CONTENTS

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

Editorial
Gambling and health: uncomfortable bedfellows
Max Abbott

Original Articles
Problem gambling risk factors and associated behaviours and health status: results from the 2002/03 New Zealand Health Survey
Kylie Mason, Richard Arnold

Problem gambling: patients affected by their own or another’s gambling may approve of help from general practitioners
Sean Sullivan, Ross McCormick, Michael Lamont, Alison Penfold

Comparison of plasma adiponectin levels in New Zealand Māori and Caucasian individuals
Brett Shand, Peter Elder, Russell Scott, Nicola Poa, Christopher M Frampton

Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance and impaired fasting glucose. Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand
Gerhard Sundborn, Patricia Metcalf, Robert Scragg, David Schaaf, Lorna Dyall, Dudley Gentles, Peter Black, Rodney Jackson

Alcohol assessment: the practice, knowledge, and attitudes of staff working in the general medical wards of a large metropolitan hospital
Justin Pulford, Ross McCormick, Amanda Wheeler, Patrick Firkin, Ian Scott, Gail Robinson

Case Reports
An international surgical collaboration for the management of pulmonary artery sarcoma: a New Zealand experience
Yaso Kathiravel, David Westwood, Jeff Macemon, Harsh Singh

Hepatic portal venous gas in a patient with pneumatosis intestinalis
Chih-Hao Shen, Heng-Cheng Chu, Wei-Kuo Chang, You-Chen Chao, Tsai-Yuan Hsieh

100 Years Ago in the NZMJ
Medical Benevolent Fund and the Defence Union
Proceedings

Medical Image
A vascular phenomenon
Utpal Nandy, George I Varughese, Yelin L Hock

Methuselah
Selected excerpts from Methuselah

Letters
ACC on the back pain article by Crawford et al—and Response
Kimberly-Anne Ford

Medical students denied access to normal vaginal deliveries
Name and affiliation withheld upon request

Acknowledging best practice in medical education
Andrew Carson-Stevens, Iain J Robbé

Systemic health system problems
John Cook

Metoprolol-induced hyperkalaemia in chronic respiratory acidosis
Andrei M Beliaev, Warren Smith

Lessons from Hong Kong and other countries for outdoor smokefree areas in New Zealand?
Nick Wilson, George Thomson, Richard Edwards

Reply to the editorial “World No Tobacco Day (31 May 2007)—did anybody notice?”
Judy Li, Michele Grigg

Obituary
Diana Manby Mason
Alan Guibal Bradford

Notice
Medical Benevolent Fund

Erratum
Arthur Leslie Batt [obituary]
NZMJ
Problem gambling risk factors and associated behaviours and health status: results from the 2002/03 New Zealand Health Survey

Kylie Mason, Richard Arnold

This study used the results of the 2002/03 New Zealand Health Survey to investigate problem gambling in New Zealand adults. The results of this study show that approximately 1.2% of New Zealand adults aged 15 years and over were problem gamblers in 2002/03. Māori and Pacific (mostly of Samoan, Tongan, Niuean, or Cook Islands origin) peoples were significantly more likely to be problem gamblers than other people. Key risk factors for problem gambling were being of Māori or Pacific ethnicity, being aged 25–34 years, living alone, being employed, and being less qualified. Compared to other people, problem gamblers were significantly more likely to be daily cigarette smokers, and to have potentially hazardous drinking behaviour (an established pattern of drinking that has a high risk of causing physical or mental health problems in future, but has not yet done so). Problem gamblers were also more likely to have worse self-rated health (based on their own perception of their health) compared to other people.

Problem gambling: patients affected by their own or another’s gambling may approve of help from general practitioners

Sean Sullivan, Ross McCormick, Michael Lamont, Alison Penfold

1580 patients (attending their GPs in Auckland, Taranaki, and Rotorua practices for non-gambling reasons) completed questionnaires to identify if they were affected by their own, or a family member’s gambling, and whether they saw their GP as suitable to help them for these gambling problems. A minority of problem gamblers or affected family members viewed their GPs as unsuitable, raising the possibility of a large alternative resource to specialist gambling treatment services for these people. Both problem gamblers and their affected families were found to be more depressed than other patients, however no other health issue that patients attended their GP for was found to be an indicator to check for problem gambling. Patients appeared willing to disclose their gambling problems when attending their GP, and although a relatively small proportion of family members affected by gambling access specialist treatment services, there appears to be a substantially higher proportion of family members who may see their GP as a source of help for their gambling issues.
Comparison of plasma adiponectin levels in New Zealand Māori and Caucasian individuals
Brett Shand, Peter Elder, Russell Scott, Nicola Poa, Christopher M Frampton

Adiponectin is a hormone made by fat cells that improves insulin function. Adiponectin levels in the blood are determined by both the genetic makeup and body size of an individual. Low adiponectin levels increase the risk of a person developing Type 2 diabetes. This study compared adiponectin levels in the blood of Māori and Caucasian people—matched for age, gender, and body size and shape. The study found the Māori people had marginally lower adiponectin levels than their Caucasian counterparts. It is possible these lower adiponectin levels may be a contributing factor to the higher incidence of type 2 diabetes found in the Māori population.

Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance and impaired fasting glucose. Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand
Gerhard Sundborn, Patricia Metcalf, Robert Scragg, David Schaaf, Lorna Dyall, Dudley Gentles, Peter Black, Rodney Jackson

This article describes the prevalence of different diabetes states amongst European, Māori, and Pacific people. It outlines burden of disease of diabetes describing the expected proportion of undiagnosed diabetes by ethnic group. It was also found that Body Mass Index (BMI) eliminates ethnic differences that were found for newly diagnosed diabetes.

Alcohol assessment: the practice, knowledge, and attitudes of staff working in the general medical wards of a large metropolitan hospital
Justin Pulford, Ross McCormick, Amanda Wheeler, Patrick Firkin, Ian Scott, Gail Robinson

Many hospital admissions are alcohol-related. The hospital is also an ideal venue in which to provide brief alcohol interventions to risky drinkers. However, identifying which patients are suited to a brief alcohol intervention, or the degree to which excessive alcohol consumption may be involved in a patient’s presenting condition, requires an accurate alcohol assessment. This study therefore sought to examine the accuracy and clinical utility of standard alcohol assessment practice in a New Zealand hospital. The findings suggested that hospital staff reliably asked patients about their alcohol use, but were not supported or skilled enough to do so in a way that allowed patient alcohol consumption to be accurately determined.
Gambling and health: uncomfortable bedfellows

Max W Abbott

Gambling has been a leading growth industry for 20 years, particularly in countries such as New Zealand where electronic gaming machines (EGMs) and urban casinos were widely introduced.1 Approximately NZ$2 billion was spent (lost) in New Zealand on major forms of gambling last year—$5.5 million per day.

Like alcohol, gambling is Janus-faced. Among other things it deals entertainment, pleasure, companionship, distraction, and dreams with one hand. And it dispenses financial ruin and a trail of personal, family, and social devastation with the other.

No comprehensive cost-benefit analysis of gambling has been undertaken in New Zealand. Studies elsewhere weight health impacts (on the cost side of the equation) heavily, particularly those related to problem gambling. Like gambling, problem gambling has a long pedigree. It is graphically portrayed in novels by Dickens and Doestevsky. However, it was not until 1980 that ‘pathological gambling’ entered the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-III).

Although having features in common with substance dependence and misuse, pathological gambling is classified as a disorder of impulse control.

Essential characteristics of pathological gambling include:

- A continuous or periodic loss of control over gambling.
- A progression, in gambling frequency and amounts wagered, in the preoccuption with gambling and in obtaining moneys with which to gamble.
- Continuation of gambling involvement despite adverse consequences.

Adverse consequences occur in many spheres—mental and physical health, family and interpersonal relationships, work, education, and recreational pursuits. Because money is the primary currency of this ‘addiction’, financial hardship and ruin are commonplace, as is criminal activity to finance gambling and gambling debt. In New Zealand prison surveys, 15% of males and 26% of females report offences of this type.2,3

The first (1991) New Zealand gambling survey estimated that 0.9% to 1.6% of New Zealand adults currently experienced serious gambling problems (probable pathological gamblers).4 A similar number had less serious problems. Males, young adults, and marginalised groups (Maori, Pacific, unemployed, low income) had particularly high prevalence rates. Few reported seeking help. Those who did turned to friends, family members, and Gamblers Anonymous.

A second national survey was conducted in 1999.5 Since 1991, casinos had been opened in Christchurch and Auckland, EGM numbers increased markedly, and total gambling expenditure doubled. A national gambling helpline was established (1993) and counselling services developed (from 1994) in major centres.
Contrary to expectation, the 1999 study did not find a prevalence increase. While Māori and Pacific people remained at high risk, the profile of problem gamblers had changed.

In the investigators’ words, problem gambling “had aged, feminised, and gone a bit upmarket.” A substantial increase was found in help-seeking. Informal sources remained important, but almost as many consulted specialist gambling services, health professionals, and alcohol and drug services. Nevertheless, most did not seek help of this type, including those with serious problems.

Since 1999, four more casinos have been established, EGM numbers further increased and additional forms including Internet gambling introduced or expanded. Total gambling expenditure has again doubled. What is the impact of this on problem gambling and related gambling harms?

Before returning to this question, it is worth noting that New Zealand was the first country to conduct a national problem gambling survey and develop nationwide problem gambling services. It has retained its pioneering role with the Gambling Act 2003. This far-reaching legislation arose out of concern about loose control in the gambling environment and gambling-related harms. It places gambling within a public health framework and seeks to contain growth and minimise harm. Implementation is the joint responsibility of the Department of Internal Affairs (regulation) and the Ministry of Health (prevention, treatment, research, and evaluation).

From 1999 to 2003, strong growth continued in specialist gambling service consultations. However, in 2005, new helpline callers reduced by a third; and new counselling clients by a fifth. Reasons are not established, but the Gambling Act 2003 and Smokefree Environments Act 2004 are probably implicated. Gambling expenditure, particularly on non-casino EGMs, also decreased for the first time. Similar changes occurred in Victoria, Australia following the introduction of a smoking ban and some other regulatory measures.\(^6\)

The Ministry of Health reports:

…It remains to be seen, however, whether the decreases are the start of a new trend, a transient period of consumer and industry adaptation, or simply an outlier in the established increasing trend… (p4)\(^7\)

Although cautionary caveats are in order, this rapid decline in help-seeking occurred while specialist services were being further expanded and promoted. Change of this magnitude is without precedent in the addiction or mental health fields. Certainly it would be a novelty if we were considering hip replacements! It would be helpful to know what is going on.

While expenditure and help seeking provide some information, their relationships with problem gambling are complex. Unfortunately there has been no national prevalence survey since 1999, although one is planned. This should help clarify whether problem levels have changed during the past decade.

Two articles in this issue of the Journal make important contributions to our understanding of problem gambling and underline its significance for GPs and other health professionals.
Mason and Arnold analyse gambling data from the 2002/03 New Zealand Health Survey. Half the problem gamblers smoked daily and two-thirds reported increasing smoking while gambling—a possible explanation for the impact of smoking bans in EGM and other gambling venues?

More than half of the gamblers studied engaged in hazardous alcohol consumption. Problem gamblers also reported worse health across a range of domains. Risk factors were similar to 1999, confirming the changes since 1991. Regrettably the study used a non-standard problem gambling measure that precluded comparison with the 1991 and 1999 surveys.

Sullivan and colleagues surveyed patients from 16 GP practices and found 7.5% experienced gambling problems. About twice as many said they were affected by another person’s gambling. Both groups had significantly elevated depressive symptoms. Māori, Pacific, Chinese, and ‘other ethnicity’ patients were at high risk. This implies previous methodologies might not have been sufficiently sensitive to problems among some Asian and recent migrant groups. There was a high level of participation in the study and apparent acceptance of GPs as a potential source of help. This suggests that primary health providers could effectively offer screening, referral, and a range of interventions to assist the majority of problem gamblers and concerned others who do not currently access specialist services.

The two studies, while showing robust associations with a range of health measures, do not indicate temporal sequence or causation, for example whether depression contributes to or results from gambling problems. For screening purposes this does not matter. However, advancing scientific understanding and informing public health and clinical interventions requires more sophisticated examination of these connections. Prospective studies are particularly important in this regard and, while few, are starting to help untangle the complex web of relationships between the agent gambling, other environmental factors, and individual psychology and biology.

There are indications that progress is being made with the Gambling Act 2003 and related initiatives to reduce gambling-related harm. However, agent, environment, and ‘host’, like rust, never sleep. And not only problem gamblers are addicted to gambling—so too are governments and communities that receive significant gambling revenue. The true measure of public health resolve comes when it is sustained in the face of reduced rents (taxes, levies and grants) to the beneficiary.

Competing interests: None.

Author information: Max W Abbott, Pro Vice-Chancellor, Dean, Professor, and Director, Gambling Research Centre, Faculty of Health and Environmental Sciences, Auckland University of Technology (AUT), Auckland

Correspondence: Professor Max Abbott, Pro Vice-Chancellor and Dean, Faculty of Health and Environmental Sciences, Auckland University of Technology, North Shore Campus, Private Bag 92006, Northcote, Auckland 1020. Fax: (09) 921 9706; email: max.abbott@aut.ac.nz

References:


Problem gambling risk factors and associated behaviours and health status: results from the 2002/03 New Zealand Health Survey

Kylie Mason, Richard Arnold

Abstract

Aims To investigate the extent of current problem gambling in New Zealand, and the risk factors, addictive behaviours, and self-rated health status associated with problem gambling.

Methods Analysis of the gambling questions from the 2002/03 New Zealand Health Survey, which interviewed 12,529 people aged 15 years and over, and included increased sampling of Māori, Pacific, and Asian people.

Results Approximately 1.2% (95% confidence interval: 1.0–1.5) of the New Zealand adult population were found to be current problem gamblers, representing an estimated 32,800 (26,200–39,400) people. Risk factors for problem gambling included being of Māori or Pacific ethnicity, being aged 25–34 years, living alone, being employed, and being less qualified. Problem gambling was significantly associated with potentially hazardous drinking behaviour, daily cigarette smoking, and worse self-rated health, as measured on several SF-36 health domains.

Conclusions Māori and Pacific peoples were at significantly greater risk of being problem gamblers than other people, particularly among those people who gambled. Associations between gambling problems and health problems and/or risk behaviours suggest compounded problems from comorbidity. This evidence may be useful in informing policy and public health programmes to reduce the harmful impact of problem gambling on individuals and communities, and in addressing the inequalities evident in gambling-related harm.

With the increasingly wide availability of gambling opportunities in New Zealand, the issue of problem gambling has become a more immediate social and public health concern. Problem gambling is characterised by a loss of control over one’s gambling, and can cause significant problems for the gambler and those around them. In New Zealand, problem gambling is formally recognised as a public health issue, and the Ministry of Health has responsibilities for the prevention and minimisation of gambling-related harm under the Gambling Act 2003.1

The prevalence of problem gambling in the population is measured with a population survey, generally using a set of questions about gambling (‘gambling screen’). Surveys investigating problem gambling were carried out in New Zealand in 1991 and 1999, funded by the Department of Internal Affairs.2,3 Using a revised version of the South Oaks Gambling Screen (SOGS-R), the 1999 New Zealand Gaming Survey found that approximately 1.3% of the population were problem gamblers (including probable pathological gamblers), representing 22,000–51,000 adults in New Zealand.3
Risk factors for current problem gambling included being in paid employment, being of Māori or Pacific ethnicity, and lacking formal qualifications.

These risk factors have also found to be significant for problem gambling in other studies. In particular, there is evidence to suggest that stressed populations, such as indigenous and ethnic minority groups, have higher proportions of problem gamblers than the general population.\(^6,7\) Other studies have found risk factors for problem gambling include being male,\(^6,8,9\) being of lower socioeconomic status,\(^10\) having lower education levels,\(^6,9,11,12\) and being employed.\(^11\)

Significant associations have been found between problem gambling and health status. Studies have found that, compared to non-problem gamblers, problem gamblers showed significantly higher rates of poor or fair health, mental health problems, and psychological distress.\(^4,13\) Furthermore, correlations have been found between problem gambling and other addictive behaviours, such as alcohol abuse\(^10,13-15\) and daily smoking.\(^13,16\)

In the New Zealand context, a small survey in the New Zealand Gaming Survey series in 1999 found associations between problem gambling and both hazardous drinking and daily cigarette smoking.\(^17\) No association was found between problem gambling and mental health, as measured by the General Health Questionnaire (GHQ-12). However, these results were based on a response rate of 45%, and confidence intervals were not calculated due to the complexity of the survey design.

The aims of this paper were to estimate the current prevalence rate of problem gambling in New Zealand, identify risk factors predictive of problem gambling, and test whether problem gambling is associated with other addictive behaviours or self-rated health status. Further results from the analysis of the gambling section of the 2002/03 New Zealand Health Survey are presented in a Ministry of Health publication.\(^18\)

**Methods**

This current research used the results of the 2002/03 New Zealand Health Survey (NZHS), a national survey about the health of the general adult population in New Zealand. The survey included questions about gambling participation and problems associated with gambling, as well as a wide range of health questions.\(^19\)

The survey used a complex multi-stage design with stratification and clustering. The target population in the general survey was all non-institutionalised adults aged 15 years and over, living in permanent private dwellings. The sample comprised of 12,529 respondents, and had a response rate of 72%. Oversampling amongst ethnic groups in the survey resulted in sample sizes of 4120 Māori, 908 Pacific peoples, and 1172 Asians (prioritised ethnicity\(^20\)).

The survey was administered through face-to-face interviews conducted by trained and experienced interviewers. Pre-survey letters were sent to selected households before the interviewer visited the house, and up to 10 callbacks were made to each selected household. The interviews for the survey were carried out between September 2002 and January 2004.

In the gambling section of the survey, all respondents were firstly asked which, if any, of the following gambling activities they had participated in during the past 12 months: Lotto, Instant Kiwi, Daily Keno, casinos, non-casino gaming machines, horse and dog races, sports betting, Housie (bingo), 0900 gambling, and Internet games. Other gambling activities, such as mah-jong, raffles, and non-casino card games, were not included.

In population surveys, the prevalence of problem gambling is typically measured with a gambling screen, which is a set of questions used to classify respondents’ levels of problematic gambling. Gambling screens include the South Oaks Gambling Screen (SOGS),\(^21\) screens based on the DSM-IV
criteria for pathological gambling,22 the two-question Lie/Bet screen,23 the Early Intervention Gambling Health Test (EIGHT) screen,24 the Gamblers Anonymous (GA) screen, and the Canadian Problem Gambling Index (CPGI).25 However, there is currently no “gold standard” gambling screen in use. The choice of gambling screen can affect the estimate of problem gambling rates, with some screens giving more conservative prevalence rates than others.

When the 2002/03 New Zealand Health Survey questionnaire was developed, it was considered that no existing gambling screen fulfilled the criteria of being short enough to include in a general health survey, while minimising respondent burden, but still measuring a wide range of negative gambling effects. For this reason, the Ministry of Health and a contracted technical specialist chose to develop a 10-question gambling screen for this survey, which included questions validated as part of several different screens, including the SOGS and DSM-IV screens (Table 1). The screen also included a past-12 month version of the Lie/Bet screen, which consists of two questions about whether the respondent had (i) felt the need to lie about their gambling, or (ii) felt the need to bet more and more; at least one positive response indicates problematic gambling.

The lifetime version of the Lie/Bet screen has since been validated in a community sample in Norway, with the screen showing good specificity and sensitivity relative to the DSM-IV.26 Note that the choice of developing a new gambling screen for the 2002/03 NZHS potentially limits comparison with the results from other gambling screens. This is discussed further in the results section below.

Table 1. 2002/03 New Zealand Health Survey gambling screen questions

<table>
<thead>
<tr>
<th>Gambling screen question</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 In the last 12 months, have you ever felt worried or depressed after playing any of those games? (referring to the list of gambling activities)</td>
<td>EIGHT</td>
</tr>
<tr>
<td>2 In the last 12 months, has anyone been worried or concerned enough to ask you about your gambling?</td>
<td>SOGS, EIGHT</td>
</tr>
<tr>
<td>3 In the last 12 months, have you ever gone into debt or borrowed money or had your credit card owing, from money spent on gambling?</td>
<td>SOGS; similar to GA</td>
</tr>
<tr>
<td>4 Do you feel that you have ever had a problem with gambling?</td>
<td>SOGS; similar to EIGHT</td>
</tr>
<tr>
<td>5 (If answered ‘yes’ to question 4) And in the last 12 months?</td>
<td>Similar to SOGS and EIGHT</td>
</tr>
<tr>
<td>6 In the last 12 months, have you said you were winning from gambling when in fact you lost?</td>
<td>SOGS</td>
</tr>
<tr>
<td>7 In the last 12 months, have you felt you would like to stop gambling but didn’t think that you could?</td>
<td>SOGS; similar to DSM</td>
</tr>
<tr>
<td>8 In the last 12 months, have you felt guilty or bad for doing wrong because of your gambling?</td>
<td>SOGS; similar to GA and EIGHT</td>
</tr>
<tr>
<td>9 In the last 12 months, have you felt at any time, the need to bet more and more money?</td>
<td>Lie/Bet; similar to SOGS, DSM, EIGHT</td>
</tr>
<tr>
<td>10 In the last 12 months, have you had to lie to people important to you about how much you gambled?</td>
<td>Lie/Bet; similar to DSM, SOGS and EIGHT</td>
</tr>
</tbody>
</table>

The gambling screen was only administered to respondents who reported spending over NZ$30 on gambling during at least 5 weeks in the previous year. Asking only the people who were likely to have gambling problems, indicated by the respondent spending a minimum of this conservative amount of time and money on gambling, helped to minimise the respondent burden for the health survey, for which the median time taken to complete the questionnaire was 60 minutes.19 This type of approach of only administering gambling screens to certain gamblers has also been used in other gambling...
prevalence surveys. In total, 613 respondents answered the gambling screen in the 2002/03 New Zealand Health Survey, representing 4.9% of the survey sample, or 3.7% of the population. People were classified as problem gamblers if they: (i) answered ‘yes’ to at least one of the two Lie/Bet questions (Q9 and 10); (ii) felt that they had had a problem with gambling in the last 12 months (Q5); or (iii) answered ‘yes’ to several of the other questions (in particular, at least five of Q2-3 and 5-8, or at least three of Q1-3 and 6-8). Current prevalence rates refer to the past 12 month’s rates; this time period was used in preference to a 6-month time period, as it produces fewer false negatives.

Gambling screen respondents were also asked questions about their normal weekly gambling expenditure. If they used alcohol or tobacco, they were also asked whether their use of alcohol or tobacco increased or decreased when they gambled. These analyses did not take into account the initial alcohol consumption or tobacco use before gambling.

The demographics covered in the survey were based on the 2001 Census questions, and included sex, age, ethnicity, country of birth, education level, income, employment status, household characteristics, income support, and socioeconomic deprivation. Ethnicity in this report refers to prioritised ethnicity, in the order of Māori, Pacific, Asian, and European/Other.

Socioeconomic deprivation was measured with the area-based NZDep2001 Index of Deprivation, transformed into quintiles, from NZDep 1 (least deprived) to NZDep 5 (most deprived). Self-rated mental and physical health were measured with a short-form questionnaire, the SF-36. Potentially hazardous drinking behaviour was indicated by a score of eight or more on the AUDIT (Alcohol Use Disorders Identification Test) screen.

This research used methods appropriate for the analysis of complex surveys. Results were weighted to represent the general New Zealand adult non-institutionalised population aged 15 and over living in permanent private dwellings. Point estimates are given with 95% confidence intervals, calculated using the delete-a-group jack-knife method. Logistic regression was used to determine risk factors for problem gambling, and also to test whether problem gambling was associated with other addictive behaviours and health status, while controlling for potential confounding variables.

For the analysis of self-rated health, the SF-36 scores were transformed into dichotomous outcomes (0,1) due to skewed distributions. Significance testing was carried out using the Pearson chi squared test for model comparison, and the Wald test for parameter values of individual explanatory variables. The Wald test used the weighted regression estimate and jackknifed standard error to calculate a test value (assuming a standard normal distribution), from which a p-value was obtained.

Results

Gambling and problem gambling—The estimated prevalence rate of problem gambling was 1.2% (1.0–1.5) of the New Zealand adult population, representing 32,800 (26,200–39,400) people.

The descriptive analysis presented in Table 2 shows inequalities in gambling-related problems experienced in the population, with Māori and Pacific peoples having significantly higher rates of problem gambling than other people. In particular, these results suggest that the Pacific population experience a bimodal pattern of gambling, with low participation rates—but among Pacific people who gamble, very high rates of problematic gambling. No differences in gambling participation were found across levels of socioeconomic deprivation, although people in the most deprived areas had significantly higher rates of problem gambling than people in the least deprived areas.

While gambling participation rates were lower among the younger and older age groups (15–24 years and 65+ years), no significant differences were found in participation between people aged 25–65 years. However, problem gambling rates were highest among people aged 25–34 years.

Multiple regression found that significant risk factors for problem gambling in New Zealand included being aged 25–34, being of Māori or Pacific ethnicity, being employed, being less qualified, and living alone (Table 3). Interestingly, model
selection procedures found ethnicity to be a superior explanatory variable than socioeconomic deprivation. As a result, deprivation was determined not to be a significant risk factor for problem gambling when included in the model with ethnicity, and was thus removed from the model.

Table 2. Gambling and problem-gambling prevalence rates in New Zealand, 2002–2003

<table>
<thead>
<tr>
<th>Population group</th>
<th>Past-year gamblers</th>
<th>Problem gamblers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% (95% confidence interval)</td>
<td>% (95% confidence interval)</td>
</tr>
<tr>
<td></td>
<td>Among population</td>
<td>Among past-year gamblers</td>
</tr>
<tr>
<td>Total</td>
<td>69.4 (68.2–70.6)</td>
<td>1.2 (1.0–1.5)</td>
</tr>
<tr>
<td>Male</td>
<td>70.7 (69.0–72.5)</td>
<td>1.6 (1.1–2.0)</td>
</tr>
<tr>
<td>Female</td>
<td>68.1 (66.4–69.9)</td>
<td>0.9 (0.7–1.2)</td>
</tr>
<tr>
<td>European/Other</td>
<td>71.8 (70.5–73.1)</td>
<td>0.8 (0.6–1.1)</td>
</tr>
<tr>
<td>Māori</td>
<td>74.1 (71.1–77.0)</td>
<td>3.3 (2.1–4.4)</td>
</tr>
<tr>
<td>Pacific peoples</td>
<td>53.9 (48.1–59.6)</td>
<td>3.8 (1.9–5.7)</td>
</tr>
<tr>
<td>Asian peoples</td>
<td>40.4 (35.8–45.0)</td>
<td>*</td>
</tr>
<tr>
<td>Aged 15–24 years</td>
<td>58.8 (55.6–62.1)</td>
<td>1.5 (0.7–2.2)</td>
</tr>
<tr>
<td>25–34 years</td>
<td>72.0 (69.2–74.8)</td>
<td>2.3 (1.4–3.3)</td>
</tr>
<tr>
<td>35–44 years</td>
<td>72.6 (70.4–74.8)</td>
<td>1.1 (0.6–1.5)</td>
</tr>
<tr>
<td>45–54 years</td>
<td>75.6 (73.2–78.1)</td>
<td>1.3 (0.7–1.9)</td>
</tr>
<tr>
<td>55–64 years</td>
<td>72.1 (69.2–75.0)</td>
<td>0.7 (0.3–1.1)</td>
</tr>
<tr>
<td>65+ years</td>
<td>64.4 (61.8–67.0)</td>
<td>*</td>
</tr>
<tr>
<td>NZDep01 quintile 1 (least deprived)</td>
<td>68.7 (65.7–71.6)</td>
<td>0.8 (0.3–1.3)</td>
</tr>
<tr>
<td>NZDep01 quintile 2</td>
<td>71.0 (68.1–74.0)</td>
<td>0.9 (0.2–1.3)</td>
</tr>
<tr>
<td>NZDep01 quintile 3</td>
<td>71.9 (69.3–74.5)</td>
<td>0.9 (0.4–1.4)</td>
</tr>
<tr>
<td>NZDep01 quintile 4</td>
<td>68.5 (65.2–71.9)</td>
<td>1.7 (1.0–2.4)</td>
</tr>
<tr>
<td>NZDep01 quintile 5 (most deprived)</td>
<td>66.8 (62.9–70.7)</td>
<td>1.9 (1.3–2.4)</td>
</tr>
</tbody>
</table>

*Suppressed due to low numbers; results considered unreliable.

Problem gambling and other addictive behaviours—Table 4 shows that problem gambling was significantly associated with the addictive behaviours of daily smoking and potentially hazardous drinking behaviour (as found by the AUDIT screen). These results show that problem gamblers have high rates of smoking compared to other people, with 58.3% being daily smokers. This association remained statistically significant when controlling for other variables with regression analysis (sex, age, ethnicity, NZDep01 quintile, household size, education, and employment status).

Problem gamblers were also more likely to increase the amount smoked while gambling (61.2%) compared to non-problem gamblers (32.4%).

A key finding was that approximately half of all problem gamblers have potentially hazardous drinking behaviour, compared to only one in six non-problem gamblers. Regression analysis showed that problem gamblers are four times more likely to have potentially hazardous drinking behaviour than people who are not problem gamblers (when controlling for sex, age, ethnicity, NZDep01 quintile, household size, education, and employment status).

In addition to this, problem gamblers were three times more likely than other people to have tried marijuana in their lifetime.
### Table 3. Risk factors for problem gambling

<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>1.00</td>
</tr>
<tr>
<td>Female</td>
<td>0.66 (0.43–1.01)</td>
</tr>
<tr>
<td>European/Others</td>
<td>1.00</td>
</tr>
<tr>
<td>Māori</td>
<td>3.51 (2.05–6.00)**</td>
</tr>
<tr>
<td>Pacific</td>
<td>4.97 (2.44–10.11)**</td>
</tr>
<tr>
<td>Asian</td>
<td>1.73 (0.71–4.18)</td>
</tr>
<tr>
<td>Aged 15–24 years</td>
<td>3.70 (1.41–9.70)**</td>
</tr>
<tr>
<td>25–34 years</td>
<td>6.02 (2.68–13.51)**</td>
</tr>
<tr>
<td>35–44 years</td>
<td>2.86 (1.28–6.39)**</td>
</tr>
<tr>
<td>45–54 years</td>
<td>3.16 (1.28–7.80)*</td>
</tr>
<tr>
<td>55+ years</td>
<td>1.00</td>
</tr>
<tr>
<td>Household size of 1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3.42 (1.44–8.10)**</td>
</tr>
<tr>
<td>3</td>
<td>1.87 (0.93–3.79)</td>
</tr>
<tr>
<td>4</td>
<td>1.42 (0.70–2.89)</td>
</tr>
<tr>
<td>5 or more</td>
<td>0.93 (0.41–2.13)</td>
</tr>
<tr>
<td>Employed</td>
<td>1.00</td>
</tr>
<tr>
<td>Not employed</td>
<td>0.59 (0.37–0.92)*</td>
</tr>
<tr>
<td>No qualification</td>
<td>1.00</td>
</tr>
<tr>
<td>School qualification</td>
<td>0.70 (0.37–1.34)</td>
</tr>
<tr>
<td>Vocational or trade qualification</td>
<td>0.76 (0.44–1.32)</td>
</tr>
<tr>
<td>Degree</td>
<td>0.27 (0.08–0.89)*</td>
</tr>
</tbody>
</table>

*p <0.05; **p <0.01

### Table 4. Summary of associations between problem gambling and other addictive behaviours

<table>
<thead>
<tr>
<th>Addictive behaviour</th>
<th>Problem gamblers % (95% CI)</th>
<th>Non-problem gamblers % (95% CI)</th>
<th>Odds ratio (95% CI)a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily cigarette smoker</td>
<td>58.3 (46.5–70.0)</td>
<td>22.5 (21.3–23.6)</td>
<td>2.96** (1.68–5.21)</td>
</tr>
<tr>
<td>Has potentially hazardous drinking behaviour (AUDIT)</td>
<td>53.5 (41.8–65.1)</td>
<td>16.8 (15.7–17.8)</td>
<td>4.05** (2.23–7.34)</td>
</tr>
<tr>
<td>Has tried marijuana</td>
<td>71.9 (61.8–82.0)</td>
<td>38.4 (37.1–39.7)</td>
<td>3.06** (1.76–5.34)</td>
</tr>
<tr>
<td>Increases tobacco amount while gambling</td>
<td>61.2 (47.8–74.2)</td>
<td>32.4 (19.5–45.3)</td>
<td>4.49* (1.32–15.21)</td>
</tr>
<tr>
<td>Increases alcohol amount while gambling</td>
<td>13.2 (5.3–21.0)</td>
<td>6.3 (1.7–10.9)</td>
<td>1.60 (0.28–9.04)</td>
</tr>
</tbody>
</table>

*a Odds ratio given for problem gamblers compared to reference category of people who are not problem gamblers (who have an odds ratio of 1). Odds ratios for all analyses in the table were calculated with logistic regression, controlling for the following variables: sex, age, ethnicity, NZDep01 quintile, household size, education and employment status. b Odds ratios also controlled for potentially hazardous drinking behaviour. c Only among those who smoked and who answered the gambling screen. d Only among those who had consumed alcohol in the last year and who answered the gambling screen. *p < 0.05; **p < 0.01.
Problem gambling and self-rated health status—Associations were measured between problem gambling and self-rated health (measured by the SF-36 screen). Table 5 presents the results from a regression analysis, giving the odds ratio and significance of problem gambling as an explanatory variable, in modelling the probability of having worse self-rated health (indicated by being below the cut-off score) for the individual SF-36 scales. For all SF-36 models, the explanatory variables were sex, age, ethnicity, education, NZDep quintile, and household size.

These results show that problem gambling is significantly associated with having worse self-rated health in several health domains. Problem gamblers are 2.6 times more likely to have worse self-rated mental health status than non-problem gamblers. Similarly, problem gambling was associated with worse self-rated health in the Role Emotional, Vitality, and General Health domains.

Table 5. Associations between self-rated health and problem gambling

<table>
<thead>
<tr>
<th>SF-36 Health domain</th>
<th>Cut-off score</th>
<th>Odds ratio of being below cut-off score (95% CI) a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical functioning</td>
<td>80</td>
<td>0.97 (0.57–1.67)</td>
</tr>
<tr>
<td>Role physical</td>
<td>90</td>
<td>1.62 (1.02–2.56)*</td>
</tr>
<tr>
<td>Bodily pain</td>
<td>60</td>
<td>1.22 (0.75–2.00)</td>
</tr>
<tr>
<td>General health</td>
<td>50</td>
<td>2.12 (1.16–3.89)*</td>
</tr>
<tr>
<td>Vitality</td>
<td>50</td>
<td>2.14 (1.25–3.66)**</td>
</tr>
<tr>
<td>Social functioning</td>
<td>90</td>
<td>1.52 (1.00–2.32)</td>
</tr>
<tr>
<td>Role emotional</td>
<td>90</td>
<td>2.36 (1.45–3.84)**</td>
</tr>
<tr>
<td>Mental health</td>
<td>80</td>
<td>2.60 (1.65–4.10)**</td>
</tr>
</tbody>
</table>

a Odds ratios given for problem gamblers compared to the reference category of people who are not problem gamblers, who have an odds ratio of 1. *p <0.05; **p <0.01.

Table 6. Comparison of problem gambling rates from Lie/Bet screen and 2002/03 New Zealand Health Survey gambling screen

<table>
<thead>
<tr>
<th>Population group</th>
<th>Problem gambling prevalence % (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lie/Bet screen</td>
</tr>
<tr>
<td>Total</td>
<td>1.0 (0.8–1.2)</td>
</tr>
<tr>
<td>Male</td>
<td>1.2 (0.8–1.6)</td>
</tr>
<tr>
<td>Female</td>
<td>0.8 (0.6–1.1)</td>
</tr>
<tr>
<td>European/Other</td>
<td>0.7 (0.4–0.9)</td>
</tr>
<tr>
<td>Māori</td>
<td>2.5 (1.7–3.3)</td>
</tr>
<tr>
<td>Pacific</td>
<td>3.8 (1.9–5.6)</td>
</tr>
<tr>
<td>Aged 15–24 years</td>
<td>1.3 (0.6–2.0)</td>
</tr>
<tr>
<td>25–34 years</td>
<td>1.8 (1.0–2.5)</td>
</tr>
<tr>
<td>35–44 years</td>
<td>0.9 (0.5–1.3)</td>
</tr>
<tr>
<td>45–54 years</td>
<td>1.1 (0.5–1.7)</td>
</tr>
<tr>
<td>55–64 years</td>
<td>0.6 (0.2–1.1)</td>
</tr>
</tbody>
</table>

Note: Results for Asian peoples and people aged 65+ years were suppressed due to low numbers.
Evaluation of problem gambling screen—The gambling screen showed good internal reliability, which indicates that the individual questions in the screen are consistent in measuring the same underlying construct. Internal reliability is measured with Cronbach’s alpha, which has a recommended minimum value of 0.7 to indicate good internal reliability.\textsuperscript{32} Compared to this, the 2002/03 NZHS Gambling Screen showed very good internal reliability, with a value for Cronbach’s alpha of 0.91. In addition, the 2002/03 New Zealand Health Survey gambling screen included a past-year version of the two-question Lie/Bet screen. A comparison of prevalence estimates from the full 2002/03 NZHS screen (including the Lie/Bet screen) and estimates from the Lie/Bet screen alone, shows similar prevalences, although the Lie/Bet estimates are somewhat lower (Table 6).

Discussion

The survey results showed that problem gambling is a significant health problem in New Zealand, which disproportionately affects Māori and Pacific peoples in particular.

Māori have both higher gambling participation rates and higher problem gambling rates than the national average. While gambling participation is lower amongst Pacific peoples than the national average, Pacific peoples also have significantly higher problem gambling rates than the rest of the population. These results suggest that Pacific peoples who gamble are significantly more likely to experience problems from their gambling than other gamblers. This supports previous evidence for ‘bimodal gambling patterns’ amongst some minority groups.\textsuperscript{3,33}

While almost two-thirds of problem gamblers live in the 40% most deprived areas in New Zealand, socioeconomic deprivation was not a significant risk factor for problem gambling when controlling for other variables. However, it should be noted that ethnicity and deprivation are highly correlated in New Zealand, with Māori and Pacific peoples more likely to live in more deprived areas; this means that the effects of ethnicity and deprivation cannot be fully separated.

Results show that problem gambling is significantly correlated with potentially hazardous drinking and tobacco smoking. Since this survey was conducted, new smoke-free laws have been introduced in New Zealand that ban smoking in all licensed premises. Given that non-casino gaming machines are generally located in licensed premises, these laws may have affected both the gambling behaviour of smokers, and the amount smoked by gamblers. The impact of these laws on the prevalence of problem gambling could be investigated in future, given the strong association found in this study between problem gambling and smoking.

This study found significant associations between problem gambling and worse self-rated health status, indicating that problem gambling may impact on many sectors of the gambler’s life, particularly their mental health and overall feelings of well-being and vitality. These results suggest that health professionals could screen for problem gambling, if someone presents for other associated health matters such as mental health issues or alcohol addiction.

The results from this study showed similar prevalence rates for problem gambling, and similar associations between problem gambling and both hazardous drinking and cigarette smoking, to those found in the 1999 New Zealand Gaming Survey.\textsuperscript{3} Despite
the differing methodologies, the results from the 2002/03 NZHS also identify similar risk factors for problem gambling to those identified in the 1999 survey,\(^3\) in particular finding Māori and Pacific people more at risk from problem gambling. The results thus also confirm that there has been a change from the risk factors identified in the 1991 survey\(^2\) where being male, a young adult (18–24 years) and being unemployed were significant risk factors for problem gambling.

Furthermore, this study found similar problem gambling risk factors to studies carried out in other jurisdictions, in particular the risk factors of having lower education levels and being employed,\(^6,9,11,12\) although the risk factors of being male and of lower socioeconomic status were not significant as they were in other studies.\(^6,8–10\) The associations found in this study between problem gambling, and daily smoking and alcohol consumption support similar findings in other studies.\(^10,13–16\)

Researching population prevalence rates of problem gambling with surveys has some general limitations. Responses to gambling questions may not be reliable, due to difficulties in recalling gambling behaviour for the past year, different interpretations of expenditure, or over- or under-estimating gambling expenditure. Studies have raised the possibility that some heavy gamblers do not report their gambling habits and spending correctly, as they may be in denial, or be wishing to conceal their gambling from other people.\(^3,34\) For these reasons, estimates for the prevalence of problem gambling may in general be conservative.

Only some survey respondents were administered the gambling screen; specifically, all those people who had spent $30 or more on gambling on five or more weeks of the past 12 months. This may have resulted in conservative estimates for the prevalence rate of problem gambling, as the gambling screen may not have been administered to those gamblers who binge, or who underreported their gambling expenditure.

In addition, the use of a newly developed gambling screen resulted in some limitations, particularly in comparisons with previous surveys. This gambling screen has not been tested for consistency with other gambling screens, nor has it been clinically validated, although it showed very good internal reliability. This study found that the results from the gambling screen were consistent with those from the Lie/Bet screen alone, although the Lie/Bet screen gave somewhat lower prevalence rates. While the prevalence rates are similar in this study (1.2%) compared to previous New Zealand studies (1.3% from the 1999 National Prevalence Survey\(^3\)), care should be taken when comparing the results of this study with those from other gambling research, due to the use of different problem gambling measures.

**Conclusion**

This study provides further evidence about problem gambling in the New Zealand community, which may be useful in informing policy and public health programmes. In particular, this study showed that Māori and Pacific peoples are at greater risk from problem gambling than others, and furthermore that problem gambling is significantly associated with other risk behaviours and self-rated health status. These results may be useful in addressing the disproportionate harm that occurs from problem gambling, particularly in the identified at-risk population groups.
While the 2002/03 NZHS Gambling Screen was not a validated gambling screen and has not been used in other studies, it still measures a construct of problem gambling, and is useful in the analysis of risk factors and associations with problem gambling.

The results from this study are consistent with earlier findings, in particular finding similar risk factors for problem gambling as the 1999 New Zealand national survey. The 2006/07 New Zealand Health Survey has included the Canadian Problem Gambling Index (CPGI), which has been used in several population surveys in countries such as Canada and Australia.

Indeed, the inclusion of the CPGI in the next national health survey will provide opportunity for further investigations into problem gambling in New Zealand in future.

**Competing interests:** None.

**Disclaimer:** Views and conclusions in this article are those of the authors, and may not reflect those of the Ministry of Health.

**Author information:** Kylie Mason, Advisor (GeoHealth/Statistics), Public Health Intelligence, Ministry of Health, Wellington; Richard Arnold, Senior Lecturer, School of Mathematics, Statistics and Computer Science, Victoria University of Wellington, Wellington

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**Correspondence:** Kylie Mason, Public Health Intelligence, Ministry of Health, PO Box 5013, Wellington. Email: Kylie_Mason@moh.govt.nz

**References:**


Problem gambling: patients affected by their own or another’s gambling may approve of help from general practitioners

Sean Sullivan, Ross McCormick, Michael Lamont, Alison Penfold

Abstract

Aims To identify the health effects, including depression, on problem gambling patients and family members, and their perception of their GP as a help provider for problem gambling.

Methods 1580 patients from practices in Auckland, Taranaki, and Rotorua completed an anonymous questionnaire containing brief screens for problem gambling, effects on family of gambling, and depression. Patients were asked to assess their GP as a help provider for problem gambling.

Results 7.5% of patients were positive for problem gambling, ranging from 3% of NZ European patients to 24% of Pacific patients; 18% of patients were affected by another’s gambling. Less than one in four problem gambling patients, and one in three family positives, did not perceive their GP as a suitable help provider for problem gambling issues. Problem gambling patients were more likely than other patients to approve their GP as a help-provider. Patients affected by problem gambling were more depressed than other patients. No other disease indicators were found. Patients over 54 years are less likely than others to be problem gamblers.

Conclusions Problem gambling is associated with depression in patients. GPs are an important complementary resource for brief interventions for gambling problems, and for some possibly a more acceptable alternative than attending specialist problem gambling treatment providers.

Gambling problems have been associated with many health conditions including depression, anxiety, alcohol abuse, and suicidal ideation—and a problem gambler is more likely to be a cigarette smoker.1-5

The 2002/3 NZ Health Survey identified that problem gambling is ‘significantly associated with worse self-rated health in several health domains’ and 1.2% of the population were ‘worried or depressed after gambling’.6 This accorded with a recent large overseas study (n=43,093) that concluded that problem gambling was associated with tachycardia, angina, cirrhosis, and other liver disease—and was associated with higher medical utilisation and treatment in hospital emergency departments.1

In addition to health problems, many social and financial problems develop which may impact upon the health of both the gambler and their family. For instance, in 2005 in New Zealand, 2875 new clients accessed a specialist gambling helpline and 2714 new clients accessed counselling services.7 Less than one in three clients were family members of problem gamblers, despite an estimate that there may be at least seven others affected by each problem gambler.8
It has been estimated that between 3% and 11% of those suffering problem gambling will seek help from specialist services, but that the majority of those affected will not seek help from any service specifically for gambling issues.\(^8-9\)

The Ministry of Health has now, since the passing of the Gambling Act 2003, assumed responsibility for the minimisation of harm arising from gambling. In its 3-year strategic plan, it has identified that ‘a key focus will continue to be on using training to increase the capability of primary health and social services to carry out problem gambling screening’ p16.\(^10\)

General practitioners (GPs) believe it a legitimate role for them to address the effects of gambling problems upon their patients.\(^11-12\) Practitioners participating in the Practice Review Activity part of this study\(^13\) gave positive feedback as to the importance of identifying health problems arising from problem gambling. There was some concern, however, that they may not have the time to screen for gambling and to deal with any previously unknown morbidity.

The aims of this study were to ascertain whether patients would self-identify as being adversely affected by their own gambling, or by the gambling of others, whether these patients would be more likely to be depressed, and whether patients perceive GPs as suitable to provide help for gambling problems.

**Methods**

Four Primary Health Organisations (PHOs) in Auckland (2), Taranaki, and Rotorua continuously invited their patients over a 1- to 4-week period to complete a questionnaire incorporating a brief problem gambling screen developed for General Practice (the Eight Screen; Sullivan 1999),\(^14-16\) a screen to identify those affected by others’ gambling (the COGS Screen; Sullivan 2002),\(^17\) and a two-question depression screen (Whoolley et al 1997).\(^18\)

The PHOs were approached with the assistance of the Department of General Practice and Primary Health Care, The University of Auckland, and 16 practices agreed to participate. Upon presenting to their GP’s practice, the practice receptionist referred all patients aged 16 years or older to information sheets and posters about the study—and invited the patient to complete the questionnaire anonymously, and return it folded to the receptionist for later collection. Patients were invited to discuss their responses with their GP if they had concerns after completing the questionnaire.

Patients were asked whether they considered their doctor could help address gambling problems, and responded by selecting one of three responses (no, maybe/uncertain, or yes). Demographic information was also sought.

Gambling screen results were compared with depression screen findings and with patients’ perceptions of their GP as help-providers for gambling problems.

Ethics approval for the project was obtained from the University of Auckland Human Subjects Ethics Committee (reference 2003/384).

**Results**

1580 patients participated—comprising 1075 in Auckland, 286 in Rotorua, and 219 in Taranaki. Receptionists at the practices reported that few, if any, refused to participate in the study because of its content, with the only refusals due to poor health, not having brought spectacles, or insufficient time to complete the questionnaire between arriving and their GP being available.

The majority of participants were female (58%; n=914), with males comprising 36% (n=563), and a further 7% (n=103) not disclosing their gender.

There was a widespread ethnic range of participants.
Ethnicity of patients approximately reflected that of the general New Zealand (NZ) population, with Māori comprising 12.5% of participants, NZ European 51.5%, Pacific 9.5%, Chinese 9.5%, Indian 3.5%, and ‘other ethnicities’ 3.5%; 10% did not disclose their ethnicity.

Demographic variable effect on problem gambling was analysed using a generalised linear mixed model, with GP practice included as a random effect to allow for clustering caused by practice. A similar model was used to determine the association between depression, problem gambling and the effects of another’s gambling. 118 patients were positive on the gambling screen (7.5%; 108 did not complete the gambling screen and the percentage is based upon the assumption that these were negative for problem gambling). Gender of problem gambling positives were similar (male 7.6%, female 7.9%) and gender was not found to influence problem gambling (p=0.75).

Those with gambling problems were less likely to be over 54 years of age than younger (15 to 34 years p=0.05 OR 2.4, 35 to 54 years p=0.02 OR 2.6). Fifty percent of those scoring as positive on the problem gambling screen were Community Services Card (CSC) holders, compared with 38% of all patients in the survey—although this difference was not statistically significant (p=0.09). The ethnicity of problem gambling positives is shown in Table 1.

<table>
<thead>
<tr>
<th>Ethnicity of patients</th>
<th>Numbers within that ethnicity identified as problem gambling screen positives</th>
<th>Percentage of patients positive identified as within that ethnic group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Māori</td>
<td>14</td>
<td>7% (14/200)*</td>
</tr>
<tr>
<td>Pacific</td>
<td>37</td>
<td>24% (37/152)*</td>
</tr>
<tr>
<td>NZ European</td>
<td>21</td>
<td>3% (21/713)</td>
</tr>
<tr>
<td>Chinese</td>
<td>20</td>
<td>13% (20/150)*</td>
</tr>
<tr>
<td>Indian</td>
<td>5</td>
<td>10% (5/51)</td>
</tr>
<tr>
<td>Other ethnicity</td>
<td>9</td>
<td>17% (9/54)*</td>
</tr>
<tr>
<td>Ethnicity missing</td>
<td>12</td>
<td>8% (12/160)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>118</strong></td>
<td><strong>7.5% (118/1580)</strong></td>
</tr>
</tbody>
</table>

*Gambling problems were statistically more likely for Māori (p=0.02, OR 2.5), Pacific (p<0.0001, OR 8.8), Chinese (p<0.0001, OR 5.3), and other ethnicities (p=0.0003, OR 4.0) when compared with NZ European.

Problem gambling was found to affect the presence of depression (p=0.0008, OR 2.4). Of those positive on the gambling screen, 63.5% scored as positive on the depression screen (yes to at least one of the two depression questions), and 40% answered yes to both depression questions. This compares with 31.2% of those scoring as negative on the problem gambling screen that scored positive on the depression screen. Of these problem gambling negatives, 17.6% answered yes to both depression questions.

Patients were asked to describe their reason for presenting that day to the clinic. Of the 118 problem gambling screen positives, 5 presented for medical certificates, 6 for depression (compared with 31 overall presenting for depression), 2 for ‘addiction’, and 2 for migraines. Forty-three (36%) of problem gambling positives did not disclose their reasons for presenting that day to the clinic.
When compared with patients in general, there were no obvious indicators that would suggest the need to immediately question a patient about problem gambling.

Patients were asked to respond in the COGS family screen if they had ever been negatively affected by another’s gambling (Table 2).

**Table 2. Affected by another’s gambling? Responses to COGS gambling screen (n=1561)**

<table>
<thead>
<tr>
<th>COGS Question</th>
<th>Number responding positive (%)</th>
<th>Number positive on depression screen (% positive of those responding to the COGS question)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I don’t know for sure</td>
<td>117 (7%)</td>
<td>61 (52%)</td>
</tr>
<tr>
<td>Yes, in the past</td>
<td>122 (8%)</td>
<td>64 (52%)</td>
</tr>
<tr>
<td>Yes, currently</td>
<td>42 (3%)</td>
<td>29 (69%)</td>
</tr>
<tr>
<td>No, never</td>
<td>1280 (81%)</td>
<td>368 (29%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>1561</strong></td>
<td><strong>368 (29%)</strong></td>
</tr>
</tbody>
</table>

Of those possibly affected by another’s gambling, 278 provided a range of current effects of that gambling. Twenty-nine percent responded that it didn’t affect them any longer, while others worried (26%), were nervous (5.5%), believed it affected their health (4.5%), found it difficult to talk about (6%), were concerned about their family’s safety (8%), or were uncertain about its current effect on them (21%). Most (68.5%) required no help at this stage, however 16.5% wanted appropriate information, 5.5% wanted to talk in confidence, and 9.5% wanted support or help.

Those affected by another’s gambling were found to be more likely to be affected by depression (p=0.0001, OR 2.2).

Patients were asked to respond to whether they thought their doctor could help with gambling problems (caused by their own gambling or the gambling of another). See Table 3.

**Table 3: Perception of GPs being help-providers for effects of problem gambling**

<table>
<thead>
<tr>
<th>Response as to whether their doctor could help with gambling problems</th>
<th>COGS positives (%)—i.e. those affected by another’s gambling (n=263 responses; 18 responses missing)</th>
<th>Gambling screen positives (%) (n=113; 5 responses missing)</th>
<th>All patients (%) (n=1394; 186 responses missing)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>35 (13%)</td>
<td>25 (21%)*</td>
<td>210 (13%)</td>
</tr>
<tr>
<td>Maybe/uncertain</td>
<td>137 (52%)</td>
<td>60 (51%)</td>
<td>572 (36%)</td>
</tr>
<tr>
<td>No</td>
<td>91 (33%)</td>
<td>28 (24%)</td>
<td>612 (39%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>263</strong></td>
<td><strong>113</strong></td>
<td><strong>1394</strong></td>
</tr>
</tbody>
</table>

*Positive association

Problem gamblers were more likely than others to consider their GP to be an appropriate help provider for problem gambling (p=0.007, OR 1.9), but those affected by another’s gambling did not appear to share this view (p=0.53).
Conclusion

This is the first study to determine the effects of gambling in a GP patient population upon both those who gamble and their families. Patients readily participated in screening for gambling problems and were willing to disclose gambling problems in the GP setting, albeit anonymously. Those who were problem gamblers comprised 7.5% of the participating patients, with two-thirds being positive on a depression screen. This depression prevalence rate was twice that found in those screening negative for problem gambling. Those affected by another’s gambling were also more likely to be depressed than non-affected patients.

Eighteen percent of patients appeared to be affected by another’s gambling, with many being unclear about exactly how they were affected. This is unsurprising as problem gambling is often difficult to detect in family members, especially in determining whether it is in the past or has recurred. Over two-thirds of these family members of problem gamblers appeared to be currently affected with a range of concerns (many health-related).

The prevalence of problem gambling for different ethnicities reported here is similar to other studies, with higher risk found amongst Māori and Pacific peoples. The higher risk with Chinese patients for problem gambling identified in this study has not been found in NZ epidemiological studies. This may be due to a previous Chinese community reluctance to disclose the presence of an issue that may reflect negatively on that community. In the current study many of the participating Chinese patients were attending a practice that specialised in this ethnic group. This may have helped improve patient participation and feeling safe around disclosure.

Patients who were problem gambling were equally likely to be male or female. Specialist treatment services report many female patients reporting problem use of gambling machines. Although problem gambling and poverty has been shown to be associated, in this study being the holder of a Community Services Card (CSC) was found not to be associated with problem gambling. Therefore neither gender nor being the holder of a CSC card is a good indicator to screen for problem gambling, however being over 54 years appeared to reduce the risk for problem gambling and to signal a reduced need for problem gambling screening in that older age group.

With insufficient responses to presenting reasons, and the apparent lack of trends between positive problem gambling and presenting reasons, our study found no indication of presenting issues that would increase the need to screen except of course for patients presenting with depression. However the high level of possible depression identified (not only with problem gamblers and their family, but with those not affected by problem gambling), contrasted with the relative absence of depression or associated issues as a patient presenting reason to GPs.

GPs have identified that they saw problem gambling as within their field of work, but have questioned whether they had sufficient time to address gambling issues, especially amongst those who are identified as affected by problem gambling when presenting for other issues.

One in four problem gambling patients see their GP as an appropriate help-provider for problem gambling. The currently undecided half of those affected by their own or another’s gambling might be encouraged to support this view with more information.
Up to one-third of those affected by problem gambling hold a definite view against this role for a GP.

Selection of the PHOs and their practices were not randomised and this study may therefore not be generalisable to the extent that such a process would have provided. It is possible that our distribution to three centres may have partly mitigated against some of the effects of non-randomisation. We note that the prevalence of problem gambling identified in this study, particularly for Pacific patients, was considerably higher than has been identified in a second patient study.

The authors of the second study used a briefer screen for gambling embedded in a multi-item instrument3. The gambling screen used in our study has been validated for a range of New Zealand settings,15 while the prevalence of problem gambling amongst the Pacific population, and others, has been found in a third study to be within the range identified in our study.19

Raising awareness amongst patients of a GP’s role in providing help for both the problem gambler and their family; developing awareness amongst GPs of the prevalence of these problems amongst patients; and training in the use of specialist problem gambling screens may overcome the relative absence of clinical indicators to screen shown by this study.

Composite screens,21 such as the CHAT (a multi-item general practice tool), may assist to overcome the identification issues that exist in addressing what is a behavioural addiction that affects the almost 1 in 12 GP patients who problem gamble and the 1 in 6 patients who may be an affected family member of a problem gambler.

The Ministry of Health views primary health providers as an important resource for those affected by problem gambling. Our findings support this view and suggest general practitioners offer a viable helping alternative for problem gamblers and their affected family members.

In future, and with patient and GP education and support, problem gamblers and their families may come to see their GP as the most accessible health professional available to assist them address a behaviour and its effects that they may be reluctant to address elsewhere.

Competing interests: None.

Author information: Sean Sullivan, Psychologist, Abacus Counselling Training & Supervision Ltd, Auckland; Ross McCormick, Director, Goodfellow Unit, The University of Auckland, Auckland; Michael Lamont, Chair, Mangere Community Health Trust & Mangere Health Resources Trust, Auckland; Alison Penfold, Director, Abacus Counselling Training & Supervision Ltd, Auckland

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Correspondence: Sean Sullivan, Abacus, PO Box 90710 Auckland Mail Centre, Auckland. Fax: (09) 360 6357; email: sean@acts.co.nz
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19. Abbott M, Volberg R. Taking the pulse on gambling and problem gambling in New Zealand: a report on Phase One of the 1999 National Prevalence Survey. Wellington: Department of
Internal Affairs; 2000.


Comparison of plasma adiponectin levels in New Zealand Māori and Caucasian individuals

Brett Shand, Peter Elder, Russell Scott, Nicola Poa, Christopher M Frampton

Abstract

Aims Adiponectin is an adipocytokine with insulin-sensitising and anti-atherosclerotic effects. Low plasma adiponectin levels are known to predispose to the development of Type 2 diabetes mellitus. Given the increased prevalence of Type 2 diabetes in the Māori population in New Zealand, we carried out a study to compare plasma adiponectin levels between non-diabetic Māori and Caucasian subjects.

Methods Plasma adiponectin levels were measured in 111 pairs of non-diabetic Māori and Caucasian individuals, matched for gender, age (±6 years), body mass index (±4 kg/m²), waist circumference (±8 cm), and presence or absence of insulin resistance. Other data collected included anthropometric measurements, indices of glycaemic control and insulin sensitivity and plasma lipid profile. The data were analysed using paired t tests, Wilcoxon signed rank tests and correlation, and linear regression analyses.

Results Statistical analysis showed the two ethnic groups were well matched with the exception of smoking habits, intercurrent medications, and clinically insignificant differences in HbA1c and total cholesterol levels. Mean plasma adiponectin levels were significantly lower in the Māori group compared with the Caucasian group (7.32±SD 4.02 µg/ml vs 8.32±SD 4.15 µg/ml; p=0.03). The prevalence of abnormally low plasma adiponectin levels (≤4.0 µg/ml) was two times higher in Māori than in Caucasians. The difference in mean plasma adiponectin levels of 1.0 µg/ml between the two groups was relatively small and less than the normal biological variability for adiponectin measured in our laboratory. In both ethnic groups, there was a significant correlation between plasma adiponectin levels and gender and characteristics of the metabolic syndrome, but not with age, percentage body fat, or smoking habits.

Discussion These results indicate that Māori people tend to have lower plasma adiponectin levels than Caucasian people of similar age, body shape, and insulin sensitivity. The reason(s) for this ethnic difference remain unclear, but may be related to differences in body composition or genetic control of adiponectin synthesis. Prospective studies are required to determine the etiological importance of these low adiponectin levels in the Māori population.

Since its discovery by Scherer and co-workers in 1995, adiponectin has attracted increasing clinical and research attention as a key hormone in the aetiology of the metabolic syndrome, insulin resistance and Type 2 diabetes.

Adiponectin is a cytokine produced exclusively by white adipose tissue that influences several pathways of glucose and fatty acid metabolism—including the insulin signalling cascade, glucose uptake, hepatic gluconeogenesis, and skeletal muscle fatty acid oxidation and clearance. There is also evidence that adiponectin
has an anti-atherogenic action by down-regulating the expression of adhesion molecules and reducing the attachment of activated macrophages to the endothelium.⁵

Adiponectin exists in the serum as both high and low molecular weight complexes consisting of 18 and 3 monomers, respectively. Recent evidence suggests that the larger complexes have greater bioactivity.⁶,⁷

The central role of adiponectin in the aetiology of Type 2 diabetes has been confirmed in numerous cross-sectional studies,⁴,⁸ including our own,⁹ that have demonstrated a close relationship between decreased adiponectin levels and factors that constitute the metabolic syndrome.

In addition, several prospective studies have shown that low plasma adiponectin level predispose to the development of Type 2 diabetes.¹⁰⁻¹² Plasma adiponectin levels are therefore used increasingly as a marker of the metabolic syndrome and incipient Type 2 diabetes, with the assay being reliable and precise and subject to relatively low biovariability.¹³,¹⁴

In line with many countries in the world, New Zealand is experiencing a marked increase in the prevalence of obesity, the metabolic syndrome and Type 2 diabetes, with this increase being especially evident in the Māori and Polynesian populations. Epidemiological studies have shown the prevalence of known Type 2 diabetes is nearly three times higher in Māori compared to Caucasians (8.0% vs 2.9%, respectively).¹⁵

Several factors have been proposed to account for this increased burden of diabetes in the Māori population including greater obesity, a younger age at diagnosis, poorer glucose control and diabetes knowledge, and socioeconomic and cultural barriers to treatment.¹⁶ It is also possible that genetic and/or biochemical differences influence the disparity in diabetes prevalence, although evidence indicates that Māori and Polynesians are not intrinsically insulin resistant as a group, unlike other ethnic groups at high risk of Type 2 diabetes.¹⁷

Two epidemiological studies have demonstrated that ethnic groups with an increased prevalence of Type 2 diabetes have decreased levels of plasma adiponectin.⁸,¹⁸ Weyer and coworkers⁸ reported that Pima Indians had significantly lower adiponectin levels compared to Caucasians matched for adiposity, but that this difference was absent when the data were adjusted for the degree of insulin sensitivity.

Likewise, Valsamakis, and coworkers¹⁸ showed ethnic differences in serum adiponectin levels between male Caucasian and Indo-Asian subjects, matched for body mass index (BMI). To determine whether low plasma adiponectin levels were a contributing factor in the increased prevalence of Type 2 diabetes in the Māori population, we carried out a study comparing levels in non-diabetic Māori and Caucasian subjects. To negate the effects of other factors known to influence adiponectin levels, the paired subjects in the study were matched as closely as possible for age, body size and shape, and degree of insulin sensitivity.

**Methods**

**Study design**—The study compared plasma adiponectin levels in 111 pairs of Māori and Caucasian subjects, matched for gender, age (±6 years), BMI (±4 kg/m²), waist circumference (±8 cm), and presence or absence of insulin resistance, defined as a homeostasis model assessment percent sensitivity index (HOMA%S)¹⁹ <70%.
The matching for waist circumference ensured that subjects classified as obese—according to the criteria of the National Cholesterol Education Program - Adult Treatment Panel III (NCEP-ATPIII)—were paired with subjects also meeting these criteria (males >102 cm, females >88 cm). Similarly, subjects classified as insulin resistant, defined by a HOMA%S <70%, were matched with subjects with a similar degree of insulin resistance.

Subjects with either a BMI >45 kg/m², diabetes mellitus (fasting glucose level ≥7.0 mmol/L), or a prior cardiovascular disease such as myocardial infarction, ischaemic heart disease or symptomatic peripheral vascular disease were excluded from the study.

Enrolment of subjects—Enrolment of the 111 Māori subjects in the study was carried out primarily using advertisements in local newspapers and by contact with Māori health workers and community groups (n=95). The remaining 16 Māori subjects were enrolled as part of a 3-year longitudinal study on adiponectin we are currently undertaking at our research unit.

The majority of Caucasian subjects selected as matched pairs were recruited from a prospective study on adiponectin currently in progress (n=93), with the remaining 18 subjects being selected from the Christchurch Hospital Lipid Out-patient Clinic database. Māori ethnicity was defined according to the criteria used in the 2001 New Zealand population census, as having a Māori birth parent or grandparent. Signed, informed consent was obtained from all the subjects prior to enrolment in the study, with the study protocol being approved by the Canterbury Ethics Committee.

Collection of clinical data—The study participants were requested to fast for 10 hours before attending a morning appointment between 8:00–10:00am, at which a brief medical and medication history was obtained, followed by measurement of height, weight, BMI, percentage body fat (Bioimpedance method, Body fat analyser, Tanita Corp., Tokyo, Japan) and sitting blood pressure (measured in duplicate after 5 min of rest using a sphygmomanometer and expressed as mean arterial pressure).

The study participants were classified into 3 groups according to the number of metabolic syndrome characteristics recorded (0, 1–2, 3–5), as defined by the NCEP-ATPIII criteria (fasting glucose ≥5.6 mmol/L; fasting triglyceride ≥1.7 mmol/L; HDL-cholesterol <1.0 mmol/L (males), <1.3 mmol/L (females); waist circumference >102 cm (males), >88 cm (females); blood pressure ≥130 / ≥85 mmHg). The proportions of subjects in each ethnic group with impaired fasting glucose (IFG), hypertension, and dyslipidaemia were also calculated.

Collection of biochemical data—Venous blood samples were collected for the measurement of plasma adiponectin and other biochemical parameters. The fasting adiponectin levels in plasma samples stored at -30°C were measured in duplicate using a radioimmunoassay (Linco Research, St Charles, MO, USA).

To minimise interassay variation, the samples from each pair of subjects were assayed in the same batch. We have shown previously that this assay has an intra-assay coefficient of variation of 8.8%. Plasma glucose, total cholesterol, HDL-cholesterol, and triglyceride levels were measured using an Aeroset analyser (Abbott Laboratories, Abbott Park, IL, USA); plasma insulin by an electrochemiluminescent immunoassay (Roche Diagnostics, Indianapolis, IN, USA); and HbA1c by a DCA 2000 analyser (Bayer, Tarrytown, NY, USA). Plasma LDL-cholesterol levels were calculated by the Friedewald equation, and insulin sensitivity assessed using the HOMA%S index.

Statistical analysis—Descriptive statistics were calculated to describe the clinical and biochemical variables. As many of the variables had a non-Gaussian distribution the data were expressed as median and inter-quartile range. Comparison of the two paired ethnic groups was carried out using either paired tests for normally distributed data (age, glucose, HbA1c and lipid fractions) or the Wilcoxon signed rank test for data with a skewed distribution (adiponectin, anthropometric indices, insulin and HOMA%S). McNemar’s Chi-squared test was used to compare proportionality in categorical variables between the two ethnic groups.

The associations between adiponectin and other variables were investigated using Spearman’s rank correlation coefficient. Stepwise multiple regression analysis was then carried out, using plasma adiponectin level as the dependent variable and variables showing a significant association (p<0.10) in the univariate correlation analysis and also HbA1c as the independent variables.

To examine the influence of smoking habits on plasma adiponectin levels, a further regression analysis of the combined data was carried out using smoking habits, ethnicity, and gender as the independent
variables. All the statistical analyses were carried out using Statistix (Analytical Software, Tallahassee, FL, USA), with significance being inferred when p < 0.05.

**Results**

**Clinical and biochemical characteristics of the two ethnic groups**—The data of the two ethnic groups are summarised in Table 1. The median values and interquartile distribution of the majority of variables were similar in the two ethnic groups, with the exception of a small but significant increase in HbA1C levels in the Māori group, and marginally higher levels of total cholesterol and triglyceride in the Caucasian group.

**Table 1. Clinical and biochemical characteristics of the two study groups. The data are expressed as either median and interquartile range or percentages**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Maori (n=111)</th>
<th>Caucasian (n=111)</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender M/F</td>
<td>38 / 73</td>
<td>38 / 73</td>
<td></td>
</tr>
<tr>
<td>2Age (yr)</td>
<td>41.5</td>
<td>33-54</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>30.7</td>
<td>26.6-35.1</td>
<td>0.57</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>96</td>
<td>85-107</td>
<td>0.51</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>41</td>
<td>33-55</td>
<td>0.07</td>
</tr>
<tr>
<td>Biochemical parameters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3Fasting glucose (mmol/L)</td>
<td>5.1</td>
<td>4.8-5.4</td>
<td>0.16</td>
</tr>
<tr>
<td>4Fasting insulin (pmol/L)</td>
<td>52</td>
<td>31-78</td>
<td>0.16</td>
</tr>
<tr>
<td>5HbA1C (%)</td>
<td>5.6</td>
<td>5.3-5.8</td>
<td>0.003</td>
</tr>
<tr>
<td>6Insulin resistance (HOMA%S)</td>
<td>90</td>
<td>57-147</td>
<td>0.18</td>
</tr>
<tr>
<td>7Total cholesterol (mmol/L)</td>
<td>5.2</td>
<td>4.6-6.0</td>
<td>0.05</td>
</tr>
<tr>
<td>8HDL-cholesterol (mmol/L)</td>
<td>1.36</td>
<td>1.14-1.53</td>
<td>0.42</td>
</tr>
<tr>
<td>9Triglyceride (mmol/L)</td>
<td>1.4</td>
<td>0.9-2.0</td>
<td>0.11</td>
</tr>
<tr>
<td>5Proportion of subjects with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 ATP III criteria</td>
<td>23%</td>
<td>17%</td>
<td>0.58</td>
</tr>
<tr>
<td>1-2 ATP III criteria</td>
<td>53%</td>
<td>59%</td>
<td></td>
</tr>
<tr>
<td>3-5 ATP III criteria</td>
<td>24%</td>
<td>24%</td>
<td></td>
</tr>
<tr>
<td>4Hypertension</td>
<td>34%</td>
<td>43%</td>
<td>0.24</td>
</tr>
<tr>
<td>5Dyslipidaemia</td>
<td>18%</td>
<td>33%</td>
<td>0.02</td>
</tr>
<tr>
<td>6Impaired fasting glucose</td>
<td>4%</td>
<td>3%</td>
<td>0.71</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>69%</td>
<td>91%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Currently smoking</td>
<td>31%</td>
<td>9%</td>
<td></td>
</tr>
</tbody>
</table>

a P value for comparison between Māori and Caucasian groups.
b Analysed using paired t tests.
c Analysed using the Wilcoxon signed rank test.
d Analysed using McNemar’s Chi-squared test.
e Hypertension defined as mean arterial pressure >100mmHg or on anti-hypertensive medication.
f Dyslipidaemia defined as cholesterol:HDL-cholesterol ratio >5.0 and plasma triglyceride >1.7mmol/L or taking lipid-lowering agents.
g Impaired fasting glucose defined as fasting glucose level ≥6.1 – <7.0 mmol/L.²¹

Only a small number of the subjects had IFG (6.1–7.0 mol/L; 4 Māori and 3 Caucasians). The same proportion (24%) of subjects in each ethnic group were classified as having the metabolic syndrome, defined as three or more NCEP-ATPIII criteria. A greater proportion of the Caucasian group had hypertension or...
dyslipidaemia than the Māori group, whereas the proportion of current cigarette smokers was 3.5 times higher in the Māori group. The differences in prevalence of dyslipidaemia and cigarette smoking between the two groups were statistically significant.

Plasma adiponectin levels in the two ethnic groups—Figure 1 shows the distribution of plasma adiponectin levels in the two ethnic groups. The mean, median and upper and lower quartiles of plasma adiponectin levels were all higher in the Caucasian group, with a difference in the means between the two groups of 1.0 μg/ml (Caucasians 8.32±SD 4.15 μg/ml vs Māori 7.32±SD 4.02 μg/ml; p=0.03).

Figure 1. Scattergrams of plasma adiponectin levels in the Māori and Caucasian study groups—matched for age, gender, body size and shape, and insulin sensitivity. (The mean, median, and quartile adiponectin levels in the two ethnic groups are shown)

A greater proportion of Māori subjects had a plasma adiponectin level <4.0 μg/ml, (Māori 18.9% vs Caucasian 9.0%; p=0.06), a level that represents the lower limit of the normal range established in our laboratory (Unpublished data). A small proportion of subjects in both groups had raised adiponectin levels >12 μg/ml (Māori 8.1% vs Caucasian 11.7%; p=0.37).
As expected, the median and interquartile range of plasma adiponectin levels were significantly higher in females subjects in both ethnic groups compared with their male counterparts (Māori; females 7.3 (5.2-11.3) µg/ml vs males 4.6 (3.3–6.7) µg/ml, p<0.001: Caucasian; females 8.1 (6.5–10.7) µg/ml vs males 6.8 (4.2–8.2) µg/ml, p=0.002).

Cigarette smoking was associated with a trend of lower median adiponectin levels in both ethnic groups, but in neither instance was this reduction statistically significant (Māori; smokers 6.4 µg/ml vs non-smokers 7.2 µg/ml, p=0.54: Caucasian; smokers 6.5 µg/ml vs non-smokers 7.3 µg/ml, p=0.62).

Relationship between plasma adiponectin levels and other clinical and biochemical parameters—The correlations between adiponectin and the other clinical and biochemical variables in the two groups are summarised in Table 2. As expected, in both ethnic groups low levels of plasma adiponectin were associated with increases in BMI, waist circumference, fasting glucose and triglyceride levels, and number of NCEP-ATPIII criteria, while high levels were associated with greater insulin sensitivity and raised HDL-cholesterol levels.

Surprisingly, there was no relationship between adiponectin levels and percentage body fat in either group. There was also no relationship (in either group) between adiponectin levels and age, HbA1C, total cholesterol levels, or smoking habits.

Step-wise multiple regression analysis showed that the significant independent determinants of plasma adiponectin levels in the Māori group were gender and BMI (both p<0.001), with these two variables accounting for 24% of the variation in adiponectin levels.

In the Caucasian group, the significant independent determinants were HDL-cholesterol (p<0.001) and glucose levels (p=0.001), accounting for 30% of the observed variation. Linear regression analysis of the combined data, using gender, ethnicity, and smoking habits as the independent variables, confirmed that both gender (p<0.001) and ethnicity (p=0.05) were significant determinants of plasma adiponectin levels, whereas smoking habits were not (p=0.26).

Table 2. Correlation between plasma adiponectin and clinical and biochemical parameters in the two ethnic groups.

<table>
<thead>
<tr>
<th>Correlation with adiponectin</th>
<th>Spearman’s correlation coefficient (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Māori</td>
</tr>
<tr>
<td>Age</td>
<td>0.12</td>
</tr>
<tr>
<td>BMI</td>
<td>-0.37 ***</td>
</tr>
<tr>
<td>Waist</td>
<td>-0.42 ***</td>
</tr>
<tr>
<td>% Body fat</td>
<td>0.02</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>-0.21 *</td>
</tr>
<tr>
<td>HbA1C</td>
<td>-0.04</td>
</tr>
<tr>
<td>Insulin resistance (HOMA%S)</td>
<td>0.33 ***</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>-0.14</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>0.34 **</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>-0.25 *</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>-0.24 *</td>
</tr>
<tr>
<td>Number of ATP III criteria</td>
<td>-0.28 **</td>
</tr>
<tr>
<td>Smoking habits</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

Significance of Spearman’s rank correlation coefficient: * p≤0.05; ** p≤0.01; *** p≤0.001.
Discussion

The results of this study demonstrate that New Zealand Māori people tend to have lower levels of plasma adiponectin than their Caucasian counterparts, matched for gender, age, body size, and shape and insulin sensitivity. Reduced production of adiponectin, especially the high molecular weight polymeric form of the compound, occurs during the development of the metabolic syndrome, with low plasma levels known to predispose to the development of Type 2 diabetes. Our results suggest that inherently low adiponectin levels may be a factor contributing to the increased prevalence of Type 2 diabetes in the Māori population, although further more comprehensive and long-term prospective studies are required to confirm this possibility.

The mechanism underlying the reduced adiponectin levels we observed in our Māori study group remains unknown and is likely to be multifactorial. In their comparative study on Pima Indians and Caucasians, Weyer and coworkers found that ethnic differences in adiponectin levels could be almost completely explained by variations in insulin sensitivity, but not by differences in adiposity. In contrast, Valsamakis et al reported that differences in adiponectin levels between Indo-Asian and Caucasian subjects was attributable primarily to differences in central obesity and HDL-cholesterol levels.

To negate the affect of these factors, the subjects in our study were matched for BMI, waist circumference, and degree of insulin sensitivity. Our finding of an ethnic difference in adiponectin levels was therefore more likely to be attributable to either differences in body fat distribution or ethnic variability in the control of adiponectin synthesis and complex formation.

While percentage body fat was marginally higher in the Māori group (and therefore may have contributed to lower adiponectin levels), the lack of correlation we observed between adiponectin and percentage body fat does not support this possibility.

With regard to variability in the genetic control of adiponectin production, it has been shown that adiponectin level is a highly heritable trait in humans, with several quantitative trait loci for adiponectin being identified in the three exons of the APM1 gene. Approximately 16 single nucleotide polymorphisms (SNPs) have been described in the APM1 gene, although studies in different ethnic groups including Japanese, French, Italians and Germans have shown marked inconsistencies between these SNPs and adiponectin levels. Further studies on the role of these frequent genetic variants in the control of adiponectin production are therefore required, and to this end, we are examining the linkage between haplotypes of these SNPs and plasma adiponectin levels and metabolic syndrome characteristics in both the Māori and Caucasian study groups.

Recent evidence also suggests that ethnic variations in proportionality between high and low molecular weight complexes of adiponectin may contribute to the increased
risk of diabetes in certain populations, with low serum levels of the high molecular weight being associated with an increased risk of Type 2 diabetes.\textsuperscript{31}

Several studies have also shown that smokers have low plasma adiponectin levels,\textsuperscript{32,33} and as the incidence of current smokers was markedly higher in our Māori study group it is possible that smoking may have contributed to the reduction in adiponectin levels.

Although our analyses showed that adiponectin levels were marginally lower in smokers in both ethnic groups, we were unable to demonstrate any significant independent relationship between smoking habits and adiponectin levels in our correlation and regression analyses. However, given the high incidence of smoking in Māori,\textsuperscript{34} we consider further studies on the potentially adverse effects of smoking on the production of adiponectin and related cytokines are warranted in this population.

Our study also raises the question as to whether or not the lower plasma adiponectin levels observed in the Māori study group represents a physiologically significant reduction. The relative importance of this reduction can be assessed by comparison with the reference change value (RCV). This value defines the change in an analyte in two consecutive samples that is greater than normal biological variability.\textsuperscript{35} This variability comprises both within-subject and within-assay variation.

In an earlier study using a nested analysis of variance, we found the RCV for plasma adiponectin was 1.7 µg/ml for overweight subjects with the metabolic syndrome and approximately two-fold higher at 3.6 µg/ml in healthy, lean controls.\textsuperscript{14} These values are higher than the mean difference of 1.0 µg/ml we observed between the Māori and Caucasian groups, the majority of whom were moderately overweight.

To put this in context, the mean difference reported by Weyer et al\textsuperscript{8} in their comparative study on Pima Indians was 3.0 µg/ml, while the difference in median values between Caucasian and Indo-Asian subjects reported by Valsamakis and coworkers\textsuperscript{18} was 1.6 µg/ml. The difference in adiponectin levels we observed in our study was therefore less than the RCV and smaller than the differences reported in similar ethnic studies.

The study had several limitations. The first of these was that approximately one-sixth of the Caucasian group were recruited from a Lipid Disorders outpatient clinic, whereas all the subjects in the Māori group were recruited from the general population. While it is possible this difference in recruitment strategies may have influenced our findings, it would be anticipated that the increased prevalence of dyslipidaemia found in the Caucasian group would be associated with lower adiponectin levels, thereby minimizing any difference in adiponectin levels observed between the two groups.

The second limitation was the finding of a significant difference in baseline HbA\textsubscript{1C} levels between the two groups. However, as there was no correlation between adiponectin and HbA\textsubscript{1C} levels, small differences in HbA\textsubscript{1C} levels would be expected to have only minimal influence on a multivariate regression analysis.

Thirdly, given the high fasting glucose levels in a small of the study participants, it is possible that some of these individuals may have been diagnosed with diabetes on the basis of an oral glucose tolerance test (OGTT). For logistical reasons in our study, we
used the ADA diagnostic criteria of a fasting glucose level >7.0 mmol/L to diagnose diabetes.  

In conclusion, this study demonstrated that Māori people tend to have lower plasma adiponectin levels than Caucasian people of similar age, body shape, and insulin sensitivity.

While the magnitude of this reduction in adiponectin levels was relatively small and less than normal biological variability, further long-term prospective studies are required to determine the aetiological significance of this ethnic difference in the development of insulin resistance and Type 2 diabetes.

**Competing interests:** None.

**Author information:** Brett Shand, Scientist¹, Peter Elder, Steroid Chemist², Russell Scott, Physician¹, Nicola Poa, Researcher³, Christopher M Frampton, Biostatistician⁴

1. Lipid and Diabetes Research Group, Christchurch Hospital, Christchurch  
2. Canterbury Health Laboratories, Christchurch  
3. Molecular Psychiatry Research Group, Christchurch  
4. Department of Medicine, Christchurch Hospital, Christchurch

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**Correspondence:** Dr Brett Shand, Scientist, Lipid and Diabetes Research Group, Hagley Building, Christchurch Hospital, Christchurch. Fax:(03) 364 0457; email: brett.shand@cdhb.govt.nz

**References:**


Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance, and impaired fasting glucose. Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand

Gerhard Sundborn, Patricia Metcalf, Robert Scragg, David Schaaf, Lorna Dyall, Dudley Gentles, Peter Black, Rodney Jackson

Abstract

Aim To estimate the prevalence of new and known diabetes mellitus, impaired glucose tolerance (IGT), and impaired fasting glucose (IFG) by ethnic group in Auckland.

Methods The Diabetes Heart and Health Survey (DHAH) was a cross-sectional population based survey and was carried out in Auckland between January 2002 and December 2003, inclusive. Participants answered a self-administered questionnaire to assess whether they had previously diagnosed diabetes. Those participants who were not previously diagnosed with diabetes were then given a glucose tolerance test (GTT) to determine diabetes status.

Results Of the total sample 6.7% were previously diagnosed (known) with type 2 diabetes, and a further 2.6% were newly diagnosed. Within the ethnic groups Europeans had the lowest level of both new and known diabetes followed by Māori and then Pacific people (mostly of Samoan, Tongan, Niuean, or Cook Islands origin). The proportions of new/known diabetes by ethnicity were 1.8%/3.9% for Europeans, 3.8%/12.0% for Māori, and 4.0%/19.5% for Pacific. Only Pacific were found to have a significantly greater relative risk (RR) than Europeans of being newly diagnosed with diabetes, particularly in the <45 (RR 11.6), and 45–54 year (RR 4.2) age groups. Compared to Europeans, Māori had a significantly greater risk of known diabetes in the 45–54 (RR 6.4) and 55–64 (RR 4.1) year age groups, while Pacific had a significantly greater risk in all age groups which ranged from RR 2.5 in those aged 65+ to RR 9.3 in the 55–64 year age group. For Europeans and Māori, the greatest proportions of diabetes occurred in the 65+ year age group, however for Pacific this occurred in the 55-64 year age group. IFG levels were only found to be significantly different from Europeans in Māori aged 45–54, and Pacific aged 45–54 and <45 years. IGT levels were only found to be significantly different from Europeans in Pacific aged 45–54 years.

Conclusions The prevalence of diabetes was 2.8 times greater for Māori, and 4.1 times greater for Pacific compared with Europeans. However for every two European people with previously diagnosed diabetes there was approximately one (0.92) person in the community undiagnosed while for every three Māori people with diagnosed diabetes was one Māori person undiagnosed. For every five Pacific with diagnosed diabetes there was just over one (1.1) Pacific person undiagnosed.
In 2003, one in 23 New Zealand (NZ) adults had self-reported diabetes mellitus.\textsuperscript{1} The prevalence of diabetes was significantly higher in Māori (males 6.9\%, females 5.1\%) and Pacific (males 8.0\%, females 12.0\%) populations [mostly of Samoan, Tongan, Niuean, or Cook Islands origin; hereafter termed ‘Pacific’] than in NZ Europeans (males 2.6\%, females 2.1\%).\textsuperscript{1, 2} It has been suggested that for every person diagnosed with diabetes there is another person in the community undiagnosed.\textsuperscript{3}

In November 2005, approximately 125,000 people had diagnosed diabetes; therefore according to this prediction the true diabetic population in NZ exceeds 250,000 people. It is predicted that more than 7,500 new people will be diagnosed with diabetes in 2006 and that more than 1,700 deaths will be attributable to diabetes.\textsuperscript{4}

For Māori and Pacific, diabetes related-mortality rates are 10 times higher than for European people.\textsuperscript{5} A recent review of the epidemiology of diabetes in NZ lists the prevalence of known and undiagnosed diabetes from various NZ surveys and has called for a nationally agreed strategic plan on how to best monitor and control diabetes.\textsuperscript{6}

Previous New Zealand studies and surveys that have been used to estimate the prevalence of diabetes have used self-report data or have been workforce surveys\textsuperscript{1, 7-9} or are now more than a decade old.\textsuperscript{10} Self-report surveys are likely to underestimate diabetes prevalence as those who have diabetes and are yet to be diagnosed will be missed (under-reporting) and workforce surveys will be biased due to the ‘healthy worker’ effect. Moreover the majority of these surveys included insufficient samples of Māori and Pacific to allow for meaningful ethnic comparisons.

The South Auckland diabetes project (1991-4) is a useful comparison for this study as it was also a population based survey and had good numbers of both Māori and Pacific.\textsuperscript{11}

The Diabetes Heart and Health (DHAH) survey has attempted to overcome these problems by using a population-based study design that included Glucose Tolerance Tests (GTT) for all non-diabetic participants. Targeted sampling of Māori and Pacific was undertaken to generate large representative samples of these communities.

The purpose of this study is to describe and compare ethnic differences in the prevalence of new and known diabetes mellitus, and impaired glucose tolerance and impaired fasting glucose levels.

**Methods**

The DHAH survey was a cross-sectional study that surveyed people aged 35–74 years, between January 2002 and December 2003. All participants were selected from within the Auckland region. Of the 4049 participants, 1014 were Māori, 1011 were Pacific, and 1745 were of European ethnicity.

Adults were recruited using two sampling frames: one was a cluster sample where random starting point addresses were obtained from Statistics New Zealand and the probability of selection was proportional to the number of people living in that mesh block (response rate 61.3\%); and the other was a random sample taken from the November 2000 Auckland electoral rolls stratified into 5-year age bands and included all people living in the Auckland area, with the exception of the Franklin and Rodney electorates (response rate 65\%). Participants were interviewed in places close to where they lived and all completed a self-administered questionnaire and a series of health measures. The 19 people who refused to have a GTT were excluded. Asians were also excluded.

Classification of ethnicity first gave priority to Māori ethnicity and followed an ‘ever-Māori’ approach used to improve undercounts in health data sets\textsuperscript{12}, followed by Pacific and Asian while all other
participants formed the European comparison group, as used by Statistics New Zealand. Ethical approval was obtained from the Health and Disability Ethics Committees.

All participants received information in the mail with instructions not to eat any food from 10pm onwards the night before their survey was scheduled and to drink water only. Included in the information pack was a sterile urine container that was used to collect an early-morning urine sample (midstream). Most participants were contacted by phone prior to their survey appointment where instructions were explained again and any queries answered. On arrival at the survey location (from 8am to 10am) and after initial consent was given, a fasting blood sample was taken from all participants.

Participants were then asked whether they had been diagnosed with diabetes, and if so how old they were when they were first told, and what their current treatment was. Those who did not have previously diagnosed diabetes mellitus were then asked to complete a 2 hour Glucose Tolerance Test (GTT). This involved them having a drink consisting of 75g glucose after their initial blood test. A final blood test was then scheduled to be taken 2 hours after the first. During the wait-time, participants were asked to fill in other survey questionnaires and to not physically exert themselves in any way or consume any food or drink with the exception of water. Glucose samples were collected into fluoride tubes and stored on ice until taken to the lab for analyses.

Fasting blood samples were assayed using enzymatic methods, plasma glucose was measured using commercial reagents (Roche Products [NZ]), HbA1c was measured by high performance liquid chromatography, and micro-albumin was measured using an immunoturbidmetric method.

Categorisation of glucose tolerance status was evaluated by 1998 WHO criteria using fasting glucose ≥ 7.0 mmol/L or 2-hour post glucose load of ≥ 11.1 mmol/L for diabetes; fasting glucose < 7.0 mmol/L and 2-hour glucose between 7.8 and 11.0 mmol/L for Impaired Glucose Tolerance (IGT) and fasting glucose of 6.1-6.9 mmol/L for Impaired Fasting Glucose (IFG). All participants were then classified as ‘known’ (from their past history), ‘new-ly diagnosed, having ‘IGT’ or ‘IFG’ or ‘normal’ glucose functioning.

Leisure exercise was assessed using a three-month physical activity recall questionnaire. One question asked if participants had engaged in any vigorous activity at least once a week, in the past three months, long enough, that caused them to breathe hard or sweat. The other question asked if they had engaged in any moderate activity (that did not cause them to breathe hard or sweat).

Statistical analysis was undertaken using SAS version 9.1. Participant data were weighted according to the sampling frame that they were obtained from and means, standard errors and prevalence’s calculated using dual frame sampling methodology. SAS survey procedures (SURVEYMEANS, SURVEYREG, SURVEYFREQ AND SURVEYLOGISTIC) were used to calculate weighted means, adjusted means, percentages and odds ratios, respectively. The Rao-Scott modified Pearson Chi-squared test was used where appropriate with the reference category being the Europeans, because they constituted the largest sample. Odds Ratios were converted to Relative Risks as described by Zhang and Yu.

Results

The demographic characteristics are shown in Table 1. For the total study population, 48.0% were male; 27.0% aged <45 years, 26.4% aged 45–54 years, 24.4% aged 55–64 years, and 22.3% aged 65+ years; 26.9% of the participants were Māori, 26.8% were Pacific, and 46.3% were of European ethnicity.
Table 1. Characteristics of the study sample

<table>
<thead>
<tr>
<th>Variables</th>
<th>Age range</th>
<th>European</th>
<th>Māori</th>
<th>Pacific†</th>
<th>European</th>
<th>Māori</th>
<th>Pacific†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>&lt; 45</td>
<td>193</td>
<td>106</td>
<td>148</td>
<td>196</td>
<td>163</td>
<td>188</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>211</td>
<td>113</td>
<td>133</td>
<td>212</td>
<td>166</td>
<td>147</td>
</tr>
<tr>
<td></td>
<td>55-64</td>
<td>219</td>
<td>115</td>
<td>119</td>
<td>236</td>
<td>130</td>
<td>110</td>
</tr>
<tr>
<td></td>
<td>65+</td>
<td>240</td>
<td>112</td>
<td>87</td>
<td>238</td>
<td>109</td>
<td>79</td>
</tr>
<tr>
<td>BMI*</td>
<td>&lt; 45</td>
<td>27.4 (26.8-27.9)</td>
<td>31.1 (29.8-32.4)</td>
<td>33.7 (32.5-34.9)</td>
<td>26.8 (26.0-27.6)</td>
<td>30.2 (28.8-31.7)</td>
<td>35.6 (34.5-36.6)</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>27.9 (27.2-28.6)</td>
<td>30.9 (29.1-32.6)</td>
<td>33.2 (31.7-34.7)</td>
<td>27.1 (26.4-27.8)</td>
<td>30.4 (28.2-32.7)</td>
<td>36.3 (34.5-38.0)</td>
</tr>
<tr>
<td></td>
<td>55-64</td>
<td>27.5 (27.0-28.0)</td>
<td>29.0 (28.3-29.8)</td>
<td>32.8 (31.6-34.1)</td>
<td>27.8 (27.1-28.5)</td>
<td>32.9 (30.8-35.1)</td>
<td>35.9 (33.5-38.3)</td>
</tr>
<tr>
<td></td>
<td>65+</td>
<td>27.5 (26.9-28.1)</td>
<td>29.9 (28.9-30.9)</td>
<td>31.0 (29.3-32.7)</td>
<td>27.8 (27.0-28.6)</td>
<td>28.9 (27.8-29.9)</td>
<td>33.7 (32.6-34.9)</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>&lt; 45</td>
<td>96.2 (94.7-97.6)</td>
<td>101.4 (98.2-104.5)</td>
<td>105.4 (102.8-108.8)</td>
<td>85.0 (83.2-86.8)</td>
<td>92.1 (89.1-95.1)</td>
<td>101.7 (99.3-104.1)</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>98.4 (96.8-99.9)</td>
<td>102.7 (99.3-106.1)</td>
<td>106.4 (103.0-109.8)</td>
<td>86.2 (84.5-87.9)</td>
<td>92.0 (88.1-95.9)</td>
<td>105.2 (102.4-107.9)</td>
</tr>
<tr>
<td></td>
<td>55-64</td>
<td>98.0 (96.7-99.3)</td>
<td>99.4 (97.5-101.3)</td>
<td>106.9 (103.7-110.1)</td>
<td>89.1 (87.5-90.8)</td>
<td>100.3 (94.5-106.1)</td>
<td>105.1 (103.0-107.2)</td>
</tr>
<tr>
<td></td>
<td>65+</td>
<td>99.5 (98.0-100.9)</td>
<td>103.2 (101.0-105.3)</td>
<td>105.8 (99.9-111.7)</td>
<td>90.7 (88.8-92.6)</td>
<td>90.4 (88.1-92.6)</td>
<td>108.2 (101.8-114.5)</td>
</tr>
<tr>
<td>Total number</td>
<td>863</td>
<td>446</td>
<td>487</td>
<td>882</td>
<td>568</td>
<td>524</td>
<td></td>
</tr>
</tbody>
</table>

*Body mass index = weight (kg$^2$) ÷ height$^2$ (m$^2$); †Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.
The proportions of impaired fasting glucose (IFG), impaired glucose tolerance (IGT), newly diagnosed, previously diagnosed (known), and total diabetes are shown in Figure 1. Europeans had the lowest proportions, Pacific had the highest, and Māori were intermediate for prevalence of all diabetes states. Māori had 2.8 times higher and Pacific 4.1 times higher prevalence of total diabetes mellitus compared to Europeans.

Figure 1. Prevalence of diabetes states by ethnicity adjusted for age and sex

Lifestyle, socioeconomic status and demographic characteristics by diabetes status are presented in Table 2. These proportions have been adjusted for age, sex, and ethnicity. Those with abnormal diabetes status generally had significantly higher BMI, were older and exercised less. Compared to those with new or known diabetes and IGT categories, the IFG subgroup was not as distinctly differentiated from the ‘normal’ group.
Table 2. Percentage and mean demographic characteristics by diabetes status; adjusted for age, sex, and ethnicity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal 80.9%</th>
<th>IFG 7.1%</th>
<th>IGT 2.7%</th>
<th>New diabetes 2.6%</th>
<th>Known diabetes 6.7%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male†</td>
<td>52.0%</td>
<td>42.8%</td>
<td>42.4%*</td>
<td>49.3%</td>
<td>54.0%</td>
</tr>
<tr>
<td>Smoker</td>
<td>15.2%</td>
<td>21.1%</td>
<td>15.2%</td>
<td>15.9%</td>
<td>19.5%</td>
</tr>
<tr>
<td>Mod-ex</td>
<td>68.3%</td>
<td>65.8%</td>
<td>62.0%</td>
<td>47.8%**</td>
<td>61.5%</td>
</tr>
<tr>
<td>Vig–ex</td>
<td>27.5%</td>
<td>22.3%</td>
<td>10.9%***</td>
<td>10.9%**</td>
<td>15.6%**</td>
</tr>
<tr>
<td>Good† health</td>
<td>88.8%</td>
<td>86.4%</td>
<td>80.5%***</td>
<td>79.5%</td>
<td>75.7%***</td>
</tr>
<tr>
<td>Education</td>
<td>64.0%</td>
<td>52.9%</td>
<td>57.2%</td>
<td>52.2%</td>
<td>57.2%</td>
</tr>
<tr>
<td>Age (years)‡</td>
<td>49.6</td>
<td>52.7**</td>
<td>56.5***</td>
<td>54.1***</td>
<td>57.3***</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.6</td>
<td>29.8***</td>
<td>30.5***</td>
<td>30.4***</td>
<td>30.2***</td>
</tr>
</tbody>
</table>

*0.01 <p< 0.05; ** 0.001<p<0.01; *** p<0.001 compared to ‘Normal’ group; †Not adjusted for sex; ‡Not adjusted for age; IGT: impaired glucose tolerance; IFG: impaired fasting glucose; **Mod-ex: Participated in moderate exercise at least 1 × per week in past 3 months; Vig–ex: Participated in vigorous exercise at least 1 x per week in past 3 months.; Good† health: Rated personal health as good or better.

Table 3 compares the risk of having newly diagnosed or known diabetes by ethnic group. Māori aged 45–54 and 55–64 years were found to have significantly higher risk of known diabetes compared to Europeans (RR: 6.4 and 4.1 respectively). For Pacific, all age groups had a significantly higher risk of known diabetes than Europeans, with the highest being in the 55–64 year age group (RR: 9.3).

Only Pacific participants in the <45, and 45–55 year age groups were found to have a significantly higher risk of new diabetes status (RR: 11.6 and 4.2 respectively) compared to Europeans.

Table 3. Relative risk (RR) of new and known diabetes by ethnic group, adjusted for sex

<table>
<thead>
<tr>
<th>Age group</th>
<th>Diabetes</th>
<th>European RR (%)</th>
<th>Māori RR (95% CI) (%)</th>
<th>Pacific RR (95% CI) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;45 years</td>
<td>New</td>
<td>1.0 (0.2)</td>
<td>4.97 (0.48-47.36)</td>
<td>11.61 (1.43-82.28)*</td>
</tr>
<tr>
<td></td>
<td>Known</td>
<td>1.0 (1.4)</td>
<td>2.60 (0.89-7.27)</td>
<td>5.88 (2.02-15.48)**</td>
</tr>
<tr>
<td>45–54 years</td>
<td>New</td>
<td>1.0 (2.2)</td>
<td>1.38 (0.52-3.56)</td>
<td>4.16 (1.87-8.68)***</td>
</tr>
<tr>
<td></td>
<td>Known</td>
<td>1.0 (2.5)</td>
<td>6.37 (2.88-12.66)***</td>
<td>7.01 (3.38-13.13)***</td>
</tr>
<tr>
<td>55–64 years</td>
<td>New</td>
<td>1.0 (2.7)</td>
<td>2.82 (0.68-9.93)</td>
<td>2.19 (0.90-5.07)</td>
</tr>
<tr>
<td></td>
<td>Known</td>
<td>1.0 (4.4)</td>
<td>4.12 (2.18-7.16)***</td>
<td>9.33 (5.73-13.41)***</td>
</tr>
<tr>
<td>65+ years</td>
<td>New</td>
<td>1.0 (3.5)</td>
<td>2.01 (0.93-4.16)</td>
<td>0.99 (0.34-2.75)</td>
</tr>
<tr>
<td></td>
<td>Known</td>
<td>1.0 (12.5)</td>
<td>1.55 (0.97-2.37)</td>
<td>2.46 (1.18-4.26)*</td>
</tr>
</tbody>
</table>

*0.01 <p< 0.05; **0.001<p<0.01; ***p<0.001 compared to Europeans.
The highest proportion of new diabetes was observed in the Pacific 45-54 age group of 6.9% (Europeans: 2.2%, Māori: 2.5%). The largest proportion of previously diagnosed (known) diabetes was also reported by the Pacific ethnic group, aged 55-64 of 38.2%. This is compared to Europeans 4.4%, and Māori 17.4%.

The only significant difference in IGT was observed in the Pacific ethnic group aged 45-54 years compared to the Europeans. For IFG the only significant differences were observed in Māori aged 45-54, and Pacific aged <45 and 45-54 years. Generally a clear trend was observed where Pacific had the highest risk and Māori intermediate for both IGT and IFG compared to Europeans. However two exceptions to this trend existed. The 55-64 IFG Māori group and the 65+ IFG/IGT Pacific groups reported lower (but not significantly different) risks compared to Europeans.

### Table 4. Relative risk (RR) of impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) by age group and ethnicity, adjusted for sex

<table>
<thead>
<tr>
<th>Age group</th>
<th>European RR (%)</th>
<th>Māori RR (95% CI) (%)</th>
<th>Pacific RR (95% CI) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;45 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IGT</td>
<td>1.0 (3.4)</td>
<td>1.40 (0.64-2.95) (4.6)</td>
<td>1.49 (0.71-3.01) (4.5)</td>
</tr>
<tr>
<td>IFG</td>
<td>1.0 (1.0)</td>
<td>1.96 (0.47-7.87) (1.8)</td>
<td>4.24 (1.16-14.32)* (3.7)</td>
</tr>
<tr>
<td>45–54 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IGT</td>
<td>1.0 (6.0)</td>
<td>1.18 (0.65-2.11) (5.9)</td>
<td>2.27 (1.26-3.88)** (10.5)</td>
</tr>
<tr>
<td>IFG</td>
<td>1.0 (1.8)</td>
<td>3.18 (1.03-9.06)* (4.9)</td>
<td>4.31 (1.36-12.22)* (6.0)</td>
</tr>
<tr>
<td>55–64 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IGT</td>
<td>1.0 (7.5)</td>
<td>1.40 (0.83-2.30) (8.7)</td>
<td>1.58 (0.83-2.86) (7.5)</td>
</tr>
<tr>
<td>IFG</td>
<td>1.0 (3.8)</td>
<td>0.55 (0.20-1.50) (1.7)</td>
<td>1.56 (0.64-3.61) (3.6)</td>
</tr>
<tr>
<td>65+ years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IGT</td>
<td>1.0 (15.8)</td>
<td>1.04 (0.64-1.63) (14.4)</td>
<td>0.76 (0.19-2.33) (9.6)</td>
</tr>
<tr>
<td>IFG</td>
<td>1.0 (3.4)</td>
<td>1.43 (0.60-3.25) (4.0)</td>
<td>0.58 (0.18-1.81) (1.6)</td>
</tr>
</tbody>
</table>

*0.01 <p< 0.05; ** 0.001<p<0.01; *** p<0.001 compared to Europeans.

Figure 2 shows the cumulative proportion of newly diagnosed diabetes mellitus by age group and ethnicity. This graph shows that Māori follow a similar trend to Europeans in spite of having a higher prevalence of both new and known diabetes mellitus. The Pacific ethnic group however follow a clearly different path. For Pacific, the same proportion of newly diagnosed participants were diagnosed approximately ten years earlier compared to both Māori and Europeans. For example 80% of newly diagnosed Pacific participants had been diagnosed by the 45-54 age group. This occurred closer to the 55-64 age-group for both Māori and Europeans.
Table 5 presents the findings of 2 multivariate models for newly diagnosed diabetes mellitus. Model 1 shows that being Māori or Pacific increases the odds of being newly diagnosed with diabetes mellitus compared with Europeans. However, only Pacific ethnicity was statistically significant. After adjustment for Body Mass Index (BMI) in Model 2 both odds ratios for Māori and Pacific ethnicity dropped making Pacific ethnicity now insignificant.

**Table 5. Multivariate odds ratios (95% CI) for ‘newly’ diagnosed diabetes mellitus**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.05 (1.03-1.07)</td>
<td>1.06 (1.03-1.08)</td>
</tr>
<tr>
<td>Male</td>
<td>1.53 (0.91-2.59)</td>
<td>1.67 (0.99-2.86)</td>
</tr>
<tr>
<td>Māori</td>
<td>2.00 (0.96-4.16)</td>
<td>1.48 (0.63-3.47)</td>
</tr>
<tr>
<td>Pacific</td>
<td>2.98 (1.78-4.97)</td>
<td>1.57 (0.85-2.90)</td>
</tr>
<tr>
<td>BMI</td>
<td>–</td>
<td>1.11 (1.06-1.15)</td>
</tr>
</tbody>
</table>

Model 1 includes age, male, Māori and Pacific; Model 2 includes Model 1 plus BMI.

Table 6 shows 2 multivariate models for previously diagnosed (known) diabetes mellitus. Model 1 show 4 times higher odds for Māori and 6.6 times higher odds for Pacific of previously diagnosed diabetes compared to Europeans. Adjustment for BMI in Model 2 saw a reduction of the odds in both Māori and Pacific compared to Europeans, but did not eliminate ethnic differences in people with previously diagnosed diabetes mellitus.
Table 6. Multivariate odds ratios (95% CI) for ‘known’ diabetes mellitus

<table>
<thead>
<tr>
<th>Variables</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.07 (1.06-1.09)</td>
<td>1.08 (1.06-1.10)</td>
</tr>
<tr>
<td>Male</td>
<td>0.88 (0.64-1.21)</td>
<td>0.99 (0.72-1.36)</td>
</tr>
<tr>
<td>Māori</td>
<td>4.01 (2.62-6.14)</td>
<td>2.94 (1.89-4.58)</td>
</tr>
<tr>
<td>Pacific</td>
<td>6.64 (4.47-9.87)</td>
<td>3.73 (2.38-5.84)</td>
</tr>
<tr>
<td>BMI</td>
<td>–</td>
<td>1.10 (1.07-1.23)</td>
</tr>
</tbody>
</table>

Model 1 includes age, male, Māori and Pacific; Model 2 includes Model 1 plus BMI.

Discussion

It has been frequently reported that between a third and a half of all diabetes in the community remains undiagnosed and that this may be experienced more by Pacific people. The common mantra that ‘for every known case of diabetes there is another undiagnosed in the community’, is not supported by our study and has significant public health implications in estimating the projected burden of undiagnosed diabetes in New Zealand by ethnic group.

In the current study, the proportion of Europeans that were newly diagnosed with diabetes was equal to 46% of those with known diabetes. This suggests that for every two European people with previously diagnosed diabetes there approximately one (0.92) person in the community undiagnosed.

For Māori and Pacific, the proportions that were newly diagnosed with diabetes were equal to 32% and 21% respectively, of those with known diabetes. This suggests that for every three Māori and every five Pacific people with previously diagnosed diabetes there would one person in the community undiagnosed (0.96 for Māori, 1.05 for Pacific).

In contrast, a study that measured new and known diabetes in adults aged 40–70 years in South Auckland during 1991-94, found that the proportion of new diabetes in Europeans was equal to 52% of known. For Māori and Pacific, these proportions were 77% and 81% respectively. These findings suggest that diabetes screening for Māori and Pacific have improved considerably over the past decade.

Prevalences of previously diagnosed diabetes found in this study had similar ethnic patterns to those reported in the 2002/03 NZ Health Survey. However the self-reported prevalences of previously diagnosed diabetes tended to be considerably lower in the NZ Health Survey. In absolute terms, the DHAH survey prevalences were higher than the NZ Health Survey data by approximately 1.4% for Europeans, 4.0% for Māori, and 9.5% for Pacific ethnic groups. These differences are in part due to the differing age structure of each survey. The NZ Health survey sampled from 15 years and above compared to 35 years for the DHAH. These surveys were both conducted during 2002/03.

In contrast to a cross-sectional survey carried out in South Auckland from 1992-1995 and the Workforce Survey in 1988-1990, Pacific people now have a poorer profile than the Māori population. The South Auckland study reported age adjusted rates of known diabetes of 5.2% for Europeans, 7.3% for Māori and 6.0% for Pacific peoples compared to 3.9%, 12.0%, and 19.5%, respectively, in the DHAH.
For those with previously diagnosed (known) diabetes, Pacific aged 55-64 reported the largest relative risk (RR: 9.33 compared to Europeans), this compares to Pacific aged 50-54 (RR: 11.8) from the Workforce Survey. Prevalences of known diabetes by age group and ethnicity followed expected trends with Pacific people having the highest prevalences and Māori intermediate.

However the patterns in RR were more mixed for new diabetes. Māori had the highest RR for new diabetes in the 55-64 and 65+ age groups with Pacific intermediate. For the <45 and 45-54 age groups Pacific had the highest risk. This could be due to both earlier onset of diabetes in Pacific and less robust screening for diabetes in Māori compared to Pacific in the younger age groups. Risk of new diabetes in Māori ranged from 1.38–4.97 compared to Europeans and for Pacific they ranged from 0.99–11.61. The largest risk for new diabetes was found in the Pacific age group of <45 years (RR: 11.61) which was also the case for the Workforce Survey (RR: 9.5).

The Workforce Survey conducted during 1988-90 reported newly diagnosed diabetes in 1.7% of Europeans, 9.7% Māori, and 7.7% Pacific people. However, these were likely to be lower than the general population as they were employees (healthy worker effect). Prevalences found in the DHAH were 1.8% for Europeans, 3.8% Māori, and 4.0% Pacific.

The marked decrease in Māori of newly diagnosed diabetes is interesting and may be the result of improved health and improved access to healthcare with the emergence of many new Māori healthcare providers over the past two decades, resulting in earlier detection of diabetes. Measurement and classification of Māori ethnicity could also have influenced this difference. This study used an ‘ever-Māori’ approach to assign ethnicity which decreases the likelihood of under-reporting.

A limitation of this study is that using Electoral role based and cluster sampling frames did not allow for ethnic specific response rates to be determined. Although the overall response rate was not as high as in previous Auckland risk factor studies, it has been shown in the Atherosclerosis Risk in Communities Study that response rates lower than those in our study produced relatively small errors in the estimates of prevalence of common cardiovascular disease risk factors.

Participants with known diabetes were more likely to engage in moderate exercise when compared to those newly diagnosed. This suggests that once a diagnosis is made increased physical activity may have been recommended to these people.

Having received further tertiary education had a protective association with diabetes. Levels of tertiary education were lower for all categories of impaired glucose tolerance when compared to the ‘normal’ reference group. (Table 2) This may be due to education leading towards higher socio-economic status, and also an increased awareness of healthy lifestyles, diabetes risk factors and symptoms.

The difference observed in the cumulative proportions of new diabetes between Pacific and non-Pacific ethnic groups (Figure 2) showed that a larger proportion of Pacific people generally experience earlier onset of diabetes. This difference equates to Pacific people being diagnosed up to 10 years earlier than Europeans and suggests that Pacific people will live with diabetes and its complications significantly longer and/or have earlier mortality. This figure could also imply that the Māori population may follow a more similar disease profile/pattern to Europeans than Pacific.
It is important to note that the age at diagnosis is not necessarily the age of
development of diabetes, and that the time between development and diagnosis may
vary between ethnic groups.

BMI and age were found to be the most significant factors associated with people
newly diagnosed with diabetes, IGT, and IFG (Table 2, 5) which supports the focus
that many health campaigns have on prevention and control of obesity to lower the
prevalence of diabetes. Model 2 from Table 5, showed that adjusting for BMI alone
reduced ethnic differences in new diabetes prevalence. However, although adjustment
for BMI in people previously diagnosed with diabetes mellitus reduced the odds in
both Māori and Pacific people compared to Europeans, it did not eliminate these
ethnic differences (Table 6).

Increasing and maintaining health promotion programmes centred on living healthy
lifestyles (nutrition and activity) to keep a healthy BMI will continue to be the most
appropriate method to prevent and manage diabetes in New Zealand.

Competing interests: None.

Author information: Gerhard McDonald-Sundborn, Research Fellow in Pacific Health; Patricia Metcalf, Senior Lecturer in Biostatistics; Robert Scragg, Associate Professor of Epidemiology; David Schaff, Senior Research Fellow in Pacific Health; Lorna Dyall, Senior Lecturer in Māori Health; Dudley Gentes, Research Fellow in Māori Health; Peter Black, Associate Professor of Medicine; Rodney Jackson, Professor of Epidemiology, Section of Epidemiology and Biostatistics, School of Population Health
University of Auckland, Auckland

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Correspondence: Gerhard Sundborn, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Private Bag 92019, Auckland 1.
Fax: (09) 3737 503; email: g.sundborn@auckland.ac.nz

References:


Alcohol assessment: the practice, knowledge, and attitudes of staff working in the general medical wards of a large metropolitan hospital

Justin Pulford, Ross McCormick, Amanda Wheeler, Patrick Firkin, Ian Scott, Gail Robinson

Abstract

Aims To measure the prevalence of routine alcohol assessment; to assess its clinical utility in the general medical wards of a large urban hospital; and to assess medical and nursing staff knowledge with regard to standard drink measures and recommended drinking limits as well as their attitudes towards alcohol assessment.

Methods The prevalence of alcohol assessment and the clinical utility of the resulting information was determined via a retrospective file review (n=109). The knowledge and attitudes of medical and nursing staff were measured via questionnaire (n=106).

Results The file review data indicated 78% (±7.25) of patients admitted to the general medical wards were queried with regard to their alcohol consumption. However, the clinical utility of the recorded information was generally poor and the accuracy questionable. Only 12% of questionnaire respondents were able to accurately identify the standard drink equivalents for beer, wine, and spirits and only 8% were able to accurately identify the recommended drinking limits (per drinking occasion and per week) for both males and females. Attitudes towards alcohol assessment were positive.

Conclusions Patient alcohol consumption is frequently assessed, but the clinical utility of the resulting information is limited. The use of a structured alcohol screen and the provision of appropriate staff training are recommended.

Research indicates that between 20–25% of adult New Zealanders regularly exceed recommended limits for responsible alcohol consumption. The resulting costs to the individual, the individual’s family, and society at large are significant. For example, excessive alcohol consumption is implicated in a range of health problems, fuels domestic violence, and is thought to cost New Zealand over one billion dollars per annum in lost productivity alone.

Specialist alcohol treatment is one means by which these costs may be allayed. Adherence to an evidenced-based, specialist treatment intervention can effectively reduce alcohol consumption and the associated harms. Relatively brief, yet effective, interventions are also available that can be delivered opportunistically in non-specialist settings.

Typically consisting of simple advice regarding safe drinking behaviour, these brief interventions primarily target those people with mild-to-moderate alcohol-related problems who do not require specialist assistance. Nevertheless, a brief intervention may also facilitate entry into a specialist alcohol treatment service if required.
The hospital is an ideal venue for brief intervention. Many people are admitted to hospital for accidents or health problems related to excessive alcohol consumption,\textsuperscript{11,12} and the effectiveness of a brief alcohol intervention has been demonstrated in a range of hospital departments.\textsuperscript{13–15} Furthermore, as excessive alcohol consumption is a legitimate health issue, it is unlikely that persons admitted to a hospital would object to discussing their drinking behaviours.

Given the opportunistic nature of brief intervention, however, successful provision is reliant on an effective alcohol assessment in the first instance. The health professional must elicit information pertaining to the type, quantity, and frequency of an individual’s alcohol consumption. A working knowledge of standard drink measures and recommended drinking limits is also needed in order to inform an accurate assessment.

Little is known about alcohol assessment in New Zealand-based hospitals. National standards for routine alcohol assessment in a hospital setting do not exist, and the prevalence or utility of routine alcohol assessment practice in New Zealand hospitals have remained largely unexamined. It is difficult to determine, therefore, whether current methods of alcohol assessment would usefully support brief intervention.

This paper reviews the prevalence and utility of routine alcohol assessment practice in the general medical wards of a large, metropolitan hospital. The knowledge of ward staff with regard to standard drink measures and recommended drinking limits were assessed as were their attitudes towards working with alcohol affected individuals. All data were collected as a prelude to a larger project that sought to promote brief intervention within the study setting. Our research findings provide insight into hospital-based alcohol assessment and the ability of the latter to support brief intervention initiatives.

**Method**

The study was conducted in the general medical wards of Auckland City Hospital. Auckland City Hospital is the largest hospital in New Zealand and serves a catchment population of 430,000. The hospital contains four general medical wards to which approximately 11,000 patients are admitted per year. Standard practice at the time of this study was to assess alcohol consumption via a specific question included in the ‘social history’ section of the patient admission form. This question was simply worded, “alcohol (amount and duration)” and a space was provided in which to enter the resulting information. The admission form was typically completed by a medical staff member, although, nursing staff would complete the social history section of the admission form on occasion.

The admission form contained no instruction on how the requested information might best be elicited or what level of alcohol consumption might be considered problematic. There was no supporting resource material that might assist in these tasks. Eliciting appropriate information and making a determination based on this information (as to the excessiveness or otherwise of the reported consumption) was dependent on the expertise of the admitting staff member.

We assessed how commonly the alcohol question was asked of patients and the utility of the resulting information by a retrospective file review. 120 files from clients admitted to the four general medical wards during a 3-month period (April–June 2004) were randomly selected for review (40 files were selected for each month sampled).

Random selection was employed, as a large number of patients (approximately 2750) were admitted during this period. The sample size afforded a margin of error of plus or minus (±) 7.25%, at a confidence level of 95%, for the prevalence calculation (described below). Once each file was located, the patient admission form was sought and a determination made (yes/no) as to whether any information had been recorded in response to the alcohol assessment question.
The presence of recorded information was taken as evidence the assessment question had been asked. The reported prevalence of alcohol assessment was based on this yes/no measure. All recorded information was then copied verbatim and subsequently reviewed for references to the quantity and frequency of an individual’s alcohol consumption and to the use of standard drink measures or type of alcohol consumed.

The knowledge of medical and nursing staff with regard to standard drink measures and recommended drinking limits as defined by the Alcohol Advisory Council (ALAC) was assessed via a series of multiple choice questions. Four response options were provided for the standard drink questions and six response options were provided for the recommended drinking limits.

The correct answers were:

- 1 standard drink is equal to a 330 ml can of beer (at 4% alcohol), 100 ml glass of table wine, and 30 ml of straight spirits;
- Recommended drinking limits for males are no more than 21 standard drinks in any 1 week and no more than 6 on any one drinking occasion—and for females they are no more than 14 standard drinks in any 1 week and no more than 4 on any one drinking occasion.

The attitudes of medical and nursing staff in regard to both alcohol assessment and intervention were measured via the shortened version of the alcohol and alcohol problems perception questionnaire (SAAPPQ).

**Table 1. Demographic profile of questionnaire sample**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nurses</th>
<th>Doctors</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>9% (7/77)</td>
<td>57% (13/23)</td>
<td>20% (20/102)</td>
</tr>
<tr>
<td>Female</td>
<td>91% (72/79)</td>
<td>44% (10/23)</td>
<td>80% (32/02)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-29</td>
<td>33% (27/82)</td>
<td>29% (7/24)</td>
<td>32% (34/06)</td>
</tr>
<tr>
<td>30-39</td>
<td>32% (26/82)</td>
<td>25% (6/24)</td>
<td>30% (32/06)</td>
</tr>
<tr>
<td>40-49</td>
<td>27% (22/82)</td>
<td>29% (7/24)</td>
<td>27% (29/06)</td>
</tr>
<tr>
<td>50+</td>
<td>8% (7/82)</td>
<td>17% (4/24)</td>
<td>11% (11/06)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>40% (29/72)</td>
<td>50% (10/20)</td>
<td>42% (39/92)</td>
</tr>
<tr>
<td>Maori</td>
<td>6% (4/72)</td>
<td>0% (0/20)</td>
<td>4% (4/92)</td>
</tr>
<tr>
<td>Pacific Island</td>
<td>8% (6/72)</td>
<td>0% (0/20)</td>
<td>7% (6/92)</td>
</tr>
<tr>
<td>Asian</td>
<td>31% (12/72)</td>
<td>40% (8/20)</td>
<td>33% (30/92)</td>
</tr>
<tr>
<td>Other</td>
<td>15% (11/72)</td>
<td>10% (2/20)</td>
<td>14% (13/92)</td>
</tr>
</tbody>
</table>

Sample sizes vary between variables as not all participants answered each question.

The SAAPPQ is a validated, 10-item, 7-point rating scale designed to measure a health professional’s motivation or willingness to work with drinkers (“motivation”); their expectations of work satisfaction with these clients (“work satisfaction”); their feelings about their adequacy of their knowledge and skills in working with these clients (“role adequacy”); the extent to which they feel they have the right to work with drinkers (“role legitimacy”); and their self-esteem in this specific task (“task specific self-esteem”).16,17

The knowledge and attitude questions were included in a survey sent to all medical (n=49) and nursing staff (n=145) employed in the general medical wards at the time of the study. Completed anonymous
surveys were collected in identifiable boxes on each participating ward. All potential participants were followed up by a second mail-out. The final response rate did not differ between the two occupational groups: 57% (82/145) of nursing staff returned surveys compared to 49% (24/49) of medical staff. This gave an overall response rate of 55% (106/194). Selected demographic characteristics of the sample are presented in Table 1.

Results

Practice—A patient admission form was located in 91% (109/120) of files randomly selected for review. Information pertaining to patient alcohol consumption was recorded in response to the standard alcohol assessment question in 78% (85/109) of these files. The latter figure may be considered the prevalence rate of alcohol assessment in the study setting (±7.25%). The level of information recorded in these 85 files was typically sparse. One or two worded responses such as “nil”, “not much” or “occasional” were provided in 71% (60/85) of cases.

The recorded response indicated current alcohol use in 54% (46/85) of cases and abstinence in the remaining 46% (39/85). Information pertaining to the quantity and frequency of alcohol consumption was specified in 14 out of the 46 files in which current alcohol use was indicated. Examples included: “1 glass wine/day”, “1–2 standard [drinks]/day” and “3 jugs of beer per week; binge, only on Saturday”.

Eight of these 14 files accounted for the type of alcohol consumed and 3 used standard drink measures. The remaining 32 files in which current alcohol use was indicated provided minimal detail. Either the quantity or frequency of alcohol consumption was recorded, but never both and rarely in a useful form. Examples included: “now and again”, “alcohol socially”, and “occasional glass of wine”. Four of these 32 files accounted for the type of alcohol consumed. No references to standard drink measures were evident.

Overall, excessive alcohol consumption was indicated in 5% (4/85) of files in which alcohol assessment information had been recorded.

Knowledge—Knowledge of standard drink equivalents—for spirits, wine and beer; and recommended weekly and single session drinking limits—were measured via a series of multiple choice questions. Table 2 outlines the percentage of respondents, by occupational group and for the total sample, who identified correct responses.

Overall, 12% of respondents were able to accurately identify standard drink equivalents for all three alcohol sources and 8% were able to accurately identify the recommended weekly and single session drinking limits for both men and women.

Chi-squared analysis revealed medical staff were more likely to answer both information sets correctly ($\chi^2=4.677$, df=1, p=0.031 and $\chi^2=24.643$, df=1, p<0.001, respectively).
Table 2. Percentage of participants who correctly identified standard drink quantities and recommended drinking limits

<table>
<thead>
<tr>
<th></th>
<th>Total (n = 106)</th>
<th>Nurses (n = 82)</th>
<th>Doctors (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Standard Drink Quantities</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spirit</td>
<td>53%</td>
<td>43%</td>
<td>88%</td>
</tr>
<tr>
<td>Wine</td>
<td>26%</td>
<td>24%</td>
<td>33%</td>
</tr>
<tr>
<td>Beer</td>
<td>50%</td>
<td>42%</td>
<td>79%</td>
</tr>
<tr>
<td><strong>All Correct</strong></td>
<td>12%</td>
<td>9%</td>
<td>25%</td>
</tr>
<tr>
<td><strong>Drinking Limits</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per occasion – Male</td>
<td>13%</td>
<td>6%</td>
<td>38%</td>
</tr>
<tr>
<td>Per occasion – Female</td>
<td>13%</td>
<td>4%</td>
<td>46%</td>
</tr>
<tr>
<td>Weekly – Male</td>
<td>28%</td>
<td>12%</td>
<td>83%</td>
</tr>
<tr>
<td>Weekly – Female</td>
<td>25%</td>
<td>20%</td>
<td>88%</td>
</tr>
<tr>
<td><strong>All Correct</strong></td>
<td>8%</td>
<td>1%</td>
<td>33%</td>
</tr>
</tbody>
</table>

Table 3. Mean SAAPPQ scores

<table>
<thead>
<tr>
<th>SAAPPQ Domain</th>
<th>Total</th>
<th>Nurses</th>
<th>Doctors</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Work Satisfaction</td>
<td>3.4</td>
<td>3.4</td>
<td>3.2</td>
<td>0.346</td>
</tr>
<tr>
<td>Task Specific Self Esteem</td>
<td>4.8</td>
<td>4.8</td>
<td>4.8</td>
<td>0.651</td>
</tr>
<tr>
<td>Motivation</td>
<td>4.1</td>
<td>4.2</td>
<td>3.8</td>
<td>0.026*</td>
</tr>
<tr>
<td>Role Legitimacy</td>
<td>5.2</td>
<td>5.0</td>
<td>6.0</td>
<td>0.001**</td>
</tr>
<tr>
<td>Role Adequacy</td>
<td>4.0</td>
<td>3.8</td>
<td>4.7</td>
<td>0.003**</td>
</tr>
<tr>
<td><strong>Overall Composite Score</strong></td>
<td>4.3</td>
<td>4.2</td>
<td>4.5</td>
<td>0.021*</td>
</tr>
</tbody>
</table>

**Attitudes**—Attitudes of ward staff with respect to alcohol assessment and intervention were measured via the SAAPPQ. On this scale, a score of 3.5 is considered a neutral response with higher scores indicating a more positive attitude (and, conversely, lower scores indicating a more negative attitude).
Overall, the mean score in four out of the five domain areas and the mean composite SAAPPQ score (the mean score across all five domains) were indicative of positive attitudes (Table 3). “Work satisfaction” was the only domain area in which the mean response fell below a score of 3.5. Statistically significant differences between medical and nursing respondents were evident on three out of the five domain scores (statistics based on Mann-Whitney U test, p value reported in Table 3).

Medical respondents reported more positive attitudes on the “role legitimacy” and “role adequacy” domains and less positive attitudes on the “motivation” domain when compared to nursing respondents. The difference between medical and nursing staff on the composite SAAPPQ score also reached a level of statistical significance.

Discussion

This paper sought to examine the alcohol assessment practice, knowledge, and attitudes of staff working in the general medical wards of a large, metropolitan hospital. It was anticipated that the findings would provide some insight into the prevalence and utility of hospital-based alcohol assessment and the ability of the latter to support brief intervention initiatives.

The results suggest patient alcohol consumption is routinely assessed, 78% of files reviewed, but the usefulness of the resulting information may be low. The latter was evidenced by the type of alcohol assessment information recorded, the inference of the recorded information, and participant response to the multi-choice questions.

Recorded alcohol assessment information comprised one or two word responses such as “nil”, “not much”, or “occasional” in 71% of cases. The clinical utility of this type of information is limited. A “nil” response may refer to a temporary period of alcohol abstention or abstinence as a long-term lifestyle choice. Similarly, an “occasional” response may refer to a glass of wine with dinner once or twice a year or to regular binging on hard liquor at celebratory events.

An accurate assessment requires detailed information regarding the length of reported abstinence or the type, quantity, and frequency of reported alcohol consumption. Detailed alcohol consumption data were only provided in 14 cases.

The recorded alcohol assessment information also indicate that 46% of patients abstained from alcohol consumption and that only 5% exceeded recommended drinking guidelines. These figures are almost certainly incorrect.

National household survey data indicate that 85% of adult New Zealanders consume alcohol at least once per year and that between 20–25% regularly exceed recommended drinking levels. Similarly, prevalence studies conducted in hospital settings indicate that between 16–26% of admissions screen positive for alcohol misuse.

It would appear that the standard alcohol assessment question in the study setting, therefore, was largely ineffective. It remains possible that the recorded responses were not an accurate reflection of the alcohol assessment actually conducted. Ward staff may have completed a thorough and accurate alcohol assessment, yet failed to record the results in a clinically useful manner.
Responses to the multi-choice questions suggest this was unlikely. Only 12% of respondents were able to correctly answer all three of the standard drink questions and only 8% of respondents were able to correctly identify all four of the recommended drinking limits questions.

Medical participants were significantly more likely to provide correct responses as compared to their nursing counterparts (25% vs 9% and 33% vs 1%, respectively); however, the majority of respondents in both groups were unable to do so. The general lack of knowledge in these areas may explain why references to the use of standard drink measures were only evident in three files.

The attitudinal data indicate that, whilst standard alcohol assessment practice may be poor, both medical and nursing respondents considered alcohol assessment to be a legitimate part of their professional role. Both groups also reported relatively high “task specific self esteem” and “composite SAAPPQ” scores. The former suggests most respondents felt comfortable providing an alcohol assessment, even if further training was seemingly required, and the latter suggests attitudes towards alcohol assessment were largely positive.

The “work satisfaction” domain received the lowest mean scores, suggesting alcohol assessment and intervention were not considered a particularly rewarding activity. Between-group differences in attitude were detected. Nursing respondents reported greater motivation for alcohol assessment, yet the medical respondents considered it a more legitimate part of their professional role and saw themselves as more adequately prepared to carry it out. The “knowledge” data confirmed the latter, but additional training and support were clearly needed by both groups.

Based on these findings it is difficult to draw any conclusion other than standard alcohol assessment practice in the study setting was seriously limited. The alcohol assessment question “alcohol (amount and duration)” was too broad and non-specific to elicit clinically meaningful data and staff knowledge of standard drink measures and recommended drinking limits was sparse at best. Nevertheless, compliance with the existing assessment question was high and attitudes towards alcohol assessment were largely positive. Thus, a platform for effective alcohol assessment was present: the medical and nursing staff were in the habit of regularly seeking alcohol consumption information and were supportive of this practice.

If the existing alcohol assessment question was replaced by a more effective alternative and if appropriate training and support resources were provided to staff, then the standard might improve. It is the recommendation of this paper, therefore, that the use of a structured, brief alcohol screen should be considered in the hospital setting. Validated screens that have previously been used for this purpose include the consumption scale of the Alcohol Use Disorders Identification Test (AUDIT)\textsuperscript{19}, the CAGE\textsuperscript{20} and the Short Michigan Alcohol Screening Test (SMAST).\textsuperscript{21}

An appropriate training program should also be introduced to support staff in the alcohol screening process. Adhering to a recognised alcohol screening process would improve the standard of assessment and would usefully support possible brief intervention initiatives in the process.

The findings presented in this paper are unlikely to be unique to the study setting. A similar standard of alcohol assessment is probably evident in most hospitals across
New Zealand. Research would be needed to confirm this of course; however, the lack of any National standard for hospital-based alcohol assessment or similarly instructive guidelines suggests the general quality of alcohol assessment may be variable at best.

The use of a validated, brief alcohol screen should therefore be considered on a wider scale. Hospital wards throughout the country could review their existing assessment practice and make changes as appropriate. Any assessment practice that relies on a non-specific question such as “alcohol (amount and duration)” or the expertise of medical or nursing staff may be found wanting if the findings reported in this study are common throughout New Zealand.

As a final point, it is worth noting that this study investigated an area that has received minimal research attention. Further research into the prevalence and utility of routine alcohol assessment practice in hospital settings is needed. Future research could improve on some of the limitations inherent in the study design. Major limitations included the relatively low rate of medical respondents to the knowledge and attitude questions and the reliance on recorded information as a measure of alcohol assessment prevalence and utility.

A number of the medical respondents who completed the survey questions were also rotated onto the hospital ward after the retrospective file review. This raises the possibility that the review findings may not have been an accurate reflection of the medical respondent’s assessment practice.

**Competing interests:** Professor Ross McCormick was a board member on the JE Caughey Trust and provided medical advice to the now defunct Beer, Wines and Spirit Council of New Zealand at the time of this study. Dr Ian Scott was Deputy Chair of the Alcohol Advisory Council of New Zealand (ALAC) and an elected member of the Auckland District Health Board at the time of this study.

**Disclaimer:** The views of the authors do not necessarily represent the views or the policies of the Alcohol Advisory Council of New Zealand (ALAC), the Accident Compensation Corporation (ACC), the JE Caughey Trust, or Auckland City Hospital.

**Author information:** Justin Pulford, Senior Researcher, Clinical Research & Resource Centre (CRRC), Waitemata District Health Board (DHB), Auckland; Ross McCormick, Director, Goodfellow Unit, University of Auckland and Chair of New Zealand Section of the Chapter of Addiction Medicine, RACP, Auckland; Amanda Wheeler, Co-Director, CRRC, Waitemata DHB, Auckland; Patrick Firkin, Senior Researcher, CRRC, Waitemata DHB, Auckland; Ian Scott, Lead Medical Officer, Detoxification Services, Community Alcohol and Drug Services, Waitemata DHB, Auckland; Gail Robinson, Co-Director, CRRC, Waitemata DHB, Auckland

**Acknowledgements:** This study was conducted with funding obtained from the Alcohol Advisory Council of New Zealand (ALAC), the Accident Compensation Corporation (ACC), the JE Caughey Trust, and Auckland City Hospital. The authors also thank John Henley (Clinical Director of the Admission and Planning Unit, Auckland City Hospital) for supporting the study initiative as well as the following members of the research team: Julia Davies, Michele Yeoman, Sadiqa Khan, Mary Routledge, and Trudy Hall.

**Correspondence:** Justin Pulford. Clinical Research & Resource Centre (CRRC), Level 3, Snelgar Building, Private Bag 93115, Henderson, Waitakere 0650. Fax: (09) 838 1883; email: Justin.Pulford@waitematadhb.govt.nz
References:

An international surgical collaboration for the management of pulmonary artery sarcoma: a New Zealand experience

Yaso Kathiravel, David Westwood, Jeff Macemon, Harsh Singh

Abstract

We present the case of a 73-year-old man with a pulmonary artery sarcoma successfully treated as a result of an international surgical collaboration. The tumour was initially deemed to be unresectable due to a lack of local expertise managing cardiac malignancies. Since the patient was unable to travel to a specialist centre in the United States, he was initially offered only palliative therapy. However, two surgeons with experience of treating malignant cardiac tumours travelled to New Zealand specifically to perform a potentially curative resection of his tumour. This case suggests that there should be an emphasis placed on the development of internationally acceptable protocols for the treatment of rare conditions and improved local access to overseas surgical expertise.

Primary pulmonary artery sarcomas (PAS) are extremely rare tumours of the cardiorespiratory system. Current literature advocates surgical resection as the best treatment option. In surgery, the best clinical outcomes are achieved when the number of patients being treated is sufficient for expertise to be maintained. Patients requiring treatment for rare conditions typically travel to specialist centres for treatment. We describe the international surgical collaboration that enabled a patient with PAS to have his tumour resected locally in New Zealand.

Case report

A 73-year-old man who was a non-smoker was referred to the respiratory physicians with a 4-month history of progressively worsening wheeze and dyspnoea. A chest radiograph revealed a left hilar lung mass suspicious of malignancy. A computerised tomography (CT) scan (Figure 1) demonstrated an endobronchial lesion in the left upper lobe with associated left upper lobe collapse. A necrotic tumour arising from the left upper lobe was biopsied during bronchoscopy.

Histology revealed a non-epithelial malignancy. Repeat CT scans and magnetic resonance imaging (MRI) over the following 6 months revealed a progressive abnormality arising in the left main pulmonary artery with distal propagation into the left lung. A presumptive diagnosis of left pulmonary artery sarcoma was made.

In view of the extensive nature of the tumour the patient received a course of radiotherapy. However, subsequent CT scans showed signs of tumour progression.

The consensus opinion amongst thoracic surgeons consulted in New Zealand and Australia was that the tumour was unresectable and that the patient should be treated palliatively. However, a further opinion from the University of Texas M D Anderson Cancer Center was sought and two surgeons with experience of treating malignant cardiac tumours were invited to perform the surgery in Christchurch.
The patient was placed on cardiopulmonary bypass. The left pulmonary artery at the bifurcation, including a cuff of right pulmonary artery, was excised and samples sent for frozen section. The margins showed only fibrous tissue. A pulmonary homograft was sutured to complete communication between the right pulmonary artery and the pulmonary trunk. Left pneumonectomy and lymph node sampling was performed. A pre-operative angiogram had demonstrated severe triple vessel coronary artery disease so coronary artery bypass grafting was also undertaken.

The patient made an excellent recovery and was discharged 11 days postoperatively. Histology confirmed PAS with no lymph node involvement. Follow-up investigations showed no evidence of recurrent or metastatic disease at 12 months.

**Discussion**

Primary PAS is rare with less than 200 cases reported since Mandelstamm first described it in 1923. The estimated incidence of PAS is 0.001–0.03% with a slight female preponderance (1:1.3). Reported age at presentation ranges from 13–86 years with a mean age of 48 years.

Primary PAS are malignant mesenchymal tumours that arise from the intima of the pulmonary artery with a predilection for the dorsal region of the pulmonary trunk. They generally propagate in the direction of flow with progressive obstruction of the
pulmonary outflow tract. There may be direct invasion of an adjacent bronchus or pulmonary metastases may arise from tumour emboli, but extrathoracic disease is unusual.\(^6\)

Clinical presentation is non-specific and typically mimics pulmonary thromboembolic disease with symptoms of dyspnoea, chest pain, cough, and haemoptysis.\(^7\) Unilateral absence of blood flow on ventilation-perfusion scans, pedunculated lesions exhibiting a “to-and-fro” motion on pulmonary angiography or lack of response to anticoagulation should raise the suspicion of PAS.

MRI with gadolinium-diethylenetriamine pentaacetic acid enhancement has been proposed as a way of distinguishing tumour from thrombus.\(^8\) PAS may also be diagnosed on histological examination of thromboendarterectomy specimens.

Only radical surgical resection improves clinical symptoms and offers the chance of prolonged survival.\(^1\) Early lesions may be completely excised using endarterectomy. However, full-thickness resection of the pulmonary artery with reconstruction of the pulmonary outflow tract provides a better margin of resection.

For patients with extensive mediastinal involvement or metastatic disease a limited tumour resection or bypass procedure may offer significant palliation and survival benefit. The prognosis is mainly dependent on local recurrence. Median length of survival without intervention is 1.5 months. Surgical resection improves survival to approximately 12 months and there is limited evidence that this may be extended by neoadjuvant and/or adjuvant irradiation and chemotherapy.\(^7\)

The rarity of cardiac malignancies means that there are few surgeons with hands-on experience of their management. The best outcomes are achieved at designated specialist units with a sufficient caseload to enable expertise to be accrued.

In the case above, an attempt was initially made to send the patient to the United States. However, the New Zealand Ministry of Health was unwilling to pay for the patient to undergo surgery in Texas in view of the indeterminate length of stay (and hence cost) of the postoperative recovery period. Videoconferencing and the transmission of scans electronically permitted the case to be discussed preoperatively.

While it is not unusual for surgeons from the developed world to donate their time and skills to perform operations in developing nations, this case is a unique example of surgeons travelling to another developed country specifically to operate on a rare condition. This was only feasible due to adequate local postoperative care provision, including the presence of a cardiac intensive care unit.

The pulmonary homograft was obtainable within New Zealand and since this was not a novel procedure local ethical approval was not required. Recognition of the visiting surgeons’ qualifications by the Medical Council of New Zealand was also necessary for them to obtain registration and indemnity insurance.

The major advantage of undertaking the procedure locally was that the patient obtained access to potentially curative surgery when previously he had been only assigned palliative treatment. The utilisation of local resources also minimised costs while the local cardiothoracic surgeons were presented with an exceptional learning opportunity. The drawbacks for the visiting surgeons were the unavailability of their usual operating team and the short duration of their stay in New Zealand. As a result,
the patient was aware that the visiting specialists would only be available for the first 48 hours postoperatively.

Overall, this case suggests that in the era of globalisation there should be an emphasis placed on the development of internationally acceptable protocols for the treatment of rare conditions with improved local access to overseas surgical expertise.

**Author information:** Yaso Kathiravel, Registrar; David A Westwood, Registrar; Jeff B Macemon, Registrar; Harsh P Singh, Consultant; Department of Cardiothoracic Surgery, Christchurch Public Hospital, Christchurch

**Acknowledgements:** No external financial support was received with this project.

**Correspondence:** Harsh Singh, Department of Cardiothoracic Surgery, Christchurch Public Hospital, Private Bag 4710, Christchurch. Fax: (03) 364 1361; email: harsh@xtra.co.nz

**References:**

Hepatic portal venous gas in a patient with pneumatosis intestinalis

Chih-Hao Shen, Heng-Cheng Chu, Wei-Kuo Chang, You-Chen Chao, Tsai-Yuan Hsieh

Case report

A 76-year-old man was sent to the Emergency Department with 10-day abdominal fullness and dull pain. He was known to suffer from chronic atrial fibrillation. On examination, he had a tachycardia, hypotension, and a distended abdomen with vague pain and absent bowel sounds. Laboratory examinations disclosed lactic acidosis and a high serum creatine phosphokinase level (1890 U/L).

Abdominal ultrasound study revealed hepatomegaly and diffuse hyperechoic particles over both lobes of the liver (Figure 1). Contrast-enhanced computed tomography scan of abdomen at portal venous phase showed free gas in the portal and mesenteric veins (Figure 2), consistent with hepatic portal venous gas (HPVG). There were also dilatation, bubble-like pneumatosis, and mucosal necrosis of small bowel in left middle abdomen (Figure 3).

Pneumatosis intestinalis (PI) resulting from mesenteric ischemia was speculated. The patient was too critical to be stabilised for surgical intervention and eventually died of septic shock.

Figure 1. Abdominal ultrasound study showing diffuse hyperechoic particles
Figure 2. Portal venous phase of contrast-enhanced computed tomography scan showing free gas in the portal veins

Figure 3. Computed tomography scan showing dilatation, bubble-like pneumatosis, and mucosal necrosis of small bowel in left middle abdomen
Discussion

HPVG—a rare but potentially lethal condition providing clues in diagnosing patients with acute abdomen—typically exists in the peripheral and nondependent part of the liver. The pathogenesis of HPVG is not well known. It has been proposed that metabolism of intestinal bacteria, swallowed air, and intestinal gases all contribute to the formation of HPVG.

Both ischaemic and non-ischaemic causes contribute to the emergence of HPVG. Mesenteric ischemia or infarction with or without PI causes a high mortality rate and needs urgent laparotomy.¹ Prolonged and extensive bowel ischaemia results in a poor prognosis.

Non-ischaemic causes of HPVG, however—such as increased intraluminal pressure (i.e. obstruction, blunt abdominal trauma, and iatrogenic luminal dilatation), focal lesions of bowel wall (i.e. carcinoma, ulcer, and inflammatory bowel disease), and pathogenic translocation of intestinal bacteria—are related to a favourable clinical outcome.²

Author information: Chih-Hao Shen, Physician, Department of Internal Medicine; Heng-Cheng Chu, Assistant Professor, Division of Gastroenterology, Department of Internal Medicine; Wei-Kuo Chang, Associate Professor, Division of Gastroenterology, Department of Internal Medicine; You-Chen Chao, Professor, Division of Gastroenterology, Department of Internal Medicine; Tsai-Yuan Hsieh, Chief; Division of Gastroenterology, Department of Internal Medicine; Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan

Correspondence: Tsai-Yuan Hsieh, Chief, Division of Gastroenterology, Department of Internal Medicine, Tri-Service General Hospital, No. 325, Sec 2, Cheng-Kung Rd., Neihu 114, Taipei, Taiwan. Fax: +886 2 87927139; email: horace.shen@msa.hinet.net

References:


Medical Benevolent Fund and the Defence Union

Published in the N Z Med J 1907;6(24).

The Committee of the New Zealand MEDICAL BENEVOLENT FUND and the New Zealand MEDICAL DEFENCE FUND, LTD., wish to before the notice of medical men residing in the Dominion the existence of the two in the hope of their seeing their way to becoming members of either one or both of them.

The BENEVOLENT FUND has for its object “The relief of Legally Qualified Medical Men and their Families under severe and urgent distress, occasioned by sickness, accident, or other calamity.”

The annual subscription is £1, or the subscriber may become a Life Member by paying a single subscription of £10. By the rules of the Fund the Committee is not able to deal with the capital until £1,000 has accumulated. There is now about £900 subscribed towards that sum.

With regard to the DEFENCE UNION, the funds so far have been only small, and the most the Committee has been able to do is to assist in paying the legal expenses of medical men who have been in the unfortunate position of defendant in blackmail cases.

The ultimate end the Committee hopes to attain is to be able to take up any deserving case and defend it in the Law Courts, as is done by the Defence Union at Home. The annual subscription is £1, but the. Committee considers it unnecessary to call up the whole amount every year, and fixed the yearly sum of 5/- per member, the other 15/- to be called up if required. This ruling applies only to members of the N.Z. Branch of the B.M.A.; other subscribers pay £1 annually, and are not liable to further calls.

The Union has now been in existence over 10 years, and has been able during that time to pay wholly or in part the legal expenses of several medical men, but with a larger membership more could be done, and the Committee hopes that every medical man in New Zealand will see the need of joining the Union.

The Honorary Secretary, Dr. W. Irving, of Christchurch, will be glad to give all particulars.

The New Zealand Medical Benevolent Fund is one to which every doctor ought to devote the modest pound per annum which is asked of him. He who either by favor of fortune or by a life of arduous toil has gained a competence for himself, should remember that there are always less fortunate brethren to consider; and the young one who is fighting for a position should not forget that this is the cheapest form of insurance that he can go in for.

The Defence Fund has happily had but few calls made on it, but any day some serious need for it may arise, and the mere fact that there is such a fund, proving that we are ready and able to stand by a professional brother when being wrongly attacked, is a powerful protection.

The effects of hypertonic saline infusion on lipopolysaccharide-induced endothelial cell activation

J Cursons¹, Dr F Van Haren¹,², Dr R Cursons³, Dr J Sleigh¹,²

1. Waikato Clinical School, Auckland University
2. Intensive Care Unit, Waikato Hospital
3. Molecular Genetics Laboratory, Waikato University

Hypertonic saline solution (HSS) has been identified for several years as a promising treatment against the development of sepsis in patients exposed to gram-negative bacterial infections. There are several methods of action which make HSS an effective treatment. These include physiological improvements such as reduced cellular and tissue oedema, improved haemodynamics, as well as cellular immunomodulation in both neutrophils and endothelial cells (ECs). This altered immune response further produces an attenuation of the excessive immune activation which characterises sepsis. Despite several studies investigating HSS in the treatment of sepsis, few investigate the effects of HSS on EC gene expression. To investigate the ex vivo effects of HSS on ECs, a system has been developed to allow the perfusion of umbilical cord veins in whole umbilical cord preparations. This system allows the investigation of an isolated EC immune response, in the absence of white blood cell effects. By exposing the umbilical vein to lipopolysaccharide (LPS) to induce EC immune activation, the altered gene expression produced by HSS has been investigated in this unique model with promising results. Hypertonic saline appears to attenuate the increase in expression for a variety of genes upon exposure to LPS, including: adhesion molecules such as E-selectin and intercellular adhesion molecule-1; proinflammatory cytokines such as interleukin-1β and interleukin-6; chemokines such as interleukin-8; and procoagulants such as tissue factor. The altered expression of these genes which are vital in the development of an immune response, indicates that the immunomodulatory effects of HSS on ECs may play a key role in the efficacy of HSS as a treatment for sepsis.
Epidemiology of thyroid disease in Hamilton general practice

Veronique Gibbons¹, John Conaglen¹, Steven Lillis¹, Vignesh Naras², Ross Lawrenson¹

1. Waikato Clinical School, University of Auckland, Hamilton
2. Wellington School of Medicine, Otago University, Wellington

Background—Thyroid dysfunction is reported to be common within the community. While the prevalence of thyroid dysfunction is reasonably well known in other countries, very limited New Zealand data is available.

Aims—This study reports on the prevalence and incidence of thyroid dysfunction in a population-based sample of adults (derived from general practice), and compares the findings with previously published data.

Methods—General practices in Hamilton with a combined population of 21464 patients participated in the study. A retrospective cross-sectional review was conducted using computerised searches using diagnostic codes for thyroid dysfunction; prescribing data of those receiving thyroid or thyroid function altering medication; and lab data to find all patients with a recognised thyroid disorder. Data was verified against computerised and hand-held records.

Findings—Our study established the incidence of thyroid dysfunction in the community of 1.8 per 1000 and prevalence of 3.2%. Our prevalence data is similar to national and international literature with the burden of thyroid dysfunction being greater in women and in the older population.

Diabetes patients in Waikato & their hospital admissions

Grace Joshy, Ross Lawrenson, Peter Dunn

Aims—To examine the profile of diabetes patients registered with Waikato Regional Diabetes Service (WRDS) in 2005 and to estimate their hospital admission rates.

Methods—The WRDS provides secondary diabetes care and also performs retinal screening in the Waikato DHB region. Hence the register is thought to be near complete for Waikato. Waikato diabetes prevalence estimates were calculated. Hospital admissions of all diabetes patients registered and alive in 2005 were looked at.

Results—A total of 9936 patients (including 796 new patients) were registered in 2005. In 2005, the diabetes population increased by 7% from the previous year. The number of diabetes patients has been growing at a rapid pace with 11% increase in 2004 and 10% in 2003. The 2005 patients were predominantly European (67%), followed by Maori (20%), Indian (2.6%), Pacific Islander (2.3%) and Other Asian (1.6%). The population was of mean age 61 ± 15.9 years and mean duration of diabetes 9.6 ± 8.5 years. 86% had Type 2 diabetes and 51% were male. Europeans are diagnosed with Type 2 diabetes at a mean age of 59 years where as other ethnic groups are diagnosed a nearly decade earlier (Maori 48, Indian 49, Pacific & Asian 50 years). Although Indians outnumber Pacific Islanders in the WRDS database, ethnic
specific prevalence estimates for Indians, Other Asians could not be calculated due to lack of population estimates for these groups in the public domain. Ethnicity is self-selected by patients and is different from multiple ethnicity selection in census.

The diabetes patients had a total of 6275 admissions in 2005 including 3287 day admissions. This resulted in 20,637 inpatient days in 2005. Maori patients have significantly high renal admission risk (odds ratio of 9) compared with Europeans (Table 2). But they do not have an increased risk for coronary artery disease (CAD) admissions or cerebrovascular disease (CVD) admissions.

Table 1. Age-specific diabetes prevalence estimates by ethnicity for Waikato in 2005

<table>
<thead>
<tr>
<th>Age</th>
<th>Other</th>
<th>Maori</th>
<th>Pacific</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-9</td>
<td>0.1%</td>
<td>0.0%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
<tr>
<td>10-19</td>
<td>0.4%</td>
<td>0.2%</td>
<td>0.3%</td>
<td>0.3%</td>
</tr>
<tr>
<td>20-29</td>
<td>0.8%</td>
<td>0.5%</td>
<td>0.7%</td>
<td>0.7%</td>
</tr>
<tr>
<td>30-39</td>
<td>1.0%</td>
<td>1.4%</td>
<td>1.1%</td>
<td>1.1%</td>
</tr>
<tr>
<td>40-49</td>
<td>1.9%</td>
<td>4.0%</td>
<td>5.1%</td>
<td>2.3%</td>
</tr>
<tr>
<td>50-59</td>
<td>4.0%</td>
<td>10.9%</td>
<td>10.2%</td>
<td>5.0%</td>
</tr>
<tr>
<td>60-69</td>
<td>7.8%</td>
<td>18.4%</td>
<td>19.7%</td>
<td>9.0%</td>
</tr>
<tr>
<td>70-79</td>
<td>11.4%</td>
<td>19.8%</td>
<td>17.1%</td>
<td>12.0%</td>
</tr>
<tr>
<td>80+</td>
<td>10.0%</td>
<td>17.3%</td>
<td>10.2%</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Odds ratios (95% CI) for hospital admissions in 2005

<table>
<thead>
<tr>
<th>Variables</th>
<th>All Admissions</th>
<th>Renal*</th>
<th>CVD*</th>
<th>CAD*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female vs Male(=1)</td>
<td>1.06 (0.96 - 1.16)</td>
<td>0.76 (0.47 - 1.21)</td>
<td>0.92 (0.60 - 1.40)</td>
<td>0.73 (0.55 - 0.98)*</td>
</tr>
<tr>
<td>Type 1 vs Type 2(=1)</td>
<td>1.23 (1.03 - 1.48)*</td>
<td>2.25 (1.08 - 4.70)*</td>
<td>0.71 (0.25 - 1.99)</td>
<td>0.76 (0.41 - 1.41)</td>
</tr>
<tr>
<td>Maori vs European(=1)</td>
<td>1.34 (1.18 - 1.51)*</td>
<td>9.64 (5.55 - 16.76)*</td>
<td>1.11 (0.62 - 1.99)</td>
<td>1.29 (0.89 - 1.86)</td>
</tr>
<tr>
<td>Pacific vs European(=1)</td>
<td>0.62 (0.42 - 0.91)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian vs European(=1)</td>
<td>0.59 (0.38 - 0.93)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indian vs European(=1)</td>
<td>0.73 (0.52 - 1.03)</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Adjusted for age, duration of diabetes, gender, ethnicity and type of diabetes
*Analysis include European and Maori patients only; * p<0.05.

After co-variate adjustment, Maori, Pacific and Indian patients have significantly higher risk of vision threatening retinopathy compared with Europeans (odds ratio 1.53, 2.18 & 2.86 respectively). Diabetes patients aged 40+ have higher mortality rates compared with NZ total population (Figure 1). But the rates are lower than expected based existing literature, especially for 80+ age group.
Conclusion—The number of diabetes patients in Waikato has been increasing at a rapid pace in the recent years, around 7-10% per year. The Indian diabetes patients in Waikato have outnumbered Pacific Islanders. Diabetes and related complications result in high hospital admission rates. The growing number of patients and their hospital admission rates will increase the burden on health care resources. The high rates of renal admissions among Maori diabetes patients require further study.

New Zealand psychologists’ perceptions and opinions on the use of the current classification systems of mental disorders

Raksha Lutchman, Graham Mellsop, Joanna McClintock, Karma Galyer, Lauren Gaffaney

For nearly 100 years the World Health Organisation and the American Psychiatric Association have been developing the International Classifications of Diseases (ICD) and the Diagnostic and Statistical Manual (DSM) respectively. The planning for the next versions, ICD-11 and DSM-V, are currently underway. The use of the current classifications in clinical practice raises a number of issues concerning the “goodness of fit” between diagnostic concepts, cultural considerations and many aspects of clinical practice. This study examined the perceptions and opinions psychologists’ have about the current classifications.

Objective—The current diagnostic classifications, ICD-10 and DSM IV, developed to provide a world-wide common language in psychiatry with priority to empirical evidence and reliability. This study examines the impact and utility of these tools from a psychological perspective; it explores their usefulness, impact on clinical and social practices, and evaluate their relevance in psychological assessments, formulation and treatment. It also investigated future directions for classification systems.

Method—An anonymous postal survey of 480 psychologists was undertaken. Questionnaires were posted to all registered psychologists. One month after the initial mailing, reminders were sent with a closure date two weeks later. The analysis was performed using SPSS - statistical package (SPSS for Windows 14.0™).
Results—01 Questionnaires were returned, completed. Frequencies, descriptive data and trends were analysed. Results indicate that most of the psychologists practising in New Zealand primarily use and prefer the DSM because that is what they were trained on. It provides a common language to clinicians and they find it to be a universal diagnostic system. About a third use the ICD-10 because they found it straightforward and “culturally sensitive”.

Psychologists make limited use of DSM’s multi-axes, with more routine use of axis I and II. Many found axes IV and V too vague and of little use in their clinical practice. Some clinicians complement the classification system by using the Tikanga Maori Model and the Te Whara Tapa Wha Model for case formulations and treatment planning.

The proposed goals of a classification system they valued most highly were reliable inter-clinician communication, conveying information about aetiology/pathogenesis and for facilitating research. The DSM classificatory system was most preferred and the ICD was the least preferred, yet most psychologists’ preferred option was to prioritise axes useful in guiding treatment or case management.

Conclusions—Although the current classification system is in keeping with their stated goals and objectives, New Zealand psychologist’s hold the perception that the system is not representative of psychological explanations of clients’ presentation. Their opinions are based on concerns that the classification system is too limited, overly compartmentalised and pathologised. It follows the medical model and individualisation is lost. The psychologists’ would like a classification system that considers cultural identity and ethnic belief systems. One which can accommodate individual differences, can have more behavioural definition and systematic description, less complex in theory and it must inform and guide treatment.

Indicators of outcome after primary angioplasty for myocardial infarction (PAMI) in high-risk ST-elevation MI: Waikato Hospital experience

N Swanson, G Devlin, S Holmes, C Nunn

Waikato Hospital, Hamilton, New Zealand

Introduction—Waikato Hospital has had a PAMI program serving a population of approximately 170,000 since 1994. Patients taken to cath lab in Waikato tend to exclude “low-risk” patients with limited inferior myocardial infarcts and so represent a higher-risk PAMI cohort. Long-term survival after PAMI has not been widely studied, although studies in the US and Europe suggest overall (i.e. including lower risk MI patients) 5-year survival of 78-80%. We have studied factors associated with adverse long-term survival.

Methods—The PAMI database for patients treated in Waikato Hospital from 1996 (n=553) was analysed. Baseline demographic information and incidence of shock on admission shock were analysed, together with GIIb/IIa antagonist or stent usage. Patients with at least three-year follow-up were divided into an Early (1996-9) and a
Late (2000-3) group and survival recorded using Kaplan-Meier curves (XLSTAT software).

**Results**—5, 8 & 10-year survival rates were 76.5% (n=318), 72.4% (n=146) & 70.7% (n=52) respectively. The presence of cardiogenic shock (n=47) was strongly associated with adverse outcomes with survival 51% (n=44) at one year (v. 90.3% in non-shock patients). Despite this very poor initial progress, patients with cardiogenic shock at presentation who survived past one year had a relatively good prognosis with 42% (n=30) still alive at five years (v.80% non-shock patients). 5-year survival was not significantly better between Early and Late phase patients (74.2% v. 78.2% respectively, log-rank test for survival curves p=0.26). Increased stent (35.9% v. 70%, p<0.001) and GIIb/IIIa (32.5 v. 59.5%, p<0.001) use was seen between these two time periods. Survival was not associated with GIIb/IIIa use, but stent use was associated with greater 5-year survival (80.4% v. 70.2%, log-rank test for survival curves p=0.01).

**Conclusions**—Cardiogenic shock led to poor outcomes immediately, but was not a driver of mortality over longer term follow-up. Stent use was associated with improved survival. Survival after PAMI remains high over the very long-term and Waikato results are similar to international comparisons, even in a higher-risk cohort.
Complications of percutaneous coronary interventions (PCI) in the real-world: the Waikato Hospital experience

N Wijesinghe, S McAlister, C Sebastian, S Heald, CM Nunn, HA Charleson, HF McAlister, GP Devlin

Department of Cardiology, Waikato Hospital, New Zealand

Background—The use of PCI has revolutionised the treatment of coronary artery disease over the past two decades. However, this intervention has a well-defined morbidity and mortality. The aim of this study was to ascertain the incidence of major complications during PCI in our cardiac catheterisation laboratory.

Method—Retrospective review of medical records and complication forms from all consecutive patients who had PCI at Waikato Hospital between 01.01.2000 to 31.12.2006. Patients who had primary angioplasty for ST elevation myocardial infarction were excluded.

Results—A total of 3425 patients had PCI during this period. They included 73% men (mean age: 60.9 years) and 27% women (mean age: 64.6 years). The procedure included 98.8% coronary angioplasty, 1% intra-coronary pressure gradient measurement and 0.2% intra-vascular ultra sound scanning. The arterial access site was 99.77% femoral, 0.2% radial and 0.03% brachial artery. Closure devices were used in 2.4% patients (1.3% Angioseal, 0.8% Perclose and 0.3% Starclose devices) at the arteriotomy site. Procedure-related mortality was 0.23%. Other major complications included 0.17% cardiac arrest, 0.17% myocardial infarction, 0.11% acute stent occlusion, 0.23% dissection of coronary artery, 0.11% urgent coronary bypass surgery, 0.34% ongoing ischemia, 0.15% cerebrovascular accidents (0.06% strokes and 0.09% transient ischemic attacks), 0.06% arrhythmia, 0.51% vascular access complications (0.28% bleeding, 0.17% pseudoaneurysm, 0.06% dissection), 0.23% contrast reaction and 0.11% haemodynamic complications. Overall major complication rate was 2.37% (24:1000).

Conclusion—PCI carries a small but definite risk of morbidity and mortality. Our complication rates were comparable with published data.
A vascular phenomenon

Utpal Nandy, George I Varughese, Yelin L Hock

A 43-year-old, right-handed Caucasian lady, who worked with air-powered screwdrivers, was referred with symptoms of numbness in her hands more marked on the right side. She also complained of intermittent redness and pallor of her right hand on exposure to cold. She had no symptoms or signs of generalised illness. On examination, reduced peripheral pulses in the right upper limb were noted. An aortogram was performed (Figure 1).

Figure 1

What is the diagnosis?
Diagnosis—Takayasu’s arteritis with Raynaud’s phenomenon

Digital subtraction arch aortogram (Figure 1) demonstrates marked delay in filling of the right carotid and subclavian vessels, secondary to occlusion of the right brachiocephalic trunk (arrow). The right subclavian and carotid arteries fill via collaterals. There is a stenosis at the origin of the left subclavian artery (arrowhead).

A diagnosis of Takayasu’s disease was made, and arterial biopsy showed inflammatory cell infiltration of the vessel wall (Figure 2), confirming the diagnosis. Takayasu’s arteritis is more often seen in oriental women and patients present with systemic symptoms like fever, fatigue and malaise, and a raised ESR. The patient also had superimposed Raynaud’s phenomenon.

Figure 2. Arterial biopsy showing inflammatory cell infiltration of vessel wall

Author information: Utpal Nandy, Specialist Registrar in Genitourinary Medicine, Leicester Royal Infirmary, Leicester, UK; George I Varughese, Specialist Registrar in Diabetes, Endocrinology & General (Internal) Medicine, Queen’s Hospital, Burton-on-Trent, UK; Yelin L Hock, Consultant Histopathologist, Manor Hospital, Walsall Hospitals NHS Trust, Walsall, UK

Correspondence: Dr G I Varughese, Specialist Registrar in Endocrinology & General (Internal) Medicine, Diabetes Centre, Queen’s Hospital, Belvedere Road, Burton-on-Trent DE13 0RB, UK. Email: georgeiv@doctors.org.uk
Medical journals and guns

Richard Smith, former editor of the *BMJ*, is upset over the relationship of Reed Elsevier and the arms trade. This company publishes not only *The Lancet* but also 2000 other medical and scientific journals.

The scientific and medical part of Reed Elsevier’s business is the most profitable: in 2005 its sales totalled £1436 million, or 28% of total Reed Elsevier sales, and its profits were £449 million, or 37% of the company’s total profits.

What annoys Smith is the point that through its subsidiary, Reed Exhibitions, Reed Elsevier runs arms fairs in Britain, the United States, the Middle East, Brazil, Germany, and Taiwan.

Methinks, Smith has a valid point. To be fair to *The Lancet*, he points out that its editors did ask the publisher to divest itself of the arms sales in 2005—they didn’t.

*Journal of the Royal Society of Medicine 2007;100:114–5*

Atrial fibrillation (AF) and strokes

Those with non-valvular AF have an annual stroke rate of 5% which may be lowered by two-thirds if the patient may be safely anticoagulated. In an excellent review, Graeme Hankey and colleagues delve into this important topic. The risk can be evaluated more exactly and this paper points out that the ischaemic stroke risk is best estimated with the CHADS<sub>2</sub> score (Congestive heart failure, Hypertension, Age ≥ 75 years, Diabetes, 1 point each; prior Stroke or transient ischaemic attack, 2 points).

Those with a score of 0 have an annual rate of 1.9% and risk rate escalates with extra points reaching 18.2% when the CHADS<sub>2</sub> score is 6. This information should ease the decision-making with respect to anticoagulation. However, they caution against the use of warfarin in the 85+ age group.

*Med J Aust 2007;186:197–202*

Management of stable coronary disease

It is generally accepted that percutaneous coronary intervention (PCI) often improves the management and outcome of unstable angina. It is, however, uncertain whether PCI is similarly beneficial to those with stable angina. This report concerns a randomized trial involving 2287 patients who had objective evidence of myocardial ischaemia and significant coronary artery disease.

Half received optimal medical therapy and PCI and the other half optimal medical therapy alone. The results of this trial indicate that PCI did not reduce the risk of death, myocardial infarction, or other major cardiovascular events when added to optimal medical therapy.

Chronic obstructive pulmonary disease (COPD)—what defines abnormal lung function?

The definition of COPD has evolved over time from one based on a clinical diagnosis of chronic bronchitis or anatomical findings of emphysema to one based on the presence of abnormal lung function.

Most of us will recall COPD is defined spirometrically by a forced expiratory volume in 1 second (FEV₁) being less than 70% of the forced vital capacity (FVC)—preferably without significant improvement after bronchodilator treatment. However, as the FEV₁/FVC ratio declines with age, using the fixed ratio to define COPD may “overdiagnose” COPD in older populations.

This paper reviews the outcome of approximately 5000 COPD patients 65 years of age or older who might have been thus “overdiagnosed”. Their conclusion is that the time-honoured FEV₁/FVC ratio is still valid.


Multidrug resistance

Chris Higgins reports from the MRC Clinical Sciences Centre, Hammersmith Hospital, London on this very important subject. He points out that the acquisition of multidrug resistance is a serious impediment to improved healthcare.

How does this happen? Apparently frequently due to active transporters that pump a broad spectrum of chemically distinct, cytotoxic molecules out of cells, including antibiotics, antimalarials, herbicides and cancer chemotherapeutics in humans. The best known of these is mammalian P-glycoprotein, which was identified 30 years ago.

Circumventing the causes of multidrug resistance has failed, and in dealing with the bacteria versus antimicrobial conflict, Harris believes a better tactic would be to cease the profligate misuse of antibiotics.

Agreed.

Nature 2007;446:749–57
ACC on the back pain article by Crawford et al

Crawford et al’s recently published article Exploring General Practitioner Identification and Management of Psychosocial Yellow Flags in Acute Low Back Pain (NZMJ 2007 May 18;120(1254); http://www.nzma.org.nz/journal/120-1254/2536) has generated both interest and concern at Accident Compensation Corporation (ACC).

We hope the article and this letter will provide an opportunity to pursue academic debate on the use and validity of clinical guidelines, and on the diagnosis and management of psychosocial yellow flags in low back pain.

In brief, it is our view that the article makes a number of unfounded claims, which we wish to refute. For example:

“GPs do not understand psychosocial yellow flags and do not use the yellow flags guideline”

On the contrary, for example, a recent case study of 461 general practitioners revealed that 74% of GPs consistently identified psychosocial yellow flags in their responses on the management of acute low back pain cases (ACC, 2005).

“ACC's dissemination of guidelines consists only in mailing out information to GPs...there is no strategy for guideline dissemination and implementation”

On the contrary, the Development Unit has a comprehensive strategy for disseminating and implementing guidelines and achieving best practice. For psychosocial yellow flags in acute low back pain, the strategy was initiated through a nationwide launch and media campaign, and included: Case Studies of general practitioners, chiropractors, and physiotherapists; Individual Feedback Reports; Reviews; Continuous Medical Education sessions, a DVD, and a CDrOm.

“The guidelines are prescriptive and do not reflect the way GPs work”

They are guidelines, and as such, they are not prescriptive. Furthermore, they were developed through extensive consultation with GPs and professional organisations. The latest Acute Low Back Pain Guide is endorsed by the New Zealand Guidelines Group, the New Zealand Society of Physiotherapists, the Royal New Zealand College of General Practitioners, and the New Zealand Register of Osteopaths.

“The guidelines are not based on evidence”

The guidelines were, in fact, based on a review of all available international literature at the time of publication. Moreover, the Acute Low Back Pain Guide has been updated twice since the original publication date, based on the latest research (The most recent guideline was published in October, 2004).

“Using guidelines doesn’t improve outcomes”

On the contrary, as evidenced in our Individual Feedback Responses and Case Studies, the dissemination of guidelines does result in behaviour change among...
providers. In theory, this change in provider practice results in decreased chronicity in low back pain sufferers.

Kimberly-Anne Ford
Project Manager, Case Studies and Best Practices
Development Unit, Research and Development
Accident Compensation Corporation

Response

We thank K-A Ford for the points she outlined on our qualitative study.

We agree with her that general practitioners (GPs) understand the ‘Yellow Flags’. However, they chose not to use the Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain. Our GP participants recognised the personal relationships they built with their patients to be fundamental in identifying and managing any biopsychosocial problems. This was of key importance in assessing and managing any symptoms, particularly if psychosocial components were present. Established relationships with their patients allowed them to manage ‘Yellow Flags’ in ways that they individually felt appropriate.

Ford stated that “a recent case study of 461 General Practitioners revealed that 74% of GPs consistently identified psychosocial yellow flags in their responses on the management of acute low back pain cases (ACC, 2005).” We note with interest in the case study cited, that one question asked: “What investigations would you do?” Here, 50% of the respondents actually ordered a variety of haematological and microbiological tests, and diagnostic imaging. Prescriptions for medication for pain and inflammation were given by 99% of the respondents. No management strategies for the ‘Yellow Flags’ that had been correctly identified by the respondents were described.

The study participants themselves did not describe any awareness of a distinct strategy (from ACC) regarding guideline dissemination and implementation. They portrayed the implementation and dissemination of guidelines (by ACC) to be not ideal. ACC case studies and follow-ups were acknowledged in the body of the original unabridged text by the authors but not by the study participants. Nevertheless, all the study participants expressed a marked lack of usefulness of the guidelines. They saw the Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain as unduly mechanistic and prescriptive, with little relevance to the context of their daily work.

We acknowledge that the development of the Acute Low Back Pain Guideline (ALBPG) was subjected to a high level of scrutiny, and as such is often identified as being ‘evidence-based’. However, the New Zealand Guidelines Group (NZGG) stated that they were “unable to endorse the Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain as evidence-based best practice”. The NZGG felt the information in the document was misleading, in that the ‘Yellow Flags’ section of the Guideline was a consensus document and that the systematic literature review only applied to the ‘Red Flag’ section. Consequently, their endorsement by the NZGG was downgraded to a “best practice guideline”.

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Ford stated that the dissemination of guidelines resulted in behaviour changes amongst providers as evidenced in your individual feedback responses and case studies. That in theory, this change in provider practice should result in decreased chronicity in low back pain sufferers. Firstly, this is level 4 evidence. The publication of better quality research evidence in New Zealand to support this theory is eagerly awaited.

To date we have not found any substantial evidence in the literature that use of the ‘Yellow Flags’ Guideline by GPs (other than identification) has resulted in reduced chronicity in low back pain sufferers.

Once again, thank you for the opportunity to debate these issues.

Cameron Crawford
Postgraduate Student, Department of Rehabilitation
Wellington School of Medicine, University of Otago
Wellington

Kathleen Ryan
Senior Research Fellow, Institute of Health and Community Studies
Bournemouth University
Bournemouth, UK

Edward A Shipton
Academic Head, Department of Anaesthesia
Christchurch School of Medicine and Health Sciences, University of Otago
Christchurch
Medical students denied access to normal vaginal deliveries

This is a reply to Misty Curry’s letter “Would somebody please have a normal vaginal delivery?” published in NZMJ 15 June 2007;120(1256).

http://www.nzma.org.nz/journal/120-1256/2595

I have to agree with Miss Curry. I am currently a 4th-year house officer. I trained in the UK and had a total of 9 weeks training as a student in Obstetrics and Gynaecology in 5 years at Medical School. My obstetrics experience was slightly enlarged with a 3-month run 18 months ago and I have just started a 6-month run with the intention of a career in Obstetrics and Gynaecology. My experience of vaginal deliveries over 9 years of medical training is a grand total of 1, as of 4 days ago. I am very familiar with Caesarean sections, in a variety of elective and emergency situations, and have even observed a few instrumental deliveries.

As a 5th-year student (final year in the UK), I was categorically denied the opportunity to observe a vaginal birth in preference to the midwifery student who sauntered into the labour ward after I had spent 4 hours trying to impress the midwives and prove I was worth something. There would obviously be too many people in the room without a lowly, useless medical student poking around.

Shortly before the mother-to-be started pushing, her IV cannula fell out and needed re-sitting. After the midwife loudly complained about having the call the SHO to insert a new cannula, I offered to save her the hassle and re-site the cannula myself. She accepted and then, with a guilt-ridden expression on her face, offered to let me observe the process. The birthing room must have been instantly re-modelled allowing room for me. I was allowed into the room but heavy emphasis was put on the midwifery students (there were now two of them) getting prime views. I was only allowed the leftover spaces.

I saw a vaginal delivery through the gaps between other bodies, and those spaces were so small I don’t remember seeing anything other than some hair and blood.

I have managed to build a good rapport with the Midwives I work with and, as they are all aware of my keenness on a career in Obstetrics and Gynaecology, they are willing to involve me in births. I suspect this enthusiasm may be swayed by the fact I am already a qualified doctor and one day, who knows, I may be their Consultant on-call.

Name and affiliation withheld upon request
Acknowledging best practice in medical education

Postgraduate medical training of recently qualified junior doctors has long thrown up challenges for medical educators. Iwona Stolarek identifies that junior doctors have reported limited perceived confidence when performing intimate examinations following the first 6 months of postgraduate training—which highlights the importance of skill practice, assessment, and informative feedback.

The acquisition of practical wisdom, phronesis, encapsulates positive contributions (role models, maturity and self-directed learning) and negative influences (the pedagogical approach in medical schools and the harshness of an adaptive healthcare system leading to cynicism) on the naive medical student, through to the competent and experienced medical practitioner. Despite the model’s limitations, coupled with the understanding of how adults learn effectively, such as through the adult learning theory of andragogy, they provide the platform for considering potential contributors and barriers to be addressed in the deliverance of postgraduate medical education.

Attainment of intimate examination experience, such as during the bimanual examination of the female patient learnt in the gynaecology setting, carries additional dilemmas such as the consideration of overstepping patient autonomy, access to adequate numbers of consenting females with or without general anaesthesia, and a gender-gap. These factors can limit the practical opportunities in this specialty as a medical student and as a junior doctor, and the knock-on effect could be to contribute to a poor confidence rating for the skilled examination.

Stolarek measured the perceived self-confidence of postgraduate house surgeons; our own recent research (due to be submitted for publication) has indicated the importance of making the distinction between confidence and competence—medical students and junior doctors showing much unease in committing towards the latter quality. We conducted a focus group study involving medical students (n=17) and separately first-year junior doctors (n=7) in which the doctors perceived limited skill progression in intimate examinations. However there was a strong awareness of the need to seek assistance from an experienced colleague when undertaking a relatively unpractised physical examination.

The Department of Health in the United Kingdom, launched a strategy called Modernising Medical Careers (MMC) in 2005 to provide a career structure for doctors through a reform in postgraduate medical education. MMC has streamlined training and has set explicit standards in assessing the competency of medical graduates, by establishing a formal training-based national curriculum, and providing regular workplace-based assessments against explicitly defined standards of competence for the 2 years as a junior doctor. Likewise, the attempt to modernise and explicitly standardise training programmes (for a safer patient population) could be adopted across all the healthcare professions.
It is crucial to universally acknowledge examples of best practice, in addition to the identification of barriers, to deliver a balanced curriculum to improve existing training schedules that facilitate improved confidence and competence of junior doctors. We would therefore agree with Stolarek that we should consider alternate means for skills’ training and further that we suggest that investing efforts in the provision of postgraduate education could have a positive impact on the professionalism of junior doctors and on a safer patient population.

Andrew Carson-Stevens  
Medical Student  
(carsonstevensap1@cardiff.ac.uk)

Iain J Robbé  
Clinical Senior Lecturer  
Cardiff University, Cardiff  
Wales, UK

References:
Systemic health system problems

Dear Pete and Helen,

I am not saying management of medicine is easy.

But there is something wrong. It would appear obvious from the inside, where I have been for the last 36 years, where the lesions lie.

I was chairperson of the Area Health Board GP Liaison Committee in Canterbury when the implementation of Gibbs report was tried. I was around when restructuring of the restructuring (no it is not a typo error, it should be repeated 5 times) of the structures of management in the health care through the 90s was tried and I, as you have, experienced the frustration we all have had of the huge cost rises in health care not having the desired result of better penetration of delivery.

I have some observations.

Prior to 1980 there was a distance between doctors and the needs of the community but at the time this was the norm. Change was in the air. A dramatic rise in technology occurred with the development of scanners, ability to solve more problems, and newer pharmacology. At this stage in the medical part of the health industry there was still some ownership and reward for that ownership. This ownership was returned in the way of goodwill—being unpaid on-call; going the extra mile for your patient; having direct discussion from GP to Consultant; and a community-type responsibility in the wards with a stable structure of medicine, nursing, and paramedical staff. I am not saying this was all good, but people were communicating with people and direct accountability was there.

I was part of the 1970/80s consultation process, or should I say part of the farce of the process. I choose not to be a part during the 90s. All of a sudden we had a large number of middle management, well-meaning transient experts appearing out of nowhere, and the process of distancing began. Goodwill was lost as ownership was lost. The extra mile and good communication were lost, and control of costs was lost as layering of accountability hit the wards and other infrastructures.

General Practice was a little isolated because the cost escalation was not so dramatic. Of latter times, micromanagement of doctors in General Practice crept in and now we have arrived at the thick end of the wedge where there is a significant perception in General Practice that salaries are the way it will go. Capped fees and expensive review processes (over, in some cases, a few cents) have reinforced this viewpoint. This will complete the disenfranchisement for me and many others like me. I will leave the workforce earlier than I expected.

The stable medical workforce is aging, the wonderful variety of new practitioners has brought with it huge communication and management issues. This situation has, in part, been fuelled by the fiscal issues of new local graduates. The introduction of upskilled nurses as a new force in primary care is to be commended, but these people are at great risk as their training is often woefully inadequate and at time experimental; reliant at times on the experiences from older practitioners. Protocled
disease management is a road that is expensive and caution signs should be everywhere.

There has never been this chaos and disharmony in our medical workforce.

Strikes—what on earth is going on?

Failure to access medical care by lower decile and other community groups, failure to deal to waiting lists, removal of deserving folks from the lists—all this is unnecessary.

I am not suggesting for a minute that I have any idea of the political intricacies you folks have to deal with, but I do have a few suggestions for the part I do understand.

Re-introduce ownership to the people who do the work.

Put the experienced intellectual firepower at the front door of the health care industry—e.g. specialists as front-line ED staff; the house officer strike gives us some insight in to the efficiency gains of this manoeuvre.

We have to get it right at the first point of contact, teaching by experience come second to providing world-class health care to the individuals in the small population we have. How many health boards do we have and how many repetitions of waste can we afford?

We don’t need many more specialists on a world scale, but we do need them, and to get them we need to pay world rates.

Where did the last $400 million go?

Sanity save New Zealand.

John Cook
GP, Pegasus Health
Christchurch
Metoprolol-induced hyperkalaemia in chronic respiratory acidosis

The combination of nonselective beta-adrenergic antagonists with acute respiratory acidosis can cause severe hyperkalaemia.\(^1\) On the other hand, it is not known whether cardio-selective beta-blockers in patients with respiratory acidosis can also increase blood potassium concentration.

We investigated this hypothesis in a cross-sectional study of 327 adult patients.

Mr C aged 44 years was admitted to the Auckland City Hospital in October 2004 with acute bacterial endocarditis of the tricuspid valve secondary to intravenous morphine abuse and multiple localised intrathoracic pus collections. Soon after, the patient’s *Enterobacter cloacae* empyemas were drained under CT guidance and he became afebrile.

The patient’s medications consisted of methadone orally for morphine withdrawal, meropenem intravenously, paracetamol, amitriptylline, ondansetron, and omeprazole. However, because of persistent sinus tachycardia, the dose of metoprolol CR had been increased from 47.5 mg to 95 mg per day. Three days later, the patient’s blood biochemistry results yielded a raised potassium level up to 6.9 mmol/L, normal creatinine (creatinine clearance = 80.1 ml/min) and creatinine kinase levels, and mildly deranged liver function tests.

The patient’s full blood count revealed presence of anaemia (Hb = 90 g/L) and leukocytosis (WCC = 20.06 × 10\(^9\)/L, segment neutrophils 17.85 × 10\(^9\)/L). An arterial blood gas analysis demonstrated an acute on chronic respiratory acidosis with type 2 respiratory failure with pH of 7.277 and potassium concentration of 6.4 mmol/L.

To treat the hyperkalaemia, the patient was started on frequent salbutamol nebulisers and calcium resonium tablets, but potassium concentration still remained elevated for 3 consecutive days. Then a combination of an increased dose of metoprolol and respiratory acidosis was suspected as a possible cause of the hyperkalaemia. Metoprolol, salbutamol, and calcium resonium were subsequently discontinued and the dosages of methadone were significantly reduced. As a result, blood potassium concentration normalised.

To find out whether hyperkalaemia of 6.4 mmol/L or higher can be caused by acidosis alone, 1869 subsequent blood gas specimen of 326 successive patients admitted to the hospital in January 2007 were reviewed. Inclusion criterion for the study was pH range of blood samples from 7.25 to 7.29. Exclusion criteria for the study were venous and capillary blood, age less than 5 years, and a raised serum creatinine together with potassium concentration.

Thirty-seven arterial blood test results of 21 patients were obtained. They were used as a control group. The Stata/SE (version 9.2) software package was used for statistical analysis. One sample t-test, and tests for skewness and kurtosis, were deployed to evaluate the study hypothesis.
The mean pH in the control group was 7.274, and the standard deviation was 0.016 (95% CI: 7.269–7.279). The pH distribution had skewness of -0.25 and kurtosis of 1.47, and was not normally distributed: kurtosis test of normality yielded p<0.001 and joint skewness/kurtosis test had p<0.001. One sample t-test of having pH 7.277 demonstrated t(36) = -1.1532, p>0.05.

95% CI of the arterial blood tests in the control group included the value of 7.277. The mean potassium level in the control group was 4.23 mmol/L, standard deviation 0.56 mmol/L (95% CI: 4.05–4.42 mmol/L). See Figure 1.

The skewness of potassium distribution was -0.068 with kurtosis of 2.195. Skewness and kurtosis tests for normality of potassium distribution in the control group showed p=0.847 for skewness and p=0.235 for kurtosis thus confirming a normal potassium distribution pattern. The probability of having potassium level of 6.4 mmol/L and higher in the control group by chance was calculated with a one-sample t-test. It showed t(36) = -23.680, p<0.001 thus confirming that acidosis in the control group is highly unlikely to account for such a high serum potassium concentration.

Beta-adrenergic agents have been implicated in regulation of potassium homeostasis. For example, beta$_2$-adrenergic agonists by stimulating the Na$^+$, K$^+$-ATPase pump and insulin secretion endorse potassium uptake by the cells, thus decreasing plasma potassium concentration.

Albuterol, a nebulized formulation of beta$_2$-agonist, is widely used clinically in acute management of hyperkalaemia.
In animal experiments it has been shown that an acute respiratory acidosis and nonselective beta-blockers can produce an elevation of plasma potassium concentration.\(^1\) Nonselective beta-blockers can also cause hyperkalaemia when other predisposing conditions such as renal impairment, heart failure, and hypoaldosteronism are present.\(^1,5–7\)

Importantly, acidosis by itself can lead to an elevation of plasma potassium concentration.\(^8\) Therefore, to distinguish potassium rising effect of a combination of moderate dose of metoprolol CR with chronic respiratory acidosis from acidosis alone, we investigated the probability of having plasma potassium concentration of 6.4 mmol/L and higher in the control group.

It has been shown that in the control group hyperkalaemia to the degree of what our patient had is highly unlikely to be explained by acidosis. Thus, it can be concluded that a combination of a moderate dose of metoprolol CR with chronic respiratory acidosis may be the possible cause of patient’s hyperkalaemia.

Andrei M Beliaev
House Officer
(andreib@adhb.govt.nz)

Warren Smith
Associate Professor, Consultant Cardiologist
Greenlane Cardiothoracic Surgical Unit
Auckland City Hospital, Auckland

References:

Lessons from Hong Kong and other countries for outdoor smokefree areas in New Zealand?

On a recent stay in Hong Kong, one of us (NW) noted the striking success of outdoor smoking restrictions (introduced in January 2007). No smoking was observed in four smokefree parks and two smokefree beaches, during a total of eight visits to these places (in May 2007). This was despite these outdoor settings being well frequented by adults and during fine weather on all occasions. Furthermore, no cigarette butts were observed in any of these sites. The smokefree signage at the entrances to these outdoor settings was very noticeable and often included large banners (e.g. Figure 1).

Figure 1: Typical signage for an outdoor smokefree area in Hong Kong in May 2007

Hong Kong law bans smoking in the open areas of hospitals (both public and private); open areas of all schools, including university campuses; public bathing beaches; public swimming pools (pool areas and spectator stands); the Hong Kong Stadium, Hong Kong Wetland Park, and Mongkok Stadium (turf pitch areas and spectator stands); and at public transport interchanges. The Hong Kong Housing Authority has banned smoking in all common areas of public rental housing estates, from April 2007. These common areas of the large estates include ‘roads, pedestrian paths, … gardens, play areas and sports grounds’
with ‘no more than five smoking areas on each estate,’ … each of ‘about 5 square metres’. 4

Worldwide, there are a number of jurisdictions where smoking is banned outside on beaches, in parks, playgrounds, stadiums, bus shelters, the outdoor sections of hospitality venues, and in the outdoor areas of the whole town of Calabasas, California. 1,5 These smokefree areas include all Californian public playgrounds, 6 and all park, sports fields, playgrounds, beaches, and bus shelters in Mosman, Sydney. 7 A number of jurisdictions ban smoking near building entrances, including Washington State in the USA. 8

There are three main arguments in favour of banning smoking in outdoor areas:

- Preventing adverse role-modelling for children;
- Preventing exposure to secondhand smoke; and
- Reducing litter from smoking-related materials.

The example of smoking by adults is a crucial factor in youth starting smoking and quitting. 9–15 Increased smokefree areas change norms about smoking, and reinforce to smokers and youth the severe risks from tobacco use. 16

The New Zealand Government’s *Framework for Reducing Smoking Initiation in Aotearoa-New Zealand* 17 has increased the policy emphasis in New Zealand on reducing the exposure of children to smoking behaviour, in order to reduce smoking uptake. One avenue to decrease this exposure is the introduction of smokefree playgrounds, parks, and other outdoor areas.

Besides the risks from the example of smoking by others, the evidence base around hazardous air pollution from outdoor smoking in various settings is also growing. This work indicates levels of fine particulates (PM$_{2.5}$) that are sometimes at hazardous levels. 18–21 There has also been preliminary work on elevated PM$_{2.5}$ levels in outdoor smoking areas of hospitality venues in New Zealand. 22

Smoking-related materials (particularly cigarette butts) are also leading components of litter. 23 Furthermore, discarded butts can constitute a fire-hazard in some outdoor settings.

There is a need to expand the evidence base, for example by conducting evaluations of the outdoor smoking restrictions that have already been introduced in New Zealand. These currently include the grounds of all schools, some council-owned parks (e.g in South Taranaki and Upper Hutt), the grounds of some hospitals, stadiums, and a university campus (Massey).

Research is also desirable to clarify the potential benefits of reducing outdoor smoking in areas frequented by children (e.g. parks) as part of the long-term denormalisation of smoking and to avoid role-modelling of smoking behaviour.

But while more research is clearly desirable, a precautionary approach suggests a need for further restrictions now, especially for parks and sports fields used by children and for semi-enclosed smoking areas in hospitality settings. We encourage the Cancer Society and the Health Sponsorship Council to continue their work with councils for smokefree parks. There is also a need for additional civil society action to get councils to adopt smokefree by-laws for hospitality settings.
Acknowledgements: This work was partly completed within the “Reducing Smoking Around Children” research project, funded by the Health Research Council of New Zealand.

Competing interests: The authors have all previously undertaken work for the Ministry of Health and/or non-governmental agencies working to improve tobacco control.

Nick Wilson, George Thomson, Richard Edwards
Department of Public Health
University of Otago, Wellington
(nick.wilson@otago.ac.nz)

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Reply to the editorial “World No Tobacco Day (31 May 2007)—did anybody notice?”

We thank the editorial authors, Lutz Beckert and Roland Meyer, for their support of New Zealand’s Quitline smoking cessation service. The authors comment on the decrease in Quitline callers between 2001 and 2005. They sourced their data from a recent article on first-time Quitline callers, but didn’t highlight that this paper referred to first-time (i.e. new) Quitline callers only. The decrease in new Quitline callers between 2002 and 2005 could be a hangover from the introduction of subsidised Nicotine Replacement Therapy (NRT) in late 2000, which resulted in a huge increase of callers to the Quitline.

While it is true that there has been a decrease in new caller numbers, we wish to point out that the Quitline also provides a service to relapsed callers. Relapsed callers are those who have enrolled on the Quitline programme in the past but are re-contacting the service to give quitting another go. The proportion of all callers who were classified as relapsed callers increased from 17% in 2002 to 24% in 2003 and 28% in 2004 and 2005. While the number of new callers decreased between 2003 and 2005, the number of total callers remained steady at about 32,000 per annum.

Data from 2006 shows that there were 24,070 new callers and 8485 relapsed callers registered with the Quitline—a total of just over 32,000 callers in that year.

Judy Li and Michele Grigg
Researchers, The Quit Group
Wellington

References:


Diana Manby Mason

Prominent doctor and anti-abortion campaigner (29 July 1922 – 5 June 2007)

Diana Mason, OBE, was half of a dynamic marriage with the late writer, playwright and actor Bruce Mason. They were both acclaimed in their fields—she the sciences, he the arts—and were high profile anomalies for their era. Dr Mason was a doctor when women interested in medicine were usually nurses and Bruce Mason was a writer when that was not the done thing for a man.

Even more unusually, Dr Mason who possibly had little choice if she were to fully encourage Bruce’s artistic pursuits, which she did, was the breadwinner for the family. She initially struggled with the concept but finally decided that “the sciences would support the arts.”

But she was far more than a financial helpmate. She practised as a doctor for more than half a century and made a long, deep contribution to women and children, recognised with the OBE in 1977. She was also a flamboyant part of Wellington’s cultural life, guaranteed to stand out in the crowd at orchestra or theatre events, tall, proud and magnificently got up.

Dr Mason was born in Wellington, her father an import-exporter and her mother an energetic, intelligent woman, who not content with being a housewife, ran a physical culture studio for women and believed people needed to be fit. Dr Mason remembered her father pedalling a bicycle in the laundry to grind wheat sold in her mother’s studio.

Her interest in health arose in those early days but her father expected this would be the basis for a career in nursing. Not for his strong-minded, intellectually able daughter. She would be a doctor.

Her education and her propensity for asserting herself had begun early in her life when, at 3, she hammered on the door and demanded to be let into the kindergarten her mother had begun in Karori for the benefit of her older sister. Consequently, the two girls, 18 months apart, went through school together. Dr Mason was Dux at Marsden College and at university at 16 and, despite not having taken science subjects, flew through medical intermediate in 1 year.

She met her husband when she was 17 and he 18. He “slowly bewitched her,” she recalled in an anthology of successful New Zealand women in which she was included years later, “by his quicksilver mind, and outpouring of words and his stunning piano playing.” Later they corresponded voluminously while he was away during World War II. It took a year after he came back for them to get to know and treasure each other again.
She qualified for Medicine on 14 December 1945, and they were married shortly afterwards. Dr Mason worked for 2 years as a house surgeon in Wellington Hospital then, planning on going to Britain, worked as Dr Bill Shirer’s practice assistant in Newtown for 2 years. By the time she had enrolled in Great Ormond Street Hospital for Sick Children for postgraduate study, the couple’s first child was 3 months old.

In London they met up with theatre luminaries Richard and Edith Campion and lived with them in Chelsea. London was an excellent place for Dr Mason to further her career but their 3 years there were a time of rejection and frustration for her husband, who failed to find a place in theatre, journalism or music and ended up teaching children involved in screen and stage work.

In a moment when she was fed up with exams, and realising London was not the place for Bruce, the decision was made to return to New Zealand. Before long, and after a time on her parents’ orchard in Tauranga, she was back in the Riddiford St surgery and her husband was writing from home. Legend has it that he was one of New Zealand’s first house husbands. His indomitable wife usually cooked the dinner, through when the doctor was called to deliver a baby he could turn a hand at the stove.

Soon after her return to New Zealand Dr Mason had realised that her heart was no longer in paediatrics, but that obstetrics was becoming her passion. She delivered thousands of babies—“enough for a whole town,” she once remarked. She never lost her infectious joy at being there at the moment life kicked in.

Her immovable belief in the sanctity of the lives she ushered into the world led her to head the anti-abortion Society for the Protection of the Unborn Child (SPUC) in the 1970s. She and her family were vilified for her high-profile adherence to a view that was usually considered peculiar to Roman Catholicism.

Her intense belief was driven by philosophy rather than religion. She considered herself a “nominal Anglican” and not at all religious. She eventually left—her views unchanged—to serve 9 years on the New Zealand Medical Disciplinary Committee.

She was also strongly against euthanasia, which she believed too open to abuse. She profoundly believed that no one had the right to take human life.

Her OBE was awarded for services to obstetrics, particularly to young single mothers at the Alexandra Maternity Hospital and Home for Unmarried Mothers where she was medical superintendent (1958–78). It was an era when young single women were almost expected to give up their babies for adoption. For Dr Mason it was the flipside of her abhorrence of abortion. She saw it was doing what she could for women who had a baby they believed they could not look after. She also illegally provided contraception to girls under 16 where she saw a need. She advocated “family planning rather than child destruction.”

Through her long career till her husband died of cancer in 1982 the couple led rich social lives, read, travelled and attended the capital’s notable theatrical and musical events.

After Mr Mason was diagnosed with cancer and had decided on no further treatment, the couple took an extended overseas trip to all the places they had previously enjoyed together.
She and the children were with him when he died in the art, book and music-filled old Kilbirnie house they had shared. Long after his death she said that his absence left her with “an aching void that might have surprised even him, and that nothing—no music, no golf, no family, no friends, ever fills.” That remained true. Such a vibrant meeting of minds and such an exceptional marriage encompassing two such forceful careers would have been impossible to consign to history, though she did her best to be a whole Diana, as a friend had exhorted, rather than half of Bruce and Diana.

She gave up working reluctantly at 78 and enjoyed University of the Third Age lectures on music, Shakespeare and art history.

She moved out of the Kilbirnie house in 2005 and spent time in an apartment and then her last year in the Rita Angus complex where she was already known to all the nursing staff and had been doctor to many of the residents.

Dr Mason is survived by three children, five grandchildren and five great-grandchildren.

This obituary was written by Diana Dekker and appeared under the heading Mason more than just a helpmate in the Dominion Post (Wellington) on 7 June 2007. We are grateful to the Dominion Post for permission to reproduce it here.
Alan Guibal Bradford

Anaesthetist (19 October 1923 – 10 April 2007)

Dr Alan Bradford, universally known as Brad, was Nelson’s first and long-serving specialist anaesthetist.

Brad was born in Sheffield UK, where his father held professorial posts in education and psychology. His unusual middle name derived from Channel Islands maternal antecedents.

He was educated at Netheredge School, then studied medicine in Sheffield during the staff depleted wartime years, graduating MBChB in 1947.

He then served his 2 years National Service in the RAF in Berlin and Northern Scotland.

He was awarded a Fulbright Scholarship for 1949, spending the year in the USA in hospitals in Elizabeth (New Jersey) and Bute (Montana).

He came to New Zealand in 1952, working his passage as ship’s doctor. He had spells in general practice in Collingwood, Ranfurly, and Plimmerton, then took a registrar post in obstetrics at National Women’s Hospital Auckland in 1954. However, he resolved to return to England at the completion of the year, as he “saw the sunrise too often for my liking”.

Four weeks prior to Brad’s scheduled departure, final-year Otago medical student Kay Johnstone began her obstetrics attachment. In what must have been a whirlwind 6 weeks they became engaged, Kay sat her finals, then they married. They remained in Auckland, Brad switching to anaesthesia, Kay training in child psychiatry.

Brad completed his anaesthesia training in Auckland, one of the early trainees of the recently structured Faculty of Anaesthetists, Royal Australian College of Surgeons. He qualified FFARACS in 1960, and was secretary/treasurer of NZSA in 1960–61. They then spent 4 years in Melbourne, Brad having an appointment at the Royal Victorian Eye & Ear Hospital, plus private practice involving ever increasing time spent travelling between various hospitals with the resultant long hours away from home.

Looking for a smaller centre lifestyle and perhaps with Brad nostalgic for the earlier time spent in Collingwood, in 1965 they both accepted positions in Nelson—Brad as the city’s first specialist anaesthetist and Kay as the Superintendent of Braemar Psychopaedic Hospital.

The Braemar Superintendent’s residence, with its lawns, tennis court, and treed backdrop, became the centre of a social round hitherto not experienced by the Nelson Medical community. Brad, tall, tweeded, every inch the English country gentleman, contrasted with Kay’s exuberant and edgy fashion style.
Anaesthesia in Nelson in 1965 was provided on a sessional basis by visiting general practitioners, of varying seniority, whose expertise was acquired on the job and who used a very limited range of drugs and techniques. To quote the then hospital superintendent, “Brad proceeded to gently elevate the standard of general practice anaesthesia”. Being the town’s first specialist meant acquiring the operating sessions associated with the most demanding clinical cases and the more demanding surgical personalities.

One visitor enchanted by the Bradfords’ hospitality was Professor Eckenhoff of Chicago, touring in 1968 as the ASA/NZSA visitor. At his invitation Brad spent 1970 in Eckenhoff’s department at Northwestern University, returning to Nelson with new expertise in regional techniques, not then commonly used in New Zealand.

Brad had infinite patience, and unfailing tact, courtesy, and equanimity, making him a reassuring presence for surgeons, theatre staff, and patients. He considered that Nelson Hospital’s best interests were served if it could attract as house surgeons the most confident and ambitious of new graduates (who might return to specialist posts), and that the absence of a registrar tier allowed early responsibility for these young doctors. Arguably this was valid reasoning in the far off days before student loans and shift work for juniors.

Thus Brad declined approaches to include Nelson in registrar training schemes, the consequence being that he spent his entire career up to retirement at 65 taking first call at nights and weekends. The demands of call evolved considerably, becoming more onerous as surgical throughput expanded beyond the daytime capacity of the 1950s design theatre suite. On the other hand, as the older generation of GP anaesthetists progressively retired, the load became shared by specialist colleagues, a second in 1972, third in 1980, and fourth in 1984.

After Brad’s retirement, Kay continued working, latterly as Nelson Hospital Chief Medical Officer. On her retirement from this post, they moved to Hamilton where Kay continues to work in child psychiatry. Sadly, Brad’s retirement was marred by the onset of Parkinson’s disease.

Brad is survived by Kay, sons Charles and George, and daughter Polly, a Medical Officer in Psychiatry in Wellington.

Allan Grant (Anaesthetist, Nelson-Marlborough District Health Board) wrote this obituary.
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
Arthur Leslie Batt [obituary]

An question mark was inadvertently introduced by the NZMJ Production Editor into a quotation in the penultimate paragraph of the Arthur Leslie Batt obituary published in N Z Med J. 2007 (15 June);120(1256). http://www.nzma.org.nz/journal/120-1256/2600 (full text), pdf, and Full Contents pdf

We apologise to Dr Brabazon, the Batt family, and our readers for this typographical error.

Please refer to the above links for the corrected copy, and replace page 127 in the Full Contents pdf.

NZMJ