients over 60 years (Figure 5).

A breakdown of the specific aetiology of sports-related TSCI are shown in Table 2. Team ball sports (eg, rugby) remained the most common sporting cause overall (20%), however rates varied significantly with age. Team ball sports (eg, rugby) was the most common sporting mechanism in the 16–30 age group, causing 31% of sporting injuries, while water sports (eg, diving) was the most common cause in 31–45 year-olds (23%) and wheeled non-motorsports (eg, mountain biking) was most common in ages 46–75 (37%).

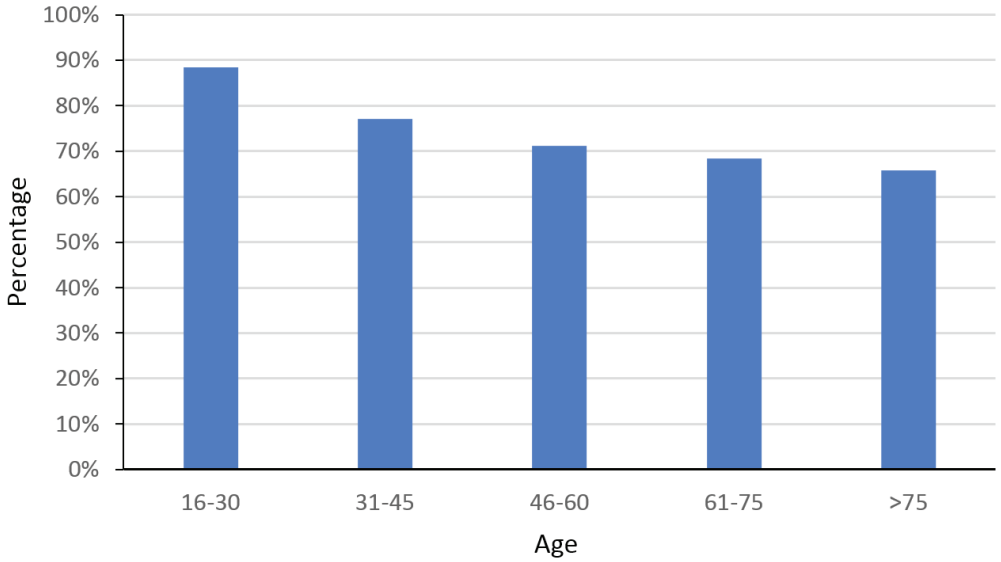
Surgical intervention

Over the last 10 years the surgical rate remained stable, with 77% (range 66–83%) of patients receiving surgical management. Surgical management was most common in the 16–30 age group (88%). Rates of surgical intervention declined with age (Figure 6).

Table 2: Detailed sports aetiology.

Sport	Total	%
Team ball sports eg, rugby, soccer, basketball	36	20%
Wheeled non-motorsports eg, mountain biking, cycling, skateboarding	32	18%
Individual water sports eg, diving, surfing, swimming	32	18%
Wheeled motor sports eg, motorcross, off-roading	19	11%
Ice or snow sports eg, snowboarding, skiing	15	9%
Equestrian sports	13	7%
Aero sports eg, paragliding, skydiving	11	6%
Other eg, adventure, acrobatic, boating	19	12%

Figure 6: Percentage of traumatic spinal cord injury (TSCI) patients who underwent surgery.



Duration of hospital admission

The length of stay (LOS) reduced from a median of 105 days in 2007 to 77 days in 2016 (Figure 7), a trend evident at both individual rehabilitation centres. The LOS did not appear to be influenced by ethnicity, with New Zealand Europeans (93 days), Māori (96 days) and Pacific Islanders (91 days) sharing similar timeframes.

Neurological injury level

Cervical level injuries were most common, accounting for 54% of all injuries (52% C1-4 and 48% C5-8), while thoracic injuries accounted for 28% of TSCI and 18% of patients had a lumbo-sacral injury. Cervical injuries were more common in the older age groups, peaking at 65% (57% C1-4, 43% C5-8) in the 61–75 age group (Figure 8).

Figure 7: Median length of stay (days) Traumatic Spinal Cord Injury (TSCI).

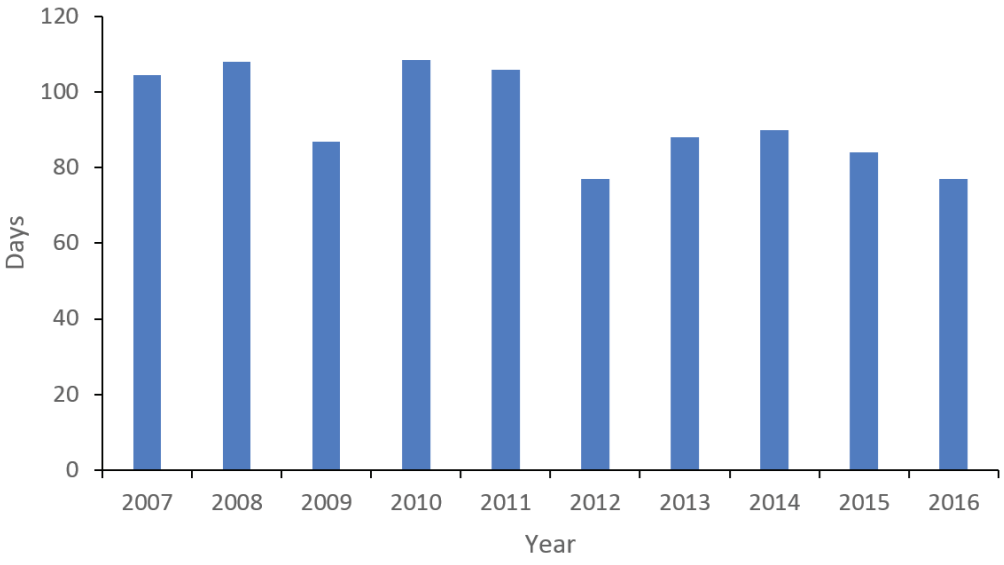
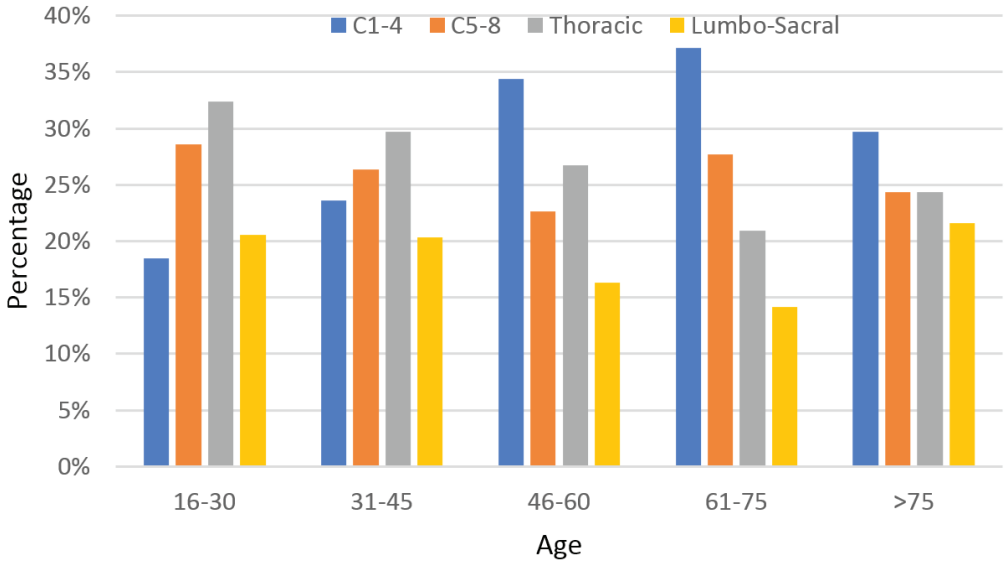


Figure 8: Age stratified neurological level of traumatic spinal cord injury (TSCI).



People identifying as Pacific Island and Māori had a higher proportion of cervical injuries, with 76% (54% C1-4, 46% C5-8) of Pacific and 61% (58% C1-4, 41% C5-8) of Māori patients with a cervical injury, compared to only 50% (50% C1-4, 50% C5-8) of New Zealand European patients.

Impairment level on discharge

Over the 10-year period there was a decrease in the proportion of patients with an AIS-A impairment score on discharge. In 2007, 40% of patients were discharged with an AIS A, in 2016 this number decreased to 30%. Over the same time period the proportion of patients with AIS D increased from 34 to 49% (Figure 9). AIS A injuries were more common in younger patients (41% of patients aged between 16–30 (Figure 10) compared with the >75 age group (11%).

Discharge destination

The number of patients discharged home following rehabilitation steadily declined over the study period from a high of 88% in 2008 to 77% in 2016. Patients being discharged to their own home was less common in older adults (only 51% for people aged >75). However, the rates of patients discharged home did not differ across ethnicities (New Zealand European (78%), Māori (78%) and Pacific Islander (86%)).

Figure 9: Percentage of patients by discharge AIS score.

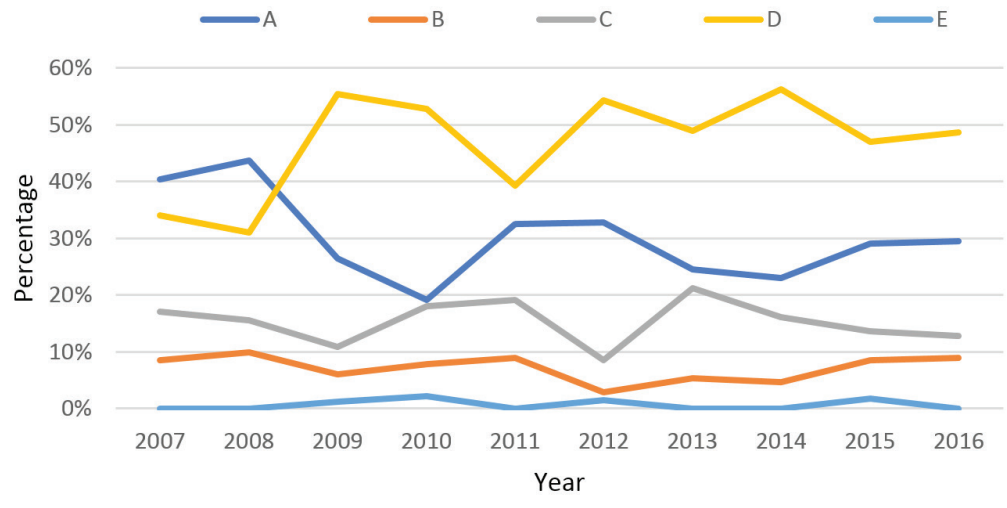
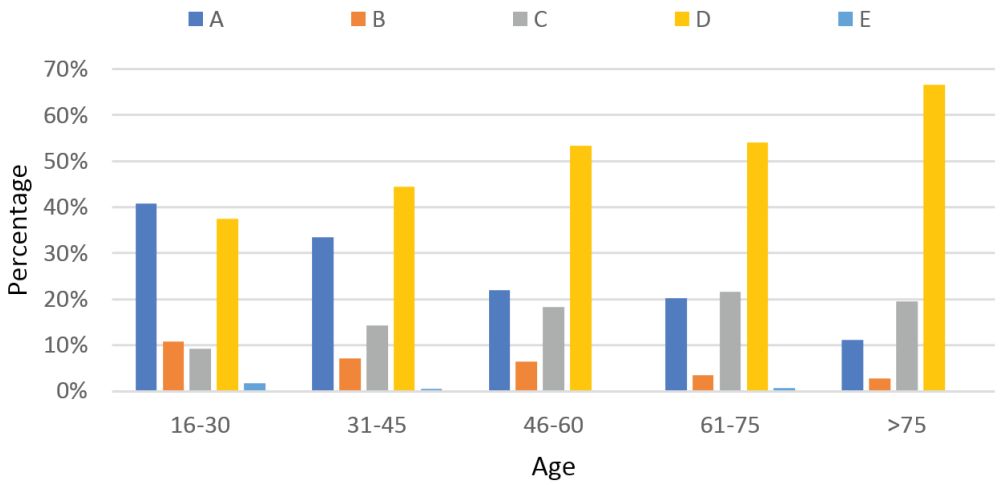


Figure 10: Age stratified AIS score on discharge.



Discussion

This study is the largest and most comprehensive report to date on the epidemiology and demographic trends of TSCI in New Zealand patients.[5,6] The mean annual incidence of TSCI in New Zealand over the 10-year period is 22 (range 17–30) per million people. This rate is lower than previously reported (49 per million people in 1998) but is consistent with the current estimated global-incident rate of 23 per million.[5,6,12]

The demographic of TSCI in New Zealand has changed over the last 10 years. While TSCI in men (34 per million) is three times more frequent than women (11 per million), the incidence is increasing faster in women (9% per year) than for men (6% per year). Epidemiological studies of TSCI in Japan have also noted a reduced male predominance, with the male to female ratio reducing from 4:1 in 1990, to 2:1 more recently (2004–2013).[15] Over the 10-year study period the mean age of patients sustaining a TSCI increased from 43 to 48 years. Concordantly incidence rates in the older age groups showed the biggest change, increasing 11% and 10% per year in the 55–74 and >75 age groups respectively.

Over the 10-year study period the leading causes of TSCI were falls (32%), transport (32%) and sports (22%). Comparatively, in 1993 the reported leading causes of TSCI in New Zealand were transport (54%), falls (24%) and sports (11%).[5,12] The pattern of decreasing transport-related TSCI and increasing number of TSCI from falls is consistent with other developed countries.[2,4,12,14,15] Age influences aetiology, with falls more common in older adults (the cause for 70% of patients over the age of 75), while transport remains the most frequent mechanism in younger (16–30 years) cohorts.

A 2016 systemic review identified New Zealand as having the third highest proportion of TSCI as a result of sports injuries worldwide (behind Russia and Fiji) based on a rate of 20% reported in 1993.[5,17] This study's recorded proportion of TSCI due to sports injuries of 22% remains high and would maintain New Zealand's standing in this comparison. Globally, rugby is responsible for 23% of all sporting-related TSCI and its popularity in New Zealand has been suggested as the reason for the high rates of sporting-related TSCI in this country.[5] In 1993, Dixon et al[5] reported rugby caused 74% of sporting TSCI in New Zealand.[5] In comparison we identified team ball sports (including rugby) to be responsible for 20% of all sports-related TSCI in New Zealand over the recent 10-year period.[17] Internationally, diving is the most prevalent cause of sports-related TSCI (35%)[17], however this was not the case in our New Zealand population (18%). Water-related incidents caused 4.6% of all TSCI in New Zealand, a rate almost half that of Australia (9.0%).[12] While the global rate of TSCI related to cycling is recorded as 5.5%, we found wheeled non-motorsport (including cycling and mountain biking) represented 18% of sporting-related TSCI in New Zealand.[17] A recent study has shown cycling to be increasingly

popular in New Zealand with hospital admissions from cycling injuries increasing 17% per year from 2012–2016.[19]

Cervical TSCI was the most common level of injury, occurring in 54% of patients. This level of injury is more common in older adults, with 62% of patients over 60 having a cervical TSCI compared to 47% of patients aged between 16–30 years. This is consistent with other global studies, including a Canadian study that found the proportion of cervical injuries was 88% in people >75 years, versus 46% in patients aged <35 years.[2] Our study found cervical injuries were more common in Pacific Islanders (76%) and Māori (61%) compared to New Zealand Europeans (50%). The higher incidence of cervical TSCI may be due to anatomical differences in the spine, with Māori having 1mm and Pacific Islanders having 2mm smaller cervical canal than New Zealand Europeans.[20] Overall, the majority of patients received surgery for their TSCI (77%). The highest proportion of patients who had surgery were in the younger age groups (89% in under 30 years), consistent with recent Canadian studies.[1,2] Low-energy incomplete (particularly AIS D) cervical TSCI's are often managed non-operatively and the probable reason for the lower surgical rates in the older age groups (66% in >75 years).[1,2]

One key finding of the study was that rates of TSCI in Māori were 1.8 times greater than New Zealand Europeans and increasing at a faster rate than both New Zealand Europeans and Pacific Islanders. Further work is needed to understand the basis for this disproportionate representation in Māori, given Pacific Islanders have a lower rate of injury despite also having smaller cervical canals than New Zealand Europeans.

Demographic information from this study maybe useful to identify preventative strategies aimed at reducing the numbers of TSCI. While we did not stratify aetiology data by gender, the increasing rates in both older adults and female patients are possibly explained by an increasing rate of trauma in older adult women from low energy falls resulting in incomplete cervical TSCI (eg, central cord syndrome), a trend noted worldwide.[2,4,13–15] While the reason for increasing falls in older adults is multifactorial, most falls happen at home. Therefore, programmes to raise awareness and improve home safety and design are important to consider in future health resource allocation.[4] In addition, a focus on cycle safety may need to be a priority in New Zealand.

There seems to have been significant progress in reducing the number of rugby and transport-related TSCI. "RugbySmart" was introduced in New Zealand by Accident Compensation Corporation (ACC) and New Zealand Rugby Union (NZRU) in 2001, which focused on educating rugby participants about physical conditioning, injury management and safe techniques in the contact phases of rugby. This coincided with a reduction in the rate of SCI arising from scrums in rugby union.[21] In 2007, new rugby scrum laws were implemented, which have been implicated in the reduced rate of rugby-related TSCI in South Africa[17] and may partly explain the reduced rate in our study. Further analysis of the cohort of people injured playing rugby is planned. The reduction in transport-related TSCI is postulated to be a result of improved car safety (seatbelts, air bags etc), improved roading and more awareness/advice on road safety.[2,4,15] Of note, the New Zealand Ministry of Transport reports a 41% decrease in both morbidity and 50% reduction in mortality from motor vehicle crashes from 1993 to 2016.[18]

The strengths of this study include the large patient numbers and the 10-year timeframe. However, the majority of data were retrospectively gathered and thus limited by the extent and quality of data initially recorded and recovered. The prospective collection of NZSCIR data from 2016 onwards will negate these issues in the future. The data in this study may underestimate the true figures as some TSCI patients in New Zealand, specifically patients with minor deficits, are not always referred for SCI rehabilitation.

This study represents the first significant epidemiology publication based on information from the recently established NZSCIR. These insights help health providers understand our current patient population and have highlighted areas where further analysis may identify

opportunities to improve outcomes. It has initiated further studies on the over-representation of Māori patients in this cohort and the changing status of rugby-related injuries. It directs health professionals and funders to anticipate a future where older adults will make up an increasing proportion of patients requiring treatment and rehabilitation. Collecting identical data to our parent Canadian-wide registry provides a unique opportunity to continually compare demographics and treatment practices on a country-wide basis with a respected and established international leader in SCI research.

Conclusion

This study provides the most current, extensive and accurate epidemiology review of TSCI in New Zealand and has identified important demographic trends in this patient cohort. The incidence of TSCI is increasing particularly in older adults, women and Māori. Falls are the most common cause of TSCI particularly in older adults, while cervical TSCI are the most common level of injury, with higher rates in Māori and Pacific Islanders. We confirmed a high proportion of sporting-related TSCI compared to other countries, with the highest rates in team ball sports (eg, rugby), individual water sports (eg, diving) and wheeled non-motorsports (eg, cycling/mountain biking). Despite previously recorded high rates of rugby-related TSCI in New Zealand, these have moderated. Future New Zealand public health resourcing and prevention strategies need to be cognisant of the trends demonstrated.

Competing interests:
Mrs Croot is one of two NZ Spinal Cord Injury Registry (NZSCIR) Coordinators. She is involved with consenting participants, data collection, data entry, registry oversight and maintenance, as well as data access requests for the NZSCIR. She was not involved in the data extraction for this paper. Dr Nunnerley reports funding from Canterbury Orthopaedic Services during the conduct of the study.

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REFERENCES:

1. Pickett G, Campos-Benitez M, Keller J, Duggal N. Epidemiology of traumatic spinal cord injury in Canada. Spine. 2006; 31(7):799–805.
2. Lenehan B, Street J, Kwon B, et al. The epidemiology of traumatic spinal cord injury in British Columbia, Canada. Spine. 2012; 37(4):321–329.
3. Kumar R, Lim J, Mekary R, et al. Traumatic spinal injury: Global epidemiology and worldwide volume. World Neurosurgery. 2018; 113:345–e363.
4. Beck B, Cameron P, A Braaf, S, et al. Traumatic spinal cord injury in Victoria, 2007–2016. Med. J. Aust. 2019; 210:360–366.
5. Dixon G, Danesh J, Caradoc-Davies T. Epidemiology of spinal cord injury in New Zealand. Neuroepidemiology. 1993; 12(2):88–95.
6. Derrett S, Beaver C, Sullivan M, et al. Traumatic and non-traumatic spinal cord impairment in New Zealand: incidence and characteristics of people admitted to spinal units. Injury Prevention. 2012; 18(5):343–346.
7. Howard-Brown C, Civil I. New Zealand Spinal Cord Registry: a new milestone. NZ Med J. 2016 15; 129(1438):6–7.
8. Noonan VK, Kwon BK, Soril, L, et al. The Rick Hansen Spinal Cord Injury Registry (RHSCIR): a national patient-registry.
9. Croot T and Young L. New Zealand Spinal Cord Injury Registry Protocol. 2019:v2.6.
10. Biering-Sørensen F, DeVivo M, Charlifue S, et al. International Spinal Cord Injury Core Data Set (version 2.0) - including standardization of reporting. Spinal Cord, 2017; 55(8):759–764.
11. ASIA Impairment Scale and the ISNCSCI Motor and Sensory Exam. (2019). http://scireproject.com/outcome-measures/outcome-measure-tool/american-spinal-injury-association-impairment-scale-ais-international-standards-for-neurological-classification-of-spinal-cord-injury/#1467983894-080-2c29ca8d-88af
12. Lee B, Cripps R, Fitzharris M, et al. The global map for traumatic spinal cord injury epidemiology: update 2011, global incidence rate. Spinal Cord. 2014; 52(2):110–116.
13. Kehoe A, Smith J, Edwards A, et al. The changing face of major trauma in the UK. Emerg Med J. 2015; 32(12):911–915.
14. Beck B, Cameron P, Lowthian J, et al. Major trauma in older persons. BJS Open. 2018; 23(5):310–318.
15. Tafida M, Wagatsuma Y, Ma E, et al. Descriptive epidemiology of traumatic spinal injury in Japan. Journal of Orthopaedic Science. 2018; 23(2):273–276.
16. Isles S, Christey G, Civil I, Hicks P. The New Zealand Major Trauma Registry: the foundation for a data-driven approach in a contemporary trauma system. NZ Med J. 2017 Oct 6; 130(1463):19–27.
17. Chan C, Eng J, Tator C, Krassioukov A. Epidemiology of sport-related spinal cord injuries: A systematic review. The Journal of Spinal Cord Medicine. 2016; 39(3):255–264.
18. Transport.govt.nz. Road crash statistics | Ministry of Transport. [online] Available at: http://www.transport.govt.nz/mot-resources/road-safety-resources/roadcrashstatistics/
19. Singh N, Joe N, Amey A, et al. Cycling-related injuries and cycling promotion: a trauma service perspective. NZ Med J. 2019; 132(1494):41–48.
20. Faraj S, Coldham GJ, Doyle A, Baber P. Ethnic variation in cervical spine diameter. Orthopaedic Proceedings. 2015; 88-B:No.SUPP_II
21. Quarrie KL, Gianotti SM, Hopkins WG, Hume PA. Effect of nationwide injury prevention programme on serious spinal injuries in New Zealand rugby union: ecological study BMJ 2007; 334:1150.

Healthcare-associated *Staphylococcus aureus* bacteraemia: time to reduce the harm caused by a largely preventable event

Sally A Roberts, Nikki Grae, Sharmini Muttaiyah, Arthur J Morris

ABSTRACT

Staphylococcus aureus disease is associated with significant morbidity and mortality and of concern, it disproportionally affects Māori and Pacific Peoples. New Zealand has high rates of skin and soft tissue infection caused by *S. aureus.* Healthcare-associated *S. aureus* bacteraemia (HA-SAB) accounts for a significant proportion of all *S. aureus* bacteraemia events. Measurement of HA-SAB has been reported in New Zealand for over 20 years but it has not been linked to quality improvement interventions to reduce the rate. It has been used as an outcome measure for the Hand Hygiene New Zealand programme; however, a recent review of submitted data questioned the accuracy of it. This has been addressed. National programmes such as the Health Quality & Safety Commissions Hand Hygiene New Zealand and the Surgical Site Infection Improvement programme have led to reduced harm from healthcare-associated infections. Interventions targeted at reducing the HA-SAB rate, such as bundles of care for insertion and maintenance of vascular access devices and skin and nasal decolonisation of staphylococci prior to surgery, are urgently required.

Staphylococcus aureus disease is associated with significant morbidity and mortality.[1] Several studies from Australia and New Zealand looking at outcomes for *S. aureus* bacteraemia (SAB) across all ages have shown a 30-day all-cause mortality of 20%; highest in the older age group.[2–5] Over the last 30 years in New Zealand, there has been a relentless increase in the incidence of skin and soft tissue infection caused by *S. aureus,* largely driven by an increase in community-associated methicillin-susceptible *S. aureus* infections.[6–8]

Healthcare-associated *S. aureus* bacteraemia (HA-SAB) is defined as an episode of *S. aureus* bacteraemia occurring 48 hours after hospitalisation (not present or incubating on admission) or occurs in the context of an indwelling medical device, within 30 days of surgery (or 90 days of surgery involving implantable devices), within 48 hours of a related invasive instrumentation or incision or is associated with neutropaenia associated with cytotoxic therapy. The onset of infection for 60–70% of all SAB episodes occurs in the community, but about half of these infections are associated with recent hospitalisation, surgery or a procedure, or associated with a medical device; most commonly a vascular access device.[4] The more common sources of HA-SAB include vascular access devices, surgical site infections, lower respiratory tract infections and skin and soft tissue infections.[9–11] Patients who develop infections often have a longer length of stay, may die and often require further interventions contributing to increased healthcare costs.[12]

There is significant ethnic disparity associated with the burden of *S. aureus* disease in New Zealand; the incidence of both invasive and non-invasive *S. aureus*

disease, even after adjusting for socioeconomic deprivation, is highest among Māori and Pacific Peoples. In particular, Māori are three times more likely and Pacific Peoples are five times more likely than non-Māori and non-Pacific peoples to have *S. aureus* skin and soft tissue infections.[6,7] For Māori and Pacific Peoples the rates of SAB are two and four times that of European New Zealanders, respectively.[2,3]

To quote Florence Nightingale "The very first requirement in a hospital is that it should do the sick no harm". We agree with this sentiment. Reporting of district health board (DHB) hospital-acquired bloodstream infections (BSI) and HA-SAB rates has been considered an important indicator of quality of care for over 20 years, but little consideration has been given to implementing interventions known to reduce the rate of either. A significant proportion of healthcare-associated infections, such as HA-SAB, are preventable, and to continue to report on this in the absence of any nationally-led initiatives to reduce these events is a missed opportunity. The Health Quality & Safety Commission Infection Prevention and Control programme has been effective at improving compliance with hand hygiene and reducing surgical site infections for hip and knee arthroplasties and cardiac surgery. To support our view that action needs to be taken, we have summarised the history of healthcare-associated BSI surveillance in New Zealand, and provided examples of actions taken to reduce HA-SAB rates in other jurisdictions. We then recommend the main interventions that need to be delivered at a national level to reduce the ongoing harm caused by HA-SAB.

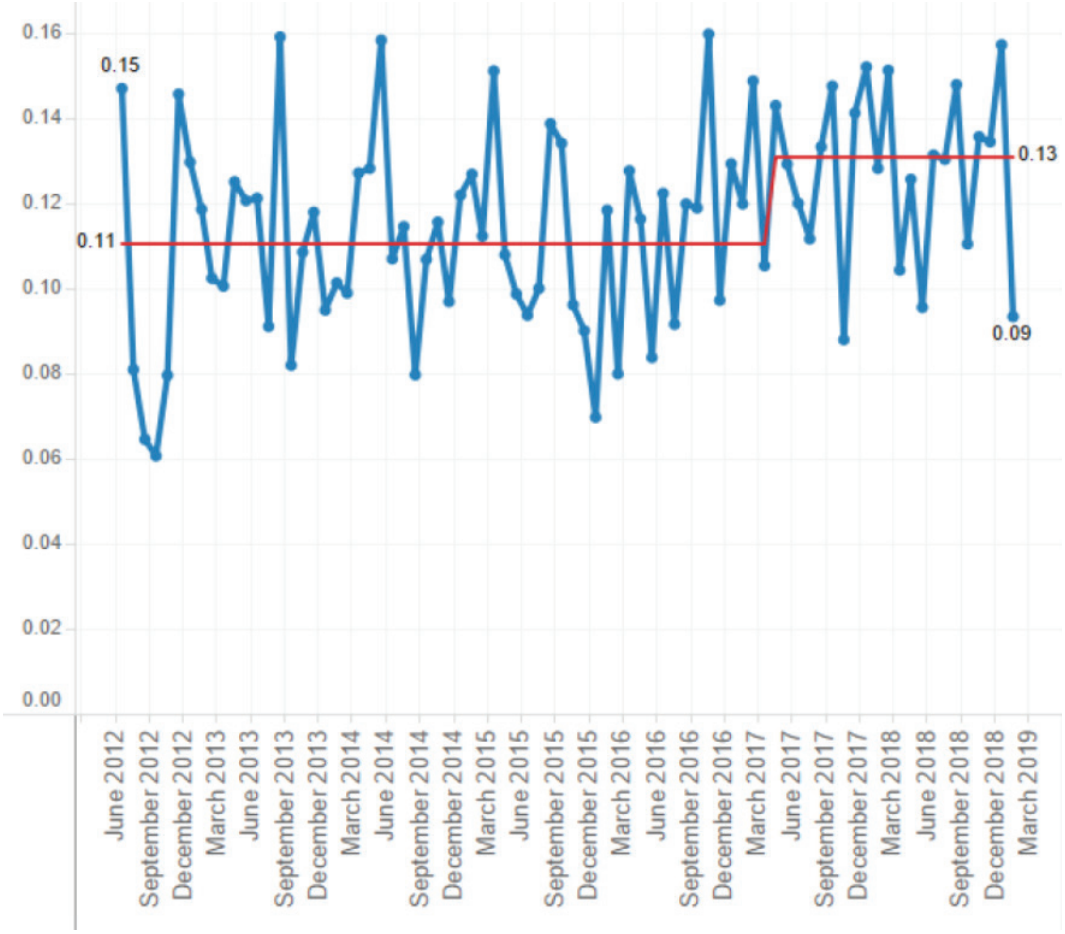
In New Zealand, hospital-acquired BSI surveillance was started in the late 1990s to provide important national data about this serious and potentially preventable event. It was established as part of the Key Performance Indicators programme set up by the Crown Company Monitoring and Advisory Unit in 1999. Subsequently, reporting shifted to the Ministry of Health's Hospital and Health Services Balanced Scorecard Performance Indicator Framework. The Australian Council on Healthcare Standards definition for hospital-acquired blood stream infections was adopted and episodes of BSI occurring more than 48 hours after admission to hospital were included. Within the health sector the reliability of this data was questioned because of inconsistencies in the way district health boards (DHB) defined such infections and reporting of results were not viewed as useful.[13]

In the early 2000s, surveillance was restricted to monitoring of HA-SAB episodes only. As with the previous surveillance programme the case definition was provided but no training or monitoring of the application of the case definition was incorporated into the programme. Both the Ministry of Health's National Quality Improvement Programme (NQIP) hand hygiene project and the Health Quality and Safety Commission's (the 'Commission') Hand Hygiene New Zealand programme have subsequently used the reported HA-SAB rate as the outcome marker for their programmes. Improvement in hand hygiene compliance was matched with improvement in the HA-SAB rate at Auckland DHB but this has not been replicated at a national level despite significant improvements in hand hygiene compliance.[14,15]

In 2017 the Commission assessed the accuracy of individual DHB reporting of the denominator (bed-days) for the HA-SAB rate. Analysis revealed inconsistencies in reported numbers; both under- and over-reporting of bed days so it decided that the denominator data would be obtained from the National Minimum Data Set (NMDS). The Commission developed an implementation guide to support and standardise the application of the definition, which included flow charts and a set of clinical scenarios providing guidance on how to apply the HA-SAB definition.[16] Webinars were held to educate and support consistency in the application of the definition. This was in keeping with the recommendation by World Health Organization guidance on core competencies of infection prevention and control programmes that "a national training programme for performing surveillance should be established to ensure the appropriate and consistent application of national surveillance guidelines and corresponding implementation toolkits".[17]

Following this work the aggregated national rate of HA-SAB has increased and remains stable at a rate of 0.13 HA-SAB per 1,000 bed days (Figure 1). The increase can

Figure 1: Outcome marker, healthcare-associated *Staphylococcus aureus* bacteraemia per 1,000 bed days by month, March 2012–March 2019.



be attributed to increased HA-SAB rates at several referral DHB.

The rate of HA-SAB is used as a national performance indicator in countries such as Australia and Scotland, and as a measurement of the process of care to improve outcomes for patients with SAB by individual hospitals in other countries.[18–21]

In 2008 the Australian Health Ministers endorsed the reporting of HA-SAB cases occurring in public hospitals by states and territories as part of performance reporting under the National Healthcare Agreement. The performance benchmark for public HA-SAB was set at no more than 2.0 per 10,000 days of patient care for acute care public hospitals by 2011–12 in each state and territory.[18] Between 2012–13 and 2015–16 the rate of hospital-associated SAB has decreased from 0.94 cases to 0.74 cases per 10,000 days of patient care. The overall rate of HA-SAB in all public hospitals in Australia for 2016–17 was 0.76 cases per 10,000 days of patient care.[18]

The burden of HA-SAB in Australia sits within principal referral hospitals. Principal referral hospitals provide a very broad range of highly-specialised services and have large patient volumes. These hospitals accounted for 54% of all events and for 37% of patient care under surveillance and in 2016–17 the rate ranged from 1.01–1.23 HA-SAB cases per 10,000 days of patient care. Unlike Australia, there has been no subset analysis of New Zealand HA-SAB events based on hospital size or complexity to determine where the burden of diseases is located.

Despite similarities between the definition used for determining the rate of HA-SAB in Australia and HA-SAB in New Zealand the two rates are not comparable for a number of reasons. Firstly, a significant proportion

of hospital beds in Australia are within the private sector; in the 2016–17 reporting period a total of 89 private hospitals reported HA-SAB data accounting for only 14.1% of all known private hospitals.[18] The national HA-SAB rate in private hospitals who had reported data was lower than the national benchmark at 0.38 cases per 10,000 days of patient care. Secondly, patients in Australia can move between privately and publicly funded care with limited linkage of the care provided. In contrast, in New Zealand there are a limited number of private surgical beds, and in situations where complications arise the patient is often transferred to or re-admitted to a DHB hospital. And thirdly, New Zealanders have a national health index number and this allows individuals to be uniquely identified for the purposes of treatment and care, and for maintaining medical records in DHB hospitals. This supports sharing of information between DHB healthcare providers about events such as HA-SAB.

Healthcare-associated infections were nominated as a priority area by the Australian Commission for Safety and Quality in Health Care (ACSQHC) and a range of national and local initiatives have been established to reduce the occurrence with leadership provided by ACSQHC. These initiatives include a national hand hygiene improvement programme, Hand Hygiene Australia, national infection control guidance which includes information on managing medical devices such as vascular access devices and urinary catheters, building capacity to address skill and knowledge gaps, an antimicrobial stewardship initiative and a national surveillance initiative to monitor healthcare-associated infections.

Likewise, in 2010 when the Commission was established the infection prevention and control programme was one of the first programmes implemented. This led to the reinvigoration of the Hand Hygiene New Zealand programme, a national initiative to reduce central line associated blood stream infections in intensive care and high dependency units in New Zealand, Target CLAB Zero, and the establishment of a national surgical site infection improvement programme.[23–25]

The recent work to improve the consistency of the application of the HA-SAB definition has resulted in a more accurate outcome measure to monitor improvement in response to these quality improvement initiatives. Hand hygiene plays a significant role in reducing the transmission of bacteria between patients and surfaces within a healthcare setting. The Hand Hygiene Australia programme has shown a reduction in HA-SAB in Australian hospitals eight years after implementation.[26] Hospitals where a number of improvement interventions have been implemented have shown sustained reductions in methicillin-resistant SAB.[27] It should be acknowledged that improving hand hygiene compliance alone is not the only activity associated with a reduction in HA-SAB rates. Unpublished New Zealand data indicates that about 50% of all HA-SAB are associated with vascular access devices, and 20% are associated with surgery or other procedures (personal communication, N. Grae). About 15% have no clear source. Interventions shown to improve the adherence to best practice for the insertion and maintenance of central and peripheral vascular access devices are associated with reduced HA-SAB.[28,29] More recently the Commission has piloted a perioperative staphylococcal decolonisation bundle, termed 'the anti-staph bundle', to reduce the risk of *S. aureus* surgical site infections in patients undergoing cardiac surgery and hip and knee arthroplasties. Preliminary results look promising (personal communication, N. Grae) and are in keeping with the research showing that anti-staph bundles reduce *S. aureus* infections.[30]

In conjunction with sustaining hand hygiene compliance other quality improvement initiatives targeting interventions known to reduce HA-SAB are required. The major areas for improvement in process are the use of vascular access devices; especially long-term central vascular devices and peripheral intravascular catheters (PIC). The latter devices are in common use; nearly half of all adult inpatients at Auckland District Health Board have a PIC, of which 20% had no apparent clinical indication (personal communication, S. Muttaiyah). Implementation of the 'anti-staph bundle' as part of the perioperative care for a wider

range of surgical procedures and across all DHBs should also be considered. This intervention has been shown to reduce SSI rates and is cost-effective.[31]

Māori carry an unacceptable burden of *S. aureus* disease and we need to work in partnership with Māori to reduce this inequity both at a secondary and primary healthcare level.[6–8] Māori children have higher rates of colonisation with *S. aureus* and as a consequence, higher rates of skin and soft tissue infection.[32] There is limited knowledge about the rate of *S. aureus* colonisation in adults in New Zealand. One study of mostly young people (age range 15–24) showed that 18% had nasal colonisation with *S. aureus* but the study had a number of limitations.[33] We argue that adults residing in households with children colonised with *S. aureus* may also have higher rates of *S. aureus* colonisation increasing their risk of healthcare-associated *S. aureus* infection should they require admission. Colonisation with *S. aureus* increases the risk of surgical site infections three- to ten-fold.[34] A better understanding of the *S. aureus* colonisation across all age and ethnic groups is required to better inform prevention strategies.

To reduce the burden of healthcare-associated infections caused by *S. aureus* in New Zealand there needs to be a commitment at a national level to implement interventions aimed at reducing them. A collaborative quality improvement approach should be taken to share expertise and experiences across all DHBs and the private surgical hospital sector; the production of guidelines alone will not be effective. National programmes such as 'Target CLAB Zero' and the SSII programme have shown reduced harm to patients and similar initiatives to reduce peripheral vascular access device infections are needed. These programmes should be linked across primary, secondary and tertiary care and be co-designed with consumers to increase the likelihood of success. In conjunction there needs to be an increased focus on improving skin care in young New Zealanders, particularly young Māori and Pacific children. In healthcare settings the most obvious areas to target include the practices of inserting and maintaining vascular access devices and reducing skin colonisation with *S. aureus* prior to any surgery.

Reducing the rates of HA-SAB is crucial to improve the outcomes for all accessing healthcare in New Zealand. No more need for counting: time for action.

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Nil.

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REFERENCES:

1. Tong SYC, Davis JS, Eichenberger E, Holland TL, Fowler VG. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin Microbiol Rev. 2015; 28:603–660.
2. Hill PC, Birch M, Chambers S, et al. Prospective study of 424 cases of Staphylococcus aureus bacteraemia: determination of factors affecting incidence and mortality. Int Med J. 2001; 31:97–103.
3. Hill PC, Wong CGS, Voss LM, et al. Prospective study of 125 cases of Staphylococcus aureus bacteraemia in children in New Zealand. Pediatr Infect Dis J. 2001; 20:868–73.
4. Turnidge JD, Kotsanas D, Munckhof W, et al. Staphylococcus aureus bacteraemia: a major cause of mortality in Australia and New Zealand. Med J Aust. 2009; 191:368–73.
5. McMullan BJ, Bowen A, Blyth CC, et al. Epidemiology and mortality of Staphylococcus aureus bacteraemia in Australian and New Zealand children. JAMA Pediatr. 2016; 170:979–86.
6. O'Sullivan CE, Baker MG, Zhang J. Increasing hospitalisations for serious skin infections in New Zealand children, 1990–2007. Epidemiol Infect. 2011; 139:1794–804.
7. Williamson DA, Zhang J, Ritchie SR, Roberts SA, Fraser JD. Baker MG. Staphylococcus aureus in New Zealand, 2000–2011. Emerg Infect Dis. 2014; 20:1156–61.
8. Williamson DA, Lim A, Thomas MG, et al. Incidence, trends and demographics of Staphylococcus aureus infections in Auckland, New Zealand, 2001–2011. BMC Infect Dis. 2013; 13:569.
9. Bishara J, Goldberg E, Leibovici L, Samra Z, Shaked H, Mansur N, Pail M. Healthcare-associated vs. hospital-acquired Staphylococcus aureus bacteremia. Int J Infect Dis. 2012; 16:e457–463
10. Collignon P, Cruikshank M, Dreimanis D. Staphylococcus aureus bloodstream infections: an important indicator for infection control. Healthcare Infect. 2009; 14:165–71.
11. Worth L, Spellman T, Bull A, et al. Staphylococcus aureus bloodstream infection in Australian hospitals: findings from a Victorian surveillance system. Med J Aust. 2014; 200:282–4.
12. van Hal SJ, Jensen SO, Vaska VL, Espedido BA, Paterson DL, Gosbell IB. Predictors of mortality in Staphylococcus aureus bacteraemia. Clin Microbiol Rev. 2012; 25:362–86.
13. Report of the Controller and Auditor-General. Management of Hospital-Acquired Infection. Office of the Auditor-General New Zealand, Vol 1. June 2003 ISBN 0-478-18105-1.
14. Roberts SA, Sieczkowski C, Campbell T, Balla G, Keenan A. Implementing and sustaining hand hygiene culture change programme at ADHB. NZ Med J. 2012; 125(1354):75–85.
15. Freeman JT, Dawson L, Jowitt DM, et al. The impact of the Hand Hygiene New Zealand programme on hand hygiene practices in New Zealand public hospitals. NZ Med J 2016; 129(1443):67–76.
16. Health Quality & Safety Commission New Zealand. Implementation guide for the surveillance of Staphylococcus aureus bacteraemia (SAB). August 2017 Accessed 2nd July 2019 http://www.hqsc.govt.nz/assets/Infection-Prevention/Hand-Hygiene/PR/HHNZ_HA_SAB_Implementation_Guide.pdf
17. Guidelines on core components of infection prevention and control programmes at the national and acute health care facility level. Geneva: World Health Organisation; 2016. Licence: CC BY-NC-SA.3.0 IGO
18. Australian Institute of Health and Welfare 2017. Staphylococcus aureus bacteraemia in Australian public hospitals 2016–17: Australian hospital statistics. Health services series no. 83. Cat. No. HSE 198. Canberra AIHW ISBN 978-1-76054-272-6.
19. Murdoch F, Danial J, Morris AK, et al. The Scottish enhanced Staphylococcus aureus bacteraemia surveillance programme: the first 18 months of data in adults. J Hosp Infect. 2017; 97:133–9.
20. Dendle C, Martin RD, Cameron DR, et al. Staphylococcus aureus bacteraemia as a quality indicator for hospital infection control. Med J Aust. 2009; 191:389–92.
21. Rosa R, Wawrzyniak A, Sfeir M, Smith L, Abbo LM. Performance of care and outcomes in patients with Staphylococcus aureus bacteraemia. J Hosp Med. 2016; 11:27–32.
22. Lam JC, Gregson DB, Robinson S, et al. Infectious diseases consultation improves key performance metrics in the management of Staphylococcus aureus bacteraemia: a multicentre cohort study. JAMMI 2019; 4:24–32.

23. Gray J, Proudfoot S, Power M, Bennett B, Wells S, Seddon M. Target CLAB Zero: a national improvement collaborative to reduce central line-associated bacteraemia in New Zealand intensive care units. NZ Med J 2015; 128(1421):13–21.

24. Morris AJ, Panting AL, Roberts SA, Shuker C, Merry AF. A new surgical site infection improvement programme for New Zealand: early progress. NZ Med J 2015; 128(1414):51–9.

25. Morris AJ, Roberts SA, Grae N, Hamblin R, Shuker C, Merry AF. The New Zealand Surgical Site Infection Improvement Programme: a national quality improvement programme reducing orthopaedic surgical site infections. NZ Med J 2018; 131(1479):45–56.

26. Grayson ML, Stewardson AJ, Russo PL, et al, Effects of the Australian National hand Hygiene Initiative after 8 years on infection control practices, healthcare worker education, and clinical outcomes: a longitudinal study. Lancet Infect Dis. 2018; 18:1269–77.

27. Evans ME, Kralovic SM, Simbartl LA, Jain R, Roselle GA. Eight years of decreased methicillin-resistant Staphylococcus aureus healthcare-associated infections associated with a Veterans Affairs prevention initiative. Am J Infect Control. 2017; 45:13–6.

28. Kok J, O'Sullivan MV, Gilbert GL. Feedback to clinicians on preventable factors can reduce hospital onset Staphylococcus aureus bacteraemia rates. J Hosp Infect. 2011; 79:108–14.

29. Blauw M, Foxman B, Wu J, Rey J, Kothari N, Malani AN. Risk factors and outcomes associated with hospital-onset peripheral intravenous catheter-associated Staphylococcus aureus bacteraemia. OFID 2019; 6:1–6.

30. Ma N, Morris AJ, Grae N, Roberts SA, Systematic review of a patient care bundle in reducing staphylococcal infections in cardiac and orthopaedic surgery. ANZ J Surg 2017; 87:239–46.

31. Rennert-May E, Conly J, Smith S, et al. A cost-effective analysis of mupirocin and chlorhexidine gluconate for Staphylococcus aureus decolonization prior to hip and knee arthroplasty in Alberta Canada compared to standard of care. Antimicrob Res Infect Control 2019; 8:113.

32. Hobbs MR, Grant CC, Thomas MG, et al. Staphylococcus aureus colonisation and its relationship with skin and soft tissue infection in New Zealand Children. Eur J Clin Microbiol Infect Dis 2018; 37:2001–10.

33. Best N, Fraser JD, Rainey PB, Roberts SA, Thomas MG, Ritchie SR. Nasal carriage of Staphylococcus aureus in healthy Aucklanders. NZ Med J 2011; 124(1332):31–9.

34. Sakir A, Brégeon F, Mège JL, Rolain JM, Blin O. Staphylococcus aureus nasal colonisation: an update on mechanisms, epidemiology, risk factors and subsequent infections. Front Microbiol. 2018; 9:2419.

Nutrition guidelines for dental care vs the evidence: is there a disconnect?

Sarah Hancock, Caryn Zinn, Grant Schofield, Simon Thornley

ABSTRACT

Dental caries is the most common chronic childhood disease in New Zealand. Concurrently, obesity and related chronic metabolic diseases are the most challenging public health problems of modern times. There is considerable evidence that a common dietary behaviour—high frequency consumption of sugar- and starch-containing foods—is the principal aetiological factor for both dental caries, and presentation of children and young people with increased adiposity or obesity. Conversely, consumption of full-fat dairy products by children and young people is associated with reduced risks of dental caries and obesity. Government-endorsed dietary guidelines for young people correctly provide recommendations to decrease intake of high-sugar foods. However, recommendations are provided to increase the frequency of consumption of sugar- and starch-containing foods as children age, and to choose low-fat dairy produce. We contend that this advice directly contradicts evidence of the dietary causes of both dental caries and obesity. This advice also does not reflect evidence regarding observed associations between the consumption of full-fat dairy produce and reduced dental caries and obesity. We present evidence to support our contention that important elements of New Zealand's dietary guidelines have been established without due consideration of the entirety of the evidence, including that which is updated, recent or evolutionarily. Given the epidemics of dental caries and metabolic disease are ongoing public health challenges in New Zealand and share common dietary causes, guidelines for healthy eating should limit refined sugar- and starch-containing foods and encourage intake of full-fat dairy items.

Dental caries is the most common chronic childhood disease in New Zealand.[1] The most recent data from the Ministry of Health (MOH) showed that 40.5% of five-year-old children were diagnosed with dental caries in 2017, with a higher prevalence observed among Māori and Pacific children compared to children of other ethnicities. In addition, New Zealand is experiencing an obesity epidemic; New Zealanders are the third most overweight and obese population among countries included in the OECD.[2] Approximately 67% of all New Zealanders over 15 years of age are overweight and the prevalence of obesity is 27%.[1]

There is now increasing scientific and public health debate that the common dietary behaviour—the high frequency consumption of sugar and starches and a concurrent reduced intake of nutrient-rich whole foods—is central to the development of dental caries, malnutrition in young children[3] and obesity in older children.[4] It is proposed that the consumption of ultra-processed sugars and starches leads to increased insulin secretion and dysregulation of the satiety hormone leptin, resulting in a cycle of increased hunger and frequent food consumption that promotes weight gain.[5] MOH-endorsed dietary guidelines for healthy eating in children and young people in New Zealand for healthy weight-management[6] correctly includes advice to limit consumption of high-sugar foods. However, these guidelines include advice for children and young people to derive most of their energy from grain-based foods and to increase the frequency of consumption of these foods as children move into adolescence. In addition, recommendations to consume dairy products involve promotion of low-fat options, despite evidence that consumption of full-fat dairy products is associated with benefits

for both oral health,[7] and in weight status and markers of cardiometabolic health in children and adolescents.[8] Policy in New Zealand for dental health is focused on prevention of caries in young children with a substantial emphasis on oral hygiene promotion, and recommendations for the consumption of a diet that broadly follows MOH guidelines.

We contend that such recommendations do not reflect the best available evidence for both healthy weight management and maintenance of optimal oral health. Given dental caries is the most common chronic childhood disease in New Zealand, it is imperative that recommendations for healthy eating for New Zealanders reflect the evidence around diet and nutrition for the prevention of dental caries alongside that for other conditions. The consideration of the hormonal theory of metabolic health may resolve the apparent contradictions between the evidence of dietary causes of caries and obesity, and advice provided in dietary guidelines. What follows is an appraisal of the evidence and the state of the current dietary guidelines for children and young people relating to three key areas of diet in dental caries and obesity—sugar, sugar and starch, and full-fat dairy products.

Diet and dental caries

Dental caries can be defined as the localised destruction of susceptible dental hard tissues by the acidic by-products from bacterial metabolism of fermentable carbohydrates.[9] Carious lesions on teeth form through a complex interaction between acid-producing bacteria and consumption of fermentable sugars and is influenced by a range of host factors including saliva and dental plaque. Reports from the MOH from 2017 indicate that 40.5% of New Zealand children present with dental caries at age five years. Further, the prevalence of caries measured in young adolescents in New Zealand is estimated to be 38.6%.[1]

The relationship between high dietary intakes of sugar (sucrose) and dental caries is well established. An extensive systematic review of the relationship between sugar intake and dental caries was conducted by Moynihan and Kelly.[10] Positive associations between free sugar intake and dental caries were found in 42 out of 50 studies in children, and in all five studies of adults. These links are both epidemiologically and biologically indicated through several lines of evidence, including studies on the action of bacteria that reside in the oral cavity on a range of sugars in the mouth.[11] Time-series analyses of country-level datasets show low levels of dental caries prior to the consumption of refined sugar, and within-person observational studies also show the central role of sugar in the aetiology of dental caries.[10]

Other fermentable carbohydrates, particularly sugar- and starch-containing foods are also associated with an increased risk of dental caries. In the Western diet, processed starches are found in a wide range of foods and constitute a high percentage of total dietary carbohydrate.[9] Studies of the human pH response to a range of starches and enamel/dentin demineralisation show that both sugars and starches are associated with cariogenic activity.[12] The total time that plaque pH remains below critical levels of 5.5 upon exposure to processed starch- and sugar-containing foods exceeded that of foods containing high levels of sucrose alone. Through this extended retention of starch-derived sugars in plaque, the effects of other simultaneously present sugars may be prolonged; in which event the consumption of starch has a co-cariogenic effect with that of other sugars. The results from prospective cohort studies show that foods with a relatively low level of sugar but a high proportion of starch were associated with increased caries risk,[7] and starch has been identified as an effects modifier in the relationship between dental caries and foods with low sugar levels.[7]

Conversely, results from prospective cohort studies[7,13] and systematic reviews indicate that consumption of full-fat dairy products by children and young people is associated with reduced risks of dental caries and obesity. Neutral or inverse associations were observed in a systematic review by Dror and Allen[14] between consumption of milk and dairy products in children and adolescents and dental caries incidence, body fatness and hypertension.[14] In addition, dairy product intake was associated with benefits for bone mineralisation and blood pressure. A recent systematic review of 43 cross-sectional studies and 31 prospective studies by Dougkas et al[8] found

that intake of milk and other dairy products are consistently found to be either not associated or inversely associated with obesity and indicators of adiposity in children, and that there is little evidence to support advice to limit consumption of dairy products for children on the grounds that they may promote obesity.[8] Another review by Guo et al[15] found a neutral or moderate inverse association between dairy consumption and the risk of developing type-2 diabetes.

The main strategy for the prevention of tooth decay adopted worldwide has been to target children, on the assumption that if caries is prevented in this group, the burden of dental disease would be reduced for all. Strategies for oral health promotion in New Zealand include teaching effective oral hygiene practices, facilitating early access to preventative dental services, promoting use of topical fluorides in toothpaste and promoting healthy eating. However, young New Zealanders present with caries, despite the promotion of a range of oral healthcare practices for the prevention of tooth decay, and caries increases through adolescence and adulthood.[16] The focus of conventional dental treatment is primarily on the endpoint of disease and remedial work.[17] We argue that the current dental delivery system is not effective in achieving health improvements because this approach does not fully consider the primary cause of disease, the impact of diet and dietary behaviours on dental caries, and the common risk factors for dental caries, other oral diseases and obesity.

Dental caries, obesity and common risk factors

There is mixed evidence about the relationship between the prevalence of dental caries and obesity in children and adolescents. On the one hand, studies in young children have shown that early childhood caries are associated with reduced growth and malnutrition due to insufficient consumption of nutrient-rich food to meet the metabolic and growth needs of children less than two years of age.[18] On the other hand, there are stronger associations observed between dental caries, and increased adiposity and obesity in older children and adolescents. Several systematic reviews have examined the cross-sectional relationship between dental caries and increased adiposity using anthropometric measures, including body mass index (BMI) and waist-hip ratio.[19] Hayden et al[20] reported a statistically significant relationship between adolescent obesity and dental caries (effect size 0.104, p=0.049).[20] Similarly, in Chen et al,[21] sensitivity analysis work showed obese individuals presented with more dental caries than normal-weight children in their primary teeth (WMD 0.52, 95% CI 0.17 to 0.87, p=0.026). Finally, in a recent prospective cohort study by Li et al, longitudinal associations were observed between dental caries and central obesity measured by a waist-hip ratio among adolescents aged 15–18 years.[22]

An important caveat when evaluating studies of food consumption, dental caries and obesity, is that much of the evidence comes from cross-sectional and prospective cohort studies. Hence, the observed correlations and relationships cannot be regarded as causal and the findings should be interpreted with some caution.

There is emerging evidence that the 'calories in, calories out' model of healthy weight management is too simplistic and ignores the complex nutritional, metabolic and hormonal effects of food. The complex hormonal, neural and microbial feedback systems are likely to be critical in the regulation of satiety, energy partitioning and consequently, body composition. Very young children with decayed teeth may not achieve satiety from meals comprised of a high proportion of nutrient-poor, processed foods, which in turn could then lead to between-meal snacking,[23] and abnormally high levels of insulin secretion.[5] High levels of insulin production are also associated with the disruption of the satiety hormone leptin, which regulates energy balance and inhibits hunger by signalling to the brain when satiety is achieved from food consumption.[5] The consequence of the disruption to leptin promotes a cycle in which hunger and food consumption increases, leading to weight gain.[5] Another point is that the frequent consumption of refined snacks comprising sugar and starches is also associated with food addiction characterised by similar behavioural, neurochemical and brain activation responses similar to those of substance abuse.[24]

Treatments for obesity include dietary interventions, medication, advice to engage in physical exercise, and for morbid obesity: bariatric surgery. There is general agreement within the scientific literature that effective interventions for obesity should comprise dietary interventions to improve metabolic health and achieve weight loss through the consumption of nutrient-rich whole foods,[25] long acknowledged to be associated with benefits for dental health and longevity in studies of dietary lifestyles both evolutionarily and modern.[26]

The predominant dietary prescription in weight loss trials and considered as "best practice" by the MOH is a diet based on a moderate-to-high carbohydrate, moderate protein and low fat intake, with a specific restriction on saturated fat, in the context of an energy deficit.[27] However, there is compelling evidence from randomised controlled trials that a restricted carbohydrate intake improves adiposity, glucose metabolism and lipid markers over and above that of the mainstream guidance system.[28] These results suggest that a carbohydrate-restriction model should feature prominently in the guidance strategy for promoting health and managing health conditions, and perhaps even be considered the first-line approach in the management of obesity. This approach typically includes less frequent eating, which may impart a dual benefit for caries prevention.

Dietary advice in New Zealand, and contradictions with scientific evidence

New Zealand dietary recommendations for both optimal oral health and weight management usually include advice to adhere to mainstream food and nutrition guidelines. "The Food and Nutrition Guidelines for Healthy Children and Young People (Aged 2–18 years)" are derived from a background paper produced with the purpose of providing background information for health practitioners who provide nutrition advice and develop nutrition programmes.[6] These guidelines, endorsed by the MOH, also provide the basis for developing health education resources that are for use by the general public and focus on this age group.

A significant feature of these guidelines is advice to prepare foods or choose pre-prepared foods that are low in sugar, especially added, and to limit the intake of drinks such as cordials, fizzy drinks (including diet drinks and sports drinks) and refined clear fruit juices. The authors of the guidelines recognise that foods high in sugar generally provide very few vitamins and minerals relative to their energy content and that limiting consumption of these foods can be difficult because they are widely available, often inexpensive and heavily marketed to children and young people. The guideline authors also correctly acknowledged the displacement of healthier foods from the diet, given that these foods and drinks contribute to 20% of total energy intake.

However, features of these recommendations are the inclusion of cereals and other processed carbohydrates including breads and baked goods, and strong encouragement to consume low-fat dairy products wherever possible. Additional advice is provided for increasing the frequency of consumption of carbohydrate-containing foods for young people. The advice offered in these guidelines supports high and frequent consumption of carbohydrates. This means that promoted foods include processed items such as breads and cereals, as defined by the NOVA classification system which classifies foods in relation to their degree of processing.[29] Further, included in the MOH food suggestions are refined carbohydrates (such as cornflakes) and foods listed in the highest NOVA category of ultra-processed foods, such as plain sweet biscuits and tortillas. This advice for children contradicts evidence of the dietary causes of dental caries and contradicts the nutritional, metabolic and hormonal theory of metabolic health as the cause of obesity. Consumption of breads, cereals and other ultra-processed refined sugar- and starch-containing foods are associated with dental caries,[7] is implicated in insulin resistance[5] and is associated with obesity in older children and adolescents.[20]

Since the introduction of dietary guidelines to reduce fat consumption, data from a range of reviews suggests the replacement of saturated fat with sugars and starches are correlated with worldwide obesity and diabetes epidemics.[30] Recommendations to consume low-fat dairy products were based on the hypothesis that saturated fats were the primary dietary cause of cardiovascular

disease. Although the anti-cariogenic properties of milk products are acknowledged in the guidelines, recommendations about dairy intake promote low-fat dairy products, in direct opposition to epidemiological evidence. Full-fat dairy products have not been associated with obesity in child and adolescent populations, and studies indicate that whole milk consumption is associated with favourable effects on body composition and lipid profiles of children.[8,14] In Table 1, specific recommendations provided in the guidelines that relate to healthy eating for weight management and prevention of dental caries are summarised.

Table 1: Specific food and nutrition guidelines for healthy eating in children and young people pertaining to dietary factors and evidence for prevention of obesity and dental caries in young people.

Dietary factor	**Weight management**		**Optimal oral health**
Carbohydrate intake	"Eat a variety of foods from each of the four major food groups each day…" • … breads and cereals, increasing wholegrain products as children increase in age.		"Eat a combination of foods at each meal, including whole grains…" "Good oral hygiene and minimising intake of cariogenic foods and drinks are key behaviours in preventing dental caries."
	Recommendations for the "breads and cereals" food group		
	Specific foods included	***Recommendation (per day)***	***Serving size examples***
	All breads, cereals, rice and pasta (increasing wholegrain options as children age)	Pre-schoolers: at least 4 servings Children: at least 5 servings Young people: at least 6 servings	1 medium slice of bread (26 g) 1 roll (50 g) 1 pita pocket or tortilla (50–80 g) 2 breakfast wheat biscuits (34 g) ½ cup muesli (55 g) ½ cup porridge (130g) 1 cup cornflakes (30 g) 1 cup cooked pasta or rice (150 g) 4 grainy crackers (40 g) 2 plain sweet biscuits (14 g) 1 cup plain popcorn
Fat intake	**Weight management**		**Optimal oral health**
	"Eat a variety of foods from each of the four major food groups each day… • milk and milk products or suitable alternatives, preferably reduced or low-fat options. "Reduced or low-fat milk and milk products are the best choices because these foods include less saturated fat, and often more protein and calcium than high-fat alternatives."		"Eat a combination of foods at each meal, including whole grains, vegetables and fruit." "Anti-cariogenic foods and drinks are those that promote tooth remineralisation. They include foods high in calcium, phosphate and protein, such as milk and milk products."
	Recommendations for the "milk and milk products" food group		
	Specific foods included	***Recommendation (per day)***	***Serving size examples***
	Milk (includes calcium-fortified milk alternatives), cheese and yoghurt (choose low-fat options)	Pre-schoolers and children: at least 2–3 servings Young people: at least 3 servings	Glass of milk or calcium-fortified milk alternative (250 ml), one pot of yoghurt (150 g) 2 slices of cheese (40 g)

Table 2: Specific food and nutrition guidelines for healthy eating in children and young people relating to meal patterns and evidence for prevention of obesity and dental caries in young people.

	Weight management	**Optimal oral health**
Food consumption frequency	"Three meals and two to three small snacks, at regular times during the day, are recommended for children and young people." "Continuous eating or 'grazing' is not recommended." "Examples of healthy and nutritious snacks are: fruit, yoghurt, vegetable sticks with a low-fat dip (eg, hummus or yoghurt-based dips), mini-sandwiches, mini homemade 'pizzas', 'mousetraps' (toasted cheese and yeast extract spread on bread), nuts and milk."	"Have no more than six meals (including snacks) per day to allow time for teeth to re-mineralise between meals." "Choose snacks such as yoghurt and cheese, which are low in fermentable carbohydrate and promote tooth remineralisation." "Limit sugary foods and drinks, especially those that remain in the mouth for an extended time or are more likely to stick to teeth, for example, hard or chewy sweets, dried fruit, roll-ups and lollipops."

Part 2 of "The Food and Nutrition Guidelines for Healthy Children and Young People (Aged 2–18 years)"[6] outlines recommendations for meal patterns of New Zealand children and young people. Three meals plus 2–3 small snacks are recommended during the day at regular times for children and young people. Examples of healthy and nutritious snacks are, according to the guideline: fruit, yoghurt, vegetable sticks with a low-fat dip (eg, hummus or yoghurt-based dips), mini homemade 'pizzas', 'mousetraps' (toasted cheese and yeast extract spread on bread), nuts and milk. Frequent consumption of carbohydrate-containing foods is also associated with increases in insulin production, down-regulation of leptin hormone action and consequent metabolic dysregulation that promotes an increase in appetite and an alteration of weight homeostasis towards weight gain.[17]

The recommendation to consume food at a frequency of up to six eating occasions daily contradicts a well-established body of evidence showing a strong relationship between a high-frequency food consumption, and dental caries and the onset of obesity in young people. The inclusion of mini sandwiches and mini homemade "pizzas" as recommended snack foods is also questionable given the high carbohydrate load of these foods. This advice is not informed by evidence that shows clear relationships between the frequent intake of fermentable carbohydrates and the increased risk of dental caries, and other disorders attributable to a high carbohydrate diet. On the contrary, we could find no direct evidence to support both increased consumption of wholegrain products as children age, and to eat from four food groups as stipulated in the guidelines outlined in Table 1. We could not find evidence to support the recommended daily consumption of four, five and six servings per day of breads and cereals of breads for pre-schoolers, children and young people, respectively. In addition, given the anti-cariogenic properties of milk and other full-fat dairy products above, the advice to consume carbohydrate-containing foods as snacks rather than dairy-based food items is likely to be counterproductive.

Conclusions

In conclusion, we have presented evidence that the consumption of foods containing sugar and starches is the principal aetiological factor in dental caries. Intake of these foods are implicated in the increased prevalence of both malnutrition and obesity

in children and young people. However, the guidelines for "healthy eating" for children and young people have been produced without consideration of the entirety of evidence relating to the role of not only sugars but also starches, in the aetiology of dental caries and obesity. Furthermore, the evidence relating to reductions in dental caries and adiposity, and markers of cardiometabolic health in children with higher intakes of full-fat dairy products is not reflected in national guidelines. Not only do the dietary guidelines not incorporate the available evidence on healthy eating to prevent dental caries and obesity in young New Zealanders, much of the advice contradicts epidemiological evidence relating to risk factors for dental caries and obesity. Given that the epidemics of dental caries and obesity are a significant and ongoing public health challenge in New Zealand, it is imperative that the guidelines for healthy eating for young New Zealanders incorporate the best dietary advice to improve their health. It is time to update the guidelines and include a dental focus to limit sugar and starch intake and encourage intake of full-fat dairy products to prevent the epidemics of dental caries and obesity, which share a common cause.

Competing interests:
Dr Zinn and Professor Schofield are co-authors of a book series titled "What The Fat", which assumes a low carbohydrate, healthy fat nutrition approach.

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REFERENCES:

1. Ministry of Health, Annual update of key results 2017/18 New Zealand Health Survey. 2019, Ministry of Health: Wellington.
2. Beaglehole R. Sugar sweetened beverages, obesity, diabetes and oral health: a preventable crisis. Pac Health Dialog. 2014; 20(1):39–42.
3. Clarke M, Locker D, Berall G, et al. Malnourishment in a population of young children with severe early childhood caries. Pediatr Dent. 2006; 28(3):254–9.
4. Chi DL, Luu M, Chu F. A scoping review of epidemiologic risk factors for pediatric obesity: Implications for future childhood obesity and dental caries prevention research. J Public Health Dent. 2017; 77 Suppl 1:S8–S31.
5. Weiss R, Bremer AA, Lustig RH. What is metabolic syndrome, and why are children getting it? Ann N Y Acad Sci. 2013; 1281:123–40.
6. Ministry of Health, NZ, Food and Nutrition Guidelines for Healthy Children and Young People (Aged 2–18 years): A background paper. Ministry of Health. 2012, Partial revision February 2015.
7. Campain AC, Morgan MV, Evans RW, et al. Sugar-starch combinations in food and the relationship to dental caries in low-risk adolescents. Eur J Oral Sci. 2003; 111(4):316–25.
8. Dougkas A, Barr S, Reddy S, et al. A critical review of the role of milk and other dairy products

in the development of obesity in children and adolescents. Nutr Res Rev. 2019; 32(1):106–127.

9. Selwitz RH, Ismail AI, Pitts NB. Dental caries. Lancet. 2007; 369(9555):51–9.
10. Moynihan PJ, Kelly SA. Effect on caries of restricting sugars intake: systematic review to inform WHO guidelines. J Dent Res. 2014; 93(1):8–18.
11. Loesche WJ, Rowan J, Straffon LH, et al. Association of Streptococcus mutants with human dental decay. Infect Immun. 1975; 11(6):1252–60.
12. Kashket S, Zhang J, Van Houte J. Accumulation of fermentable sugars and metabolic acids in food particles that become entrapped on the dentition. J Dent Res. 1996; 75(11):1885–91.
13. Chankanka O, Marshall TA, Levy SM, et al. Mixed dentition cavitated caries incidence and dietary intake frequencies. Pediatr Dent. 2011; 33(3):233–40.
14. Dror DK, Allen LH. Dairy product intake in children and adolescents in developed countries: trends, nutritional contribution, and a review of association with health outcomes. Nutr Rev. 2014; 72(2):68–81.
15. Guo J, Givens DI, Astrup A, et al. The Impact of Dairy Products in the Development of Type 2 Diabetes: Where Does the Evidence Stand in 2019? Adv Nutr. 2019; Epub ahead of Print: (nmz050, http://doi.org/10.1093 advances nmz050).
16. Broadbent JM, Thomson WM, Poulton R. Trajectory patterns of dental caries experience in the permanent dentition to the fourth decade of life. J Dent Res. 2008; 87(1):69–72.
17. Jatrana S, Crampton P, Filoch, S. The case for integrating oral health into primary health care. The New Zealand Medical Journal. 2009; 122(1301):43–52.
18. Sheiham A. Dental caries affects body weight, growth and quality of life in pre-school children. Br Dent J. 2006; 201(10):625–6.
19. Aas JA, Griffen AL, Dardis SR, et al. Bacteria of dental caries in primary and permanent teeth in children and young adults. J Clin Microbiol. 2008; 46(4):1407–17.
20. Hayden C, Bowler JO, Chambers S, et al. Obesity and dental caries in children: a systematic review and meta-analysis. Community Dent Oral Epidemiol. 2013; 41(4):289–308.
21. Chen D, Zhi Q, Zhou Y, Tao Y, et al. Association between Dental Caries and BMI in Children: A Systematic Review and Meta-Analysis. Caries Res. 2018; 52(3):230–245.
22. Li LW, Wong HM, McGrath CP. Longitudinal Association between Obesity and Dental Caries in Adolescents. J Pediatr. 2017; 189:149–154.
23. Drummond BK, Meldrum AM, Boyd D. Influence of dental care on children's oral health and wellbeing. Br Dent J. 2013. 11): E27. doi:10.1038/sj.bdj.2013.533
24. Lennerz B, Lennerz JK. Food Addiction, High-Glycemic-Index Carbohydrates, and Obesity. Clin Chem. 2018; 64(1):64–71.
25. Troesch B, Biesalski HK, Bos R, et al. Increased Intake of Foods with High Nutrient Density Can Help to Break the Intergenerational Cycle of Malnutrition and Obesity. Nutrients. 2015; 7(7):6016–37.
26. Woelber JP, Bremer K, Vach K, Konig D, et al. An oral health optimized diet can reduce gingival and periodontal inflammation in humans - a randomized controlled pilot study. BMC Oral Health. 2016; 17(1):28–35.
27. Ministry of Health, New Zealand. Clinical Guidelines for Weight Management in New Zealand Children and Young People. 2016. Wellington, New Zealand.
28. Feinman RD, Pogozelski WK, Astrup A, et al. Dietary carbohydrate restriction as the first approach in diabetes management: Critical review and evidence base. Nutrition. 2015; 31(1):1–13.
29. Monteiro CA, Cannon G, Levy RB, et al. Ultra-processed foods: what they are and how to identify them. Public Health Nutr. 2019; 22(5):936–941.
30. Harcombe Z. Dietary fat guidelines have no evidence base: where next for public health nutritional advice? Br J Sports Med. 2017; 51(10):769–774.

Osteomyelitis following an undisplaced basal skull fracture

Kate Seddon, Christopher Low

Central skull base osteomyelitis is usually associated with malignant otitis externa or iatrogenic trauma, although cases without these precipitants have been described in at-risk patients (ie, diabetic, immunocompromised).[1–3] We are not aware of any reported cases of central skull base osteomyelitis following an undisplaced fracture of the temporal bone.

Case report

A 71-year-old male presented with an undisplaced fracture of the right squamo-mastoid portion of the temporal bone on CT scan, following an accidental fall. He is on insulin for type 2 diabetes. On clinical examination, he had a GCS 15/15 and a right haemotympanum. He was observed in a neurosurgical tertiary unit and was discharged after 10 uneventful days.

He re-presented the next day with a head injury following another fall. Clinically, he had incomplete cranial nerves IV and VII palsy and a right hearing loss with a perforated right tympanic membrane. A repeat CT head and an MRI showed a new acute subdural haemorrhage and extension of the basal skull fractures to the sphenoid sinus and pterygoid plate.

He was discharged with a low-grade right earache and non-specific headache. His right ear drum healed within weeks, with ongoing middle ear effusion. Neurologist and maxillofacial surgical opinion found no specific cause of his pains.

Eight months later he presented with gradual worsening of his headaches and fevers; he was treated empirically with systemic antibiotics for suspicion of meningitis. The treatment was discontinued when the lumbar puncture results were negative.

A month later he was readmitted with progressive worsening and severe headaches with raised inflammatory markers (CRP 107, WBC 14.0, Neut 11.8). He had bilateral ear effusions but no abnormality of the ear canals. An MRI brain and high-resolution contrast-CT head showed inflammation and destruction of the right central skull base with an associated soft tissue swelling and sigmoid sinus thrombosis, consistent with central skull base osteomyelitis. He was started on an eight-week course of IV metronidazole and cefepime. His headaches and inflammatory markers improved dramatically.

Discussion

Central skull base osteomyelitis is usually associated with malignant otitis externa or a complication of an invasive procedure. Cases without these precipitants have been reported, with the source of infection hypothesised as arising from the sinuses or blood.[3] Patients often have systemic risk factors,[3] two of which were present in this case: diabetes and old age.

The most common symptom of skull base osteomyelitis is non-specific headache, which can delay diagnosis, as in this case. Additional symptoms arise from complications; including cranial neuropathies, thrombosis and meningitis, which often occur late in the disease course.[2] In many cases inflammatory markers are normal;[2] however,

raised markers in our patient increased the suspicion of an occult infection.

Often blood cultures are negative and the pathogen is not identified. Previous cases associated with malignant otitis externa have often found *Pseudomonas aeruginosa* and gram-positive bacteria to be the common pathogens.[1,2]

CT imaging is generally regarded as a poor diagnostic modality for skull base osteomyelitis. MRI is the modality of choice[2] and helped to clinch the diagnosis in this case (Figure 1). Scans that are helpful in monitoring the progress of the treatment include WBC-labelled scans, gallium scintigraphy and PET-CT.

Osteomyelitis is treated aggressively with long-term IV antibiotics, with or without surgical debridement.[4] The antibiotic of choice is usually broad spectrum when the organism is not identified.

Figure 1: CT head taken following the patient's second fall. Right-sided otic sparing undisplaced fracture of the right squamo-mastoid portion of the temporal bone (1), extending to the sphenoid sinus (containing air fluid level) (2).

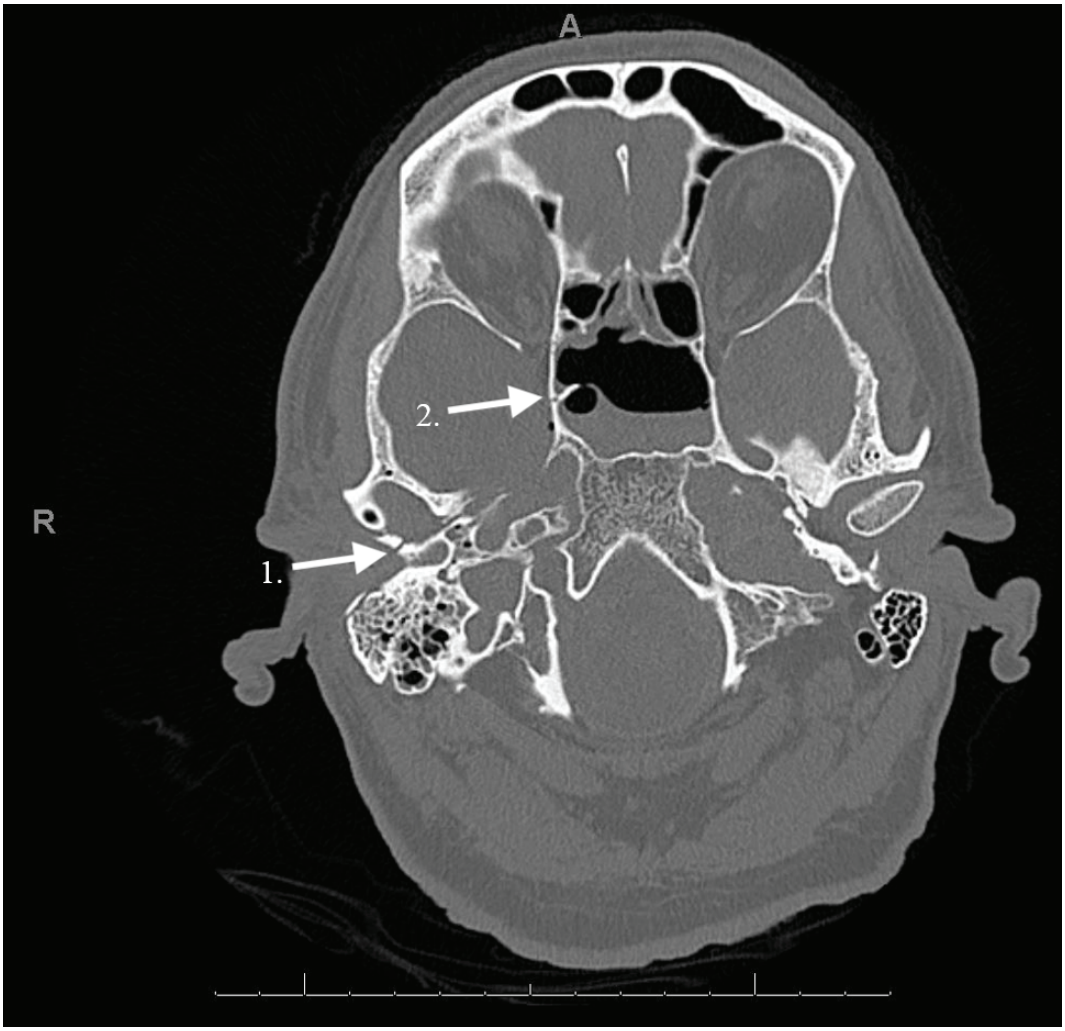

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Figure 2: CT head taken nine months after initial presentation. Soft tissue swelling anterior to the clivus (1), bony destruction anterior right sided aspect of clivus (2). Typical appearance of central skull base osteomyelitis.

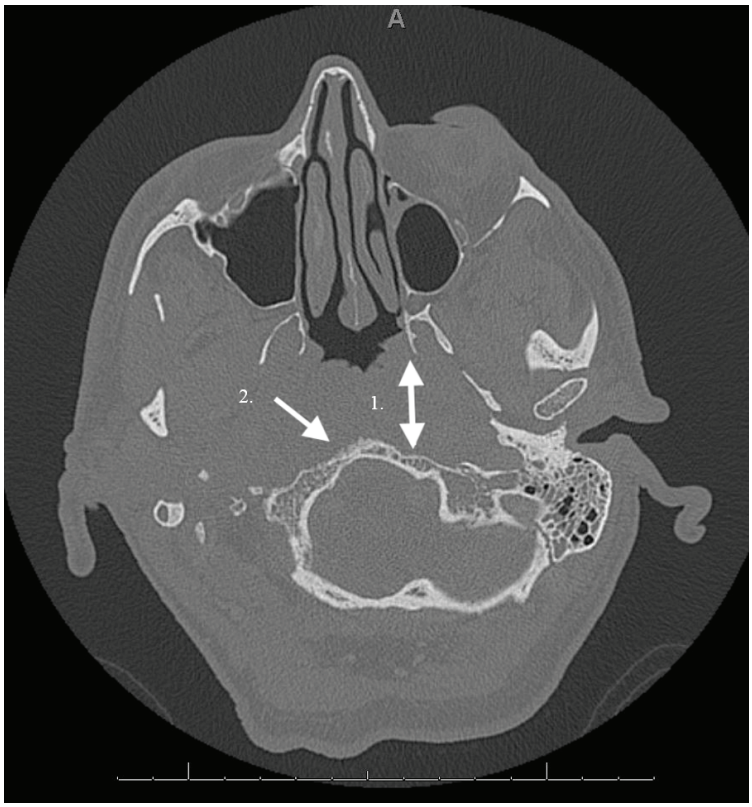
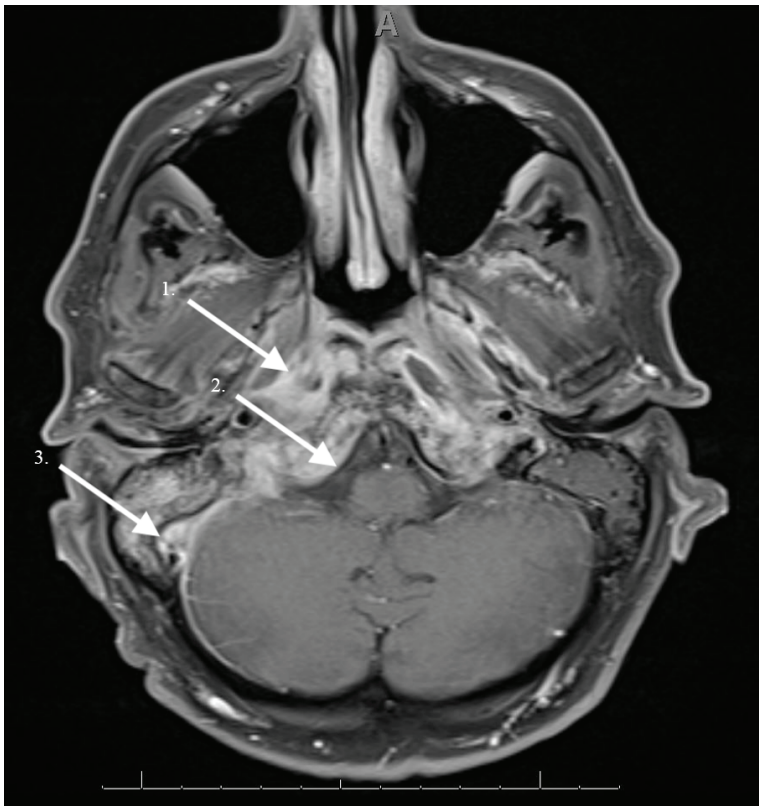


Figure 3: MRI brain (T1) taken nine months after initial presentation. Enhancement of the skull base and anterior soft tissue (1), loss of cortical bone right sided clivus (2), right sigmoid sinus thrombus (3). Typical appearance of central skull base osteomyelitis.



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https://www.nzma.org.nz/journal-articles/osteomyelitis-following-an-undisplaced-basal-skull-fracture

REFERENCES:

1. Chang, Fishbein, Holliday. Central Skull Base Osteomyelitis in Patients without Otitis Externa: Imaging Findings. American Journal of Neuroradiology, 2003; 24(7):1310–1316.
2. Khan, Quadri, Kazmi, Kwatra, Ramachandran, Gustin, Farooqui, Suriya Zafar. Presentations, a Comprehensive Review of Skull Base Osteomyelitis: Diagnostic and Therapeutic Challenges among Various. Asian Journal of Neurosurgery, 2018; 13(4):959–970.
3. Clark M, Pretorius P, Byren I, Milford C. Central or Atypical Skull Base Osteomyelitis: Diagnosis and Treatment. Skull Base, 2009; 19(4):247–254.
4. Baruah, Kumar, Haque. Acute Osteomyelitis in Closed Fracture of Adult Humerus- Case Report and Review of Literature. IOSR Journal of Dental and Medical Sciences, 2016.

The projected burden of knee osteoarthritis in New Zealand: healthcare expenditure and total joint provision—a response

Haxby Abbott, Ross Wilson

We thank Professors Gywnne-Jones and Hooper for their correspondence in regard to our projections of demand for total knee joint replacement (TKR) to 2013–2038.[1] They are correct that the estimates are based on the first TKR, and any revision of that TKR, for primary osteoarthritis (OA) in the population resident in New Zealand as of 2013. The estimates do not include unicompartmental knee replacements (UKR), subsequent contralateral TKR or TKR for indications other than primary OA.

The main purpose of our analysis was to investigate to what extent healthcare costs (including TKR demand) will increase as a result of increasing population obesity in New Zealand.[2] This focus required delimiting the population to that for which we had reliable population data and evidence of the association between obesity, OA and those effects. These delimitations were reported in the paper (and its supplementary material), and our correspondents are right to point out that they must be explicitly acknowledged when interpreting the estimates, such as when making important decisions about workforce planning and formulating strategies for mitigating the public health burden of OA. Based on our reported proportional increases, the total number of knee replacements required by 2038 may exceed 13,300, an increase of around 6,000 or more than 80% from 2013.

It is worth noting, as reported in our paper, that our projections are based on increasing demand for TKR due to projected changes in population ageing and obesity rates, and do not capture "supply side" changes in health sector funding or ability to perform the number of surgeries required. The significant increase in funding for elective orthopaedic surgeries ($30 million over three years) made in Budget 2015 explains much of the remaining difference (after accounting for our inclusion criteria as above) between projected and actual provision of TKR in 2017.

We agree with our correspondents' interpretation, as originally made by Hooper et al,[3] that continuing to meet the increasing demand for provision of TKR in New Zealand will be enormously challenging, even insurmountable, based on these independently validated current projections.[1,3] When taking into account the additional surgeries demanded by indications outside our base case delimitations (ie, subsequent contralateral TKR surgeries [around 25% of TKRs[4]], UKR surgeries and arthroplasties for reasons other than primary OA, eg, rheumatoid arthritis, trauma, other pathology etc. [which account for around 15% of TKRs[4]]), the implications for workforce training and health system planning are stark.

The projected future burden of OA is alarming. Among the top 20 causes of disability-related life-years lost in adults, many (such as cardiovascular causes and cancer) have shown decreases between 1990 and 2018, but OA has shown among the greatest rises.[5] This is largely driven by people living longer with OA, rising obesity rates, increasing injury rates and earlier onset of OA. Restricted relative supply of

TKR would further add to the tally. Acknowledging that OA is a chronic, long-term condition, it is clear that a unidimensional, ambulance-at-the-bottom-of-the-cliff approach will not be adequate. We agree with our correspondents that a more coordinated approach that includes prevention, public health initiatives and effective non-operative treatments, including body-weight management and exercise therapy at the early- and mid-stages of OA, as well as adequate provision of optimally-timed TKR for advanced-stage OA, all have an important role in the management of knee OA.

Our research centre plans to host a New Zealand Osteoarthritis Summit in June 2020, where attendees will propose and plan a course of action to help address the rising burden of OA, and the response to it of the New Zealand public healthcare system. We invite representatives from orthopaedic surgery and all other interested parties to collaborate with us in this initiative.

Competing interests:
Nil.

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REFERENCES:

1. Wilson R, Abbott JH. The projected burden of knee osteoarthritis in New Zealand: healthcare expenditure and total joint provision. N Z Med J 2019; 132(1503):53–65.
2. Wilson R, Abbott JH. Age, Period, and Cohort Effects on Body Mass Index in New Zealand, 1997–2038. Australian & New Zealand Journal of Public Health. 2018; 42(4):396–402.
3. Hooper G, Lee AJJ, Rothwell A, Frampton C. Current trends and projections in the utilization rates of hip and knee replacement in New Zealand from 2001 to 2026. N Z Med J 2014; 127(1401):82–93.
4. New Zealand Joint Registry. Sixteen year report: January 1999 to December 2015. 2016 Oct. http://nzoa.org.nz/system/files/Web_DH7657_NZJR-2014Report_v4_12Nov15.pdf accessed 06/11/2019.
5. GBD Compare. http://vizhub.healthdata.org/gbd-compare/ accessed 06/11/2019.

A Case of Melaena Neanatorum

By ALEX. T. McCAW, F.R.C.S.

The following case of melaena neonatorum may be of interest, after Dr. Horrax's case published in your issue of December:—

The child, a female, well formed, weighing nine pounds, was delivered without forceps, after a normal labour.

The mother, aged 36, is healthy, except for a history of furunculosis and a uterus with multiple fibroids.

This is her second child, the first being a healthy boy now three and a-half years old.

Immediately after birth the child was a good colour, but movements and cry were less marked than usual and the breathing seemed shallow. About an hour after birth the child became deeply cyanosed, the veins were distended, and the breathing sounded asthmatical.

I could get no mucus from the respiratory tract, but smart slapping with the child held up by the feet and hot and cold douches gradually wrought an improvement, and in twelve hours, with occasional relapses into a blue condition, the child became normal in colour.

There were no murmurs in the heart; later, after the melaena, a systolic murmur over the whole cardiac area developed, but disappeared in a few days, leaving the sounds clear.

Forty hours after birth the child had a small haematemesis followed by a small dark melaena, which had been preceded by several normal stools. A few hours after the first slight melaena there was a profuse dark one. The melaena was repeated four times during the next twenty-four hours and the child became anaemic and collapsed.

The stools in the next twenty-four hours gradually decreased in size and became normal in appearance. There has been no recurrence of the haemorrhage, and the child, now twelve days old, is pale and has a pulse of 160, but is otherwise normal.

I first gave horse serum (antidiphtheritic, the only form I could get) by mouth, and then coagulose and normal horse serum when it came to hand. I also gave calcium lactate and liquor ferri perchloridi.

The child was able to take the breast, and, except when very collapsed, sucked well. While giving the serum I had some gelatin in 10 per cent. solution twice sterilised and injected 10 c.c.s. into the subcutaneous tissues of the back. According to Knopfelmacher, Cautley, and other authorities, the best results follow the use of gelatin, and in this case the commencement of improvement coincided with the injection.

As to the cause in this case, I could eliminate spurious melaena from the mother's nipples or discharges, or from the child's nose or mouth. There was no demonstrable disease that could account for symptomatic melaena. There is no syphilitic taint in the parents. The father has a distant cousin who is a haemophiliac, but, owing to the child's sex, the early onset is of symptoms, the absence of other haemorrhages, and the absence of history on the mother's side, haemophilia is negatived. Sepsis, the probable cause of the haemorrhagic diathesis of the new-born, was not the cause, as there were no other symptoms of septicaemia, and, again, the onset was too early. There are no physical signs now of congenital heart or lung conditions.

There was no palpable tumour of the abdomen. Ulceration of the stomach or duodenum was possible, but I believe in this case the melaena was due to hyperaemia and stasis in the gastro-intestinal mucosa due to postpartum asphyxia, which was probably caused by inhalation of mucus or discharges.

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