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This Issue in the Journal

Original Articles

Adverse reactions to food in New Zealand children aged 0–5 years
Christine Crooks, Rohan Ameratunga, Maia Brewerton, Michelle Torok, Stephen Buetow, Shannon Brothers, Clare Wall, Penny Jorgensen

This study examined food allergy symptoms in a sample of infants attending Plunket Clinics in Auckland and Wellington. Food allergy symptoms were very common and eczema was a common problem in young infants across all ethnic groups. Very few children were tested for food allergy, even though some had severe symptoms. This study shows there is an urgent need for funding further studies on food allergy in New Zealand.

Inappropriate trace element testing in the Auckland region of New Zealand
David Song, Barry Palmer, Stephen du Toit, James S Davidson

This paper reports that the number of trace element tests done by Labplus, the Auckland City Hospital laboratory, increased markedly from 2004 to 2008. The most commonly requested tests were blood levels of zinc, copper, selenium and mercury. The majority of test requests were from a small minority of doctors, who were mostly associated with “alternative” or “holistic” types of medical practice. The paper discusses the appropriate medical uses of these tests, and concludes that the majority of the tests requested have been unnecessary and of little or no benefit to patients. The paper suggests that this wastage of public health resources could be addressed by carefully defining the clinical situations in which these tests should be used.

Does health status matter for the risk of injury?
Ruth Cunningham, Kristie Carter, Jennie Connor, Jackie Fawcett

Injuries which disrupt usual activities for more than a week are common and represent a significant impact on society from injury which is not captured by health care statistics. We found a strong relationship between having poor self-rated health or a high number of chronic diseases and an increased risk of injury. The information provided by this study gives some indication of level of this aspect of injury burden, and may be useful in planning interventions targeted at those most likely to experience such injuries.
SF-36v2 norms for New Zealanders aged 55–69 years
Christine Stephens, Fiona Alpass, Merel Baars, Andy Towers, Brendan Stevenson

A survey of the health of older New Zealanders aged 55–69 showed that there were few differences in mental and physical health between the sexes in this age group, which may indicate a closing of the gap between men and women’s health status. Physical health scores generally decrease with age as expected but mental health scores increase with age. This trend toward improved mental health as people age has also been shown in international studies.

Evaluation of Pacific obstetric and gynaecological ultrasound scanning capabilities, personnel, equipment and workloads
Hemal Kodikara, Jenny Mitchell, Alec Ekeroma, Peter Stone

This is the first survey of obstetric and gynaecological (O&G) ultrasound in the Pacific and identified training, quality equipment, and service needs. The survey also identified the most appropriate types of training courses for those engaged in O&G ultrasound in the Pacific. Theoretical clinical and technical training is needed but hands-on practical training is most favoured. The future of utilising ultrasound to enhance clinical outcomes needs to be part of any expansion of ultrasound in the Pacific.

The prevalence of diabetes among adults aged 40 years and over in Fiji
Garry Brian, Jacqueline Ramke, Louise Maher, Andrew Page, John Szetu

This paper reports a 2009 survey that estimates how many adults aged 40 years and over living in Fiji have diabetes: 99,000 people (41% of the people in this age group). In this age group: Indian Fijians have approximately twice the risk of having diabetes compared with Melanesian Fijians; females have almost twice the risk compared with males; and, risk increased with increasing age. Management of diabetes is likely to be poor in a developing country such as Fiji because of scant resources. Unless something is done about improving diagnosis and management of diabetes in the community, with passing time there is likely to be an increasing, and perhaps eventually unmanageable, burden of diabetes-related complications (e.g. blindness, loss of lower limbs, kidney problems) that will put great strain on medical services, communities and individuals.

Estimating diabetes prevalence in South Auckland: how accurate is a method that combines lists of linked health datasets?
Simon Thornley, Roger Marshall, Gary Jackson, James Smith, Wing-Cheuk Chan, Craig Wright, Dudley Gentles, Rod Jackson

Linked health information (drug use, hospital diagnoses, and laboratory testing) were anonymously linked to estimate the (adult) burden of diabetes in South Auckland. About 1 in 14 adults were estimated to have diabetes in South Auckland using this method.
This figure was similar to national survey estimates and a list of people known to have longstanding diabetes. A statistical technique, known as “capture-recapture” suggests that the actual burden of diabetes in this area is much higher—about 1 in 11 adults. Our findings suggest that about 1 in 30 adults in South Auckland has diabetes but is not yet diagnosed.

**Assessment of obesity in New Zealand Chinese: a comparative analysis of adults aged 30–39 years from five ethnic groups**  
Ji Y J Wen, Elaine C Rush, Lindsay D Plank

We know that risk for diseases such as diabetes and cardiovascular disease increases with increasing body size as measured by body mass index (BMI). The risk for Asian is higher at a lower BMI than European. The immigrant Asian population in New Zealand is diverse and not homogeneous. There are differences between Asian Indian and Chinese origin people in disease prevalence and this may be related to differences in fatness, fat distribution and body proportions. For the same height and weight, Chinese had more body fat than European and less than Asian Indian. There were also differences in relative arm and leg lengths and distribution of fat and muscle. Health policy that uses BMI cutoffs to identify risk and inform treatment needs to consider ethnic disparities in the relationships between body size, fatness and other risk factors.

**Review Article**

**Can imaging determine if a rotator cuff tear is traumatic?**  
Khalid D Mohammed, Ben Wilkinson, Chethan Nagaraj

Traumatic rotator cuff tears are entitled to treatment through the ACC scheme in New Zealand. The imaging report is considered by the ACC in determining causation of the rotator cuff tear. This paper reviews the relevant historic and contemporary scientific literature on shoulder imaging in the context of causation of rotator cuff tears. Some imaging findings indicate a significant rotator cuff tear has been present for a long time, others findings may not have relevance. The common criteria in the literature for determining if a rotator cuff tear was traumatic was a history of a traumatic event with onset of symptoms.
Rotator cuff imaging and the Accident Compensation Corporation (ACC)

Michael Caughey

This issue of the Journal contains an article entitled Can imaging determine if a rotator cuff tear is traumatic? by Khalid Mohammed and colleagues.¹ The authors are to be congratulated on what is a comprehensive review of imaging of the rotator cuff largely as it relates to rotator cuff tears.

What has stimulated interest in this topic? According to Accident ACC Minister Nick Smith, between 2004 and 2008 there was a 57% increase in ACC claim costs which was deemed unsustainable.

Largely through more critical review of applications for surgery by the clinical advisory panel of the Elective Surgical Unit in Dunedin, a significant reduction in ACC-funded operations occurred. Between January and June 2008, 18,294 operations were undertaken compared to 20,679 in the same period this year. This represents a reduction of 2385 operations or minus 11%. Initially shoulder surgery and in particular repairs of the rotator cuff were especially affected by the high decline rate.

The incidence of rotator cuff tears increases progressively with age and a recent meta-analysis of patient prevalence of atraumatic asymptomatic rotator cuff tears indicated that 10% of the population at the age of 55 has a full thickness rotator cuff tear which increases to 30% by the age of 75.² Thus when particularly an older patient presents with a full thickness rotator cuff tear following an accident determining if the tear is truly traumatic or a pre-existing condition rendered symptomatic by the accident (as the clinic advisory panel frequently asserts) may be problematic.

Clearly the patient history is critical with the mechanism of injury and a force sufficient to tear the rotator cuff being key elements. The force required to damage the rotator cuff in an 80-year-old is significantly less than that of a 40-year-old in the same way that the force required to fracture a femoral neck is considerably less. Any acute loss of function is clearly an important feature of the history. Specific strength testing for the components of the rotator cuff at initial presentation is critical particularly for acute tears.

What percentage of rotator cuff tears requiring surgery are traumatic in onset? Probably the best local information comes from The Rotator Cuff Registry. This study is an initiative of the New Zealand Shoulder and Elbow Society and since March 2009 its goal has been to recruit all patients in the country undergoing rotator cuff repair. To date, 3000 patients have been enlisted making it easily the largest study of its kind worldwide. Pain and activity level questionnaires are filled out by patients preoperatively and at 6, 12 and 24 months postoperatively.

The surgeon fills out a 2-page operating day questionnaire detailing exactly what was done and this looks as if it will provide powerful information on best practice in managing cuff tears—e.g. double vs single row repair, arthroscopic vs mini-open vs
open repair, management of SLAP tears, the biceps tendon and the AC joint as well as
the influence of NSAIDs, smoking, and physiotherapy on the surgical outcome. In the
“Event details” pre-op form patients are asked “Is your shoulder problem the result of
an accident?” In a recent analysis of the data 90% of patients replied “Yes”.

The accuracy of the history of injury as detailed by the patient has been called into
question. In discussions between the NZ Shoulder and Elbow Society and ACC
Representatives the latter group has indicated that in some cases they observe the
patient’s history of injury evolving in magnitude with time. Hence the pursuit of
potentially objective information that imaging may provide.

Heavy reliance has been placed on such information by the Clinical Advisory Panel in
coming to decisions regarding patients’ eligibility for ACC-funded elective surgery.
Oftentimes this has outweighed the evidence of a strong history of injury and obvious
clinical findings of a rotator cuff tear. Thus the significance of acromial shape,
upward migration of the humeral head, cystic change in the greater tuberosity, degree
of tendon retraction, and the degree of atrophy and fatty infiltration of the parent
muscle have been closely scrutinised in both the aetiology and likely chronicity of
rotator cuff tears.

There are two areas of ongoing discussion and debate I would like to focus on in this
editorial. First, how relevant is acromial morphology? Second, I stress the difficulty
of differentiating between primary and secondary impingement.

Probably the most quoted study on the relevance of acromial shape is that presented in
1986 by Bigliani and Morrison. In the 140 cadaveric shoulders dissected type 1 or
flat acromia were associated with a 3% rate of cuff tears, type 2 or curved acromia
with a 24% rate of cuff tears and type 3, or hooked acromia with a 73% rate of cuff
tears. While an apparently convincing correlation subsequent studies, notably one by
Gill and associates showed no significant association between type 3 acromia and
rotator cuff tears in patients over 50 when age adjusted.

They suggested both the presence of type 3 acromia and rotator cuff tears were age-
related with no true causal relationship. Mohammed et al also allude to the papers By
Stehle et al and Bright et al questioning the reliability and reproducibility of
radiological assessment of the acromion. Being a three dimensional structure
assessment in more than one plane is important.

With regard to partial thickness tears Mohammed et al note “numerous reports of
articular surface tears being two to three times more common than bursal surface
tears” which is not what would be expected if primary impingement was the
mechanism. Bursal side partial thickness tears are more likely to occur with
subacromial impingement.

Impingement has been very commonly cited as a cause to decline applications for
ACC funding for rotator cuff repair. However once a patient has sustained a rotator
cuff tear, the glenohumeral kinematics are altered. A dynamic balance exists between
the powerful deltoid driving the humeral head proximally and the supraspinatus
countering this force. If weakened through tearing of the tendon, the head migrates
upward.
Ken Yamaguchi at Washington University has demonstrated that once tears increase to a size larger than 1.5 cm measurable superior humeral head migration occurs. It is very likely that smaller tears will have a subtle if not measurable effect.

Following partial thickness articular-sided tears where joint fluid bathes the torn tendon and healing rarely occurs bursal hypertrophy may provide continuity between the tendon and the humerus beyond the supraspinatus footprint. This thickened bursa frequently evident on ultrasound coupled with subtle upward migration of the humeral head may result in impingement not previously present, particularly if the subacromial space is limited.

Hence secondary impingement occurs as a direct result of the tear and this should not be interpreted as a primary impingement problem resulting in entitlement to surgery being declined.

**Competing interests:** None.

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Cartwright Inquiry correspondence in the NZMJ: enough is enough

Frank A Frizelle

In 2010, the NZMJ published several papers commenting on the *The Report of the Cervical Cancer Inquiry 1988* by the then District Court Judge Silvia Cartwright.¹ The Inquiry, as many will know, was into the management of cervical dysplasia at National Woman’s Hospital. The published papers have focused on various interpretations of data related to the Inquiry, the personalities involved, the significance of the Inquiry, and more recent commentaries on the Inquiry including a book by Professor Bryder. In addition, the role of the Inquiry in developing the current medicolegal structure in New Zealand—especially the role of the Health and Disability Commissioner (HDC)—was explored by Ron Paterson in his editorial.²

The many papers (editorials and letters) that we published—22 years later—explored a wide range of issues relevant to the 1988 Report and I have tried to give authors and responders a free hand (within the limits that an editor must set). I feel now, however, that few constructive comments are coming from the continued barrage of email correspondence sent to me.

As I have pointed out already to some who have contacted me, you do not have to convince me of whatever point of view you have, I am an editor not a referee. Any further letters published in the NZMJ on this topic must provide new insights into the issue, not just re-stating what an author previously said (or did not say). Indeed, these letters generally seem to have become tedious and repetitive and do not provide any new insight. Like children fighting it now appears that some authors believe that the person who provides the last comment is considered ‘right’. This sort of behaviour is not uncommon, and at various times on certain topics I have rejected letters when they no longer contribute to the discussion. Indeed, many journals have time limits, such as the NEJM which will accept letters for only 3 weeks following publication of an article. I have used a more open approach and have generally accepted correspondence up to 3 months later if it contributes to the discussion. We are well past that point now. On this topic I now feel that ‘enough is enough’ and I will only publish another letter if it provides new information.

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Food allergies in children: common but overlooked

Rodney P K Ford

How big is the food allergy problem in New Zealand, and what should we be doing about it? These are the questions at the heart of the article by Christine Crooks et al. They state, “We have commenced studies to determine the burden of adverse reactions to food and food allergy in New Zealand.” This pilot study documents parental/caregiver reported adverse reactions to food, experienced by children under five years of age, who were attending child-health nurse clinics.

Their main findings were: adverse food reactions are reported by a large number of parents (40%); that a wide variety of foods and a wide spectrum of symptoms are implicated; and that almost none (3%) of these children with suspected food reactions had been investigated or had any medical management help.

The motivating factors to document this problem are that more and more children are now suffering from food allergy/intolerances, which is a world-wide phenomenon; but despite this, strong medical scepticism persists about the existence of illness caused by food allergy. They wrote: “Some medical practitioners remain sceptical about the role of food allergies in a number of clinical syndromes, such as atopic dermatitis, colic and gastro-oesophageal reflux in infancy, despite an increasing body of evidence that food allergy can contribute to these conditions.”

Consequently, patients and caregivers who bring up the subject of food allergy during a medical consultation are more often than not, brushed off. Perhaps this is due to a lack of confidence or knowledge about what to do. So, when parental experiences of caring for a child with medically diagnosed severe food allergies were investigated, the authors found, “The general lack of support experienced by these families from healthcare professionals is a significant concern both for primary and tertiary health care providers. Multidisciplinary support is required for these families, and currently there is a lack of healthcare professionals with the knowledge to support these families.”

This has also been my experience. I was the first paediatrician to conduct double-blind studies with children who were suspected as having food allergy. This was done in an academic environment of cynicism. Disappointingly, over the last 30 years, food allergy remains poorly recognised and managed. So why does this attitude persist, globally?

The problem could stem from Louis Pasteur, the French chemist and microbiologist, the instigator of the germ theory of disease. After over a century of tackling infectious diseases, it is a difficult transition to acknowledge that what we are eating can cause a similar spectrum of symptoms. Food allergic/intolerance reactions are responsible for a variety of symptoms involving the skin, gastrointestinal tract, and respiratory tract and may be due to IgE-mediated and non-IgE-mediated mechanisms. But it is all
too easy to dismiss these common food allergy symptoms as “it’s just a virus” or “you’ll grow out of it”.

In addition, it is the common foods (cow’s milk, egg, peanuts, soy, wheat and gluten) that cause most of the adverse reactions. As these are the core-foods in our diets, that someone could react to them seems implausible. Moreover, that food allergens can pass through a mother’s breast milk and can cause both immediate and delayed reactions, could appear beyond belief—but they do. Finally, to add to the clinical pot, gluten has recently been discovered to cause a lot more clinical harm beyond coeliac disease.

But food allergy/intolerance indeed does affect around one-in-ten children, some with life-threatening reactions. Surveys show that 2% of children react to cow’s milk, 2% to egg, 3.3% to peanut and 3.8 to any nut, that 1% have coeliac disease and up to 10% of people suffer from a gluten-sensitivity.

Patients are demanding to be heard. The authors want to create a much better and bigger awareness of the food allergy issues. The Allergy New Zealand organisation (www.allergy.org.nz) is doing a wonderful job advocating for improved diagnostic and management services, and safe healthy food for people with food allergy/intolerance. My initiative has been to set up an “eClinic” (www.DrRodneyFord.co.nz) to help lay and medical people work through the food allergy/intolerance pathway.

There is still a big job ahead: to help clinicians understand the principles of diagnosis and management of food allergy/intolerance in childhood. Measuring the burden of adverse reactions to food and food allergy in New Zealand is a good start. When a child presents with an ongoing illness, perhaps think more about foods and less about bugs.

Competing interests: None.

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References:
Adverse reactions to food in New Zealand children aged 0–5 years
Christine Crooks, Rohan Ameratunga, Maia Brewerton, Michelle Torok, Stephen Buetow, Shannon Brothers, Clare Wall, Penny Jorgensen

Abstract

Aim The aim of this study was to describe parent/caregiver-reported adverse reactions to food in children aged 0–5 years in New Zealand.

Method A cross-sectional survey was undertaken in clinics conducted by the Royal New Zealand Plunket Society, which is the major healthcare provider for New Zealand’s Well Child programme. Parents/caregivers of 110 (65%) children participated.

Results Of the 44 children who experienced an adverse reaction to food, only four were clinically evaluated and had undergone diagnostic testing. Two other children were hospitalised following systemic symptoms. Neither was tested for food allergy. 18 (16%) children had physician diagnosed eczema.

Conclusion Within the limitations of this small study, the data indicated adverse reactions to foods are a public health concern in New Zealand and may be under investigated even in children with severe symptoms. These children remain at increased risk of continued morbidity. Based on this preliminary study further research on food allergy in New Zealand is warranted.

Adverse reactions to food (AFR) in children are a source of increasing concern worldwide. Adverse reactions to foods are common and can be classified as either intolerance or food allergy (FA). Food intolerance is not mediated by the immune system and in general children are not at risk of severe reactions. In contrast, FA is an immunological adverse reaction to food, triggered by either an IgE- or a non-IgE-mediated immune mechanism.\(^1\) IgE mediated FA is generally rapid in onset, occurring within minutes to two hours.\(^2\)

The severity of IgE mediated symptoms varies from mild oral symptoms to life-threatening anaphylaxis which can include cutaneous, respiratory, ocular and gastrointestinal responses.\(^3\) A retrospective, case-based Australian study identified adverse reactions to foods as the major cause of anaphylaxis in children presenting to an emergency department.\(^4\) Non-IgE-mediated FA symptoms typically involve sub-acute or chronic symptoms isolated to the gastrointestinal tract.\(^5\)

Modern diets have become increasingly diversified with international migration and the manufacture of novel foods.\(^6\) Changes in dietary exposure have resulted in changing FA patterns with increasing sensitisation to a growing variety of foods.\(^7\) Recent data further suggest ethnic-specific FA patterns, which may be a result of culture-bound dietary intake.\(^8\)
International data indicate that the incidence of FA is highest during the first 3 years of life, when approximately 5–6% of children may be affected. The prevalence of FA has not been systematically studied in New Zealand (NZ). From the 2006–07 NZ Health Survey, 120,600 children (14.1%) aged from birth to 14 years were estimated to have eczema that had been diagnosed by a physician. FA is frequently associated with eczema in infants.

We have commenced studies to determine the burden of AFR/FA in NZ. The aim of this study was to describe parental/caregiver reported adverse reactions to food experienced by children under 5 years of age attending Plunket clinics in NZ. All children in NZ are entitled to free well child services. Most children accessing the Well Child/ Tamariki Ora programme attend clinics conducted by the Royal New Zealand Plunket Society (Plunket Society). They can consult a Plunket nurse at a clinic or home at: 4 to 6 weeks, 6 to 9 weeks, 3 months, 5 months, 9 months, 15 months, 2 years, and 3 years of age.

Methods

Design—The design of the survey was cross-sectional. An interviewer assisted questionnaire was developed and administered to families attending four Plunket clinics in the Auckland and Wellington regions. Three urban Plunket clinics were selected from areas known to have diverse ethnic populations. A fourth more rural clinic (Tuakau) was also selected. The questionnaire included questions about AFR symptoms, recall of foods associated with a reaction, and demographic details. It was based on previous surveys of FA symptoms in children. Acute onset AFR symptoms are likely to be IgE mediated. It is more difficult to associate delayed reactions including eczema with food consumption without diagnostic testing. The AFR symptoms in the questionnaire included those occurring within two hours of consumption such as urticaria, angioedema, pruritus, gastrointestinal symptoms (including, diarrhoea, emesis, flatulence, offensive stool, reflux), rhinitis, asthma, and anaphylaxis. The presence of eczema reported by the parent/caregiver was also recorded.

Participant recruitment—Study materials (posters and information sheets) were made available in the clinic a month ahead of the planned interviews. The interview procedures were explained to the clinic nurses before the survey and their assistance was sought to recruit participants. All parents/caregivers attending the clinic with children aged up to 5 years were invited to participate following their consultation with the nurse. This included those scheduled for a visit as well as casual attendees. Siblings of children attending the clinics were also invited to join the study. Presentation of study material and subsequent recruitment were completed at the same Plunket visit. An explanation of the study was given by the interviewer, informed consent was obtained, and the survey was administered.

Data collection and analysis—Clinics with a separate interview room/area were selected to allow for privacy. These clinics were also known to have high attendance rates. The survey was performed from September to November 2009. The questionnaire was administered by the same interviewer who first established the child’s age. The parent/caregiver was shown photographs of urticaria, eczema and angioedema and asked whether they had identified symptoms and signs listed in the questionnaire within two hours of their child consuming food (including breast milk).

Ethnicity was coded according to the subgroupings of the 2006/2007 NZ Health Survey Child Questionnaire and using the total response method. This method records all ethnic groups with which the parent/caregiver identifies and so percentages exceed 100 percent. A question about the ease of answering was included at the end of the questionnaire.

All data were entered into a Microsoft Excel spreadsheet and descriptive statistics are reported. Inferential statistics were performed to assess maternal (and child) differences between the groups of children who were reported to have reacted adversely to food or not.

Ethics approval—The study methods were reviewed by the Royal New Zealand Plunket Society and the Auckland District Health Board Research office. Ethics approval was provided by the Ministry of Health’s Multi-Regional Ethics Committee (MEC09/47/EXP).
Results

Participation rate—152 children were scheduled for a visit with the nurse at the Plunket clinic on the days the interviews were performed. Parents/caregivers of 110 (65%) participated in the survey, comprising 97 (64%) of the booked appointments as well as an additional 13 (72%) casual attendees.

Table 1 compares socio-demographic characteristics of the respondent parents/caregivers (and their child) between the groups of children for whom adverse reactions to food were reported or not. No statistically significant differences at the 5% level were detected between these groups on any of the variables reported.

Table 1. Prevalence of parent/caregiver responses about adverse reactions to food by child and maternal characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total (n=110)</th>
<th>Adverse reactions ¹ (n=44)</th>
<th>No adverse reactions (n=66)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>Ethnicity of child²</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>73 (66.4)</td>
<td>28 (63.6)</td>
<td>45 (68.2)</td>
</tr>
<tr>
<td>Māori</td>
<td>21 (19.1)</td>
<td>8 (18.2)</td>
<td>13 (19.7)</td>
</tr>
<tr>
<td>Pacific peoples³</td>
<td>28 (25.5)</td>
<td>14 (31)</td>
<td>14 (21)</td>
</tr>
<tr>
<td>Asians⁴</td>
<td>25 (22.7)</td>
<td>12 (27.3)</td>
<td>16 (24.2)</td>
</tr>
<tr>
<td>Other</td>
<td>4 (3.6)</td>
<td>0</td>
<td>4 (6.1)</td>
</tr>
<tr>
<td>Age (months)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>16.7±15.2</td>
<td>18.0 ± 15.8</td>
<td>15.8±14.8</td>
</tr>
<tr>
<td>Range</td>
<td>1.0 – 67.4</td>
<td>1.7 – 67.4</td>
<td>1.1 – 56.9</td>
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<tr>
<td>Gender</td>
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<td></td>
</tr>
<tr>
<td>Male</td>
<td>59 (53.6)</td>
<td>22 (50.0)</td>
<td>37 (56.1)</td>
</tr>
<tr>
<td>Female</td>
<td>51 (46.4)</td>
<td>22 (50.0)</td>
<td>29 (43.9)</td>
</tr>
<tr>
<td>Birth order of child</td>
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<td></td>
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</tr>
<tr>
<td>1</td>
<td>62 (56.9)</td>
<td>23 (52.3)</td>
<td>39 (60.0)</td>
</tr>
<tr>
<td>2</td>
<td>26 (23.9)</td>
<td>14 (31.8)</td>
<td>12 (18.5)</td>
</tr>
<tr>
<td>3-7</td>
<td>19 (17.2)</td>
<td>5 (11.4)</td>
<td>13 (19.6)</td>
</tr>
<tr>
<td>Adopted</td>
<td>2 (1.8)</td>
<td>1 (2.3)</td>
<td>1 (1.5)</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>3.5±0.6</td>
<td>3.53 ± 0.6</td>
<td>3.5±0.57</td>
</tr>
<tr>
<td>Range</td>
<td>1.4 – 5.5</td>
<td>1.38 – 4.50</td>
<td>1.9 – 5.5</td>
</tr>
<tr>
<td>Gestation (weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>39.6±1.9</td>
<td>39.9±2.2</td>
<td>39.4±1.70</td>
</tr>
<tr>
<td>Range</td>
<td>28-42</td>
<td>28-42</td>
<td>34-42</td>
</tr>
<tr>
<td>Mother's highest educational level attained</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NCEA⁵secondary school qualification or equivalent</td>
<td>46 (41.8)</td>
<td>17 (38.6)</td>
<td>29 (43.9)</td>
</tr>
<tr>
<td>Post secondary school qualification</td>
<td>64 (58.2)</td>
<td>27 (61.4)</td>
<td>37 (56.1)</td>
</tr>
</tbody>
</table>

¹Urticaria, angioedema, pruritus, gastrointestinal symptoms (including, diarrhoea, emesis, flatulence, offensive stool, reflux), rhinitis, asthma, and anaphylaxis, and worsening of eczema symptoms; ²All ethnic groups with which the child identifies; ³Pacific peoples: (Samoan, Cook Island Māori, Tongan, Niuean, Fijian, Tokelauan, Fijian/Indian/Zimbabwean); ⁴Asians (Chinese, Indian, Japanese, Philippine, Iraqi); ⁵Other (includes other European); ⁶National Certificate of Educational Achievement.

Foods reported by the parent/caregiver to be associated with adverse reactions—29 different foods were associated with adverse reactions reported by the
parent/caregiver. These have been grouped according to the main allergen they contain in common (Table 2). For example the ‘Dairy’ group includes cow’s milk, cows’ milk formula, cheese, ice cream, yoghurt (contained fruit), and goats’ milk formula.

Table 2. Parent/Caregiver-reported adverse reaction to a food by their young child, symptoms, diagnosis and ease of answering questionnaire

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=44</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food groups associated with reactions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dairy</td>
<td>27</td>
<td>61.4</td>
</tr>
<tr>
<td>Breast milk</td>
<td>18</td>
<td>40.9</td>
</tr>
<tr>
<td>Foods containing multiple allergens</td>
<td>15</td>
<td>34.1</td>
</tr>
<tr>
<td>Fruit/Vegetables</td>
<td>8</td>
<td>18.2</td>
</tr>
<tr>
<td>Egg</td>
<td>3</td>
<td>6.8</td>
</tr>
<tr>
<td>Crustacean</td>
<td>2</td>
<td>4.5</td>
</tr>
<tr>
<td>Tree nut</td>
<td>2</td>
<td>4.5</td>
</tr>
<tr>
<td>Soy</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Could not identify food</td>
<td>5</td>
<td>11.4</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angioedema</td>
<td>5</td>
<td>11.4</td>
</tr>
<tr>
<td>Eczema</td>
<td>33</td>
<td>75.0</td>
</tr>
<tr>
<td>Hay fever</td>
<td>2</td>
<td>4.5</td>
</tr>
<tr>
<td>Life-threatening reaction (vomiting and aspiration)</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Pruritus</td>
<td>17</td>
<td>38.6</td>
</tr>
<tr>
<td>Gastrointestinal symptoms (including, diarrhoea, emesis, flatulence, offensive stool, reflux)</td>
<td>13</td>
<td>29.5</td>
</tr>
<tr>
<td>Urticaria</td>
<td>12</td>
<td>27.3</td>
</tr>
<tr>
<td>Who diagnosed food allergy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allergy specialist</td>
<td>3</td>
<td>6.8</td>
</tr>
<tr>
<td>Paediatrician</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Not investigated</td>
<td>40</td>
<td>90.9</td>
</tr>
<tr>
<td>Diagnostic test (n=4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Specific IgE (RAST)</td>
<td>2</td>
<td>50.0</td>
</tr>
<tr>
<td>SPT</td>
<td>2</td>
<td>50.0</td>
</tr>
<tr>
<td>Diagnosed allergies (n=4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cat</td>
<td>2</td>
<td>50.0</td>
</tr>
<tr>
<td>Cow’s milk</td>
<td>3</td>
<td>75.0</td>
</tr>
<tr>
<td>Dust mite</td>
<td>2</td>
<td>50.0</td>
</tr>
<tr>
<td>Egg</td>
<td>3</td>
<td>75.0</td>
</tr>
<tr>
<td>Peanut</td>
<td>4</td>
<td>100.0</td>
</tr>
<tr>
<td>Soy</td>
<td>3</td>
<td>75.0</td>
</tr>
<tr>
<td>How easy to answer were the questions asked you today? (n=89)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very Easy</td>
<td>72</td>
<td>80.9</td>
</tr>
<tr>
<td>Easy</td>
<td>17</td>
<td>19.1</td>
</tr>
</tbody>
</table>

1 Radioallergosorbent testing; 2 Skin prick testing.

It was not possible to identify the nature of the allergenic constituent in the foods containing a mixture of potential allergens. For example, commercially produced Spaghetti Bolognese contains tomato, wheat, cheese and traces of egg, while home-made scrambled egg may contain egg, cows’ milk or soy milk. In addition some parents were not able to recall the brand of food consumed; one child who reacted to a ‘baby cereal’ was subsequently diagnosed with allergies to soy, cows’ milk and
peanut. Another parent noticed their child reacted to strawberry yoghurt but tolerated other dairy. It was also not possible to determine the nature of the allergen causing reactions to breast milk.

**Symptoms**—Forty percent (44/110) of the children were reported to have experienced an adverse reaction to food and 64% (28/44) of them experienced the reaction within 2 hours of the consumption. Two children were hospitalised after experiencing AFR symptoms.

Thirty three children were reported to have eczema. Eczema was diagnosed by a doctor (family physician/ general practitioner) in 18/110 (16%) children. In this small sample of children the percentage with doctor diagnosed eczema (14%) is higher than reported in the 2006/2007 New Zealand National Health survey.11

Eczema symptoms were reported to worsen within two hours of food consumption in 10 children. Two of these 10 children had physician diagnosed eczema.

Eczema symptoms were reported to improve in 6/6 children after their diet was altered. Parents/caregivers modified their child’s diet by attempting to eliminate suspected food allergens. Diet changes were recommended by a physician for one of these six children. No allergy testing was recommended for this child.

Eczema symptoms improved in 3/3 children after their mothers altered their diet while breastfeeding. Diet changes were recommended by a physician for two of these mothers. Again, no allergy testing was undertaken. No parent/caregiver in this study received advice from a dietitian; the reason for this was not investigated.

**Diagnosis**—Food allergy was investigated and diagnosed in only four (9%) of the 44 children whose parent/caregivers reported AFR symptoms and eczema. FA was not investigated in 40 (91%) of the 44 children whose parent caregiver reported AFR symptoms, including the two who were hospitalised.

**Discussion**

Self-reported FA has become more frequent in developed countries in recent years.17,18 The reasons for the increase are still poorly understood.19 There have been no previous community based studies of AFR/FA in New Zealand, yet anecdotal evidence suggests that AFR/FA is a problem in the community, which is not being addressed. Patients calling the Allergy New Zealand helpline and those on internet forums indicate they are uncertain where to seek further assistance.20

The health status of children attending Plunket clinics has been investigated previously,21 however there are some limitations to this approach. Attendance at Plunket clinics wane after second and subsequent pregnancies, therefore it will not be possible to measure the occurrence of AFR in younger siblings by this method. Plunket nurses report transportation and language barriers are problems which impede clinic attendance and will further affect selection bias. Plunket data of attendance at the clinics in this study indicated 30-80% of contacts are visited at home. Future surveys of AFR in children seen by Plunket nurses require procedures to include those visited at home.

This study had a 65% participation rate. It is uncertain whether the children who did not participate in the study had a similar rate of food related symptoms. If
parent/caregivers of the children who experienced FA symptoms were more likely to participate in the study, surveys of this nature may overestimate the prevalence of AFR in Plunket attendees.

Parents/caregivers may inaccurately recall the food related to an adverse reaction, since FA affects multiple organ systems including the gut, skin, respiratory tract, and causes anaphylaxis, while some IgE mediated reactions are delayed, particularly eczema. Therefore parental reporting of FA can be higher compared to that confirmed by food challenges (11.8% compared to 2.5 %). This study was based on self-report (no clinical or lab confirmation). For this reason we limited the probable FA case definition to try to capture reactions most likely to be FA and we asked about reactions within 2 hours of food consumption.

Personal interview is a common method of collecting original data for epidemiological studies. Interviewers may reduce errors by encouraging a better response rate. Alternatively errors may be introduced by their presence, manner, method of administration, or method of recording responses. The interviewer for this study was an experienced dietary interviewer. The questionnaire achieved its purpose of collecting data about reported adverse reactions to food experienced within two hours of consumption and about the presence of eczema. Most (80.9%) parents/caregivers found the questionnaire very easy to complete and the remaining (19.1%) found it easy to complete. However, the ability of the questionnaire to capture the presence of food allergy has not yet been assessed. Clinical and laboratory validation is planned for future research.

Despite these limitations, our preliminary data suggest that AFR symptoms are a public health concern in NZ. Given that 28/110 (25.5%) children experienced AFR within two hours of food consumption and another 10/110 (9%) had worsening eczema symptoms, FA may be at least as common as reported overseas. We cannot however validate this inference because no allergy testing or food challenges were undertaken in this study.

Interestingly 18/44 (40%) children experienced an adverse reaction after consuming breast milk. This study was not designed to investigate the association of maternal dietary intake during lactation with FA symptoms. The benefits of dietary restriction during breastfeeding are still unknown as studies to date have been limited by their small size and methodological considerations. However the data suggests the effect of maternal diet in lactation on the development of food allergy requires further investigation.

Parents may have difficulty identifying the cause of an adverse reaction as 5/44 (11.4%) could not associate reactions with a food. Alternatively it is possible that food was not the cause. Clinical assessment and diagnostic testing are required to prevent ongoing morbidity.

Our most important finding is that only 4/44 (11%) of children with reported AFR symptoms appear to have been clinically evaluated and undergone diagnostic testing. All four children were found to be sensitised to food allergens. Clinical evaluation and diagnostic testing are crucial in identifying food allergen(s). Children may be allergic to multiple foods. For example, one child reacted to a commercial baby cereal and upon clinical evaluation had a positive skin prick test to egg and a positive specific
IgE results to egg and peanut. Without testing, the first manifestation of peanut allergy may have been anaphylaxis.

It is also of concern that two children were admitted to hospital with probable systemic allergic reactions to food and yet no testing was undertaken to identify food allergy. If these children have food allergies, they remain at risk for continued and possibly severe reactions.

Lack of clinical consultation and laboratory confirmation of FA can also result in unnecessary elimination diets, which can pose nutritional sequelae for the children. Unsupervised avoidance diets, when followed by breastfeeding mothers is also a risk for nutrient deficiency for both mother and infant. Failure to thrive is commonly seen in children experiencing FA as a result of multiple foods being removed from their diet. It is very important for children with a defined food allergy to consume a nutritionally restricted but balanced diet and for their growth to be monitored.

In this study 6% (6/89) parent/caregivers reported modifying their children’s diets without advice from a physician or dietitian. Furthermore, three breastfeeding mothers in this study eliminated foods from their own diet to improve their infants’ symptoms. Moreover, none of the families including the four children diagnosed with FA had received advice from a dietitian. The study did not investigate the reason for this. The inclusion of a paediatric dietitian in the health professional team managing their FA should be an integral part of their treatment.

This study also confirms previous international data that AFR symptoms are not confined to children of European origin. Our study has shown that AFR symptoms occur in ethnic minorities as well as European children. In this study, the frequency of AFR was 8/21 (38%) in Māori children. None of the Māori children with reported AFR symptoms was clinically investigated. Māori have on average the worst health status of any ethnic group in New Zealand. For example Māori experience greater morbidity associated with asthma. Our study suggests AFR could be a significant source of morbidity in Māori children and further investigation is required to reduce health disparities.

The frequency of children with reported AFR was also high for Pacific peoples (Samoan, Cook Island Maori, Tongan, Niuean) (12/24, 50%), and Asian ethnicities (Chinese, Indian) (10/20, 50%). These data suggest FA impacts on the health status of all ethnic groups in NZ.

Among the children in this study 16% (18/110) had physician diagnosed eczema compared to a previous New Zealand study whose authors reported a physician diagnosed prevalence of 14% among children aged 0 to 14 years. Our finding may be of concern because up to 40% of children with eczema can have a food allergic trigger. However our observation is limited by the small sample size and the possibility of selection bias since only 65% of possible children participated.

More than half of the children (18/33, 54.5%) with eczema were being treated by a physician. Nearly a third (5/18, 27.8%) of these children continued to have problems with their skin despite being prescribed topical therapy. One possible explanation for this observation is undiagnosed FA. Without testing, allergic triggers for eczema could not be identified in these participants. The results from this study suggest that further investigation of FA as a cause of eczema is warranted.
In conclusion, results from this preliminary study suggest that AFR may be an important public health concern for diverse ethnic groups in NZ. Lack of medical assessment and diagnostic testing may place a substantial proportion of children with AFR symptoms at continued risk for reactions. Further development and validation of the tools to research AFR in New Zealand is required. There is an urgent need to investigate the epidemiology, diagnosis, and prevention of FA in New Zealand to reduce morbidity, improve child health, and reduce the burden to health costs.

**Competing interests:** None.

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**References:**


Inappropriate trace element testing in the Auckland region

David Song, Barry Palmer, Stephen du Toit, James S Davidson

Abstract

Aim To understand recent changes in trace element test usage in the Auckland region of New Zealand.

Methods Laboratory records of trace element tests between 2004 and 2008 were analysed. A questionnaire was sent to a frequent requestor group to elicit reasons for requesting trace element tests.

Results The annual number of trace element test requests increased by 3.5-fold over the study period. The increase was largely due to a 2.8-fold increase in serum copper, a 3.8-fold increase in serum zinc, and a 3.4-fold increase in serum selenium tests. Most of the increase was accounted for by a small number of requestors, mainly general practitioners. An outlier group of 24 requestors was identified who were responsible for ordering 55% of serum copper, 61% of serum zinc, 63% of serum selenium and 66% of blood mercury tests in the last year of the study. Responses to the questionnaire suggest that among the outlier group the reasons for requesting serum zinc, copper and selenium tests are not evidence-based.

Conclusion The majority of trace element tests performed in the Auckland region appear to be non-evidence-based, and represent a significant wastage of public laboratory resources. This suggests that laboratories could achieve significant savings in expenditure by clearly defining appropriate indications for performing trace element tests.

The trace elements zinc, copper and selenium are necessary for many biochemical functions. Deficiencies of these elements occur in the settings of malnutrition and malabsorption, and measurement of serum levels are useful in the management of patients with gastrointestinal disorders and especially in parenteral nutrition. Measurement of serum and urine copper levels are also useful in the diagnosis and management of Wilson's disease and in rare genetic disorders of copper metabolism. These tests are also of value in occasional cases of zinc, copper and selenium poisoning.

Measurement of whole blood and urine mercury are of value in monitoring workplace exposure and in mercury poisoning.

Unless there is a high pre-test probability of deficiency (i.e. a predisposing condition such as gastrointestinal disease), or toxicity (e.g. workplace exposure or suspicion of Wilson's disease) it has not generally been considered useful to measure serum copper, zinc, selenium or blood mercury in patients in general practice.

Despite this, we report a substantial increase in trace element testing by general practitioners in the Auckland region from 2004 to 2008, and describe the results of a questionnaire aimed at understanding the reasons for this increase.
Methods

Labplus is the tertiary referral laboratory for trace element testing for Auckland City Hospital and other hospitals in the Auckland region of New Zealand, and serves a population of approximately 1.5 million. Serum zinc, serum and urine copper, serum selenium, whole blood mercury and urine mercury results from 1 September 2004 to 31 August 2008 were retrieved from the laboratory information system and analyzed to identify trends in trace element testing. These were plasma and serum samples submitted in the course of routine patient care, and had been collected at varying times of day. Serum and plasma samples were treated interchangeably and are referred to collectively as "serum" in this paper.

Serum zinc and copper were measured by flame atomic absorption spectroscopy using a GBC Avanta instrument with an air-acetylene flame. Serum selenium and urine copper were measured by graphite furnace atomic absorption spectroscopy using a Perkin Elmer 4110ZL instrument, which was also used to measure urine mercury and whole blood mercury by flow injection cold vapour atomic absorption spectroscopy. The laboratory participates in the external quality assurance program for trace elements run by Quality Control Technologies Pty Ltd and was accredited by International Accreditation New Zealand to standard ISO15189 during the period of the study.

A questionnaire was sent to the top 24 requestors of trace elements tests, excluding gastroenterologists and surgeons. The participants were told that the laboratory had noted a substantial increase in trace element testing and the purpose of the questionnaire was to determine the reasons for this increase. Participants were asked about their reasons for requesting serum zinc, copper and selenium levels. Several reply options were provided, as well as space for free text replies. Participants were also asked to describe their type of practice as: conventional, holistic or integrative, anti-ageing medicine or "other".

Additional information about the type of medical practice of the top 24 requestors was obtained from material emanating from the practitioners themselves: the names of the practices, their websites and letterheads.

An estimate of the number of general practitioners in the Auckland region was provided by the Medical Council of New Zealand.

Results

Between 2004 and 2008 the annual number of trace element test requests increased by 3.5-fold (Table 1). The increase was largely due to a 2.8-fold increase in serum copper, a 3.8-fold increase in serum zinc, and a 3.4-fold increase in serum selenium tests.

<table>
<thead>
<tr>
<th>Year</th>
<th>Serum copper</th>
<th>Urine copper</th>
<th>Serum zinc</th>
<th>Blood mercury</th>
<th>Urine mercury</th>
<th>Serum selenium</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004–2005</td>
<td>794</td>
<td>119</td>
<td>2082</td>
<td>0</td>
<td>62</td>
<td>242</td>
<td>3299</td>
</tr>
<tr>
<td>2005–2006</td>
<td>1089</td>
<td>101</td>
<td>2922</td>
<td>77</td>
<td>72</td>
<td>314</td>
<td>4575</td>
</tr>
<tr>
<td>2006–2007</td>
<td>1763</td>
<td>101</td>
<td>5069</td>
<td>275</td>
<td>72</td>
<td>932</td>
<td>8212</td>
</tr>
<tr>
<td>2007–2008</td>
<td>2223</td>
<td>84</td>
<td>7826</td>
<td>375</td>
<td>86</td>
<td>821</td>
<td>11415</td>
</tr>
</tbody>
</table>

The distribution of test requests was markedly skewed, with a relatively small number of requestors being responsible for the majority of requests. The top 27 requestors comprised 21 general practitioners, 2 surgeons, one gastroenterologist, one psychiatrist, one paediatrician and one pain specialist. The surgeons and gastroenterologist were excluded from the "frequent requestor" outlier group because
their patients are likely to have malabsorption or parenteral nutrition in whom trace element tests are appropriate. The remaining 24 requestors are referred to as the "top 24".

Figures 1–4 show that the number of tests ordered by the top 24 increased markedly between 2004 and 2008. In the 2007-2008 year, the top 24 requestors were responsible for ordering 55% of serum copper, 61% of serum zinc, 63% of serum selenium and 66% of blood mercury tests.

Although responsible for ordering the majority of trace element tests, these 24 medical practitioners represent only approximately 1.5% of the 1566 GPs practising in the Auckland region.

The distribution of serum zinc results of patients from the top 3 zinc requestors was not different from that expected for a normal population, with few results outside the reference interval (Figure 5). The distribution showed a 2.5\textsuperscript{th} percentile of 9.1 µmol/L, which is very similar to a healthy US population from the NHANES II study, where the 2.5\textsuperscript{th} percentiles for non-fasting serum zinc were 9.0 µmol/L in females and 9.3 µmol/L in males.\textsuperscript{1}

The distribution of all serum zinc results requested during that year was similar, except for a larger tail of low serum zinc results (Figure 5). Analysis of these low serum zinc results indicated that they largely represent patients with hypoproteinemia and/or gastrointestinal disorders, many of whom were hospitalised.

**Figure 1. Serum copper tests performed during the study period**
Figure 2. Serum zinc tests performed during the study period

Figure 3. Serum selenium tests performed during the study period
Figure 4. Whole-blood mercury tests performed during the study period. The test was introduced during the 2005-6 year.

Figure 5. Distribution of serum zinc levels in patients from the top 3 requestors (dotted line) compared with all patients (solid lines), for the 2007-2008 year of the study. Vertical dotted lines show the reference interval.
Figure 6. Distribution of serum selenium levels in patients from the top 3 requestors (dotted line) compared with all patients (solid lines), for the 2007-2008 year of the study. Vertical dotted lines show the reference interval.

The distribution of serum selenium results of patients from the top 3 selenium requestors showed few results outside the reference interval (fig. 6). Low selenium results were largely found in hospitalised patients and in requests from gastroenterologists. There was a long tail of high serum selenium results (>2.0 umol/L) which is likely to represent people on selenium supplements.

The distribution of serum copper and whole blood mercury results of patients from the top 3 requestors of these tests were not different from that expected for a healthy population, with few results outside the reference interval, and also did not differ from the distribution of results from all requestors (data available on request).

Questionnaire—Of the 24 requestors who were sent the questionnaire, 15 responded. Tables 1 and 2 show the percentage of the 15 respondents giving the indicated responses.
Table 2. Responses to the questionnaire

<table>
<thead>
<tr>
<th>Reasons for requesting serum zinc</th>
<th>67%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc deficiency is common in New Zealand</td>
<td></td>
</tr>
<tr>
<td>Zinc deficiency is a common cause of depression</td>
<td>13%</td>
</tr>
<tr>
<td>Zinc deficiency is associated with low immune function</td>
<td>67%</td>
</tr>
<tr>
<td>To see if zinc supplements are required</td>
<td>47%</td>
</tr>
<tr>
<td>To monitor the patient’s use of zinc supplements</td>
<td>33%</td>
</tr>
<tr>
<td>To enable calculation of the zinc/copper ratio</td>
<td>27%</td>
</tr>
<tr>
<td>To optimise my patient’s health and not necessarily to detect deficiency or toxicity</td>
<td>27%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reasons for requesting serum copper</th>
<th>33%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc/copper ratio is useful</td>
<td></td>
</tr>
<tr>
<td>To test for Wilson’s disease</td>
<td>13%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reasons for requesting serum selenium</th>
<th>40%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Selenium deficiency is common</td>
<td></td>
</tr>
<tr>
<td>Selenium supplementation will/may reduce the incidence of certain cancers</td>
<td>33%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of practice (self-described)</th>
<th>33%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conventional general practice</td>
<td></td>
</tr>
<tr>
<td>Holistic or integrative or alternative medical practice</td>
<td>27%</td>
</tr>
<tr>
<td>Anti-aging medicine</td>
<td>13%</td>
</tr>
<tr>
<td>Interest in autism/ADHD</td>
<td>20%</td>
</tr>
</tbody>
</table>

The most common reasons for serum zinc testing cited by the questionnaire respondents were the notions that zinc deficiency is common in New Zealand and that this is associated with low immune function. Consistent with this, the use of zinc supplements appears to be common among this group of practitioners. The belief that the serum zinc/copper ratio is clinically useful was expressed by a third of the respondents. None of the respondents cited gastrointestinal disease or parenteral nutrition as reasons for testing trace elements.

Additional information on the type of medical practice was obtained from the practitioners' websites and letterheads. Thirteen of the top 24 requestors (54%) described themselves as practising holistic, integrative, anti-ageing, complementary, or anthroposophic forms of medicine, chelation therapy, Mind-Body medicine, or "bio-identical" hormone therapy. Two others described themselves as having a special interest in "biomedical" treatments for autism.

Discussion

Our findings show a marked increase in laboratory testing for the trace elements zinc, copper, selenium and mercury from 2004 to 2008. To understand the reasons for this increase, and because of concern that it might reflect inappropriate laboratory test requesting practice, we analysed the distribution of test requests and sent a questionnaire to the most frequent requestors.

The main findings were (a) that the majority of the increase was attributable to a small number of requestors, and (b) that the requestors were largely identified with unconventional (alternative) types of medical practice.

A belief that zinc deficiency is common in the general population of New Zealand appears to be the reason for the majority of serum zinc tests. It seems likely that the serum zinc level may be used as an indicator of the need for zinc supplements. Is
there any evidence that zinc deficiency is common in this population, or that routine serum zinc measurements are justified?

Zinc balance in humans is maintained by a homeostatic mechanism which regulates its absorption and excretion.\textsuperscript{2,3} When dietary zinc intake is low, zinc absorption can increase to nearly 100\%, while urinary and faecal excretion fall to low levels.\textsuperscript{4} This adaptation allows zinc balance to be maintained with Zinc intakes as low as 2–3 mg/day.\textsuperscript{5–7}

Zinc deficiency occurs when this homeostatic mechanism fails. This may occur in patients with malabsorption or chronic diarrhoea, if total parenteral nutrition (TPN) is given without adequate zinc supplementation, in malnutrition or eating disorders, in patients receiving chelation therapy (e.g. for iron overload) and in acrodermatitis enteropathica, a rare inherited defect of intestinal zinc transport.\textsuperscript{2,8} Severe zinc deficiency is associated with stunted growth, decreased immunity, skin lesions and poor wound healing.\textsuperscript{9,10}

Since a wide range of foods including meat, fish, shellfish, nuts, seeds, legumes and whole-grain cereals are rich in zinc,\textsuperscript{10} deficiency does not occur in people who consume a balanced diet and have normal gastrointestinal function. Vegetarians may be theoretically more likely to become deficient because zinc from plant sources is less bioavailable due to the presence of phytic acid which inhibits its absorption.\textsuperscript{11} However in practice serum zinc levels are similar in vegetarians and nonvegetarians, and adverse effects from zinc deficiency have not been demonstrated in vegetarians in developed countries.\textsuperscript{11} In a survey of risk of zinc deficiency at the population level, New Zealand is classified as a low risk country.\textsuperscript{9}

A separate but related question concerns the utility of serum zinc concentration as an index of zinc status. Plasma zinc falls in severe zinc deficiency but it is a poor marker of marginal zinc deficiency.\textsuperscript{1,12} During experimental zinc depletion, serum zinc concentrations do not fall consistently unless the zinc intake is below 3 mg/day.\textsuperscript{13} The utility of serum zinc measurements is further compromised because the serum zinc level is influenced by other factors unrelated to zinc status. As serum zinc is 98\% protein-bound, zinc levels are low in hypoproteinaemic patients, without indicating zinc deficiency.\textsuperscript{14} In addition, the proteins to which zinc is bound (albumin and alpha-2 macroglobulin) are negative acute phase reactants: their concentrations decrease in response to inflammation.\textsuperscript{1} For this reason low serum zinc is a non-specific finding in a variety of disease states and does not indicate zinc deficiency in these settings.\textsuperscript{15,16}

The notion that the serum copper/zinc ratio is clinically useful appears to be the reason for the increase in copper requests. An increase in the copper/zinc ratio has been reported in numerous disease states.\textsuperscript{15,17–21} The increase in the serum copper/zinc ratio in many diseases occurs because serum copper has a positive acute phase response (increases) and serum zinc has a negative acute phase response (decreases) in inflammatory states. Thus the serum copper/zinc ratio has no diagnostic value other than as a non-specific marker of disease.

Plasma zinc levels are often low in major depression, and there is evidence that this may be due to activation of an inflammatory response in this disorder.\textsuperscript{16,22} There is no convincing evidence that zinc deficiency is associated with depression, or that measuring serum zinc is of any benefit to these patients.
A large number of zinc, copper and mercury tests are requested on children with autism spectrum disorder. The belief that biochemical imbalances including trace elements are the cause of autism, and that their correction by means of supplements and/or chelation therapy can be used to treat the disorder, is known as the "Biomedical approach" to autism. This approach has been thoroughly discredited, and there is no convincing evidence for any trace element imbalance in autism. Current guidelines for the investigation of autism spectrum disorder do not include the measurement of trace elements.

Prior to 1990 the selenium status in the New Zealand population, especially in the South Island, was low due to the low selenium content of our soils, but has since improved, while remaining lower than some countries. However, clinically significant selenium deficiency has not been found in the general population of New Zealand, and is confined to patients with malnutrition or malabsorption.

Epidemiological studies which demonstrated an association between low serum selenium levels and increased incidence of some cancers and mortality as well as an early randomised controlled trial suggested that selenium supplements could reduce the incidence of prostate and other cancers. This led to a major randomised controlled trial which has conclusively shown that selenium supplementation does not produce any reduction in prostate cancer or cancer of any type.

The reasons for requesting blood mercury tests were not examined in the questionnaire. However, information provided on the request forms, written information emanating from the practitioners, as well as communications between one of the authors (J.D.) and the requestors suggests that the main reasons were a belief that mercury toxicity, particularly due to amalgam dental fillings, is an important cause of fatigue, cognitive decline, memory loss, Alzheimer's disease, depression and autism. In fact there is no convincing evidence that mercury has any causal relationship to any of these conditions.

The marked increase in trace element testing in Auckland mirrors the growth in use of dietary supplements and alternative/complementary medicine in New Zealand and reflects the type of medical practice of the most frequent trace element test requestors. The majority of zinc, copper, selenium and mercury tests are requested for reasons which are not evidence-based. The inappropriate laboratory testing of trace elements represents a significant waste of public sector health care resources. The unit costs of trace element tests in this laboratory ranged from NZ$26 for serum zinc to NZ$49 for whole blood mercury. This suggests that laboratories could achieve significant savings in expenditure by clearly defining appropriate indications for performing trace element tests.

Competing interests: None.

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References:
Does health status matter for the risk of injury?

Ruth Cunningham, Kristie Carter, Jennie Connor, Jackie Fawcett

Abstract

Background Poor health status as a risk factor for injury has not been well elucidated. This study aims to investigate the overall risk of injury and the association between health status and risk of injury in New Zealand.

Methods We used data from the Survey of Families, Income and Employment (SoFIE) (n=18,955). The outcome measure was self-report of “an injury in the past 12 months which stopped you doing your normal activities for more than 7 days. Health status variables were prior self-rated health and number of comorbid conditions. Logistic regression was used to quantify the association adjusting for confounders, overall and for men and women separately.

Results 12% of respondents reported an injury in the past 12 months. Injury was more common in men, young people, and Māori respondents. A linear relationship between worsening health status and increasing injury risk was evident. A strong crude relationship was found of poor self-rated health and the risk of subsequent injury (OR 1.72 95%CI 1.48–2.01), and between 2 or more comorbid diseases and injury (OR 1.70 95%CI 1.47–1.96). The odds ratio was unchanged after adjustment for confounders and health status variables.

Conclusions Injuries which disrupt usual activities for more than a week are common and represent a significant impact on society from injury which is not captured by health care statistics. People with pre-existing medical conditions are at increased risk of injury and should be targeted for injury prevention activities.

Injury is a leading cause of premature death in New Zealand (NZ), with about 1100 deaths resulting from injury annually. Injury is also a cause of significant morbidity, which has implications for health services, but also for society more broadly in terms of the costs of time off work or school and the care required by those who are incapacitated by injury.

The degree of morbidity caused by injury can be characterised in a variety of ways, depending on both the source of the information and severity threshold used. For example the recent report *The Impact of Injury* used hospitalisation data to estimate morbidity, and the report focused only on injuries with significant threat to life. The NZ Health Survey on the other hand report the number of self-reported injuries for which “any” medical attention had been sought.

Both of these measures capture only those who access health care services for injuries. Not all of those incapacitated by injury for a significant period of time will access health services. Alternative measures of morbidity that are independent of health services access are therefore useful in characterising the societal burden of injury.
For effective injury prevention policy it is also important to understand the characteristics which are associated with an increased risk of injury. Several studies have found that poor health or comorbid disease is an important risk factor for injury. However much of the evidence relates specifically to an increased risk of falls in the elderly with underlying medical conditions, and there is limited evidence about the relationship between health status and risk of injury in other age groups. Moreover, health service utilisation data is often used to measure both comorbidity burden and injury occurrence, which may result in an overestimate of the strength of the relationship. As injury can cause a decline in health status itself, it can be difficult to establish the direction of causality where data is cross-sectional.

This study uses data from a longitudinal survey to examine the relationship between self-reported health status one year prior to injury assessment and comorbidity and the risk of injury. The injury outcome used was self-report of injury causing disruption to normal activities for more than a week. The use of this measure provides an opportunity to estimate the degree to which injury causes functional disruption in NZ.

The aims of this study were to:

- Estimate the incidence of injury causing disruption to activities for more than a week in the NZ population.
- Test the hypothesis that poor self rated health and diagnosed medical conditions are independently associated with an increased risk of subsequent injury.
- Test whether the association between health status and injury risk differs by sex.

Methods

Data

This study utilised data from the Survey of Families, Income and Employment (SoFIE). SoFIE is a representative household panel longitudinal survey of the usually resident population living in private dwellings in NZ, conducted between 2002 and 2010. The SoFIE sample comprised approximately 11,500 private households (response rate of 77%) with over 22,000 adults (aged 15 years or older) responding in Wave 1 (October 2002 to September 2003), reducing to just over 20,000 in Wave 2 (91%) and over 18,000 in Wave 3 (83% of Wave 1 responders).

Annual face to face interviews were used to collect information on income, employment, education, household and family status, demographic factors, and self-rated health. In Wave 3 an additional health module was asked, collecting information on health related quality of life, psychological distress, injury and history of comorbid diseases. Written consent was also requested from participants to link their records with cancer registrations and hospitalisations data from the NZ Health Information Service (NZHIS).

The current analysis used the first three Waves of SoFIE data and was restricted to original sample members who responded in Wave 3 (asked in 2004–05), aged 15 years or older (N=18,950: SoFIE data Wave 1 to 4 Version 6 was used).

Measures

Injury—The health module included four injury-related questions:

- In the last 12 months have you had an injury that stopped you from doing your usual activities for more than a week? (yes/no)
If yes:
- In the last 12 months did you have more than one injury? (yes/no)
- What type of injury was that? (if more than one injury, the most recent that stopped you doing your activities for more than a week) (1. injury from traffic accident; 2. sports injury; 3. other type of injury)
- Where did that injury occur? (1. at home; 2. at work; 3. at another place).

Demographic and socioeconomic variables—Demographic and socioeconomic variables were treated as confounders in this analysis. The demographic variables age, sex, and prioritised ethnicity were taken from the Wave 3 interview. Socioeconomic variables were taken from the Wave 2 interview (prior to the injury question), and were equivalised household income categorised into quintiles, labour force involvement, the highest level of education, and the NZ Deprivation (NZDep2001) index. However as these variables were not found to have a significant effect on injury risk on initial regression modelling they were not included in the final model.

Health status—Self-rated health, asked at Wave 2, was used as an indicator of health status prior to injury (coded as excellent, very good, good or fair/poor). The number of chronic diseases reported at Wave 3 was grouped into a variable with levels 0, 1–2, and more than 2 comorbidities. Participants were asked “have you ever been told by a doctor that you have…?” and the chronic diseases asked about were asthma, hypertension, high cholesterol, heart disease, diabetes, stroke, migraine and psychiatric disorders (depression and schizophrenia). Year of diagnosis was also recorded, and as most had been diagnosed more than a year prior to the Wave 3 interview, these were assumed to be prior to the injury.

Statistical analysis
All analyses were conducted using SAS v8.2 software within the Statistics NZ data lab, Wellington. The numbers of respondents are random rounded to the nearest multiple of five, with a minimum value of 10, as per Statistics NZ confidentiality protocol. Cross-tabulations were used to demonstrate the associations of demographic, socioeconomic and health status variables with risk of injury. Logistic regression analyses were used to estimate the association of health status with injury after adjustment for demographic confounders (age, sex and ethnicity). The analyses were also stratified by sex to investigate the possibility of effect modification by sex. The Wald test for heterogeneity was used to test for difference in the regression results between males and females.

Results
Overall, 12.5% of the SoFIE population reported at least one injury during the 12 months before the Wave 3 interview that prevented them from doing their usual activities for more than a week.

Table 1 shows the characteristics of the SoFIE population, and of those reporting injury, and the results of multivariate analysis of the odds of injury.

The first column shows the gender, age, ethnicity and health status distribution of the SOFIE population. Column two shows the characteristics of those who reported an injury. The proportion reporting injury was greater in males (14%) than females (12%), greater among Māori (15%) than among European (13%), Pacific (9%) and Asian (7%) ethnic groups, and greater at younger and older ages than in middle age.

The proportion reporting injury was also examined by socioeconomic variables* (data not shown), and did not vary markedly across socioeconomic strata, with the exception of labour market status, where injury was most common amongst those in employment.

*Footnote: equivalised household income categorised into quintiles, labour force involvement, the highest level of education, and the NZ Deprivation (NZDep2001) index.
Table 1 Characteristics of those reporting injury, and odds of injury by demographic and health status factors

<table>
<thead>
<tr>
<th>Demographic and health characteristics</th>
<th>All N=18,265</th>
<th>Injury n=2360</th>
<th>Univariate</th>
<th>Odds ratios adjusted for age, sex, ethnicity</th>
<th>Fully adjusted*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>8395</td>
<td>1185</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Female</td>
<td>9870</td>
<td>1175</td>
<td>0.82 (0.75–0.90)</td>
<td>0.82 (0.76–0.90)</td>
<td>0.80 (0.73–0.87)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15–24</td>
<td>2775</td>
<td>465</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>25–34</td>
<td>2575</td>
<td>320</td>
<td>0.71 (0.61–0.83)</td>
<td>0.71 (0.61–0.83)</td>
<td>0.68 (0.58–0.80)</td>
</tr>
<tr>
<td>35–44</td>
<td>3640</td>
<td>490</td>
<td>0.78 (0.68–0.89)</td>
<td>0.77 (0.67–0.89)</td>
<td>0.72 (0.62–0.83)</td>
</tr>
<tr>
<td>45–54</td>
<td>3410</td>
<td>405</td>
<td>0.68 (0.59–0.78)</td>
<td>0.67 (0.58–0.78)</td>
<td>0.59 (0.51–0.68)</td>
</tr>
<tr>
<td>55–64</td>
<td>2705</td>
<td>290</td>
<td>0.60 (0.51–0.70)</td>
<td>0.58 (0.50–0.68)</td>
<td>0.47 (0.40–0.55)</td>
</tr>
<tr>
<td>65+</td>
<td>3155</td>
<td>390</td>
<td>0.71 (0.61–0.82)</td>
<td>0.69 (0.59–0.79)</td>
<td>0.49 (0.42–0.57)</td>
</tr>
<tr>
<td>Prioritised ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ/European</td>
<td>14,585</td>
<td>1920</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Maori</td>
<td>1970</td>
<td>295</td>
<td>1.17 (1.02–1.33)</td>
<td>1.11 (0.97–1.27)</td>
<td>1.05 (0.92–1.21)</td>
</tr>
<tr>
<td>Pacific</td>
<td>790</td>
<td>75</td>
<td>0.68 (0.54–0.87)</td>
<td>0.65 (0.51–0.83)</td>
<td>0.65 (0.51–0.83)</td>
</tr>
<tr>
<td>Asian/Other</td>
<td>920</td>
<td>65</td>
<td>0.51 (0.39–0.66)</td>
<td>0.48 (0.37–0.61)</td>
<td>0.49 (0.38–0.64)</td>
</tr>
<tr>
<td>Self-rated health (Wave 2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent</td>
<td>7005</td>
<td>785</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Very good</td>
<td>5885</td>
<td>730</td>
<td>1.12 (1.00–1.24)</td>
<td>1.18 (1.06–1.32)</td>
<td>1.14 (1.02–1.27)</td>
</tr>
<tr>
<td>Good</td>
<td>3770</td>
<td>555</td>
<td>1.35 (1.21–1.52)</td>
<td>1.53 (1.35–1.73)</td>
<td>1.41 (1.25–1.60)</td>
</tr>
<tr>
<td>Fair/Poor</td>
<td>1605</td>
<td>285</td>
<td>1.69 (1.46–1.96)</td>
<td>2.01 (1.72–2.35)</td>
<td>1.73 (1.47–2.03)</td>
</tr>
<tr>
<td>Index of chronic comorbid disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No comorbid</td>
<td>8310</td>
<td>925</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>1–2 comorbid</td>
<td>8145</td>
<td>1120</td>
<td>1.27 (1.15–1.39)</td>
<td>1.35 (1.22–1.48)</td>
<td>1.27 (1.15–1.40)</td>
</tr>
<tr>
<td>&gt;2 comorbid</td>
<td>1810</td>
<td>310</td>
<td>1.65 (1.43–1.90)</td>
<td>1.96 (1.69–2.28)</td>
<td>1.66 (1.42–1.94)</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, ethnicity, self-rated health, index of comorbid disease.
After adjustment for age, sex and ethnicity (column 4) the relationship between age and sex and the odds of injury did not change, but the increased odds of injury in Māori was no longer significant. After further adjustment for health status variables (final column) the odds of injury in Māori attenuated further and a gradient of reducing odds of injury with increasing age became apparent.

Table 2 shows the distribution of type and place of most recent injury. About 32% of injuries occurred at home with 21% at work and 47% at other places. 28% of injuries were sports injuries, 4% traffic injuries, and 68% had another external cause.

Table 2. Type and location of (most recent) injury in the 2360 people who reported an injury in the past 12 months

<table>
<thead>
<tr>
<th>What type of injury</th>
<th>At another place</th>
<th>At home</th>
<th>At work</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>col %</td>
<td>row %</td>
</tr>
<tr>
<td>Other injury</td>
<td>405</td>
<td>36.49</td>
<td>25.31</td>
</tr>
<tr>
<td>Sports injury</td>
<td>635</td>
<td>57.21</td>
<td>94.78</td>
</tr>
<tr>
<td>Traffic injury</td>
<td>70</td>
<td>6.31</td>
<td>77.78</td>
</tr>
<tr>
<td>Total</td>
<td>1110</td>
<td>750</td>
<td>500</td>
</tr>
</tbody>
</table>

*col% = column percent (over place of injury); row% = row percent (over type of injury).

The bottom half of Table 1 presents the relationship between health status and risk of injury. There was an increasing risk of injury with worsening health and an increasing number of chronic conditions. The unadjusted (crude) odds ratios (column 3) show that less than excellent self-rated health and the presence of comorbid conditions were significantly associated with an increased risk of injury.

The crude odds of injury were also increased with the presence of each chronic condition asked about (data not shown). The fourth column shows the odds ratios for the association after adjustment for age, sex and ethnicity, and it can be seen that the association strengthens after adjustment for these factors (this will be partly explained by effect modification present for age and sex, see results below). The final column shows the odds ratio for injury after adjustment for age, sex, ethnicity and mutual adjustment for health status variables.

The relationship between health status and the risk of injury attenuates after mutual adjustment for health status variables, and are similar to the unadjusted estimates. This attenuation suggests that self-reported health is correlated with reporting comorbid disease. However, both health status variables remained significantly associated with injury risk suggesting that they are also capturing different dimensions of health which are both important for injury risk. Those reporting fair or poor health at Wave 2 had a 74% increase in the odds of injury in the subsequent year compared to those reporting excellent health, while those with two or more chronic conditions had a similarly increased odds compared to those with no comorbidities.

The results of stratified analysis by sex are presented in Table 3, as crude odds ratios, after adjustment for age and ethnicity, and then after mutual adjustment for health factors. The associations of age, ethnicity and health status with injury all vary by sex.
Table 3. Logistic regression results; odds of injury by sex

<table>
<thead>
<tr>
<th>Demographic characteristics (Wave 3)</th>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Univariate</td>
<td>Adjusted for age &amp; ethnicity</td>
</tr>
<tr>
<td></td>
<td>Full Multivariate*</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15-24</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>25-34</td>
<td>0.69 (0.56–0.85)</td>
<td>0.68 (0.55–0.84)</td>
</tr>
<tr>
<td>35–44</td>
<td>0.68 (0.57–0.83)</td>
<td>0.68 (0.56–0.82)</td>
</tr>
<tr>
<td>45–54</td>
<td>0.53 (0.44–0.65)</td>
<td>0.53 (0.43–0.64)</td>
</tr>
<tr>
<td>55–64</td>
<td>0.40 (0.32–0.50)</td>
<td>0.39 (0.31–0.49)</td>
</tr>
<tr>
<td>65+</td>
<td>0.47 (0.38–0.58)</td>
<td>0.46 (0.37–0.56)</td>
</tr>
<tr>
<td>Prioritised ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ/European</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Maori</td>
<td>1.40 (1.17–1.69)</td>
<td>1.24 (1.03–1.50)</td>
</tr>
<tr>
<td>Pacific</td>
<td>0.74 (0.52–1.05)</td>
<td>0.66 (0.46–0.94)</td>
</tr>
<tr>
<td>Asian/Other</td>
<td>0.57 (0.40–0.81)</td>
<td>0.49 (0.35–0.70)</td>
</tr>
<tr>
<td>Self-rated health (Wave 2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Very Good</td>
<td>1.00 (0.86–1.16)</td>
<td>1.12 (0.96–1.30)</td>
</tr>
<tr>
<td>Good</td>
<td>1.12 (0.95–1.32)</td>
<td>1.41 (1.18–1.68)</td>
</tr>
<tr>
<td>Fair/Poor</td>
<td>1.35 (1.09–1.68)</td>
<td>1.90 (1.50–2.40)</td>
</tr>
<tr>
<td>Index of comorbid disease</td>
<td>No comorbid</td>
<td>1</td>
</tr>
<tr>
<td>1–2 comorbid</td>
<td>1.14 (1.00–1.30)</td>
<td>1.29 (1.13–1.48)</td>
</tr>
<tr>
<td>&gt;2 comorbid</td>
<td>1.33 (1.07–1.65)</td>
<td>1.88 (1.49–2.37)</td>
</tr>
</tbody>
</table>

*Adjusted for age, ethnicity, self-rated health, index of comorbid disease.
In men the odds of injury are highest amongst the youngest age group, while for women the odds are highest for the oldest age group. Māori men have significantly greater odds of injury than European men, while Māori and European women have similar odds. Pacific and Asian women have a much lower odds of injury compared to European women than their male counterparts do compared to European men. After mutual adjustment for age and ethnicity, the odds of injury were similar.

There is a much stronger crude association of injury and poorer self-rated health in women than in men (Wald p-value=0.001), and of injury and comorbidity if there were more than two comorbidities reported (Wald p-value=0.002). After adjustment for age and ethnicity, the association in women remains similar, but strengthens in men. After mutual adjustment for health factors, the associations between injury and self-reported health and comorbidities attenuate, especially amongst women, and become more similar between men and women.

The odds ratio for injury in those with fair or poor health compared to excellent health was 1.86 (1.49–2.32) for women and 1.65 (1.29–2.11) for men (Wald p-value=0.456). The odds ratio for injury in those with 2 or more chronic conditions compared to no chronic conditions was 1.70 (1.38–2.10) for women and 1.61 (1.26–2.06) for men (Wald p-value=0.734).

**Discussion**

This study found that 12.5% of the study population reported an injury causing disruption of normal activities in the past 12 months. The risk of injury increased with worsening self-reported health and with increasing numbers of comorbid conditions. The relationship between both demographic and health status factors and injury odds varied by sex. Amongst men injury was less common with advancing age, while for women the opposite was true. The relationship between health status and odds of injury was stronger in women, although this difference was not significant after adjustment for confounding and health.

**Characterising the burden of non-fatal injury**

Most data on the burden of injury in NZ rely on health care access to identify injuries. Hospitalisation data are the most common source, and while these are useful for characterising life-threatening injuries, many less serious injuries will not be included that may still have significant impacts on people’s lives. In NZ, another data source is from the Accident Compensation Corporation (ACC) which funds health treatments (96% of claims), income replacement and other social support services for injured people. However, access to health services is generally required to enter an ACC claim, and detailed information is only collected on the very small proportion of claimants who require further support. As mentioned previously the NZ Health Survey also asks about injuries for which medical help had been sought.

There are several reasons why an alternative measure such as used in this study is useful. Firstly, it identifies a different subset of those injured, as some of those whose injury impacts on their normal activities for more than a week may not seek medical attention. Cryer and Langley found that only a small proportion of those who have extended time off work from an injury compensated by ACC had been admitted to hospital for their injury. Similarly, less than 6% of the respondents in this study
reporting an injury had a hospital admission for injury over the same period, and while a greater number may have sought primary care assistance, it is likely that not all will have.

The interruption of normal activities for at least a week represents a significant impact for society that is not captured in health care access statistics. By only counting injuries where health services are accessed, any associations found may be biased by the characteristics of those who are more likely to access health services. For example, if an ethnic group are less likely to access health services than other groups, then their rate of injury may be spuriously low when only counting medically attended injuries. This is less of a problem for life threatening injuries, where medical attention is almost universally sought.

As other estimates of injury burden use such different measures, the estimated proportion of the population experiencing injury over 12 months may not be comparable to those found in this study. For example the 1996/97 NZ Health Survey found that 27% of adults reported an injury requiring medical treatment in the preceding 12 months.3 ACC receives 1.7 million claims per year (equivalent to 40% of the NZ population, although this will include multiple claims), and pays 120,000 entitlement claims annually19 (approximately 3% of the population).

More interesting however is the comparison of demographic characteristics between injury causing functional impairment and other measures of injury burden. The 1996/97 NZ Health Survey also found that injury was more common amongst men than women and more common in young adults.3 Injury prevalence was similar between Māori and European groups and lower for Pacific and “other” groups. These results are in line with the findings in this paper. However Māori, Pacific and Asian NZers have lower ACC claim rates than Europeans of the same age, which may be due partly to problems with accessing the ACC system by these groups.20

**Health status and injury**

The finding of a higher risk of injury in those with poorer health status or chronic illness is similar to that found in other studies. A Finnish study found that the risk of adolescent injury was associated with poor self-reported health, pre-existing disability or chronic illness, depression, and the number of complaints suffered.21 A Canadian study also found that those hospitalised for injury had higher Charlson Comorbidity Index scores, and higher rates of hospital admissions and physician claims in the 12 months prior to injury than a matched non-injured cohort. However we are not aware of any previous study that has established this relationship using NZ data. Those who seek health services for injury may be more likely to seek health services in general. This may result in overestimation of the strength of the association.

The current study is more generic than others as it included all adults (aged 15 years or more) and all types of illness and injury. Many studies looking at the association between health status and injury risk have focused on specific populations e.g. investigating this association in the elderly10–12,22 or in children and adolescents.21,23 Other studies have focused on the association between specific types of chronic disease such as stroke, or diabetes10,12 and specific types of injury such as those due to falls.22,24 This study demonstrates that the association between injury and health status is present across the age spectrum and for injury overall. Only limited information
was collected on injury type in this study (sports, traffic and “other”) so analysis for specific types of injury was not performed. Small numbers also limited the analysis of injury risk for individual chronic diseases, and so this was not performed.

The current study asked specifically about being prevented from doing normal activities. The presence of chronic disease is associated with functional limitation and disability and is a predictor of poor functional outcome following injury. Therefore, it is possible that those who were in poor health or suffering from chronic illness prior to their injury would be more easily prevented from doing their normal activities than those in excellent health. It is likely that the association found here is stronger than it would be had a different measure of injury been used.

While those with fair or poor health had a substantially increased risk of injury compared to those with excellent health, it is worth noting that this group only represent 12% of all those reporting injury. Most injuries occur to people with excellent or very good self reported health status (who are the majority of the population).

**Strengths and limitations of the study**

The main strengths of this analysis are that it was conducted on a large population-based sample representative of NZ, and the temporality of the variables included in the model; self rated health and socioeconomic variables were taken from the Wave 2 interview (12 months prior to Wave 3), so prior to the period during which the injury occurred.

Attrition is a limitation in using longitudinal data. In Wave 3 of the SoFIE study, 83% of the original sample members were re-interviewed. When this is combined with the household response rate at Wave 1 of 77%, the effective response rate is 64%. However, the attrition within the SoFIE study is low compared with other population-based longitudinal panel surveys. Recent studies using longitudinal data have shown that attrition has minimal effect on certain exposure outcome relationships over time.

Another limitation of this study is that only limited information on the external cause of injury was recorded, and this only related to the most recent injury if there had been more than one injury in the 12 months. Therefore, the examination of the complex relationships between demographic, socioeconomic and health characteristics and the risk of different types of injury was limited.

There may have been variation in reporting between individuals. Participants were asked to recall any injuries over the past 12 months, and so there may be recall bias present. The 12% incidence estimate is therefore likely to be an underestimate, although stopping usual activities for more than a week is a fairly serious and probably memorable event. The question about injury asked in the survey requires a subjective judgement of what constitutes stopping normal activities, and so there may have been considerable variability in the severity and type of injuries being reported.

Whether an injury stops an individual from performing their normal activities or not may relate more to the individual and their perception of what they can and can’t do, or the nature of their normal activities, than to the characteristics of the injury itself. Therefore, the reported injuries are likely to be heterogeneous.
Conclusions

Injuries which disrupt usual activities for more than a week are common and represent a significant impact on society from injury which is not captured by health care statistics. Characterising those who are likely to have injuries that disrupt normal functioning in terms of age, sex and ethnicity, as well as health status, may contribute to the development of appropriate injury prevention interventions. Although not always causing a burden on the health care system, such injuries cause considerable time off work and associated costs. Often family members or others will be required to help injured individuals with normal activities, and so preventing them also has considerable social benefit. The information provided by this study gives some indication of level of this aspect of injury burden, and may be useful in planning interventions targeted at those most likely to experience such injuries.

Competing interests: None.

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References:


SF-36v2 norms for New Zealanders aged 55–69 years

Christine Stephens, Fiona Alpass, Merel Baars, Andy Towers, Brendan Stevenson

Abstract
Owing to an ageing population there is growing interest in research to improve the health of older New Zealanders. To facilitate the use of the internationally used SF-36 (version 2) measure of health and quality of life for this work in New Zealand we provide norms and comparative data from the first wave in a longitudinal study of a representative sample of New Zealanders aged 55–69 years. The use of the normative data from this study will facilitate comparisons of results from small clinical samples of older people with the general New Zealand population and international populations. The norms are also available for use in calculating summary physical and mental health summary scores for data from clinical trials and national surveys.

The first ‘baby-boomers’ (a demographic bulge born between 1946 and 1960) will reach 65 in 2011 and the movement of this group into retirement and older age will present new issues in health and health care research. Because of this ageing population there is growing interest in studies of the health and well-being of older New Zealanders.

To facilitate the use of the Medical Outcomes Study Short Form questionnaire version 2 (SF-36v2) for this work in New Zealand we provide norms and comparative data from the first data wave in a longitudinal study of a representative sample of New Zealanders aged 55–69 years.

The SF-36 Health Survey is a widely used reliable self-reported measure of generic health status. Designed for use in clinical practice and research, health policy evaluations, and general population surveys, it has been widely applied to assess changes and differences in mental and physical health in medical intervention studies and has been shown to be valid for use in older people. Because of improved wording and lay-out, and the changed number of responses in some questions, the second version of this measure minimises ambiguity and allows for greater comparability between cultural adaptations and translations. At the same time scores can still be directly compared to SF-36 version 1. Scores from eight sub-scales measuring different aspects of health may be standardised, and also summarised to provide scores for two underlying factors of physical and mental health. When used as a baseline and outcome measure in smaller samples Ware and colleagues strongly recommend that these measures are standardised using population norms to provide the basis for national and international comparisons, and also improve the validity of observations within the sample under study.

The authors of the scale also stress the importance of the norms used being as up to date as possible, and Scott et al, point to the variations in scores across cultures so that local norms are required.
In New Zealand, norms on the SF-36 (version 1) sub-scales for the general population from the New Zealand national health survey (1996/7) have been published by Scott et al., who also assessed the appropriateness of the SF-36 for the New Zealand population. However, more recent normative data using SF-36v2, and norms for particular population groups, such as older people, are now required.

The Health Work and Retirement study (HWR) is a longitudinal study of older New Zealanders funded by the Health Research Council of New Zealand. The first wave of data collection for the HWR involved a representative sample of 6662 New Zealanders aged between 55 and 70 years. In this paper we provide normative data from this sample on SF-36v2 sub-scales and Physical and Mental Health summary scores for the whole sample as well as those for 5 year age groups, ethnicity and gender. Sub-group comparisons are given for physical health summary scores and mental health summary scores.

**Method**

**Sampling method**—The population of interest for the study was New Zealanders aged 55 to 70 who are generally in the later stages of work life or early stages of retirement (there is no legal age of retirement in New Zealand, although a universal superannuation scheme provides a pension from age 65). There are approximately 814,464 New Zealanders aged 50 to 69, with 65790 (8%) of those identifying as Māori, the indigenous people of Aotearoa/New Zealand.

The New Zealand Electoral Roll was the source for sample selection. Registration on the roll is mandatory for all citizens eligible to vote in government elections and in 2007, 96% of all eligible New Zealanders were registered. Equal probability random sampling procedures were used to select two independent samples to represent the general population (N=5264) and the Māori population (N=7781).

Māori were over-sampled for this study using the Māori descent indicator on the general and Māori electoral rolls. This was done to maximise participant recruitment and provide sufficient numbers for statistical analysis in later data collection waves. In total 13,045 55–70 year olds were surveyed. The total response rate (after exclusions, e.g. unable to be contacted, deceased, or institutionalised, N=551) was 53.32% (N=6662). The response rate for the general population was 61% and for the Māori sample, 48%. Both samples have similar gender and age group proportions to the general and Māori populations respectively when compared to the 55–69 year-old population from the 2006 Census.

Because of the over-sampling, the total sample was weighted to represent the New Zealand population. A post-stratified weighting variable according to primary ethnicity was applied to the present analyses based on the population estimates from the 2006 Census for the 55–69 year old age group.

In the weighted sample age was well distributed with 2422 (40.5%) aged 55–59, 1905 (31.9%) aged 60–64, and 1651 (27.6%) aged 65–69. There were 219 missing and 513 who were outside the 55–69 age range and accordingly not included in the present analysis (N=5978). The numbers (rounded) for ethnicity were: European descent (4153), Māori (461), Pasifika (199), Asian (265), other (864), and missing (i.e. did not report ethnicity; 40). In regard to gender, 2856 (47.8%) were males and 3103 (51.9%) females, (22 missing).

**Data collection method**—The postal survey used multiple contact points to maximise participation.

1. A brief pre-notice letter was sent to inform potential participants about their selection and the questionnaire study.
2. One week later, a questionnaire which included the SF-36 items and other measures for the Health Work and Retirement Longitudinal Study (see http://hwr.massey.ac.nz), a detailed information sheet and a free-post return envelope were sent.
3. At 3 weeks a reminder postcard was sent to the whole sample.
4. At 6 weeks a replacement questionnaire was sent to all non-respondents.
5. At 11 weeks a final postcard was sent to all non-respondents.
These procedures were approved by the Massey University Human Ethics Committee.

**Measures and analysis**—The SF-36v2 comprises 36 items grouped into eight sub-scales each examining a different dimension of health (physical function, role limitations for physical problems, bodily pain, general health perception, vitality, social functioning, role limitations for emotional problems and general mental health).

The eight sub-scales of the SF-36v2 were calculated using algorithms provided by the developers. For all measures of the eight SF-36v2 sub-scales, scores were transformed to scales of 0 to 100, with higher scores reflecting better health and fewer role limitations.

In addition, the 8 sub-scales were combined using principle components (orthogonally rotated) derived coefficients to form two components assessing physical (PCS) and mental health (MCS). Each physical and mental component summary score was standardised (using standard deviations from the present representative study) with lower scores implying poorer health.

Cronbach’s alpha coefficients were used to test internal consistency of the 8 SF-36v2 subscales and these are reported in Table 1. Cronbach’s alpha exceeded .80 in all but one subscale (social functioning with alpha .67).

**Table 1. Cronbach’s alpha internal consistency statistics for the eight sub-scales of the SF-36v2**

<table>
<thead>
<tr>
<th>Sub-scale</th>
<th>No. of items</th>
<th>N</th>
<th>Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical function</td>
<td>10</td>
<td>6478</td>
<td>0.92</td>
</tr>
<tr>
<td>Role – physical</td>
<td>4</td>
<td>6491</td>
<td>0.95</td>
</tr>
<tr>
<td>Bodily pain</td>
<td>2</td>
<td>6521</td>
<td>0.90</td>
</tr>
<tr>
<td>General health</td>
<td>5</td>
<td>6457</td>
<td>0.83</td>
</tr>
<tr>
<td>Vitality</td>
<td>4</td>
<td>6498</td>
<td>0.85</td>
</tr>
<tr>
<td>Social function</td>
<td>2</td>
<td>6653</td>
<td>0.67</td>
</tr>
<tr>
<td>Role emotional</td>
<td>3</td>
<td>6424</td>
<td>0.92</td>
</tr>
<tr>
<td>Mental health</td>
<td>5</td>
<td>6491</td>
<td>0.81</td>
</tr>
</tbody>
</table>

Mean differences between groups were tested using one-way ANOVA. Owing to different group sizes and unequal variances the Welch statistic was used to test equality of means, and if significant differences were found, post hoc Tamhane’s t tests (p<0.05), assuming a non-normal distribution and unequal variances, were performed.

Each variable had some missing data so Ns for every analysis vary and are reported in the results.

**Results**

Table 2 provides means and standard deviations for each of the eight sub-scales of the SF-36v2 in the whole sample (55–69 years of age). Means and standard deviations are also provided across three different age groups: 55–59 years, 60–64 year and 65–69 years.

PCS and MCS scores for this group are also provided in Table 2. The Welch test of equality of means revealed that the three age groups differed significantly on PCS scores ($F(2, 3372)$=44.83, p<.001). Post hoc comparisons using the Tamhane test indicated significant differences between all three group means, with PCS scores decreasing as age increased. One-way ANOVA also showed a significant difference between age groups for MCS scores ($F(2, 3494)$=15.16, p<0.001). Post hoc analysis revealed a significant difference between the 55–59, and 60–64, and the oldest (65–69) sub-group, with the oldest sub-group scoring higher than the two younger groups.
Thus, mean PCS and MCS scores across the three age groups showed a decrease in physical health and an increase in mental health scores as people age (see Figure 1).

**Figure 1. PCS and MCS mean scores across three age groups**

![Graph showing PCS and MCS mean scores across three age groups](image)

**Note:** Cases weighted for ethnicity.

Mean scores and standard deviations broken down by three ethnic groups (i.e. New Zealand European, Māori, and Pasifika) and across the three different age groups are shown in Table 3. The three ethnic groups were found to have significantly different scores on both PCS, $F(2, 331.50)=67.22, p<.001$, and MCS $F(2, 325.70)=30.17, p<.001$. Post hoc comparisons indicated that the mean PCS score for New Zealand Europeans was significantly different from Māori and Pasifika Peoples. Māori also differed significantly from Pasifika Peoples. The same pattern was observed for the MCS summary scores. Here, the mean MCS score for New Zealand Europeans significantly differed from Māori and Pasifika Peoples. However, Māori and Pasifika Peoples did not differ significantly on the Mental Health summary score.
Table 2. SF-36v2 Mean scores (SD) on eight sub-scales for whole sample (with 95% confidence intervals [CI]) and across three age groups

<table>
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<tbody>
<tr>
<td>55–69</td>
<td>5536</td>
<td>79.32 (22.81)</td>
<td>81.49 (24.17)</td>
<td>71.12 (23.75)</td>
<td>71.12 (21.63)</td>
<td>65.57 (19.64)</td>
<td>85.22 (22.48)</td>
<td>87.37 (20.63)</td>
<td>80.71 (15.78)</td>
<td>50.00</td>
<td>50.10</td>
</tr>
<tr>
<td>95%CI</td>
<td></td>
<td>78.73–79.91</td>
<td>80.87–82.11</td>
<td>70.51–71.73</td>
<td>70.56–71.68</td>
<td>65.07–66.07</td>
<td>84.65–85.80</td>
<td>86.84–87.90</td>
<td>80.31–81.11</td>
<td>49.75–49.85</td>
<td></td>
</tr>
<tr>
<td>55–59</td>
<td>2296</td>
<td>81.50 (22.52)</td>
<td>84.48 (22.94)</td>
<td>72.10 (23.33)</td>
<td>72.02 (21.66)</td>
<td>65.41 (19.69)</td>
<td>85.33 (22.32)</td>
<td>87.77 (20.30)</td>
<td>79.93 (16.13)</td>
<td>51.22</td>
<td>49.52</td>
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<tr>
<td>60–64</td>
<td>1743</td>
<td>79.93 (21.78)</td>
<td>81.69 (23.87)</td>
<td>70.76 (23.92)</td>
<td>70.52 (21.60)</td>
<td>65.32 (19.65)</td>
<td>84.97 (22.55)</td>
<td>87.74 (20.54)</td>
<td>80.23 (16.16)</td>
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<tr>
<td>65–69</td>
<td>1497</td>
<td>75.33 (23.88)</td>
<td>76.70 (25.55)</td>
<td>70.05 (24.14)</td>
<td>70.42 (21.60)</td>
<td>66.10 (19.61)</td>
<td>85.36 (22.66)</td>
<td>86.33 (22.30)</td>
<td>82.42 (14.63)</td>
<td>48.14</td>
<td>51.17</td>
</tr>
</tbody>
</table>

Note: Range 0–100, sample size varies due to missing data; Cases weighted for ethnicity. Group Ns are different from total count because weighted Ns are rounded.
Table 3. SF-36v2 Mean scores (SD) for the eight sub-scales, PCS and MCS across ethnicity groups whole sample (with 95% confidence intervals [CI]) and across three age groups

<table>
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<td>NZ Eur</td>
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<td>82.38 (23.80)</td>
<td>71.97 (23.55)</td>
<td>71.98 (21.36)</td>
<td>65.92 (19.65)</td>
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<td>88.79 (19.80)</td>
<td>81.45 (15.39)</td>
<td>50.22 (9.60)</td>
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<tr>
<td>95%CI</td>
<td></td>
<td>79.73–81.10</td>
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<td>71.25–72.70</td>
<td>71.32–72.64</td>
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<tr>
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<td>1491</td>
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<td>70.22 (21.21)</td>
<td>65.42 (19.56)</td>
<td>85.66 (22.53)</td>
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<td>Māori</td>
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<td>66.61</td>
<td>66.74 (23.53)</td>
<td>62.39 (20.54)</td>
<td>75.60 (25.14)</td>
<td>79.03 (25.22)</td>
<td>77.69 (17.22)</td>
<td>47.39 (10.60)</td>
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<td>95%CI</td>
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<td>64.13–68.98</td>
<td>64.53–68.96</td>
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<td>76.21–80.99</td>
<td>76.67–81.40</td>
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<td>77.22 (26.12)</td>
<td>68.03 (26.03)</td>
<td>68.22 (23.29)</td>
<td>62.23 (20.89)</td>
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<td>70.89 (28.80)</td>
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<td>65.95 (24.44)</td>
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<td>76.81 (27.15)</td>
<td>77.63 (26.56)</td>
<td>77.24 (17.73)</td>
<td>46.87 (10.79)</td>
<td>47.27 (11.22)</td>
</tr>
<tr>
<td>65–69</td>
<td>108</td>
<td>66.95 (28.06)</td>
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<td>63.29 (19.83)</td>
<td>78.87 (25.88)</td>
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<td>78.67 (16.52)</td>
<td>45.42 (10.61)</td>
<td>48.43 (10.67)</td>
</tr>
<tr>
<td>Pasifika</td>
<td>155</td>
<td>61.69 (30.05)</td>
<td>61.26 (28.57)</td>
<td>57.83 (25.79)</td>
<td>59.26 (21.14)</td>
<td>63.74 (18.16)</td>
<td>70.75 (26.12)</td>
<td>70.83 (25.79)</td>
<td>73.71 (17.90)</td>
<td>42.51 (8.78)</td>
<td>45.81 (9.47)</td>
</tr>
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<td>95%CI</td>
<td></td>
<td>57.29–66.09</td>
<td>57.16–65.36</td>
<td>54.17–61.49</td>
<td>56.13–62.40</td>
<td>61.08–66.40</td>
<td>67.12–74.38</td>
<td>66.97–74.70</td>
<td>71.09–76.34</td>
<td>41.13–43.89</td>
<td>44.32–47.30</td>
</tr>
<tr>
<td>55–59</td>
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<td>58.33 (31.13)</td>
<td>61.84 (27.94)</td>
<td>57.62 (28.42)</td>
<td>64.34 (19.18)</td>
<td>62.37 (18.14)</td>
<td>73.21 (23.05)</td>
<td>66.42 (24.11)</td>
<td>72.63 (20.29)</td>
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</tr>
<tr>
<td>60–64</td>
<td>56</td>
<td>63.44 (31.41)</td>
<td>67.16 (27.67)</td>
<td>56.25 (18.29)</td>
<td>56.80 (17.45)</td>
<td>66.22 (14.80)</td>
<td>68.75 (27.27)</td>
<td>75.49 (22.02)</td>
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<td>46.83 (8.20)</td>
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<tr>
<td>65–69</td>
<td>36</td>
<td>64.65 (26.09)</td>
<td>51.14 (28.89)</td>
<td>60.55 (29.89)</td>
<td>53.30 (27.24)</td>
<td>62.73 (22.03)</td>
<td>69.32 (29.71)</td>
<td>70.37 (33.60)</td>
<td>78.09 (17.89)</td>
<td>40.35 (11.13)</td>
<td>46.80 (11.80)</td>
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</tbody>
</table>

Note: Range 0–100, sample size varies due to missing data. Cases weighted for ethnicity. Group Ns are different from total count because weighted Ns are rounded.
Table 4. SF-36v2 mean scores (SD) for the eight sub-scales, PCS and MCS across gender whole sample (with 95% confidence intervals [CI]) and across three age groups

<table>
<thead>
<tr>
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<tbody>
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<td></td>
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<tr>
<td>55–69</td>
<td>2658</td>
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<td>71.88</td>
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<td>85.17</td>
<td>87.52</td>
<td>81.32</td>
<td>50.24</td>
<td>50.30</td>
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<tr>
<td></td>
<td></td>
<td>(22.16)</td>
<td>(23.97)</td>
<td>(23.48)</td>
<td>(21.21)</td>
<td>(19.65)</td>
<td>(21.89)</td>
<td>(20.52)</td>
<td>(15.45)</td>
<td>(9.23)</td>
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<td></td>
<td>80.10–</td>
<td>80.72–</td>
<td>71.01–</td>
<td>69.69–</td>
<td>65.92–</td>
<td>84.36–</td>
<td>86.76–</td>
<td>80.74–</td>
<td>49.89–</td>
<td>49.95–</td>
</tr>
<tr>
<td></td>
<td></td>
<td>81.76</td>
<td>82.50</td>
<td>72.76</td>
<td>71.27</td>
<td>67.38</td>
<td>85.97</td>
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<td>81.89</td>
<td>50.59</td>
<td>50.65</td>
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<td>72.98</td>
<td>71.70</td>
<td>66.78</td>
<td>85.27</td>
<td>88.37</td>
<td>80.63</td>
<td>51.58</td>
<td>49.87</td>
</tr>
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<td>792</td>
<td>80.63</td>
<td>80.99</td>
<td>70.21</td>
<td>68.83</td>
<td>65.57</td>
<td>84.43</td>
<td>87.81</td>
<td>80.47</td>
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<tr>
<td></td>
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<td>(21.99)</td>
<td>(24.75)</td>
<td>(25.54)</td>
<td>(21.55)</td>
<td>(20.30)</td>
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<td>65–69</td>
<td>750</td>
<td>77.32</td>
<td>76.29</td>
<td>72.03</td>
<td>70.41</td>
<td>67.59</td>
<td>85.79</td>
<td>85.93</td>
<td>83.20</td>
<td>48.76</td>
<td>51.24</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>55–69</td>
<td>2859</td>
<td>77.83</td>
<td>81.35</td>
<td>70.43</td>
<td>71.69</td>
<td>64.59</td>
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<td>87.27</td>
<td>80.17</td>
<td>49.76</td>
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<td>(24.36)</td>
<td>(23.96)</td>
<td>(22.00)</td>
<td>(19.58)</td>
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<td>(20.72)</td>
<td>(16.01)</td>
<td>(9.86)</td>
<td>(9.75)</td>
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<td>69.57–</td>
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<td>63.88–</td>
<td>84.46–</td>
<td>86.52–</td>
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<td>49.40–</td>
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<td>83.38</td>
<td>71.33</td>
<td>72.36</td>
<td>64.23</td>
<td>85.43</td>
<td>87.26</td>
<td>79.36</td>
<td>50.87</td>
<td>49.24</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(23.75)</td>
<td>(24.37)</td>
<td>(24.44)</td>
<td>(22.87)</td>
<td>(20.35)</td>
<td>(23.02)</td>
<td>(20.86)</td>
<td>(16.04)</td>
<td>(10.01)</td>
<td>(9.96)</td>
</tr>
<tr>
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<td>82.24</td>
<td>71.19</td>
<td>71.90</td>
<td>65.12</td>
<td>85.38</td>
<td>87.69</td>
<td>80.07</td>
<td>50.17</td>
<td>50.90</td>
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<td>65–69</td>
<td>740</td>
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<td>77.01</td>
<td>68.04</td>
<td>70.38</td>
<td>64.43</td>
<td>84.88</td>
<td>86.73</td>
<td>81.55</td>
<td>47.48</td>
<td>51.07</td>
</tr>
</tbody>
</table>

Note: Range 0–100, sample size varies due to missing data; Cases weighted for ethnicity; Group Ns are different from total count because weighted Ns are rounded.
### Table 5. Comparison of the SF-36v2 mean scores (SD) for the eight sub-scales between 55–64 year olds in the HWR survey with 55–64 year olds in the New Zealand Health Survey (2006/7)

<table>
<thead>
<tr>
<th>Survey</th>
<th>Physical Function</th>
<th>Role Physical</th>
<th>Bodily Pain</th>
<th>General Health</th>
<th>Vitality</th>
<th>Social Function</th>
<th>Role Emotional</th>
<th>Mental Health</th>
</tr>
</thead>
<tbody>
<tr>
<td>HWR</td>
<td>79.3 (78.7–79.9)</td>
<td>81.5 (80.8–82.1)</td>
<td>71.1 (70.5–71.7)</td>
<td>71.1 (70.5–71.6)</td>
<td>65.6 (65.0–66.1)</td>
<td>85.2 (84.7–85.8)</td>
<td>87.4 (86.8–87.9)</td>
<td>80.7 (80.3–81.1)</td>
</tr>
<tr>
<td>New Zealand Health Survey</td>
<td>81.3 (80.1–82.6)</td>
<td>83.2 (81.9–84.5)</td>
<td>73.0 (71.6–74.5)</td>
<td>73.6 (72.5–74.6)</td>
<td>65.0 (64.1–66.0)</td>
<td>89.0 (88.0–90.1)</td>
<td>94.6 (93.9–95.4)</td>
<td>84.6 (83.9–85.2)</td>
</tr>
</tbody>
</table>
In Table 4, normative data is provided for males and females for the sample as a whole and across three age groups. When examining the sample as a whole, no significant difference was found in scores on PCS and MCS, \( F(1, 5514.53) = 3.47, \text{ns}, \) and \( F(1,5514.75)=2.04, \text{ns} \), respectively. Only PCS scores differed significantly between males and females aged 55–59, \( F(1, 2559)=4.39, p<.05 \), with males reporting better physical health. No significant gender differences were found for the other age groups on PCS or MCS scores.

**Discussion**

Reliability analysis indicates good internal consistency of the eight sub-scales of the SF-36v2. Reliability statistics for the two role functioning sub-scales and vitality are higher than those previously reported for the general New Zealand population, whereas the social functioning scale shows lower reliability. Although social functioning has only two items, the items are very similar in wording and it is not immediately apparent why this group was not responding consistently. Given past reliable findings this subscale may be used with confidence while this anomalous finding is tested in future surveys of this population.

Validity of the scales for use with this population is also supported by the results for health outcomes and for ethnic differences. For the sample as a whole, physical health summary scores decreased and mental health summary scores increased with age. The observation that physical health deteriorates across these age groups is not surprising. The observation that mental health initially improves with age after 55 may not be expected by those unfamiliar with ageing populations, yet is well supported by previous findings for the general New Zealand population and in international samples.

The SF-36v2 was used in the 2006/7 New Zealand Health Survey, a survey of the whole population. Comparison of the 55–64 year old age group with the HWR 55–64 year old age group (see Table 5) shows that physical health scores are comparable, although slightly higher, while mean mental health sub-scale scores (social functioning, role emotional, and mental health) are higher in the New Zealand Health survey. The higher mental health scores in this smaller sample of the older population support the trends shown in the present data.

Comparison of three ethnic sub-groups, New Zealanders of European descent, Māori and Pasifika Peoples, revealed significant differences in both physical and mental health. Overall, New Zealand Europeans reported better physical and mental health than Māori and Pasifika Peoples. These results are consistent with those from earlier studies of the SF36 in New Zealand.

These differences have also been reliably observed in other studies in New Zealand using different indicators of health outcomes, including mortality. However, Scott et al. have questioned the validity of the factor structure of these summary scores (which attempt to separate mental and physical health as separate constructs) for Māori and Pasifika People. They found that the expected factor structure was not reproduced for older Māori and this suggests that comparisons using the summary scores as presently constructed should be treated with caution, and other ways of
using the responses to SF-36v2 items must be investigated. Scott et al. also showed that these scores are not valid for Pasifika peoples.

In addition, the SF-36v2 mean scores for Pasifika peoples in these data are not appropriate for use as normative data since there are low numbers, no indication of particular ethnicity, and wide confidence intervals in the results. However these results do suggest areas for ongoing enquiry using more focussed samples.

An apparent difference for this older age group compared with general populations, is less marked gender differences. Unlike results reported previously for the general New Zealand population and similar U.K. samples, data from our sample of New Zealanders aged 55–69 did not indicate significant sex differences on either physical or mental health summary scores. Differences were found only for the youngest age group (aged 55–59) and only for the physical health summary score. This finding indicates the importance of examining different population groups, as norms for certain groups can differ from the general population and patterns of change (such as improving mental health in general) may be revealed.

The present indication of a narrowing of the gap between male and female self-rated health may also be reflected in United Nations estimates of a difference in male and female mortality in New Zealand of less than 3 years by 2020.

The representativeness of the present data may be limited because of a low response rate from the selected population. Although the response rate of 61% for New Zealand Europeans was high for a postal survey, questions remain regarding the characteristics of those who did not respond. Because the sample in this study reflects census data on key variables such as age groups and gender, and owing to the oversampling of Māori, we have confidence that this large sample can indicate trends and differences in health in the population.

The SF-36v2 is now one of the most well used measures of health and quality of life in survey and clinical research. Although the measure has its critics and limitations, an advantage of its widespread use is the ability to compare results internationally and within local populations. The use of the norms from this study will facilitate the comparison of sub-scale data collected from small clinical samples of older Māori and European New Zealanders with the general New Zealand population and international populations of those aged 55 to 69 years. The norms are also available for use in calculating summary physical and mental health summary scores for data from national surveys (as opposed to using norms generated in the US or other countries). These norms may be used in any research with those aged 55–69 years and within three different age groups.

Competing interests: None.

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Acknowledgements: These data were collected with the assistance of research funding from the Health Research Council of New Zealand. The authors also acknowledge the very helpful contributions of the two anonymous NZMJ reviewers.

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References:

Evaluation of Pacific obstetric and gynaecological ultrasound scanning capabilities, personnel, equipment and workloads

Hemal Kodikara, Jenny Mitchell, Alec Ekeroma, Peter Stone

Abstract

Aims There are no published data on the coverage, training or experience of ultrasound services in the Pacific. This study aimed to obtain information on the knowledge, experience and training of ultrasound operators and scanning equipment and workloads in the Pacific region.

Methods Participants for the survey were recruited by post, via the Pacific Society of Reproductive Health (PSRH) website and at the PSRH conference. Questions obtained information on ultrasound scanning capabilities, personnel, equipment and workloads in the Pacific region.

Results 30 respondents from 17 hospitals in 11 countries provided completed questionnaires. Close to 50% of the responses were from Fiji. The majority of respondents were sonographers or obstetricians. Lack of transvaginal probes (7/17) in some facilities limit accuracy of early pregnancy scanning. 17/17 respondents felt an advanced course would be the preferred type of course.

Conclusion There is a sound basic level of ultrasound being performed in the Pacific region. A multimodal training programme, incorporating a practical hands-on course based in New Zealand, combined with CD/published materials appears to be the best method of developing more advanced skills in order to optimise antenatal care in the region.

Ultrasound in pregnancy is one of the most important advances in antenatal and obstetric care. Routine ultrasound can provide real benefit to patients when it is included in antenatal care programmes designed to improve maternal and neonatal health, and as in developed countries it could become a standard procedure in developing countries. This is limited by a number of factors including lack of resources, training and infrastructure in addition to the problems of not having adequate maternity services in place which could benefit from the information that ultrasound may provide.

The Pacific region geographically covers a vast area and is very heterogeneous in ethnic/cultural make up and also in basic health indices. The proportion of skilled birth attendants (SBA) at delivery is a general indicator of quality of antenatal/perinatal care. 61.2% of births in Melanesia, 93.9% in Micronesia and 97.6% of Polynesia were attended by SBA. This is reflected in the perinatal mortality rate which varies from 45/1000 in Melanesia to 13/1000 in Micronesia and 20/1000 in Polynesia. This demonstrates the likely differences in antenatal care coverage among nations in the Pacific which is important when considering coverage/need/provision of ultrasound services.

1 The proportion of skilled birth attendants (SBA) at delivery is a general indicator of quality of antenatal/perinatal care. 61.2% of births in Melanesia, 93.9% in Micronesia and 97.6% of Polynesia were attended by SBA. This is reflected in the perinatal mortality rate which varies from 45/1000 in Melanesia to 13/1000 in Micronesia and 20/1000 in Polynesia. This demonstrates the likely differences in antenatal care coverage among nations in the Pacific which is important when considering coverage/need/provision of ultrasound services.
There are no published data on the coverage, training or experience of ultrasound operators in the Pacific. Surveys of ultrasound knowledge and training requirements have been conducted in rural/remote Australia in order to assess educational need.\(^3\) It is imperative to obtain this information for the Pacific to plan for allocation of further resources, further training and ongoing education in ultrasound if such services are to be developed in the region.

**Method**

Ultrasound operators or centres from eleven countries in the Pacific were identified via regional hospitals in each country and were asked to complete a self-administered written questionnaire in 2007. Personal contact was made with key individuals. The questionnaire aimed to evaluate the demographics, qualifications, and experience of persons performing ultrasound. In addition, information was sought as to the workload, type of scanning performed and ultrasound equipment available. The questionnaire also looked at the preferences of ultrasound operators in the Pacific region about the location and content of educational and practical courses that would be deemed useful.

The questionnaire was in the form of a series of tick boxes plus the option for short responses. In addition to this, the questionnaire provided opportunity for respondents to identify current problems with ultrasound scanning in their region and to make any further comments/suggestions for improvement of services.

The questionnaire was distributed by mail, as well as being distributed at a meeting of the Pacific Society of Reproductive Health (PSRH) and was also made available as a web-based document through the PSRH web site. The PSRH secretariat followed up all personnel likely to be associated with obstetric and gynaecological ultrasound in the Pacific in an attempt to obtain as many responses as possible.

**Results**

Thirty ultrasound operators from seventeen regional centres, in 11 countries provided responses to the questionnaire.

The countries in Micronesia from which survey response were received were Kiribati and the Federated States of Micronesia. In Melanesia, responses were obtained from Papua New Guinea (PNG), Vanuatu, the Solomon Islands and Fiji. In Polynesia, the countries participating in the survey were Tuvalu, Tonga, Cook Islands, American Samoa and Niue.

The proportion of responses from the 11 countries is shown in Figure 1. The mean age of ultrasound operators was thirty nine (range 24 to 60). Forty-six percent were male.

In Figure 2, the professional background of the ultrasound operators is shown.

**Experience and training**—The median level of ultrasound experience for respondents was 7 years (range 2–25 years). Overall 25 (83\%) respondents had some formal training. Training occurred throughout the Pacific and this is shown in Figure 3 below.
Figure 1. Proportion of survey responses from each country (%)

Figure 2. Professional training of ultrasound operator (%)

Figure 3. Location of previous ultrasound training (%)

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Training was provided by varying groups including World Health Organization, radiologists and obstetricians. Eleven (37%) were certified in ultrasound with 12 (40%) engaged in some form of continuing education. These data are shown for each country in Table 1.

Table 1. Training and experience of ultrasound operators by country

<table>
<thead>
<tr>
<th>Country</th>
<th>No. of operators</th>
<th>Trained</th>
<th>Certified</th>
<th>Continuing education</th>
<th>Experience (mean in years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Samoa</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>Cook Islands</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Fiji</td>
<td>15</td>
<td>12</td>
<td>13</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Kiribati</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Niue</td>
<td>2</td>
<td>2</td>
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<td>1</td>
<td>Not stated</td>
</tr>
<tr>
<td>Papua New Guinea</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>Solomon Islands</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>9.5</td>
</tr>
<tr>
<td>Tonga</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Tuvalu</td>
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<td>0</td>
<td>Not stated</td>
<td>Not stated</td>
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</tr>
<tr>
<td>Vanuatu</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>7</td>
</tr>
</tbody>
</table>

In a self-appraisal of expertise, 21 (70%) respondents felt they were ‘competent’ in ultrasound, with 7 (23%) having a ‘basic’ knowledge (3 not responding).

**Equipment and workload**—The ultrasound workload varied widely between services with a range of 20 to 2000 patients seen per month which is shown below in Figure 4.

**Figure 4. Number of patients seen per hospital per month**
A variety of transducer types were available, however transvaginal probes were available in only 59% (10/17) of centres including 16/17 centres performing scans to diagnose ectopic pregnancy. Portable scans were also not commonly available with just over 47% (8/17) of respondents having access to this.

Table 2 describes the availability of transvaginal or portable equipment in comparison with early pregnancy scanning.

Table 2. Availability of transvaginal probe/portable scanner and levels of scanning in early pregnancy by location

<table>
<thead>
<tr>
<th>Location</th>
<th>Transvaginal probe</th>
<th>Ectopic scanning</th>
<th>Miscarriage scanning</th>
<th>Portable scanner</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Samoa</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Cook Islands</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiji (CWM Hospital)</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiji (GPO Suva)</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiji (Labasa Hospital)</td>
<td>Yes</td>
<td></td>
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<tr>
<td>Fiji (Nakasi)</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Fiji (Nausori)</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiji (Samabula)</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kiribati</td>
<td>Yes</td>
<td></td>
<td></td>
<td>Not listed</td>
</tr>
<tr>
<td>Micronesia</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Niue Foou Hospital</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Papua New Guinea</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solomon Islands</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Tonga</td>
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<td></td>
<td>Not listed</td>
<td></td>
</tr>
<tr>
<td>Tuvalu</td>
<td>Not listed</td>
<td></td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Vanuatu (Port Vila)</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vanuatu (Santo)</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>10/17</strong></td>
<td><strong>16/17</strong></td>
<td><strong>12/17</strong></td>
<td><strong>8/17</strong></td>
</tr>
</tbody>
</table>

Types of scanning—Respondents were asked about their current ultrasound practice. All operators were performing early and mid pregnancy scans (for dating, anatomy etc), while 29/30 respondents were performing scans late in pregnancy. Similar high rates were found in gynaecological scans with 97%, 90% 100% and 93% of operators reporting scanning in the diagnosis of suspected ectopic pregnancy, vaginal bleeding, abdominal masses and abdominal pain respectively.

Future training—Twenty-seven (90%) respondents expressed an interest in an ultrasound training course and 28 (93%) saw a need for courses to train new/more staff. Respondents were asked whether a basic, advanced or refresher type of course was preferred. An ‘advanced’ type of course (fetal abnormality diagnosis, gynaecological pathology) was by far the most popular type of course requested. The preferred type of course by ultrasound centre is shown in Table 3.
Table 3. Preference for type of course by location

<table>
<thead>
<tr>
<th>Location</th>
<th>Basic</th>
<th>Advanced</th>
<th>Refresher</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Samoa</td>
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<td>Yes</td>
</tr>
<tr>
<td>Cook Islands</td>
<td>Yes</td>
<td>Yes</td>
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</tr>
<tr>
<td>Fiji (CWM Hospital)</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
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<tr>
<td>Fiji (GPO Suva)</td>
<td>Yes</td>
<td></td>
<td></td>
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<tr>
<td>Fiji (Labasa Hospital)</td>
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<tr>
<td>Fiji (Nakasi)</td>
<td>Yes</td>
<td></td>
<td></td>
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<tr>
<td>Fiji (Nausori)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Fiji (Samabula)</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kiribati</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Micronesia</td>
<td>Yes</td>
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<tr>
<td>Niue Foou Hospital</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<td>Papua New Guinea</td>
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</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>6/17</strong></td>
<td><strong>17/17</strong></td>
<td><strong>9/17</strong></td>
</tr>
</tbody>
</table>

Respondents were asked their preferred course teaching method. They favoured a multimodal course delivery method with more than half requesting a course featuring Internet, face-to-face and practical components. A solely Internet-based course was not favoured (3.3%) as while 27 respondents (90%) had access to a computer only 23 (77%) had access to reliable Internet. This is shown in Figure 5.

**Figure 5. Participants preferred course delivery method (%)**

![Figure 5](image)

Respondents were offered a number of locations for a possible course location with more than 90% willing to travel to New Zealand (Figure 6). However, only 40% of respondents felt that their government or department would be able to meet the required travel and accommodation costs.
Respondents were given the option of describing the current problems they have in their ultrasound practice. Twenty-three of 30 respondents provided information which was coded and grouped into the categories shown below in Figure 7.

The comments section allowed for suggestions for improving the current services: Sixteen out of thirty respondents provided information which was coded and grouped into the categories shown below in Figure 8.
Discussion

This is the first study surveying and evaluating obstetric and gynaecological ultrasound services in the Pacific. Informal comments prior to this study had suggested that in addition to the need to train more people capable of providing high quality obstetric and gynaecological ultrasound, there was a desire on the part of current personnel to be able to both up skill and engage in continuing professional development. The challenge then was to define more precisely the needs and consider effective ways of meeting these. The approach to the evaluation was not dissimilar to a “market” survey because it was apparent that there were diverse and in places very basic needs. Before designing any teaching programme in such an environment, it was essential to determine what content would be applicable and which learning modalities would be appropriate.

Worldwide, much of the ultrasonography that is currently performed is conducted by individuals with little or no formal training. Training these users should be a priority as a significant improvement in ultrasonography skill level will lead to an improvement in health outcomes due to more accurate performance. However lack of resources and infrastructure means that worldwide there are limited education opportunities for those in developing countries. Options for providing these opportunities include didactic lectures, reading from textbooks, CD-ROM and interactive online media and hands-on experience away from the site of work. Clearly cost is an issue and in the future satellite and other communication links may open new opportunities for online distance learning.

A pilot obstetric ultrasound workshop for rural doctors in Australia which used a combination of face-to-face workshops, teaching CDs and manuals, and an online obstetric ultrasound module, showed a clear improvement in confidence in performing ultrasound compared to their pre-workshop levels. The Manual of Diagnostic ultrasound produced by the World Health Organization is an example of published materials aimed to educate operators in developing countries; nevertheless the need for “extensive supervised training” is highlighted by the organization.
Given the large geographical area in which participants of this survey practice, a centralized teaching programme, or alternatively an online or CD-based programme would appear to be the most suitable for this population. Of the 30 respondents, approximately one-third identified themselves as obstetricians, one-third as sonographers and one-tenth were radiologists.

Most operators felt competent and were experienced in basic aspects of obstetric and gynaecologic ultrasound, a high proportion with formal training. Therefore a training programme while briefly reviewing ultrasound physics and basic skills should aim to provide a fairly advanced level of training. However “lack of knowledge” accounted for approximately 30% of the ongoing problems cited by respondents with a significant proportion not involved in any ongoing education on ultrasound. Addressing this lack of knowledge by developing an ultrasound education programme with a continuing education component is thus a priority.

This clear need for ongoing education and training was backed by a demand by ultrasound operators with 90% interested in further ultrasound training courses. 40% of comments on how to improve current services included further education and training as a target area. All ultrasound centres identified an ‘advanced’ level of course as the desired course type. A multimodal course type with face-to-face lectures, practical aspects and Internet was the preferred option. Close to 90% of respondents stated they would be willing to travel to a major centre such as in New Zealand for a course. The advantages of teaching in a large centre would include access to a higher patient volume than would be available in most centres in the Pacific excepting Fiji or Papua New Guinea.

It would appear that despite the potential advantages of an Internet-based course (given the vast physical distances between centres) the lack of reliable Internet facilities would limit this option currently. Computer access was high (90%) so a CD or DVD-based education format remained a possibility. Ultrasound teaching programmes frequently involve the use of large computer files as used in video clips or multiple images and in many areas the information technology currently available would not permit the use of such files. This would be expected to improve over time and resurvey in the future would be suggested by the authors.

As expected of lower income countries, a heavy workload, lack of resources and equipment were expected to be important limiting factors in ultrasound practice in the Pacific and this was upheld in the data. Limited or faulty equipment similarly accounted for 28% of problems. Only 40% of respondents felt that their government would provide financial support to travel for further education/training. Addressing this issue of lack of resources would appear to be a critical step in progressing ultrasound services in the Pacific and should parallel an increase in ultrasound training.

The importance of this is captured in the data on transvaginal probe availability; only 10/17 centres have transvaginal scanning capability despite early pregnancy bleeding and diagnosis of ectopic pregnancy scanning being quoted as one of the commonly performed type of scan. When asked to comment on how services could be improved, increasing numbers of staff, equipment and addressing lack of resources accounted for 42% of suggestions.
Limitations of the data include the high proportion of responses (approximately 50%) from Fiji which limits the generalisability of the data. As Fiji (in Suva particularly) has had a high volume obstetric and gynaecological ultrasound service for sometime, the data shown on desire for advanced courses rather than basic ones has been influenced by the needs of Fijian sonographers. Furthermore, responses were not available from some countries in the Pacific (Samoa for example).

Nevertheless, these data provide preliminary information on the current state of ultrasound services in the region and permit the completion of an ultrasound teaching and Continuing Professional Development programme supported by the Asia Pacific Committee of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists.

A multimodal training programme, incorporating a practical hands-on course based in New Zealand, combined with CD/published materials appears to be the best method of achieving this at present. However, in perhaps that most disadvantaged nation in the region, Papua New Guinea, running a basic startup course may well be the best solution for that environment because this survey has highlighted marked local differences in needs. Internet-based approaches may have a role in the future when more reliable Internet access is available in this region.

Competing interests: None.

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References:
The prevalence of diabetes among adults aged 40 years and over in Fiji

Garry Brian, Jacqueline Ramke, Louise Maher, Andrew Page, John Szetu

Abstract

Aim To estimate the prevalence of diabetes among adults aged ≥40 years in Fiji, and determine the demographic characteristics associated with this diagnosis.

Method During a population-based survey, participant glycosylated haemoglobin (HbA1c) was determined and physician diagnosis of diabetes self-reported. HbA1c ≥6.5% or claimed previous diagnosis, independent of HbA1c, defined presence of diabetes. Results were extrapolated to the whole population. Predictors of risk for diabetes were investigated using logistic regression models.

Results Of those enumerated, 1381 participated (73.0%). For 1353 with either a history of diabetes or valid HbA1c, prevalence of diabetes was 44.8% (95%CI 42.2–47.5). Adjusting for age and domicile, Indians had significantly higher risk of diabetes than Melanesians among males (OR 2.02, 95%CI 1.37–2.97, p<0.001) and females (OR 1.99, 95%CI 1.44–2.73, p<0.001). Females were at greater risk than males among Melanesians (OR 1.75, 95%CI 1.30–2.36, p<0.001) and Indians (OR 1.94, 95%CI 1.33–2.84, p<0.001). Risk increased with age for both genders and ethnicities, adjusting for ethnicity and domicile, then gender and domicile. The ethnicity-gender-age-domicile adjusted prevalence of diabetes among adults aged ≥40 years in Fiji was 41.0% (95%CI 38.4–43.6): 99,000 people.

Conclusion As identified in 1970, diabetes continues to be a substantial population health problem in Fiji.

The theory of epidemiological transition of populations subjected to economic development and “Westernisation” concerns the shift from infectious and nutrient deficiency to degenerative causes of disease and mortality. However, the transition should not be thought of as unidirectional or uniform across a nation’s population. As a country develops economically, those who maintain a more traditional lifestyle—usually characterised as rural and active, with lower calorie and fat intake—are generally regarded as being at lesser risk of degenerative diseases and their sequelae. But the reality is frequently more complicated.

Development and its impact are uneven. People, whether rural or urban dwellers, do not simultaneously access the benefits and risks of development. For example; initially at least, social arrangements, work practices, diet and activity may change, but lack of access to medical care frequently does not. The result may be a substantial burden of both infectious and chronic non-communicable diseases—which developing-country individuals and governments can ill afford to treat or manage.

Fiji is a South Pacific biracial island nation of 837,300 people (240,700 aged ≥40 years, being 50.0% female, 51.5% Melanesian, 42.6% Indian, 5.8% other ethnicity,
and 50.6% rural dwellers). It has a medium Human Development Index rating, which decreased in 2007, and is ranked 108 of 182 countries. There is continuing high morbidity from infectious disease, and chronic degenerative diseases are becoming more prevalent. Diabetes, which was declared a major public health problem in 1970, is an example of the latter.

In 1967 the reported prevalence of diabetes for urban indigenous Melanesian Fijians was 0.6%, with 5.7% for urban Asian Indo-Fijians. A 1980 survey using fasting blood glucose and oral glucose tolerance testing amongst adults aged ≥20 years found the age-standardised prevalence of diabetes for indigenous Fijian males to be 1.1% and 3.5%, and 1.2% and 7.1% for females, for rural and urban dwellers, respectively. For the Indian population, these were 12.1% and 12.9%, and 11.3 and 11.0%.

By 2002, based on a fasting blood sugar methodology, the prevalence was reported to be 16.0±3.1% of adults aged 25–64 years: increasing with age, and higher in urban than rural dwellers. The prevalence for Melanesian Fijians was 11.5%, with 21.2% for Indo-Fijians. No difference was associated with gender. The National Non-communicable Diseases Strategic Plan 2004–2008 was initiated as a result of the diabetes and other disease findings of this survey. Seven years on, a repeat survey is timely as Fiji navigates its epidemiological transition.

This paper reports glycosylated haemoglobin (HbA1c) data collected during the Fiji Eye Health Survey 2009 (FEHS2009). It provides an estimate of the prevalence of diabetes for the survey sample and for the Fiji population aged ≥40 years, and examines the predictors of risk of diabetes.

**Method**

**Sampling plan**—The sample frame (188,800 people aged ≥40 years; 50.3% female; 49.4% Melanesian Fijian, 44.9% Indo-Fijian, and 5.7% of other ethnicity; 43.2% rural dwellers) included all eight provinces of Viti Levu, Fiji’s main island, where 79.1% of the population reside. Using an anticipated prevalence of vision impairment of 11% in the target population, absolute precision of ±2.2% (20% relative difference), with 95% confidence, a design effect of 1.4 and a response rate of 80%, the sample size was determined to be 1354 persons. From the sample frame, 34 clusters of 40 people were required. Across Viti Levu, using national census data, the clusters were selected through probability proportionate to size sampling.

**Enumeration**—A single FEHS2009 survey team visited all clusters during September to November, 2009. Using a random process, the team leader identified the first household to be targeted in each cluster. Thereafter, consecutive households were approached and eligible people enumerated by trained local fieldworkers until the 40 participants for that cluster were enrolled. If an eligible person was absent, with no prospect of returning during the team’s time in the cluster, the absentee’s demographic and socioeconomic data were elicited from an available relative in the household or a knowledgeable adult in an adjacent household.

**Questionnaire and examination**—Participants attended a central facility, typically a community hall. An interview-based questionnaire, developed in English, translated into Fijian and Hindi, and back translated to ensure veracity, was administered. Respondents were invited to declare if a previous personal diagnosis of diabetes had been made by a doctor. HbA1c was determined using a point of care DCA 2000+ analyser (Siemens / Bayer, Munich, Germany).

**Study definition**—Diabetes was defined as present if HbA1c ≥6.5% or if a previous physician diagnosis of diabetes had been claimed, independent of HbA1c.
Data analysis—Data were de-identified and entered into a specifically designed database during the survey, with subsequent extensive but random checking for entry integrity. Prior to analysis, missing and outlier data were checked against the survey forms. Descriptive analyses were performed using SPSS Statistics 17.0 (SPSS Inc, Chicago, IL) and OpenEpi 2.3 (www.openepi.com). Logistic regression models were conducted in SAS using PROC GENMOD (SAS Institute Inc, Cary, NC). Statistical significance was accepted at p<0.05. Post hoc ratio survey weights based on national census data (2007) were used to adjust the sample prevalence estimates for ethnicity, gender, age and urban/rural domicile, and to extrapolate the findings to those aged ≥40 years across the entire country.

Ethical considerations—The Fiji National Research Ethics Review Committee convened by the Fiji Ministry of Health approved this study and its methodology. Consent was obtained from village chiefs prior to survey commencement in each cluster. Participants provided written acknowledgement of informed consent prior to data collection and examinations, including point of contact blood analysis. Communications occurred in English, Fijian or Hindi, depending on the participant’s preference.

Results

Of the 1892 eligible people enumerated, 1381 participated (73.0%). However, 27.2% (139/511) of nonparticipants were from just 5 (14.7%) clusters. Most (63.6%) nonparticipants were not at home, with 39.7% (129/325) of these away for work. Immobility or illness prevented 5.5% (28/511) attending. Others refused to participate because their eye or vision problem was already being managed (2.3%) or because there was no perceived problem (1.6%).

Of the 1381 participants, 222 (16.1%) claimed a previous personal diagnosis of diabetes had been made by a doctor. Of these, 107 were Melanesian, 106 were Indian, and 9 were of other ethnicities.

Of the 1159 participants who denied having diabetes, a valid HbA1c was not recorded for 28 (2.4%). Sporadic omission or analyser error (including sample anaemia) was responsible for 14. Logistical difficulties at one cluster accounted for the others. HbA1c was not documented for 2.9% (21/725) of Melanesians and 1.8% (7/396) of Indians. Those without HbA1c measurements were more likely to be younger (mean ages 49.9 and 54.8 years: t=2.47, p=0.01) and rural dwellers (p=0.01), but there was not gender bias (p>0.99).

For the 1131 participants with a valid HbA1c, the mean was 6.5% (95%CI 6.4–6.6) (Figure 1). This included 704 Melanesians, 389 Indians, and 38 of other ethnicities for whom the mean HbA1c were 6.5% (95%CI 6.4–6.6: minimum 4.6%), 6.6% (95%CI 6.4–6.7: minimum 3.8%), and 6.5% (95%CI 6.0–6.9: minimum 5.2%), respectively. Seven (0.6%) HbA1c were reported as 14.0%, the underestimating maximum determination capable by the DCA 2000+ analyser. HbA1c ≥6.5% was recorded for 212 indigenous Fijians, 161 Indo-Fijians, and 11 of other ethnicities.
Figure 1. Distribution of haemoglobin A1c (mean±SD: 6.5±1.3%) among 1131 adults aged ≥40 years in Fiji who denied a personal diagnosis of diabetes

For the 1353 survey participants with a previous diagnosis or valid HbA1c measurement, the prevalence of diabetes (defined as present if HbA1c ≥6.5% or if a previous physician diagnosis of diabetes had been claimed, independent of HbA1c: n=606) was 44.8% (95%CI 42.2–47.5). That for indigenous Fijian (319/811), Indian (267/495) and other ethnicity (20/47) participants was 39.3% (95%CI 36.0–42.7), 53.9% (95%CI 49.5–58.3), and 42.6% (95%CI 29.5–56.7), respectively.

Adjusting for age and domicile, Indians had a significantly higher risk of diabetes than Melanesians for both males (OR 2.02, 95%CI 1.38–2.97, p<0.001) and females (OR 1.99, 95%CI 1.44–2.74, p<0.001) (Table 1). Also, females were at greater risk than males for both Melanesians (OR 1.75, 95%CI 1.30–2.36, p<0.001) and Indians (OR 1.94, 95%CI 1.33–2.84, p<0.001) (Table 2). Adjusting for ethnicity and domicile (Table 1), and gender and domicile (Table 2), increasing risk of diabetes occurred with increasing age for both genders and both ethnicities.
Table 1. Predictors by gender of diabetes among adults aged ≥40 years in Fiji

<table>
<thead>
<tr>
<th>Variables</th>
<th>n</th>
<th>%</th>
<th>Adjusted^ Odds Ratio (95% Confidence Interval)</th>
<th>P value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>38</td>
<td>20.1</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>50–59</td>
<td>81</td>
<td>45.5</td>
<td>3.33 (2.08–5.31)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>60–69</td>
<td>68</td>
<td>47.6</td>
<td>3.68 (2.25–6.01)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>≥70</td>
<td>29</td>
<td>42.6</td>
<td>3.19 (1.74–5.86)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
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</tr>
<tr>
<td>Melanesian</td>
<td>114</td>
<td>32.3</td>
<td>1.00</td>
<td>–</td>
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<tr>
<td>Indian</td>
<td>96</td>
<td>46.2</td>
<td>2.02 (1.38–2.97)</td>
<td>&lt;0.001**</td>
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<tr>
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<td>6</td>
<td>35.3</td>
<td>0.93 (0.32–2.69)</td>
<td>0.897</td>
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<tr>
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<td>128</td>
<td>36.7</td>
<td>1.29 (0.88–1.89)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
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<td>88</td>
<td>38.4</td>
<td>1.00</td>
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<tr>
<td>Females‡</td>
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<tr>
<td>Age (years)</td>
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<tr>
<td>40–49</td>
<td>107</td>
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<td>50–59</td>
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<td>45</td>
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<td>1.83 (1.12–2.99)</td>
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<td>14</td>
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</tr>
<tr>
<td>Domicile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>200</td>
<td>50.9</td>
<td>1.10 (0.81–1.50)</td>
<td>0.551</td>
</tr>
<tr>
<td>Urban</td>
<td>188</td>
<td>49.7</td>
<td>1.00</td>
<td>–</td>
</tr>
</tbody>
</table>

^Adjusted for age, ethnicity and domicile; †Significance accepted at p<0.05; ‡ Multivariate analysis excluded 2 Melanesian females for whom age was unknown.

Table 2. Predictors by ethnicity of diabetes among adults aged ≥40 years in Fiji

<table>
<thead>
<tr>
<th>Variables</th>
<th>n</th>
<th>%</th>
<th>Adjusted^ Odds Ratio (95% Confidence Interval)</th>
<th>P value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melanesian Fijian‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
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<td></td>
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</tr>
<tr>
<td>Male</td>
<td>114</td>
<td>32.3</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>Female</td>
<td>203</td>
<td>44.7</td>
<td>1.75 (1.30–2.36)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>71</td>
<td>25.8</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>50–59</td>
<td>144</td>
<td>55.4</td>
<td>2.37 (1.64–3.45)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>60–69</td>
<td>95</td>
<td>52.5</td>
<td>3.39 (2.26–5.08)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>≥70</td>
<td>40</td>
<td>38.8</td>
<td>1.89 (1.16–3.06)</td>
<td>0.011*</td>
</tr>
<tr>
<td>Domicile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>131</td>
<td>35.3</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>Urban</td>
<td>186</td>
<td>42.7</td>
<td>1.35 (1.00–1.81)</td>
<td>0.048*</td>
</tr>
<tr>
<td>Indo-Fijian</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Male</td>
<td>96</td>
<td>46.2</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>Female</td>
<td>171</td>
<td>53.8</td>
<td>1.94 (1.33–2.84)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>69</td>
<td>39.0</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>50–59</td>
<td>103</td>
<td>58.9</td>
<td>2.34 (1.51–3.62)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>60–69</td>
<td>63</td>
<td>64.3</td>
<td>3.24 (1.91–5.49)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>≥70</td>
<td>32</td>
<td>71.1</td>
<td>4.14 (2.01–8.53)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Domicile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>196</td>
<td>53.4</td>
<td>1.00</td>
<td>–</td>
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<tr>
<td>Urban</td>
<td>71</td>
<td>55.5</td>
<td>0.86 (0.56–1.32)</td>
<td>0.489</td>
</tr>
<tr>
<td>Other ethnicity</td>
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<td></td>
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<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>6</td>
<td>35.3</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>Female</td>
<td>14</td>
<td>46.7</td>
<td>1.91 (0.46–7.96)</td>
<td>0.376</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>5</td>
<td>33.3</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>50–59</td>
<td>11</td>
<td>73.3</td>
<td>5.99 (1.09–32.86)</td>
<td>0.039*</td>
</tr>
<tr>
<td>60–69</td>
<td>2</td>
<td>20.0</td>
<td>0.56 (0.08–3.91)</td>
<td>0.558</td>
</tr>
<tr>
<td>≥70</td>
<td>2</td>
<td>28.6</td>
<td>0.78 (0.10–5.92)</td>
<td>0.807</td>
</tr>
<tr>
<td>Domicile</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>1</td>
<td>25.0</td>
<td>1.00</td>
<td>–</td>
</tr>
<tr>
<td>Urban</td>
<td>19</td>
<td>44.2</td>
<td>1.22 (0.10–15.53)</td>
<td>0.879</td>
</tr>
</tbody>
</table>

^Adjusted for gender, age, and domicile; †Significance accepted at p<0.05; ‡ Multivariate analysis excluded 2 Melanesian females for whom age was unknown.
The ethnicity-gender-age-domicile adjusted prevalence of diabetes for adults aged ≥40 years across all of Fiji was 41.0% (95% CI 38.4–43.6): affecting an estimated 99,000 people.

**Discussion**

The majority of non-participants were so for reasons not likely to be associated with HbA1c level or diabetes. Nor was the difference of their mean age (53.1±10.1 years) from that of participants (55.4±10.5 years) likely to be associated with a risk differential of having diabetes. The ethnicity composition was similar for both groups ($\chi^2=3.50$, $p=0.17$). However, nonparticipants were more likely to be male ($p=0.001$), and therefore at lower risk of diabetes. Data from the 511 nonparticipants were not included in the survey analysis.

The use of plasma glucose concentration, either fasting or after oral glucose, is standard clinical practice for the diagnosis of diabetes in individuals. Presenting HbA1c $\geq$6.5% may displace this,$^{10,11,12}$ As a population screening tool, HbA1c has manifest practical advantages over plasma glucose. These include avoiding the imprecisions and inconvenience of self-declared fasting and, when required, a 2-hour glucose value. However, there are concerns about its use in screening,$^{11,12,13}$ including the impact of haemoglobinopathies and iron deficiency anaemia.

The application of HbA1c to population screening has been demonstrated.$^{14,15}$ Further, the DCA 2000+ analyser has shown utility for point of care population-based screening in difficult conditions, and good concordance with laboratory estimates of HbA1c.$^{16}$ Therefore, the FEHS2009 determined to use point of contact HbA1c as a screening test for diabetes, with, understanding the inherent limitations,$^{10,12,17}$ a threshold of $\geq$6.5% for diagnosis.$^{12}$

Although uncommon in this survey, a claimed previous diagnosis of diabetes may be associated with an HbA1c $<6.5\%$, whether due to excellent glycaemia control, mistaken declaration of diabetes or incorrect diagnosis. Consequently, mindful of the limitations of the HbA1c methodology, including the 6.5% threshold, and the possible small over-estimation of accepting a previous diagnosis in those with HbA1c $<6.5\%$, for the purpose of calculating the prevalence of diabetes, the presence of the disease was accepted for every person who claimed previous diagnosis, independent of their HbA1c, and for all others with an HbA1c $\geq 6.5\%$.

The small number (n=28: 2.4%) of participants for whom HbA1c was not recorded was unlikely to significantly influence the calculated prevalence of diabetes. This is particularly given that the gender composition was comparable ($p>0.99$) for the groups with and without HbA1c, that the mean age difference was unlikely to be clinically significant, and that, on multivariate analysis, domicile did not significantly influence the presence of diabetes.

Survey methodology differences—particularly relating to diagnosis of diabetes and age sampling—preclude direct comparison with previous studies in Fiji.$^{6-8,18}$ However, the elevated risk of diabetes for the Indian population remains a constant.
The current study also found increasing age and female gender were associated with greater risk. Although epidemiological transition theory links increasing health risk with increasing urbanisation, and urbanisation continues to increase in Fiji (51% of total population in 2007), urban domicile was not significantly associated with the presence of diabetes in this study.

Logistics dictated that the sample frame was limited to Fiji’s main island. Local advice was that circumstances for most of the people on the other 100 or so permanently inhabited islands (20.9% population) were not materially different from those living away from the larger population centres on Viti Levu. The authors have accepted this, and made extrapolations from the sample to the entire Fijian population aged ≥40 years.

Adjusting for ethnicity, gender, age and domicile, there were approximately 99000 people (41%) with diabetes. This is substantially more than the International Diabetes Federation’s 2010 estimate of 38800 with type 2 diabetes in the 40-79 year age group. The latter excludes the at-risk 0.7% (n=5700) of the population aged ≥80 years. However, this does not account for the difference. It seems likely that the burden of diabetes in Fiji is greater than anticipated.

Competing interests: None.

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References:

Estimating diabetes prevalence in South Auckland: how accurate is a method that combines lists of linked health datasets?

Simon Thornley, Roger Marshall, Gary Jackson, James Smith, Wing-Cheuk Chan, Craig Wright, Dudley Gentles, Rod Jackson

Abstract

Aims To assess the accuracy of a method for estimating adult diabetes prevalence that combines linked, routine health datasets in South Auckland, New Zealand.

Methods We used a simple algorithm that combined records of laboratory testing, drug dispensing and hospital diagnoses applied to National Health Index-linked health data in South Auckland to estimate the prevalence of diabetes in 2007. We investigated the sensitivity of this ‘combined list’ algorithm against a gold standard of individuals with diagnosed diabetes enrolled in a Chronic Care Management programme (CCMP). We also assessed the sensitivity of this algorithm against an estimated diabetes population generated using capture-recapture methods.

Results From the combined-list algorithm, 25,797 (7.2%) South Aucklanders aged 15 years and over had diabetes. During this period, 10,725 patients were enrolled in the CCMP. The combined list algorithm correctly identified (sensitivity) 10,351/10,725 (96.5%) of those enrolled. When we used the capture-recapture estimated diabetes population as an alternative gold standard, 34,418 [9.5%] of South Aucklanders 15 years and over had diabetes, with the sensitivity of the combined list method falling to about 75% (25,797/34,418).

Conclusion Linked health data provide reasonably accurate estimates of diabetes prevalence in a New Zealand population; particularly for cases with longstanding or complicated disease.

With the global rise of obesity and Type 2 diabetes in the last 30 years in New Zealand and elsewhere, timely and accurate estimates of the burden of this disease are required to plan health interventions and assess their influence on disease prevalence. In areas where prevalence of diabetes is thought to be high, reliable estimates are required.

In New Zealand, the highest concentration of diabetes is likely to be in Counties Manukau District Health Board (CMDHB), given the high proportion of Māori and Pacific peoples; and the high level of socioeconomic deprivation in this district health board (DHB) population. This administratively defined region encompasses most of South Auckland, and is one of 21 geographically defined DHBs in New Zealand, with a population of about 464,500.

Accurate, cost-effective and timely measurement of diabetes prevalence in CMDHB would help determine the effect of prevention programmes by allowing time trends to be studied. Such information has been extrapolated from national health status.
surveys that allow regional estimates to be calculated, by assuming nationally aggregated ethnic specific rates of disease apply to a local population. Such methods are, however, limited in a number of ways. For example, health status surveys are potentially biased by high levels of non-response (e.g. the 2006/07 New Zealand Health Survey had a non-response rate of 32% in adults\(^2\)). People who do not respond to such surveys, generally, have poorer health than responders. Moreover, the national survey is only carried out every 4 to 5 years, so that few measurements are available for time trend analyses.

Analysis of health care data may more accurately estimate disease prevalence than survey methods, given that its quality has recently improved substantially. From 2005, the proportion of records linked to the National Health Index (NHI)—a unique national individual identifier—has been greater than 90%. Linked health data now allows us to study population trends in health care in New Zealand by combining records of hospital diagnoses from admissions, drug dispensing and laboratory test events, along with mortality. Linked data of primary care enrolments and outpatients attendances are also available, although the coverage is less comprehensive.

These data can generate population prevalence estimates of diabetes using two alternative methods. The first is an individual “combined list estimate”, or algorithm, that counts the number of people with diabetes-related activities appearing on different databases (diagnosis at hospital discharge, history of laboratory tests, or pharmaceutical dispensing). An alternative approach uses model based ‘capture-recapture’ methods that can estimate the extent of undercount associated with the ‘combined-list’ procedure.\(^3,4\)

The strength of the former (combined list) approach is that it requires fewer assumptions to be made, and simply counts individuals who are recorded with indicators of diabetes related care in health service databases. It is more likely to identify people with more serious conditions that result in more contact with health services.

The alternative (capture-recapture) approach has some important advantages over the combined list approach but also important weaknesses. The strength of capture-recapture methods is their potential to estimate the number of people who are missing from databases, either because of incomplete recording or because they have never been identified. Their main weakness, however, is the requirement to assume that the counts from lists, along with intersections between lists, conform to specific probability distributions (Poisson) specified in statistical (log-linear) models.

Therefore capture-recapture estimates need to be interpreted with considerable caution and we have used this method as a secondary validation tool, rather than as our primary method, for estimating diabetes prevalence.

Methods

Background and study design—We used routine health data sources to calculate the prevalence of diabetes in residents of CMDHB, identifying the same individual on each database by their NHI number. Every New Zealander has a unique NHI alphanumeric code that is encrypted and used to anonymously link different databases. This identifier is now linked to most routinely collected national health databases. Appearance in at least one of these databases defined a denominator population, from which, cases of diabetes are presumed to occur. Then, cases of diabetes were drawn from a subset of this population.
To define the denominator population, we selected people who appeared on any of the following databases: drug dispensing, hospital discharge diagnoses (the National Minimum Data Set or NMDS), primary care enrolment or laboratory test claims. This population is the ‘health contact population’ for CMDHB in 2007, and forms the denominator for all subsequent calculations. Individuals who had a death recorded in mortality data during this period were removed.

The population was thus limited to those residing within CMDHB, who had appeared in at least one of the previously mentioned data sources in 2007, were aged ≥15 years, and were alive throughout the year. Ethnicity data was taken from 2nd quarter 2007 primary health organisation (PHO) enrolment using a prioritised method in the following order—Māori, Pacific, South Asian and Other. The “Other” ethnicities group was comprised mainly of New Zealand Europeans, with only small numbers of non-European ethnic groups. Following usual practice, we have, therefore, combined these groups under the ‘Other’ category. NZDep2006, an area-based measure of socioeconomic deprivation, was derived from the census area unit recorded in PHO enrolment data.

**Diabetes prevalence using the combined list method**—A diagnosis of diabetes in an individual was determined by a “combined-list” method. That is, an individual was classified as having diabetes if they appeared on at least one of the following three databases:

- Diabetes hospital discharge diagnosis (1998–2007) with ICD 10 codes of E10-E14 or ICD-9 equivalent 250 (diabetes codes), or O24.0 to O24.3 (diabetes in pregnancy) excluding all ICD 10:O24.4 (diabetes arising in pregnancy);
- Three or more HbA1c test claim records during 2006 and 2007 in the laboratory claims database;
- Dispensing of diabetes drugs (May 2005 to 2007) including insulin and all oral hypoglycaemic agents (metformin, glibenclamide, gliclazide, glipizide, and pioglitazone).

The combined list prevalence estimate was then calculated as the number of people with diabetes divided by the health contact population.

**Chronic care management diabetes population**—We investigated the sensitivity of the combined list estimate of prevalence against a population known to have diabetes, from their enrolment in the CMDHB Chronic Care Management (CCMP) programme (taken from 2001 until the end of 2007). To qualify for inclusion in CCMP, individuals must have either Type 1 or Type 2 diabetes with evidence of increased risk for macrovascular outcomes—such as HbA1c ≥9%, blood pressure ≥150/90 mmHg, current smoker, nephropathy (urinary albumin ≥300 mg/L), vision threatening retinopathy, previous cardiovascular disease event, total cholesterol ≥6 mmol/L, or two or more admissions for diabetes to a medical ward for ≥5 bed-days in the last year. Such people are, therefore, more likely to have higher risk of complicated and more advanced disease than people with diabetes who were not enrolled.

**Capture-recapture estimated diabetes population**—A shortcoming of the combined list estimate of prevalence is the possibility that some cases of diabetes may occur but not appear on any of the lists, because they have mild, diet-managed disease; or they have not been diagnosed with diabetes; or because of unreliable recording. In contrast, capture-recapture methods generate an estimated total prevalence without all cases necessarily being recorded in health records. People with diabetes not “caught” or identified on annual lists are estimated by statistical modelling of the degree of overlap between diabetes lists to provide an estimate of the number “not caught”. This number of people with unrecorded diabetes is then added to the number of observed cases to generate the total disease prevalence.

In zoological and epidemiological research, capture-recapture methods may help estimate all ‘cases’ in a population. Biologists initiated these methods to measure the abundance of wildlife populations, by capturing a sample of animals, marking them, and then recapturing a further sample at a later date. An estimate of the total, including the unobserved population, is made by counting the proportion of marked to unmarked animals caught in the second catch. Such techniques have been adapted for epidemiology by using records of health service use as capture occasions. Episodes of “capture” may include hospital discharge diagnoses, drug dispensing, general practitioner diagnosis records, laboratory test use or hospital outpatient diagnosis.

We used the Rcapture utility of the R-project to apply log-linear models to capture-recapture methods. Such models address the problem of dependence between lists that is often encountered in an epidemiological context, where ‘capture’ methods vary and are often closely related to each other. For
example, if a patient is diagnosed with diabetes in hospital, such an event is likely to increase the probability of the subject being treated with diabetes drugs, and consequently, appearing on a second list. In contrast, repeated trapping methods, used to estimate animal abundance, often yield independent samples.

To account for between list dependence, interaction terms, between capture occasions, were included in models. Selecting the model used to assess the total numbers with diabetes in the population involves the trade-off between model fit and parsimony. Initially, models with high order interaction terms were computed, with progressively simpler models assessed.

The minimally adequate model, that best fits the data, with a penalty built in for increasing parameters, was judged by comparing Akaike’s Information Criterion (AIC), Chi-square statistics, and plots of Pearson residuals with predicted values for competing models. Scaled rectangle diagrams (like Venn diagrams) display overlap in the datasets used for the combined list and capture-recapture methods and were plotted using SPAN.3

Capture-recapture information was derived from three separate lists of diabetes patients:

- Pharmaceutical claims for medicines used to treat diabetes;
- Laboratory claims in 1 year with more than 2 HbA1c claims in 1 year; and
- A diagnosis of diabetes in hospital discharge records.

List appearance was limited to the 2007 calendar year, unlike in the combined list analysis, to allow for the possible reversal of diabetes status; to standardise sampling; and to conform to the assumption of a closed population (to simplify model construction).

Results

We identified 359,413 people aged ≥15 years living in CMDHB with a health contact in 2007, however, 2437 died in that year, leaving 356,976 individuals for analysis as the denominator population (similar to the 2006 census extrapolated estimate of about 348,000 in this age band). Of this population, 10,600 (3.0%) had a diagnosis of diabetes recorded in the last 9 years in hospital records. 17,991 (5.0%) had three or more HbA1c tests recorded in the 2-year interval between 1 Jan 2006 and 31 Dec 2007; and 21,633 (6.1%) had at least one drug dispensing for diabetes-related medication between May 2005 and 31 Dec 2007.

Overall (see Table 1), the combined list estimate of diabetes prevalence was 7.2% (25,797/356,976); compared with capture-recapture estimates, which were about 30% higher at 9.5% (34,418/356,976). Marked variation occurred in the prevalence of diabetes by age and ethnic group for both the combined list and capture-recapture estimates. In middle-age (35 to 54) the combined list frequency of diabetes is between 1 in 20 and 1 in 10, then it increases to between one in seven to 1 in 5 people in the 55 to 75 age group.

Of the ethnic groups, Pacific and South Asian people had the highest prevalence by both estimation methods. South Asian people show the largest difference between the two estimation methods (12.3% by combined list compared to 20.5% by capture-recapture); but the precision of the South Asian capture-recapture prevalence is poor due to small numbers. There was no evidence of an increasing socioeconomic status gap between the combined list and capture-recapture estimates.

Of those in the denominator (health contact) population, 10,725 individuals (Table 1) were enrolled in the diabetes CCM module until the end of 2007. The combined list correctly identified (sensitivity) 10,351/10,725 (96.5%) of these people.
Considerable overlap was observed between the three diabetes indicators, and those known to have diabetes in the CCMP diabetes register (Figure 1). The areas of the three coloured boxes represent the size of each of the three lists used to define diabetes status, while the numbers relate to the mutually exclusive non-overlapping or overlapping categories within the combined lists. Most people were identified from drug dispensing records.

Table 1. Sociodemographic characteristics of people with diabetes: i. ‘diagnosed’ by the Combined-list (algorithm) method; ii. estimated by the Capture-recapture method; iii. enrolled in the Chronic Care Management (CCMP) diabetes programme: presented as numbers (and percentages) of the 2007 Counties Manukau population aged 15 years and over (n=359,413)

<table>
<thead>
<tr>
<th>Variables</th>
<th>i. Combined list estimates of diabetes prevalence: n (%)*‡‡</th>
<th>ii. Capture-recapture estimates of diabetes prevalence: % (95% CIs) ††</th>
<th>iii. Patients with diabetes enrolled in the CCMP: n (%) ‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>13,223 (7.0)</td>
<td>9.3 (8.6 to 10.2)</td>
<td>5491 (2.9)</td>
</tr>
<tr>
<td>Male</td>
<td>12,572 (7.4)</td>
<td>10.0 (9.3 to 11.1)</td>
<td>5234 (3.1)</td>
</tr>
<tr>
<td>Age category</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 to 24</td>
<td>496 (0.7)</td>
<td>1.0 (0.24 to 1.8)</td>
<td>106 (0.1)</td>
</tr>
<tr>
<td>25 to 34</td>
<td>1221 (1.9)</td>
<td>2.7 (2.0 to 5.4)</td>
<td>360 (0.6)</td>
</tr>
<tr>
<td>35 to 44</td>
<td>3258 (4.5)</td>
<td>7.3 (5.7 to 11.2)</td>
<td>1318 (1.8)</td>
</tr>
<tr>
<td>45 to 54</td>
<td>5487 (9.2)</td>
<td>12.7 (11.0 to 15.9)</td>
<td>2446 (4.1)</td>
</tr>
<tr>
<td>55 to 64</td>
<td>6863 (15.9)</td>
<td>20.0 (18.3 to 22.7)</td>
<td>2955 (6.8)</td>
</tr>
<tr>
<td>65 to 74</td>
<td>5264 (20.6)</td>
<td>27.2 (24.5 to 31.4)</td>
<td>2289 (9.0)</td>
</tr>
<tr>
<td>over 75</td>
<td>3208 (17.8)</td>
<td>22.3 (20.2 to 25.5)</td>
<td>1251 (6.9)</td>
</tr>
<tr>
<td>Ethnic group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>3310 (7.6)</td>
<td>8.9 (8.2 to 10.0)</td>
<td>1737 (4.0)</td>
</tr>
<tr>
<td>Pacific</td>
<td>7661 (11.1)</td>
<td>13.7 (12.6 to 15.4)</td>
<td>4013 (5.8)</td>
</tr>
<tr>
<td>South Asian</td>
<td>2510 (12.3)</td>
<td>20.5 (15.3 to 36.9)</td>
<td>917 (4.5)</td>
</tr>
<tr>
<td>Other†</td>
<td>12,316 (5.5)</td>
<td>7.6 (7.0 to 8.6)</td>
<td>4058 (1.8)</td>
</tr>
<tr>
<td>NZDep2006#</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 and 2 least deprived</td>
<td>3413 (5.1)</td>
<td>7.3 (6.1 to 9.7)</td>
<td>937 (1.4)</td>
</tr>
<tr>
<td>3 and 4</td>
<td>2668 (5.2)</td>
<td>8.0 (6.6 to 11.1)</td>
<td>785 (1.5)</td>
</tr>
<tr>
<td>5 and 6</td>
<td>2023 (5.5)</td>
<td>7.3 (6.1 to 9.9)</td>
<td>612 (1.7)</td>
</tr>
<tr>
<td>7 and 8</td>
<td>4187 (8.0)</td>
<td>8.0 (6.6 to 11.1)</td>
<td>1741 (3.3)</td>
</tr>
<tr>
<td>9 and 10 most deprived</td>
<td>13,501 (9.0)</td>
<td>11.1 (9.6 to 13.6)</td>
<td>6650 (4.5)</td>
</tr>
<tr>
<td>Total</td>
<td>25,797 (7.2)</td>
<td>9.6 (9.1 to 10.3)</td>
<td>10,725 (2.3)</td>
</tr>
</tbody>
</table>

*Includes 70 persons with unknown gender and 78 persons with unknown NZDep2006 status; †Other is mainly European ethnic group; *NZDep2006 – area based measure of socioeconomic deprivation; ‡ CCMP is the Chronic Care Management Programme for people with diabetes who are considered at high risk of re-admission to hospital; ††As this method provides a modelled assessment of people with diabetes we have presented prevalence information, rather than absolute numbers; ‡‡ CIs not presented for Combined list prevalence estimates as all are less than 0.5%.
The number of people assumed to have diabetes in the three databases used for the combined list estimates were also used to derive the overall capture-recapture estimates of diabetes prevalence. As the data for the latter analyses were restricted to 2007 only (discussed in the Methods section), we used two or more HbA1c tests in 1 year as laboratory evidence of diabetes, compared with the combined list method, in which three tests in 2 years as laboratory evidence of diabetes. The model fit for the 2007 capture-recapture estimate of diabetes prevalence was optimal using a log-linear model that included two-way interactions for all lists.

The best fit (indicated by lowest AIC - see Methods - with least discordance between observed and expected values and plots of residuals and predicted values) was 34,418 (95% CI 32,523 to 36,812). This translates to an estimated diabetes prevalence of 9.6%, or about 8,600 more cases compared to the combined list method. If the
capture-recapture results (point estimate) were assumed to be the best estimate of the number of people with diabetes in this population, then the combined list method would have identified 75% (25,797/34,418) of all people aged 15 years and over with diabetes in the CMDHB.

Discussion

Our method for estimating the prevalence of diabetes, that combines linked routine health databases, provides a reasonably accurate estimate of known diabetes prevalence. Such a method shows good concordance with capture-recapture estimates and a list of people enrolled in a diabetes management programme. Local surveys have reported diabetes prevalence consistent with our combined-list estimates. For example, the New Zealand Health Survey 2006/7, reported an adult (age ≥15 years) prevalence of diagnosed diabetes in CMDHB of 26,400, very similar to our combined-list prevalence (25,797).

As the combined list method can only identify people known to have diabetes – because they have to be recorded in a routine health dataset for a ‘diabetes-related’ activity - it will miss undiagnosed diabetes. This shortcoming is illustrated by the higher estimated prevalence using the capture-recapture method, that is able to estimate the size of this unidentified group. When compared to the capture-recapture estimate, the undercount using the combined list approach was about 25%, or 8600 people with diabetes.

The gap we observed between the two methods of estimating diabetes prevalence is consistent with an earlier (2002/03) community survey, undertaken in the same geographic region, that reported an undercount of 33% in European, 25% for Māori and 17% for Pacific, and a weighted mean undercount of about 28%. Taken together, these estimates suggest that undiagnosed diabetes rates have been relatively constant in the interval years, despite effort to improve rates of diabetes detection.

Gaps between the combined list and capture-recapture prevalence may help identify subgroups of the population with high levels of undiagnosed diabetes. The largest gaps observed between the two prevalence estimates were for South Asian people although the precision of the capture-recapture estimates were poor. To better assess such gaps, we plan to combine data from several years or use larger populations to generate the necessary statistical power to investigate this observation further.

However, if our estimates were correct, a significant population of about 1,700 South Asian people in CMDHB, had diabetes, but were not diagnosed during 2007.

The interpretation of the apparent differences in prevalence between the two methods is limited by the validity of capture-recapture methods, which is contingent on several underlying assumptions, we introduced earlier. One assumption, of a closed population, is approximated here by limiting records of health care use to one year. Another includes between-list independence, which is questionable in an epidemiological context, as the lists used in the modelling are, frequently, not independent. We addressed this problem with log-linear models, that has been discussed at length in another summary. In short, such models are designed to account for between list dependence through the inclusion of interaction terms, with our final model including beta coefficients for all two-way dependence between the three lists. With three lists used in the capture-recapture, this level of dependence was
the maximum possible, given that one interaction term (three-way in our study) must be dropped, to solve the equation. Capture-recapture models are also dependent on the assumption that counts of people with diabetes on lists, and between-list intersections, are distributed according to a Poisson probability model. Therefore, the capture-recapture prevalence we calculated should be interpreted with considerable caution, and we do not suggest using such results as the best estimate of diabetes prevalence.

The combined list method will produce more conservative estimates of diabetes prevalence and some people with mild diabetes, managed by diet alone, are likely to be missed. We compared the combined list method to a gold standard of patients with diabetes enrolled in a chronic care management programme. CCMP patients represent a subgroup of people with diabetes with increased risk of complications given the eligibility criteria for the programme. Using this standard, our combined list method performed well, which is not surprising because, as discussed above, the combined list method is more likely to identify people with more longstanding and complicated disease.

The use of historical data, and differing time periods for each dataset (using as much data as was available), could potentially lead to mismatches, for example through out-migration. We controlled for this by ensuring that individuals were all present in CMDHB in 2007 for at least one health contact. Also, other work has shown that as the health datasets improve in quality over time, the health contact population begins to approximate Census-based estimates.

Both methods prevalence estimates assume that the diabetes-related activities recorded in the three databases accurately identify people with the disease. The weakest indicator of a diabetes diagnosis is likely to be that based on HbA1c test records. These may be requested (repeatedly) in patients suspected, but not fulfilling, the diagnostic criteria for diabetes. Although not recommended as a screening test in national guidelines, HbA1c tests are increasingly used for this purpose. When used to monitor glycaemic control, protocols recommend a 3 to 6 monthly testing interval. We used three HbA1c tests in 2 years to define diabetes for the combined list estimate, based on a receiver operating characteristic curve, which used CCMP status as the ‘gold standard’ comparator (Figure 2). The curve shows that optimal performance (assuming the relative costs of false positives to false negatives are identical) is achieved at a level of three, per 2-year period. Such performance may change as HbA1c testing becomes more frequently used to screen for diabetes status (already recommended in the US).

Of the people identified with diabetes using the combined list, 10.5% (2,714/25,797) were defined as a result of HbA1c test records only. An arguably, stricter definition of diabetes, based on two HbA1c measures in 1 year rather than three in 3 years, was applied to the capture-recapture definition, as the databases used were all restricted to 1 year.
Internationally, diabetes prevalence studies have reported both combined list and capture-recapture methods, although not together. The combined list estimate method has been used previously in Denmark\(^{12}\) to describe time trends in diabetes prevalence between 1995 and 2006. The researchers used a similar technique to the one we describe; however, five or more blood glucose measurements in a year, rather than three HbA1c measures in 2 years, were used as laboratory evidence of diabetes. They also included diabetes outpatients visits as one of their lists. A similar prevalence study from Ontario was based on a rule that included a diabetes diagnosis in hospital discharge or outpatient records in the last 2 years.\(^{13}\) The sensitivity of 96.5% for the combined list method in our study is higher than that reported in the Danish (85%)\(^{12}\) and Canadian (86%) studies,\(^{14}\) but, as discussed, this may be overestimated due to the
severity of disease in our gold standard group of patients in the CCMP. Our capture-recapture result suggests the combined list method has a sensitivity of about 75% for all diabetes.

The Casale Monferrato study in northwest Italy used capture-recapture log-linear models, similar to ours, to monitor the incidence of diabetes between 1988 and 2000.\textsuperscript{15} Data from diabetes clinics, hospital discharge records, prescribing and sales of reagents and strips were combined to calculate population estimates. Comparing individual to capture-recapture methods, they found a case-ascertainment of about 80%; which was consistent with our estimate of 75%. In the United Kingdom, similar rates of list undercount to the Italian study have been reported in capture-recapture studies of diabetes prevalence.\textsuperscript{4,16}

Our study shows that diabetes prevalence, derived from combining linked health databases, is similar to survey-based estimates in South Auckland. Further, the discrepancy between combined list and capture-recapture diabetes prevalence estimates appears greatest among people who identify as South Asian. In this area, primary care screening programmes for diabetes are currently targeting Pacific and Māori people, yet the greatest proportion of undiagnosed diabetes may now exist in the South Asian population.

Given the small numbers of people in many ethnic subgroups in our study, we had insufficient power to investigate these groups adequately, and so plan to repeat the current study in larger populations. In addition, the incomplete nature of the CCMP diabetes list did not allow us to estimate the specificity of the combined list method. We therefore plan to examine this issue with a more complete “gold standard” list of people with diagnosed diabetes drawn from a large, primary care database.

\textbf{Competing interests:} None.

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References:
Assessment of obesity in New Zealand Chinese: a comparative analysis of adults aged 30–39 years from five ethnic groups

Ji Y J Wen, Elaine C Rush, Lindsay D Plank

Abstract

**Aims** To compare the relationships between body mass index (BMI) and percentage body fat (%BF) in New Zealand Chinese aged 30–39 years and their European, Māori, Pacific Island, and Asian Indian counterparts.

**Methods** Healthy Chinese (M20, F23) were selected to cover a wide range of BMI. European (M29, F37), Māori (M23, F23), Pacific Island (M15, F23), and Asian Indian (M29, F25) volunteers were drawn from existing data. Total and regional body fat and arm and leg lengths were measured by dual-energy X-ray absorptiometry.

**Results** For fixed BMI, Chinese had a higher %BF than European and less %BF than Asian Indian. At a %BF equivalent to a BMI of 30 kg/m\(^2\) in Europeans (WHO threshold for obesity), BMI values for Asian Indian and Chinese women were 5.8 and 2.2 units lower than European, respectively, and for Asian Indian and Chinese men, 8.2 and 3.0 units lower. Chinese had relatively shorter arm and leg lengths than Asian Indians and Europeans with a significantly higher ratio of central fat mass to limb fat mass.

**Conclusions** The %BF-BMI relationships for Asian Indian and Chinese differ from Europeans and from each other and different BMI obesity thresholds may be required for these Asian ethnic groups.

The body mass index (BMI) classifications for overweight (≥25 kg/m\(^2\)) and obesity (≥30 kg/m\(^2\)) recommended by the World Health Organization (WHO) are intended as thresholds for reflecting risk for Type 2 diabetes and cardiovascular disease.\(^1\) The intention is that they are used as a basis for informing health policy and triggering public health actions. They were based, however, on the relationship between BMI and morbidity and mortality in Western populations and the WHO recognised that the risk for Asian populations was higher at lower BMI.\(^1\)

There was also recognition that the cut-offs for observed risk varied across Asian populations and BMI thresholds specific to different Asian populations were not advocated by the WHO. For these populations, retention of the ‘Western’ threshold for obesity may mask the true prevalence of this condition and the associated disease risk.

Immigrant Asian populations in New Zealand (NZ) as quantified in the most recent census (2006) are dominated by Chinese (147,570 or 3.7% of the total population) and Asian Indian (2.6%) with other Asians making up 2.2%.\(^2\) The NZ Ministry of Health applied BMI obesity cut-offs of 30 and 25 kg/m\(^2\) to Asian (15+ years of age)
data collected in the 2002/3 NZ Health Survey\(^3\) acknowledging the lower obesity cut-off of 25 suggested by the 2004 WHO consultation on Asian populations.\(^1\)

For Chinese males obesity prevalence increased from 4.0 to 20.1% for these respective cut-offs. For Asian Indian males the corresponding prevalence data were 7.1 and 34.2%. Such a reduction in the obesity cut-point clearly has significant implications for health policy. The Ministry of Health acknowledged the limited data supporting BMI cut-offs for obesity that are specific for Asians and for Asian ethnic subgroups.

Studies carried out in Singapore\(^4\) and Canada\(^5\) provide the only published data we are aware of that directly compare immigrant Chinese and Asian Indian populations. Among Singaporean adults aged 18–75 y, for the same BMI, Asian Indians had markedly higher percent body fat (%BF) compared to Chinese.\(^4\) BMI cut-offs for obesity of about 26 for Indians and 27 for Chinese were equivalent in %BF terms to a BMI of 30 for Europeans.

Differences between these ethnic groups in body build (frame size and leg length) appeared to explain these results. The Canadian study of adults aged 30–65 y showed that Asian Indians had more body fat in absolute terms and Chinese less body fat than their European counterparts at a given BMI.\(^5\)

We recently published a comparison of the %BF-BMI relationship in NZ European, Māori, Pacific Island and Asian Indian ethnic groups.\(^6\) This highlighted the marked differences between Asian Indians and Europeans in this relationship with a BMI of 26 in Asian Indian women and 24 in Asian Indian men suggested as the appropriate obesity cut-points. The present study was designed to extend this comparison to NZ-domiciled Chinese.

Specifically, in adults aged 30–39 y we aimed to:

- Compare the BMI-body fatness relationships in NZ Māori, Pacific Island, Asian Indian and European ethnic groups with NZ Chinese; and
- Compare data on muscularity, bone mass, fat distribution and leg length in Chinese with those from the other ethnic groups.

**Methods**

Ethics approval for this study was obtained from the Auckland University of Technology Ethics Committee, and informed written consent was provided by all participants.

Recruitment of Chinese volunteers was designed to achieve similar numbers in both male and female groups between the ages of 30 and 39 y and to achieve a wide range of BMI in each group. Recruitment was by personal contact, advertisement, or through existing networks of the recruiter (JW). Chinese ethnicity was self-defined and was at least the ethnic group of all 4 grandparents. Chinese was used to describe people of Chinese origin from mainland China, Taiwan, Singapore or Malaya. All participants needed to have been resident in New Zealand for at least 3 years to allow adaptation to the local environment (including food supply and physical activity patterns). Women who were breastfeeding, pregnant or likely to be pregnant were excluded as were individuals who lifted weights more than once a week, had major health conditions, were unwell at the time of the measurements, or used anabolic steroids or other drugs that may affect body composition.

Participants visited the body composition laboratory in the Department of Surgery, University of Auckland, between December 2006 and March 2007. Height and weight were measured with participants wearing light clothing and no shoes. An estimated clothing weight was subtracted. Whole-body composition (fat mass, fat-free soft tissue and bone mineral content [BMC]) and whole-body
bone mineral density (BMD) measurements were made using a single dual-energy X-ray absorptiometry (DXA) machine (model DPX+ with software version 3.6y, GE-Lunar, Madison, WI) with subjects lying supine in light clothing. Fat-free mass was estimated as the sum of fat-free soft tissue and BMC. %BF was calculated as 100 × fat mass / (fat mass + fat-free mass).

Regional analysis of the DXA scans was carried out to obtain appendicular skeletal muscle mass (ASMM), calculated as total leg and arm (appendicular) mass minus leg and arm fat mass and wet bone mass. The latter was estimated as BMC divided by 0.55. Central fat mass was defined as total fat mass minus appendicular fat mass. Abdominal fat was obtained from analysis of a region of interest positioned with the lower horizontal border on top of the iliac crest and the upper border approximately parallel with the junction of the T12 and L1 vertebrae. The sides of this region were adjusted to include the maximum amount of abdominal tissue. A region of interest of identical height placed over the thighs with the upper horizontal border positioned immediately below the ischial tuberosities was used to obtain fat content of the thighs. The lateral margins were adjusted to follow the shape of the thighs. Bone lengths were determined using the right side of the DXA image. Lengths of the humerus, radius, femur, tibia, and total skeletal length were calculated from the x and y co-ordinates on the digitized image of proximal and distal points on the bones. Leg bone length was defined as the sum of femur and tibia bone lengths, while arm bone length was defined as the sum of humerus and radius bone lengths. Total skeletal length (DXA height) was measured as the distance from the apex of the cranium to the plantar surface of the calcaneus bone.

These same techniques were applied in previously published work comparing European, Māori, Pacific Island and Asian Indian ethnic groups. Subjects aged 30–39 y were extracted from these data and used for comparison with the Chinese participants in the current study.

The results are expressed as mean ± SD unless stated otherwise. Between-group differences in subject characteristics were tested using one-way ANOVA followed by pairwise comparisons if a significant F test was obtained. Analysis of covariance was used to adjust body composition results for comparison across ethnic groups. As there is a curvilinear relationship between %BF and BMI, BMI was log-transformed before linear regression analysis. Similarity of regression slopes among the ethnic groups was verified by examining the significance of the interaction between the covariate(s) and the group variable. Data were analysed using SPSS, version 14.0 (SPSS Inc, Chicago, IL). Results with P values <0.05 were considered significant.

Results

Forty-three Chinese (23 female, 20 male) were recruited and underwent the body composition analysis. Their characteristics are summarised in Tables 1 and 2 along with the previously acquired data from European, Māori, Pacific Island and Asian Indian ethnic groups in the 30–39 y age range. Chinese women were lighter than their European, Māori, Pacific Island and Asian Indian counterparts with lower %BF than Māori, Pacific Island and Asian Indian women. Chinese men were lighter than European, Māori and Pacific Island men while their %BF was lower than Asian Indian but not significantly different from European, Māori and Pacific Island men.

After adjustment for body weight and height, the Chinese women and men had significantly less FFM, ASMM and BMC than their European, Māori and Pacific counterparts and significantly more FFM than the Asian Indian women and men (Table 3). Controlling for weight, BMD did not differ significantly between Chinese and European women and men. Chinese men had significantly higher BMD than Asian Indian men. Among the five ethnic groups, after adjustment by DXA height, Chinese had the shortest leg and arm bone lengths.

After adjustment for weight and height, abdominal fat mass was significantly higher in Chinese than European, Māori and Pacific women and men and thigh fat mass was significantly lower in Chinese than Asian Indian men and women but not significantly different from European, Māori and Pacific women and men (Table 3).
### Table 1. Characteristics of 131 women aged 30–39 y from five ethnic groups in New Zealand

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Age (y)</td>
<td>34 ± 3 (30-39)</td>
<td>34 ± 2 (30-38)</td>
<td>34 ± 3 (30-39)</td>
<td>35 ± 3 (30-39)</td>
<td>36 ± 2 (31-39)</td>
<td>0.142</td>
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<tr>
<td>Weight (kg)</td>
<td>66.5 ± 13.6 (49.1-102.0)*</td>
<td>79.1 ± 16.5 (52.2-108.3)*</td>
<td>89.1 ± 19.0 (60.2-136.9)*</td>
<td>67.3 ± 11.3 (47.1-91.9)*</td>
<td>54.5 ± 6.7 (44.4-67.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>164.9 ± 5.9 (151.5-176.5)*</td>
<td>161.9 ± 5.4 (150.5-173.0)</td>
<td>162.7 ± 5.8 (153.5-172.5)</td>
<td>158.7 ± 5.3 (142.5-166.4)</td>
<td>160.0 ± 5.3 (150.8-169.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg.m⁻²)</td>
<td>24.5 ± 5.2 (18.6-39.4)</td>
<td>30.1 ± 5.9 (20.9-40.5)*</td>
<td>33.6 ± 6.7 (22.6-46.0)*</td>
<td>26.9 ± 5.3 (17.7-38.1)*</td>
<td>21.2 ± 2.1 (18.1-26.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>44.38 ± 4.70 (35.20-58.54)*</td>
<td>46.82 ± 5.91 (37.90-57.83)*</td>
<td>52.26 ± 8.69 (34.38-72.64)*</td>
<td>37.26 ± 3.44 (31.66-43.12)</td>
<td>37.51 ± 3.15 (32.45-42.19)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>22.16 ± 10.88 (9.21-49.13)</td>
<td>32.45 ± 11.83 (14.70-53.99)</td>
<td>36.74 ± 12.61 (16.74-62.89)</td>
<td>29.86 ± 9.71 (11.95-50.72)</td>
<td>16.38 ± 4.53 (7.93-24.95)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FM (%)</td>
<td>31.7 ± 9.4 (16.6-50.4)</td>
<td>39.8 ± 7.2 (27.7-53.9)*</td>
<td>40.3 ± 7.4 (27.1-53.4)*</td>
<td>43.4 ± 7.7 (25.7-56.7)*</td>
<td>29.9 ± 5.5 (17.7-37.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMC (kg)</td>
<td>2.58 ± 0.32 (1.97-3.32)*</td>
<td>2.62 ± 0.31 (2.13-3.21)*</td>
<td>2.88 ± 0.40 (2.02-3.78)*</td>
<td>2.23 ± 0.26 (1.69-2.63)</td>
<td>2.21 ± 0.21 (1.84-2.72)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMD (g cm⁻²)</td>
<td>1.16 ± 0.08 (1.05-1.33)</td>
<td>1.19 ± 0.08 (1.05-1.37)</td>
<td>1.28 ± 0.09 (1.04-1.41)*</td>
<td>1.15 ± 0.08 (1.00-1.28)</td>
<td>1.14 ± 0.06 (1.04-1.29)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ASMM (kg)</td>
<td>17.43 ± 2.42 (12.91-24.77)*</td>
<td>18.27 ± 2.77 (13.86-23.61)*</td>
<td>20.48 ± 3.40 (13.58-26.83)*</td>
<td>14.61 ± 1.70 (11.30-17.29)</td>
<td>13.99 ± 1.34 (12.07-15.96)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ASMM (%)</td>
<td>26.7 ± 4.0 (19.5-34.1)</td>
<td>23.4 ± 2.7 (17.7-27.0)*</td>
<td>23.4 ± 3.1 (17.8-30.2)*</td>
<td>22.2 ± 3.4 (15.9-28.0)*</td>
<td>26.1 ± 2.0 (23.2-30.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Central FM/ApFM</td>
<td>0.95 ± 0.23 (0.51-1.60)*</td>
<td>1.20 ± 0.20 (0.87-1.61)</td>
<td>1.18 ± 0.16 (0.94-1.47)</td>
<td>1.06 ± 0.21 (0.77-1.59)*</td>
<td>1.36 ± 0.27 (0.91-2.02)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM (kg)</td>
<td>1.54 ± 1.11 (0.33-4.19)</td>
<td>2.80 ± 1.29 (1.08-5.46)*</td>
<td>3.04 ± 1.27 (1.15-6.20)*</td>
<td>2.57 ± 1.04 (0.58-4.83)*</td>
<td>1.40 ± 0.46 (0.46-2.13)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM (% of FM)</td>
<td>6.3 ± 1.8 (3.5-9.5)*</td>
<td>8.4 ± 1.2 (5.8-10.2)</td>
<td>8.1 ± 1.1 (5.9-11.4)</td>
<td>8.4 ± 1.5 (4.8-10.7)</td>
<td>8.4 ± 1.1 (5.8-10.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM (% of MAR)</td>
<td>29.6 ± 12.4 (9.0-53.2)</td>
<td>42.8 ± 8.3 (26.7-55.9)*</td>
<td>42.5 ± 8.2 (26.8-57.1)*</td>
<td>45.6 ± 8.8 (19.8-57.6)*</td>
<td>33.8 ± 8.1 (15.3-44.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thigh FM (kg)</td>
<td>2.39 ± 0.97 (1.15-5.09)*</td>
<td>3.19 ± 0.97 (1.72-4.97)*</td>
<td>3.39 ± 1.04 (1.71-5.29)*</td>
<td>3.22 ± 1.02 (1.77-6.01)*</td>
<td>1.69 ± 0.37 (1.02-2.33)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thigh FM (% of FM)</td>
<td>11.4 ± 1.8 (7.8-15.0)</td>
<td>10.1 ± 1.4 (8.2-12.5)</td>
<td>9.4 ± 0.9 (8.1-11.4)</td>
<td>10.9 ± 1.5 (8.4-14.8)</td>
<td>10.6 ± 1.7 (6.8-14.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM/Thigh FM</td>
<td>0.59 ± 0.25 (0.24-1.20)*</td>
<td>0.86 ± 0.22 (0.47-1.20)</td>
<td>0.88 ± 0.18 (0.58-1.40)</td>
<td>0.80 ± 0.23 (0.33-1.27)</td>
<td>0.83 ± 0.23 (0.39-1.59)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Arm length (cm)</td>
<td>52.51 ± 2.98 (46.90-58.04)*</td>
<td>51.40 ± 2.73 (47.43-56.82)*</td>
<td>53.37 ± 2.93 (46.99-57.90)*</td>
<td>52.51 ± 2.84 (45.73-58.90)*</td>
<td>49.09 ± 2.79 (43.78-53.31)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Leg length (cm)</td>
<td>77.24 ± 4.45 (67.79-87.73)*</td>
<td>74.38 ± 4.44 (66.64-88.82)</td>
<td>77.57 ± 3.97 (68.23-85.45)*</td>
<td>75.51 ± 3.82 (64.34-82.57)</td>
<td>73.90 ± 3.37 (69.23-83.82)</td>
<td>0.003</td>
</tr>
</tbody>
</table>

**Note:** Values are mean ± SD. Range in parentheses. *Significantly different to Chinese (P<0.05). **Abbreviations:** BMI, body mass index; FM, fat mass; BMC, bone mineral content; BMD, bone mineral density; ASMM, appendicular skeletal muscle mass; ApFM, appendicular fat mass; AbFM, abdominal fat mass; MAR, mass of abdominal region.
Table 2. Characteristics of 116 men aged 30–39 y from five ethnic groups in New Zealand

<table>
<thead>
<tr>
<th>Variable</th>
<th>European (N=29)</th>
<th>Māori (N=23)</th>
<th>Pacific (N=15)</th>
<th>Asian Indian (N=29)</th>
<th>Chinese (N=20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>34 ± 2 (30-38)*</td>
<td>33 ± 3 (30-39)*</td>
<td>34 ± 2 (31-38)</td>
<td>35 ± 3 (30-39)</td>
<td>36 ± 2 (30-39)</td>
<td>0.017</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>79.8 ± 7.8 (64.0-97.4)*</td>
<td>88.2 ± 14.0 (62.0-114.8)*</td>
<td>92.1 ± 9.1 (81.5-108.0)*</td>
<td>72.6 ± 12.6 (48.5-106.4)*</td>
<td>70.5 ± 11.3 (46.6-95.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.5 ± 6.5 (166.0-191.0)*</td>
<td>174.3 ± 6.8 (156.0-185.0)</td>
<td>171.3 ± 6.7 (154.0-179.0)</td>
<td>169.1 ± 7.7 (151.4-181.5)</td>
<td>171.0 ± 4.4 (162.7-177.2)</td>
<td>0.001</td>
</tr>
<tr>
<td>BMI (kg.m⁻²)</td>
<td>25.6 ± 2.1 (21.4-30.9)</td>
<td>29.1 ± 4.5 (18.9-37.7)*</td>
<td>31.5 ± 4.1 (26.0-42.2)*</td>
<td>25.4 ± 4.4 (18.7-39.6)</td>
<td>24.1 ± 3.5 (17.5-32.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>63.23 ± 6.69 (51.74-83.22)*</td>
<td>66.47 ± 8.03 (52.36-82.31)*</td>
<td>68.81 ± 3.86 (59.87-74.17)*</td>
<td>49.35 ± 7.96 (30.58-64.68)</td>
<td>54.28 ± 6.08 (41.25-64.74)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>17.12 ± 6.82 (4.82-26.47)</td>
<td>22.38 ± 8.93 (7.15-39.30)</td>
<td>23.66 ± 7.33 (11.39-37.48)*</td>
<td>23.39 ± 8.58 (7.87-48.09)*</td>
<td>16.03 ± 7.08 (4.57-32.24)</td>
<td>0.001</td>
</tr>
<tr>
<td>FM (%)</td>
<td>21.0 ± 7.6 (6.3-33.2)</td>
<td>24.5 ± 7.0 (11.4-34.9)</td>
<td>25.2 ± 5.6 (13.6-35.3)</td>
<td>31.6 ± 7.7 (14.0-50.5)*</td>
<td>21.9 ± 7.2 (9.7-35.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMC (kg)</td>
<td>3.35 ± 0.43 (2.65-4.38)*</td>
<td>3.47 ± 0.37 (2.60-4.18)*</td>
<td>3.44 ± 0.39 (2.60-4.03)*</td>
<td>2.60 ± 0.39 (1.93-3.58)</td>
<td>2.85 ± 0.35 (2.08-3.48)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMD (g.cm⁻²)</td>
<td>1.26 ± 0.08 (1.10-1.44)</td>
<td>1.31 ± 0.08 (1.06-1.41)*</td>
<td>1.33 ± 0.05 (1.25-1.40)*</td>
<td>1.16 ± 0.09 (1.04-1.38)*</td>
<td>1.23 ± 0.09 (1.02-1.37)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ASMM (kg)</td>
<td>26.76 ± 3.25 (20.48-37.68)*</td>
<td>28.48 ± 4.21 (21.42-36.81)*</td>
<td>29.86 ± 2.34 (24.21-33.64)*</td>
<td>21.30 ± 3.68 (12.61-29.97)</td>
<td>22.07 ± 2.83 (16.47-28.16)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ASMM (%)</td>
<td>33.4 ± 3.3 (26.5-38.6)</td>
<td>32.3 ± 3.3 (26.9-40.0)</td>
<td>32.5 ± 3.0 (27.3-37.9)</td>
<td>29.5 ± 3.8 (20.3-36.7)</td>
<td>31.7 ± 3.1 (25.5-36.2)</td>
<td>0.001</td>
</tr>
<tr>
<td>Central FM/ApFM</td>
<td>1.33 ± 0.21 (0.97-1.74)*</td>
<td>1.55 ± 0.29 (0.96-2.14)*</td>
<td>1.38 ± 0.19 (0.95-1.65)*</td>
<td>1.45 ± 0.27 (0.86-1.98)*</td>
<td>1.81 ± 0.40 (1.10-2.56)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM (kg)</td>
<td>1.38 ± 0.67 (0.30-2.60)</td>
<td>2.03 ± 0.95 (0.40-4.16)</td>
<td>2.10 ± 0.83 (0.65-3.44)</td>
<td>2.28 ± 0.94 (0.69-3.53)*</td>
<td>1.58 ± 0.78 (0.31-3.27)</td>
<td>0.001</td>
</tr>
<tr>
<td>AbFM (% of FM)</td>
<td>7.8 ± 1.2 (4.8-9.9)*</td>
<td>8.8 ± 1.4 (5.6-11.7)</td>
<td>8.7 ± 1.5 (5.6-11.4)</td>
<td>9.7 ± 1.2 (6.5-11.5)</td>
<td>9.5 ± 1.3 (6.3-11.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM (% of MAR)</td>
<td>24.5 ± 10.0 (5.7-41.9)</td>
<td>31.2 ± 9.2 (10.7-46.2)</td>
<td>30.0 ± 7.0 (13.1-40.5)</td>
<td>39.1 ± 7.9 (16.7-53.4)</td>
<td>29.6 ± 9.9 (10.9-46.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thigh FM (kg)</td>
<td>1.51 ± 0.56 (0.47-2.48)</td>
<td>1.90 ± 0.68 (0.78-3.22)*</td>
<td>2.03 ± 0.54 (1.07-2.99)*</td>
<td>2.00 ± 0.76 (0.71-3.99)*</td>
<td>1.32 ± 0.51 (0.49-2.39)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thigh FM (% of FM)</td>
<td>9.0 ± 1.0 (6.6-11.4)</td>
<td>8.7 ± 0.9 (7.2-10.9)</td>
<td>8.7 ± 0.9 (7.4-10.9)</td>
<td>8.6 ± 1.3 (6.7-12.5)</td>
<td>8.6 ± 1.6 (6.4-13.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AbFM/Thigh FM</td>
<td>0.88 ± 0.21 (0.42-1.39)*</td>
<td>1.03 ± 0.22 (0.52-1.43)</td>
<td>1.01 ± 0.25 (0.52-1.54)</td>
<td>1.16 ± 0.27 (0.52-1.62)</td>
<td>1.15 ± 0.30 (0.47-1.73)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Arm length (cm)</td>
<td>56.83 ± 2.72 (51.53-62.00)*</td>
<td>57.72 ± 3.44 (49.56-63.92)*</td>
<td>56.78 ± 3.14 (51.18-61.58)*</td>
<td>56.59±2.12 (48.52-66.30)*</td>
<td>53.61 ± 2.62 (49.37-59.70)</td>
<td>0.002</td>
</tr>
<tr>
<td>Leg length (cm)</td>
<td>83.14 ± 3.91 (75.91-89.97)*</td>
<td>81.55 ± 4.86 (68.26-90.29)</td>
<td>81.28 ± 4.92 (72.15-90.45)</td>
<td>81.30 ± 4.02 (74.06-88.42)</td>
<td>79.71 ± 2.87 (74.56-85.98)</td>
<td>0.086</td>
</tr>
</tbody>
</table>

**Note:** Values are mean ± SD. Range in parentheses. *Significantly different to Chinese (P<0.05). **Abbreviations:** BMI, body mass index; FM, fat mass; BMC, bone mineral content; BMD, bone mineral density; ASMM, appendicular skeletal muscle mass; ApFM, appendicular fat mass; AbFM, abdominal fat mass; MAR, mass of abdominal region.
In both women and men, abdominal-to-thigh fat ratio was higher in Chinese than Europeans, but not significantly different from Asian Indians (Tables 1 and 2). In addition, Chinese men had the highest central-to-appendicular fat mass ratio among the five ethnic groups, and this ratio was significantly higher for Chinese women than European and Asian Indian women (Tables 1 and 2).

The relationships between %BF and the logarithm of BMI for each ethnic group were separately analysed by sex and are shown in Figures 1 and 2. For women, the slope of the regression of %BF on log\(_{10}\) (BMI) for European was steeper than for Pacific (P=0.037) while the slopes for European, Māori, Asian Indian and Chinese did not differ significantly. The regression equation for Pacific females alone was:

\[
%BF=67.60 \log_{10}(\text{BMI}) – 62.29
\]

(Standard error of estimate [SEE]=4.53%, R\(^2\)=0.65)

Covariance analysis showed that there was no significant difference between European and Māori in the elevations of the regression lines (P=0.79) and the common slope regression equation for European, Māori, Asian Indian and Chinese women was:

\[
%BF=86.00 \log_{10}(\text{BMI}) – 86.89 + 8.06 \text{ group1} + 2.84 \text{ group2}
\]

(SEE=4.25%, R\(^2\)=0.81)

…where group1 is coded as 1 for Asian Indian, 0 otherwise and group2 is coded as 1 for Chinese, 0 otherwise. Hence, at fixed BMI, %BF in Asian Indian is 8.1% higher and in Chinese, 2.8% higher, than in European and Māori.

At a BMI of 30 for European (and Māori) women the predicted %BF (40.1%) equates to a BMI of 33 for Pacific, 24 for Asian Indian, and 28 for Chinese women (Table 4).

For men, there were no significant differences in the slopes of the regression lines for the five ethnic groups. However, as for the women, covariance analysis showed that the elevations of the regression lines for Māori and European men did not differ significantly (P=0.54) and the common slope regression equation for all five ethnic groups was:

\[
%BF=84.97 \log_{10}(\text{BMI}) – 98.97 – 2.92 \text{ group1} + 11.70 \text{ group2} + 3.91 \text{ group3}
\]

(SEE=5.08%, R\(^2\)=0.63)

…where group1 is coded as 1 for Pacific, 0 otherwise, group2 is coded as 1 for Asian Indian, 0 otherwise, and group3 is coded as 1 for Chinese, 0 otherwise. Hence, at fixed BMI, %BF in Pacific is 2.9% lower, in Asian Indian is 11.7% higher and in Chinese, 3.9% higher, than in European and Māori.

At a BMI of 30 for European (and Māori) men the predicted %BF (26.5%) equates to a BMI of 32 for Pacific, 22 for Asian Indian, and 27 for Chinese women (Table 4).
Table 3. Body composition and limb length of women and men in five ethnic groups adjusted for weight and height within each sex

<table>
<thead>
<tr>
<th>Variable</th>
<th>European</th>
<th>Māori</th>
<th>Pacific</th>
<th>Asian Indian</th>
<th>Chinese</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FM (kg)</td>
<td>26.15 ± 0.54</td>
<td>28.39 ± 0.67</td>
<td>23.62 ± 0.74*</td>
<td>31.31 ± 0.64*</td>
<td>27.57 ± 0.72</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>44.52 ± 0.55*</td>
<td>44.63 ± 0.68*</td>
<td>47.17 ± 0.76*</td>
<td>39.22 ± 0.65*</td>
<td>42.43 ± 0.74</td>
</tr>
<tr>
<td>BMC (kg)</td>
<td>2.52 ± 0.04*</td>
<td>2.56 ± 0.05*</td>
<td>2.73 ± 0.06*</td>
<td>2.35 ± 0.05</td>
<td>2.38 ± 0.05</td>
</tr>
<tr>
<td>BMD (g.cm-2) †</td>
<td>1.18 ± 0.01</td>
<td>1.17 ± 0.01</td>
<td>1.23 ± 0.02</td>
<td>1.16 ± 0.01</td>
<td>1.18 ± 0.02</td>
</tr>
<tr>
<td>ASMM (kg)</td>
<td>17.37 ± 0.27*</td>
<td>17.41 ± 0.33*</td>
<td>18.45 ± 0.37*</td>
<td>15.51 ± 0.32</td>
<td>16.00 ± 0.36</td>
</tr>
<tr>
<td>AbFM (g)</td>
<td>2030 ± 67*</td>
<td>2189 ± 87*</td>
<td>1738 ± 92*</td>
<td>2623 ± 79</td>
<td>2475 ± 89</td>
</tr>
<tr>
<td>Thigh FM (g)</td>
<td>2717 ± 78</td>
<td>2695 ± 96</td>
<td>2317 ± 107</td>
<td>3334 ± 93*</td>
<td>2600 ± 104</td>
</tr>
<tr>
<td>Arm length (cm)‡</td>
<td>51.68 ± 0.36*</td>
<td>51.82 ± 0.44*</td>
<td>52.96 ± 0.44*</td>
<td>53.51 ± 0.43*</td>
<td>49.32 ± 0.44</td>
</tr>
<tr>
<td>Leg length (cm)‡</td>
<td>75.81 ± 0.39*</td>
<td>75.10 ± 0.48</td>
<td>76.87 ± 0.48*</td>
<td>77.23 ± 0.47*</td>
<td>74.29 ± 0.48</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FM (kg)</td>
<td>18.66 ± 0.75*</td>
<td>17.63 ± 0.85*</td>
<td>15.15 ± 1.10*</td>
<td>26.44 ± 0.77*</td>
<td>21.22 ± 0.92</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>61.48 ± 0.77*</td>
<td>62.59 ± 0.88*</td>
<td>64.81 ± 1.13*</td>
<td>53.46 ± 0.79*</td>
<td>58.32 ± 0.94</td>
</tr>
<tr>
<td>BMC (kg)</td>
<td>3.22 ± 0.06*</td>
<td>3.34 ± 0.06*</td>
<td>3.39 ± 0.08*</td>
<td>2.78 ± 0.06*</td>
<td>2.98 ± 0.07</td>
</tr>
<tr>
<td>BMD (g.cm-2) †</td>
<td>1.26 ± 0.01</td>
<td>1.28 ± 0.02</td>
<td>1.29 ± 0.02</td>
<td>1.18 ± 0.01*</td>
<td>1.25 ± 0.02</td>
</tr>
<tr>
<td>ASMM (kg)</td>
<td>25.94 ± 0.42*</td>
<td>26.69 ± 0.48*</td>
<td>28.02 ± 0.62*</td>
<td>23.20 ± 0.43</td>
<td>23.94 ± 0.51</td>
</tr>
<tr>
<td>AbFM (g)</td>
<td>1583 ± 87*</td>
<td>1566 ± 99*</td>
<td>1206 ± 127*</td>
<td>2547 ± 89*</td>
<td>2091 ± 106</td>
</tr>
<tr>
<td>Thigh FM (g)</td>
<td>1642 ± 74</td>
<td>1566 ± 84</td>
<td>1403 ± 109</td>
<td>2206 ± 76*</td>
<td>1689 ± 90</td>
</tr>
<tr>
<td>Arm length (cm)‡</td>
<td>55.66 ± 0.42*</td>
<td>57.62 ± 0.46*</td>
<td>57.80 ± 0.58*</td>
<td>57.50 ± 0.42*</td>
<td>53.35 ± 0.50</td>
</tr>
<tr>
<td>Leg length (cm)‡</td>
<td>81.50 ± 0.44*</td>
<td>81.41 ± 0.48*</td>
<td>82.71 ± 0.60*</td>
<td>82.57 ± 0.43*</td>
<td>79.35 ± 0.52</td>
</tr>
</tbody>
</table>

Note: Values are mean ± SEM. *Significantly different to Chinese (P<0.05). Abbreviations: AbFM, abdominal fat mass; ASMM, appendicular skeletal muscle mass; BMC, bone mineral content; BMD, bone mineral density; FM, fat mass. †Adjusted for weight only. ‡Adjusted for DXA height only.

Table 4. Comparison of European body mass index (BMI) and corresponding percent body fat with estimated BMI equivalents for Māori, Pacific, Asian Indian and Chinese derived from regression equations for predicting percent body fat from BMI

<table>
<thead>
<tr>
<th>European BMI (kg.m⁻2)</th>
<th>Body fat (%)</th>
<th>Māori</th>
<th>Pacific</th>
<th>Asian Indian</th>
<th>Chinese</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>25.0</td>
<td>20.0</td>
<td>19.6</td>
<td>16.1</td>
<td>18.5</td>
</tr>
<tr>
<td>25</td>
<td>33.3</td>
<td>25.0</td>
<td>26.0</td>
<td>20.1</td>
<td>23.2</td>
</tr>
<tr>
<td>30</td>
<td>40.1</td>
<td>30.0</td>
<td>32.8</td>
<td>24.2</td>
<td>27.8</td>
</tr>
<tr>
<td>35</td>
<td>45.9</td>
<td>35.0</td>
<td>39.9</td>
<td>28.2</td>
<td>32.4</td>
</tr>
<tr>
<td>40</td>
<td>50.9</td>
<td>40.0</td>
<td>47.2</td>
<td>32.2</td>
<td>37.1</td>
</tr>
<tr>
<td>45</td>
<td>55.3</td>
<td>45.0</td>
<td>54.9</td>
<td>36.3</td>
<td>41.7</td>
</tr>
<tr>
<td>50</td>
<td>59.2</td>
<td>50.0</td>
<td>62.7</td>
<td>40.3</td>
<td>46.3</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>11.6</td>
<td>20.0</td>
<td>21.6</td>
<td>14.6</td>
<td>18.0</td>
</tr>
<tr>
<td>25</td>
<td>19.8</td>
<td>25.0</td>
<td>27.1</td>
<td>18.2</td>
<td>22.5</td>
</tr>
<tr>
<td>30</td>
<td>26.5</td>
<td>30.0</td>
<td>32.4</td>
<td>21.8</td>
<td>27.0</td>
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<tr>
<td>35</td>
<td>32.2</td>
<td>35.0</td>
<td>37.9</td>
<td>25.5</td>
<td>31.5</td>
</tr>
<tr>
<td>40</td>
<td>37.2</td>
<td>40.0</td>
<td>43.3</td>
<td>29.1</td>
<td>36.0</td>
</tr>
<tr>
<td>45</td>
<td>41.5</td>
<td>45.0</td>
<td>48.7</td>
<td>32.8</td>
<td>40.5</td>
</tr>
<tr>
<td>50</td>
<td>45.4</td>
<td>50.0</td>
<td>54.1</td>
<td>36.4</td>
<td>45.0</td>
</tr>
</tbody>
</table>
Figure 1. Relation between percentage body fat (%BF) and BMI for 37 European (blue circles), 23 Māori (brown triangles), 23 Pacific (green triangles), 25 Asian Indian (squares) and 23 Chinese women (red circles)

Note: The linear regressions are: %BF=95.8log_{10}(BMI) – 100.5 (r^2=0.75) for European; %BF=74.0log_{10}(BMI) – 69.1 (r^2=0.76) for Māori; %BF=67.6log_{10}(BMI) – 62.3 (r^2=0.65) for Pacific; %BF=77.0log_{10}(BMI) – 66.0 (r^2=0.69) for Asian Indian; %BF=98.3log_{10}(BMI) – 100.4 (r^2=0.56) for Chinese.

Figure 2. Relation between percentage body fat (%BF) and BMI for 29 European (blue circles), 23 Māori (brown triangles), 15 Pacific (green triangles), 29 Asian Indian (squares) and 20 Chinese men (red circles)

Note: The linear regressions are: %BF=103.8log_{10}(BMI) – 125.0 (r^2=0.24) for European; %BF=86.6log_{10}(BMI) – 101.8 (r^2=0.70) for Māori; %BF=80.6log_{10}(BMI) – 95.3 (r^2=0.60) for Pacific; %BF=79.3log_{10}(BMI) – 79.4 (r^2=0.51) for Asian Indian; %BF=94.3log_{10}(BMI) – 107.9 (r^2=0.71) for Chinese.
Discussion

In this study we undertook for the first time a comparison of the body composition and %BF-BMI relationships between NZ European, Māori, Pacific, Asian Indian and Chinese ethnic groups. Our earlier analysis\(^6\) showed the wide disparity in the relationships between %BF and BMI between the first four of these ethnic groups with Pacific and Asian Indian occupying the two extremes.

The important finding of the present work was that Chinese differed from Asian Indian and the other ethnic groups in this relationship. At a fixed %BF equivalent to a BMI of 30 kg.m\(^{-2}\) in Europeans (40% for women, 27% for men), BMI values for Chinese were 2–3 units lower and for Asian Indians 6-8 units lower. For a BMI of 30 kg m\(^{-2}\), body fat in Chinese women was 43% and in Asian Indian women, 48%, while in men these fatness levels were 30% and 38%, respectively.

The higher %BF in Asian Indian compared to Chinese at a fixed BMI is in agreement with the findings of Deurenberg-Yap et al\(^4\) in Singaporean Indian and Chinese adults and Lear et al\(^5\) in Canadian South Asian and Chinese aged 30-65y. Our results also are consistent with a number of other studies that have compared European populations to Chinese.\(^9\)-\(^11\) For the same BMI, %BF was higher in Singaporean Chinese,\(^9\) Hong Kong Chinese\(^10\) and Taiwanese\(^11\) adults than in Europeans. BMI in Chinese equivalent to a BMI of 30 in Europeans was 25-27 kg.m\(^{-2}\), compared to 27-28 in the current work.

Differences in body build may explain, at least in part, the ethnic differences in the %BF-BMI relationships. In individuals with the same %BF, those with shorter legs relative to their height will have higher BMI. Larger frame (stockier) individuals will tend to have lower %BF than their smaller frame counterparts with the same BMI because of higher muscle and bone mass in the former.\(^12\)

Deurenberg et al\(^12\) explained differences between Singaporean Chinese and Indians on the basis of relative leg length and measures of frame size. In the present study, relative leg length was shorter in Chinese than the other ethnic groups. While we did not measure frame size, the DXA results provide measures of bone mass and limb muscle. Both BMC and ASMM were lower in Chinese and Asian Indian than the other ethnic groups.

There is increasing evidence that risk of obesity-related diseases is high at low BMI levels in Asian Indian and Chinese populations, justifying a lowering of the cut-off threshold for obesity for these ethnic groups. For example, Asian Indians with ‘normal’ BMI (<25 kg.m\(^{-2}\)) have high cardiovascular disease risk\(^13\) and Singaporean Indians and Chinese have significantly elevated cardiovascular disease risk factors at a BMI of 27 kg.m\(^{-2}\).\(^14\) In a representative sample of 2319 adult Singaporean Chinese only 3.8% of the females and 3% of the males had a BMI≥30 kg.m\(^{-2}\) yet mortality from CVD in Singapore is comparable to that found in Western countries.\(^15\)

Data reported by the NZ Ministry of Health\(^3\) showed that Asian Indians had significantly higher and Chinese significantly lower cardiovascular disease (CVD) hospitalisation and mortality rates than the total population. Based on data from the 2002/03 NZ Health Survey the prevalence of self-reported doctor-diagnosed diabetes
was more than three-fold higher for Asian Indians than the total population and, for Chinese, not significantly different from the total population.\(^3\)

These Health Survey data also showed that obesity prevalence, using the standard 30 kg.m\(^{-2}\) BMI threshold, was 7.1% and 14.8% for Asian Indian males and females, respectively, compared with 20.1% and 21.7% for males and females in the total population. For Chinese males, obesity prevalence was 4%, and for females the numbers were too small for a reliable prevalence figure. A clear discordance exists between CVD and diabetes rates and the obesity prevalence figures, particularly in Asian Indians but also in Chinese.

The higher %BF levels seen in Asian Indian and Chinese populations compared to European at the same BMI provide one possible reason for the elevated risk of obesity-related disease at the lower BMI levels. In addition, the propensity for abdominal adiposity found in Asian populations may be important. Central obesity is closely associated with risk for CVD and Type 2 diabetes in Asian Indians.\(^{16-18}\)

Notably, in the present study Chinese had significantly higher central to appendicular fat mass ratio than all other ethnic groups. The fact that both relative arm and leg lengths were shortest in Chinese may provide part of the explanation for this finding. Asian Indians however had the highest abdominal fat after controlling for weight and height.

The ratio of abdominal to thigh fat did not differ significantly between Chinese and Asian Indians, but was significantly higher than in Europeans. The results indicate the tendency for greater central fat accumulation in the Chinese and Asian Indian groups. Larger, longitudinal studies are required to explore the importance of ethnic differences in %BF, fat distribution, ASMM and relative leg length on disease risk.

This report has focused particularly on comparison of Chinese with the other ethnic groups, and, most importantly, the Asian Indian group since a detailed comparison of the four non-Chinese groups, over the adult age range, has appeared elsewhere.\(^6\)

Strengths of the present study include the application of a well-validated body composition methodology and the same machine for all measurements.

The primary aim of the present work was to examine the ethnicity-dependence of the relationships between body size and fatness for which it was desirable that wide ranges of BMI and %BF were achieved within each gender/ethnic subgroup rather than representativeness of the wider populations. A limitation was the restricted BMI range for the Chinese women which extended only to 27 kg.m\(^{-2}\).

This report provides the first evidence that in the NZ adult population there are marked disparities in the %BF-BMI relationships between the principal ethnic groups and, in particular, between Chinese and Asian Indian which together make up 74% of the Asian population in this country.\(^2\) While this evidence must be regarded as preliminary, pending confirmation in studies covering a wider age range in the Chinese group, it is, nevertheless, in general agreement with similar investigations of Chinese and Asian Indian adults carried out elsewhere.

Our results emphasise the limitations of universal BMI cut-off points for determination of percentage body fat and obesity when applied across ethnic groups. They point to the need for a reduction in the obesity thresholds for Asian populations in this country and for a distinction to be recognised between Asian Indian and
Chinese ethnic groups in terms of these thresholds. Such reductions in the BMI cut-offs for obesity clearly have major implications for health policy but may be essential if these groups are not to be disadvantaged under policies that target obesity and its treatment.

**Competing interests:** None.

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**References:**


Can imaging determine if a rotator cuff tear is traumatic?

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The purpose of this review paper is to clarify if imaging studies can determine whether a rotator cuff tear is traumatic or atraumatic in aetiology. The impetus for this review is the important issue of entitlement for Accident Compensation Corporation (ACC)-funded treatment in New Zealand.

In this paper we review specific information that can be reported on plain X-rays, ultrasound scans, CT and MRI scans. Literature searches have been undertaken to investigate radiological features that have been speculated to be associated with atraumatic rotator cuff disease. The historical and recent relevant research has been reviewed. Conclusions are based on literature evidence. The review includes some definitions and grading systems so the reader may better understand the topic.

Acromial morphology

Historical—In 1934 Codman, considered acromial morphology was a potential cause of rotator cuff tendon pathology. Codman’s conclusions were based on observation of cadaver shoulders.

In 1972 Neer published his results of anterior acromioplasty in 50 shoulders. He postulated that impingement on the tendinous portion of the rotator cuff by the coracoacromial ligament and anterior acromion was responsible for the syndrome of chronic impingement. It is important to note however that this study excluded acute traumatic tears, there was no radiographic assessment of the acromion and the study did not prove causation.

Bigliani and Morrison presented a cadaveric study on the morphology of the acromion in 1986. They examined 140 cadaveric shoulders with a mean age of 74 years. They identified three types of acromion with scapular lateral X-rays. 17.1% were type 1 (flat), 42.9% type 2 (curved) and 39.3% type 3 (hooked). 24% of the cadaver shoulders had a full thickness rotator cuff tear. 3% of the rotator cuff tears were in type 1 acromions, 24% in type 2 and 70% in type 3 respectively. The type 2 (curved) acromion was the most common. Only 8 of the 60 specimens with type 2 acromions had a full thickness rotator cuff tear (13%). There was no information regarding pre-morbid history of shoulder symptoms or trauma.

Another acromial morphological variation that has been considered to have an association with rotator cuff pathology is the os acromiale. This is an anatomical variation in which the ossification centres of the acromion do not fuse with bone. In 1984 Mudge et al published a review of 8 patients with rotator cuff tears who also had os acromiale.

Recent research—A prerequisite for a radiological classification to be useful is the classification being reliable and reproducible. Stehle et al’s cadaveric radiological study demonstrated significant variation in the appearance of the acromion with
projectional changes. Bright et al examined reliability of assessment of acromial morphology concluding that there was ‘unacceptable variability of interpretation and grading of radiographs’. Ogawa et al examined the relationship of subacromial spurs to rotator cuff tear and tear morphology in cadavers, patients with cuff tears and a control group. Subacromial spurs were classified as small (<5 mm), medium (5 mm–9 mm) and large (≥10 mm). A large amount of information is presented in the results and many aspects are relevant to this paper. Spur size increased with increasing age. The authors concluded ‘The presence of a small spur has no diagnostic value for rotator cuff tears. Spurs measuring 5 mm or more, however, are of diagnostic value because of the high rate of association with bursal-side tear, complete tears limited to the supraspinatus tendon, or massive tears.’

Pearsall et al, however, found no association between acromial morphology and rotator cuff tears. They concluded that acromial shape and AC joint changes are not predictive of a full thickness rotator cuff tear. Standard shoulder radiographs in these patients however, did demonstrate greater tuberosity sclerosis, osteophytes, subchondral cysts, and osteolysis that are not noted in patients without shoulder symptoms.

Gill et al identified a strong association between rotator cuff pathology and acromial morphology when no adjustments for patients age were made. However stratified univariate analysis did not demonstrate an independent association between full thickness rotator cuff tears and type 3 acromions in patients over 50 years of age. The authors concluded that ‘Previous reports linking full-thickness cuff tears to acromial morphology may simply have been describing the association of type III acromions and cuff tears with aging, rather than a true causal relationship.’

The association between os acromiale and cuff pathology has been re-examined by Ouellette et al in 2007; 42 patients with os acromiale who were compared with age and gender matched controls with MRI. They were unable to demonstrate an association between os acromiale and tears of the rotator cuff.

Conclusions—The historical concept that acromial morphology is a significant contributor is based upon clinical and cadaver observations, not studies designed to assess causation. More recent studies have demonstrated that acromial morphology is unreliably and inconsistently assessed by radiographs. There is an increasing incidence of subacromial spurs and rotator cuff pathology with normal aging. Although there are conflicting conclusions in different studies, some studies report that patients with a large subacromial spur are more likely to have rotator cuff pathology. Evidence of a causal relationship is lacking. An os acromiale cannot be considered an aetiological factor in rotator cuff tears.

Acromioclavicular joint arthrosis

Historical—Neer, in his landmark paper of 1972, mentioned that excrescences on the under surface of the AC joint may potentially impinge upon the cuff.

Recent research—Shubin Stein et al examined the MRI appearances of symptomatic and asymptomatic AC joints. Reactive bone oedema was a more reliable predictor of symptoms from the AC joint than was the severity of AC arthritis on MRI.
Relevant to our review is the extremely high prevalence of AC arthritis on imaging studies. The prevalence of AC joint arthritis using MRI was 82% in asymptomatic shoulders. In those less than 30 years, 68% had MRI features of arthritis, while the prevalence rose to 93% in those over 30 years. The authors concluded that radiological AC joint arthritis may not be clinically relevant.\textsuperscript{11}

Needell et al\textsuperscript{12} demonstrated AC joint arthritis correlated more closely with age than MRI evident tendon abnormalities. In their study, AC joint arthritis was seen in 89% of shoulders in those over 40 years and 76% of shoulders overall.\textsuperscript{12} Approximately two-thirds of those with normal tendons had AC arthritis.

**Conclusions**—Contemporary studies show that radiographic appearances of AC joint arthrosis are common and increase in prevalence with age. Imaging findings of AC arthrosis can be considered normal for age in patients over 40 years of age and may be found as an asymptomatic finding in younger age groups as well. There is no evidence that AC arthritis causes rotator cuff tears or indicates the presence of a rotator cuff tear.

**Greater tuberosity changes**

**Historical**—In 1934 Codman, in a cadaver study, described an association between partial thickness rotator cuff tears and eburnation at the sulcus, or interval between the articular cartilage and greater tuberosity.\textsuperscript{1,13} No radiographic correlation was made. In 1964 Cotton et al performed a cadaver study with radiology and dissection.\textsuperscript{14} The authors concluded that cysts were indicative of tears. Sclerosis and cortical thickening in the absence of other changes were not found to be a significant predictor of cuff tearing. Unfortunately only 6/136 of the radio graphically normal shoulders were dissected for comparison. Other groups have reported similar findings associating tuberosity sclerosis, cortical thickening and cyst change with rotator cuff tears. Unfortunately, the majority of these are either anecdotal or lack controls.\textsuperscript{15}

**Recent research**—In 1999, Huang et al published an MRI and radiological study titled ‘Greater tuberosity changes as revealed by radiography: Lack of clinical usefulness in patients with rotator cuff disease.’\textsuperscript{15} The authors concluded ‘Cortical thickening of the greater tuberosity and subcortical sclerosis are not associated with rotator cuff disease. For some observers, identifying cyst-like lesions is associated with rotator cuff disease, but the clinical usefulness of the observation is limited by high interobserver variability and poor positive predictive value.’\textsuperscript{15}

McCuailey et al examined bone marrow oedema in the greater tuberosity on MRI scans. Bone marrow oedema was a rare finding. The authors concluded that traumatic rotator cuff tear with oedema may result from avulsion forces on the supraspinatus insertion.\textsuperscript{16}

Williams et al examined the relationship greater tuberosity cysts and both age and rotator cuff tears. Cysts were present in 70% of patients and were 7 times more frequent in the posterior aspect of the tuberosity than the anterior aspect of the tuberosity. Tuberosity cysts and rotator cuff tears did not appear to be related.\textsuperscript{17}

In 2007, Fritz et al published on the relationship of greater tuberosity cysts and rotator cuff disorders. MRI better demonstrates the position of the cysts than plain X-rays.
Posterior cysts were more common, occurring in 56.7% of shoulders and showed no correlation with age or rotator cuff disorders.

Anterior cysts occurred in 22.7% of shoulders and were strongly associated with rotator cuff disorders with 94% of patients with anterior cysts having a rotator cuff disorder (p<0.001). They defined rotator cuff disorders as including tendinopathy (a surgical diagnosis of the appearance of the tendon), partial thickness tears and full thickness tears. With regard to full thickness tears of the rotator cuff, 48% of those with anterior cysts had a rotator cuff tear.13

Conclusions—Greater tuberosity cysts are common. The location of cysts is more accurately determined on MRI or CT scans than plain X-rays. Posterior cysts are more common and show no correlation with rotator cuff disorders. Anterior cysts are less common and are probably associated with the presence of rotator cuff disorders. Full thickness rotator cuff tears were found in 48% of patients with anterior greater tuberosity cysts in one study of symptomatic shoulders evaluated by MRI and surgery. Cortical thickening and subcortical sclerosis are not seen more frequently in shoulders with rotator cuff disease than those without rotator cuff disease.

Reduced acromiohumeral interval

Historical—The acromiohumeral interval (AHI) has been used in the assessment of potential rotator cuff disease since Golding recommended its routine measurement on AP shoulder radiographs in 1962.18 Cotton used it as one of his radiological diagnostic criteria in his cadaver study.14

Recent research—Gruber et al published level 1 evidence in 2009, demonstrating that the assessment of AHI on standardized anteroposterior X-rays is reliable and reproducible.18 This study did not assess the effect of radiographic projectional errors. The patient was standing with their arm beside them in external rotation and the palm of the hand facing forwards.

Recently Saupe et al examined the relationship between the AHI and abnormalities of the rotator cuff tendons as assessed by MR arthrography.19 Of those with an AHI less than or equal to 7 mm, 90% had a full thickness supraspinatus tear, 67% a full thickness infraspinatus tear and 43% a full thickness subscapularis tear. Additionally 71% showed MR evidence of fatty atrophy.19

Nove-Josserand et al examined the factors affecting the AHI and coracohumeral interval (CHI) in patients with rotator cuff tears having surgery. The AHI was assessed on 20° caudal tilt AP X-rays, with the arm in neutral rotation with the patient relaxed. A distance of less than 7 mm was considered abnormal. The CHI was assessed on preoperative CT scans, measuring the narrowest distance between the tip of the coracoid process and the humeral head. The position of the arm was not found to significantly alter this distance. A distance of less than 6 mm was considered abnormal.20

Multiple tendon tears were more likely to have a reduced AHI with 45% of combined supraspinatus, infraspinatus and subscapularis tears having a reduced AHI. No shoulder with an isolated rupture of supraspinatus or subscapularis had a reduced AHI. A reduced CHI was present in 24% of patients with a combined tear of supraspinatus and subscapularis, but was only present in 5% of shoulders with no
subscapularis tears and 7% of patients with an isolated subscapularis tear. Fatty
degeneration Grade 3 or worse was associated with reduced AHI and CHI. Shoulders
with symptoms for more than 5 years were more likely to have a reduced AHI
(p=0.002). More than one-third of the patients with more than 5 years symptoms had
a reduced AHI. 20

Conclusions—Recent studies support the long held assumption that reduced AHI is
correlated with rotator cuff tears. Measurement is reliably reproducible on
standardized X-rays. Reduced AHI is associated with both fatty atrophy and
symptoms that may be of more than 5 years duration.

Cuff tear arthropathy

Historical—The first documented description of the typical changes of the entity now
known as rotator cuff arthropathy was by Adams and Smith in the 1850s.21, 22

In 1983 Neer et al introduced and defined the term ‘cuff tear arthropathy’, the
essential features being the presence of a rotator cuff tear and glenohumeral
osteoarthritis, characterized by the presence of a massive rotator cuff tear, superior
humeral head migration, acetabulisation of the acromion, collapse of the humeral
head articular surface and erosion of the superior glenoid.21, 23

Neer hypothesised that both mechanical and nutritional factors were responsible for
the development of cuff tear arthropathy and estimated the prevalence of cuff tear
arthropathy to be approximately 4% of patients with rotator cuff tendon tears. The
duration of symptoms in his series prior to ranged between 2 and 20 years, with a
mean of 9.8 years.

In 1981 an association between a cuff tear arthropathy-like condition and microscopic
basic calcium phosphate crystals deposited in the synovium and adjacent structures
was described and is known as Milwaukee shoulder.21, 22

Hamada et al, in 199024, proposed a grading system for RCA based primarily on
reduced acromiohumeral interval (AHI) as follows:

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

Recent research—Rockwood et al followed shoulders with massive irreparable tears
of supraspinatus and infraspinatus post surgery for an average of 6.5 years.25 None
progressed to cuff tear arthropathy. Apoil and Augereau, however, reported that more
than 25% of 56 shoulders that had undergone debridement of a degenerative lesion of
the rotator cuff had developed cuff tear arthropathy 10 years after surgery.21

In 2007 Zingg et al reported the outcomes of patients with massive rotator cuff tears
treated non operatively, followed for a mean of 4 years. Tears were classified as
reparable if the fatty infiltration was stage 2 or lower and the AHI was 7 mm or more
and irreparable if the fatty infiltration was stage 3 or greater and the AHI was less
than 7 mm. As in other publications the classification of a traumatic tear was based on the patient history of a traumatic event. 84% reported a traumatic event.

The time between acute injuries and diagnosis averaged 23 months. Although most patients in this group maintained satisfactory shoulder function, the radiological appearances deteriorated significantly. Over the follow-up period (mean 4 years), glenohumeral osteoarthritis progressed (p=0.014), the AHI decreased by 2.6 mm (p=0.01), fatty infiltration increased by one grade and the size of the tear increased (p=0.003). Half of the rotator cuff tears that were graded as reparable initially were graded irreparable at final follow up.26

Rotator cuff tears in primary osteoarthritis are uncommon. Edwards et al reported an incidence of 7% partial thickness tears and 7% full thickness tears in patients having shoulder replacements for primary osteoarthritis.27

Conclusions—Cuff tear arthropathy appears to be an uncommon sequelae of massive rotator cuff tears that develops over many years. The exact incidence of cuff tear arthropathy following massive rotator cuff tears and the duration from tear to onset of significant arthropathy is long but unknown.

Fatty muscle degeneration

Early research—CT and MRI were initially used to assess muscles in neuromuscular disease and spinal disorders. In 1989, Goutallier et al presented on fatty muscle degeneration.28 The authors proposed the following 5 grade staging system, using CT scans to assess the rotator cuff muscles

Stage 0—Normal muscle, no fatty streak
Stage 1—The muscle contains some fatty streaks
Stage 2—The fatty infiltration is important, but there is muscle than fat
Stage 3—There is as much fat as muscle
Stage 4—More fat than muscle is present

In 1994, Goutallier et al29 reported that significant fatty degeneration of infraspinatus was associated with severe functional impairment and that fatty degeneration of infraspinatus did not improve after repair. The authors concluded that ‘it is probably better to operate on wide tears before irreversible muscle damage’. Their data suggests that significant (worse than stage 2) degeneration of infraspinatus was uncommon in tears that has been symptomatic for less than 6 months although they commented ‘successive preoperative CT scans performed in more recent patients have shown that the infraspinatus can degenerate in several months or even weeks.’29

Nakagaki et al published a histological cadaveric study in 199630 concluding that ‘the fatty degeneration in the supraspinatus muscle after cuff tear was found to have a strong association with the degree of retraction of the tendon fibres’.

Recent research—Oh et al evaluated the reliability of MRI arthrography and CT arthrography. MRI arthrography had more interobserver reliability than CT arthrography. Interobserver and intraobserver reliability was poor.31 The investigators chose to assess fatty degeneration on oblique sagittal CT and T1 weighted MRI images where the scapular body, spine and base of the coracoid process form a ‘Y’.
Williams et al investigated the most reliable plane of imaging to identify fatty infiltration of supraspinatus. They recommended the axial plane, which had good intraobserver agreement and moderate interobserver agreement. The authors also evaluated the ‘tangent sign’ described by Zanetti et al. The tangent sign is evaluated on the sagittal plane at the most lateral image where the scapular spine is in contact with the scapular body. A line is drawn tangential to the scapular spine and the coracoid. The tangent sign is positive if the supraspinatus does not reach above this line.

Williams et al found a positive tangent sign is an indicator of muscle atrophy and advanced fatty infiltration. The authors described a new sign called the ‘fish backbone sign’. In this sign the supraspinatus looks like a fish backbone in the axial view and this indicates Goutallier grade 3 fatty infiltration.

Berthouet et al reviewed massive rotator cuff tears in patients younger than 65 years. They did not find a correlation between AHI and duration of symptoms. There was a significantly higher rate of fatty infiltration of the infraspinatus muscle (> stage 2) in patients with a long duration of symptoms (p<0.05). There was a significantly higher rate of infraspinatus fatty degeneration in patients with no history of trauma (p<0.05). However, 25% of patients with short duration symptoms and a history of trauma had greater than Goutallier stage 2 fatty infiltration in infraspinatus. The authors placed importance on the patient’s history of trauma and duration of symptoms to define the study groups.

Conclusions—The Goutallier classification system is used to stage fatty degeneration of the rotator cuff muscles. CT and MRI can be used to assess fatty degeneration. The axial image is best for evaluating supraspinatus. However, it should be noted that the potential for interobserver and intraobserver variation is high, especially with a variety of imaging sequences and observer experience. Significant infraspinatus fatty degeneration, greater than Goutallier stage 2, is more common if symptoms have been present for more than 6 months, although it may occur with a duration of symptoms of less than 6 months.

Bursal changes

Historical—We were unable to find literature indicating that bursal thickening was a sign of non traumatic rotator cuff disease.

Recent research—Teeffey et al investigated the sonographic differences in acute and chronic full thickness rotator cuff tears. Once again the diagnosis of an acute rotator cuff tear (RCT) was based on the patient’s history.

‘An acute RCT was considered to be present when (1) the clinical history revealed a distinct injury within 6 months from the time of operation in a previously asymptomatic shoulder and (2) the operative findings showed blunt, frayed cuff edges, tendon quality and thickness comparable to those of an intact cuff, and a freely mobile cuff.’

Seventy-five percent of patients with a midsubstance tear had an acute tear. Sixty-four percent of patients with joint or bursal fluid had an acute tear; 80% of patients with a non visualised rotator cuff due to a massive tear had a chronic tear; and 73% of patients with no sonographic evidence of bursal or joint fluid had a chronic tear.
Li et al studied ultrasound appearances of bursal thickening in Stage I and Stage II subacromial impingement. They defined Neer’s classification of impingement as follows:

Stage I—Reversible oedema and haemorrhage in the bursa

Stage II—Fibrosis and thickening of subacromial soft tissue and sometimes a partial rupture of the rotator cuff

Stage III—Complete rupture of the rotator cuff

The Neer classification of impingement classifies the pathology causing symptoms defined as impingement symptoms and not the aetiology (traumatic v non traumatic). Li’s et al reported that the normal subacromial bursal thickness is considered to be less than 2 mm.

Conclusions—A mid substance rotator cuff tear, or the presence of bursal fluid in a rotator cuff tear are more commonly present in an acute tear. Absence of bursal fluid and visible cuff tissue are more likely signs of a chronic tear. There is no information regarding bursal thickening in patients with symptoms of Stage I or Stage II impingement that indicates whether the impingement is traumatic or non traumatic in aetiology.

Tendon retraction

Gerber et al examined the effect of tendon release and delayed repair on the structure of the rotator cuff muscle in sheep. The authors concluded

“Rotator cuff tendon tears lead to substantial and progressive muscular changes with a severity that is proportional to the amount of musculotendinous retraction. If muscular function is to be preserved, a repair may need to be performed before marked retraction has occurred or new or different techniques for repair need to be developed.”

Gerber’s group later used the same model to examine how some of the retraction occurs. They found the tendon end retracts into the muscle the musculotendinous junction shifting more distal relative to the tendon.

Braune reported the intraoperative appearances of acute traumatic, chronic traumatic and atraumatic rotator cuff tears in the German literature. One of the factors he examined was tendon retraction. Once again the criteria used to define a tear as traumatic was an acute traumatic incident, pain free and healthy shoulder before accident, spontaneous constantly painful shoulder after accident. An acute traumatic tear was defined as less than 12 weeks from trauma and a chronic traumatic tear more than 12 weeks.

Patients with no known trauma were considered to have degenerative tears. They noted the location of the tear, tear size and retraction. The quality of the tendon end and changes in the long head of biceps, were also noted. Retraction was graded on as follows:

Grade I—Retraction to half the distance between the greater tuberosity and the level of the neck of the humerus

Grade II—Retraction to the centre of the neck of humerus
Grade III—Retraction to between the centre of neck of humerus and the glenoid
Grade IV—retraction beyond the glenoid

Although numbers in each study group were small, Braune did not find retraction beyond the glenoid in any patients in the acute traumatic group. Isolated subscapularis tears were only seen in the traumatic groups. Haematoma may be seen in the acute traumatic group. Subluxation of the long head of biceps was more common in the traumatic group \( (p=0.007) \). Partial and complete tears of the biceps were more common in the degenerative group. Synovitis of the biceps was seen in all 3 groups.\(^{39}\)

**Conclusions**—Little has been written about tendon retraction. Animal studies demonstrate significant tendon retraction occurring within one hour of rotator cuff tendon release, whereas a human study reports that retraction beyond the glenoid rim was not seen within 12 weeks of injury. Anatomical differences in the animal models may contribute to these different observations.

**Partial thickness tears**

**Historical**—Codman described an articular surface partial thickness tear, which he labelled a ‘rim rent’ tear. His hypothesis was that intrinsic tendon degeneration was the primary lesion.\(^{1,40,41}\) Neer instead favoured a process extrinsic to the tendon itself as primarily responsible for tears and proposed that subacromial impingement was the primary cause of tears.\(^2\)

Rothman and Parke in 1965 described a ‘critical zone’ of hypovascularity of the supraspinatus tendon near its humeral attachment.\(^{40}\) They confirmed a relatively avascular zone distally and found that the earliest changes of ‘degeneration’ occurred in this zone. They emphasised that their study did not produce evidence of causation of degeneration.\(^{42}\)

In 1990 Lohr and Uhtoff reported zone of relative hypovascularity to be more pronounced on the articular surface of the tendon, extending from the myotendinous junction to within a few millimetres of the humerus. Their cadaveric study published in 1990, demonstrated a hypovascular zone extending from the myotendinous junction to within 5mm of the humeral head insertion on the articular surface of the tendon. The bursal surface in comparison has a rich vascular supply.\(^{43}\)

**Current research**—The relative incidence of partial thickness tears in clinical studies however differs, with numerous reports of articular surface tears being 2 to 3 times more common than bursal surface tears.\(^{40,41}\)

Nakajima et al in 1994 compared the histological and *ex vivo* biomechanical properties of the articular and bursal surface fibres of supraspinatus, demonstrating more organised tendon bundles on the bursal surface.\(^{44}\) Biomechanical analysis showed that the bursal surface fibres had greater deformity and tensile strength. This may explain the increased incidence of partial thickness articular surface tears following a traumatic event.\(^{40}\)

Ko et al,\(^{45}\) in a prospective histological study, concluded that articular surface tears are likely related to intrinsic factors while bursal surface tears are associated with subacromial impingement.
Payne et al performed a retrospective analysis of athletes under the age of 40 who had undergone arthroscopic treatment for partial thickness cuff tears. Two main groups were identified; Group A patients had a history of an acute traumatic event and Group B consisted of overhead throwing athletes with an insidious onset of non traumatic shoulder pain. Seventy-nine percent of the partial tears in Group A were of the articular surface and 97% of the tears in Group B were of the articular surface.

A recent meta-analysis by de Jesus et al compared MR arthrography (MRA), conventional MR (MRI) and ultrasound (US) for diagnosis of full and partial thickness cuff tears. MRA performed significantly best and MRI and US performed similarly. The sensitivity for full thickness and partial thickness tears respectively were 95% and 86% for MRA, 92% and 64% for MRI and 92% and 66% for US. The specificity for full and partial tears was 99% and 95% for MRA, 93% and 92% for MRI and 94% and 86% for US. The authors stress that the analysis encompasses studies performed over a wide range of time and that this may influence findings as technical improvements and expertise improve in each modality.

The accuracy of US in particular is highly operator dependent, especially for small partial tears.

Conclusions—The aetiology of partial thickness rotator cuff tears is complex, not fully understood and almost certainly multifactorial. It appears likely that intrinsic tendon factors including differences in tendon layer mechanical characteristics and alterations in tendon structure with age, external impingement and trauma all have a part to play in the aetiology of partial thickness tears. It is clear that trauma can cause both articular surface and bursal surface tears and this has been reported in normal tendons of young athletes. It is also probable that intrinsic tendon changes and extrinsic impingement have a part to play in articular surface and bursal surface tears respectively.

MR Arthrography remains the gold standard for the assessment of rotator cuff tears, but standard MRI and US in appropriate hands have good accuracy.

Calcific tendinitis

Historical—Calcific tendinitis of the rotator cuff refers to a syndrome of shoulder pain associated with calcific deposits within the rotator cuff tendons. Codman proposed tissue hypoxia as the primary event leading to the development of tendon calcification in 1934. This remains a popular theory. The reported rates of coexistence of cuff tears and calcification in the literature have varied greatly, with some authors reporting that the two entities are virtually mutually exclusive while others report an association.

In 1993, Jim et al in 1993 examined the incidence of cuff tears in 81 symptomatic patients with calcification and reported that a small (not defined) rather than a large amount of calcium was more likely to be associated with a cuff tear. A deficiency of these and other studies is that they fail to distinguish between calcific tendinitis and degenerative calcification.

Recent research—Hamada et al demonstrated that crystals were composed of carbonate apatite. Recent extensive reviews on the subject by Uhthoff and Hurt emphasise that the actual aetiology of calcific tendinitis is unknown. Uhthoff and Loehr in 1976 proposed a three stage process. The initial precalcific stage, the site of
future calcification is proposed to undergo cartilaginous metaplasia. The second calcific stage is proposed to consist of three distinct phases itself. During the formative phase, calcium crystals are deposited. During the resting phase, no new calcium deposits are laid down. Finally during the resorptive phase calcium deposits are reabsorbed. This is accompanied by an inflammatory infiltrate. During the post calcific stage, the tendon is repaired. The calcific and post calcific stages are often associated with pain, which is most severe during the resorptive phase. Most authors have found that rotator cuff tears and calcification rarely coexist. Loew et al studied 75 patients with calcific tendonitis with MRI scans and found only 1 patient had a partial thickness cuff tear. Rotator cuff tears in calcific tendinosis are sufficiently rare that Gotoh et al published a case report in Skeletal Radiology in 2003 of a patient with calcific tendinosis that progressed to rotator cuff tear.

Dystrophic calcification in comparison consists of linear calcific deposits at the insertions of tendons. These are thought to be secondary to degenerative changes in tendons. Conclusions—Calcific tendinosis of the rotator cuff is a condition of unknown aetiology. Tissue hypoxia near the rotator cuff insertion appears to be involved in the pathogenesis. Most studies report that cuff tears are uncommon in association with calcific tendinosis. Studies frequently fail to distinguish between different types of cuff calcification. This likely contributes to the large reported differences.

Tendinopathy

Tendinopathy is a complex subject beyond the scope of this imaging review. Lewis, in a review of rotator cuff tendinopathy in the British Journal of Sports Medicine 2009 noted that ‘tendinopathy is a generic term without aetiological, biochemical or histological implications and is used to describe pathology in, and pain arising from, a tendon.’ Lewis’s extensive literature review concludes that the pathogenesis of rotator cuff tendinopathy is multifactorial and results form a combination of intrinsic, extrinsic and environmental factors. Intrinsic degenerative changes in a tendon may occur due to overuse or overload of a tendon. Extrinsic compression from subacromial impingement may sometimes occur as a secondary phenomenon when there is tendon dysfunction.

Although the aetiology of tendinosis is likely multifactorial, age does appear to have a role in some cases. The peak age group for rotator cuff tendinopathy is the 5th to 7th decades.

Is there an accepted definition of tendinopathy on imaging studies? Is there interobserver and intraobserver reliability in reporting tendinopathy on imaging? Does tendinopathy on imaging correlate to tendinopathy histologically or biochemically? Can some tendinopathy changes be considered normal for age in older subjects? Does the presence of tendinopathy mean that a subsequent rotator cuff tear is wholly or substantially due to degeneration? We have not been able to satisfactorily answer these questions from our literature review.

Conclusions—Studies reporting imaging findings in traumatic rotator cuff tears use a history of a traumatic event with onset of symptoms as the criteria for diagnosing a traumatic rotator cuff tear. The clinical history is important.
Age related changes occur in the shoulder and rotator cuff. Some changes can be considered normal for age. These include:

- Subacromial spur formation\(^7,9\)
- AC joint arthrosis\(^11,12\)
- Possibly tendinopathy but definitions and data lacking.\(^55\)

There are some signs on imaging that indicate chronicity. The definition of chronicity varies from months to years in different conditions and studies. When these are present, in a patient with a recent history of a traumatic event, the symptoms may be substantially due to a pre-existing condition. This may be more likely if the traumatic event is a low energy event, more in keeping with an activity of daily living. It may also be more relevant if the onset of symptoms was a significant time after the perceived traumatic event. Signs that commonly, but not always, indicate chronicity include:

- Cuff tear arthropathy\(^21,22,24,26\)
- Decreased Acromiohumeral interval of 7 mm or less\(^19,20\)
- Fatty muscle degeneration\(^28,29,34\)
- Tendon retraction beyond glenoid rim\(^39\) Animal studies report immediate significant retraction however\(^37\)
- Anterior greater tuberosity cysts\(^13\)

There are some imaging features that cannot be considered significantly associated with rotator cuff tears or are unreliable in their appearance or reporting. These include:

- Acromial morphology\(^5,6,8,10\)
- Os Acromiale\(^10\)
- AC joint changes\(^11\)
- Greater tuberosity sclerosis\(^15\)
- Greater tuberosity posterior cysts\(^1,3,15,17\)
- Calcific tendinosis\(^52,53\)

There are some features that, when present, do indicate the tear is likely to be traumatic. These include:

- Bone oedema in the greater tuberosity with supraspinatus tear\(^16\)
- Midsubstance tear\(^35\)
- Bursal fluid, haematoma or debris present\(^35,39\)
- Isolated subscapularis tears\(^39\)

None of the listed features are ‘absolute’. Imaging features should be interpreted in the context of the patient’s age, history of injury and history of symptoms when considering the aetiology of a rotator cuff tear.
Competing interests: None.

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References:


New Zealand Injury Prevention Strategy: significant shortcomings after 5 years

John Langley

Injury is a serious problem in New Zealand. It results in substantial mortality and morbidity and has significant social and economic costs.\(^1\) Historically, injury prevention efforts have been fragmented with various government agencies and non-government organisations addressing a wide range of issues, often without reference to one another. The introduction of New Zealand Prevention Strategy (NZIPS) in 2003 was an attempt to address this fragmentation. The NZIPS vision is “a safe New Zealand, becoming injury free”.\(^2\)

Several government agencies are involved in leading implementation of the Strategy and its six priority areas. The strategy is collectively owned by members of the Chief Executives Forum and supported by the NZIPS Secretariat. Individual government agencies have responsibility for specific priority areas and each priority area has its own strategy. The priority areas, the lead agency for each area, and the social and economic costs for each area are presented in Table 1.

This paper examines the performance of the lead agencies to date, and more generally, the future of injury prevention in New Zealand in terms of reducing important injuries namely those that result in death, represent a high threat to life, result in significant disability, or are a high cost to society.

The 5-year evaluation report for NZIPS was released in July 2010.\(^3\) The evaluation examined progress in terms of injury outcomes by comparing 2003 with 2006 data for fatal injuries. For serious non-fatal injury, namely injuries that have a 6% chance or more of resulting in death, data for 2003 were compared with data for 2009.

The evaluation report concludes that the gains that have been made in terms of deaths, in particular in areas such as road crashes and workplace, have been due to sustained activity and investment in injury prevention over time. For serious non-fatal injury outcomes, however, the indicators for “all injury” and for all priority areas show increases, most notably assault at 50% and self-harm at 45%.

The review suggests that this contrasting situation could be due to successfully reducing the severity of injury from fatal to serious and improvements in care that have resulted increased survivability of injuries. While these explanations may be valid they fall far short of fully explaining the difference since the number of fatal injuries is small relative to serious non-fatal injuries. For example, for every fatal road traffic crash there are four to five times as many serious non-fatal injuries.
Table 1. Summary of injury costs by cost capital and priority area. Base-case estimate using official transport sector VPF; 3% discount rate; NZ $M; June 2008 prices

<table>
<thead>
<tr>
<th>Priority Area</th>
<th>Lead Government Agency</th>
<th>Treatment and Rehabilitation (1)</th>
<th>Lost Economic Contribution (2)</th>
<th>Human Costs (2)</th>
<th>Total Social and Economic Cost</th>
<th>% of Total Social and Economic Costs - All Injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Weekly Compensation</td>
<td>Lost Income to Premature Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assault</td>
<td>Ministry of Justice &amp; Social Development</td>
<td>$2.5</td>
<td>$2.2</td>
<td>$47.3</td>
<td>$227.5</td>
<td>$239.6</td>
</tr>
<tr>
<td>Falls</td>
<td>Accident Compensation Corporation</td>
<td>$53.7</td>
<td>$24.1</td>
<td>$29.2</td>
<td>$928.7</td>
<td>$1,735.2</td>
</tr>
<tr>
<td>Drowning</td>
<td>Accident Compensation Corporation</td>
<td>$0.8</td>
<td>$0.3</td>
<td>$47.9</td>
<td>$246.4</td>
<td>$295.5</td>
</tr>
<tr>
<td>Motor Vehicle</td>
<td>Ministry of Transport</td>
<td>$256.5</td>
<td>$206.4</td>
<td>$256.1</td>
<td>$1,477.0</td>
<td>$2,195.0</td>
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<tr>
<td>Suicide/Self Harm</td>
<td>Ministry of Health</td>
<td>$1.6</td>
<td>$0.5</td>
<td>$37.6</td>
<td>$1,787.4</td>
<td>$2,159.1</td>
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<tr>
<td>Workplace</td>
<td>Department of Labour</td>
<td>$349.5</td>
<td>$570.9</td>
<td>$593</td>
<td>$357.8</td>
<td>$1,347.5</td>
</tr>
<tr>
<td>Subtotal - Six priority areas</td>
<td></td>
<td>$1,143.6</td>
<td>$1,021.9</td>
<td>$831.4</td>
<td>$5,124.8</td>
<td>$8,121.8</td>
</tr>
<tr>
<td>Estimated &quot;Non- priority&quot; areas</td>
<td></td>
<td>$251.7</td>
<td>$139.2</td>
<td>$77.4</td>
<td>$1,087.1</td>
<td>$1,555.4</td>
</tr>
<tr>
<td>All injuries</td>
<td></td>
<td>$1,395.2</td>
<td>$1,161.1</td>
<td>$908.8</td>
<td>$6,211.9</td>
<td>$9,677.2</td>
</tr>
</tbody>
</table>

1) Treatment and rehabilitation costs of $1.4 billion, excluding GST, represents the economic costs for the purchase of ACC services.
2) Based on DALYs incurred from premature mortality and disability associated with injury.

There are many potential explanations for the observed trends, in part or in whole, and it is highly likely that their significance will vary by priority area. In the commentary for each of the priority areas, the evaluation report identifies some specific factors that might account for the trends.

Significant among them is that a number of lead agencies have only had implementation policies in place for a few years. Behavioural and environmental changes take time and, typically, the gains that might be expected for any specific strategy in an area may be modest. Consequently, in most cases it is too early to judge progress in terms of an indicator that seeks to capture the cumulative effect of a range of strategies in a priority area.

Irrespective of the date of implementation it is nevertheless important to determine the relevance of the policy to the outcomes being measured. It could be that strategies adopted by some of the lead agencies are not focused, or not sufficiently focused, on important injuries, or on the groups experiencing these injuries. Such an assessment was not undertaken by the evaluation.

The need for such an assessment is well illustrated by reference to the assault priority area in the context of serious non-fatal injury. Before doing this it is important to point out that the serious non-fatal injury outcome indicators for assault are labelled as provisional. This label is intended to reflect the concern that observed increases
might reflect higher levels of reporting as public awareness improves around the need to report violent acts, rather than any real increase in incidence.

Recent research suggests that there is no evidence to support this explanation and it has been recommended that the provisional status of the indicator be removed. In other words, the observed trends are considered highly likely to reflect the true trends in the incidence of serious non-fatal assault in New Zealand.

For the period 2000–2006 inclusive there were 5080 serious non-fatal assaults, 79% of which were to males. As noted in Table 1, the NZIPS lead agencies for assault are the Ministry of Justice (MoJ) and Ministry of Social Development (MSD). The specific strategies the MSD identifies for the priority area of assault is Te Rito: New Zealand Family Violence Prevention Strategy and Taskforce for Action on Violence within Families. The former was published in February 2002 and the latter has been in place since 2005.

The MoJ strategy is the Taskforce for Action on Sexual Assault, which has been in operation since 2007. The 5-year evaluation report notes that it is unclear how much co-ordination or overlap there is across these strategies.

It is difficult to see how collectively these strategies are going to make a significant impact on homicide and assault among males, where the major burden lies, given that most of this violence is not perpetrated in a family context or by a family member (the focus of the MSD strategies) and sexual assault is rarely involved (the focus of the MoJ strategy). Neither strategy makes specific reference to NZIPS.

In contrast, the Department of Labour’s 2005 Workplace Health and Safety Strategy (WHSS) specifically states that it contributes to two of the Government’s wider goals, one being implementation of NZIPS. The WHSS was reviewed in 2009. While the review report makes frequent reference to how the strategy fits under NZIPS, a supporting document presents an outcome monitoring framework that is silent on NZIPS injury outcome indicators.

Moreover, while the suggested fatality indicator aligns with that used for monitoring injury outcomes under NZIPS, the indicator suggested for the non-fatal injury, rate of work-related injury resulting in hospitalization per 100,000 workers, has been demonstrated as having major threats to validity. It was for this very reason that high threat to life indicators where chosen for the NZIPS chartbooks.

The Department of Labour is not alone in using an indicator that has a high threat to validity. In its ‘Safer Journey’s: New Zealand’s Road Safety Strategy 2010–2020, the Ministry of Transport, for one of its progress measures, uses serious injuries, defined as those hospitalised for one day or more. No reference is made to the NZIPS serious non-fatal outcome indicator. Length of stay is not a suitable proxy for severity of injury in terms of threat to life.

The failure of these recent policy-related documents to pay due regard to the NZIPS injury outcome indicators is surprising given that the NZIPS 2008–2011 Implementation Plan specifically states that “Initiatives focused on and delivered by the Chief Executives’ Injury Prevention Forum will address the issue of serious injuries…” (p12).
There can be no confusion as to what serious means in this context given that official indicators for monitoring progress in reducing serious non-fatal injuries are those that represent a high threat to life. These examples of poor alignment of policy with NZIPS raise questions about the commitment of lead agencies to NZIPS. This concern is reinforced by the evaluation report that notes that limited priority is given to injury prevention by several agencies, as it is not considered core business. This is exemplified by poor attendance at the Chief Executives Forum and the Ministerial Committee that is responsible for overseeing Government’s progress on the NZIPS strategy, with chief executives delegating representation to lower level officials who have no decision-making authority.

This situation contrasts with New Zealand’s Accident Compensation Corporation (ACC) where injury prevention is core business, mandated by legislation. ACC is the lead agency for the Falls and Drowning strategies. These strategies were developed by ACC specifically in response to the NZIPS. There is, however, a major barrier to ACC meeting its responsibilities under NZIPS.

ACC recently released its 2010–2013 Statement of Intent (SoI). As that document states, ACC has a legislative mandate, independent of NZIPS, in promoting measures to reduce the incidence and severity of personal injury. There is, however, a significant qualifier, and that is that Section 263 of the Accident Compensation Act 2001 requires that such measures only be undertaken by ACC if they are expected to lead to cost-effective reduction in levy rates. This is a major barrier to reducing injury since the cost of injury to society may be high but the cost to ACC low. Two examples illustrate these points.

ACC is the lead agency for drownings but as Table 1 shows that the human costs (non ACC costs) for drowning and near drowning exceed treatment and rehabilitation and weekly compensation costs (ACC costs) by 224 times. This reflects two issues. First, the ratio of deaths to non-fatal injury is close to 1:1. In other words, you if you get into difficulties in water you will either die or survive, and if you survive there is high probability you will be largely unscathed. Secondly, deaths cost ACC relatively little.

ACC is also the lead agency for falls where the estimated human cost @ $928m exceed ACC’s costs @ $777.3m. The question thus arises as to how can ACC exercise its wider injury prevention responsibilities, that is, pay due regard to human costs, under NZIPS? For example, a prevention programme that brings about reduction in childhood falls may have minimal benefit for ACC in terms of treatment and rehabilitation costs (and thus levy reduction) but a significant benefit in terms of reducing serious injury and human costs.

The Review is inappropriately silent on this important issue. This issue has become very salient in recent times as ACC has, with the recent change of government sought aggressively to reduce costs. Human costs will inevitably be the loser to ACC costs in this environment. This is well illustrated by the ACC recently withdrawing from the Otago Exercise Programme, an evidence-based programme aimed at reducing falls in elderly people. Given that the majority of elderly people are non-earners the impact of a falls reduction programme in this group would have a relatively small impact on weekly compensation, a major driver of levies.
Section 263 of the Accident Compensation Act, which places the levy restraint on injury prevention expenditure, also states that ACC can fund prevention activity if Parliament has appropriated money for such measures and they are included in the current service agreement. In the current economic climate such an appropriation seems very unlikely. How then is prevention expenditure which has a favourable benefit to cost ratio primarily in terms of human costs, that is minimal impact in terms of ACC costs, to be funded?

Since the inception of NZIPS, ACC has hosted and funded the NZIPS Secretariat. The authors of the evaluation report considered the appropriateness of this arrangement given that they considered the Secretariat needed to:

- Enhance its stakeholder management and information dissemination role;
- Adopt a whole-of-injury prevention policy function; and
- Design and implement strategies, policies and programmes for areas not covered by a lead agency.

The report concluded that ACC has the most direct interest in injury prevention outcomes and therefore is likely to be best placed to host the Secretariat function. It noted, however, that it was important that any policy work undertaken reflected a sector wide view of injury prevention, rather than an ACC focused view.

The existing secretariat arrangement has recently been confirmed by the Associate Minister in charge of ACC. As with previous administrations this reflects the Government’s intention that ACC lead the NZIPS. But one has to wonder about the ability of ACC to act as the leader given its recently renewed interest in only investing in injury prevention programmes that are expected to lead to cost-effective reductions in levy rates.

The Government has also made it clear that it is favourably disposed to opening parts of the accident compensation scheme to competition. This means that for some areas of injury (e.g. work injuries) there will be providers other than ACC, and they will be in competition with ACC and one another. In such an environment it is difficult to see how ACC could perform its leadership role.

Another potential explanation for the poor progress in reducing injury, especially all-injury, is that there are some significant injury issues that may not receiving the attention they deserve. As mentioned at the outset, there are six priority areas in NZIPS. There are number of dimensions on which priority areas can be decided. Several key factors influenced the original choice of six.

First, was the traditional way of describing the distribution of the burden, namely by the International Classification of Disease external cause codes, which are mixture of mechanism (e.g. car crash) and intent (e.g. self harm). Secondly, were the government organizations that have a legislative mandate to deal with specific injury problems (e.g. Department of Labour: work-related injuries) and thus can be held to account to a Minister of the Crown. Third, was that, collectively the priority areas would account for a substantial proportion of the overall injury burden. Finally, was the concern that having too many priority areas would defeat the concept of setting priorities.
The choice of the final six inevitably resulted in some tensions with some parties advocating for more priority areas, most notably injuries to children and to Māori. The latter area being promoted, in large part, on the basis of Treaty of Waitangi obligations.

The evaluation report recommends that, as NZIPS has been in place for only 5 years, the existing priority areas be unaltered. It noted, however, that injury to children and alcohol-related injury cut across most, if not all, of the existing priority areas and that stakeholders considered there was an absence of accountability for them. In addition, Māori injury and community engagement were considered issues that would benefit from increased focus.

The report recommended these four issues become focus areas and that lead agencies be required to identify specifically in their action/implementation plans ways to address these them. Given the response of some lead agencies to NZIPS to date, this is a very optimistic expectation. It also seems that one key opportunity was lost - namely to designate alcohol-related injury as a priority area. New Zealand has an Alcohol Advisory Council (ALAC) that has a legislative mandate to reduce the harm associated with alcohol consumption.

A 2005 report on the burden of death, disease and disability due to alcohol estimated that injury accounts for 51% of all alcohol-related deaths and 72% of years of life lost in New Zealand. Clearly, if ALAC is to make a significant impact on alcohol-related harm overall, it must tackle issues relevant to reducing alcohol-related injury harm.

One other potential priority area which warranted consideration and on which the evaluation report makes no mention is sporting and recreational injury. IPRU, in its submission to the evaluation, pointed out that the impact of sport and recreation injury in terms of serious non-fatal injury numbers was similar to assault and substantially greater than self-harm and near drowning. Moreover, there was only a modest (approx 30%) overlap between sports and recreation injury and the falls priority area.

The establishment of NZIPS represented a bold and internationally unique approach to injury prevention. The 5-year evaluation has raised some serious questions about its implementation. The evaluation has, however, failed to adequately address some other fundamental issues. The resolution of these issues is critical if NZIPS is to make a significant impact on reducing important injury in New Zealand.

Competing interests: None.

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References:


Drug use is a health issue

Simon J Adamson, Fraser C Todd

The Law Commission is reviewing the Misuse of Drugs Act (1975), following a request from the Associate Minister of Health. This review is long overdue. Over the past 35 years there has been a substantial change in drug use patterns, new drugs have emerged, and knowledge about the effects of different drugs and the effectiveness of different means to minimise the harms associated with drug use has increased.

The Misuse of Drugs Act (1975) was designed solely for the purpose of identifying which drugs were illegal, their level of illegality, and the corresponding penalties for a range of use, manufacture, importation and supply offences. Thus it attempted to control drug use through criminalising it. As with most other members of the United Nations, New Zealand is obliged to have legal sanctions against illicit drugs by being a signatory to the various United Nations drug conventions. Despite these widespread efforts to control drug use through the creation and enforcement of legal sanctions, however, drug use continues to be widespread. Drug use is not simply a legal issue however. It is a matter of substantial concern for the wellbeing of the individuals using drugs and for those around them who may be adversely affected by that drug use.

Health professionals and health services have had a substantial impact on this country’s response to harmful use of the legal drugs alcohol and nicotine. While the addiction treatment sector provides interventions for people using a wide range of drugs, the legal status of illicit drugs hampers the ability of health professionals to intervene. Not only may the illegality of a drug reduce the chance that a person experiencing problems with their drug use will disclose their use to a health professional, it also limits the ways health professionals can communicate with and influence drug users. Public health messages around cannabis are a good example of this.

Cannabis is the most widely used illicit drug in New Zealand. Whilst serious consequences may arise as a result of cannabis use there are many people using it who experience little or no serious harms. An important element in minimising harm around alcohol use is a moderation message. People can use alcohol moderately and are far more likely to consider reducing their drinking rather than trying to become totally abstinent. Moderation is defined by recommended upper limits for responsible drinking, such as those developed and promoted by the Alcohol Advisory Council (ALAC).

The illegality of cannabis means that it would be very difficult to undertake a public health campaign promoting moderation, and has also led to the situation where we don’t have a clear idea what would constitute moderate cannabis consumption.

The argument that decriminalisation of cannabis use would be associated with increased rates of use and harm is often used to justify its continued illegality. There is in fact little evidence that decriminalising cannabis possession is associated with
increased rates of use or increased harms and in young people in New Zealand where we have a high rate of cannabis convictions compared to many other countries, being convicted of cannabis possession does not appear to reduce a person’s cannabis use.

A hypothetical moderation message with respect to cannabis use and the actual development of the provision of safe and accessible drug injecting equipment are both examples of harm minimisation, the concept that reducing the harm associated with a behaviour should be our primary aim, rather than reduction in the behaviour per se, although clearly reduced use can form part of the means to reduce harm.

Harm minimisation arose in the 1980s as a concept in response to the HIV/AIDS epidemic. It is worth noting that this concept had not arisen at the time the Misuse of Drugs Act (1975) was passed into law. A new Act of parliament designed to address the harms of drug misuse must reflect the concept of harm minimisation. We would argue that decriminalisation of possession for personal use is a minimum step in this direction. There is good evidence to support this view.

Many countries and territories have decriminalised possession of drugs for personal use, perhaps the most notable being Portugal, which in 2001 decriminalised the possession of all drugs in favour of encouraging those with drug related problems into treatment. The results of this policy are worthy of note. While it is hard to attribute changes in the prevalence of drug use to this policy change, it is worth noting that there has not been an increase in drug use. There has, however, been a significant drop in a range of drug-related harms including new cases of HIV in drug users and drug related deaths from opiates and amphetamines.

The addiction treatment sector witnesses the harms of drug use on a daily basis. This experienced and educated workforce, a quarter to a third of whom are in recovery from the own alcohol or other drug problems, were surveyed in 2004. One question, not previously published, asked the 288 workers for their view on the legal status of cannabis. The most frequently endorsed option was decriminalisation for personal possession (38%) followed by no change to the current law or enforcement (27%), no change to current law but more lenient enforcement or sentencing (15%), increased penalties (12%) and legalisation (7%). In total, therefore, 60% favoured some sort of liberalisation of laws, particularly in regard to personal use.

Decriminalisation of drug use is not the same as legalisation, which we are not advocating. The preference that our citizens not use psychoactive drugs can and should still be promoted on the grounds that not using drugs is the best way to avoid drug related harm. By the same token, any law addressing drug use should also be designed to minimise drug related harm.

The Law Commission argues that the primary purpose of regulating drugs should be to minimise harm and with that in mind propose the decriminalisation of possession for personal use and what they describe as social supply (i.e. small scale and non-commercial). If this were the case then police, judicial and corrections resources could be directed towards those involved in the importation, manufacture and commercial distribution of drugs. The agencies then charged with addressing the use of drugs by individual New Zealanders would be health services.

The argument can be made that, just as there would be little property crime without people willing to buy stolen property there would also be no drug importation,
manufacture or dealing without people willing to use drugs, and thus personal drug use should remain a criminal offence of concern to law enforcement and the courts. There is a critical difference between these two scenarios however.

In the case of drug importation, manufacture and dealing, the victims of these offences are the drug users. Although it is acknowledged that drug use can produce victims beyond the user themselves this is also the case for gambling, alcohol and tobacco use. Many health interventions improve not only the wellbeing of the patient but also the wellbeing of those surrounding the patient.

We would therefore like to call for the enactment of new legislation that prominently reflects the reality that drug use is a health issue and acknowledges that drug illegality actively undermines the physical and mental health of drug users. The tenets of harm minimisation must hold sway if we are to achieve the greatest gain in health and wellbeing for New Zealand citizens when the Misuse of Drugs Act (1975) is laid to rest.

Competing interests: None.

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References:
Another bitter pill: a case of toxicity from DMAA party pills

Paul Gee, Suzanne Jackson, Josie Easton

Abstract

“Party pills” continue to be legally sold though the main ingredient is no longer 1-benzylpiperazine. Dimethylamylamine (DMAA) is a synthetic stimulant and is one of the main ingredients of new “BZP-free party pills”. Though patented in the 1950s as a nasal decongestant, little is known of its pharmacology via the ingested route. This case report describes a 21-year-old male who suffered a cerebral haemorrhage shortly after ingesting two capsules of DMAA.

‘Party pills’ are synthetic stimulants that emerged in New Zealand in the early 2000s. Most were 1-benzylpiperazine (BZP)-based and were widely consumed. Evidence accumulated of risk and toxicity and BZP was subsequently scheduled.1,3 Dimethylamylamine (DMAA) is one of the next generation of ‘BZP-free’ party pills. This report describes a case of a serious complication associated with the recreational use of DMAA.

Case report

A 21-year-old man was out with friends and purchased a quantity of legal party pills identified as ‘99.9%-pure DMAA’. He took the recommended dose of 2 tablets at approximately 11:30 pm along with a capsule identified as 150 mg of caffeine. He had already ingested 1 can of beer.

Within 30 minutes he developed a severe global headache and called for a friend to take him home. He subsequently became confused, incontinent of urine and vomited for 2–3 hours before falling asleep. The next morning he was drowsy and had slurred speech. He did not improve during the day so at 6 pm he was taken to the local hospital emergency department (ED).

On arrival in the ED he was confused and had slurred speech. He was disorientated in time but not person or place. He could not give a coherent history. He had a right facial droop and right-sided weakness. There was no sympathomimetic toxicity evident, however 19 hours had elapsed since ingestion. His heart rate was 65 bpm and blood pressure was 126/66 mmHg.

An urgent computed tomograph (CT) of the brain was requested and showed a large haemorrhage (66 mm × 21 mm × 31 mm) in the region of the left basal ganglia with mass effect causing 5 mm of midline shift.

A detailed examination revealed receptive and expressive dysphasia. His short term memory was impaired. He displayed constructional and dressing dyspraxias. He had impaired stereognosis. He had right-sided weakness grade 4+/5 in upper and lower limbs. He suffered a focal seizure involving his right arm and was commenced on phenytoin.
A subsequent cerebral angiogram failed to show evidence of aneurysm, arteriovenous malformation or any cerebral vasculitis to account for the haemorrhage. After 5 days he showed improvement and was transferred to a brain injury rehabilitation facility. He was discharged after 15 days of rehabilitation.

A detailed multidisciplinary assessment identified severe impairment to memory and abstract reasoning. Mild impairments of speech and right hand coordination were also noted. Identical pills obtained from the retailer were analysed and confirmed the contents as powdered 1,3 dimethylamylamine 278 mg per capsule. No caffeine, amphetamine, BZP or other stimulants were present in the sample.

**Discussion**

This is the first serious complication of DMAA reported in the medical literature. DMAA is marketed as a ‘BZP-free’ party pill. BZP was initially promoted as a safe herbal alternative to illicit street drugs. It was not scheduled or controlled by drug or food safety legislation. Concerns were expressed that these designer stimulants had toxic potential. Subsequent reports and research linked BZP to toxic seizures, renal impairment and multi-organ failure. BZP was banned in NZ and has also been scheduled in the United Kingdom and the European Union.

DMAA has been depicted as a benign herbal derivative (an extract of geranium oil—hence the alternate name ‘geranamine’). It is present at a concentration of less than 0.7% in naturally extracted geranium oil but the actual product is chemically synthesised and pure.

DMAA was patented by Eli Lilly in 1944 as a nasal delivery decongestant called ‘Forthane’. Animal studies confirmed its sympathomimetic properties and established an LD50 in rodents. Despite being sold and used in an inhaler form there is little published research on its effects in humans and none via the ingested or intravenous route.

In this case, cerebral haemorrhage is likely to be linked to the ingestion of DMAA and caffeine. The time sequence is suggestive of cause and effect. Cerebral haemorrhage is associated with both episodic and chronic stimulant use. Animal and autopsy studies show that amphetamines can induce microvascular injury and angiitis. Drug-induced hypertension may be a significant factor or may unmask underlying arteriovenous malformations or cerebral aneurysms.

In this case, no angiographic evidence of vascular anomaly was found. The background rate of intracerebral haemorrhage is ‘rare’ in people under 45 years old (less than 1.9 per 100,000 person years) and this figure is made up mostly of cases with underlying arteriovenous malformation and aneurysm. This makes haemorrhage without underlying pathology exceedingly rare. In addition a 2009 news report cited a New Zealand Ministry of Health document detailing three cases of severe headache with vomiting and one case of cerebral haemorrhage associated with DMAA use. DMAA has been legally available internationally as a bodybuilding supplement but is now being sold as a legal stimulant. DMAA has been banned by World Anti-Doping
Agency in their 2010 Prohibited list (listed under its synonym ‘methylhexaneamine’).\textsuperscript{13}

The New Zealand Ministry of Health has moved to regulate the sale of DMAA to those 18 years or older, but it is still legally available for human consumption.\textsuperscript{14}

Figure 1. DMAA capsules and packaging (brand name obscured)

![DMAA capsules and packaging](image1)

Figure 2. CT scan of the brain showing intracerebral haemorrhage

![CT scan of the brain showing intracerebral haemorrhage](image2)
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Necrotising pneumonia and pandemic influenza

Andrea Adjetey, Ruthraj Edwards

Abstract

A case of PVL-positive *Staphylococcus aureus* pneumonia in a 32-year-old female is described. This is one of the first reports of this particular organism causing pneumonia in New Zealand, although the organism has been identified before. With the likelihood of increased numbers of cases of this life-threatening infection in the future in connection with pandemic influenza, an approach to the management of patients with PVL-positive necrotising pneumonia is discussed.

Case report

A 33-year-old Māori female was brought into the accident and emergency department in the winter of 2009, complaining of shortness of breath, right-sided chest pain and haemoptysis. She reported that 1 week previously she had had the flu, from which she seemed to be getting better. However, the day before presenting she had recurrence of fever with associated rigors. She also complained of a new productive cough with blood stained sputum, and some right-sided pleuritic chest pain. She had no past medical history and did not take any regular medications, but was an active smoker, approximately 25 g of tobacco per day.

On arrival in the emergency department, she was markedly tachypnoeic with a respiratory rate of 40. Her oxygen saturations were 93% on air and she was tachycardic at 110 beats per minute. Blood pressure was low at 94/58 mmHg and she had a low grade fever at 37.6°C. Examination revealed coarse inspiratory crackles at the right lung apex.

Chest X-ray (CXR) demonstrated an infiltrate at the right apex and inflammatory markers were elevated with neutrophilia of $23.5 \times 10^9/L$ and C-reactive protein (CRP) of 513 mg/L. She was resuscitated with intravenous (IV) crystalloid, commenced on treatment for community acquired pneumonia with amoxicillin and roxithromycin and admitted to the ward.

IV fluids and antibiotics were continued over the following days. However, she was slow to improve. She continued to spike fevers up to 38.4°C, and systolic blood pressure remained between 80 and 90 mmHg despite continuing IV fluids. Oxygen saturations were low, running between 91% and 95% on air with respiratory rate between 20 and 30.

Two days following admission, blood cultures taken on arrival grew *Staphylococcus aureus* sensitive to flucloxacillin, but resistant to macrolides. Amoxicillin and roxithromycin were discontinued and IV flucloxacillin 1 gram four times daily was commenced.

Over the following days, she remained unwell. She continued to spike fevers up to 38.4°C, and systolic blood pressure remained between 80 and 90 mmHg despite continuing IV fluids. Oxygen saturations were low, running between 91% and 95% on air with respiratory rate between 20 and 30.
Repeat blood cultures were taken, but they did not yield any further information. Inflammatory markers remained high with CRP of 248 mg/L and neutrophils rising to $33.5 \times 10^9/L$. Flucloxacillin was increased to 2 g four times daily.

On day 8 following hospital admission she was continuing to spike fevers and remained hypotensive. Examination revealed right upper zone crackles and bronchial breathing, but no other signs to explain her ongoing fevers. Repeat CXR showed progression of the pneumonia, with patchy infiltrate and multilobar involvement (Figure 1).

**Figure 1. Chest radiograph 8 days after presenting to hospital**

A computed tomography (CT) chest scan was organised to investigate the possibility of a developing abscess. The CT revealed a severe, multi-lobar necrotising pneumonia (Figure 2). At this time further tests were organised to investigate other causes of a multilobar pneumonia. The possibility of tuberculosis was considered, though the patient denied a contact history.

A Mantoux test was performed and QuantiFERON-TB Gold sample was sent away for the exclusion of tuberculosis. The admission set of blood cultures which had grown *S. aureus* were also sent for further analysis to further define the strain of *Staphylococcus*. Specifically, samples were sent for PCR to detect the presence of the Panton-Valentine Leukocidin (PVL) gene. In the meantime, flucloxacillin was continued and clindamycin 300 mg IV four times daily was added.
Over the proceeding days the patient slowly improved. Her tachypnoea settled, her cough resolved and she began to feel well in herself. Her fevers ceased. Examination continued to reveal left upper zone crackles and bronchial breathing, but no heart murmurs and no other positive chest findings. Her inflammatory markers slowly decreased and on day 12 of admission, CRP had fallen to 105 mg/L, with neutrophils of $14.2 \times 10^9/L$. However, she was noted to have a haemoglobin of 75 g/L. She gave no symptoms of blood loss. She was transfused 2 units of packed red cells and continued on flucloxacillin and clindamycin.

On day 15 following admission, polymerase chain reaction (PCR) confirmed that the patient had a PVL-positive *S. aureus*. A further change of antibiotics to clindamycin and rifampicin was considered. However, as the patient was improving clinically and inflammatory markers continued to decrease, flucloxacillin and clindamycin were continued. Swabs sent to look for methicillin-sensitive *Staphylococcus aureus* (MSSA) carriage were negative.

On day 19 following admission, the patient was discharged to complete a further 2 weeks of oral antibiotics. At follow-up 8 weeks later, she was well and repeat CXR confirmed near complete resolution of the pneumonia with a small residual scar. She continues to remain well.

**Discussion**

The PVL toxin was not commonly identified in *S. aureus* until recently. PVL-producing *Staphylococcus* is emerging as a serious problem worldwide. There is no evidence-based guidelines for its management and the literature contains only approximately 100 cases worldwide with widely differing antimicrobial therapies.\(^1\)

Toxin production is an important aspect of the virulence associated with staphylococcal infections, as these toxins are likely to be responsible for the severity
of illness seen. The PVL toxin is a potent mediator of inflammation and destroys leucocytes. It has been associated with both MSSA and methicillin-resistant 
*Staphylococcus aureus* (MRSA) organisms and is a recognised cause of necrotizing pneumonia.

It is important to consider the diagnosis of PVL-positive *S. aureus* pneumonia as it is a serious infection. Mortality approaches 75% even with appropriate antibiotics, causes of death including sepsis, respiratory failure, and lung haemorrhage which may be massive and exsanguinating.\(^2\) Maximum survival has been quoted as 38%, and treatment requires special considerations.\(^1\)

There are several clinical features associated with this particular infection that may alert the physician to the possibility of a PVL-positive *S. aureus* pneumonia. It often affects fit, young patients. Presenting symptoms may include haemoptysis, hypotension, high fever, tachypnoea or severe sepsis, commonly following a recent flu-like illness.

Investigation findings which should raise the possibility of a PVL-positive pneumonia include multilobar infiltrates on CXR, leucopenia secondary to leucocyte destruction, high CRP level (>250–300 g/L), haemorrhagisis (anaemia resulting from destruction of red blood cells) and staphylococcal-like Gram-positive cocci on Gram film.

Treatment of a PVL-positive *S. aureus* pneumonia consists of IV antimicrobials. However, efficacy is decreased by reduced penetration into necrotic tissue and diminished activity in anaerobic conditions. As a result, IV flucloxacillin is generally not recommended.

Antibiotics more commonly considered include clindamycin, rifampicin, linezolid and vancomycin, together with IV immunoglobulins in severe cases.\(^3\)–\(^5\) Treatment should continue for a minimum of 2–3 weeks. The current consensus for management is outlined in the flow-diagram below.

Though *S. aureus* is an infrequent cause of community-acquired pneumonia (CAP), it is one of the more recognised causes of influenza-associated CAP. Outbreaks of influenza are likely to result in an increase in the number of staphylococcal pneumonia seen, and it is therefore important to consider this serious infection.

Early consideration of treatment for PVL-positive *S. aureus* infection following influenza is important to avoid the high mortality associated with this condition.
Figure 1. Management of patient with suspected staphylococcal pneumonia in the healthcare setting

IVIG=intravenous immunoglobulin.

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References:


Computed tomographic images of membranes in a pulmonary hydatid cyst
Prem P Gupta, Krishan B Gupta, Dipti Agarwal

A 44-year-male presented with progressive dyspnoea for 6 months, dry cough and chest discomfort for 3 months. His chest radiograph and computed tomographic (CT) thorax features, as shown here (Figures 1 to 5), were typical characteristic of a hydatid cyst.

Figure 1. Chest radiograph, postero-anterior view, showing large cystic lesion with air-fluid level having uneven interface suggesting floating membranes in cyst (water lily sign)
Figure 2. CT thorax axial view showing large cyst (size 154mm×124mm×126mm) in right lung with fluid that is having water attenuation. Unevenness of air-fluid interface is clearly visible; floating membranes of hydatid cyst being underlying cause.

Figure 3. CT thorax, axial view, showed membranes of hydatid cyst through the fluid as serpentine linear structures leading to uneven fluid level (water lily sign), a finding that is highly specific for hydatid disease. This sign appear as a result of collapse of innermost lining of the cyst (endocyst).
Hydatid disease (echinococcosis) is a parasitic disease known to affect humans and other mammals. Four *Echinococcus* species have been identified: *Echinococcus granulosus*, *E. multilocularis*, *E. vogeli* and *E. oligarthus*.

*E. granulosus* has a worldwide prevalence particularly in regions where cattle-rearing is common. *E. multilocularis* mainly occurs in central Europe, northern parts of Europe, Asia, and North America. *E. vogeli* and *E. oligarthus* are not so common and their prevalence is limited to Central and South America.
Although echinococcosis may involve any organ, liver (in 75% of cases) and lungs (in 5–15% of cases) are most frequently affected. Most of the lesions may grow until picked up on imaging modality or become large enough to compromise the functions of host organ.¹

Plain radiographs are useful in localising the lesions, and in cases with pulmonary disease, floating membrane in cyst (or water lily sign) is of high diagnostic value. Ultrasonography is a useful initial diagnostic technique; cysts with a visible split wall inside, septated cysts, or those with a honeycomb pattern are pathognomonic. CT scans usually show cyst(s) with fluid having water attenuation (3-30 HU).² The matrix represents hydatid fluid containing membranes of broken daughter cysts, scolices, and hydatid sand.

Membranes may appear within the matrix as serpentine linear structures, a finding that is highly specific for hydatid disease (water lily sign). Multilocular cysts manifest as well-defined fluid collections in a honeycomb pattern with multiple septa—a spoke wheel pattern.

Serological diagnosis include detection of anti-*Echinococcus* antibodies IgG using indirect haemagglutination test or enzyme-linked immunosorbent assay.

Usual treatment of echinococcosis includes chemotherapy using albendazole and/or mebendazole along with surgical removal of the cysts whenever feasible though multi-organs or vital organ involvement makes surgery impractical.

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**References:**


A thrilling mass

Rajesh B Dharmaraj, Abu H A Samah, Jon Griffin, Anantha Ramanathan

Clinical

An 88-year-old woman with longstanding dementia was referred to the Emergency Department with a 1-day history of dizziness, increasing confusion and unexplained hypotension.

On examination, she appeared pale and confused. Her heart rate was 85/min, blood pressure 84/57 mmHg and oxygen saturation was 98% on room air. There was a mildly tender, pulsatile mass in the central abdomen associated with an abdominal bruit on auscultation.

An urgent CT angiogram of the abdominal aorta was performed demonstrating the findings shown in Figures 1 and 2.

Figure 1. A transverse section of CT angiogram of the aorta showing a communication between the abdominal aortic aneurysm and the inferior vena cava

![CT Angiogram Image]
Figure 2. An image of the aortocaval fistula formation seen on coronal section of the CT angiogram of the aorta

What is the diagnosis and what are the management options?
Answer and discussion

The CT angiogram shows the inferior vena cava filling with contrast early in the arterial phase, a finding consistent with an aortocaval fistula. There is no evidence of intra- or extra-peritoneal rupture.

Aortocaval fistula was first described by Syme\(^4\) and is a rare but life-threatening complication of abdominal aortic aneurysm. It is found in less than 1% of all abdominal aortic aneurysms and occurs in 3–4% of ruptured aneurysms.\(^1,2\)

The clinical presentation is variable and largely depends on the size and the location of the fistula.\(^1\) The triad of low back pain, palpable abdominal aortic aneurysm and a machinery abdominal murmur is diagnostic, and may be associated with high-output cardiac failure and regional venous hypertension.

Aneurysmorrhaphy in aortocaval fistula carries an operative mortality of approximately 30%, no greater than mortality associated with other ruptured abdominal aortic aneurysms.\(^2\) Endovascular repair has been reported.\(^3\)

Given the patient’s age, comorbidities and pre-morbid status, the decision was made to proceed with non-operative management and palliation. She steadily deteriorated over the next few days and passed away on day 5 post-presentation from cardiac failure.

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On some principles of hospital management: part 1

First part of an article by Dr Colquhoun, Dunedin, published in NZMJ 1910;9(36):33–37.

Last year (1906) I brought before the Staff certain suggestions as to Hospital reform. I thought then and I think still that there are many essential elements in the relationship of the Medical profession to the Hospitals which need to be clearly defined by ourselves and which are only very vaguely comprehended by the public. Further, I think the time is ripe for the Staff to try to enunciate clearly, for their own benefit and that of the public:

1st. The principles on which Hospital aid should be given.

2nd. The reforms which are necessary in order that Hospital aid may be given most effectually and economically.

With regard to the first point, I submit the following statements as being almost axiomatic:

I. Hospital aid should be available in all cases where patients are unable to pay for suitable advice and attendance.

II. People who can afford to pay for medical attendance and advice are not entitled to Hospital aid.

In supporting the first proposition, apart from altruistic consideration, the State, if it reasons properly, is forced to provide help for the sick. Every sick man is a loss to the community while he is ill. The State is poorer by the loss of his work, he ceases to be a customer to other workers, he takes away from the common store of working powers a certain amount of labour which might be profitably applied. Individually the worker who is sick suffers loss, the amount of which it is not easy to estimate, but which is always great and often ruinous.

Another reason for State aid is that many of the diseases conditions to be dealt with are infectious. They give rise to an endless chain of similar sicknesses, and thus if not checked are a menace to the whole of the public.

The second proposition is one on which there will be in the immediate future much debate, and on which we should clear our own minds and make our position plain the public. First it may be noted that the rule ‘the hospital for the sick poor,’ has never been applied in a rigid or narrow sense. We have always given and probably will always give cheerfully medical help for nothing to classes and individuals whom nobody else supplies for nothing—people who are charged fees by the clergyman and the lawyer, who are expected to pay the landlord for the houses they live in, and the baker and butcher for what they eat.

But if the hospital is to be free to all without distinction, it is clear that Doctors must reconsider their position, and the public must understand what such a step would mean. The term ‘A Free Hospital’ is a misnomer—somebody has to pay for it.
New Zealand Society of Gastroenterology Annual Scientific Meeting, 17–19 November 2010

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Site-specific memory T cells generated from sustained antigen-release vaccination. A Highton\textsuperscript{1}, A Girardin\textsuperscript{1}, S Hook\textsuperscript{2}, R Kemp\textsuperscript{1}. \textsuperscript{1}Department of Microbiology and Immunology, Otago School of Medical Sciences, \textsuperscript{2}School of Pharmacy, University of Otago, Dunedin.

Memory cells of the immune system protect us from secondary infection of pathogens and are the underlying reason that vaccines are protective. Understanding how to generate an effective memory cell population of quantity, quality and in the correct biological location is key in having effective vaccination methods. The aims of this study were to evaluate the efficacy of sustained release vaccines and compare their ability to generate peripheral versus mucosal memory.

We investigated the generation of memory CD8+ T cells in sustained antigen-release vaccines (chitosan gel, cubosomes) and dendritic cell (DC) vaccination using the model antigen ovalbumin. Lymphocytes were collected from peripheral and mesenteric lymph nodes, spleens and Peyer’s patches of mice euthanised 30 - 40 days following subcutaneous vaccination with 25 µg ovalbumin in chitosan gel, 1 mg of ovalbumin in cubosomes, or 1x10\textsuperscript{5} DCs. Memory CD8+ T cells were identified and phenotyped by incubation with Major Compatibility class one pentamer, antibodies against CD122, CD44, CD103, CD127 and then detected through flow cytometry.

Memory CD8+ T cells (Pentamer+CD122+CD44+) were generated and sustained, in peripheral lymph nodes, at a higher level from sustained release vaccines than from dendritic cell vaccination, with 10381 ± 5939 (mean ± SEM, n = 3), 4432 ± 3657 and 500 ± 341 cells with gel, cubosome and DC vaccination, respectively. Distinct populations of memory cells were detected in peripheral and gut mucosal lymphoid organs following the different vaccine regimes. Chitosan gel vaccination gave lower CD127 expression in peripheral lymph nodes (mean fluorescent intensity: 143.7 ± 5.608) compared to Peyer’s patches (756 ± 240.5; \(P < 0.05\); ANOVA, Tukey’s post-hoc test).

These results indicate that sustained release vaccines may have advantages in developing CD8+ T cell memory populations of differential phenotype in peripheral and mucosal tissue, with chitosan gel vaccination the most successful in generating mucosal memory.
Psychological stress exacerbates colitis in interleukin-10 knockout mice. C Lai1,2,4, A Lindstrom1,2, M Thompson-Fawcett1,4, A Butt4, M Schultz1,3.
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Psychological stress is perceived to play a significant role in initiation and exacerbation of inflammatory bowel disease (IBD). We investigated the effects of psychological stress in a mouse model of IBD.

Under specific pathogen free conditions, interleukin-10 knockout (IL10-/-) mice were stressed by physical restraint for 2 hours twice a day for 7 days in 17 x 20 x 110 mm chambers. Two age groups were assessed: 4-week old disease-free juveniles and 7-week old adults with subclinical disease, and compared with controls (n = 10/group). Bodyweight, faecal pellet and faecal water content were monitored daily. After 7 days, mice were euthanized; sections of terminal ileum (TI) and proximal colon (PC) were collected and either fixed and stained in haematoxylin and eosin, then blind-scored for histological inflammation, or mounted in Ussing chambers to measure permeability.

Increased faecal pellet production in both stressed groups indicated that the restraint was associated with stress (juvenile: ratio 4:1, P < 0.001; adult: ratio 2:1, P < 0.01, one-way ANOVA, followed by Tukey’s post-hoc test). However, final bodyweight, intestinal histology and permeability in stressed juveniles did not differ from control even though faecal water content was increased (64 ± 0.5% versus 60 ± 0.7%, mean ± SEM, P < 0.001). In contrast to the juveniles, final bodyweight of stressed adults was reduced (-7.4 ± 1.1% versus -0.8 ± 0.5%, P < 0.001). Faecal water content was increased (69 ± 0.5% versus 60 ± 1.2%, P <0.001). There was histological inflammation (PC: 2.3 ± 0.3 versus 1.2 ± 0.2, P < 0.01, Mann-Whitney test). Despite obvious intestinal inflammation, permeability was unaffected (PC: 1.35 ± 0.22 versus 1.37 ± 0.11 [x10^-6 cm/s], P > 0.05).

In conclusion, psychological stress does not initiate colitis in IL10-/- juveniles but triggers a flare-up in adults without preceding intestinal barrier dysfunction.

Differential requirements for T cell receptor and gamma chain cytokine receptors in naïve and memory T cells. J Prier, A Girardin, R Kemp.
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Identifying the factors that mediate T cell survival and proliferation is necessary to fully understand immune responses. The role of gamma chain receptor cytokines, interleukin (IL)-2, IL-7 and IL-15 in maintaining naïve and memory CD8+ T cell populations is well established. Survival of naïve T cells requires signals through the T cell receptor (TCR) in concert with signals from gamma chain cytokines, while memory T cells are maintained independently of TCR signals. Here, we aimed to determine the contribution of the janus kinase/signal transducer and activator of
transcription (Jak/STAT) and phosphoinositide 3-kinase (PI3K) pathways in TCR-dependent versus cytokine-dependent signalling in memory versus naïve cells.

Lymphocytes were harvested from euthanised naïve C57BL/6 or OT-I mice or vaccinated C57BL/6 mice, then incubated with IL-2, IL-7, IL-15 or antigen in vitro. Cell survival and activation of signalling pathways was determined by incubation with specific monoclonal antibodies that were detected by flow cytometry.

Naïve CD8+ T cells showed increased levels of phosphorylated STAT5 and ribosomal S6 kinase (S6K) in the presence of IL-7 (STAT median fluorescent intensity (MFI): IL-7, 218; control, 110, S6K MFI: IL-7, 522; control, 238), indicating that IL-7 can activate both Jak/STAT and PI3K pathways. CD8+ memory T cells were enhanced in the presence of IL-15 (0.22%) and IL-2 (0.22%) but not IL-7 (0.14%) compared to the unstimulated control (0.20%). Resting CD8+ memory T cells had a higher basal level of phosphorylated STAT5 compared to control T cells (MFI: resting, 745; control, 467).

Together, these data suggest differential roles for gamma chain cytokines in maintaining naïve versus memory CD8+ T cells via activation of the Jak/STAT and PI3K pathways. Ultimately, this knowledge can be employed to manipulate the immune responses critical for protection against infectious diseases and cancer, by improving the current cell-mediated vaccine strategies.

Virtual digital measurement of facial soft tissue and bone from living subjects. L Baillie¹, P Blyth¹, I Premachandra², G Dias¹. ¹Department of Anatomy and Structural Biology, Otago School of Medical Science, University of Otago, Dunedin, ²Department of Finance and Quantitative Analysis, School of Business, University of Otago, Dunedin.

Forensic facial reconstruction uses tables of averaged Facial Soft Tissue Depth (FSTD) measurements within its technique. Large errors, estimated between 10% to 30%, are associated with these FSTDs. There have been multiple measurement techniques with various protocols and no assessment of method errors. The most common technique has used needles to probe soft tissue depths on cadavers at identified landmarks. Sources of error include estimation of underlying bone landmarks, embalming, supine position, angle and length of peg probe, tissue compression, and averaging of non-normal or highly variable values. These errors have resulted in facial reconstruction being termed facial approximation, with no legal validity. This study examined whether Cone Beam Computed Tomography (CBCT) followed by computational analysis might represent a technique to decrease error in facial reconstruction.

CBCT datasets from fourteen living volunteers were segmented into skin and bone surfaces in Osirix (osirix-viewer.com, freeware) then transferred to Blender (blender.org, freeware) as object files. Within Blender each skin and bone object file was distinct yet remained linked in the x, y and z planes. Cylindrical shapes were created as measuring pegs. Each peg was lengthened from an identified bone landmark to the skin, and in this way 42 FSTD and 17 Craniofacial measurements (CMM) were taken from each dataset. Intra-observer error was assessed by measuring three of the fourteen data sets three times, on three separate occasions. Repeatability
was calculated by taking the difference of each observation from the mean (n = 3) and deriving a 95% confidence interval for the absolute differences, the raw error. All except five of the 378 FSTDs and all 153 CMM intra-observer measurements showed less than 5% error, indicating that the technique was highly repeatable.

This study demonstrates a novel CBCT-based method for FSTD and CMM measurements that overcomes many sources of error in current methods.

Neuromuscular blocking actions of novel pinnatoxins E and F. S Hellyer, S Kerr. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

The cyclic imine toxins, gymnodimine and spirolides, are potent antagonists of both muscle type and neuronal nicotinic acetylcholine receptors and cause death within minutes by respiratory depression. This toxicity is shared by the novel cyclic imine pinnatoxins E and F (PnTx\(_{E/F}\)), which are produced by a newly discovered species of marine dinoflagellate and enter the food chain via uptake into shellfish. However, there is currently little data regarding the mechanism of action for any of the pinnatoxins, and no data at all on PnTx\(_{E/F}\). Here we investigated potential PnTx\(_{E/F}\) antagonism of nicotinic acetylcholine receptors using two in vitro tissue preparations.

Sprague-Dawley rats were euthanised and hippocampi and hemidiaphragms dissected. Compound muscle action potentials elicited by stimulation of the phrenic nerve were recorded from the hemidiaphragm in order to test PnTx\(_{E/F}\) effects on muscle type heteromeric nicotinic receptors. PnTx\(_{E/F}\) effects on \(\alpha_7\) homomeric neuronal nicotinic receptors were investigated by recording hippocampal gamma oscillations from CA1 pyramidal cells in response to tetanic stimulation of the Schaffer-collateral pathway. A crude extract of PnTx\(_{E/F}\) had no effect on hippocampal gamma oscillations (500 nM; n = 4 slices), but caused a decrease in amplitude of the hemidiaphragm compound muscle action potential to 38.2 ± 14.0% and 28.7 ± 17.7% at concentrations of 500 nM (n = 5) and 3 µM (n = 4), respectively (\(P < 0.05\) each, paired \(t\)-test). Pure pinnatoxin F also caused a dose-dependent reduction in compound muscle action potential amplitude, with decreases to 30.6 ± 14.8% and 25.1 ± 10.6% of baseline at 260 nM (n = 4) and 520 nM (n = 4), respectively (both \(P < 0.01\)).

These results show that PnTx\(_{E/F}\) blocks neuromuscular transmission and suggest that observed in vivo muscle paralysis by PnTx is due to selective antagonism of muscle type nicotinic acetylcholine receptors.

Modulation of supraoptic nuclei neuronal activity by a glial \(\gamma\)-amino butyric acid transporter. N Joe, V Scott, C Brown. Centre for Neuroendocrinology and Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

Magnocellular neurons of the hypothalamic supraoptic nucleus (SON) and paraventricular nucleus (PVN) project to the posterior pituitary gland, where they secrete the hormones, oxytocin and vasopressin, into the bloodstream in proportion to their action potential (spike) firing rate. Vasopressin inhibits diuresis (urine
production) whereas oxytocin promotes natriuresis (sodium excretion) to reduce plasma osmolality. Ambient concentrations of the inhibitory neurotransmitter, γ-amino butyric acid (GABA), underlie a sustained form of inhibition (tonic inhibition) of the activity of SON neurons. Here, we tested the hypothesis that glial cells surrounding the SON neurons modulate SON neuronal activity by regulating the ambient levels of available GABA in the extracellular space.

In vivo electrophysiology experiments were conducted on urethane-anæsthetized female virgin Sprague-Dawley rats. Extracellular recordings of firing rate were made from SON neurons from these rats where β-alanine, a selective inhibitor for the glial GABA transporter, GAT3, was administered locally into the SON via a microdialysis probe over 1 h. In the presence of β-alanine (100 mM), SON neurons decreased their firing rate from 4.9 ± 0.8 to 2.5 ± 0.4 spikes s⁻¹ (n = 6, P < 0.05; paired t-test).

Hence, it appears that glia are able to take up GABA from the extracellular space to modulate the firing activity of SON neurons. Modulation of ambient GABA concentrations might be important in ensuring that the appropriate amount of vasopressin and oxytocin is secreted to meet any changes due to physiological demands that may be placed on the organism such as a change in plasma osmolarity; this will be the subject of future study.

Preserving taste function in middle ear surgery: mapping the intraosseous course of the chorda tympani nerve. L McManus¹, P Dawes², M Stringer¹. ¹Department of Anatomy & Structural Biology, Otago School of Medical Sciences, ²Department of Medical and Surgical Sciences, Dunedin School of Medicine, University of Otago, Dunedin.

Few studies have attempted to define the intraosseous course of the chorda tympani nerve (CTN) which conveys taste sensation from the anterior two-thirds of the tongue and provides parasympathetic innervation to the submandibular and sublingual glands. Inadvertent injury of the CTN is a complication of middle ear surgery, which may result in taste alteration, dry mouth, and unpleasant sensory symptoms. The aim of this study was to accurately define the intraosseous course of the CTN.

Twenty cadaver temporal bones (seven female, mean age 72 years, three bilateral) were scanned using a SkyScan micro-computer tomography (CT) scanner. Three-D multiplanar reconstructions were generated using the software platform Amira 4. Images were oriented to Reid’s plane using data derived from the orientation of the horizontal semicircular canal. The posterior canaliculus was measured in relation to standardised bony landmarks using the image processing programme ImageJ.

Results showed that the CTN originated from the facial nerve outside the skull in 4 specimens and from within the facial canal in 16 specimens (80%). In the latter, the posterior canaliculus arose 2.1 ± 1.5 mm (mean ± SD) above the stylomastoid foramen at an angle of 19 ± 14 degrees to the parasagittal plane and 16 ± 6 degrees to the coronal plane. The posterior canaliculus was 12.6 ± 4.0 mm long with maximum and minimum diameters of 1.1 ± 0.2 mm and 0.5 ± 0.1 mm, respectively. The CTN entered the middle ear above the mid-point of the annulus of the tympanic membrane in all cases.
This microCT study provides the most accurate description to date of the anatomy of the posterior canaliculus, which houses the chorda tympani nerve. These data not only yield important anatomical information but should also assist surgeons in protecting the nerve from inadvertent injury during middle ear surgery.

**Interleukin-6 interacts with bovine adrenal medullary chromaffin cells.**

**D Sreenivasan, S Bunn. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Chromaffin cells are traditionally recognised for their role in the neuronally mediated ‘fight-flight’ response and also respond to a range of other stressful stimuli by secreting both catecholamines and neuropeptides. A relationship between the immune and stress systems is likely to be of physiological importance and a dysregulation of this interaction may contribute to a number of pathologies. While the receptor for the cytokine, interleukin-6 (IL-6), has been found within the adrenal gland of a number of species its mechanism of action has not been previously described. The aim of this study was to characterize the action of the immune-derived IL-6 on the chromaffin cells of the adrenal medulla.

Bovine chromaffin cells were isolated, purified and cultured on collagen-coated wells. Cells were then washed with physiological buffer and incubated with 1 nM IL-6 for 5-30 mins. Western blotting was employed to detect the activation of specific signalling proteins, the blots were scanned, quantified by densitometry and comparisons made between basal and IL-6 stimulated samples using a Mann-Whitney U-test.

IL-6 significantly increased the phosphorylation of signal transducer and activator of transcription (STAT)3 by approximately three fold which was maximal after 15 min ($P < 0.05, n = 5$). Immunohistochemistry showed that approximately 70% of chromaffin cells had phosphorylated STAT3 within their nuclei at this time. IL-6 also significantly increased the extracellular signal-regulated kinases (ERK)1/2 phosphorylation by approximately two fold (maximal at 5 min, $P < 0.05, n = 3$) but not the phosphorylation of two other mitogen activated protein (MAP) kinases, p38 or c-Jun N-terminal kinases (JNK).

These data provide convincing evidence that the neuroendocrine chromaffin cells are responsive to IL-6 thus suggesting a potential pathway linking immune signals with the adrenal stress response. The ability of IL-6 to activate both ERK1/2 and STAT3 may allow regulation of both acute and chronic activity of the chromaffin cells. Experiments are currently underway to identify possible changes in gene transcription occurring in response to this cytokine.

**Alcohol consumption and next-day functioning in University of Otago students.**

**M Polak, T Conner. Department of Psychology, University of Otago, Dunedin**

Excessive consumption of alcohol among tertiary students is a major public health issue. The dangers of heavy drinking, also called "binge" drinking, are well known, with both acute and long-term consequences. However, less is known about the dangers of extreme drinking, which we define as more than twice the heavy drinking...
threshold (10+ standard New Zealand drinks for women/12+ drinks for men). The current project investigated the drinking patterns of 225 University of Otago students and the effects of different levels of alcohol consumption on the next-day physical function (sleep duration and quality, tiredness, and physical illness), cognitive function (concentration problems and workload management), and emotional function (positive and negative moods and stress).

Data were collected using an internet-based daily diary procedure, in which participants (age 19.8 ± 1 years) reported their alcohol use as number of standard drinks consumed “last night” and level of functioning on a five point scale each day for 21 days. Overall, participants reported drinking alcohol on 6 out of 21 of days and consuming an average of 7.3 standard drinks per report. Heavy drinking (in excess of safe drinking levels of 4+ drinks for women and 6+ drinks for men) occurred on 3 out of 21 days and extreme drinking (in excess of 10+/12+ drinks) occurred on 2 out of 21 days. Heavy drinking, and particularly extreme drinking, predicted significant decreases in physical and cognitive functioning the next day. Safe levels of drinking were associated with no, or minimal, functional decreases. Functioning was similar regardless of whether participants abstained from drinking or drank at the safe level the night before. Drinking within safe levels did not cause any impairment, whereas heavy drinking caused significant impairment (e.g. $P < 0.001$ for ability to concentrate, $t$-test). Extreme drinking caused more severe impairment than heavy drinking (e.g. $P = 0.001$ for ability to concentrate, $t$-test). These findings suggest the need to differentiate further between heavy and extreme drinkers.
Antipyretics in the treatment of influenza

The authors of this paper point out that health authorities in both the USA and UK have produced guidelines that recommend that paracetamol or ibuprofen be used to treat fever and systemic symptoms of influenza, in both children and adults.

There is no evidence base for these recommendations and the authors have systematically searched the literature. They found no relevant human studies. They recovered 8 animal studies and somewhat alarmingly report that the risk of mortality was increased by antipyretic use in influenza-infected animals.

An increased risk was observed with aspirin, paracetamol and diclofenac. Consequently they recommend that appropriate placebo-controlled trials of antipyretic drugs in influenza be conducted.


Proton-pump inhibitors and the risk of birth defects

Gastro-oesophageal reflux is common in pregnancy. Consequently such women are likely to be treated with proton-pump inhibitors (PPIs). The question arises—could PPIs endanger the health of the foetus by causing potential birth defects? This question is reviewed in a nationwide cohort study conducted in Denmark. It involved 840,968 live births. In 5082 women exposed to PPIs in the first trimester there were major birth defects in 3.4%, compared with 2.6% in the cohort not exposed to PPIs.

This difference was not significant and consequently the researchers conclude that exposure to PPIs during the first trimester of pregnancy is not associated with a significantly increased risk of major birth defects.


Elevated troponin 1 levels in older patients after emergency orthopaedic surgery

The authors of this study have noted previously that over 50% of orthopaedic patients sustained a post-operative troponin rise. This prospective study is intended to determine the association between post-operative troponin rises and longer term (2-year) mortality after emergency orthopaedic surgery in patients over 60 years of age.

Elevated post-operative troponin levels were shown to be predictive of 1-year but not 2-year mortality in older patients undergoing emergency orthopaedic surgery. They also noted that pre-existing atrial fibrillation and post-operative renal failure were significantly associated with cardiac events. The question arises—would early intervention in the post-operative period be helpful to those with such troponin level elevation?

High-dose allopurinol in patients with stable angina pectoris

Recently (NZMJ 10 September 2010) we abstracted a paper on this topic. The authors claimed that in selected patients 600 mg of allopurinol per day was a well tolerated and safe anti-ischaemic drug for patients with angina.

This paper has provoked discordant views which argue that widespread use would be dangerous. Concerns aired include the risks of toxic epidermal necrolysis and other toxicity in the elderly, renally impaired patients who are frequently subject to polypharmacy. In reply, the authors restate and reiterate their views. Perhaps it would be safe in the younger, otherwise healthy patient.

I note with interest, and surprise, that the senior author of the original paper and his university have applied for a patent for the use of xanthine oxidase inhibitors to treat anginal chest pain.


Drug-eluting vs bare-metal stents in the management of large coronary artery disease

Drug-eluting stents have been shown to reduce re-stenosis rates when compared to bare metal stents. However, some believe that the drug-eluting stents may be more prone to the risk of late re-stenosis.

In this prospective trial, 2314 patients were randomly assigned to receive sirolimus or everolimus-eluting stents or bare metal stents. They report that at 2 years there was no significant between-group difference in the rate of death from cardiac causes or nonfatal myocardial infarction.

A patient journey and the ACC

My experience begins on 19 January 2010. A group of us were waterskiing on Lake Benmore. I had a high-speed fall while crossing the boat wake. I was immediately aware of a shoulder injury, having to raise my left instead of right hand to signal that I was otherwise well in the water.

I was aware of a severe limitation of right shoulder function from that time. It was a witnessed significant injury to a previously asymptomatic shoulder. Previous regular sporting activities included biking and tennis (during which I experienced no problems with overhead shots). I was 49 years old at the time. I am now 50.

Shortly after my return to Christchurch I consulted my general practitioner. An Accident Compensation Corporation (ACC) claim for the injury was registered on 29/01/2010. Shoulder X-ray, ultrasound and MRI were performed.

I am a practising general radiologist with interests including musculoskeletal imaging and my practice allowed me to have these without an immediate charge.

In summary, the result of imaging was:

- X-ray demonstrated no boney predisposition to rotator cuff pathology or evidence of preexisting tear.
- Ultrasound and MRI (28/01/2010) demonstrated a supraspinatus tear.
- On MRI the tear was a high grade partial tear but in a good undisplaced position.
- Associated bone oedema underlying the supraspinatus insertion and bursal fluid/inflammation were present.
- Moderate AC joint arthropathy and mild fraying of the superior labrum were also noted.

I was able to see an orthopaedic surgeon at short notice. His opinion was that there was a good chance the tear would heal in an unchanged good position with conservative management. We would check progress with another MRI scan in about 3 months.

Living with this injury to my dominant right shoulder was life-changing. I was unable to engage in usual family pastimes, perform most household chores or get things done in the garden. My sporting activities (a significant focus of my socialising outside work) ceased.

Prior to the injury most working days would include doing some ultrasound and/or procedures. For the first 6 weeks post injury my activities were limited to reading imaging examinations only. After that I did expand my work mix a little to include some of the ultrasound and procedures that I would have done pre injury, partly because of stresses on the roster, but doing them was difficult and painful. Always, at the back of my mind, was concern about the possible impact on my career.
During that time I was contacted by my ACC officer 2 to 3 times to explain that a decision had not yet been made. I was relaxed. I still hoped that my shoulder would heal without intervention and my only expenses to date had been about 2 or 3 physiotherapy appointments recommended by my surgeon.

Things changed with the second MRI scan on 08/04/2010 which demonstrated that the supraspinatus tear was not healing and was now distracted. Findings otherwise were similar. Bone oedema and bursal fluid were still present.

Surgical advice was now that an operation was necessary and looking ahead, there were limited spaces to fit me in during the next several months.

I now needed the ACC to make decisions about my initial claim and the need for surgery. A letter dated 19/04/2010 referenced “We’re sorry, we can’t approve your claim” advised that the ACC had reached the end of its legislative timeframes in which to make a decision but that they would continue to assess the claim. The letter was confusing and provided no guidance as to what would happen next or when.

My private medical insurance would not agree to pay while I may still be covered by the ACC. I then made multiple telephone calls to the ACC. The decision was finally made to accept my claim for the injury on 28/04/2010 and yet funding for surgery to repair the shoulder was declined on 11/05/2010. I was now dealing with a different officer. The medical opinion below is as it appeared on a letter to me.

“COMMENT: ANDREW HAS CLINICAL EVIDENCE OF HIS RIGHT SHOULDER SUBACROMIAL IMPINGEMENT SYNDROME * – AN INTRINSIC DEGENERATIVE CONDITION RENDERED SYMPTOMATIC”

With private medical insurance cover, I had my operation on 4 June 2010. My supraspinatus tendon was repaired, my glenoid labrum was repaired and an acromioplasty was performed. I understand that after a slow start my progress has been as expected and now in early November I have no significant limitation of function at home or at work. There are still some limitations on sporting activities.

I applied for a review of the decision not to cover surgery costs. The first written application to the ACC was declined (26/07/2010).

The letter to me included a faxed handwritten medical opinion which I was not able to read completely. Reasons to decline my application included the operative findings, tendinopathy, bursitis, impingement and, I think, preexisting symptoms. The operative findings had not been discussed with the surgeon. The surgeon’s opinion at operation was that findings were in keeping with the result of a traumatic event. Tendinopathy, implying preexisting compromise of the tendon had not been mentioned in an imaging report or the operative findings.

The second application resulted in a hearing in front of an independent reviewer on 20/08/2010. Written submissions to the reviewer and the ACC with all relevant documents prior to the hearing were required. At the hearing an ACC representative was to be in attendance, by telephone in my case.

Following the preliminaries at the beginning of the hearing I stated my case, basically talking to my written submission. The ACC officer made no comment. I wondered why things had gone that far.
Following this, the decision not to fund my right shoulder surgery was quashed. (Almost exactly 7 months after the injury.)

Through this whole experience I consider myself to have been in a privileged position:

- I was able to organise imaging and an orthopaedic assessment at short notice and at no personal expense.
- I have private medical insurance which covered the surgery costs once the ACC had made a decision either way.
- Finally as a radiologist with an interest in musculoskeletal imaging I had sufficient knowledge and confidence to persist with the potentially intimidating review process.

I continued with the review process and am writing this letter because:

- In my opinion a case of a single traumatic injury to a previously asymptomatic joint which demonstrated no evidence of previous injury or chronic condition could not have been more clear cut. Bursitis/bursal fluid (seen with most recent rotator cuff tears and an imaging finding to indicate that a tear is likely to be recent) and impingement (which was secondary to the tear in my case) repeatedly clouded the primary problem which was the supraspinatus tear.
- I now have first-hand knowledge of the potentially life-changing result of a shoulder injury particularly to a dominant upper limb (unlike others I was able to continue to work with my injury) and the stress and frustration that repeated delays/adverse decisions cause.

I would now like to do what I can to ensure that others who may be less able to argue their case and/or continue with appropriate management without ACC funding, and who clearly have a shoulder injury and require surgery, get accurate decisions quickly. The review process takes time and money and is potentially intimidating. As a result I understand that many do not see the review process through.

Andrew Slaven
Christchurch
Chalmers, Bunkle and Bryder correspondence

Reluctantly, I am rejoining the now acrimonious correspondence relating to the ‘unfortunate experiment’. I am doing so only to state again the clinical facts that Cartwright’s adversaries find so hard to accept.

In the mid-1960s a group of women with high grade cervical smear abnormalities were referred to the National Women’s Hospital (NWH) by general practitioners in the expectation their patients would receive the same conventional treatment (cone biopsy/hysterectomy) for carcinoma in-situ (CIS) their patients had received during the previous decade. Instead, those women who were directed to Dr Green’s care, were without their consent entered into a study that involved a small diagnostic biopsy, and if CIS was confirmed, observed for varying lengths of time without adequate treatment—and as we know, with disastrous consequences.1–4


The object of the study was to prove Dr Green’s belief that “CIS is not a premalignant disease” and “the patient with in-situ cancer has only the normal chance of developing future invasive cancer”.2,5

Conventional treatment—Professor Chalmers challenges us to define “conventional management” of CIS.6 Like Chalmers, I too was a resident in UK teaching and provincial hospitals in the late 1960s and early 1970s. There was no colposcopy. In my experience the standard management of women with significant cytology abnormalities was application of Lugol’s iodine stain, “blind” conisation or hysterectomy (which served both as diagnosis and treatment). Wedge biopsy was performed to exclude invasion. I support his views that there were (and still are) marked differences in the types of treatments of CIS worldwide. But I know of nowhere else where women with CIS were observed for long periods without definitive treatment.

With hindsight it is clear the NWH was a leader in the “conservative” (in this sense non-hysterectomy) approach to the treatment of CIS of the cervix. In 1958 the official NWH policy was that women with CIS should be treated with “adequate cone biopsy … providing the immediate follow up is negative and … the pathologist is satisfied that the cone biopsy has included all of the carcinomatous material”.2 Dr Green concurred with this management at this time.7

Green’s 1968 Lecture Notes to postgraduate doctors define “conventional” treatment.7 They state “Conventional treatment comprises: (a) Cone biopsy excision. This is much more commonly advocated and practised now than 5 years ago. A certain proportion continue to have doubtful or positive smears in the follow-up period … , and require further biopsy excision or even hysterectomy (b) Hysterectomy … ”

Green’s experiment in which treatment was withheld from women with CIS began in 1965 (although he only gained approval the following year). He paraphrased the study, “in 1965 the senior medical staff of the NWH initiated a project* under the supervision of the author for patients up to 35 years of age whose only abnormal
finding was positive cervical cytology. Where such a patient had no clinical, cytologic or colposcopic evidence of invasive cancer a histologic diagnosis was to be established by a punch biopsy of the most significant area of the cervix. Providing the biopsy did not remove the entire significant area, or reveal invasive carcinoma, there was to be no further treatment”.(sic)

("Project: ‘A piece of work that is carefully planned to achieve a particular aim.’ Concise Oxford Dictionary, 2006.")

These statements by Green are clear evidence of two separate and simultaneous policies.

- A recommendation for conisation or hysterectomy as “conventional” treatment.
- An experiment entailing withholding treatment for CIS (i.e. observing CIS).

Green also stated that if CIS is “removed” there is “an almost 100% chance of permanent cure”.

Doctor’s dilemma—Phillida Bunkle is correct, “the doctor’s dilemma” was caused by more than 20 years … of delays in treatments. There were no simple answers. There were. I have managed cases of multicentric lower genital tract neoplasia at NWH for 38 years and the only ones to create a dilemma were the few I inherited from Dr Green.

Multicentric lower genital tract neoplasia is uncommon but not rare. The principles of management are those of the individual tumour – each needs to be adequately treated, on its own merits, and at the earliest opportunity. With time these lesions progressively extend to involve increasingly larger areas of tissue and eventually progress to invasive cancer.

I remember the case (60/64) in question. It illustrates the natural history of untreated vaginal and vulval CIS. In 1960 the woman had a cone biopsy followed by hysterectomy with complete excision of the lesion. (Green was treating CIS at that time.) The woman had normal smears until 1964 (not positive smears as Bryder states). Biopsy of a visible vaginal lesion in 1965 revealed CIS (by this time Green was observing CIS without treatment). The woman was followed with regular (abnormal) smears and biopsies reconfirming vaginal CIS for the next 12 years when invasion occurred. In 1971 a separate vulval CIS lesion developed. This was biopsied and observed until 1977 when she underwent vulvovaginectomy. Positive margins for CIS were noted at the external urethra. This lesion progressed to invasion and the patient died from this cancer in 1981. Jordan’s dilemma was how to comment on a case that had been neglected for so many years.

Note: Photographs of a patient of Dr Green with untreated CIS progressing to cancer can be seen in Singer and Monaghan’s book Lower Genital Tract Precancer (Blackwell Scientific Publications).

Professor Ronald W Jones
Clinical Professor of Obstetrics and Gynaecology, National Women’s Hospital
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References:


Response to Elizabeth Overton’s comments about Ron Jones’ research

The treatment of women with cervical intraepithelial neoplasia 3 (CIN3) at National Women’s Hospital (NWH) in the years 1955-76 was subject to two quite separate analyses, the first published in 1984 by McIndoe et al,1 the second published in 2008 and 2010 by ourselves.2,3 Professor Ron Jones was an author of all three papers. Medical records of all women diagnosed with CIN3 at NWH in those years (not just those under Dr Green’s care) were the source of data for both analyses but as the inclusion and exclusion criteria were not quite the same, there were small differences in the final numbers. These differences are not important when considering the overall conclusions.

In the 1984 and 2010 papers, a principal outcome of interest was the difference in the risk of invasive cancer between two groups of women. In the 1984 paper the groups were defined according to whether or not women had persisting CIN3 on the basis of positive cytology two years after first diagnosis (irrespective of their treatment up to that time) whereas in the 2010 paper they were defined according to whether women, first diagnosed in the 1965-74 period, had treatment of curative intent within 6 months of first diagnosis (irrespective of their follow-up cytology). Thus the 1984 paper identified the risk resulting from failure to eradicate CIN3 within two years of diagnosis, while the 2010 paper identified the risk due to withholding or delaying treatment of curative intent.

Mrs Overton’s letter4 claims to identify a major discrepancy between the 1984 and the 2010 papers in the numbers with respect to their initial treatment. She continues the line of reasoning used by her husband Dr Graeme Overton,5 and adopted by Ms Sandercock and Dr Burls,6 in earlier letters to NZMJ. The flaw in Dr Overton’s reasoning was identified and explained in a previous letter,7 and is given again below.

Mrs Overton writes: ‘The 1984 McIndoe Paper states that of the 948 women with grade 3 cervical dysplasia reviewed, 1955–76, 923 had principal initial treatment of hysterectomy or cone excision of cervix and 25 punch or wedge biopsies.’

- Her reference (#2) identifies four places in the 1984 paper to support this statement. These are, each with the actual wording used in the paper for what she has represented as ‘principal initial treatment’:
  - Page 452, Table 1: ‘Definitive management …’
  - Page 453, para 6 and page 454, para 4: In both cases the words in the text are ‘The principal management…’
  - Page 455, para 2: This paragraph is concerned with how a final diagnosis was reached in women, with the word ‘initial’ used to describe the type of biopsy on which the earlier diagnosis had been based, the wording being “… after the initial biopsy diagnosis of CIS, by …’ (with the various types of initial biopsy being specified).
The 1984 paper does not define ‘definitive’ or ‘principal’ management but it would appear that these terms were interchangeable. It does not use either together with the word ‘initial’.

The term ‘initial treatment’ was used only once in the 1984 paper, when referring to 29 group 2 women who developed invasive carcinoma and whose detailed management was depicted in Figures 2 and 3. Of these women 14 were said to have had ‘initial treatment’ by cone biopsy and 6 by hysterectomy. However, of these 14 so-called ‘initial’ cone biopsies, 4 had been delayed 2 to 8 years after first diagnosis of CIN3.7

Table 4 of the 1984 paper, headed ‘Detailed patient management’, gave treatment information separately for women in group 1 and group 2. Women who received either cone biopsy or total hysterectomy as ‘later’ treatment were identified but ‘later’ was not defined. Of the women in group 1, 78% (n=637) had hysterectomy, amputation of the cervix or cone biopsy as their first treatment, 20% (n=165) as later treatment and 2% (n=15) had no more than a punch or wedge biopsy. By contrast, the respective numbers for women in group 2 were 49% (n=64), 44% (n=57) and 8% (n=10).

Hence, both Dr and Mrs Overton have wrongly used the term ‘principal initial treatment’ to include management that was not necessarily ‘initial’ (by the definition used in the 2010 paper) and, at least in some cases, had been delayed for some years after the original diagnosis.

Another less important difference between the numbers in the two papers is that the 1984 paper included ring biopsy (a shallow cone biopsy) with cone biopsy whereas it was separately categorised in the 2010 paper, as Dr Green had described it as not being definitive treatment.8 That this distinction was justified is clear from Table 3b and Figure 2b of the 2010 paper, which show that the risk of invasive cancer following initial management by ring biopsy was intermediate between that following cone biopsy on the one hand and punch or wedge biopsy on the other.

Mrs Overton states that the 2010 paper ‘claims to be the ‘final word’ to silence all critics of the 1984 McIndoe Paper’.

The inverted commas imply that this is a quotation from the paper. Nowhere in the paper is such a claim made; indeed, it makes no reference to ‘critics of the 1984 McIndoe paper’.

She asserts that the 2010 paper states ‘of the 948 women reviewed by McIndoe in the years 1955–76, 428 women, in the years 1965–74, had initial management in which treatment of ‘curative intent’ was deliberately withheld in unethical experiments.’

There is no such statement.

Of the 1063 women diagnosed with CIN3 at NWH in 1955-76, 422 (not 428 – probably a typographical error) were newly diagnosed in 1965-74.3 These 422 women comprised the group in whom we compared the outcomes according to their initial management (that is, within 6 months of CIN3 diagnosis) – 215 received treatment of curative intent (cone biopsy, amputation of cervix or hysterectomy), 72 a ring biopsy, 127 no more than a punch or wedge biopsy and 8 a biopsy of unknown type (Tables 2b & 3b). Thus, it was in only some of the 422 women that treatment of curative intent was deliberately withheld.
Certainly, the number of women (127) who had initial management of punch or wedge biopsy in the 2010 paper is higher than the number (25) whose definitive management was described as punch and/or wedge biopsy in the 1984 paper. However, while the 25 women had never had a procedure that was more extensive than a punch or wedge biopsy, the 127 women had no more than a punch or wedge biopsy within 6 months of their diagnosis but may have had a more extensive procedure subsequently.

Many of the 222 later treatments included under the heading of ‘definitive management’ in the 1984 paper would not have been counted as ‘initial management’ in the 2010 paper.

Mrs Overton writes: ‘Simple arithmetic confirms that it is not possible to have 422 women with treatment of “curative intent withheld” in 948 women which the 1984 McIndoe Paper states 923 had initial treatments of cone excision or hysterectomy, i.e. on their own 2010 definition ‘treatment of curative intent’

As shown above, this is based on the incorrect assumption that definitions in the 2010 paper applied also to the 1984 paper.

- ‘Definitive’ or principal’ management (1984) was not the same as ‘initial’ management (2010).
- McIndoe et al included ring biopsy with cone biopsy (1984), while ring biopsy was categorized separately in the 2010 paper.
- Procedures performed more than 6 months after the original diagnosis were taken into account in the 1984, but not in the 2010, paper.

She concludes that ‘the 2010 statistics are damaging fiction in my opinion.’

As we have shown, she has misunderstood the published information on which her letter is said to be based. These derogatory remarks about the 2010 paper are without foundation.

Our 2010 paper showed that the group of women with CIN3 not treated promptly with curative intent had a substantially increased risk of invasive cancer even though some subsequently received such treatment.

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References:


The Cartwright Legacy

Ron Paterson in an editorial on the Cartwright Legacy has perpetuated the myth that the Inquiry had revolutionized medical ethics and that after the Inquiry the patient had been put first and not the doctor. He states that there has been a “seismic” shift in relationships between doctors and their patients. This implied that there had been a general arrogant disregard of patients by the profession.

I practiced surgery in Auckland throughout the period of the Inquiry and I can assure Mr Paterson that I was unaware of any behavioural change in the management of patients before during or after the Inquiry. My observation of colleagues both within and outside the hospitals was that strict ethical standards were adhered to and that the patients’ health problems were paramount. Also patients had always been consented for procedures done where appropriate.

Since the Inquiry the risk factors have been amplified such that now patients are told that when a general anaesthesia is administered there is a possibility that they might die. Previously this would not have been a routine and the consent would be tailored according to the anticipated risk. One reason was to avoid unduly frightening the patient where the risk was extremely small. What’s new are the many wall instructions in hospitals pointing out patient’s rights.

Recently Linda Bryder, a medical historian, in her book, has brought some balance to the Cartwright Inquiry and the aftermath. She has clearly pointed out the central place the ideology of the feminist movement played in the drama that unfolded. Also the difficulty the Court had in understanding the science of in situ cervical carcinoma. The aftermath had the detrimental affect on the management of childbirth with the rapid rise in the power of midwives and the decline in GP involvement in obstetrics.

The recent death of Mr Bill Faris caused Mr Tony Baird to describe his outstanding lifetime service in the field of Obstetrics and Gynaecology; yet to have inappropriately suffered the indignity of being found guilty of “conduct unbecoming a medical practitioner” by being implicated as a member of the subcommittee that was established in 1975 at National Women’s Hospital to review the care of women with cervical dysplasia. Along with other medical staff he had no recourse to defend himself of this charge.

Lawrence Smith
Parnell, Auckland

Reference:

Malcolm Ross (Barney) Mowat

Dr Malcolm Ross Mowat (MB ChB, Dip Obst) died of a major stroke, a complication of cardiovascular disease, in the Greymouth Hospital on 5 June 2010.

Born into a farming family on 15 December 1933, Barney, as he was always known, went to school in Fairton, just north of Ashburton, and then he boarded at Timaru Boys’ High School, whose Old Boys, he argued, were just as good as those from Christ’s College, but they didn’t wear moleskins.

Barney’s father was a soldier in the Great War, who farmed successfully for many years in spite of losing an arm during action against the enemy. Barney’s teachers considered the University Entrance examinations would be too hard for him, so he went back to the farm, having passed School Certificate.

Rapidly tiring of driving a tractor, he went back to school at Ashburton High and it was there that he got his University Entrance accredited. At Canterbury University College in 1953, he successfully completed an intermediate year (with a very good mark in Botany) and that gave him the options of Agriculture, Veterinary work, Dentistry, or Medicine. Having decided (on the basis of some party-going) that doctors had the most fun, Barney went to Dunedin and entered the Medical School. He graduated in 1959.

Barney was a House Surgeon at Nelson Hospital in 1960 and 1961. He briefly worked as a Registrar in Radiology at Queen Margaret Hospital, but he went on to take a job as House Officer at the Christchurch Women’s Hospital (which was formerly known as St Helen’s) for 6 months in the course of 1962. He then joined the late Kevin O’Connor in general practice on Waimairi Road where he especially enjoyed the maternity work. About 1976 he joined the Student Health Service at the University of Canterbury.

In 1988, Barney was appointed as the Special Area Doctor at Whataroa, in South Westland. He remained in this remote coastal area as the solo practitioner, serving patients scattered from Hokitika in the north to the Haast River in the south, for over 10 years. Search and rescue work was a regular part of the practice. Together with his soul mate, Juliet, he shared an enormous love of the wilderness, painting, music and family during this time of geographical isolation.

When they moved to Hokitika, Barney continued to do occasional locums around the South Island until his health deteriorated. Late in 2009, he was hospitalised in Christchurch with Legionnaire’s disease.

Barney was an outdoors man, who spent time in Antarctica teaching snow craft. Roger Ridley-Smith writes, “It was entirely thanks to Barney that at various times I climbed Mount Rolleston, crossed the Crow Glacier, tramped the Routeburn Track and the Greenstone Valley (with one of my daughters, and Barney’s young son), and skied the length of the Tasman Glacier on one of the sublimest days of my life. I am by nature sedentary, and I was grateful to Barney for getting me going.”

He was involved in Mount Cheeseman ski field, where the growing family spent many weekends, either up on the mountain, or down by the skating rink. Barney was passionate about winter sports and encouraged his sons, Guy and Hamish, onto the ski racing circuit. Bag-piping was another great passion of Barney’s. In his earlier days he was a member of the Ashburton Pipe Band and more recently he played at many functions including Anzac commemorations. Having lost an 18-year-old brother in World War II, Anzac Day had personal significance. He was made a life member of the Hari Hari RSA. When at home, Barney celebrated life surrounded by pet animals and a flourishing vegetable garden.

An alcoholic whilst still at Medical School, Barney Mowat, when he was working as a House Surgeon, not infrequently began the day by swallowing a bottle of cough mixture, with its high alcohol content. The disease of alcoholism, and its associated craving for a variety of prescription drugs, both “uppers” and “downers”, eventually took over his life, and if he had not got help, he would have probably died.

During the 1970s, he was, as his daughter Shellie relates “in and out of mental hospitals”, and he had numerous ECT shock treatments because he was not ready to acknowledge his problem. A friend and colleague, Ian Fulton, recalls, “one of the most tragic memories I have of Barney was taking Hamish and Anna (my daughter) to visit him when he was hospitalised in Auckland and undergoing treatment for alcoholism whilst incarcerated in a six-bed ‘ward’ of fellow-travellers, his bed being an old-type army stretcher with a straw palliasse as a mattress.”

Shellie recalls that her father was eventually rescued by the veteran Alcoholics Anonymous worker, Trevor Grice, and with Grice’s help he finally addressed his drug and alcohol addictions. He went to Hanmer Hospital in 1975 as a very sick man, and there he began his recovery journey.

From then on he worked with enormous zeal for Alcoholics Anonymous until his death, and the help and support that he provided for others in distress is beyond calculation. This included helping Kiwis, and Americans, to face their addictions through his Antarctica connections. He was instrumental in setting up Dry Dock at the Christchurch Deep Freeze Base. He also paved the way for many in the medical profession to seek help. Knowing well the diabolical lure of alcohol for so many people, he adhered to complete sobriety for 35 years, attending several Alcoholics Anonymous meetings every week.
John Kent, another medical colleague, writes, “Barney was one of my oldest and dearest friends, certainly the most eccentric and with a wonderful sense of humour… I will cherish many fishing and hunting memories we shared over the years.”

On the West Coast, Barney acquired huge popularity (in 1999 he was awarded South Westland Person of the Year). About 400 people attended his funeral in Greymouth on 12 June 2010. The congregation represented his wide circle of friends, medical colleagues, patients, members of Alcoholics Anonymous, and many of the mates with whom he went hunting, shooting and fishing. After the service he was buried in the Hokitika Lawn Cemetery.

At the funeral, tributes were delivered by Hugh Bodle, Peter Adams, Gerry Hack, Paddy Kennedy and on behalf of Trevor Grice. Anyone who wishes to read these accounts of a remarkable life should send their name and email address to r.sdekkka@actrix.gen.nz

Barney is survived by his wife Juliet; his first wife Joey and three of their children (Shellie, Nicola and Hamish); and four of his five grandchildren.

Barney’s long-time friend Roger Ridley-Smith compiled this obituary, with assistance from his family.
National Heart Foundation: 2011 Grant Applications

((Libraries, print from the link above then replace this page))
Māori Cardiovascular Research Fellowship

This Fellowship is designed to support graduates who propose to engage in research to improve cardiovascular health in Māori.

The Māori Cardiovascular Research Fellowship is available for a medical graduate or non-medical graduate enrolled for a higher degree.

Preference will be given to applicants with a working knowledge of “Kaupapa Māori” and who are committed to Māori health. This fellowship is tenable in New Zealand for up to 3 years.

Application forms for the Māori Cardiovascular Research Fellowship are available on our website [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz) or from

Helen Stewart
Heart Foundation
PO Box 17160
Greenlane
AUCKLAND 1546

Ph: (09) 571 9191
Fax: (09) 571 9190

Email: HelenS@heartfoundation.org.nz

Applications close 1 June 2011
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
Complementary Therapies for Cancer: what works, what doesn't—and how to tell the difference


This book provides an evidence-based overview of the use of complementary therapies for cancer. It has been written for anyone with cancer and for health care professionals who care for such patients.

In some countries such as the United States, there are more visits to complementary and alternative medicine (CAM) practitioners than there are to primary health care doctors. Whether we like it or not, our patients are using CAM and other natural therapies, and health care practitioners need to understand the basic principles of these therapies and know which have a solid scientific basis and can be recommended.

This is particularly the case for patients with cancer, half of whom will use CAM therapies and almost all of the remainder will look at using them and seek information, often from their doctor.

With respect to health care professionals, the utility of the book is neatly captured in the powerful forward written by Dr Belinda Scott who says....”no patient should waste their valuable energy, time or money on treatments that have not been scientifically proven…it matters to me that Shaun referred to sound scientific studies when recommending or dismissing a therapy.”

In addition, the book is endorsed by the world’s leading expert on CAM therapies and the co-author of Trick or Treatment, Professor Edzard Ernst, who says in the cover notes…”this book is a much-needed assistance for vulnerable and often desperate people. It should be made available for all cancer patients who feel tempted to try some form of complementary medicine.”

The introductory chapters provide an overview of the use of CAM by people with cancer. Remarkably, a quarter of those who use CAM will use 7 or more therapies. On reflection this makes sense, given the attitude of many patients who are faced with a potentially fatal diagnosis and are determined to leave “no stone unturned” in their quest to maximise their chances of survival and maintain as high a quality of life as is possible. It is emphasised that none of the therapies discussed will cure cancer, but many can be effective in reducing symptoms and/or increasing quality of life.

Around 40 of the commonest CAM therapies that are used by people with cancer are discussed, with around half being recommended and the remainder advised against. For each therapy, the history of the treatment and the practicalities of receiving it are discussed, along with a summary of the key research findings and a graded recommendation from “highly recommended” to “definitely avoid.”
Some of the research findings are surprisingly robust, for example, a study of over 600 participants with chemotherapy-induced nausea not only demonstrated clear benefits of ginger but also determined a dose-response relationship.

There are numerous fascinating facts; for example, shark cartilage is promoted as a cancer cure on the basis that sharks do not get cancer, whereas the reality is that over 40 types of cancer have been described in sharks.

*Complementary Therapies for Cancer* is an educational and enjoyable read and is highly recommended to all health care professionals who care for patients with cancer.

Richard Beasley
Professor
Medical Research Institute of New Zealand
Wellington
Exploring the Successes of Natural Health Care


Some natural and complementary therapies are safe and effective, but most are not. The area is rife with pseudoscience, anti-science, testimonials and conspiracy theories and all these traits are well-represented in this book.

The book is based on the false premise that anecdotes are an important source of information in helping us decide if a treatment is effective or not. Anecdotes and testimonials are presented in this book to support therapies. These therapies, including colonic irrigation, reflexology, reiki, homeopathy and kinesiology, are not supported by scientific research and are mostly biologically implausible. The author, an engineer by training, is probably a well-meaning person who thinks that he is helping to disseminate important health information that doctors can or will not give to their patients.

But the information he presents is incorrect and this sort of book, which is worryingly prevalent in the new-age section of book shops, has the potential to cause harm in a number of ways. As well as promoting therapies that can directly cause physical harm, such as a ruptured bowel from colonic irrigation, the therapies promoted can lead to a vulnerable sick person having false hope, delaying proven treatments or wasting precious time and money.

A disappointing and harmful consequence of the sort of nonsense illustrated throughout this book is that many health care professionals are put off natural and complementary therapies altogether, and their patients are not told about therapies that have been shown to be safe and effective in well-conducted clinical trials, such as omega-3 fish oil, St John’s Wort, massage therapy, yoga and many others.

As a believer in free speech and the rights of others to express their opinions, no matter how ridiculous they are, I do not advocate that this book should be banned…although it is tempting.

Professor Shaun Holt
Tauranga
Ethical Issues in Governing Biobanks: Global Perspectives


Storing human tissues to support research has been an important recent advance. This book brings together international views about three aspects of the ethics of storing tissues in biobanks. The 4 editors have involved 5 other experts in writing the 16 chapters.

After an introductory chapter, the first of the 3 parts is a literature review of biobanks and current guidelines, and highlighted 3 unresolved controversies: consent, confidentiality and commercialization. Part 2 describes a qualitative study carried out from 2004 to 2006 which sampled 87 international experts, half from the US, drawn from physicians, researchers, ethicists, lawyers and philosophers. The results are presented as 10 chapters under headings such as “consent issues”, “collective consent”, “anonymization and coding”, each with an introduction and discussion of how the data contributes to current knowledge, and a bibliography.

Part 3 reports a meeting of 18 international scholars, which addressed ownership of samples, intellectual property rights, feedback to participants, disposal, and benefit sharing. The final chapter looks to the future, suggesting that biobanking is becoming normalised with a sample seen to be just another biological sample, and not “the key to every person's essence”, and more widely seen to be a public good. They support the development of common international standards.

A theme throughout the book is the dynamic and ever-changing nature of attitudes and requirements for banking, with this leading to ongoing improvement. Anyone who uses or collects human tissue for research would find this book a useful resource. However, the views reported were from 5 years ago, and there is very little about ethnicity issues, although two New Zealanders were listed as being interviewed in the study, where arguably New Zealand has led the world.

Bridget Robinson
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