CONTENTS

This Issue in the Journal
4  A summary of the original articles featured in this issue

Editorials
6  Māori health. What next?
   Julia Carr
9  Deserving of more: framing of Māori inequities in cardiovascular care remains a challenge
   Elana Curtis

Original Articles
12  Māori have worse outcomes after coronary artery bypass grafting than Europeans in New Zealand
   Tom Kai Ming Wang, Tharumenthiran Ramanathan, Ralph Stewart, Sue Crengle, Greg Gamble, Harvey White
23  Are there differences between Māori and non-Māori patients undergoing primary total hip and knee arthroplasty surgery in New Zealand? A registry-based cohort study
   Neal Singleton, Emily Buddicom, Andrew Vane, Vaughan Poutawera
31  Cross-sectional study on prevalence, causes and avoidable causes of visual impairment in Māori children
   CheeFoong Chong, Shuan Dai
39  Injury severity and 3-month outcomes among Māori: results from a New Zealand prospective cohort study
   Brett Maclennan, Emma Wyeth, Brendan Hokowhitu, Suzanne Wilson, Sarah Derrett
50  New Zealand guidelines for the diagnosis of acute rheumatic fever: small increase in the incidence of definite cases compared to the American Heart Association Jones criteria
   Nigel J Wilson, Lesley Voss, Johan Morreau, Joanna Stewart, Diana Lennon

Review Article
60  Health consequences of tobacco use for Māori—cessation essential for reducing inequalities in health
   Marewa Glover, Anette Kira, Nathan Cowie, Ron Wong, Jane Stephen, Kate Marriner
Viewpoints

74 Improving health, safety and energy efficiency in New Zealand through measuring and applying basic housing standards
Julie Gillespie-Bennett, Michael Keall, Philippa Howden-Chapman, Michael G Baker

86 SUDI prevention: a review of Māori safe sleep innovations for infants
Sally Abel, David Tipene-Leach

Clinical Correspondence

95 Shellfish-acquired Vibrio cholerae cellulitis and sepsis from a vulnerable leg
Samuel J Whittaker

98 Drug-induced subacute cutaneous lupus erythematosus due to treatment with interferon beta-1a
Sarah Buchanan, Ian Rosemergy, Paul Healy

102 Medical image. Raccoon eyes in amyloidosis
Michael Prystajecky, Habib U Rehman

104 Medical image. Mediastinal enlargement
Eiki Tayama, Hidetsugu Hori, Takanori Kono, Ken-ichi Imasaka, Yuma Motomatsu, Yukihiro Tomita

Letters

106 Coke’s anti-obesity campaign: a FIZZ or not?
Gerhard Sundborn, Simon Thornley, Rod Jackson

109 Revoke PM10 regulations
Peter W Moller

110 Additional evidence for concern about the quality of public toilets in New Zealand
Nick Wilson, George Thomson

100 Years Ago in the NZMJ

112 Treatment of the Insane (part 2)

Methuselah

113 Selected excerpts from Methuselah

Notice

115 Medical Benevolent Fund
Erratum

Incorrect Methuselah abstracts in the 12 July 2013 edition

NZMJ
This Issue in the Journal

Māori have worse outcomes after coronary artery bypass grafting than Europeans in New Zealand
Tom Kai Ming Wang, Tharumenthiran Ramanathan, Ralph Stewart, Sue Crengle, Greg Gamble, Harvey White

Patients undergoing coronary bypass surgery at Auckland City Hospital were followed for 1½ years after surgery. Māori were 8 years younger than Europeans but had more complications and higher mortality; mortality at 1 year 6.3% Māori vs 1.5% European. The reasons for this include Māori presenting later with more advanced heart disease. Steps need to be taken to ensure Māori present earlier for bypass surgery and that risk factors such as smoking rates and diabetes are reduced.

Are there differences between Māori and non-Māori patients undergoing primary total hip and knee arthroplasty surgery in New Zealand? A registry-based cohort study
Neal Singleton, Emily Buddicom, Andrew Vane, Vaughan Poutawera

Māori have the poorest health status of any ethnic group in New Zealand. The aim of this study was to determine whether there are any differences between Māori and non-Māori patients in the severity of their arthritis before and following primary total hip and knee arthroplasty surgery. We also compared general and mental health scores and to determine whether the intervention rate for Māori arthroplasty patients is appropriate. Māori patients are younger, have worse general and mental health and worse preoperative function compared with non-Māori patients. These differences are significant and ongoing education and effort is required in order to achieve earlier intervention rates and improve postoperative outcomes for Māori patients.

Cross-sectional study on prevalence, causes and avoidable causes of visual impairment in Māori children
CheeFoong Chong, Shuan Dai

Approximately 22% of blind children in New Zealand are Māori; this translates to about 1 in every 1000 Māori children have at least some degree of visual impairment. The major cause of visual impairment in this group of children is cerebral visual impairment (a condition where the eye is normal but connection from eye to brain or brain processing centres are impaired). Approximately 30% of these blindness could be avoidable. The largest avoidable cause of Māori children blindness is non-accidental injury (i.e. child physical abuse).
Injury severity and 3-month outcomes among Māori: results from a New Zealand prospective cohort study
Brett Maclennan, Emma Wyeth, Brendan Hokowhitu, Suzanne Wilson, Sarah Derrett

Māori and other indigenous populations around the world experience a disproportionate burden following injury. Previous research on injury outcomes among these populations has generally focused on particular types of injury or fatal and/or serious injuries but these are significantly outnumbered by less severe injuries, many of which result in disability. This paper examined the prevalence of various health and social outcomes following injuries that varied in type and severity among Māori. High levels of adverse outcomes were observed 3 months post-injury in all injury severity groups and differences in the prevalence of adverse outcomes 3 months after injury compared to pre-injury were statistically significant. Results highlight the importance of identifying improved strategies to prevent injury, including ‘minor’ injuries, and for appropriate rehabilitation for injured Māori, irrespective of injury severity.

New Zealand guidelines for the diagnosis of acute rheumatic fever: small increase in the incidence of definite cases compared to the American Heart Association Jones criteria
Nigel J Wilson, Lesley Voss, Johan Morreau, Joanna Stewart, Diana Lennon

From 2006 Doctors in New Zealand have used the New Zealand rheumatic fever guidelines to diagnose acute rheumatic fever (published by the Heart Foundation. www.heartfoundation.org.nz). This study shows that 12% more children were diagnosed with ARF using these guidelines compared to the American diagnostic guidelines. Diagnosis of ARF is important as treatment with penicillin prevents the development of severe heart damage.

Improving health, safety and energy efficiency in New Zealand through measuring and applying basic housing standards ((viewpoint article))
Julie Gillespie-Bennett, Michael Keall, Philippa Howden-Chapman, Michael G Baker

Substandard housing is a major problem in New Zealand, with little recognition of the important aspects of housing quality that affect people’s health and safety. The Healthy Housing Index is a practical risk assessment tool based on strong international evidence and adapted to New Zealand conditions that measures the physical characteristics of houses that affect the health and safety of the occupants.
Māori health. What next?

This edition of the New Zealand Medical Journal offers readers several papers with a focus on health outcomes for Māori, or issues relevant to Māori health.

Dr Marewa Glover et al reinforce and point out the negative impact that tobacco use has on Māori smokers and their whānau (extended families). Chong and Dai provide information on the prevalence and causes of blindness in Māori children, an area where there has been little specific research. This paper highlights the importance of non-accidental injury (NAI) and neonatal trauma as avoidable causes of blindness.

Singleton et al report differences in preoperative and postoperative function for Māori patients undergoing hip or knee replacement surgery compared to non-Māori patients having the same surgery. Maclennan et al examine health and social outcomes for working-age Māori recovering from injury.

Wilson et al test the effect of using New Zealand guidelines for the diagnosis of rheumatic fever rather than the American Heart Association Jones criteria, and remind us that rheumatic fever remains endemic among Māori and Pacific populations, with a rising incidence.

These papers each contribute to our understanding of current Māori health and disability outcomes. The analysis and reflection on outcomes for Māori in areas that have had less focus in the past, childhood blindness, post-injury rehabilitation and orthopaedic surgery, adds useful new information to the body of Māori health and health service research.

The big question is how to use these carefully constructed and reported studies, to make a difference. Each article reinforces our knowledge that Māori experience higher exposures to risk factors for poor health, more injury, more disability and poorer outcomes when they interact with health services. The results and discussion point to potential levers to change this situation. So is it changing?

As an observer with a strong interest in Māori health, the answer is: not at a pace or on a scale that is acceptable or even encouraging. However, this is not to suggest that an accelerated pace of change is not possible. The challenge raised by each of the articles is how the results can be used. How do we translate new knowledge into effective action?

Part of the challenge is to think beyond the conventional. It is so tempting to digest the written words, feel satisfied with a fuller understanding and continue to do more of what we have done.

However, hard questions need to be asked. Why are Māori children experiencing higher levels of neonatal trauma and NAI? Which Māori whānau are we talking about? Where are these problems concentrated and why? Why do Māori present with more advanced joint problems before surgery is an option? Why do Māori with high levels of pain and immobility following injury report that they are generally ok? How
will we make sure that post-injury care happens? Who is most affected and how can the insights from their lived experience inform our responses?

None of these are easy questions to answer. Underlying the results reported across the different studies in this edition are entrenched systemic drivers of disparities and poor outcomes for Māori. These include social and environmental drivers, health system factors, health professional behaviours and institutional resistance to innovation. However, there are always opportunities to drive positive change.

Whether it is through clinical governance groups, alliance contracting discussions, outreach into communities, Whānau Ora developments or service redesign, opportunities to address these factors are all around us. As health professionals, we are fortunate in having considerable information, power and networks.

After reading this thought-provoking series of articles, and considering the findings not separately but together, it is apparent that a new breakthrough is needed in Māori health.

As health professionals we know the determinants of Māori health outcomes. These include low incomes, poor housing, inadequate education, erratic employment and racism. The impacts are complex and intergenerational. We measure and plot these variables. We use them to address ‘confounding’, although in the real world they are ‘compounding’. We have our instruments of diagnosis, and of intervention. We polish and sharpen them. We have the treatments, the ‘fixes’ for individuals, but the people still suffer.

How do we generate new forces for change, and activate the next leap forward in Māori health? Given the complexity and persistent nature of the problems, new thinking and new tools need to be added to the toolkit.

Health professionals, and Māori health professionals in particular, have been leaders in highlighting disparities in Māori health outcomes and potential interventions. However, a relatively untapped resource are the whānau and Māori communities most affected by these health issues – the silent (or silenced?) partners.

How do we engage with these whānau and communities to achieve a quantum change in expectations and to design solutions, both technical and political?

Competing interests: None identified.

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References:

Deserving of more: framing of Māori inequities in cardiovascular care remains a challenge

Elana Curtis

It is well known that Māori are more likely to die from cardiovascular disease yet are less likely to receive invasive cardiovascular procedures such as angioplasty and coronary artery bypass grafts (CABG).1–5

Indeed, the inverse care law, where the availability of care is inversely proportional to the need of the population served, is operating in Aotearoa New Zealand with respect to cardiovascular disease—with those who need healthcare the most, receiving it the least.4

Wang and colleagues contribute to this growing body of evidence with their recent review of Māori access to CABG at Auckland City Hospital.6 Of concern, their findings (from a retrospective, observational, single-centre study between 2010 and 2012) document that Māori ethnicity is independently associated with 30-day mortality, surgical morbidity and lower Māori 1-year survival. In other words, in this study Māori get less, and what they do get may be of lower quality than their European counterparts.

This is important information. It builds on recent research undertaken by Māori health researchers exploring this phenomenon within the Hauora – Māori Standards of Health IV series and expands the investigation to postoperative outcomes following invasive cardiovascular procedures.7 Furthermore, the authors discuss the potential contribution of surgical urgency that may be “questionable in Māori”; the intricacies of the EuroSCORE 1 and the need for more effective strategies to reduce cardiovascular disease (such as earlier screening for Māori).

However, missing from the ‘frame’ is a discussion on the role of the clinician and the system in which clinicians must operate. Wang et al’s findings support the need to explore the role of clinician prejudice or bias (above and beyond scoring systems) particularly in contexts of greater clinical uncertainty when interacting with different ethnic groups.4

The seminal Institute of Medicine report ‘Unequal Treatment’ highlights the need to consider the health care provider role as a likely contributor to creating and maintaining ethnic health disparities.8

Van Ryn & Fu (2003) acknowledge that this task will require us to engage in what can be ‘painful questions’, noting:

…Because institutional racism (differential processes or outcomes according to race/ethnicity) is the result of the sum total of policies and procedures created and enforced, and the behaviors engaged in, by institutional members, we must ask whether health and human service providers directly contribute to these racial/ethnic disparities in care and health outcomes. If so, how does this occur? (p. 248).
Wang and colleagues have done well to identify their findings of inequity with respect to Māori access to and outcomes from CABG. The next challenge is to theorise the likely basic causes of these inequities so that further research (and therefore interventions) can be appropriately directed.\textsuperscript{10}

This means we must move from describing surface causes to the fundamental drivers of these inequities. Exploring clinician factors, including bias, will require researchers to theorise more deeply on what their findings mean, and how they can and should be discussed. This will require researchers to reflect on the complexity of what is driving ethnic disparities in survival seen across multiple disease indicators for Māori.

As noted by Hill and colleagues the structure of the health system as a whole may be driving inequitable care for Māori.\textsuperscript{11}

...Even if individual facilities in New Zealand provide equitable care, the structure of the health system as a whole may result in unequal care for Maori and non-Maori patients, a form of institutional racism and an important cause of survival disparities (p 121).

These issues are not easy—but if evidence of reduced access and reduced quality of care for Māori exist within our health system, it deserves deeper discussion.

**Competing interests:** None identified.

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**References**


Māori have worse outcomes after coronary artery bypass grafting than Europeans in New Zealand

Tom Kai Ming Wang, Tharumenthiran Ramanathan, Ralph Stewart, Sue Crengle, Greg Gamble, Harvey White

Abstract

Aims Disparities for Māori exist in New Zealand for cardiovascular risk factors, events and access to revascularisation. We compared characteristics and outcomes of coronary artery bypass grafting (CABG) between Māori and Europeans in New Zealand.

Methods Patients undergoing isolated CABG at Auckland City Hospital from July 2010–June 2012 were retrospectively analysed.

Results Of 818 patients, 82 were Māori and 444 were Europeans. Māori were younger (60.0 vs 67.9 years, p<0.001), had higher NZ deprivation index (8.5 vs 5.0, p<0.001), body mass index (32.6 vs 28.8 kg/m², p<0.001), higher prevalence of heart failure (11.0% vs 2.3%, p<0.001), diabetes (43.9% vs 24.1%, p<0.001), smoking (39.0% vs 13.1%, p<0.001), dialysis (4.9% vs 0.9%, p=0.023), lower ejection fraction (p=0.001), lower additive EuroSCORE 1 (4.1 vs 4.8, p=0.041) and longer cardiopulmonary bypass time (100 vs 89 minutes p<0.001).

Māori ethnicity was independently associated with 30-day mortality, odds ratio (OR) 6.35, 95% confidence interval 1.01–39.9, p=0.046; and surgical morbidity OR 2.05, 1.04–4.04, p=0.040. Māori had a trend for higher mortality at 1.4±0.6 years (hazards ratio 2.91, 0.92–9.20, p=0.069), 1-year mortality 6.3% vs 1.5%.

Conclusion Despite being younger, Māori undergoing CABG had more comorbidities and socioeconomic deprivation. Māori had higher mortality and complication rates. Māori should have earlier access to CABG.

Ischaemic heart disease (IHD) is the single most common cause of mortality in New Zealand (NZ) at 19.0% in 2009. Māori had 1.13 times higher age-standardised IHD death rate than non-Māori, with differences most marked in the 45–64 year old age-group (odds ratio 2.82).

Factors contributing to these ethnic inequalities include ethnic differences in the prevalence of cardiovascular risk factors and their management, cardiovascular disease and inappropriately lower rates of percutaneous and surgical revascularisation procedures than other ethnicities.

Coronary artery bypass grafting (CABG) is the optimal treatment for severe three-vessel or severe left main stem coronary artery disease, and multi-vessel disease in diabetics. Various ethnic groups worldwide have been reported to have worse outcomes after CABG. As associations between CABG outcomes and ethnicity have not been previously investigated in a New Zealand cohort, we aimed to compare the characteristics and outcomes after CABG between Māori and Europeans.
Methods

Patient selection and data collection—Ethics approval of this study was obtained from the Auckland Ethics Committee. Consecutive patients having isolated CABG without concomitant valve surgery from July 2010 to June 2012 were retrospectively identified from the cardiothoracic surgical unit database.

Ethnicity information was obtained from records associated with the national health index (NHI), which is recorded by hospital staff upon asking patients. All patients for whom Māori was recorded as an ethnic group were included in the Māori group. The second group “Europeans” included those whose ethnic group were recorded as “New Zealand Europeans” or “other Europeans”.

NZ Europeans and Europeans were combined as they had similar demographics and characteristics, which were very different to Asian, Indian, Pacific and other ethnicity, so we compared Europeans with Maori to specifically identify factors and outcomes associated with these two main groups of interest, and to identify discrepancies and strategies to reduce inequalities. Relevant clinical characteristics and outcomes were also extracted from computerised clinical records.

The following definitions were used for baseline characteristics. Socioeconomic deprivation was assigned using the New Zealand Index of Deprivation 2006 (NZDep2006) decile—a small area geographic measure derived from the 2006 Census.

As the sample in this study is relatively small we prespecified to categorise deprivation into ‘mild’ (deciles 1–4), moderate (deciles 5–7) and high (deciles 8–10) rather than use deciles or quintiles.

Angina was graded using the Canadian Cardiovascular Society Classification (CCS) and dyspnoea by the New York Heart Association Functional Classification (NYHA) for severity of heart failure.

Hypertension was defined as having been prescribed medications for lowering blood pressure, any measurement of over 140/90 mmHg prior to operation and/or a previous formal diagnosis.

Hypercholesterolaemia refers to total cholesterol >5.0 mmol, being on treatment before admission and/or a previous formal diagnosis.

A family history of coronary artery disease is defined as a first-degree relative(s) with angina, myocardial infarction, sudden cardiac death, percutaneous and/or surgical coronary intervention before the age of 55 years.

Stroke included any previous history of a neurological deficit that persisted over 24 hours and was caused by disturbance of cerebral blood supply.

Peripheral vascular disease included claudication, vascular intervention or amputation of peripheries for arterial insufficiency, aortic aneurysm and ankle brachial index <0.9 and/or imaging evidence of >50% stenosis in any peripheral artery.

Chronic respiratory diseases included use of inhaled corticosteroids for respiratory symptoms, forced expiratory volume in 1 second (FEV1) <80% on spirometry, and/or previous formal diagnosis.

Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet and Renal Disease equation using the last preoperative serum creatinine measurement.

The number of main coronary arteries with angiographic evidence of stenosis >50% was recorded. The operative risk was calculated using additive and logistic EuroSCORE I.

Operative variables collected include the number of grafts and duration of cardiopulmonary bypass and aortic cross-clamp time. Postoperative high-sensitivity troponin T (hs-TnT) was routinely measured 12–24 hours postoperatively and the peak value recorded.

The development of new Q-waves or left bundle branch block (LBBB) on postoperative electrocardiogram (ECG) was independently interpreted by two authors (TKMW and HDW). Any discrepancies were resolved by discussion to reach consensus. New regional wall motion abnormalities on postoperative echocardiograms were documented.

Perioperative myocardial infarction was defined as postoperative hs-TnT >140 ng/L (10 times 99% upper reference limit) and the ECG and/or echocardiographic criteria described earlier.

Five other surgical complications as defined by the Society of Thoracic Surgeon’s score and their composite surgical morbidity were determined. These included stroke (acute neurological deficit >24 hours due to cerebral blood supply disturbance), renal failure (new dialysis requirement or increase of creatinine to >4.0 mg/dL and >3 times last preoperative level), prolonged ventilation >24 hours, deep sternal wound infection and return to theatre for any reason.
Mortality data were checked against New Zealand’s national registry up till 31 December 2012. Medium-term mortality is defined as mortality that occurred during the follow-up period. Thirty-day mortality, medium-term mortality and composite morbidity were our main pre-specified outcomes.

**Statistical analyses**—Mean (standard deviation) or percentages were calculated for continuous and categorical variables respectively. Student t-tests and Fisher’s exact test were used were used for univariate analyses. Kaplan-Meier curves and log-rank (Mantel-Cox) test were performed for univariate longitudinal survival analysis. Logistic regression was used to calculate odds ratios (OR) for cross-sectional outcomes and Cox proportional hazards regression for hazards ratio (HR) for longitudinal outcomes, and their 95% confidence intervals (95%CI).

Baseline variables with P<0.10 in univariate analyses were included in stepwise multivariate analyses. All tests were two tailed and p-value less than 0.05 deemed statistically significant. SPSS (Version 17.0, SPSS Inc., Chicago, IL, USA) and Prism (Version 5, GraphPad Software, San Diego, CA, USA) were used for analyses.

**Results**

**Preoperative characteristics**—There were 818 patients who had isolated CABG during the 2-year study period, of which 82 (10.0%) were Māori and 444 (54.3%) were Europeans. Table 1 presents the preoperative characteristics of these patients.

<table>
<thead>
<tr>
<th>Table 1. Baseline patient characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnicity</strong></td>
</tr>
<tr>
<td><strong>Number</strong></td>
</tr>
<tr>
<td><strong>Demographics</strong></td>
</tr>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
</tr>
<tr>
<td><strong>New Zealand 2006 Index of Deprivation</strong> by decile</td>
</tr>
<tr>
<td>1–4 (least deprivation)</td>
</tr>
<tr>
<td>5–7</td>
</tr>
<tr>
<td>8–10 (most deprivation)</td>
</tr>
<tr>
<td><strong>Presentation</strong></td>
</tr>
<tr>
<td>Canadian Cardiovascular Class for angina</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td><strong>New York Heart Association Class for dyspnoea</strong></td>
</tr>
<tr>
<td>I</td>
</tr>
<tr>
<td>II</td>
</tr>
<tr>
<td>III</td>
</tr>
<tr>
<td>IV</td>
</tr>
<tr>
<td>Recent myocardial infarction within 6 weeks</td>
</tr>
<tr>
<td>Intra-aortic balloon pump (%)</td>
</tr>
<tr>
<td>Inpatient operation (%)</td>
</tr>
<tr>
<td>Waitlist – inpatient (days)</td>
</tr>
<tr>
<td>Waitlist – elective (days)</td>
</tr>
<tr>
<td><strong>Past Medical History</strong></td>
</tr>
<tr>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>Percutaneous coronary intervention</td>
</tr>
<tr>
<td>Coronary artery bypass grafting</td>
</tr>
</tbody>
</table>
Māori were younger (p<0.001), had higher body mass index (p<0.001), higher prevalence of NZ deprivation index decile 8–10 (p<0.001), higher NYHA class (p=0.004), higher prevalence of congestive heart failure (p<0.001), diabetes (p<0.001), active smoking (p<0.001), were on dialysis (p=0.023), and had lower ejection fraction (p=0.001). Māori had lower additive EuroSCORE 1 (p=0.041) but similar logistic EuroSCORE 1 (p=0.150) to Europeans.

In-hospital outcomes—Table 2 shows the operative variables and postoperative outcomes by ethnicity. There were no differences in the proportion of CABG performed off-pump and the amount of grafting. Māori had longer duration of cardiopulmonary bypass (p<0.001) and cross-clamp (p=0.005). There were no differences in length of hospital stay and discharge medications.

Māori had higher 30-day mortality (p=0.007) and composite morbidity (p=0.002) mainly due to higher rates of prolonged ventilation >24 hours (p=0.004). Causes of in-hospital mortality were cardiac shock and failure in 4, respiratory failure in 1 and ischaemic bowel in 1 patient.
Table 2. Operative variables and postoperative outcomes

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Māori</th>
<th>European</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Operation Details</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Off-pump</td>
<td>4.9% (4)</td>
<td>2.5% (11)</td>
<td>0.269</td>
</tr>
<tr>
<td>Number of distal anastomoses</td>
<td>3.3 (0.8)</td>
<td>3.2 (0.8)</td>
<td>0.285</td>
</tr>
<tr>
<td>Left internal mammary artery graft</td>
<td>96.3% (79)</td>
<td>97.7% (434)</td>
<td>0.437</td>
</tr>
<tr>
<td>Right internal mammary artery graft</td>
<td>7.3% (6)</td>
<td>5.0% (22)</td>
<td>0.419</td>
</tr>
<tr>
<td>Radial artery graft</td>
<td>31.7% (26)</td>
<td>22.3% (99)</td>
<td>0.089</td>
</tr>
<tr>
<td>Saphenous vein grafts</td>
<td>91.5% (75)</td>
<td>92.8% (412)</td>
<td>0.648</td>
</tr>
<tr>
<td>Cardiopulmonary bypass time (minutes)</td>
<td>100 (31)</td>
<td>89 (25)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cross-clamp time (minutes)</td>
<td>64 (22)</td>
<td>57 (20)</td>
<td>0.005</td>
</tr>
<tr>
<td><strong>Postoperative Outcomes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postoperative hs-TnT (ng/L)</td>
<td>625 (1000)</td>
<td>549 (783)</td>
<td>0.445</td>
</tr>
<tr>
<td>New ECG or echocardiographic changes</td>
<td>17.3% (14/81)</td>
<td>13.4% (59/439)</td>
<td>0.384</td>
</tr>
<tr>
<td>Perioperative myocardial infarction</td>
<td>14.3% (8/56)</td>
<td>13.1% (43/327)</td>
<td>0.832</td>
</tr>
<tr>
<td>Composite surgical morbidity</td>
<td>29.3% (24)</td>
<td>14.2% (63)</td>
<td><strong>0.002</strong></td>
</tr>
<tr>
<td>Stroke</td>
<td>3.7% (3)</td>
<td>1.1% (5)</td>
<td>0.114</td>
</tr>
<tr>
<td>Renal failure</td>
<td>2.4% (2)</td>
<td>1.8% (8)</td>
<td>0.659</td>
</tr>
<tr>
<td>Prolonged ventilation &gt;24hours</td>
<td>23.2% (19)</td>
<td>10.8% (48)</td>
<td><strong>0.004</strong></td>
</tr>
<tr>
<td>Deep sternal wound Infection</td>
<td>0.0% (0)</td>
<td>0.5% (2)</td>
<td>1.000</td>
</tr>
<tr>
<td>Re-operation</td>
<td>8.5% (7)</td>
<td>3.6% (16)</td>
<td>0.070</td>
</tr>
<tr>
<td>Operation to discharge (days)</td>
<td>8.6 (5.7)</td>
<td>8.2 (5.4)</td>
<td>0.528</td>
</tr>
<tr>
<td>Readmission to hospital &lt;30 days</td>
<td>18.3% (15)</td>
<td>17.1% (76)</td>
<td>0.753</td>
</tr>
<tr>
<td><strong>30-day mortality</strong></td>
<td><strong>4.9% (4)</strong></td>
<td><strong>0.5% (2)</strong></td>
<td><strong>0.007</strong></td>
</tr>
</tbody>
</table>

**Discharge medications**

<table>
<thead>
<tr>
<th></th>
<th>Māori (78/78)</th>
<th>European (434/442)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>100.0%</td>
<td>98.2%</td>
<td>0.613</td>
</tr>
<tr>
<td>Statin</td>
<td>91.0% (71/78)</td>
<td>88.0% (389/442)</td>
<td>0.565</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>76.9% (60/78)</td>
<td>75.9% (337/442)</td>
<td>1.000</td>
</tr>
<tr>
<td>ACEi or ARB</td>
<td>35.9% (28/78)</td>
<td>30.1% (133/442)</td>
<td>0.352</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>2.6% (2/78)</td>
<td>2.7% (12/442)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

hs-TnT = high-sensitivity troponin T, ECG = electrocardiogram, ACEi = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blocker

*P-values less than 0.05 are in bold, and between 0.05–0.10 are in italics.

Survival—Mean follow-up was 1.4±0.6 years with no ethnic differences (P=0.187). Figure 1 illustrates the Kaplan-Meier survival curves for Māori and Europeans.

One-year survival was of 93.7% in Māori and 98.5% European. Māori had higher medium-term mortality HR 5.64, 95%CI (1.26–25.2), p=0.02.

Multivariate analysis—Predictors with p<0.10 of 30-day mortality, medium-term mortality and composite morbidity are listed in table 3. Māori ethnicity was independently associated with 30-day mortality (OR 6.35, 95%CI 1.01–39.9, p=0.046) and composite morbidity (OR 2.05, 95%CI 1.04–4.04, p=0.040). There was also a trend for Māori to have higher medium-term mortality (HR 2.91, 95%CI 0.92–9.20, p=0.069).
Figure 1. Kaplan-Meier survival curves of isolated coronary artery bypass grafting for Māori and Europeans. Log-rank test was used to calculate hazards ratio (HR), 95% confidence interval (95% CI) and p-values.

![Kaplan-Meier survival curves](image)

HR 5.64, 95% CI (1.26-25.2), p=0.024

Table 3. Multivariate predictors of mortality and morbidity

<table>
<thead>
<tr>
<th>30-day mortality</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Māori</td>
<td>6.35</td>
<td>1.01–39.9</td>
<td>0.046</td>
</tr>
<tr>
<td>Female</td>
<td>7.42</td>
<td>1.23–44.8</td>
<td>0.029</td>
</tr>
<tr>
<td>New York Heart Association Class IV</td>
<td>8.92</td>
<td>0.99–80.4</td>
<td>0.051</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medium-term mortality</th>
<th>Hazards ratio</th>
<th>95% confidence interval</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Māori</td>
<td>2.91</td>
<td>0.92–9.20</td>
<td>0.069</td>
</tr>
<tr>
<td>New York Heart Association Class IV</td>
<td>4.13</td>
<td>0.86–19.8</td>
<td>0.075</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Composite surgical morbidity</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Māori</td>
<td>2.05</td>
<td>1.04–4.04</td>
<td>0.040</td>
</tr>
<tr>
<td>Intra-aortic balloon pump</td>
<td>5.30</td>
<td>2.45–11.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Previous coronary artery bypass grafting</td>
<td>6.92</td>
<td>1.66–28.8</td>
<td>0.007</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.81</td>
<td>0.98–3.34</td>
<td>0.059</td>
</tr>
<tr>
<td>EuroSCORE 1 logistic</td>
<td>1.07</td>
<td>1.03–1.11</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Only predictors with P<0.10 in multivariate analyses are listed.
Discussion

The novel finding of this study is that Māori have higher mortality and composite postoperative morbidity after CABG than Europeans. Most of the mortality difference was in the first 30 days, with an absolute difference of 4.4%. This difference increased slightly at 1 year to an absolute difference of 4.8%.

Operative morbidity measured by the STS composite criteria\(^\text{19}\) was 106% higher in Māori. After multivariate adjustment, Māori remained significantly more likely to have higher 30-day mortality and postoperative morbidity.

At a global level, traditional modifiable cardiovascular risk factors remain the predominant driver of ethnic discrepancies in cardiovascular disease.\(^\text{13}\) The same can be said for New Zealand, where Māori have significantly higher prevalence of cardiovascular risk factors including diabetes, smoking, hypertension and metabolic syndrome than Europeans,\(^\text{1–8}\) and also greater levels of socioeconomic deprivation. However, only some of the cardiovascular risk factors mentioned above are associated with adverse outcomes after CABG.\(^\text{19,23,24}\) In our study, adjustment for these risk factors and socioeconomic deprivation in multivariate analyses did not explain the ethnic differences in CABG outcomes.

Māori had higher prevalence of many indicators for severe heart disease, including worse NYHA class, lower ejection fraction and higher rates of congestive heart failure. Not only are all of these strong predictors of mortality after cardiac surgery,\(^\text{19,23,24}\) but they suggest a longer period of no treatment or suboptimal treatment leading to their presentation for surgery. In addition more Māori were on dialysis, indicating end-stage renal disease which is associated with high mortality rates regardless as to whether cardiac surgery is performed or not. However, after adjustment for these factors the inequalities in mortality persisted.

Time on waiting lists were similar between Maori and European groups for both inpatient and outpatient waiting-times for surgery. Europeans had a trend towards a higher proportion having inpatient surgery despite having less indicators for severe heart disease. The appropriateness of surgical urgency may be questionable in Māori.

Other studies have also found differences in outcomes after CABG across various ethnic groups. The Society of Thoracic Surgeon’s (STS) national database in the United States, identified that amongst racial groups, “Blacks” had the highest rates of composite morbidity at 19.0% and “Caucasians” lowest at 13.9%.\(^\text{19}\) Using the same STS definitions, composite morbidity rates were even higher in our cohort for Māori (29.3%).

In the STS database, operative mortality was uniform across different races (2.3–2.7%). Māori (4.9%) in our cohort had higher rates compared to Europeans (0.5%). Other studies have reported that both African American and Asians\(^\text{20}\) and South Asians alone\(^\text{18}\) have higher operative mortality and morbidity than Caucasians.

Frequently used models for operative mortality after cardiac surgery include the EuroSCOREs,\(^\text{23,24}\) which doesn’t include ethnicity, and the STS score\(^\text{19}\) which incorporates ethnicity for morbidity but not for mortality prediction.
We found the additive EuroSCORE 1 was actually lower in Māori than Europeans even though 30-day mortality was higher in Māori, suggesting that this score may put too much weight on age, the only cardiovascular risk factor higher in Europeans. On the other hand the higher prevalence of comorbidities in Māori balanced their younger age such that they had similar logistic EuroSCORE 1 to Europeans.

The logistic EuroSCORE 1 has been shown in recent studies to significantly overestimate operative mortality. In our study it was a good estimate of operative mortality in Māori patients while overestimating Europeans’ operative risk. Developing a local operative risk model taking into account the higher risk of Māori would be of value.

Our results show that rates of surgical revascularisation remain low in Māori when compared to need as evidenced by the prevalence of ischaemic heart disease morbidity and mortality.

In Māori according to the 2006 Census statistics, 12.5% of the population our hospital covered for cardiac surgery were Māori but only 10.0% of our CABG cohort were Māori. This is despite Māori having 1.3–2.0 times higher age-standardised prevalence of cardiovascular disease than others, and up to 2.8 times more IHD deaths. In addition, Māori had more advanced heart disease in our cohort.

Ethnic disparities in both percutaneous and surgical revascularisation have been previously reported with Māori having fewer interventions.

It is important that, more effective strategies are developed to reduce the ethnic disparities in cardiovascular health in New Zealand. Currently, Māori are recommended to be screened 10 years earlier than Europeans, i.e. at 35 years for male and 45 years for female.

In addition, Māori should have lower thresholds for cardiac investigations including angiography. The addition of Māori into the existing New Zealand Access and Urgency Scores for cardiac surgery should be considered to enable Māori to have greater access to surgery.

Limitations—This is a retrospective observational single-centre study. Patient’s ethnicity information was obtained from computerised clinical records. Theoretically this information is patient derived; however, ethnic misclassification in hospital datasets is known to occur and may have resulted in Māori being misclassified as a non-Māori ethnic group (i.e. incomplete ascertainment of the Māori sample) and vice versa.

We were not able to examine other factors which may have influenced postoperative outcomes such as duration of cardiac disease, delayed presentation for cardiac assessment either in the community or from the community and medical treatment before CABG.

Follow-up time was restricted as this was a recent cohort. We did not obtain data in respect to adherence following discharge from hospital but discharge medication use was high (100% for aspirin and 91% for statins).
Conclusion—There were striking disparities between Māori and Europeans in the baseline characteristics and outcomes of CABG in our contemporary cohort at Auckland City Hospital. Māori not only had higher prevalence of cardiovascular risk factors, but Māori ethnicity was also independently associated with higher 30-day mortality and composite morbidity after CABG.

Further research is required to identify factors that may explain these findings and to develop strategies to reduce such inequality.

Competing interests: None identified.

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References:


Are there differences between Māori and non-Māori patients undergoing primary total hip and knee arthroplasty surgery in New Zealand? A registry-based cohort study

Neal Singleton, Emily Buddicom, Andrew Vane, Vaughan Poutawera

Abstract

**Aim** It has been well demonstrated that Māori have the poorest health status of any ethnic group in New Zealand.\(^1\) The aim of this study was to determine whether there are any differences between Māori and non-Māori patients in the severity of their arthritis preoperatively and in their postoperative functional outcomes following primary total hip and knee arthroplasty surgery. Secondary objectives were to compare general and mental health scores and to determine whether the intervention rate for Māori arthroplasty patients is appropriate.

**Method** We compared preoperative and postoperative (1 and 5 year) Oxford and WOMAC scores, general health (SF-12 PH) and mental health (SF-12 MH) scores in all public patients who underwent primary total hip and knee arthroplasty surgery in our region between 2005 and 2009.

**Results** Māori patients are younger at the time of surgery, have higher ASA scores and worse preoperative function. They also have worse postoperative outcomes and smaller overall improvements following surgery when comparing their preoperative with postoperative scores. In terms of general health, Māori and non-Māori had similar SF-12 PH scores but worse SF-12 MH scores both pre- and postoperatively.

**Conclusion:** Māori patients are younger, have worse general and mental health and worse preoperative function compared with non-Māori patients. Both absolute and differential scores show that Māori patients also have worse postoperative outcomes compared with non-Māori patients. These differences are likely clinically significant and ongoing education and effort is required in order to achieve earlier intervention rates and improve postoperative outcomes for Māori patients.
It has been reported that there is no difference in the prevalence of arthritis between Māori and non-Māori patients. There are no previous studies comparing preoperative and postoperative outcome data between Māori and non-Māori patients undergoing total hip and knee arthroplasty surgery.

The primary objective of this study was to provide a detailed comparison of preoperative and postoperative function in Māori and non-Māori patients who underwent primary total hip and knee arthroplasty surgery.

The secondary objectives were to compare general and mental health scores between Māori and non-Māori and to determine whether the intervention rate for Māori arthroplasty patients is appropriate.

**Method**

**Study design**—Ethical Board approval was granted for this study. Data covering the period from 1 January 2005 to 31 December 2009 was collected from our regional joint registry records. Our regional joint registry was established in 2004 and collects prospective data on a wide range of orthopaedic conditions.

The registry was established by a group of orthopaedic surgeons who serve a population of approximately 250,000 people. Our regional joint registry is distinct from but complementary to the New Zealand National Joint Registry, prospectively recording preoperative functional scores, baseline demographics, operative characteristics and postoperative outcome measures for all patients undergoing arthroplasty surgery throughout the region.

The registry assesses preoperative and postoperative patient function using self-administered disease specific Oxford and WOMAC scores, and general health (SF-12 PH and SF-12 MH) questionnaires completed by patients at their preoperative clinic appointment as well as at 1 and 5 years postoperatively. We use the modified Oxford score out of 48 points.

Registry records of all public patients who underwent primary total hip and knee arthroplasty surgery during this period were reviewed.

Baseline demographics (age at time of surgery, gender, ASA score, operative side, preoperative diagnosis) were recorded and all patients were classified as either Māori or non-Māori (from self-reported preoperative questionnaires).

The two groups were then compared using disease specific functional scores (Oxford and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)) and general health scores (Short Form 12 Physical Health (SF-12 PH) and Mental Health (SF-12 MH)) both preoperatively and at 1 year and 5 years postoperatively.

Absolute scores as well as change in scores from preoperative to 1 and 5 years postoperatively were compared.

**Statistical analyses**—Data was tabulated using an Excel spreadsheet. Standard descriptive statistics including means and standard errors were used to summarise the continuous measures and frequencies and percentages for the categorical measures for Māori and non-Māori groups.

Continuous measures at each time point were compared between Māori and non-Māori groups using ANOVA and changes from baseline compared using ANCOVA with the baseline level as the covariate. Preoperative ASA scores were compared using a Mann-Whitney U test. A two-tailed p-value of <0.05 was taken to indicate statistical significance.

**Results**

2390 public patients underwent hip or knee joint replacement during the study period. There were 329 Māori patients and 2061 non-Māori patients. Not all measures were available for all patients at each time point and mean levels and sample sizes for each measure at each time point are shown in Table 1.
**Baseline demographics**—Māori comprised 13.77% (329/2390) of all patients undergoing primary total hip and knee arthroplasty surgery over this 5-year period. At the time of surgery Māori patients were younger than non-Māori patients, mean 63.25 years vs 69.90 years (p<0.001). They also had higher ASA scores, mean 2.12 vs 1.97 (p=0.001).

With regards to diagnosis, 202 Māori patients underwent total hip arthroplasty (179 osteoarthritis, 8 AVN, 9 DDH, 3 inflammatory arthritis, 1 SUFE, 2 fractures) and 127 total knee arthroplasty (119 osteoarthritis, 8 inflammatory arthritis).

In non-Māori patients there were 1,083 total hip arthroplasties (1004 osteoarthritis, 26 AVN, 21 DDH, 13 inflammatory arthritis, 5 SUFE, 14 fractures) and 978 total knee arthroplasties performed (942 osteoarthritis, 7 osteonecrosis, 19 inflammatory arthritis, 10 post-traumatic arthritis).

**Primary outcome**—Māori patients were found to have worse preoperative disease specific function compared with non-Māori (mean Oxford 10.10 vs 11.26 p=0.001, WOMAC 76.24 vs 73.54 p=0.005).

Māori patients had worse postoperative outcomes at 1 year (mean Oxford 35.86 vs 37.88 p=0.002, WOMAC 22.78 vs 18.66 p=0.002) and 5 years (mean Oxford 35.26 vs 38.15 p=0.053, WOMAC 22.79 vs 18.67 p=0.129). Māori had worse scores in all three WOMAC categories (pain, stiffness and physical function) preoperatively (mean pain score 15.66 vs 15.00 p=0.002, mean stiffness score 6.39 vs 6.23 p=0.111, mean physical function score 54.22 vs 52.46 p=0.011).

Māori again had lower scores at 1 year (mean pain score 3.97 vs 2.95 p<0.001, mean stiffness score 2.07 vs 1.83 p=0.071, mean physical function score 16.91 vs 13.87 p=0.002). There were no differences between Māori and non-Māori at 5 years (mean pain score 3.59 vs 2.86 p=0.251, mean stiffness score 2.03 vs 1.83 p=0.502, and mean physical function score 15.41 vs 13.41 p=0.373).

Both Māori and non-Māori showed sustained improvement in Oxford and WOMAC scores following surgery. Māori had smaller overall improvements following surgery when comparing their preoperative with postoperative scores at 1 year (mean change in Oxford 26.09 vs 26.55 p=0.028, WOMAC 53.56 vs 54.60 p=0.009).

There was no difference in overall improvement between the groups at 5 years (mean change in Oxford 25.86 vs 26.39 p=0.321, WOMAC 53.08 vs 53.66 p=0.408).

**Secondary outcomes**—Māori and non-Māori had similar SF-12 physical health scores both preoperatively and postoperatively (mean 26.32 vs 26.37 p=0.902 preoperatively, 41.82 vs 42.23 p=0.607 at 1 year and 41.24 vs 41.04 p=0.916 at 5 years postoperatively).

Māori had worse SF-12 mental health scores preoperatively (mean 35.36 vs 37.58 p=0.004) at 1 year (mean 50.25 vs 52.20 p=0.0120 and 5 years postoperatively (6.58 vs 51.76 p=0.001).
Table 1. Comparison of Oxford, WOMAC, SF-12 PH and SF-12 MH scores between Māori and non-Māori preoperatively and at 1 and 5 years postoperatively

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Pre-Operative</th>
<th>1 Year Post-Operative</th>
<th>5 Years Post-Operative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Mean</td>
<td>Std. Error Mean</td>
</tr>
<tr>
<td>Oxford</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Maori</td>
<td>1895</td>
<td>11.26**</td>
<td>0.14</td>
</tr>
<tr>
<td>Maori</td>
<td>304</td>
<td>10.10</td>
<td>0.32</td>
</tr>
<tr>
<td>WOMAC Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Maori</td>
<td>1920</td>
<td>73.54**</td>
<td>0.36</td>
</tr>
<tr>
<td>Maori</td>
<td>312</td>
<td>76.24</td>
<td>0.89</td>
</tr>
<tr>
<td>WOMAC Pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Maori</td>
<td>1911</td>
<td>15.00**</td>
<td>0.08</td>
</tr>
<tr>
<td>Maori</td>
<td>311</td>
<td>15.66</td>
<td>0.20</td>
</tr>
<tr>
<td>WOMAC Stiffness</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Non-Maori</td>
<td>1911</td>
<td>6.23</td>
<td>0.04</td>
</tr>
<tr>
<td>Maori</td>
<td>311</td>
<td>6.39</td>
<td>0.10</td>
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<tr>
<td>WOMAC Physical function</td>
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<td>Non-Maori</td>
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<tr>
<td>Maori</td>
<td>311</td>
<td>54.22</td>
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<td>SF-12 PH</td>
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<td>Non-Maori</td>
<td>1900</td>
<td>26.32</td>
<td>0.13</td>
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<tr>
<td>Maori</td>
<td>307</td>
<td>26.37</td>
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<tr>
<td>SF-12 MH</td>
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<td></td>
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<tr>
<td>Non-Maori</td>
<td>1900</td>
<td>37.57**</td>
<td>0.29</td>
</tr>
<tr>
<td>Maori</td>
<td>307</td>
<td>35.36</td>
<td>0.69</td>
</tr>
</tbody>
</table>

*p<0.05 compared with Maori  
**p<0.01 compared with Maori  
***p<0.001 compared with Maori  
a p<0.05 change from baseline compared with Maori  
b p<0.01 change from baseline compared with Maori

Discussion

There is a paucity of data comparing and contrasting health outcomes between Māori and non-Māori in the orthopaedic literature. Although there are numerous reports in the medical literature on the health disparities between Māori and non-Māori, there has been minimal work done in the field of orthopaedics and nothing previously published on arthroplasty surgery in Māori patients.

This study is the first of its kind comparing hip and knee arthroplasty in Māori and non-Māori. In this retrospective review of prospectively collected joint registry data a number of important findings have been established.

Our study demonstrates that Māori patients who underwent primary total hip and knee arthroplasty surgery displayed worse preoperative function than the control group of non-Māori patients. Furthermore, they showed worse functional outcomes at 1 year postoperatively and had smaller functional gains.

Māori patients were also younger and had higher ASA scores at the time of surgery and worse general and mental health both preoperatively and postoperatively.

**Methodological considerations**—The Oxford hip and Oxford knee scores are instruments for assessing hip and knee function. Both have been validated in several studies and are currently used in the New Zealand National Joint Register.7 The WOMAC score is a validated scoring system specific to locomotor capability that is widely used to evaluate patients undergoing total hip and knee arthroplasty surgery.8
The WOMAC is composed of three sections—pain, stiffness and physical function, with a higher score corresponding to poorer function. The SF-12 PH and SF-12 MH scores are validated measures that were developed using 12 items from the Medical Outcomes Study 36-Item Short-Form Health Survey (SF-36).

The SF-12 PH and SF-12 MH have been shown to accurately and efficiently reproduce the Physical Component Summary and Mental Component Summary scales of the SF-36, with higher scores indicating improved health status.

Our study has certain limitations. Although we employed several different functional measures, there are some intrinsic limitations to the use of the Oxford, WOMAC, SF-12 PH and SF-12 MH scores, which may not be able to measure subtle changes in function. A relatively small number of patients have been followed through to 5 years.

Ethnicity was taken from self-reported preoperative questionnaires which can be considered a subjective variable. Any patient who identified as Māori when completing preoperative questionnaires was grouped into the Māori group and obviously this is not a homogenous group. However, nor is the non-Māori group homogenous although by far the most common ethnicity recorded was New Zealand European/Pākehā (2037/2061 patients).

Baseline information—In our study Māori patients were found to be significantly younger than non-Māori at the time of surgery. This suggests that Māori suffer more from their arthritis at an earlier age. A large number of factors may contribute.

Obesity is one such factor, known to contribute to degenerative joint disease, and documented to be more prevalent in the Māori population.

The fact that Māori patients had significantly higher ASA scores than non-Māori is not surprising as it is well known that Māori have the poorest health status of any ethnic group in New Zealand and the ASA score is a classification system for assessing general health.

Primary outcome—In this study Māori patients had worse preoperative functional scores than non-Māori patients. There could be a number of reasons for this. Māori patients may be presenting for consideration of arthroplasty surgery at a later stage.

Delayed presentation to tertiary orthopaedic services may relate to primary care access issues, negative attitudes towards orthopaedic surgery, poor doctor-patient communication in the primary or tertiary care setting, or socio-economic constraints. Other patient comorbidities may also influence the poor functional and general health scores.

This study has shown that Māori patients have worse functional outcomes at 1 and 5 years postoperatively. This may simply be a consequence of their worse preoperative function, as it is known that a major predictor of postoperative functional outcome is preoperative function. It may also be a consequence of Māori patients presenting for surgery at a later stage with more advanced arthritis, potentially limiting their gains in functional improvement.

Furthermore, it has been reported in the literature that preoperative mental health has an effect on postoperative functional outcome and this study has found Māori to have
worse preoperative mental health which could therefore impact negatively on their postoperative functional outcomes.\textsuperscript{12,13}

Consequently, Māori have smaller overall improvements following surgery than non-Māori when comparing their preoperative with postoperative scores.

We compared our results with the New Zealand National Joint Registry and found our patient population tended to have poorer results than the general population with Māori patients having an average Oxford score of 36.89 and non-Māori 39.42 at 1 year following primary total hip arthroplasty compared with the national average of 40.61 at 6 months following surgery.\textsuperscript{14}

Our Māori population was younger at the time of surgery than the national average, mean 61.1 years compared with 66.85 years, and our non-Māori population was slightly older at 68.8 years.\textsuperscript{14}

In terms of our outcomes following primary total knee arthroplasty again our results tended be worse than the national average with our average 1 year post-primary total knee arthroplasty Oxford score being 34.29 in our Māori population and 36.24 in our non-Māori population compared with the national average of 37.28 at 6 months postoperatively.\textsuperscript{14}

Again our Māori population was younger than the national average, mean 66.65 years versus 68.47 years, whereas our non-Māori population was older at 71.11 years.\textsuperscript{14}

Numerous studies have reported on the minimal clinically important difference (MCID) which is the smallest change in score that patients perceive as meaningful. The MCID has been reported as being as low as 2 for the Oxford scores and 0.51-1.33 for the WOMAC score meaning that these differences in scores between Māori and non-Māori are not only statistically significant but also clinically meaningful and likely represent functionally perceivable differences.\textsuperscript{15,16}

The statistically significant differences in functional scores between Māori and non-Māori which were seen preoperatively and at 1 year postoperatively were not seen at 5 years postoperatively. This is likely due to the fact that the number of returned 5-year postoperative questionnaires was low.

Though the trend at 5 years remained for a worse outcome in Māori patients, these results did not reach statistical significance due to the small sample size. It has previously been reported that most of the functional improvement following primary total arthroplasty surgery takes place in the first postoperative year and then plateaus.\textsuperscript{11}

Other studies have suggested that outcome measures such as the Oxford score at 6 months is representative of later scores and consequently the developers of the Oxford score used the scores at 6 months in their validation.\textsuperscript{7}

Therefore, it is likely that the differences identified between Māori and non-Māori at 1 year would again have been evident at 5 years postoperatively given greater numbers.

**Secondary outcomes**—It is well documented that Māori have poorer general and mental health than non-Māori and therefore the findings in this study that Māori had
worse general and mental health scores both preoperatively and postoperatively was not surprising.

The MCID has been reported as 2.0-7.8 for the SF-12 scores and so again these differences highlighted between Māori and non-Māori are likely to represent true clinical differences.\textsuperscript{16}

This study has important implications at a regional level as well as at a national level. On the surface it would appear that the Bay of Plenty District Health Board is providing an appropriate level of service for our local Māori population given the rates of Māori undergoing arthroplasty surgery as a proportion of our local population (13.77\% of arthroplasty patients are Māori vs 11.16\% of the general population over the age of 50 years identifying as Māori).\textsuperscript{17} It may be, however, that the intervention rate is disproportionately low relative to the disease burden in Māori patients.\textsuperscript{18}

New Zealand has an ageing population. The 65+ year age group comprises 12\% of the current population although this is expected to increase to more than 25\% by 2030 with particularly large increases expected in the elderly Māori population.\textsuperscript{17}

It may be, however, that the intervention rate is disproportionately low relative to the disease burden in Māori patients.\textsuperscript{18}

New Zealand has an ageing population. The 65+ year age group comprises 12\% of the current population although this is expected to increase to more than 25\% by 2030 with particularly large increases expected in the elderly Māori population.\textsuperscript{17}

It is predicted that the older Māori population in New Zealand will grow by 7.1\% between 2011 and 2026 whereas the older non-Māori population will increase by only 3.3\%.\textsuperscript{19} This increase will be driven by the growth in the 65+ year age group amongst which the number of Māori is predicted to increase by 121.8\% compared with just 60.3\% in non-Māori.\textsuperscript{19} This means that by 2026 Māori are predicted to comprise 9.5\% of the older people’s population in New Zealand (up from 6.8\% in 2006).\textsuperscript{19}

Consequently, it is likely that demand for arthroplasty surgery will increase in the future particularly amongst the Māori population and as such it is important that we continue to investigate Māori health outcomes in the setting of orthopaedics and specifically arthroplasty surgery to ensure that Māori are achieving equal outcomes to non-Māori and are accessing orthopaedic arthroplasty services at an appropriate level.

The Government and the Ministry of Health have made reducing the health inequalities that affect Māori a key priority. He Korowai Oranga (The Māori Health Strategy) was published in 2002 and outlines an action plan for addressing health disparities that affect Māori by providing a framework for the direction of Māori health development in the future.\textsuperscript{20}

Orthopaedic pathology and care does not feature in this publication. This paper identifies potential disparities in orthopaedic care and outcomes for Māori patients in New Zealand and highlights the need for further work to better understand and begin to address these disparities.

Competing interests: None identified.

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5. Tatau Kura Tangata (Health of Older Maori Chart Book 2011).
17. http://www.stats.govt.nz
18. Disability and Maori in New Zealand in 2006
20. He Korowai Oranga (The Maori Health Strategy 2002)
Cross-sectional study on prevalence, causes and avoidable causes of visual impairment in Māori children

CheeFoong Chong, Shuan Dai

Abstract

Aims To provide information and comparison pertaining to visual impairment of Māori children with other children in New Zealand in particular: prevalence of blindness, causes of visual impairment, and avoidable causes of visual impairment.

Methods Retrospective data collection utilising the WHO/PBL eye examination record for children with blindness and low vision at Blind and Low Vision Education Network New Zealand (BLENNZ), Homai. Individuals not of Māori ethnicity or over the age of 16 were excluded from the study.

Results 106 blind and 64 low-vision Māori children were studied. The main cause of blindness in Māori children is cortical visual impairment. Twenty-eight percent of causes of blindness in this population are potentially avoidable with non-accidental injury as the main cause.

Conclusions The prevalence of blindness and low vision in children amounts to 0.05% and 0.03%, respectively. The prevalence and causes of childhood blindness are comparable to the other ethnic groups in New Zealand. The main difference lies in avoidable causes of blindness, which appeared to be much higher in the Māori population. The leading cause of avoidable blindness in Māori children is caused by non-accidental injuries.

The impact of childhood blindness is sizeable. Blind children have a lifetime of blindness ahead of them compared to adult onset blindness. They often require more input in terms of emotional, social and economic needs. The estimated global burden of childhood blindness is only second to cataracts.

Childhood blindness affects approximately 0.05% of child population in New Zealand. This amounts to approximately 450 blind children in the country. This figure excludes a further 350 children with low vision. Hence, approximately 800 visually impaired children currently live in New Zealand.

Children who become blind have a higher mortality rate compared to non-blind children. It was reported that 60% of blind children die within 1 year of becoming blind.

Based on New Zealand 2006 census data, New Zealand consists of six main ethnic groups, listed in order of majority:

- European (67.6%)
- Māori (14.6%)
- Other (11.1%)
• Asian (9.2%)
• Pacific peoples (6.9%)
• Middle Eastern/Latin American/African (MELAA) (0.9%)

Of the 800 visually-impaired children in New Zealand, close to 25% of them are from a single ethnic group, the Māori people, who account for about 14.6% of the total New Zealand population.3

This study aimed to provide a snapshot of the Māori children in New Zealand, in particular:

• Prevalence of blindness
• Causes of visual impairment
• Avoidable causes of visual impairment

Methods

Ethics approval sought and granted by the University of Auckland Human Participants Ethics Committee.

The Blind and Low Vision Education Network New Zealand (BLENNZ) offers multi-disciplinary support for blind and visually impaired children in New Zealand. All visually impaired children who access governmental funding and support are registered with BLENNZ.

A pilot study from retrospective data collection from 1142 records of students registered with BLENNZ was conducted. Data pertaining to medical, ophthalmic and family history were studied. Demographics including age, gender, age of onset and ethnicity were recorded.

The UNICEF definition of child is adhered to. Only students of Māori ethnicity and below the age of 16 were included in the study.

Visual acuity was measured utilising the Snellen chart whenever possible, failing that a variety age appropriate visual assessments were conducted (optokinetic drum, 100s and 1000s, forced preferential testing, picture matching).

Ophthalmic examination included slit-lamp examination, detailed fundoscopy, retinoscopy, tonometry and kinetic visual fields (Goldman perimetry) where required.

The WHO/PBL Eye Examination Record for Children with Blindness and Low Vision (ERCB) was utilised to collect data from the students’ records. Students over the age of 16 are excluded from the study. Data collected were assessed for

• Requirement of each individual children for medical/surgical management, optical correction and low-vision aids,
• Assess preventable, reversible and treatable causes of childhood visual loss.

In individuals where there were 2 causes for visual impairment, the main cause of visual loss was recorded. If both causes of visual impairment are deemed equally significant, the most preventable or treatable cause was recorded.
The definition of avoidable causes of visual impairment used in this study encompasses both preventable and treatable causes of visual impairment. Preventable causes include:

- Causes that can be prevented all together; primary prevention
- Causes potentially preventable with early intervention; secondary prevention
- Causes where blindness or visual impairment can be reversed; tertiary prevention

**Results**

The demographics of Māori children enrolled with BLENNZ are summarised in Table 1. There is a slight male preponderance in Māori childhood blindness with 117 males to 87 females. Of the Māori school children, 106 of them are blind while a further 64 children have low vision.

**Table 1. Demographics of Māori children enrolled with Blind and Low Vision Education Network New Zealand (BLENNZ)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>None-Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Blind</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>34</td>
<td>54</td>
<td>10</td>
<td>106</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Male</td>
<td>21</td>
<td>33</td>
<td>6</td>
<td>57</td>
</tr>
<tr>
<td>Female</td>
<td>13</td>
<td>21</td>
<td>4</td>
<td>49</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average (Years)</td>
<td>10.9</td>
<td>9.1</td>
<td>9.5</td>
<td>7.2</td>
</tr>
<tr>
<td>Median (Years)</td>
<td>10.5</td>
<td>9.2</td>
<td>10.7</td>
<td>6.3</td>
</tr>
<tr>
<td>Range (Years)</td>
<td>4.3–15.9</td>
<td>1.7–16.0</td>
<td>3.5–14.3</td>
<td>0.4–15.3</td>
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<tr>
<td><strong>Onset of visual impairment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Since birth</td>
<td>21</td>
<td>47</td>
<td>8</td>
<td>76</td>
</tr>
<tr>
<td>1st Year</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>1-15 years</td>
<td>6</td>
<td>4</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Unknown</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>10</td>
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<tr>
<td><strong>Other disability</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>None</td>
<td>24</td>
<td>31</td>
<td>2</td>
<td>19</td>
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<tr>
<td>Hearing impairment</td>
<td>2</td>
<td>4</td>
<td>0</td>
<td>7</td>
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<td>Developmental delay</td>
<td>8</td>
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<td>7</td>
<td>64</td>
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<td>Physical disability</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>34</td>
</tr>
<tr>
<td>Epilepsy</td>
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<td>3</td>
<td>36</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td>4</td>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td><strong>Previous ophthalmic surgery</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>22</td>
<td>45</td>
<td>9</td>
<td>93</td>
</tr>
<tr>
<td>Cataract</td>
<td>4</td>
<td>6</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Corneal graft</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Strabismus</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Enucleation</td>
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<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

BLENNZ = Blind and Low Vision Education Network New Zealand.
Concomitant disabilities are more commonly seen with increasing levels of visual impairment. Forty-three percent of children with moderate visual impairment have associated disability compared to 82% of blind Māori children.

Ninety blind Māori children were bilaterally affected and a further 10 were unilaterally affected. Māori children with low vision are mostly bilaterally affected while only 3 were unilaterally affected.

Table 2. Causes of visual impairment and blindness in Māori children enrolled at BLENNZ, by anatomical site of the abnormality

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Moderate</th>
<th></th>
<th>Severe</th>
<th></th>
<th>Blind</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unilateral</td>
<td>Bilateral</td>
<td>Unilateral</td>
<td>Bilateral</td>
<td>Unilateral</td>
</tr>
<tr>
<td>Whole Globe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phthisis</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Anophthalmos</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Microphthalmos</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cornea</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corneal scar</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Keratoconus</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Corneal dystrophy</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lens</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cataract</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aphakia</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Uvea</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aniridia</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Coloboma</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Retina</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retinal dystrophy</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Albinism</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ROP</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Retinoblastoma</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Optic Nerve</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrophy</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hypoplasia</td>
<td>0</td>
<td>9</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Globe Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myopia</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Amblyopia</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>CVI</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>0</td>
<td>6</td>
<td>0</td>
<td>1</td>
<td>0</td>
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<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

ROP = Retinopathy of prematurity; CVI = Cerebral visual impairment.
The leading causes of blindness in Māori children are cerebral visual impairment (CVI) (41.0%) followed by optic nerve atrophy (19.0%) and optic nerve hypoplasia (13.0%).

Low-vision Māori children have a completely different mix of causality compared to causes of blindness; optic nerve hypoplasia (18.8%), nystagmus (10.9%), retinopathy of prematurity (ROP), optic nerve atrophy and CVI (9.4% each) (Table 2).

Avoidable causes of blindness for Māori children amounts to 28.5% of Māori childhood blindness. The leading causes of blindness in Māori children are non-accidental injury (NAI) (24.5%), neonatal trauma/asphyxia (15.1%) and neonatal infections (9.4%). Avoidable causes of low vision in Māori children are predominantly caused by aphakia (18.2%), corneal scarring (18.2%) and neonatal trauma/asphyxia (13.6%)

Table 3. Avoidable causes of visual impairment and blindness in Māori children

<table>
<thead>
<tr>
<th>Treatable/Reversible/Preventable Causes</th>
<th>Moderate visual impairment</th>
<th>Severe visual impairment</th>
<th>Blind</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non accidental injury</td>
<td>0</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>Accidental injury</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Neonatal trauma/asphyxia</td>
<td>2</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Neonatal infections</td>
<td>1</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Retinopathy of prematurity</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Amblyopia</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Cataract</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aphakia</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Corneal scarring</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 4. Causes of severe visual impairment and blindness in children, by anatomical site of the abnormality

<table>
<thead>
<tr>
<th>Site of abnormality</th>
<th>Causes of severe visual impairment and blindness (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Auckland data</td>
</tr>
<tr>
<td>Whole globe</td>
<td>1.1</td>
</tr>
<tr>
<td>Glaucoma</td>
<td>1.9</td>
</tr>
<tr>
<td>Cornea</td>
<td>3.0</td>
</tr>
<tr>
<td>Lens</td>
<td>3.4</td>
</tr>
<tr>
<td>Uvea</td>
<td>6.5</td>
</tr>
<tr>
<td>Retina</td>
<td>28.9</td>
</tr>
<tr>
<td>Optic nerve</td>
<td>13.7</td>
</tr>
<tr>
<td>Other</td>
<td>41.4</td>
</tr>
<tr>
<td>Number of children examined</td>
<td>263</td>
</tr>
</tbody>
</table>
Discussion

The significant impact and preventable nature of childhood blindness has not gone unnoticed by the World Health Organization (WHO): “The Right to Sight” programme, a global initiative of WHO and the International Agency for the Prevention of Blindness was established with an aim to eliminate avoidable childhood blindness by the year 2020.

The global prevalence of blindness in children was approximated to be 0.75/1000 children. The report found higher prevalence of childhood blindness in lower income regions as well as varying causes of visual impairment with varying levels of socioeconomic development.

The prevalence of blindness and low vision in Māori children in New Zealand amounts to 0.01% and 0.007% respectively. Correcting for ethnic specificity, the prevalence of blindness and low vision within Māori children is 0.5/1000 and 0.3/1000 respectively. This prevalence figures are comparable to the prevalence of visual impairment in Auckland children.

The three leading anatomical sites contributing to blindness in Māori children according to the WHO/PBL ERCB were other, optic nerve and retina. This is fairly similar to the anatomical causes of visual impairment in the Auckland child population (Table 4).

Table 5. Comparison on avoidable causes of blindness in Māori and other ethnic groups

<table>
<thead>
<tr>
<th>Treatable/Reversible/Preventable Causes of blindness</th>
<th>Māori ethnicity</th>
<th>Auckland data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non accidental injury</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>Accidental injury</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Motor vehicle accident</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Neonatal trauma/asphyxia</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Neonatal infections</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Glaucoma</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Retinopathy of prematurity</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Amblyopia</td>
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<td>0</td>
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<tr>
<td>Cataract</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aphakia</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Corneal scarring</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Although the causes and prevalence of visual impairment are comparable between the Auckland children and Māori children, the key difference lies in the avoidable causes of visual impairment (Table 5).

Avoidable causes accounts for 18% of blindness in Auckland children but in Māori children, this figure is close to 29%. The most significant difference in avoidable causes is in NAI, which accounts for 25% of all avoidable blindness in Māori
children. This is an underestimation of the prevalence of NAI in New Zealand as this study only included surviving NAI victims with visual impairment. From previous clinical audit (Dai 2008 unpublished), the rate of NAI mortality in New Zealand was approximately 30%.

Neonatal trauma/asphyxia contributes to 15% of avoidable blindness. This figure coincides with Auckland children data, the causes for this is likely to be multifactorial and beyond the scope of this study.

Table 6 shows non-accidental injuries sustained by Māori children and their respective visual outcome.

Table 6. Injuries sustained and visual outcome of Māori children with NAI

<table>
<thead>
<tr>
<th>Case</th>
<th>Injuries</th>
<th>Visual outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Acute and chronic SDH, multiple left rib fractures, global brain injury, superficial bruising (head, abdomen, chest)</td>
<td>Blind</td>
</tr>
<tr>
<td>2</td>
<td>Acute and chronic SDH; right retinal haemorrhage, bilateral tibial metaphyseal fractures</td>
<td>Blind</td>
</tr>
<tr>
<td>3</td>
<td>Acute on chronic SDH; severe diffuse brain injury; bilateral retinal haemorrhages</td>
<td>Blind</td>
</tr>
<tr>
<td>4</td>
<td>Bilateral fronto-parietal subacute SDH, greater on right</td>
<td>Blind</td>
</tr>
<tr>
<td>5</td>
<td>Bilateral SDH acute on chronic; multiple retinal haemorrhages; diffuse brain injury</td>
<td>Severe visual impairment</td>
</tr>
<tr>
<td>6</td>
<td>Bilateral SDH, extensive brain injury, left retinal haemorrhage and retinoschisis</td>
<td>Blind</td>
</tr>
<tr>
<td>7</td>
<td>Bilateral SDH, left TBI, bilateral severe retinal haemorrhage with left RD</td>
<td>Blind</td>
</tr>
<tr>
<td>8</td>
<td>Bilateral SDH, Retinal haemorrhages, global developmental delay</td>
<td>Blind</td>
</tr>
<tr>
<td>9</td>
<td>Bilateral acute SDH; right parietal skull fracture; multiple rib fractures; multiple long-bone fracture right retinal haemorrhages and retinoschisis</td>
<td>Blind</td>
</tr>
<tr>
<td>10</td>
<td>Shaken baby syndrome</td>
<td>Blind</td>
</tr>
<tr>
<td>11</td>
<td>Acute on chronic SDH, TBI</td>
<td>Blind</td>
</tr>
<tr>
<td>12</td>
<td>Acute on chronic SDH</td>
<td>Blind</td>
</tr>
</tbody>
</table>

NAI=non-accidental injury; SDH=subdural haemorrhage; TBI=traumatic brain injury; RD=retinal detachment.

In conclusion, the demographics, prevalence and causes of visual impairment are fairly similar comparing Māori children with children of other ethnicity in Auckland. The main difference lies in avoidable causes of blindness, which appeared to be much higher in the Māori population.

The cause of this discrepancy between Māori children and other ethnic groups are likely to be multifactorial. This requires further research, which is beyond the scope of this study.

The leading cause of avoidable blindness in Māori children is caused by NAI followed by neonatal trauma. In order to eradicate avoidable blindness in children by the year 2020 as proposed by “The Right to Sight” programme, further resources and work will need to be channelled to manage neonatal trauma and asphyxia.
Competing interests: None identified.

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References:

3. Chong C, Dai S. Prevalence and causes of childhood blindness in Auckland, New Zealand. (Data yet to be published)
Injury severity and 3-month outcomes among Māori: results from a New Zealand prospective cohort study

Brett Maclennan, Emma Wyeth, Brendan Hokowhitu, Suzanne Wilson, Sarah Derrett

Abstract

Aims To examine the prevalence of health and social outcomes pre- and 3 months post-injury, and the association between New Injury Severity Scores (NISS) and 3-month outcomes, for the Māori cohort of the Prospective Outcomes of Injury Study.

Methods New Zealand residents were recruited from the Accident Compensation Corporation’s entitlement claims register and participants interviewed at 3 months post-injury. Those who reported Māori ethnicity (n=566) were included in the Māori cohort.

Results States indicative of favourable health were less prevalent among the cohort post-injury than pre-injury for all measures examined. Approximately half the cohort were experiencing difficulties walking 3 months after their injury, over two-thirds a level of pain or discomfort, and more than half a level of psychological distress. The prevalence of disability was 49%. The prevalence of some adverse outcomes increased with increasing NISS but a high level of problems were still experienced by those classified as having a ‘minor’ injury. Nonetheless, a majority of the cohort were satisfied with life and they considered themselves to be of good to excellent overall health.

Conclusions Findings emphasise the importance of injury prevention and appropriate post-injury care to reduce the burden experienced by Māori due to injury.

Injury is a leading contributor to the burden of death and disability in several countries including New Zealand. The 2006 New Zealand Household Disability Survey found that one in six residents aged 15 years and over was living with a disability. In nearly one-third of cases injury was the reported cause, and among those aged 15-64 years it was the leading cause of disability.

Māori, New Zealand’s indigenous population, comprise 15% of the country’s total population and experience a disproportionate burden following injury. Those aged 15-64 years are at greater risk of mortality (RR: 2.29) and hospitalisation (RR: 1.62) from unintentional injury than non-Māori in this age group.

Prevalence of disability due to injury is also higher among Māori (31.4%) than non-Māori (29.3%) aged 15 years and over. Kingi and Bray have previously qualitatively explored Māori perceptions of disability with 15 participants. Themes identified included the impact of colonial history on knowledge of Māori language and culture, difficulties of access to healthcare providers, traditional foods and resources, and the disabling effect of this impact on reaching and maintaining hauora (i.e., optimal health and well-being). Negative socioeconomic factors were also perceived as disabling, more so than functional impairment. Their study points to the impact of
broader sociohistorical factors on perceptions of Māori disability, which reflect Māori models of overall health and wellbeing. One model, Te Whare Tapa Whā (literally, a four-sided house), for example, advocates that Māori health can be encapsulated through taha wairua (the spiritual dimension), taha hinengaro (the mental dimension), taha tinana (the physical dimension) and taha whānau (the social relationship dimension). A particular focus of POIS is to provide robust and relevant evidence about injury outcomes for Māori. Such work is important to inform efforts aimed at reducing adverse injury outcomes for Māori.

The aims of this paper, therefore, are to examine the prevalence of a range of health and social outcomes pre-injury and 3 months post-injury, and to examine the prevalence of 3 month post-injury outcomes in relation to injury severity.

**Methods**

The POIS cohort was recruited from claimants on the entitlement claims’ register at the Accident Compensation Corporation (ACC), New Zealand’s comprehensive “no-fault” insurer for both residents and visitors to the country who sustain an injury. If an injury is serious enough to be likely to necessitate a week or more away from paid work, or if supports such as home help and transportation (e.g., to and from work or a medical appointment) are required, the injured person is placed on ACC’s entitlement claimants register. Those on the register (excluding those whose injuries were the result of self-harm or sexual assault) were eligible to participate if they were New Zealand residents aged 18-64 years (inclusive), and living in one of five regions from throughout New Zealand. The aim of the POIS was to identify factors leading to disability following injury with a focus on outcomes among the usual working-age population. Disability among younger and older New Zealanders is also of concern. We limited the eligible age group as participation in paid employment is an important indicator of outcomes following injury, and selecting the working-age population allowed us to use consistent measures (rather than requiring versions for youth or older people) for all participants and because people in this working-age group report the greatest prevalence of disability. The regions, which include a mix of urban and rural areas, were: Auckland City, Manukau City, Gisborne and the provinces of Otago and Southland. They were selected to provide a broad range of communities in terms of sociodemographic characteristics and to ensure that sufficient Māori were recruited.

Claimants in these areas were posted a letter by ACC inviting them to take part in the study. Members of the university research team were then provided with the details of those who did not opt out of being invited to participate in an interview. A total of 2856 (59%) gave consent and participated in the 3 month post-injury interview phase conducted between December 2007 and August 2009. Ethical approval of POIS was granted by the New Zealand Health and Disability Multi-region Ethics Committee (MEC/07/07/093). Further details on the study protocol are provided elsewhere.
Participants were interviewed up to four times post-injury. The focus of this paper is on the first interview conducted 3 months, on average, following injury. At this interview participants were asked an array of questions about their injury and personal characteristics (pre- and post-injury). This information was primarily collected via telephone although a small number of interviews were administered face-to-face or by post.\(^{16}\)

Data on ethnicity was obtained using the ethnicity question from the 2006 New Zealand Census of Population and Dwellings which allows participants to report belonging to one or more ethnic groups.\(^ {19}\)

For the purpose of these analyses, those who reported Māori ethnicity, whether or not they reported additional ethnicities, were included in the Māori cohort (n=566).\(^ {17}\)

Injury severity was measured using Abbreviated Injury Scale (AIS) scores derived from ACC diagnosis data.\(^ {15}\) The sum of the squares of each participant’s three highest AIS scores was calculated to provide their NISS.\(^ {20}\) Participants were then grouped for analysis into NISS 1-3 (one or more minor injuries), NISS 4-6 (one moderate injury with or without other minor injuries) and NISS>6 (two or more moderate injuries or one or more serious injuries).

NISS were unable to be calculated for 23 Māori participants. Those admitted to hospital or treated at an Emergency Department for 3 hours or more within seven days of the injury event were identified from the National Minimum Dataset (a nationwide dataset of hospital discharge information) and were classified as having been ‘hospitalised’ as a result of their injury.\(^ {15}\)

A range of variables measuring psychological, physical and social well-being pre- and post-injury was examined. General health was assessed by asking participants to rate their overall health on a five-point scale (poor to excellent) and their health now compared to 1 year previous (much/somewhat better, same, much/somewhat worse).

The EQ-5D\(^ {21}\) was used to assess health status in relation to mobility, problems with self-care, ability to perform usual activities, experience of pain or discomfort, and experience of anxiety or depression. Information on psychosocial health was ascertained using the Kessler 6\(^ {22}, 23\) to provide a measure of psychological distress in the previous 4 weeks (categorised as: low 1–3; moderate 4–12; high 13–24), and three items enquiring about overall happiness (very happy, fairly happy, not too happy), satisfaction with life (completely satisfied, mostly satisfied, neither satisfied nor dissatisfied, mostly dissatisfied, completely dissatisfied) and satisfaction with social relationships (completely satisfied, mostly satisfied, neither satisfied nor dissatisfied, mostly dissatisfied, completely dissatisfied).

The last two variables were categorised as ‘satisfied’ (the first two response options) and ‘not satisfied’ (last three response options) for analyses. Disability status was measured using the 12-item WHODAS II, those with a summed scoring \(\geq 10\) being defined as having a disability.\(^ {15}, 24\) Participants were also asked how their recovery was meeting their expectations (much/somewhat better than expected, as expected, much/somewhat worse than expected).

Stata/SE v12.1 software was used to analyse the data.\(^ {25}\) Percentage estimates and 95% confidence intervals were produced to determine the pre- and post-injury (3 months) prevalence of a range of health and social outcomes. The prevalences of post-injury outcomes by injury severity were also estimated.

**Results**

The median time to interview for the group (n=566) was 3.1 months post-injury (IQR: 2.5–4.1 months). Females comprised just over one-third (34%) of the Māori cohort. The median age of participants at interview was 38 years (IQR: 28–48 years).

Approximately one in eight (12%) reported having ‘not enough’ household income pre-injury to meet their everyday needs (e.g. accommodation, food, clothing), just over one in four (28%) had ‘just enough’, and the remainder had ‘enough’ (40%) or ‘more than enough’ (20%). Further sociodemographic and health information on the Māori cohort is provided elsewhere.\(^ {26}\)

The majority (85%) of injuries sustained by the cohort were of minor to moderate anatomical severity and less than 1.5% of participants had a ‘severe’ injury as defined...
by a NISS of 16 or more (Table 1). The median NISS was 4 (IQR = 1-5). Just over one-quarter (27%) were hospitalised as a result of their injury.

Table 1. Description of injuries

<table>
<thead>
<tr>
<th>Variables</th>
<th>n¹</th>
<th>% of cohort²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Injury region and nature</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head and/or neck</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracranial</td>
<td>17</td>
<td>3.0</td>
</tr>
<tr>
<td>Superficial</td>
<td>18</td>
<td>3.2</td>
</tr>
<tr>
<td>Spine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sprain or dislocation</td>
<td>95</td>
<td>16.8</td>
</tr>
<tr>
<td><strong>Upper extremity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fracture</td>
<td>81</td>
<td>14.3</td>
</tr>
<tr>
<td>Open wound</td>
<td>36</td>
<td>6.4</td>
</tr>
<tr>
<td>Sprain or dislocation</td>
<td>78</td>
<td>13.8</td>
</tr>
<tr>
<td>Superficial</td>
<td>36</td>
<td>6.4</td>
</tr>
<tr>
<td><strong>Lower extremity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fracture</td>
<td>89</td>
<td>15.7</td>
</tr>
<tr>
<td>Open wound</td>
<td>33</td>
<td>5.8</td>
</tr>
<tr>
<td>Sprain or dislocation</td>
<td>146</td>
<td>25.8</td>
</tr>
<tr>
<td>Superficial</td>
<td>34</td>
<td>6.0</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e.g. burn, crush injury</td>
<td>102</td>
<td>18.0</td>
</tr>
<tr>
<td><strong>Injury severity (NISS)³</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–3</td>
<td>241</td>
<td>44.4</td>
</tr>
<tr>
<td>4–6</td>
<td>220</td>
<td>40.5</td>
</tr>
<tr>
<td>&gt;6</td>
<td>82</td>
<td>15.1</td>
</tr>
</tbody>
</table>

¹ Total exceeds 566 as some participants had multiple injury types
² Percentage among the 566 participants
³ NISS were unable to be calculated for 23 participants
⁴ Percentage among the 543 participants for whom a NISS could be derived

**General health**

More than two-thirds of the cohort described their overall health as being ‘very good’ or ‘excellent’ prior to their injury and only 8% felt it was ‘fair’ or ‘poor’ (Table 2). Three months post-injury, a greater proportion (26%) reported ‘fair’ or ‘poor’ overall health. More than 40% of the cohort felt their health at 3 months post-injury was worse than 12 months prior.

**EQ-5D health status**

Close to one-half of the cohort was experiencing problems with mobility and one-quarter problems with self-care at 3 months post-injury (Table 2). Over half reported trouble performing usual activities at 3 months, 70% were experiencing pain or discomfort, and more than one-quarter were experiencing anxiety or depression. For each of the five dimensions, a statistically significant increase was observed in the proportions reporting EQ-5D problems post-injury compared to pre-injury.
Table 2. Health status of Māori participants pre- and 3 months post-injury

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre-injury</th>
<th>Post-injury (3 months)</th>
<th>( \chi^2 ) (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( n ) % (95% CI)</td>
<td>( n ) % (95% CI)</td>
<td></td>
</tr>
<tr>
<td><strong>General health</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall health</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Excellent/very good</strong></td>
<td>383 68 (64, 72)</td>
<td>229 41 (37, 45)</td>
<td>101.2 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Good</strong></td>
<td>136 24 (21, 28)</td>
<td>185 33 (29, 37)</td>
<td></td>
</tr>
<tr>
<td><strong>Fair/poor</strong></td>
<td>45 8 (6, 10)</td>
<td>148 26 (23, 30)</td>
<td></td>
</tr>
<tr>
<td><strong>Health compared to 1 year ago</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Better</strong></td>
<td>– –</td>
<td>114 21 (17, 24)</td>
<td></td>
</tr>
<tr>
<td><strong>Same</strong></td>
<td>– –</td>
<td>210 38 (34, 42)</td>
<td></td>
</tr>
<tr>
<td><strong>Worse</strong></td>
<td>– –</td>
<td>226 41 (37, 45)</td>
<td></td>
</tr>
<tr>
<td><strong>EQ-5D health status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mobility</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>No problems walking</strong></td>
<td>529 93 (91, 96)</td>
<td>307 54 (50, 58)</td>
<td>225.5 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Some problems walking</strong></td>
<td>37 7 (4, 9)</td>
<td>254 46 (42, 50)</td>
<td></td>
</tr>
<tr>
<td><strong>Confined to bed</strong></td>
<td>– 5</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Self-care</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>No problems with self-care</strong></td>
<td>554 98 (97, 99)</td>
<td>428 76 (72, 79)</td>
<td>122.0 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Some problems with self-care</strong></td>
<td>12 2 (1, 3)</td>
<td>129 24 (21, 28)</td>
<td></td>
</tr>
<tr>
<td><strong>Unable to wash/dress self</strong></td>
<td>– 9</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Usual activities</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>No problems performing</strong></td>
<td>532 94 (92, 96)</td>
<td>250 44 (40, 48)</td>
<td>328.9 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Some problems performing</strong></td>
<td>33 6 (4, 8)</td>
<td>269 56 (52, 60)</td>
<td></td>
</tr>
<tr>
<td><strong>Unable to perform</strong></td>
<td>1 47</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pain or discomfort</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>None</strong></td>
<td>504 89 (87, 92)</td>
<td>172 30 (27, 34)</td>
<td>405.8 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Some</strong></td>
<td>57 11 (8, 13)</td>
<td>351 70 (66, 73)</td>
<td></td>
</tr>
<tr>
<td><strong>Extreme</strong></td>
<td>4 42</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Anxiety or depression</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>None</strong></td>
<td>523 93 (90, 95)</td>
<td>407 72 (68, 76)</td>
<td>81.7 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Moderate</strong></td>
<td>37 7 (5, 10)</td>
<td>144 28 (24, 32)</td>
<td></td>
</tr>
<tr>
<td><strong>Extreme</strong></td>
<td>5 14</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Psychosocial health</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Psychological distress (Kessler 6)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Low (0–3)</strong></td>
<td>– –</td>
<td>250 45 (41, 49)</td>
<td></td>
</tr>
<tr>
<td><strong>Moderate (4–12)</strong></td>
<td>– –</td>
<td>244 44 (40, 48)</td>
<td></td>
</tr>
<tr>
<td><strong>High (13–24)</strong></td>
<td>– –</td>
<td>60 11 (8, 13)</td>
<td></td>
</tr>
<tr>
<td><strong>Overall happiness</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Very happy</strong></td>
<td>281 50 (46, 54)</td>
<td>162 29 (25, 32)</td>
<td>102.1 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Fairly happy</strong></td>
<td>263 46 (42, 51)</td>
<td>282 50 (46, 54)</td>
<td></td>
</tr>
<tr>
<td><strong>Not too happy</strong></td>
<td>22 4 (2, 5)</td>
<td>122 22 (18, 25)</td>
<td></td>
</tr>
<tr>
<td><strong>Satisfaction with life</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Satisfied</strong></td>
<td>521 93 (90, 95)</td>
<td>402 71 (68, 75)</td>
<td>85.1 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Not satisfied</strong></td>
<td>42 7 (5, 10)</td>
<td>161 29 (25, 32)</td>
<td></td>
</tr>
<tr>
<td><strong>Satisfaction with global social relationships</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Satisfied</strong></td>
<td>529 94 (92, 96)</td>
<td>455 81 (78, 84)</td>
<td>42.8 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Not satisfied</strong></td>
<td>35 6 (4, 8)</td>
<td>108 19 (16, 22)</td>
<td></td>
</tr>
<tr>
<td><strong>Disability (WHODAS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>No (0–9)</strong></td>
<td>519 92 (90, 94)</td>
<td>283 51 (47, 55)</td>
<td>234.8 (&lt;0.001)</td>
</tr>
<tr>
<td><strong>Yes (10+)</strong></td>
<td>44 8 (6, 10)</td>
<td>273 49 (45, 53)</td>
<td></td>
</tr>
<tr>
<td><strong>Recovery course</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Better than expected</strong></td>
<td>– –</td>
<td>263 48 (44, 52)</td>
<td></td>
</tr>
<tr>
<td><strong>As expected</strong></td>
<td>– –</td>
<td>141 26 (22, 29)</td>
<td></td>
</tr>
<tr>
<td><strong>Worse than expected</strong></td>
<td>– –</td>
<td>144 26 (23, 30)</td>
<td></td>
</tr>
</tbody>
</table>
Table 3. Prevalence of 3-month outcomes by injury severity

<table>
<thead>
<tr>
<th>Variables</th>
<th>NISS* 1–3 (n = 241)</th>
<th>Injured Severity NISS 4–6 (n = 220)</th>
<th>NISS &gt; 6 (n = 82)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%   (95% CI)</td>
<td>n</td>
</tr>
<tr>
<td>General health</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor overall health</td>
<td>65</td>
<td>21</td>
<td>9  (5, 12)</td>
</tr>
<tr>
<td>Health worse than 1 year ago</td>
<td>226</td>
<td>84</td>
<td>36  (30, 42)</td>
</tr>
<tr>
<td>Any problems with mobility</td>
<td>259</td>
<td>90</td>
<td>37  (31, 43)</td>
</tr>
<tr>
<td>Any problems with self-care</td>
<td>138</td>
<td>47</td>
<td>20  (14, 25)</td>
</tr>
<tr>
<td>Any problems performing usual activities</td>
<td>316</td>
<td>109</td>
<td>45  (39, 52)</td>
</tr>
<tr>
<td>Any pain or discomfort</td>
<td>393</td>
<td>151</td>
<td>63  (57, 69)</td>
</tr>
<tr>
<td>Any anxiety or depression</td>
<td>158</td>
<td>61</td>
<td>25  (20, 31)</td>
</tr>
<tr>
<td>Psychosocial health</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High psychological distress</td>
<td>60</td>
<td>25</td>
<td>11  (7, 15)</td>
</tr>
<tr>
<td>Not too happy</td>
<td>122</td>
<td>45</td>
<td>19  (14, 24)</td>
</tr>
<tr>
<td>Not satisfied with life</td>
<td>161</td>
<td>58</td>
<td>24  (19, 30)</td>
</tr>
<tr>
<td>Not satisfied with social relationships</td>
<td>108</td>
<td>46</td>
<td>19  (14, 24)</td>
</tr>
<tr>
<td>Disability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>273</td>
<td>99</td>
<td>42  (35, 48)</td>
</tr>
<tr>
<td>Recovery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worse than expected</td>
<td>144</td>
<td>55</td>
<td>24  (18, 29)</td>
</tr>
</tbody>
</table>

* NISS (New Injury Severity Score) could not be calculated for 23 participants; NB: The total number (n) in each NISS group varies by injury outcome.
Psychosocial health

A moderate level of psychological distress was being experienced by nearly half the cohort 3 months post-injury and one in 10 were experiencing a high level of psychological distress (Table 2). Pre-injury information on general psychological well-being was not collected using the Kessler 6. Half the cohort reported that they were ‘very happy’ pre-injury and 46% reported being ‘fairly happy’, however, fewer participants were ‘very happy’ post-injury and more were ‘not too happy’.

Nonetheless, 71% were satisfied with life and 81% satisfied with global social relationships at 3 months, although these percentages were lower than pre-injury prevalence rates of 93% and 94%, respectively.

Disability

Prior to injury the prevalence of disability (WHODAS≥10) was 8% (Table 2). This had increased to 49% at 3 months post-injury.

Recovery

In terms of self-perceived recovery from the sentinel injury, about half believed they were recovering ‘better than expected’ 3 months post-injury, however, one-quarter reported their recovery as being ‘worse than expected’ (Table 2).

Prevalence of outcomes by injury severity (NISS)

General health—The proportion considering their health to be worse than 12 months prior increased with increasing injury severity. A quarter (24%) of those in the highest injury severity group considered their health to be ‘poor’ at 3 months post-injury compared to 9% in the lower severity groups (Table 3).

EQ-5D health status—The tendency to report mobility problems at 3 months increased with increasing injury severity (Table 3). There was no discernible difference between the three injury severity groups for self-care difficulties. Like mobility problems, problems performing usual activities, pain or discomfort, and anxiety or depression also tended to increase with increasing injury severity (although with overlapping 95% confidence intervals in some cases). The prevalence of these problems in the low severity group was still considerable, ranging from 20% to 63% across the five EQ-5D items.

Psychosocial health—A third (34%) of participants in the high injury severity group were ‘not too happy’ 3 months post-injury compared to a fifth in the lower severity groups (Table 3). A higher proportion (37%) in this group was also not satisfied with life at 3 months compared to the lowest group (20%) although differences between the groups were not statistically significant. Psychological distress and satisfaction with relationships did not vary substantially by injury severity.

Disability and recovery progress—Disability prevalence at 3 months post-injury ranged from 42% in the lowest injury severity group to 63% in the highest (Table 3). Participants’ views on recovery progress did not vary substantially by injury severity.
Discussion

High levels of adverse outcomes were observed 3 months post-injury among the POIS Māori cohort. Almost half were experiencing problems with mobility, a majority were having difficulties performing their usual activities, and most were suffering some or extreme pain or discomfort. Over half were experiencing a level of psychological distress.

For all the measures for which we could make pre- and post-injury comparisons, there was a statistically significant increase in the prevalence of adverse outcomes at 3 months. The prevalence of some of these problems increased with injury severity but those classified as having sustained a minor injury were not immune to adverse outcomes.

A substantial proportion in each injury severity group reported their health was worse 3 months after their injury compared to 1 year ago. Nonetheless, a majority of the cohort considered themselves to be at least ‘fairly happy’, satisfied with their social relationships and life in general, and of ‘good’ if not ‘very good’ or ‘excellent’ overall health.

Previous research into injury outcomes, among both Māori and non-Māori, has tended to focus on particular injury types or people sustaining ‘severe’ injuries that have resulted in hospitalisation.\(^\text{17}\) Fatal and serious non-fatal injuries are only a small part of the “injury iceberg”.\(^\text{27, 28}\) These injuries are far outnumbered by those of minor to moderate severity and many of these less severe injuries result in disability.\(^\text{27, 28}\)

Recruiting participants from the ACC entitlement claimants’ register in POIS has allowed examination of longitudinal outcomes among those incurring injuries of ‘minor’ or ‘moderate’ NISS severity, most of which did not result in a hospital visit. It is a strength of the study that recruitment of Māori with lower NISS-severity injuries via ACC was possible. Inclusion of Māori with such injuries in the POIS reveals that the burden in terms of adverse outcomes is not restricted to those with higher NISS severity alone.

It is important to note that following letters of invitation being sent to potential participants on our behalf by ACC, the study was conducted independently of ACC (the insurer) thereby reducing any incentive for participants to exaggerate adverse outcomes in order to maximise compensation entitlements.

The POIS cohort was not designed to be a representative sample of all ACC claimants or the general or Māori populations and the results may not be generalisable to these larger groups. However, this does not diminish our finding that a significant proportion of the POIS Māori cohort, many of whom sustained injuries of low-to-moderate severity, was experiencing adverse injury outcomes at 3 months.

A potential source of recall bias was that pre-injury health information was collected retrospectively at the 3 month interview. Participants may have overestimated their pre-injury health status unaware that they were comparing it to their post-injury health status. This would lead to changes pre- to post-injury being inflated, however, comparisons of reported health status as measured by the EQ-5D revealed only small differences between pre-injury health and health at 5 and 12 months for those POIS
participants who had recovered from their injury. This suggests that bias from using recalled pre-injury health information is likely to be minor.

Our finding that three-quarters of the Māori cohort considered their health at 3 months to be ‘good’ to ‘excellent’ suggests that many had adapted to life with injury. This is consistent with Kingi and Bray’s finding that external factors were considered more disabling than any functional impairment. It is possible that for our participants, happiness and satisfaction with life in the face of marked adverse outcomes (e.g., pain or discomfort) was aided by positive social relationships (e.g., support from whānau and social networks).

These positive relationships, combined with other factors (e.g., self-efficacy), may have been promoting favourable health and well-being overall. This premise resonates with Māori models of health which are based on a number of aspects collectively contributing to total well-being (e.g.,) and which will be the focus of future research.

The purpose of this study was to describe a range of pre-injury and 3 month post-injury outcomes, by injury severity, in the POIS Māori cohort. It addresses a dearth of research examining injury outcomes for Māori across a range of injury types and severities. We intentionally did not compare findings between Māori and non-Māori nor take into account potential confounders. Focussing specifically on outcomes for Māori provides greater insight into areas that require further attention for this particular group and helps avoid a ‘deficit model’ approach.

The findings from this research will inform future regression analyses that control for pre-injury characteristics (e.g., existing comorbidities) and potential confounders in order to identify potentially modifiable factors that can be addressed to improve post-injury outcomes for Māori. Analysis of data at 12 and 24 months post-injury will also permit examination of whether, and how, injury outcomes for Māori differ from the 3 month post-injury period.

To the best of our knowledge, this study is the first to have examined the prevalence of various health and social outcomes following injury among an indigenous population. It reveals that injuries among Māori, including those of minor severity, can lead to adverse physical and psychological outcomes 3 months later. This may be due to the fact that injuries defined by NISS (a threat-to-life measure) as minor-to-moderate can still result in a high level of disability.

The findings in this paper highlight the importance of identifying improved strategies to prevent injury, including ‘minor’ injuries, and for appropriate rehabilitation for injured Māori, irrespective of injury severity.

Competing interests: None identified.

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**References:**

25. StataCorp LP, College Station, TX, USA.
New Zealand guidelines for the diagnosis of acute rheumatic fever: small increase in the incidence of definite cases compared to the American Heart Association Jones criteria

Nigel J Wilson, Lesley Voss, Johan Morreau, Joanna Stewart, Diana Lennon

Abstract

Aim The aim of the study was to compare utilisation of the New Zealand guidelines for the diagnosis of acute rheumatic fever (ARF) compared to the American Heart Association Jones criteria in a cohort of children

Method Retrospective review of 79 consecutive hospital diagnosed cases of ARF referred for secondary penicillin prophylaxis. The 2006 New Zealand guidelines for ARF were applied to the cohort and the diagnostic classification compared to classification using the American Heart Association 1992 Jones criteria. Cases were defined as definite, probable, possible or not ARF. The New Zealand guidelines use subclinical (echocardiographic) carditis as a major criterion of ARF. Monoarthritis, if associated with anti-inflammatory medicine usage likely preventing polyarthritis, is also accepted as a major criterion.

Results Sixty-six cases were considered to be possible, probable or definite first episode of occurrence ARF. Utilisation of the New Zealand guidelines resulted in 16% (CL 7–29%) more cases defined as definite ARF than using American Heart Association 1992 Jones criteria (59/66 cases vs 51/66 cases). Polyarthitis was the most frequent presenting symptom. Of those classified as definite ARF, 11% had monoarthritis with anti-inflammatory usage.

Clinical carditis was present in 55% and subclinical carditis in 30%. The utilisation of subclinical carditis as a major criterion influenced the diagnosis to become definite ARF in 8% of the cohort only, as the remainder had polyarthritis or Sydenham’s chorea as a major criterion.

Conclusion Utilisation of New Zealand guidelines for the diagnosis of ARF result in a modest increase (16%) in cases classified as definite ARF compared to the 1992 Jones criteria.

Acute rheumatic fever (ARF) remains endemic among Māori and Pacific populations in New Zealand with rising incidence in these groups. Recurrences of ARF often lead to established rheumatic heart disease (RHD) with significant morbidity, early mortality and expense.

Recurrences can be prevented by secondary penicillin prophylaxis using intramuscular benzathine penicillin (BPG) so it is of great importance that cases of ARF are not misdiagnosed or underdiagnosed. In the USA where RF is no longer endemic, the American Heart Association (AHA) have, appropriately for their country, moved to increase the specificity of diagnosis with successive revisions of the Jones criteria.
Conversely, New Zealand\textsuperscript{12,15} and Australia\textsuperscript{13} have introduced guidelines aimed at increasing the sensitivity of diagnosis for ARF mainly by accepting subclinical carditis\textsuperscript{14} and broader definitions of arthritis for major criteria of ARF.

A landmark New Zealand RCT for primary prevention of rheumatic fever using treatment of GAS throat infection in a cohort of children in a high RF prevalence population in New Zealand was published in 2009.\textsuperscript{16} The case ascertainment committee for this study comprised a general paediatrician, an infectious diseases paediatrician and a paediatric cardiologist who decided whether a case met the 1956 and 1965 Jones criteria for definite or probable ARF (analysis A) or the 1992 Jones criteria (analysis B).\textsuperscript{16}

A large database of clinical and laboratory features for ARF cases in this study was gathered from the records from KidzFirst Hospital, South Auckland and Starship Children’s Hospital, Auckland with a the diagnoses of ARF, RF or RHD over the 4-year period 1998–2001 inclusive.

The aim of the current study was to use this database to compare the diagnosis of definite ARF using the New Zealand guidelines\textsuperscript{12,15} to that using the latest American Heart Association Jones Criteria, the 1992 update.\textsuperscript{11} The secondary aim is to highlight the signs and symptoms for the diagnosis of ARF, for which there is no laboratory test. Diagnosis of ARF remains a clinical decision, often with diagnostic difficulty.

\textbf{Methods and definitions}

The hospital records for all children attending one of the randomised controlled trial study schools, admitted to hospital within the study period of 1998–2001 and with a discharge diagnosis of acute rheumatic fever were reviewed.\textsuperscript{16} Those identified as being RHD or recurrences were removed and those incorrectly diagnosed as RF identified.

Files were reviewed meticulously looking at the patient’s referral letter, the admitting officer’s notes, the paediatric junior and senior staff notation on clinical findings on admission and during the hospital stay.

Echocardiograms were reviewed in full if the echocardiographic report was ambiguous or where the report indicated minor degrees of mitral or aortic valve regurgitation to determine whether regurgitation was pathological or not.\textsuperscript{14} Laboratory results were available at source if they were not recorded in the clinical file. This study\textsuperscript{16} had received Auckland Regional ethics committee approval.

The differences between the New Zealand guidelines criteria and the 1992 Jones criteria are shown in Table 1. The main modification made to the AHA Jones 1992 update\textsuperscript{11} for the New Zealand guidelines is the acceptance of echocardiographic evidence of carditis as a major manifestation.

In addition monoarthritis is accepted as the presenting feature if there was a history of NSAID use that was likely to have aborted classical migratory polyarthritis.\textsuperscript{12}

\begin{itemize}
  \item Arthralgia
  \item Fever >38\degree C
  \item Elevated acute phase reactants (ESR>50, CRP>30)
  \item Prolonged PR interval on ECG (related to age\textsuperscript{12})
\end{itemize}

The diagnosis of definite ARF is made when there is either 1 major and 2 minor manifestations or 2 major manifestations plus (always) evidence of a preceding GAS infection.

The New Zealand guidelines\textsuperscript{12} for ARF use the following definitions:
Table 1 Major criteria used for acute rheumatic fever (ARF)

<table>
<thead>
<tr>
<th>New Zealand guidelines</th>
<th>1992 Jones criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Migratory polyarthritis</td>
<td>Migratory polyarthritis</td>
</tr>
<tr>
<td>History of hip or knee pain precluding weight bearing accepted as arthritis</td>
<td>Arthritis must be medically observed</td>
</tr>
<tr>
<td>Monoarthritis if there was a history of non-steroidal anti-inflammatory usage</td>
<td>Monoarthritis not accepted</td>
</tr>
<tr>
<td>Clinical carditis</td>
<td>Clinical carditis</td>
</tr>
<tr>
<td>Subclinical carditis</td>
<td>Subclinical carditis not accepted</td>
</tr>
<tr>
<td>Chorea</td>
<td>Chorea</td>
</tr>
<tr>
<td>Erythema marginatum</td>
<td>Erythema marginatum</td>
</tr>
<tr>
<td>Subcutaneous nodules</td>
<td>Subcutaneous nodules</td>
</tr>
</tbody>
</table>

Definition of arthritis—Arthralgia was defined as pain in a joint without signs of swelling, tenderness or decreased range of movement. Arthritis is “classified as swelling of the joint in the presence of two or more of the following: limitation of movement, hotness of the joint and pain in the joint and/or tenderness”.

A history of arthritis of any joint was accepted as arthritis even in the absence of observation by a medical observer if that history was clear cut. A history of hip pain precluding weight bearing was taken as arthritis rather than arthralgia.

Careful note was made about the use of anti-inflammatory drugs. “Monoarthritis may be a presenting feature if there was a history of NSAID use early in the course of the illness” where monoarthritis was recorded but there was no evidence of use of anti-inflammatory usage this was not a major criterion. These definitions allowed a consistent approach to defining arthralgia, monoarthritis and polyarthritis.

Definition of carditis—The presence of a pansystolic murmur at the cardiac apex combined with echocardiographic mitral regurgitation was defined as carditis. The presence of a diastolic murmur at the upper left sternal border combined with echocardiographic aortic regurgitation was defined as carditis.

Echocardiographic mitral regurgitation (MR) or aortic regurgitation (AR) meeting agreed criteria as pathological MR or AR in the absence of a typical murmur was defined as subclinical carditis and used as a major criterion.

Importantly, when clinicians judged there was clinical MR or AR but subsequent echocardiography showed there was not, then the patient was defined as NOT having carditis. The presence of tricuspid or pulmonary regurgitation in the absence of left sided valve regurgitation was not regarded as carditis.

Evidence of recent Group A streptococcal infection—Evidence of a recent streptococcal infection by anti-streptococcal antibody titres is required for a definite case. A positive streptococcal throat culture in the absence of streptococcal serological support is allowed for a probable case.

American Heart Association definitions 1992 Jones Update criteria definitions—“Polyarthritis, the most frequent but benign major manifestation, is almost always migratory unless aborted by premature administration of anti-inflammatory mediation.”

We interpreted this statement to mean that monoarthritis is not accepted as a major criterion, reinforced by the subsequent 2002 statement of the American Heart Association. Carditis can be diagnosed as a major criterion only when there is clinically audible mitral regurgitation or aortic regurgitation. Indolent carditis is defined as ARF when the carditis is of insidious onset and of slow progression.

‘Probable ARF is defined by the American Heart Association as 1 major and 2 minor with the inclusion of a preceding group A streptococcal infection as a minor manifestation. Possible ARF is defined as strong clinical suspicion of ARF, but insufficient signs and symptoms to fulfil diagnosis of definite or probable ARF. These same definitions are used in New Zealand.

Recurrence of ARF—In a person with a known past ARF or RHD: 2 major or 1 major and 2 minor or 3 minor manifestations plus evidence of a recent GAS infection.

Rheumatic heart disease—Defined as structural mitral valve changes including, unequivocal marked valve thickening, subchordal restriction, prolapse of the valve tips or excessive mitral leaflet movements or restricted leaflet motion with mitral stenosis. Acute inflammatory markers, by definition, are normal in these patients.
Results

The files of 79 patients with a diagnosis of rheumatic fever were examined. These consisted of 54 of Pacific ethnicity, 23 Māori and 2 of unknown ethnicity. Six cases previously referred to the RF register had established RHD, three were recurrent ARF and four did not have ARF, leaving 66 cases identified as definite, probable or possible first occurrence of ARF fever.

Fifty-nine cases were definite ARF using the New Zealand guidelines criteria (Table 2) compared to 51 definite ARF using the 1992 Jones update. Overall, for this cohort of children with an acute illness considered as ARF, utilisation of the New Zealand guidelines resulted in 16% (CL 5–29%) more cases defined as definite ARF than using AHA 1992 Jones criteria.

Table 2. Diagnostic categories of 79 children with hospital discharge of ARF

<table>
<thead>
<tr>
<th>New Zealand guidelines criteria</th>
<th>AHA Jones 1992 Update</th>
</tr>
</thead>
<tbody>
<tr>
<td>59 Definite#</td>
<td>51 Definite</td>
</tr>
<tr>
<td>3 Probable</td>
<td>3 Probable</td>
</tr>
<tr>
<td>4 Possible</td>
<td>12 Possible</td>
</tr>
<tr>
<td>4 Not ARF *</td>
<td>4 Not ARF *</td>
</tr>
<tr>
<td>6 RHD</td>
<td>6 RHD</td>
</tr>
<tr>
<td>3 ARF recurrence</td>
<td>3 ARF recurrence</td>
</tr>
<tr>
<td>79 Total</td>
<td>79 Total</td>
</tr>
</tbody>
</table>

*It was recommended to the clinicians responsible for these 4 patients with an incorrect diagnosis of ARF that secondary BPG prophylaxis be discontinued

# 5 cases used subclinical carditis as a major criterion, 5 cases used monoarthritis as a major criterion (2 cases used subclinical carditis and monoarthritis).

Arthritis—59 of 66 (89%) patients had either arthritis or arthralgia at presentation. Of the 66 with a final diagnosis of ARF, arthritis was present in 41 cases and arthralgia in 18 cases.

Classical migratory polyarthritis of large joints was present in 32 (48%) Of these, two patients who had observed arthritis in one joint only but with an additional history of hip pain were defined as polyarthritis by the New Zealand guidelines.

Monoarthritis was found in nine cases, with seven of these having record of “early” anti-inflammatory usage (Table 3). These seven (11%) of the cohort were judged to be aborted migratory polyarthritis using the New Zealand guidelines as there was evidence of a recent streptococcal infection, they had minor criteria and no alternative cause of reactive arthritis was found. There was supporting cardiac features in five of these seven cases with clinical carditis (2 cases) and subclinical carditis (3 cases). Two of the seven had received NSAIDs prescribed by family doctors and five had been given them in hospital.

The influence of monoarthritis—The incidence of monoarthritis with anti-inflammatory usage was 11% (7/66). Two of these seven cases had clinical carditis so the influence of monoarthritis on the diagnosis to categorise as definite ARF was 5 of 66 or 8% (Table 3)
Table 3. Comparison of New Zealand Guidelines and American Heart Association Major Criteria for ARF

<table>
<thead>
<tr>
<th>Variables</th>
<th>New Zealand guidelines criteria</th>
<th>1992 Jones criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Migratory polyarthritis</td>
<td>32</td>
<td>30</td>
</tr>
<tr>
<td>History hip pain as 2nd joint</td>
<td>2</td>
<td>0 by definition</td>
</tr>
<tr>
<td>Monoarthritis + anti – inflammatory medication</td>
<td>7 *</td>
<td>0 by definition</td>
</tr>
<tr>
<td>Clinical carditis</td>
<td>36 †</td>
<td>36 †</td>
</tr>
<tr>
<td>Subclinical carditis</td>
<td>20 #</td>
<td>0 by definition</td>
</tr>
<tr>
<td>Chorea</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Erythema marginatum</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Subcutaneous nodules</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

* influence on diagnosis to change the case to definite ARF in 5 cases.
# influence on diagnosis to change the case to definite ARF in 5 cases.
† includes 3 cases of indolent carditis.

Carditis—The incidence of clinical carditis was 55% (36/66), subclinical carditis 30% (20/66) and no carditis 14% (10/66). The proportion of MR and AR are shown in Table 4.

Table 4. Numbers of cases showing MR and AR

<table>
<thead>
<tr>
<th>Variables</th>
<th>Clinical carditis n=36</th>
<th>Subclinical carditis n=20</th>
</tr>
</thead>
<tbody>
<tr>
<td>isolated MR</td>
<td>13</td>
<td>5</td>
</tr>
<tr>
<td>isolated AR</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>MR and AR</td>
<td>20</td>
<td>12</td>
</tr>
</tbody>
</table>

MR = mitral regurgitation; AR = aortic regurgitation.

The influence of subclinical carditis on the diagnosis of ARF—There was a 30% incidence of subclinical carditis. (Table 3 and 4) However there was another major criterion, polyarthritis (12 cases) or chorea (1 case) in 13 of these 20. In these cases the criteria for definite ARF was already met and the presence of subclinical carditis reinforced the diagnosis only.

Subclinical carditis was used as the major criterion in 7 cases who presented with monoarthritis (2 cases) or polyarthralgia (5 cases) influencing the category to definite ARF in 5 cases and to possible ARF in 2 cases. Thus the influence of subclinical carditis to become definite ARF was 5 of 59 or 8% of the cohort only. (Table3)

Cases wrongly diagnosed as ARF—Two cases had arthralgia, subclinical carditis, elevated acute phase reactants but no evidence of recent group A streptococcal infection. One case had arthralgia, no other minor or major criteria and borderline streptococcal serology.

The 4th case had met the definition of probable ARF but the arthritis was atypical not responding to aspirin. Serology was consistent with hepatitis B reactive arthritis. Thus, for the overall cohort of 79 patients, 5% were overdiagnosed as ARF.
Discussion

Utilisation of New Zealand guidelines for rheumatic fever\textsuperscript{12,15} resulted in 16\% more cases being diagnosed as definite ARF compared with the American Heart Association 1992 Jones update criteria.\textsuperscript{11}

This modest increase should allay fears that guidelines that have been developed in high prevalence regions to increase sensitivity for ARF will result in a large increase in case numbers. Nevertheless, the findings have implications for management as definite cases of ARF in New Zealand receive BPG secondary penicillin for 10 years or until aged 21 years.

In contrast, cases defined as possible ARF in New Zealand are either prescribed 5 years secondary prophylaxis or are followed without prophylaxis.

This study confirms that application of the New Zealand guidelines have increased the sensitivity for ARF. In the 1990s age-specific rates of ARF for Māori aged 5–14 were 41/100,000 and for Pacific children 84/100.00\textsuperscript{12} and these rates are rising.\textsuperscript{1,2}

Unfortunately, over many years in New Zealand we have seen problems with under diagnosis of ARF. For example several children with an acute illness presenting with monoarthritis but without culture growth after joint aspiration, returned later with severe rheumatic heart disease requiring cardiac surgery.

Conversely, not all Māori or Pacific children presenting with joint symptoms have ARF. Four children in this series appear to have been misdiagnosed by the hospital clinicians.

The sensitivity and specificity of any set of new criteria for ARF cannot be determined as there is no laboratory or objective test for ARF. The denominator of all patients in whom the diagnosis was considered during the 4 year period of the study could not be determined as only those patients that had RF (ARF or RHD) on the hospital discharge codes or those referred to the RF register were identifiable.

An Australian study\textsuperscript{17} found that utilisation of monoarthritis, subclinical carditis and low grade fever increased the diagnosis of ARF by 20\% compared to the AHA 1992 update\textsuperscript{11}. They reported that the diagnosis of ARF was influenced in 8 of 67 cases (12\%) by monoarthritis and 6 of 67 cases (9\%) by subclinical carditis,\textsuperscript{17} similar to our findings in New Zealand. Thus, the Australian guidelines have also resulted in increased sensitivity for ARF, justified in light of the extremely high rates in Aboriginal children.\textsuperscript{13}

Although the 1992 update\textsuperscript{11} did not accept echocardiography as the sole criterion for carditis, some aspects of the update do broaden sensitivity. Índolent carditis was described, and isolated chorea did not require confirmation of a recent group A streptococcal infection. Recurrences of ARF were addressed.

Arthritis—This study confirms that joint inflammation is the commonest presenting symptom\textsuperscript{12,30} and polyarthritis the commonest major criterion of ARF with nearly 90\% of the cohort having joint symptoms.

We accept a reliable history of arthritis without medical confirmation, and define history of hip pain precluding weight bearing as evidence of arthritis not arthralgia.
We found a high incidence of monoarthritis and it was concluded that 7 of 9 such patients had received “prematurely” anti-inflammatory drugs likely aborting evolution to polyarthritis. This judgement is made more confidently by clinicians experienced in the management of rheumatic fever.\textsuperscript{23}

These patients invariably had polyarthralgia as well as evidence of a recent GAS infection to support the aetiology as ARF. We suspect that some of the hospital clinicians who prescribed anti-inflammatory drugs before the diagnosis of ARF was certain were likely influenced by the concomitant presence of carditis, whether clinical or subclinical, but this is speculative in this retrospective study. However, it is important that the adage of not prescribing aspirin or other NSAIDs early in suspected ARF continues to be taught and followed.

Paracetamol or codeine can usually provide adequate pain relief for arthritis but will not stop the evolution to polyarthritis with its greater diagnostic certainty as a major criterion of ARF.

In New Zealand, over the counter NSAIDs such as ibuprofen are available from pharmacies and retail shops, and is a continued concern for both underdiagnosis as well as complicating the diagnosis of ARF.

The New Zealand guidelines for rheumatic fever\textsuperscript{12,15} stress the importance for paediatricians to search for alternative causes of arthritis when the presentation is atypical. Non-rheumatic causes of reactive arthritis including rubella, post rubella vaccination, parvovirus, hepatitis B, cytomegalovirus and Ebstein-Barr virus. Bacterial arthritis may occur with staphylococcal, Neisseria gonorrhoea, Yersinia and Mycoplasma infection.

Over many years we have observed all these infections in patients with a differential diagnosis of ARF. Indeed, in the current cohort one patient had reactive arthritis following hepatitis B infection and one patient with possible ARF with a GAS infection also had evidence of parvovirus infection. Some have observed monoarthritis without NSAID having been given and in Australia this is now accepted as a major criterion.\textsuperscript{13}

In the New Zealand setting monoarthritis without NSAID use has rarely been observed (2 cases in this series) and we have not used this as a major criterion of ARF. When children present to family doctors or to hospital with monoarthritis with a high fever the most important diagnosis to exclude initially is septic arthritis. Those with septic arthritis usually have significant toxicity and high fever.

In contrast, in our population and others,\textsuperscript{17,30} fever is not high in ARF. Finally it is important that there is unequivocal supporting evidence of a recent streptococcal infection when the monoarthritis and anti-inflammatory use is attributed to ARF. (Table 3)

Carditis—We are also confident to include subclinical carditis as a major criterion.\textsuperscript{12,15} Our experience with Doppler echocardiography in ARF over the past two decades\textsuperscript{14,23–27} has helped paediatricians determine whether arthritis symptoms in a child with raised streptococcal titres are more or less likely to represent ARF.

The present study showed a high incidence of clinical carditis (55\%) and subclinical carditis (30\%). The incidence of subclinical carditis in our region has been steady at
about 30% for a decade (R Nicholson, personal communication, 2010) but this is the first study from our group to show the influence of subclinical carditis as a major criterion on the diagnosis of ARF.

For patients presenting with polyarthritis as the major criterion, the finding of subclinical carditis helps support the diagnosis, is useful for counselling families, but does not influence the diagnosis. We found that in only in a third of those with subclinical carditis cases did this lead to a diagnosis of definite ARF.

Where echocardiography is available but the patient is from a non-endemic population for ARF, clinicians are less likely to be experienced assessing carditis both clinically and by echocardiography. Great care should be taken not to over diagnose what constitutes pathological mitral or aortic regurgitation.14

The implication for those with a diagnosis of definite or probable ARF in New Zealand is to commence long term secondary BPG prophylaxis for 10 years or until aged 21 years.17,28,29 Delivery of BPG is every 28 days and is highly effective to prevent recurrences of ARF when delivery is achieved using the registry to prescribe the penicillin, with delivery by a responsive well coordinated team of public or district health nurses. The actual penicillin failure rate is very low at 0.07 patient recurrences per 100 patient years.8

The actual recurrence rate is 1.4/100 patient years as some of the population are transient or non-compliant. Those patients with a diagnosis of possible ARF receive BPG for 5 years12 but the decision to commence prophylaxis is often influenced by family perception of the importance of the diagnosis.

Study limitations—The study was retrospective so some patient data may have been misinterpreted or not recorded.

Conclusions

In this New Zealand study 79 children who were part of an RCT to treat sore throats had been labelled as ARF at hospital discharge. Further inspection identified 66 of these as definite, probable of possible ARF first occurrences. Utilisation of the more liberal New Zealand guidelines for the diagnosis of ARF resulted in 16% (CL 5–29%) more definite ARF cases than using the 1992 Jones criteria.

Monoarthritis was the presenting symptom in 11% of cases and when this occurred in association with anti-inflammatory usage, it was accepted as a major criterion of ARF. The incidence of subclinical carditis was 30% but this influenced the diagnosis in only 8%. In high prevalence populations such as Māori and Pacific children in New Zealand, underdiagnosis of ARF is more of an issue than overdiagnosis.

This study shows how the broadened definitions of carditis and arthritis influenced diagnosis by increasing sensitivity in a population in whom ARF continues at an unacceptably high incidence.

Combined with a successful secondary penicillin prophylaxis programme, these guidelines will result in fewer recurrences of ARF and consequently help reduce the prevalence of severe RHD in Māori and Pacific populations.
Competing interests: None identified.

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Acknowledgements: We thank Elizabeth Wilson who reviewed the cases of monoarthritis and Ross Nicholson who provided a second review of echocardiograms with subclinical carditis.

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References:

Health consequences of tobacco use for Māori—cessation essential for reducing inequalities in health

Marewa Glover, Anette Kira, Nathan Cowie, Ron Wong, Jane Stephen, Kate Marriner

Abstract

Aim Tobacco use remains the largest preventable cause of death and disease in New Zealand. The aim of this paper was to identify all known health consequences of smoking, including exposure to other people’s smoke, focusing on Māori.

Method A review of the scientific literature, ‘grey’ literature, and, Government health data and reports.

Results Smoking has been causally linked with cardiovascular disease (CVD), many cancers, and several respiratory diseases, and, rates are higher for Māori than non-Māori. There are many consequences for smokers loved ones, including, pregnancy and birth complications, SUDI, and increased respiratory infections, cancers and CVD for children and adults. Māori have higher rates of still-birth and SUDI.

Conclusion This paper summarises all health consequences, to the smoker and their family. Supporting smoking cessation among Māori, particularly women and parents, may be one of the quickest pathways to health improvements for Māori.

Tobacco use remains the largest preventable cause of death and disease in many developed counties, including New Zealand (NZ). In 2006, the two main causes of death for New Zealanders (NZ) were cardiovascular disease (CVD) and cancer and both of those have been causally attributed to smoking.

Māori have higher rates than non-Māori of CVD and most cancers. Smoking plagues the smoker’s family from conception contributing to, increased risk of miscarriage, still-birth, and, ongoing respiratory problems. Exposure to secondhand smoke (SHS) is a major cause of early death and illness in non-smoking adults and children.

The consequences of smoking do not affect ethnic groups equally, due to much higher smoking prevalence among many indigenous populations, such as Aboriginal Australians and Māori than non-indigenous populations. In NZ, 45% of Māori smoke, nearly one in two Māori women and 40% of Māori men. Smoking prevalence is higher for Māori than non-Māori for adults, pregnant women and 14-15 year olds.

More Māori than non-Māori smoked at home and in cars and were exposed to SHS in the home. Māori children are at twice the risk of being exposed to SHS in the home.

Although there are many published papers on various health impacts of smoking, previous research has commonly focused on a particular subset of tobacco-related health consequences. The aim of this paper was to conduct a comprehensive review to
identify all known health consequences associated with smoked tobacco, for the smokers, in-utero fetuses, and those affected through SHS, with a focus on Māori.

The review presented in this paper was prepared for the Ministry of Health (MoH) to inform their advice to a Māori Affairs Committee conducting a parliamentary inquiry into the tobacco industry in Aotearoa (NZ) and the consequences of tobacco use for Māori.15

Method

The information in this paper was based on a review of the latest scientific literature on health topics, searches of ‘grey’ literature, and local Government health data and reports.

Key sources of evidence were online library databases: Medline, Scopus, PsychINFO and internet search engines. New Zealand specific evidence was sought from websites for the MoH, The Quit Group, Health Sponsorship Council, Action on Smoking Health, Cancer Society, Smokefree Coalition, Treasury, Customs, Health NZ, End Smoking NZ, and the Heart Foundation.

International evidence was sourced from Centers for Disease Control, The World Health Organisation, The International Agency for Research on Cancer, and websites of governmental and non-governmental organisations overseas. Given the large range of health consequences, it is not feasible to list all the search criteria. An example of search criteria used is:

To identify possible consequences, an initial search was conducted: TITLE((pregn* OR uter* OR "uter* OR gestation") and (smok* or tobacco or cigarette) and (risk or risk or adverse or hazard or hazards or consequence or consequences or threat or threats or danger)). Based on that search a range of topics and further publications were identified. For each identified topic a more thorough search was conducted, including investigating NZ/Māori context.

Results

Smoking as a cause or risk factor for mortality and morbidity—During 1996–1999 smokers had an estimated 3.9 to 7.4 years lower life expectancy than non-smokers.16 Life expectancy for the 2005–2007 period, was 70.4 years for Māori and 79 years for non-Māori,17 including both smokers and non-smokers. Smoking, which has been linked to many diseases, contributes to this lowered life expectancy.

Smoking is a known cause of CVD (Table 1a).3 Overall, Māori have higher rates of deaths and hospitalisations from CVD than non-Māori (Table 2), and heart failure mortality and hospitalisation rates are particularly high.4

Further, one of the largest differences between Māori and non-Māori age-standardised mortality rates was for hypertensive disease (age-standardised rate of 17.3 for Māori versus 3.7 for non-Māori).18

In 2000, 21% of total global cancer deaths were due to smoking.21 Tobacco smoking has been causally associated with at least 15 types of cancer.3 Compared to non-Māori, Māori have three times higher registration and mortality rates for lung, liver and stomach cancer, nearly double the rates for kidney, pancreatic and cervical cancer, and, higher rates of leukaemia and oesophageal cancer.5

A large number of respiratory diseases have been causally linked to smoking (Table 1c).3 Māori have higher risk of dying from respiratory diseases and have substantially higher rates of hospitalisation and death from COPD, asthma, and bronchiectasis than non-Māori (Table 3).4
Table 1. Consequences for smoker

<table>
<thead>
<tr>
<th>Smoking is causally associated with</th>
<th>Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Cardiovascular diseases (CVD)</td>
<td></td>
</tr>
<tr>
<td>Abdominal aortic aneurysm,</td>
<td>3</td>
</tr>
<tr>
<td>Atherosclerosis/ischaemic heart disease,</td>
<td>3</td>
</tr>
<tr>
<td>Cerebrovascular disease (stroke), and</td>
<td>3</td>
</tr>
<tr>
<td>Coronary heart disease (heart failure)</td>
<td>3</td>
</tr>
<tr>
<td>(b) Cancer</td>
<td></td>
</tr>
<tr>
<td>Lung</td>
<td>3</td>
</tr>
<tr>
<td>Stomach</td>
<td>19</td>
</tr>
<tr>
<td>Liver</td>
<td>19</td>
</tr>
<tr>
<td>Pancreas</td>
<td>19, 20</td>
</tr>
<tr>
<td>Leukaemia</td>
<td>3</td>
</tr>
<tr>
<td>Oesophagus</td>
<td>3</td>
</tr>
<tr>
<td>Cervix</td>
<td>3</td>
</tr>
<tr>
<td>Kidney</td>
<td>3</td>
</tr>
<tr>
<td>Oral</td>
<td>3, 19</td>
</tr>
<tr>
<td>Bladder</td>
<td>19</td>
</tr>
<tr>
<td>Larynx</td>
<td>19</td>
</tr>
<tr>
<td>Oro- and hypopharyngeal</td>
<td>19</td>
</tr>
<tr>
<td>(c) Respiratory disease</td>
<td></td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease (COPD)</td>
<td>3</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>3</td>
</tr>
<tr>
<td>Premature onset of and acceleration of age-related decline in lung</td>
<td>3</td>
</tr>
<tr>
<td>function</td>
<td>3</td>
</tr>
<tr>
<td>Asthma</td>
<td>3</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Incidence of CVD deaths (2000–2004) and public hospitalisations (2003–2005) for Māori and non-Māori (per 100,000)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Māori</th>
<th>Non-Māori</th>
<th>Rate ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>All CVD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>139.8</td>
<td>61.2</td>
<td>2.29</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>1,119.9</td>
<td>643.2</td>
<td>1.74</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>80.0</td>
<td>35.5</td>
<td>2.25</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>342.5</td>
<td>239.0</td>
<td>1.43</td>
</tr>
<tr>
<td>Heart failure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>2.8</td>
<td>1.2</td>
<td>2.27</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>182.7</td>
<td>39.4</td>
<td>4.64</td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>21.7</td>
<td>13.5</td>
<td>1.61</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>116.1</td>
<td>63.3</td>
<td>1.84</td>
</tr>
</tbody>
</table>

Source: 4

In 2000, 21% of total global cancer deaths were due to smoking. 21 Tobacco smoking has been causally associated with at least 15 types of cancer. 3 Compared to non-Māori, Māori have three times higher registration and mortality rates for lung, liver and stomach cancer, nearly double the rates for kidney, pancreatic and cervical cancer, and, higher rates of leukaemia and oesophageal cancer. 5
A large number of respiratory diseases have been causally linked to smoking (Table 1c). Māori have higher risk of dying from respiratory diseases and have substantially higher rates of hospitalisation and death from COPD, asthma, and bronchiectasis than non-Māori (Table 3).

Table 3. Respiratory disease deaths and public hospitalisations for Māori and non-Māori (per 100,000)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Māori</th>
<th>Non-Māori</th>
<th>Rate ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combined respiratory disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>33.8</td>
<td>13.1</td>
<td>RR 2.59</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>2249.8</td>
<td>1367.2</td>
<td>RR 1.65</td>
</tr>
<tr>
<td>COPD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>24.7</td>
<td>9.3</td>
<td>RR 2.65</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>261.1</td>
<td>73.7</td>
<td>RR 3.54</td>
</tr>
<tr>
<td>Asthma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>2.6</td>
<td>0.6</td>
<td>RR 4.10</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>457.3</td>
<td>245.2</td>
<td>RR 1.86</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>2.1</td>
<td>0.3</td>
<td>RR 6.70</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>41.7</td>
<td>11.6</td>
<td>RR 3.60</td>
</tr>
<tr>
<td>Pneumonia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>3.3</td>
<td>1.86</td>
<td>RR 1.86</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>366.5</td>
<td>222</td>
<td>RR 1.65</td>
</tr>
</tbody>
</table>

Source: Robson & Harris

Consequences of smoking in pregnancy—There is very little research into consequences of smoking while pregnant for Māori. However, the ill health effects are of particular relevance for Māori. Smoking prevalence for Māori while pregnant remains extraordinarily high, with 43% smoking at first registration with a maternity carer and 34% still smoking when discharged.

Smoking tobacco is associated with many adverse pregnancy outcomes (Table 4a), and increases the risk of negative outcomes for the unborn child (Table 4b). The ill-effect of smoking during pregnancy has ongoing effects on the newborn (Table 4c). Māori neonatal death is significantly more common than for NZ European infants and Māori have almost six times the rate of sudden unexpected death of an infant (SUDI) than non-Māori. Having been exposed to smoking in-utero has ongoing effects in to childhood and beyond (Table 4d).

In addition to physical consequences, children who had been exposed to smoking in-utero have higher symptom rates for many behavioural problems (Table 4e).
Table 4. Consequences of smoking during pregnancy

<table>
<thead>
<tr>
<th>Increased risk of adverse…</th>
<th>Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>(a) Pregnancy outcomes</em></td>
<td></td>
</tr>
<tr>
<td>Abnormal implantation of the placenta</td>
<td>24</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>25</td>
</tr>
<tr>
<td>Miscarriage</td>
<td>6</td>
</tr>
<tr>
<td>Placental lining separating from the uterus</td>
<td>24, 26</td>
</tr>
<tr>
<td>caesarean sections</td>
<td>27</td>
</tr>
<tr>
<td><em>(b) Outcomes for unborn child</em></td>
<td></td>
</tr>
<tr>
<td>Preterm delivery</td>
<td>28, 29</td>
</tr>
<tr>
<td>Small-for-gestational age (SGA)</td>
<td>30–32</td>
</tr>
<tr>
<td>Stillbirth</td>
<td>33, 34</td>
</tr>
<tr>
<td><em>(c) Ongoing outcomes for newborn child</em></td>
<td></td>
</tr>
<tr>
<td>Infant mortality</td>
<td>22, 23</td>
</tr>
<tr>
<td>SUDI</td>
<td>37, 38</td>
</tr>
<tr>
<td><em>(d) Ongoing outcomes during childhood and adulthood</em></td>
<td></td>
</tr>
<tr>
<td>Chromosome damage</td>
<td>39</td>
</tr>
<tr>
<td>Increases in abnormal chromosome changes in newborns</td>
<td>39</td>
</tr>
<tr>
<td>Respiratory response to low oxygen levels in the bloodstream</td>
<td>40, 41</td>
</tr>
<tr>
<td>Congenital heart defects</td>
<td>42, 43</td>
</tr>
<tr>
<td>Conotruncal heart defects (outflow tract defects)</td>
<td>44</td>
</tr>
<tr>
<td>Elevated blood pressure in neonates and childhood</td>
<td>45, 46</td>
</tr>
<tr>
<td>Cleft lip/palate</td>
<td>47</td>
</tr>
<tr>
<td>Respiratory infection</td>
<td>48</td>
</tr>
<tr>
<td>Chronic bronchitis, wheezing, lower respiratory tract illness</td>
<td>49, 50</td>
</tr>
<tr>
<td>Any asthma</td>
<td>8</td>
</tr>
<tr>
<td>Ear infection (after adjusting for other risk factors)</td>
<td>51</td>
</tr>
<tr>
<td>Ear infections, sub-acute ear infections</td>
<td>52</td>
</tr>
<tr>
<td>Previous ear surgery</td>
<td>52</td>
</tr>
<tr>
<td><em>(e) Behavioural problems</em></td>
<td></td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>53</td>
</tr>
<tr>
<td>Depression</td>
<td>53</td>
</tr>
<tr>
<td>Alcohol abuse</td>
<td>53</td>
</tr>
<tr>
<td>Substance abuse</td>
<td>53</td>
</tr>
<tr>
<td>Externalising behaviour problems</td>
<td>54</td>
</tr>
<tr>
<td>Attention deficit and ADHD</td>
<td>55–57</td>
</tr>
<tr>
<td>Learning disabilities and lower academic achievement</td>
<td>58</td>
</tr>
<tr>
<td>Increased criminal offending in adulthood</td>
<td>59</td>
</tr>
<tr>
<td>Increased risk of child experimentation with and uptake of smoking</td>
<td>60</td>
</tr>
</tbody>
</table>

**Consequences of exposure to secondhand smoke**—In addition to the detrimental consequences to the smoker and smoker’s fetus, smoking also has negative consequences for those living around the smoker. Māori are more likely than non-Māori to be exposed to smoking in the home, less likely to have a household smoking ban, are exposed for longer hours and are more likely to be exposed to smoking in cars.

Non-smoking pregnant women exposed to SHS had an increased risk of adverse pregnancy outcomes and well as adverse outcomes for the fetus and child (Table 5a). Children exposed to SHS are at higher risk for many ill-health consequences. As mentioned earlier, smoking during pregnancy increases the risk of SUDI. However, there is also a relationship between SUDI and postnatal exposure to smoking.
Furthermore, childhood exposure to SHS has been associated with a number of adverse conditions (Table 5b). A recent study found lower iron levels in the cordal blood of Māori neonates compared to non-Māori, which may contribute to higher rates of anaemia in Māori infants.\textsuperscript{68}

Exposure to SHS is a major cause of early mortality and illness in non-smoking adults.\textsuperscript{69,70} Non-smokers have health problems arising from SHS (Table 5c).

Table 5. Consequences of exposure to secondhand smoke

<table>
<thead>
<tr>
<th>Secondhand smoke exposure during…</th>
<th>Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Pregnancy</td>
<td></td>
</tr>
<tr>
<td>Increases risk of miscarriage and stillbirth</td>
<td>71–73</td>
</tr>
<tr>
<td>Increases risk of delivering a SGA infant</td>
<td>71</td>
</tr>
<tr>
<td>Increases risk of asthma and allergy-related symptoms in preschoolers</td>
<td>74</td>
</tr>
<tr>
<td>(b) Childhood</td>
<td></td>
</tr>
<tr>
<td>Increases risk of SUDI</td>
<td>65–67</td>
</tr>
<tr>
<td>Increases risk of respiratory diseases</td>
<td>50, 75–77</td>
</tr>
<tr>
<td>Negative impact on lung function</td>
<td>78–80</td>
</tr>
<tr>
<td>Increases prevalence of lower respiratory tract infections</td>
<td>81</td>
</tr>
<tr>
<td>Increases prevalence of pneumonia</td>
<td>82</td>
</tr>
<tr>
<td>Increases prevalence of asthma</td>
<td>50, 83, 84</td>
</tr>
<tr>
<td>Increases prevalence of pulmonary tuberculosis</td>
<td>85, 86</td>
</tr>
<tr>
<td>Increases incidence of poor heart health</td>
<td>87, 88</td>
</tr>
<tr>
<td>Is associated with aortic stiffness</td>
<td>89</td>
</tr>
<tr>
<td>Increases incidences of adult emphysema</td>
<td>88</td>
</tr>
<tr>
<td>Is related to increasing prevalence of allergic diseases in children</td>
<td>90, 91</td>
</tr>
<tr>
<td>Is linked to middle ear infections and ear drum abnormalities</td>
<td>92–94</td>
</tr>
<tr>
<td>Is linked to low HDL cholesterol levels</td>
<td>95–97</td>
</tr>
<tr>
<td>Is linked to anaemia</td>
<td>98</td>
</tr>
<tr>
<td>Increases risk of pre-adolescent smoking experimentation and current smoking</td>
<td>99</td>
</tr>
<tr>
<td>(c) Adulthood, increased risk of…</td>
<td></td>
</tr>
<tr>
<td>Respiratory problems</td>
<td>70</td>
</tr>
<tr>
<td>Lung cancer\textsuperscript{70}</td>
<td>70</td>
</tr>
<tr>
<td>CVD, stroke and heart attack</td>
<td>9</td>
</tr>
</tbody>
</table>

Discussion

This review paints a tragic picture for Māori health; the consequences of smoking for Māori are extensive across all age groups. We have identified a disturbingly long list of health consequences for the smoker and their loved ones, including unborn babies, children and other adults.

Because of the lack of available data specific to Māori, it is likely that other factors other than smoking, such as poverty or obesity, also contribute to the high prevalence of negative health outcomes for Māori. However, even if smoking isn’t the only contributor, the weight of evidence of the harmful consequences of tobacco smoking on Māori is established.

There is no doubt that reducing smoking—a modifiable behaviour—will result in a lower prevalence of early mortality and morbidity for Māori. Policy and research now
needs to move on to identifying and testing effective interventions that will most rapidly reduce smoking and reverse this tobacco-related harm.

The Māori Affairs parliamentary inquiry expressed a deep concern for the devastating consequences of the high smoking prevalence among Māori women, particularly while pregnant. Their recommendations predominantly proposed broad public health policy solutions most of which were not Māori specific. The Government agreed to the goal to move New Zealand towards becoming a smokefree nation by 2025.

In terms of reducing Māori smoking specifically, the Government response agreed that Māori will remain a priority group and repeatedly said that “the Government will continue to” do what it already does to ensure that the current media campaigns or cessation programmes effectively reach and work for Māori. However, the smoking prevalence of Māori women has not significantly declined in the past 6 years, demonstrating the insufficiency of current campaigns. We urgently need innovative interventions specifically designed for Māori, that resonate with Māori, and that will reach Māori.

Some of our other research has revealed that Māori knowledge of the full range of ill-effects of smoking in pregnancy is limited, and whilst many Māori may know about the Quitline, nicotine patches and gum, there is poor knowledge of the wider range of effective cessation products and that many are cheaply available. Māori have higher participation rates in organised competition and events than other ethnic groups, for example, Māori cultural contests such as kapa haka are well-attended and popular.

Quit and Win type interventions that incorporate competitiveness and encourage community participation may be particularly attractive to Māori people who smoke.

Rongoā Māori (traditional Māori healing) approaches to supporting smoking cessation could also be utilised to trigger widespread quitting among Māori. Pharmacotherapies for cessation, such as Bupropion have been shown to be effective for Māori. Similarly, Cytisine (a plant extract found in Golden Rain [Cytisus laburnum L] and the New Zealand Kowhai [Sophora tetrapetra L.] has a similar molecular make-up to nicotine, has been used successfully as a cessation product in central and eastern Europe and central Asia for many years, and is low priced. A recent study with Māori found that cytisine would likely be more attractive to Māori than currently available cessation products if presented as a rongoā Māori. A trial to test the efficacy and safety of cytisine is currently underway in New Zealand.

Technology based interventions, for example a mobile-phone based cessation programme, have also shown promise with Māori.

A tobacco control programme designed to reduce smoking consumption and prevalence across the whole population (that is, for the numeric majority of smokers) will not have equitable effects for Māori. The introduction of tobacco to Aotearoa has resulted in a unique epidemic that is only shared by a few other colonised Indigenous populations who also have half of their mothers smoking, for example, Australian Aboriginal and Torres Straight Island women.
The majority of the world’s women do not smoke! That means the majority of the world’s children are not exposed to the same level of smoke as many Māori children are. This paper shows the spectre that the rest of the developing world will one day face if they do not look at what the tobacco industry has achieved with Māori. We are in the unique position, if we could but gain support to design and test interventions that reduce Māori maternal smoking, to inform the global tobacco control programme hopefully before other countries begin to experience the same level of smoking-related harm.

Limitations—One of the limitations of this study is that due to the limited number of studies focusing on Māori, it is difficult to discern how much of the higher prevalence of health consequences for Māori is attributable to smoking. However, given the many consequences of smoking have been defined as causal, and given the very high smoking prevalence among Māori, it is highly likely that smoking substantially contributes to the poorer health outcomes for Māori than non-Māori.

Competing interests: None identified.

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Improving health, safety and energy efficiency in New Zealand through measuring and applying basic housing standards

Julie Gillespie-Bennett, Michael Keall, Philippa Howden-Chapman, Michael G Baker

Abstract

Substandard housing is a problem in New Zealand. Historically there has been little recognition of the important aspects of housing quality that affect people’s health and safety. In this viewpoint article we outline the importance of assessing these factors as an essential step to improving the health and safety of New Zealanders and household energy efficiency. A practical risk assessment tool adapted to New Zealand conditions, the Healthy Housing Index (HHI), measures the physical characteristics of houses that affect the health and safety of the occupants. This instrument is also the only tool that has been validated against health and safety outcomes and reported in the international peer-reviewed literature. The HHI provides a framework on which a housing warrant of fitness (WOF) can be based. The HHI inspection takes about one hour to conduct and is performed by a trained building inspector.

To maximise the effectiveness of this housing quality assessment we envisage the output having two parts. The first would be a pass/fail WOF assessment showing whether or not the house meets basic health, safety and energy efficiency standards. The second component would rate each main assessment area (health, safety and energy efficiency), potentially on a five-point scale. This WOF system would establish a good minimum standard for rental accommodation as well encouraging improved housing performance over time. In this article we argue that the HHI is an important, validated, housing assessment tool that will improve housing quality, leading to better health of the occupants, reduced home injuries, and greater energy efficiency. If required, this tool could be extended to also cover resilience to natural hazards, broader aspects of sustainability, and the suitability of the dwelling for occupants with particular needs.

Imagine a country where all the houses are warm, dry, energy efficient, safe, sustainable, and meet the needs of the occupants who live in them. Also imagine that advertisements for houses to rent or buy don’t just have icons for the number of bathrooms, bedrooms and garages but also communicate vital information about the health, safety, energy efficiency, and earthquake resilience of housing and its suitability for different groups of people including families with children, the elderly and people with disabilities.

We argue that New Zealand desperately needs a system for rating the quality of its houses. Such a system should at the very least define a minimum standard (a warrant of fitness) and provide well validated quality rating on a number of important scales to encourage improved housing performance above this minimum level. While there are costs of implementing such a system, the costs of inaction are considerably greater, and are already being borne on a day-to-day basis by many individuals and whānau (extended family).
Housing quality in New Zealand

Housing is one of the key material determinants of health and shelter is a fundamental human need. New Zealand has substandard housing, which has resulted from poor regulation of minimum housing standards and lack of maintenance. This situation has been compounded by an increasing reliance on market solutions for affordable housing and an emphasis on housing as an investment supported by imported capital and tax-breaks for landlords.

The result has been an inequitable distribution of housing assets as well as a rise in housing costs, regardless of housing quality. In addition, there are growing health inequalities between different groups in society, with negative health impacts particularly on Māori, Pacifica and low-income households.

Two-thirds of New Zealand houses have timber frames, iron roofs and are on concrete or wooden piles, and the majority of homes have single-glazed windows. One-third of homes are un-insulated and many households are inadequately heated and are colder than recommended by the World Health Organization.

In comparison to other developed countries, New Zealand households use less energy for home heating, although the percentage of the household budget spent on energy is similar.

The public health and economic consequences of poor quality housing

People in the developed world spend around 90% of their lives indoors and as most of this is spent in the home, the quality of housing affects the health and safety of the population. There are many consequences of poor housing. However, the most important in developed temperate countries such as New Zealand are probably negative effects on respiratory health and injury. Housing quality also affects how much energy is required to heat the house.

Poor quality housing and respiratory health—The link between cold, damp, mouldy housing and poor health has been highlighted in several international and New Zealand studies. Houses that are cold are also likely to be damp, which leads to the growth of moulds that can increase respiratory symptoms.

Cold, damp substandard housing changes the way in which the occupants live within a house. New Zealanders tend to crowd together in one room to stay warm, which promotes the transmission of viral infections. It has been suggested that respiratory viral infections may influence asthma frequency, with deficient immune responses to viral infections not limited to children with atopic asthma, but also present in atopic children without asthma. Furthermore, data from longitudinal studies suggest that wheezing episodes associated with viral infections early in life are a major risk factor for the development of asthma later in life.

Indoor air pollutants, such as nitrogen dioxide, produced by unflued gas heaters have also been shown to increase respiratory symptoms. Almost one-third of New Zealand households (386,000 houses) use portable unflued gas heaters to heat their homes. The use of indoor, unflued gas heating occurs disproportionately in lower socioeconomic groups, with single parent and Māori families over-represented. One of the most common chronic respiratory diseases of childhood and adolescence is asthma, which carries with it a significant impact on daily activities, including school attendance.
In New Zealand, asthma rates are among the highest in the world, with more than one-quarter of children and one-in-six adults suffering from asthma. As well as the stress associated with having a chronic illness, asthma leads to higher health service utilisation and pharmaceutical costs. The economic burden of asthma in New Zealand has been conservatively estimated at over $800 million per year.

Apart from asthma, new cases of other respiratory disorders, such as bronchiectasis, are rapidly increasing and compared to New Zealand’s neighbouring Pacific countries we have higher rates of admission for diseases such as pneumonia. New Zealand has a high incidence of preventable respiratory disease in comparison to other OECD countries.

There is good evidence to link poor quality housing and increased respiratory symptoms. A study by Keall and colleagues reported that in a sample of about 1,000 houses, people who were classified as living in the poorest quality houses, would have a 33% reduction in respiratory symptoms (relative risk 0.67 with 95% CI 0.53 to 0.85) if these people lived in the best performing houses. One way to reduce New Zealand’s burden of respiratory symptoms is to improve the quality of housing.

**Poor quality housing and injury**—Poor quality housing increases injury rates. Globally around one-third of injuries occur in the home. In New Zealand during the 2007/2008 year long period there were 715,218 claims to the Accident Compensation Corporation (ACC) as a result of unintentional injuries in the home.

Approximately 36,000 people who suffer from an injury in the home are hospitalised each year, which makes home the most common location for injuries resulting in hospitalisation. Excluding self-harm, assaults and transport accidents New Zealand has approximately 250 injury deaths a year at home.

In 2010 more than 261,000 New Zealanders were injured as a result of everyday slips, trips and falls in their homes. Collectively, the social cost of these injuries and deaths have been estimated to be around $13 billion annually (at 2008 costs), 3½ times more than are associated with road injuries, making the average social costs per home injury approximately $26,000.

One way to improve the burden of these injuries and their associated costs is to reduce injury hazards in the home. The findings of an economic analysis conducted on about 1000 houses in Taranaki indicated that, for an average of around $500, most householders can fix certain injury hazards in their homes and reduce their injury risk. For each additional home injury hazard enumerated, there was a significant associated increase in the odds ratio of a home injury of 22%.

**Poor quality housing and energy efficiency**—Poor quality housing can increase energy consumption. Excessive use of energy contributes to CO₂ emissions and climate change.

In 2003, the United States residential sector produced more than 20% of total United States energy-related CO₂ emissions. In New Zealand, houses account for approximately 13% of the country’s total energy use, with 34% of energy used on space heating and 29% used on water heating. Annually on average each New Zealand house spends around $2000 on energy and produces 2 tonnes of CO₂.

In Britain, a study of private rental housing found that retrofitting insulation was effective in terms of modelled reductions in fuel use. An Irish study modelled the
returns on domestic energy conservation opportunities and concluded that a home retrofitting programme would result in a 3:1 benefit cost ratio including energy savings, health benefits and reduction in avoidable mortality.38

In New Zealand the Housing, Insulation and Health Study showed that insulating 1350 houses, built before insulation was required, improved the occupants' health and well-being as well as household energy efficiency.39 A cost-benefit analysis of this study concluded that valuing the health gains, energy and savings on CO₂ emissions suggests that the total benefits are 1½ to 2 times the magnitude of the cost of retrofitting insulation.40

An evaluation of the roll-out of the Warm Up New Zealand: Heat Smart programme found the benefits exceeded the costs by 5-to-1; the most significant contribution came from reduced mortality in older people.41

Improving energy efficiency is one of the most constructive and cost–effective ways to address the challenges of high energy prices, lack of energy security, air pollution, and global climate change. Most importantly, improving the energy efficiency of older housing means houses are warm and comfortable to live in and the health of the occupants is improved.42

Poor quality housing and inequalities—Māori and Pacific children are over-represented in low-income families and more than half of the children living in poverty are Māori or Pacific.43 These children live disproportionately in substandard housing, which means they suffer a greater health burden as they are exposed to additional housing hazards.

Hand-in-hand with living in substandard housing, people who have lower incomes are struggling to heat their houses given it is very expensive to heat an un-insulated, damp house to a comfortable, healthy temperature. It is estimated that between 10 and 14% of New Zealand houses or some 400,000 people live in fuel poverty nationwide.42

How the Healthy Housing Index works

In New Zealand the residential housing stock is largely built, so improving the quality of the existing built environment would benefit from a diagnostic tool that identifies deficiencies in the home environment that can be addressed by retrofitted improvements.44

The Healthy Housing Index (HHI) was developed by the Housing and Health Research Programme/He Kainga Oranga and the Building Research Association of New Zealand (BRANZ) and is the only outcome-validated housing quality assessment tool available internationally. The HHI is an independent, science-based, practical tool, which translates knowledge gained from health and building science research and practical experience during a 10-year period into an ongoing system for informing housing improvement.

The assessment is based on an inspection conducted by a trained building professional. The inspection takes about an hour and involves both observation and measurement of the house. Results are recorded on a tablet computer with data from the inspection downloaded once there is an internet connection.

The assessment has two outputs. The first is a pass/fail Warrant of Fitness type of assessment as to whether the home meets basic health and safety standards. The second
output rates each major assessment area (health, safety and energy efficiency) potentially on a five-point scale (the exact form of this scale is still being refined).

In addition, a report is provided to the home-owner, which lists identified problems along with prioritised solutions and remediation options to improve the health, safety and energy efficiency of the house. Figure 1 is an example of how the HHI summary assessment could look.

Figure 1. An example of how the HHI housing assessment certificate could look

There are some operational aspects of the HHI that are still being developed. For example the exact standards required to ‘pass’ the warrant of fitness are being refined in consultation with councils and the New Zealand Green Building Council and the role of the additional scales is currently being discussed with key agencies.

An expanded workforce of inspectors needs to be trained and quality control measures put in place. There are also important details about how the information generated would be held and communicated, for example, whether it will be made available online to owners, renters and potential purchasers of properties.

It is envisaged that the HHI will be implemented in a phased manner, starting with rental housing, and initially focusing on health, safety, and energy efficiency. There are opportunities provided by new housing construction in Christchurch to provide a common metric as represented by the HHI assessment to guide design and construction quality standards.

It is expected that inspections would be carried out a maximum of once every 5 years or when a house is rented or sold. Table 1 gives examples of components assessed during a HHI inspection and indicates what components could be required to pass an inspection. It also gives examples of additional components that may be required to gain extra points on the proposed five-point scales.
It is important to note that the HHI and WOF cannot, and should not, cover every important aspect of housing relevant to its condition and impact on the health and safety of the occupants.

Firstly, even a high-quality house can be used in unhealthy and unsafe ways. A key example is household crowding, which is a function of the composition of the household in relation to the size of the house. Household crowding is a serious problem in New Zealand and is almost certainly contributing to our high rates of serious infectious diseases. However, its solution depends on increasing the supply of affordable housing so there is less pressure on low-income people to live in crowded conditions.

Secondly, the HHI and WOF cannot fully cover house conditions and maintenance, except for selective aspects that are relevant to health, safety, and energy efficiency. By analogy with motor vehicles, there is a WOF relating to the safety of the car, but in addition there is a driver’s license relating to the skills and behavior of the driver, and various vehicle inspection services covering vehicle maintenance and condition.

As noted in Table 1, some of these aspects of housing may be included in a ‘suitability statement’ associated with the house WOF, but this element requires further consideration.

### Table 1. Examples of components assessed by the Healthy Housing Index

<table>
<thead>
<tr>
<th>Name of scale</th>
<th>Outcome</th>
<th>Examples of components required to pass the WOF based on the HHI</th>
<th>Examples of additional components to gain extra points on a proposed 5-point scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Core modules</td>
<td>Health</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Respiratory diseases</td>
<td>Ceiling and under-floor insulation where possible, installed to EECA standards</td>
<td>Excess heat and cold control</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular diseases</td>
<td>Sanitary areas to include a toilet and personal washing facilities</td>
<td>Control of all indoor pollutants</td>
</tr>
<tr>
<td></td>
<td>Infectious diseases linked to enteric transmission</td>
<td>Storm-water and waste-water drainage to Council Standards</td>
<td>Sound proofing for noise</td>
</tr>
<tr>
<td></td>
<td>Adequate food preparation and storage areas, including an operational stove and oven</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Mechanical ventilation in kitchens and bathrooms</td>
</tr>
<tr>
<td></td>
<td>Adequate temperature control</td>
<td>Adequate food preparation and storage areas, including an operational stove and oven</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
</tr>
<tr>
<td></td>
<td>Adequate ventilation</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Adequate temperature control</td>
</tr>
<tr>
<td></td>
<td>Adequate cooking and food storage</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Adequate temperature control</td>
</tr>
<tr>
<td></td>
<td>Adequate heating</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Adequate temperature control</td>
</tr>
<tr>
<td></td>
<td>Adequate cooling</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Adequate temperature control</td>
</tr>
<tr>
<td></td>
<td>Adequate lighting</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Adequate temperature control</td>
</tr>
<tr>
<td></td>
<td>Adequate water heating</td>
<td>Adequate control of mould and dampness; including dry underfloor/vapour barrier and reduced moisture sources</td>
<td>Adequate temperature control</td>
</tr>
<tr>
<td></td>
<td>Safety</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Injuries and poisonings</td>
<td>Working fire alarms</td>
<td>High standards of driveway &amp; boundary fencing</td>
</tr>
<tr>
<td></td>
<td>Adequate electrical and lighting safety</td>
<td>All power outlets/ light switches safe, functional and in a good state of repair</td>
<td>High security from intruders</td>
</tr>
<tr>
<td></td>
<td>Structural soundness of house</td>
<td>Adequate electrical and lighting safety</td>
<td>Fire resistant materials &amp; design</td>
</tr>
<tr>
<td></td>
<td>Safe access to house</td>
<td>Structural soundness of house</td>
<td>High standards of fall protection</td>
</tr>
<tr>
<td></td>
<td>Adequate lighting</td>
<td>Safe access to house</td>
<td>High standards of external lighting</td>
</tr>
<tr>
<td></td>
<td>Water heating set to safe temperature</td>
<td>Adequate electrical and lighting safety</td>
<td>High standards of noise insulation</td>
</tr>
<tr>
<td></td>
<td>Ranch-sliders and low level windows to have safety visibility strips</td>
<td>Water heating set to safe temperature</td>
<td>Fire extinguishers</td>
</tr>
<tr>
<td></td>
<td>Secure storage for potential poisons</td>
<td>Ranch-sliders and low level windows to have safety visibility strips</td>
<td>Fire extinguishers</td>
</tr>
<tr>
<td></td>
<td>Basic security from intruders (locking doors/windows)</td>
<td>Secure storage for potential poisons</td>
<td>Fire extinguishers</td>
</tr>
<tr>
<td>Energy</td>
<td>efficiency</td>
<td>Financial and environmental costs</td>
<td>Energy efficient lighting</td>
</tr>
<tr>
<td></td>
<td>Ceiling and under-floor insulation where possible, installed to EECA standards</td>
<td>Financial and environmental costs</td>
<td>Sustainable heating</td>
</tr>
<tr>
<td></td>
<td>Safe and energy efficient heating</td>
<td>Financial and environmental costs</td>
<td>Sustainable heating</td>
</tr>
</tbody>
</table>
### Benefits of the Healthy Housing Index

Assessing house quality has two broad functions: first to assist house owners, renters, property managers and compliance agencies in making informed judgements about the management of individual properties; and second to provide a robust basis for policy development, compliance monitoring and research regarding the quality of housing stock.44

Adequate housing quality data in New Zealand are almost entirely absent to support decision making by local authorities and national policy agencies with responsibility for health, safety and housing quality. Quotable Value records contain basic information and some data are available from the BRANZ House Condition Survey. But there is a lack of more comprehensive quality data on individual residential properties.

The HHI focuses on building condition rather than the occupants or the way they might live within a house. The HHI provides a measure of how likely it is that occupants will suffer ill health or injuries due to a housing factor(s). Each measure included in the inspection was selected on the basis of previous local and international research, together with expert opinion. In addition each individual element has been validated against respiratory health outcomes28 and injury claims to ACC.34

### Alternative quality rating schemes for housing

There are other home rating tools available in New Zealand, but their scope is more limited, focusing mainly on the energy efficiency of the building. Several organisations that provide home rating tools and information on building maintenance, energy efficiency, and renovations are listed in Appendix 1 at the end of this paper.

The Energy Efficiency Conservation Authority (EECA) states that there is a lack of public information on energy efficiency and this is a major barrier to the uptake of insulation and efficient heating systems.46 It is also worth noting that the involvement of the building industry in supporting a housing quality measure does present issues with

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### Table: Examples of components required to pass the WOF based on the HHI

<table>
<thead>
<tr>
<th>Name of scale</th>
<th>Outcome</th>
<th>Examples of components required to pass the WOF based on the HHI</th>
<th>Examples of additional components to gain extra points on a proposed 5-point scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resilience</td>
<td>Injuries from natural hazards</td>
<td>Yet to be developed</td>
<td>Solar orientation, Thermal mass, Optimal house layout, Heating and cooling systems, Solar water heating, Window efficiency/double glazing</td>
</tr>
<tr>
<td>Sustainability</td>
<td>Financial costs, Environmental costs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suitability</td>
<td>Infectious diseases linked to household crowding</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*The suitability module is proposed to provide useful information for occupants on aspects such as the number of rooms (and therefore number of occupants the house can accommodate before it becomes crowded), disability issues (such as wheelchair access), and suitability for children, but it will not form part of the rating system.
conflicts of interest and minimises the focus on the effect of the indoor environment on health and wellbeing, as noted by the WHO in its recent report on Health in the Green Economy.  

A range of countries have developed labels and standards for green building or energy efficiency for buildings. As is mirrored in New Zealand, many international housing rating systems and assessment tools that consider energy efficiencies fail to measure housing aspects related to health impacts—beyond basic safety. Assessment of housing quality that includes health, safety and sustainability can provide a more robust basis for policy development, compliance monitoring and research on the quality of housing stock.

In 2001 the United Kingdom established a ‘Decent Homes’ standard, which states that houses should be warm, weatherproof and have reasonably modern facilities. Such homes are a key element in developing thriving, sustainable communities where crime is reduced and where employment and educational opportunities are improved.

Alongside the Housing Act, 2004 the Housing Health and Safety Rating System (HHSRS) measures housing conditions. Rather than assessing against a fixed standard, the HHSRS employs a risk assessment approach to enable risks from hazards to health and safety in dwellings to be minimised. The system applies to all dwellings, regardless of ownership.

Once it is accepted that unsatisfactory housing conditions can have a negative effect on health, it is logical to assume that there will be a cost to society. In the United Kingdom the Audit Commission has recently stated that every £1 spent on providing housing support for vulnerable people can save nearly £2 in reduced costs of health services, tenancy failure, crime and residential care.

Furthermore, a report that combines housing quality data from the HHSRS and health service costs highlights potential savings of more than £600 million a year from dealing with the most pressing housing problems, such as slips, trips, falls, mould and other treatable aspects of unhealthy housing. This saving to the health sector is thought to be around 40% of the total cost saving to society.

What needs to be done to introduce a housing quality rating tool in New Zealand

Based on more than a decade’s experience with systematically assessing the quality of approximately 3000 New Zealand houses, we see no significant scientific or technical difficulty with introducing a valid, practical and useful housing quality rating scheme for New Zealand.

In order to get the HHI to function as a housing quality rating tool, stakeholders, such as local Councils, Housing New Zealand, Ministry of Business Innovation and Employment (MBIE), District Health Boards, Ministry of Health, Accident Compensation Corporation (ACC), and Energy Efficiency Conservation Authority (EECA) need to support the implementation of the HHI.

For the HHI to become a nationwide housing assessment tool the essential requirement is national leadership at a political and agency level, and some resources and infrastructure support. The Children’s Commission’s Expert Advisory Group on Solutions to Child Poverty has called for the introduction of a WOF underpinned by a
health-based assessment tool such as the HHI and there has been cross-party support for
the policy.43
Initially the HHI could be voluntary, but legislative or regulatory measures would be
necessary if the rating system was to move from a voluntary to compulsory basis (which
would be required if it were to be applied to the most vulnerable populations - those
living in poor quality rental housing).

Conclusion
Substandard housing is a major problem in New Zealand that adversely affects the
health and safety of a large proportion of our population.

The HHI is a practical risk assessment tool that can measure the physical characteristics
of houses to assess the potential risks to the health and safety of the occupants, as well
as the energy efficiency of the house. The tool is the only outcome-validated housing
quality assessment tool available internationally. A valid housing assessment tool, such
as the HHI, can guide improved housing quality and therefore better health, a reduction
in home injuries, and an improvement in energy efficiency.

Better housing quality would yield improvements in population health, safety and the
economy more generally. There is widespread support for introducing this evidence-
based approach. The main elements needed now are high-level policy commitment and
resources to coordinate its implementation.

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Appendix 1. Examples of housing improvement sources and rating tools

www.energywise.govt.nz is an online tool which provides information about how to make a building energy efficient, through the use of insulation, draught blocking, dampness, double glazing, space-heating, thermal mass, hot water and lighting appliances.

www.smarterhomes.org.nz is a website with information to help during renovations and new builds, it is supported by the Ministry for the Environment, Department of Building and Housing, Beacon and Building Research. It provides design information about energy, water, landscaping, materials and construction.

www.rennovations.co.nz/smarter-homes is a website to use when undertaking a building project.

www.righthouse.co.nz was established in 2007 and is sponsored by Meridian Energy. It provides information on home heating, lighting, hot water, insulation, house aspect and design and electricity generation. This site is a provider of EnergyWise.

Energy Efficiency and Conservation Authority (EECA) runs Healthy Homes (heating and insulation) and www.energysmart.co.nz (heating and insulation)

The Healthy Housing Programme is run by Housing New Zealand in combination with District Health Boards. This programme aims to raise awareness of diseases, improve access to health and social services, reduce overcrowding, and reduce the risk of housing related health issues.

www.homestar.org.nz is a New Zealand Building Industry initiative which assesses homes using a 1-10 star rating system. The home rating system is a free online tool that promotes healthier, more comfortable, energy efficient homes. Homestar was launched in 2010 and during the first year approximately 5,000 people carried out the online assessment, which on average took about seven minutes. There is also a certificate assessment, which takes approximately three-to-four hours and is carried out through a third-party.

www.smarthome.net.nz provides information on technology and wiring of homes.

http://www.lifemark.co.nz/home.aspx is an independent seal of approval (Lifemark) awarded to homes that have been designed and built to achieve specific quality design standards which make them easy and safe to live in – for a lifetime.
SUDI prevention: a review of Māori safe sleep innovations for infants

Sally Abel, David Tipene-Leach

Abstract: Recent research and policy around sudden unexpected death in infancy (SUDI) have emphasised the place of safe sleeping practices within SUDI prevention strategies. Māori SUDI prevention workers have focussed on innovations around the safe sleep environment for some time now, as they have grappled with difficult to change and disproportionately high Māori SUDI rates.

The wahakura (a flax bassinet modelled on a traditional Māori infant sleeping item) was developed in 2006 aiming to mitigate some of the risks of bedsharing with vulnerable infants, in particular infants exposed to maternal smoking in pregnancy. Early wahakura projects in Gisborne and Hawke’s Bay showed high acceptability, effectiveness as an infant health promotion vehicle but difficulty maintaining a low/no cost supply for vulnerable families. The Hawke’s Bay project revealed two pathways forward: the need for robust research to ensure the safety of the wahakura and the exploration of financially viable and more readily available alternatives. Work on both pathways is currently in progress around the country, signalling New Zealand’s ongoing contribution to SUDI prevention and its potential contribution to knowledge and practices applicable to indigenous and other marginalised communities worldwide.

Recommendations by New Zealand coroners and recent publications in the New Zealand Medical Journal have highlighted the urgency of ensuring that strategies to prevent sudden unexpected death in infancy (SUDI) are well understood and effectively implemented by parents and caregivers of young babies. In addition, in June 2012, the Health Quality & Safety Commission (HQSC) wrote to all District Health Boards urging them to prioritise SUDI prevention strategies and making a number of recommendations in this regard.

The issue of infant bedsharing has come in for particular attention, with recommendations to ensure consistent safe sleep messages are given and to provide safe sleep options where necessary to families with vulnerable babies. This focus on the infant safe sleep environment has been central to Māori SUDI prevention workers for the last seven years as they have grappled with difficult to change and disproportionately high Māori SUDI rates.

In this viewpoint article we review the development of Māori initiated innovations for safer infant sleep environments, and suggest that these and other local safe sleep initiatives and research have the potential to keep New Zealand at the forefront of international SUDI prevention research and advice.

Sudden infant death syndrome (SIDS) has been defined as “the sudden unexpected death of an infant <1 year of age, with onset of the fatal episode apparently occurring during sleep, that remains unexplained after a thorough investigation, including

[Further content continues... ]
performance of a complete autopsy and review of the circumstances of death and the clinical history.”

The broader term SUDI describes “any sudden and unexpected death, whether explained or unexplained (including SIDS), that occurs during infancy. After case investigation, [SUDIs] can be attributed to suffocation, asphyxia, entrapment, infection, ingestions, metabolic diseases, and trauma (accidental or non accidental).”

New Zealand has the highest SUDI rate in the industrialised world. Over the period 2003 to 2007 there was an average of 65 SUDI deaths per year or a rate of 1.1 deaths per 1,000 live births. It is the main cause of post neonatal mortality in infants up to 1 year of age in New Zealand and although dramatic reductions in SIDS and SUDI occurred throughout the 1990s, since 2002 post-neonatal SUDI death rates have remained static.

Deaths classified as SIDS still predominate in SUDI figures but the proportion of SUDI attributed to accidental suffocation/strangulation increased steadily over the period 2005 to 2009 and was particularly high for very young babies, accounting for 61% and 38% of SUDI deaths in babies aged 0-3 weeks and 4-7 weeks respectively.

The most recent publication on accidental suffocation in New Zealand reported that, amongst the 50 deaths from suffocation in a place of sleep recorded between 2002 and 2009, the most common age of death was one month or under. While the age range of the deaths in this report was 0-24 years of age, 48% of these were infants under one year of age.

Much of SUDI prevention research and advice has been focussed on SIDS prevention, although it is believed that SIDS prevention practices can also help prevent suffocation/strangulation in bed. The classic approach to preventing SIDS deaths has been to define the risk factors, devise the appropriate messages and then design and implement an information-sharing health promotion campaign. Indeed, this has worked very well in mainly middle class, white communities in which advice to change from the prone to the back sleeping position was associated with a huge decrease in post-neonatal death in the 1990s. However, it has not been as effective amongst Māori, whose babies are now significantly over-represented.

In the period 2003 to 2007, 62% of SUDI deaths were Māori. This equates to approximately 40 deaths per annum, a rate of 2.3 deaths per 1000 live births, which is four and a half times that of Other (non-Māori, non-Pacific, non-Asian) infants whose rate is 0.52 per 1,000.

The risk of SIDS increases significantly with maternal smoking in pregnancy and with bed-sharing where the mother smoked in pregnancy. SIDS is also associated with high socio-economic deprivation.

In New Zealand high rates of smoking in pregnancy persist amongst Māori women. A recent Auckland study found that 53% of Māori mothers smoked in pregnancy compared to just 8% of a mostly European sample, confirming the relatively poor success of smoking cessation programmes among pregnant Māori women.

Although some commendable efforts have been made regarding tobacco policy and Māori smoking rates, the pervasive marketing of tobacco alongside the difficulties...
of dealing with smoking addiction in poorly resourced communities have made progress in this area very challenging.

Consequently efforts to reduce smoking in pregnancy, including exploration of new innovative approaches, should remain a primary aim of health authorities, along with strategies that take a wider approach to SUDI prevention.

For several years now Māori SIDS prevention workers have recognised that the phenomenon of ‘bedsharing where the mother smoked in pregnancy’ is deserving of specific attention. The Auckland studies showed that 21% of Māori mothers had both smoked in pregnancy and ‘always’ or ‘sometimes’ co-slept with their baby, compared with only 1% of the mostly European mothers.\textsuperscript{14,15}

Considering the difficulty of effecting smoking cessation amongst Māori women during pregnancy, attention moved towards how to increase infant sleep environment safety without necessarily banning bedsharing, the closeness of which is heralded as beneficial both for bonding and promoting breastfeeding.\textsuperscript{18,19} Also speaking to the importance of working with safety issues around bedsharing are infant deaths from accidental suffocation, which have continued to increase over time.

Māori have been shown to feature prominently in these figures, with the latest data showing that between 2002 and 2009 the Māori death rate from suffocation in the place of sleep was 8.22 times the European rate.\textsuperscript{10}

Bedsharing is relatively common amongst Māori. The two Auckland studies\textsuperscript{14,15} found that 65% of Māori mothers had bedshared for some period the night before, compared with 27% of the mainly European mothers.

Neither health promotion advice nor coroners’ frequent urging of parents to avoid bedsharing with infants less than six months of age appear to have impacted significantly on this behaviour. In addition to the bonding and breastfeeding benefits it affords, it seems that bedsharing amongst Māori is both a culturally valued behaviour\textsuperscript{20} and an infant sleeping practice that is prevalent in resource-poor homes.

The issue for Māori SUDI prevention health workers therefore became how to find a ‘safer sleep environment’ that was both culturally acceptable and practical.

**The wahakura (flax bassinet)**

A first expression of this ‘safer sleeping environment’ emerged in Gisborne in 2006. Similar to a pre-European Māori product called the pōrakaraka,\textsuperscript{21} the wahakura (‘waha’ to carry, ‘kura’ precious little object) is an approximately 72 x 34 cm bassinet-like object woven from harakeke (New Zealand flax). It comes with a thin foam mattress and a set of ‘rules’ that promote back sleeping; keeping the wahakura free of pillows, bumpers, loose blankets or toys; keeping the baby’s environment smoke-free; and banning the proximity of tired or inebriated adults, alongside the promotion of ‘every time, every place, every sleep’ usage, a return to the wahakura after feeding and sharing the ‘rules’ with every possible caregiver.

The wahakura seeks to provide a safer sleeping place for infants, particularly within a shared parental or caregiver bed. This form of maintaining closeness with baby is likely to find favour with Māori over the currently promoted bassinet beside the bed. In particular, the traditional origin and the ‘Māori flavour’ of the flax construction are
designed to appeal to the Māori mother who might otherwise reject advice not to bedshare in an unsafe fashion.

Figure 1. Wahakura (Photo credit: Kath Allen)

The development of the wahakura prototype and a trial of its production and distribution were the focus of a Te Puni Kōkiri funded project in Gisborne in 2006 and 2007. The prototype development phase determined the appropriate design and size and the type of harakeke needed to ensure sturdy sides and durability. Eighty-five wahakura were distributed through a Māori midwifery service to mothers of vulnerable Māori babies.

Two significant outcomes identified in the project audit were the high level of acceptability of the wahakura by whānau (extended family), and that participating midwives found it invaluable to successfully deliver a range of antenatal and infant health promotion messages (such as, smoke-free environments for babies and the promotion of breastfeeding) within a culturally conducive paradigm.

Most of these wahakura were subsequently either distributed for use amongst other whānau members expecting babies or became the ‘security blankets’ of the growing infant. The inability to reclaim them back into the project, therefore, led to a problem with sustainability of supply. The Gisborne project itself stalled around the expense of making further wahakura.

In the attempt to boost production skills and thereby supply, the making of wahakura was promoted by the Māori SIDS Prevention Programme (now known as Whakawhetu) as an important focus of their national SIDS prevention work from 2008. They ran a number of meetings around the country aimed at training and up-skilling weavers and health promoters around the production and use of the wahakura and subsequently regional wahakura projects developed in Northland, Auckland and Waikato.

Although these projects did not translate into a sustainable supply of wahakura for vulnerable Māori babies, a number of weavers around the country continue to make wahakura and Whakawhetu continues to promote them.
The Hawke’s Bay Tu Meke First Choice PHO Project

Building on the Gisborne wahakura work, a Hawke’s Bay Ministry of Health funded project was initiated in late 2008 by Hawke’s Bay’s former Tu Meke First Choice Primary Health Organisation (Tu Meke PHO).

The Wānanga Wahakura – Weaving Our Way to the Future project had two objectives: to further investigate the wahakura as a vehicle for antenatal health promotion delivery and to explore the viability of Māori communities producing a sustainable supply of wahakura without major external funding.

Four sites of production/distribution were trialled - a Māori midwifery practice in Hastings, a Māori Women’s Welfare League/urban marae in Napier, a Primary Health Organisation in Wairoa and a single weaver working with community networks of her own in the high deprivation Flaxmere community.

Each site confirmed that using the wahakura and its associated educational resources as a focus for delivering antenatal infant health promotion messages was very successful. The project was, however, unable to demonstrate an approach that could produce wahakura from the community in an economically sustainable fashion.

The project evaluation found a number of reasons why this was difficult. The high degree of weaving skill required and the length of time it took to make a wahakura militated against easy and ready construction. Any chance of a supply evolving without ongoing external funding was clearly not viable.

In addition, there was a paucity of people with these particular weaving skills and some constraints around supply of the appropriate types of long flax. This meant that, although mothers who had reasonable financial means or weavers in the whānau had a good chance of obtaining a wahakura, those who had neither, usually those whose babies were most vulnerable, were unlikely to be able to access one unless production was funded.

The Tu Meke PHO project was pivotal in clarifying the way forward for further development of Māori safer sleeping environments by determining two onward pathways.

Wahakura research

The first pathway determined by the Tu Meke PHO project was the development of research that might establish the safety or otherwise of the wahakura as an infant sleep environment. Consequently, collaboration between Hawke’s Bay researchers and researchers from the University of Otago and Otago Polytechnic led to the development of the Kahungunu Infant Safe Sleep (KISS) study, a Health Research Council funded three year project which was initiated in Hawke’s Bay in 2011.

This study is randomising approximately 240 mothers who attend Hawke’s Bay midwifery services with many Māori clients, to either a wahakura or bassinet as a sleeping environment, and then seeking to determine the safety and other benefits, or harm, of each.

It is designed to examine thermal environment, hypoxic events, head covering/uncovering episodes, mother-infant interaction including breastfeeding and
infant sleep time, and whether there is greater maternal and whānau ‘baby mindedness’ (the ability of the mother/whānau to observe and think about her baby’s thoughts, feelings and needs) in the assigned device.

In addition, an Eastern Institute of Technology Hawke’s Bay wahakura qualitative study, funded by a Lottery Health Research Grant, was initiated in 2012. It sits beside the KISS study and is exploring in depth Māori views about and experiences of the wahakura, including contemporary understanding of historical precursors, and how the wahakura is used in normal practice.

**Alternative infant sleeping spaces**

The other pathway that became obvious following the findings of the Tu Meke PHO project was the development of alternative infant sleeping spaces that were relatively simple and cheap to procure.

The term pēpi-pod was originally conceived by Nicola McDonald, then from the Māori SIDS team, to denote any device not made of flax that served the ‘safer sleep environment’ function of a wahakura.

It was the dramatic upheaval in Christchurch homes following the February 2011 earthquake resulting in a sudden emergence of “increased risks to babies posed by disrupted living and sleeping conditions”,26 that prompted child health advocacy programme, *Change for our Children* (CFOC), to mount a pēpi-pod response after identifying a relatively cheap item in the Plastic Box store.

The pēpi-pod, considered a “sister to the wahakura”,26 is made from the bottom section of a plastic clothes container and comes with an attractive cover, a simple mattress and a sheet/merino blanket set, along with comprehensive safe sleep education resources and instruction. It is considerably cheaper than the wahakura.

![Figure 2. Pēpi-pod (Photo credit: CFOC)](image)

_Choice for our Children_ distributed 642 pēpi-pods in the five months following the Christchurch earthquake and found they were very well received by the 100 families surveyed, many of whom valued the capacity for ‘safer bed sharing’.26
Later that year the Hawke’s Bay District Health Board (HBDHB) also launched a pēpi-pod intervention. The HBDHB Safe Sleep Action Project provides enhanced antenatal safe sleep education and a safe sleeping environment audit, with the provision of pēpi-pods for families of vulnerable babies.

An evaluation of the first 14 months, during which 345 pēpi-pods were distributed primarily to Māori and Pacific families, reported on pēpi-pod usage and retention of safe sleep knowledge and behaviours with the device.

Like Cowan et al.’s (2012) Christchurch evaluation, it found high levels of acceptability, with many mothers appreciating being able to confidently have their baby close by, in or on the parental bed.  

In early 2013 the pēpi-pod was being actively deployed in five regions: Christchurch, Hawke’s Bay, Waikato, Rotorua and Otara. Research to ascertain the safety of the pēpi-pod has yet to be undertaken, however a study involving the pēpi-pod is currently underway in South Auckland.

The Haumaru moe o te pēpi study, funded by Cure Kids and the Auckland Medical Research Foundation, is a randomised control trial comparing outcomes from an enhanced safe sleep education programme that uses pēpi-pods with those from a standard safe sleep education programme. The research is expected to be completed by the end of 2013.  

A less complex method of making a wahakura has recently been developed and is currently being utilised in a safe sleep intervention by Northland DHB. The wahakura waikawa, an equally robust but perhaps artistically less appealing item, is woven from untreated flax. With preparation and weaving time being significantly reduced, the cost of the item will likely make it considerably easier to access.

Conclusion

Over the past few years Māori and other SUDI prevention workers’ promotion of infant safer sleeping devices that can be used in the parental bed has taken place within an environment in which SUDI prevention advice has often been very anti-bedsharing. More recently there is increasing acknowledgement that blanket warnings against bedsharing are unlikely to be successful when there are well understood parental and infant benefits from this practice.  

With many Māori (and other) parents and caregivers now using a wahakura or pēpi-pod, often in the shared bed, there needs to be a distinction made when reporting on infant sleeping practices between ‘direct bedsharing’ (without an infant safe sleeping device) and ‘bedsharing with an infant safe sleeping device’.

More work is needed to refine our understanding of safety and lack of safety within the shared bed. Results from the current KISS Study in Hawke’s Bay may yield useful information in this regard. In addition, a nation-wide Health Research Council funded case control study aims, amongst other things, to explore the infant/parent shared sleeping environment closely possibly identifying safer ways to bedshare.

Earlier New Zealand research that determined the prone sleeping position as the primary risk for sudden infant death syndrome, was a major contributor to
international SIDS prevention advice that subsequently saw a dramatic decline in post-neonatal mortality in the developed world.

The persistently high rates of SIDS and SUDI deaths in largely Māori communities, however, highlight the continued significance of other SUDI risk factors, such as smoking in pregnancy, unsafe bedsharing practices and social deprivation.

A traditional Māori infant sleeping device has been revived in an attempt to mitigate some of these risks. The work to provide robust evidence for its safety is in progress as is the exploration of similar alternatives. This work is directed at ensuring that babies most vulnerable to SUDI have access to a safer sleep environment without (undue) cost.

The extent of bedsharing with vulnerable infants and Māori efforts to find a solution to this problem make New Zealand an ideal place to grapple with the ‘tail of the SIDS epidemic’.

The practical approach of utilising and adapting indigenous infant sleeping methods may be equally applicable in other countries where SIDS/SUDI persists in indigenous and other marginalised communities.

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Shellfish-acquired *Vibrio cholerae* cellulitis and sepsis from a vulnerable leg

Samuel J Whittaker

**Abstract**

The following case concerns a soft tissue *Vibrio cholerae* (*V. cholerae*) infection in a fisherman who cut his foot while retrieving his fishing dinghy. It is rare for *V. cholerae* to cause extraintestinal infection. This *V. cholera* was identified as a non-toxigenic organism. The patient was successfully treated with medical therapy at Waikato Hospital (Hamilton, New Zealand) and discharged home after 10 days.

The following case concerns a soft tissue *Vibrio cholerae* (*V. cholerae*) infection in a susceptible individual, successfully treated with medical therapy at Waikato Hospital (Hamilton, New Zealand).

**Case report**

A 55-year-old gentleman was brought in by ambulance to Waikato Hospital 48 hours after piercing the underside of his foot on an oyster shell while retrieving his fishing dinghy in the Coromandel.

This recreational fisherman had noticed increasing leg pain and swelling developing over the previous day, and presented to a peripheral centre for assessment. Whilst en-route to Waikato by ambulance, he suffered two febrile seizures, and arrived tachycardic and hypotensive with a fever of 39.3°C.

He had an obviously swollen and erythematous right leg to just below the knee. Following successful fluid resuscitation he was admitted under general medicine for careful observation and treatment.

The patient was an ex-intravenous drug user, and had previously used the punctured leg to inject. While he had not injected for more than 25 years, the leg had a significant degree of venous obstruction, and had been subject to recurrent DVT, for which the patient was currently on warfarin therapy. The patient also drank a cask of wine with his partner each day, and had an elevated GGT of 331 (0–50) U/L and an AST:ALT ratio of 2:1. The patient was not known to be immunocompromised.

IV ceftriaxone and vancomycin were altered to IV ceftazadime and oral ciprofloxacin after blood cultures grew *V. cholerae*.

The patient’s cellulitis resolved after 10 days of therapy and he was discharged home with crutches to assist mobilisation. No surgical debridement was necessary.

**Discussion**

When the laboratory culture initially identified the organism as a *Vibrio* species, it was presumed that the patient had contacted *Vibrio vulnificus* (*V. vulnificus*).
**V. vulnificus** is one of the top four marine-acquired soft-tissue infections alongside the *Aeromonas* genus, *Erysipelothrix rhusiopathiae* and *Mycobacterium marinum*.¹

*V. vulnificus* infections are the leading cause of death from shellfish ingestion in the United States, and a frequent cause of necrotising fasciitis in marine wound infections.²

Marked haemorrhagic bullae are characteristic for *V. vulnificus* soft-tissue infection.³ Antibiotic recommendations are for either IV cefotaxime or ceftazadime with IV doxycycline or ciprofloxacin.⁴,⁵

In contrast, *V. cholerae* soft-tissue infections are a relatively rare phenomenon. A PubMed search of “vibrio cholerae” and “cellulitis” outlined 16 case reports and 2 case series. A degree of hepatic impairment or immunocompromise is seen in the majority of non-gastrointestinal *V. cholerae* infections.²,⁶,⁷

It has been recommended that patients with decompensated cirrhosis should not ingest raw seafood or expose skin wounds to salt water.² Antibiotic recommendations for extraintestinal infections are as for *V. vulnificus*⁷ (above).

There are over 200 strains of the *V. cholerae* species. *V. cholerae* infections are reportable to public health. Testing in New Zealand is centred on determining if the *V. cholerae* organism is toxigenic. Types 01 and 0139 contain the cholera toxin, which elevates cAMP in enterocytes, leading to increased chloride excretion and decreased sodium resorption. This results in profuse watery diarrhoea and infectious spread.

The *V. cholera* isolated in this case was not a type 01 or 0139 organism, and therefore was not further classified. Non-type 01 *V. cholerae* are thought to be more capable of producing systemic infection, but are not usually responsible for human cholera epidemics.⁸

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Drug-induced subacute cutaneous lupus erythematosus due to treatment with interferon beta-1a

Sarah Buchanan, Ian Rosemergy, Paul Healy

Abstract
Drug-induced subacute cutaneous lupus is a very rare adverse reaction to medications. This case report describes onset of this condition caused by Interferon beta-1a, which has been rarely reported previously.

Drug-induced subacute cutaneous lupus erythematosus (DI-SCLE) is a rare, idiosyncratic adverse drug reaction. It was originally described in association with antihypertensive therapy, and these medications are still amongst the most common provoking agents. However, a growing number of other medications have been implicated in causing this condition.

Classically, DI-SCLE presents as an annular, erythematosus or papulo-squamous rash affecting predominantly sun exposed areas. It does not differ significantly from idiopathic SCLE in its biochemical or clinical characteristics. Patients frequently have anti-SSA/ Ro antibodies.

Here we discuss a case of severe DI-SCLE in a patient taking Interferon beta-1a, a very rare adverse effect of this medication.

Case report
A 33-year-old woman was under regular follow up in neurology clinic. Her history includes epilepsy diagnosed at age 16 when she developed generalised tonic-clonic seizures. Her EEG was consistent with an idiopathic generalised epilepsy.

Multiple anti-epileptic drugs were either ineffective or not tolerated, and she was started on carbamazepine 400 mg twice daily 4 years prior to this presentation, with variable seizure control.

She was diagnosed with relapsing-remitting multiple sclerosis in 2005 at age 27 after mentioning bilateral leg numbness and weakness at a routine follow up. An MRI scan of the brain was consistent with a demyelinating illness. She was treated with glatarimer acetate for 5 years; however, due to a high relapse rate, this was ceased in 2011.

In May 2011 she was commenced on subcutaneous Interferon beta-1a (Avonex) 30 mcg at weekly intervals. Although the first dose was uneventful, within 24 hours of the second injection she noted the onset of an itchy, erythematous, blanching rash which began on the trunk and spread over a period of several weeks to involve chest, back and arms, sparing the soles and palms (Figure 1).

The rash worsened with sun exposure. By the time she re-presented to neurology clinic she had received six doses of interferon.
Investigation revealed antinuclear antibodies in a homogenous pattern, at a titre of 1:160. Anti-SSA/ Ro antibodies were present. No other antibodies were positive initially, although anti-SSB/ La was present on repeat testing 6 months later.

Anti-histone antibodies were negative. Biopsy of the rash showed a patchy lichenoid inflammatory cell infiltrate and a mild perivascular chronic inflammatory cell infiltrate, consistent with subacute cutaneous lupus erythematosus. Direct immunoflourescence was negative.

She experienced no systemic symptoms suggestive of connective tissue disease. Blood counts, renal function and urinalysis were normal.

Interferon therapy was ceased immediately. She was given topical steroids and advised to avoid sunlight. With no significant improvement after 4 weeks, she was commenced on hydroxychloroquine 200 mg twice daily. This was also ineffective and the patient was then prescribed prednisone 40 mg daily.

There was gradual but clear improvement in the rash and pruritic symptoms. Carbamazepine dosing remained stable over this time.

Figure 1. The patient’s rash (A) at diagnosis, and (B) after 4 weeks of topical therapy
Discussion

Drug-induced SCLE has been precipitated by a wide range of medications, however there have been only a handful of cases implicating carbamazepine and two from Interferon beta-1a.2,4-6

Due to the striking temporal relationship between starting interferon therapy and symptom onset, interferon was thought to be the provoking agent, although carbamazepine has previously been reported to cause DI-SCLE after an unusually long incubation period of 5 years.4

Management centres around identifying and withdrawing the provocative medication, as withdrawal of the offending drug usually leads to symptom improvement or resolution within a matter of weeks.1 There are no reports of successful treatment of DI-SCLE without withdrawal of the precipitating medicine.1

As the patient did improve whilst taking the carbamazepine, it is unlikely that this was the causative medication. However, some features of this case are unusual for DI-SCLE. Firstly, the incubation time was very short—only 1 week. The two previously reported cases of interferon-beta induced SCLE both had incubation times of 20 weeks.2,6 Incubation times for DI-SCLE are generally in the range of 4 to 8 weeks but may be up to 5 years.1,2,5

In addition, symptoms persisted for an unusually long time period despite therapy. The unusual severity of this case raises the question of what role carbamazepine played in this presentation. It has been hypothesised that a succession of environmental triggers can prime patients to present with autoimmune disease.7 Carbamazepine may have been such a trigger in this patient.

A third possibility is that this was sporadic subacute cutaneous lupus. There are no clear clinical, pathological or biochemical features which distinguish between sporadic and drug-induced forms of the disease.1 However, the strong relationship between starting the drug and symptom onset make this seem less likely.

This case highlights a rare but significant side effect of treatment with interferon beta-1a which prescribers should be aware of.

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Raccoon eyes in amyloidosis

Michael Prystajecky, Habib U Rehman

An 85-year-old woman was seen in the emergency department with abdominal pain and constipation. Her medical history included dialysis-dependent chronic renal impairment, AL amyloidosis, and monoclonal IgG lambda gammopathy.

Bilateral periorbital (“raccoon eyes”) and nasolabial fold ecchymosis was noticed (see Figures 1 and 2). Raccoon eyes are due to deposition of amyloid in the capillary walls, leading to fragility of capillaries, which can burst after minor trauma like sneezing, cough, or rubbing the eyes. Raccoon eyes can be a sign of bleeding diathesis in AL amyloidosis, particularly if the patient has acquired factor X deficiency.

Focal or generalised haemorrhage is a commonly encountered clinical problem in patients with amyloidosis and can prove fatal at times. The bleeding manifestations frequently observed in patients with immunoglobulin light chain amyloidosis (AL) have been attributed to different pathogenic factors including amyloid deposits in capillary walls, deficiency of some clotting factors, and the presence of plasma components interfering with fibrin formation. However, in one study involving 100 patients, bleeding occurred frequently in the absence of abnormalities of clotting tests, suggesting that haemorrhage in amyloidosis is most often due to amyloid infiltration of blood vessels.¹

Raccoon eye sign is not pathognomonic of amyloidosis, and bilateral periorbital ecchymosis has also been described in thoracic and fractures of the skull base.²,³ The occurrence of periorbital ecchymosis, either spontaneously or after minimal trauma or Valsalva manoeuver, should raise the possibility of vascular fragility secondary to amyloidosis.

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References:

Mediastinal enlargement

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Clinical—Chest radiographs in a 74-year-old female indicated widening of the right middle mediastinum in the supine position, but not in the standing position (Figure 1). Chest computed tomography (CT) was performed (Figure 2).

Figure 1. Chest radiography

![Chest radiography](image1)

Left: supine position, right: standing position.

Figure 2. Chest computed tomography

![Chest computed tomography](image2)

Significant dilated SVC was seen (arrows). SVC: superior vena cava; Ao: aorta; PA: pulmonary artery.

What is the abnormality?
Answer—Chest CT showed a markedly dilated superior vena cava (SVC) without any evidence of obstruction in the other vessel below the dilation. SVC aneurysms are one of the rarest cases of mediastinal masses.

Most cases are usually asymptomatic. A change in the size of the lesion in chest radiography depending on posture is a feature specific to venous aneurysms.

While long-term prognosis is relatively good, even without surgery, rupture of aneurysm and fatal pulmonary embolism were rarely reported. Clear criteria for surgical indication have not been established.

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Coke’s anti-obesity campaign: a FIZZ or not?

The new campaign from Coca-Cola New Zealand to address obesity marks a potentially significant change by the iconic beverage company. In presenting this campaign Coca-Cola is acknowledging for the first time that their products play an important role in the epidemic of unhealthy weight gain.

Fast-food companies, including Coca-Cola, generally attempt to distract consumers from the harmful effects of their products by aligning themselves with health-promoting ventures such as sponsorship of sport, or by promoting their products, say, as fat-free when they are full of sugar or salt. Some have also teamed up with weight-loss companies (for example Weight Watchers and McDonalds) to enhance their image and health profile.

So is this new approach just another marketing ploy, or does it signal that the world’s most successful soft drink company is ready to address the world’s most visible epidemic by modifying the products they sell?

Coca-Cola’s campaign involves four initiatives:

1. Smaller portion sizes;
2. Calorie counts on vending machines;
3. Increased sponsorship of physical activity programmes; and

While increased sponsorship of physical activity is marketing in disguise and shouldn’t be encouraged, the other three proposals are small steps in the right direction.

Calorie counts on vending machines is not a radical change and as long as the information is highly visible and simply presented, we encourage Coca-Cola to do it now.

The other two initiatives, if taken to their logical conclusions are potential game changers. There is a significant literature that show people consume more when given larger portion sizes, thus supporting a move to smaller portion sizes as a weight-reducing strategy—but to be meaningful, Coke needs to phase out the larger portion sizes. Unfortunately the recent unsuccessful campaign to limit the size of soft drinks sold in New York City indicates that this will be strongly resisted.

In contrast, Coke’s intention to offer more low-kilojoule drinks is, with several modifications, not only likely to be the most effective initiative but also the easiest to achieve. The first modification is to change the language from ‘low kilojoule’ to ‘sugar-free.’

Aside from the calories, sugar is a concentrated form of fructose that appears to have addictive properties similar to nicotine in tobacco. So sugar intake, not just total energy, is an important health problem that must be addressed.
Secondly, to be at all meaningful, Coke’s goal of providing a wider selection of sugar-free drinks has to result in a reduction in the consumption of sugar-sweetened drinks. We will expect Coke to demonstrate that this initiative is a genuine attempt to reduce obesity.

To have any impact they need to increase the promotion and sales of their sugar-free brands (Coke Zero, Sprite Zero, Diet Coke) as their flagship products while systematically decreasing the marketing and sales of their sugary brands. In addition we will monitor and report on the introduction of sugar-free options for their entire product line.

We will also lobby Coca-Cola and the Government to support the goal of FIZZ (Fighting Sugar in Soft-Drinks), a new advocacy group fighting for a sugar-sweetened soft-drink free New Zealand by 2025. To support Coke’s sugar-free initiatives and maintain a level playing field among all beverage producers we will lobby Government to provide a regulatory environment that facilitates the substitution of sugary drinks with sugar-free alternatives. For example many hospitals and schools already have sugar-free soft drinks only policies that could be easily extended to all public institutions in New Zealand.

The next logical step would be legislation along the lines of the smoke free environments bill. Differential taxation of sugar-sweetened and sugar free soft drinks would likely also have a major impact on consumption patterns. All these initiatives have been in place for years for tobacco, so they are clearly doable.

Most public health advocates argue that sugar-sweetened soft drinks should be substituted with water and unsweetened milks. While we agree it would be ideal, this strategy hasn’t worked and whatever the real and perceived harms of sugar-free soft drinks, they are insignificant compared with the harms of sugary drinks.

Of note, several reviews have concluded that artificial sweeteners do not increase consumer’s weight\textsuperscript{6-9} and a recent review of the metabolic effects of the use of artificial sweeteners in young people found no evidence that intake of artificial sweeteners adversely affected metabolic health.\textsuperscript{10}

Our philosophy at FIZZ of substitution rather than elimination does not undermine the financial viability of the beverage companies and therefore has a greater probability of success. We plan to work with the beverage industry and will support any initiative that leads to the substitution of sugar-sweetened soft drinks with sugar-free alternatives. We believe this is the one initiative in Coca-Cola’s campaign that has the potential to meaningfully impact on obesity in New Zealand and particularly among children.

This paper has been prepared by FIZZ (Fighting Sugar in Soft-drinks) New Zealand; an advocacy group established to reduce the consumption of sugar sweetened beverages in New Zealand to zero by 2025.

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Revoke PM\textsubscript{10} regulations

A moratorium should now be placed on regulations involving atmospheric PM\textsubscript{10}. These have caused expense to us all, loss of secure home heating for many and increased risk of illness in the vulnerable.

The authoritative ‘Review on Health effects of Air-pollution’ from WHO\textsuperscript{1} blames SO\textsubscript{2} from coal burning, ozone, and in particular, the products of motor-vehicle combustion. Motor-vehicle exhaust contains “ultrafine particles, carbon monoxide, nitrous oxide, black carbon, polycyclic aromatic hydrocarbons and transition metals,” which “are likely to be responsible for the observed health effects.” “Long-term (years) exposure to PM\textsubscript{2.5} is associated with both mortality and morbidity. The evidence is weaker for PM\textsubscript{10}.”

The report only says that “exposure to residential wood combustion may be associated with respiratory and cardiovascular health.” Short-term exposure with wood smoke at a concentration normally found in a residential area with a high density of burning wood stoves caused no change in FEV\textsubscript{1} or VC and only a mild inflammatory response in atopic individuals.\textsuperscript{2}

This has profound implications for home heating and for transport planning in cities. We need to avoid intense traffic corridors through cities and progressively reduce motor vehicle traffic, particularly diesel-powered traffic, in city centres. The controls on the burning of wood, an outcome of the PM\textsubscript{10} theory, probably do nothing for health and increase the risk of exposure to cold, the main underlying factor in winter illness. They should be revoked.

There is also an ethical aspect to the issue. Professionals who undertake contract work for the government or whose research is financially supported by a Ministry can see themselves as above approach. The attractions of professional advancement, influence, and future commissions may be more potent factors for a conflict of interest than other well-recognised situations.

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References:
Additional evidence for concern about the quality of public toilets in New Zealand

We previously published in this Journal a survey indicating suboptimal provision of soap and water in council-operated public toilets (39% had no soap, 4% had no water, n=150 toilets in the lower North Island). In response to this publication, at least five journalists wrote stories on this topic. This included conducting a poll, three site surveys and interviews with council staff. This additional investigative activity raised the following points:

- An online poll in the New Zealand Herald suggested public concerns were common. That is, out of the estimated 11,950 to 12,000 votes cast in response to the question “are our public loos really that bad?”, over a third (39%) voted for “Yes, I’d rather hold on than use ‘em”. This compared to 39% voting for “they ain’t pretty – but they get the job done” and 22% for “nah – there are plenty of countries with worse ones”). While it is very difficult to interpret such online polls, there were sufficient negative responses to suggest some degree of public concern.

- A survey of public toilet blocks in a setting outside of our study area (Tauranga), found only half (3/6) had soap. This was consistent with the statement of a local council worker reporting that out of 73 toilets, 41% had no soap dispensers.

- A New Plymouth survey found no soap in any of the six toilet blocks sampled, while a South Taranaki survey found soap in all six sampled. These additional small surveys were all outside of our original study area of the lower North Island and so provide some additional data on soap availability.

- Other problems identified in two of these surveys were: lack of signage to the facility, bad smells, lack of hand drying options, lack of warm water, a missing lock, absence of toilet paper, and a toilet being clogged up.

- One Tauranga Council worker who was interviewed, stated that the provision of soap in all toilets was a goal that the Council was working towards, but that addressing damage from vandalism was its first priority. For example, he reported that one toilet block had three soap dispensers stolen in two months and hand basins ripped off the walls and smashed every few months.

This additional information adds further support to the case for improving soap provision and the overall quality of public toilet provision in this country. In some settings, such improvements may need to include facility designs that are as vandalism-proof as possible.

The benefits of better toilets not only include public hygiene, but also public convenience and maintaining the country’s reputation amongst international tourists. Because of the national level implications, central government funding and standards may be required.
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2. Backhouse M. Public toilets fail to clean up when it comes to hygiene. NZ Herald 2013;(17 June).  
Treatment of the Insane (part 2)


In Auckland, the over-crowding of the insane has been a notorious scandal. The adverse report upon the Sunnyside Mental Hospital has been presented to Parliament, and is a reproach to every politician, and a credit to an undermanned medical staff who do their best to overcome the difficulties created by the carelessness and negligence of this, and previous Governments.

Let anyone even with scant imagination picture what the hospital ward at Sunnyside must be like when it is used also for the reception of epileptics. The late Dr. MacGregor and Dr. Hay and his assistant have no powers over the public exchequer, and have to make the best of what is given them. There are medical men in the mental hospital service in New Zealand who are ornaments to that service, and to the profession to which they belong, and the blame must rest upon the right shoulders, that is, those of the politicians, whom we impeach for failing to discharge the important trust of providing proper accommodation for the insane.

Will nothing make them alive to their duties? Perhaps an evening spent out of the Parliamentary Club in an overcrowded ward of one of their madhouses would stimulate them to instant action. Let them seriously consider the matter, and adopt a comprehensive scheme, not a thing of shreds and patches, for the modernising of these institutions.

We feel certain that if a commissioner in lunacy or other competent person were imported from England to report on the state of the mental hospitals in New Zealand, public indignation would be aroused against all the politicians concerned in the maintenance of the present cruel, but cheap, system, and the doctors of the mental hospital service and the nurses and attendants would receive a just meed of praise for the wonderful way in which they have managed to carry on their work with the pitiful resources at their disposal.

Let Parliament put a sufficient vote upon the Estimates to rid itself and the country of the reproach that clamant voters may obtain what they want, but the insane must live their lives out in antiquated madhouses, the coarse and the gentle, the criminal and the well-behaved, the maniac and the melancholic, all taking their chances together—such as they are!

The present Government may depart with honour from the custom of its predecessors of attempting to patch the rotten fabric of unclassified treatment of the insane.
Cancer risk in people exposed to computed tomography scans in childhood or adolescence

The carcinogenic effect of ionising radiation has been well documented at larger doses, but not at the doses typically delivered by CT scans (5–50 mGy per organ imaged).

This data linkage study of 11 million Australians examines this matter. Between 1985 and 2005 680,211 people aged less than 20 years were recorded as having a CT scan. The researchers compared the incidence of cancer from one year after the scan with the cancer incidence rates in 10,259,469 unexposed people.

The conclusion was that the cancer incidence was increased by 24% on average; the proportional increase in cancer risk was greater after scans at younger ages. Future risks could be reduced in all populations by restricting scans to cases with a definite clinical indication, and by improving procedures to provide a diagnostic image at the lowest possible radiation risk.

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Maternal vitamin D status during pregnancy and the bone mineral content in their children

Lowered concentrations of vitamin D during pregnancy are suggested to be related to low bone-mineral content (BMC) in offspring. An important matter as rickets, osteoporosis, osteomalacia and the risk of fractures might follow. Apparently three previous studies have produced ambiguous results. This report concerns a prospective cohort study of 3960 mothers and offspring.

Vitamin D status assessments of the mothers during their pregnancies resulted in 67% of them being classified as having sufficient levels. 28% were rated as having insufficient levels and 6% having deficient levels of vitamin D. The children had their BMC measured by dual-energy X-ray absorptiometry at age 9–10 years. The BMC measurements did not differ between the offspring of mothers in the lower two groups compared with those with a sufficient vitamin D concentration.


Management of transient ischaemic attacks

Stroke is common during the first few weeks after a transient ischaemic attack (TIA) or minor ischaemic stroke. Treatment with low-dose aspirin has an established role in the management of TIA. However, combination therapy with clopidogrel and aspirin may provide greater protection against subsequent stroke than aspirin alone. This randomised, double-blind, placebo-controlled trial report concerns this issue.
Over 5000 patients were randomised within 24 hours after the onset of a minor ischaemic stroke or high-risk TIA to either clopidogrel and aspirin or aspirin and placebo. At 90 days the clopidogrel and aspirin group has a significantly lower incidence of stroke than the aspirin cohort. The incidence of moderate or severe haemorrhage was the same (0.3%) in both groups.

Medical Benevolent Fund

NZMA Members, and families of deceased Members, may apply for aid when in situations of financial hardship or distress.

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
Tel: 0800 656161
Erratum

In the previous 12 July 2013 edition, an incorrect older set of Methuselah abstracts appeared for a few days until the correct set was uploaded (*Medical emergencies on commercial airline flights, Cardiovascular events after clarithromycin use in lower respiratory tract infections, and Computed tomographic colonography versus colonoscopy*).

(Libraries and other institutions that print out the Full Contents PDF, please print out the correct 12 July 2013 Methuselah abstracts now available at [http://journal.nzma.org.nz/journal/126-1378/5745/content.pdf](http://journal.nzma.org.nz/journal/126-1378/5745/content.pdf) and replace the pages.)


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