THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

CONTENTS

© NZMA

Medical Image

Bodypacker

Methuselah

Selected excerpts from Methuselah

Letters

Buteyko pseudoscience
Shaun Holt, Richard Beasley

The Green Prescription
Janet Copeland

Chronic idiopathic singultus: is there life after cisapride?
Georg Petroianu

Medicolegal

Medical discipline - professional misconduct

Medical discipline - not guilty

Medical discipline - charge struck out

Obituary

Alister Douglas George Bowron

Notice

The Hawke's Bay Medical Research Foundation Inc: Medical Research and Study Grants

Book Reviews

Multiple sclerosis: immunology, pathology, and pathophysiology, Robert M Henderson (ed)
John Fink

Life on cripple creek: essays on living with multiple sclerosis, Dean Kramer
John Fink

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

This Issue in the Journal

Illness beliefs and adherence in diabetes mellitus: a comparison between Tongan and European patients
L Barnes, R Moss-Morris, M Kaufusi

The aim of this study was to determine the way in which Tongan and European people with Type 2 diabetes conceptualise their illness and how these beliefs relate to diabetes self-care regimens. Findings highlight differences in personal beliefs and self-care between these groups, with beliefs that characterise Tongan patients tending to be associated with poorer adherence to diet and medication taking.

Pregnancy planning by mothers of Pacific infants recently delivered at Middlemore Hospital
J Paterson, E Tumama Cowley, T Percival, M Williams

Within the Pacific Islands Families cohort study, 1365 birth mothers were interviewed six weeks after they had given birth about pregnancy planning. Forty per cent of the mothers reported that they had planned their pregnancy. Of the 60% of mothers who had not planned their pregnancies, 70.8% were not using contraception when they conceived. The accessibility and acceptability of family planning services need to be examined to ensure that they are delivered in a way that maximises choices regarding the use of contraceptives.

Regional variations in asthma hospitalisations among Maori and non-Maori
L Ellison-Loschmann, R King, N Pearce

We studied asthma hospitalisations for Maori and non-Maori living in urban or rural settings during 1994–2000. Asthma hospitalisation rates were higher in Maori than non-Maori in all age groups and the differences (though not large) were higher in rural than in urban areas. The higher admission rates among Maori may reflect differences in asthma severity as a result of reduced access to health services, which may be a more acute problem for those in rural areas.

Motor vehicle traffic crashes involving Maori
M Sargent, D Begg, J Broughton, S Stephenson, C Wright, J Baxter

The aim of this study was to describe factors associated with fatal and non-fatal motor vehicle traffic crashes involving Maori from 1980–1994, inclusive. The results were based on 8178 crash events involving 8273 vehicles and 9288 Maori casualties. Most Maori casualties were males aged less than 34 years. Most crashes occurred in fine weather, on sealed, two-way roads, in or near an urban area. Some implications of the findings and the need to improve data quality are discussed.

© NZMA

Representative case series from public hospital admissions 1998 I: drug and related therapeutic adverse events
R Briant, W Ali, R Lay-Yee, P Davis

This is an analysis of drug and related therapeutic adverse events (AEs) identified from medical records in New Zealand public hospitals in 1998. These events tended to be more common among older patients, with cardiovascular drug AEs the dominant group and inadequate monitoring and follow up the most common reason for failure to prevent. Many drug-related AEs are preventable, with better monitoring and more appropriate medication choice for individuals the most common prevention strategies.

White-tailed spider bites – arachnophobic fallout?
J Banks, P Sirvid, C Vink

Case histories of patients admitted to Christchurch Hospital were investigated to see if they reflected public concern regarding the effects of white-tailed spider bites. We found no evidence that patients developed necrotising arachnidism, a condition mistakenly attributed to the bite of white-tailed spiders. Both New Zealand and Australian scientific and medical literature were reviewed. We conclude that the public's fear of white-tailed spider bites is unfounded and, unless the (painful) bite is felt, alternative diagnoses should be considered first.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Going from bad to worse?

Frank Frizelle, Editor

We continue to practise in interesting times. Sometimes I wonder if things are going from bad to worse. New diseases keep popping up. Last year we thought SARS was bad, yet this year Asian chicken viruses threaten to be worse. Treatments we thought were good, such as HRT, turn out to be bad. The inequalities between different racial groups in regard to access and outcome in medical care are now well described, but there is little evidence that we are able to do much about them. We are increasingly recognising that adverse events are part of the faulty human system of medicine; however, in parallel, we are experiencing the increasing crimination of medical error. New laws affecting doctors, which were designed to make matters better, appear to be making matters worse. To cap all that, our young doctors are said to be leaving New Zealand as fast as they can get their medical degree.

So, should we all be despondent at the beginning of a new year, or is there reason to hope that things will improve? Last year New Zealand was fortunate not to be too affected by the SARS crisis, but this year we are told that officials are braced for a flu pandemic. Besides making the appropriate preparations, there is little we can do to protect ourselves from this threat other than cross our fingers and hope that our remote location will once again work in our favour.

Health access and outcome for different racial groups is perhaps an area over which we should feel able to exert somewhat more control. This issue of the Journal presents further studies that demonstrate the inequalities existing in New Zealand. Over the last year we have published a number of similar studies highlighting these inequalities; the real issue now is what can be done about them. The problems are complex and often relate more to the socioeconomic realities of different ethnicities than anything else, and these are difficult to change. We urgently need more research aimed at providing solutions to these inequalities, not just describing them.

Legal aspects of medical care continue to dominate the media and be of major concern to most doctors. Hard-working medical professionals are repeatedly held up by the media as bad doctors and bad people, when in truth reporters are often relaying unsubstantiated allegations. It appears increasingly likely that doctors will not be able to get name suppression for cases waiting to be heard by the Medical Practitioners Disciplinary Tribunal. The Health and Disability Commission seems to be keen avoid name suppression, as this prevents other cases related to a medical practitioner coming forward.

The Medical Council likewise seems to have forgotten that it needs to be there for both the doctor and the patient, at times withholding doctors' annual practising certificate when they have yet to appear before a tribunal. Does this mean that if the Director of Proceedings decides to charge us we must be guilty; if so, his/her role becomes increasingly critical. The HPCA Act comes into effect this year, and the influence of this is uncertain. However, it seems likely that it will at the very least increase Ron Paterson's workload.

© NZMA

The retention of doctors continues to be a problem and is a subject that the media have recently picked up. However, there always have been and always will be plenty of young doctors heading overseas. Many of them do return. New Zealand also continues to attract many quality doctors from overseas. Despite this, there are many concerning aspects about retention of doctors in the public system, with surgical specialists increasingly opting out of the public system into full-time private practice. Another worrying trend is the reduction in the number of medical students coming from families where at least one parent is a medical doctor. It is difficult not to wonder what advice doctors are giving their children about careers in medicine.

The New Zealand Medical Journal will continue to publish articles of interest to New Zealand medical practitioners, covering a broad range of subjects from Asian chicken flu to the HPCA Act and as much in between as best we can. Last year we published more original manuscripts than ever before (35% of submitted papers). We still have an average turnaround time from submission to publication of 3–4 months. The biggest delay relates to the time it takes to find available reviewers (who continue to do an excellent job). The Medical Image and Case Notes sections we launched last year appear to have been well received. We are particularly grateful to our colleagues working behind the scenes to provide material for the Medical Image, 100 Years, Methuselah and Obituaries sections.

No major changes are expected in the structure of the Journal over the next 12 months; however, as can be seen from the medical news we never know what is around the corner. By continuing to showcase research of which New Zealand can be proud, and opening up issues for serious debate, we hope to make a positive contribution in the coming year to the efforts of the medical community in addressing some of the challenges it faces.

Correspondence: Professor Frank A Frizelle, Department of Surgery, Christchurch Hospital, Private Bag 4710, Christchurch. Fax: (03) 364 0352; email: frank.frizelle@cdhb.govt.nz

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Ethnicity, acculturation and health: who's to judge?

Tony Blakely and Kevin Dew

If socioeconomic factors were the only driver of ethnic differences in mortality, Pacific people in New Zealand should have the highest mortality rates. However, Pacific people have intermediate mortality rates between Maori and non-Maori non-Pacific people[1] (although specific health problems such as infectious disease, stroke and diabetes are of pressing concern among Pacific people). Clearly, ethnic differences in health are due to more than just socioeconomic position. It is interesting to speculate whether and how Pacific *culture* has protected Pacific people from the full impact of lower socioeconomic position, and whether any such protection will be maintained into the future with their increasing acculturation. Unfortunately, culture is examined all too often in deficit terms in attempts to explain ethnic differences in health. Research and understanding of the beneficial effects of Pacific culture (and both Maori and the dominant Pakeha culture) on health is required in New Zealand, not just for its own intrinsic value but because it may identify positive policy options for reducing inequalities in health.

In the current issue of the NZMJ two papers address Pacific health issues and cultural contexts.[2,3] Neither paper defines culture, but both imply a definition of culture as a way in which 'human groups create and share explanatory systems about the world in which they live and the ways in which they act according to their shared understandings'.[4] Culture is dynamic, perhaps particularly so when confronted with different explanatory systems. The change in one culture in response to another has been termed 'acculturation', or in some situations 'assimilation'. These papers raise the issue of assimilation and its possible health benefits. Assimilation into a dominant culture has the potential to increase one's 'cultural capital', a term that relates to forms of knowledge and language that are privileged. Cultural capital is a resource that provides a return, for example, in educational success.[5] As such, cultural capital is linked to socioeconomic position and life chances.

Barnes et al focus on Type 2 diabetes (clearly a major health problem for Pacific people), and compare health beliefs and healthcare adherence among Tongan and European patients.[2] In this instance, the authors identify beliefs commonly held by Tongan people with diabetes that (from a largely Western paradigm) are unlikely to be conducive to successful treatment and control of their diabetes: beliefs that God's will and other external factors are aetiologically important for their diabetes, and that their disease is acute and cyclical rather than chronic. However, further interrogation of the data (Table 3) supports the hypothesis that such beliefs are associated with poorer adherence to diet and medication. The implicit message to healthcare practitioners is that asking about Pacific people's beliefs regarding their disease, not just their knowledge or understanding of their disease, is important. However, Barnes et al take a rather individualising model of health beliefs, where the solution is largely to alter incorrect beliefs. We could take the questioning of culture further, and ask what social roles these beliefs play. Why do some members of the Tongan community adhere to diet and medication and others not? If health beliefs sustain other important

© NZMA

social structures and interactions (such as the place that food may have in social interaction), altering the beliefs of individual patients alone is unlikely to result in substantive benefit.

Sixty per cent of pregnant Pacific mothers in the study by Paterson et al had an unplanned pregnancy.[3] If one accepts the (normative) standpoint that unplanned pregnancies are disadvantageous, and there is much empirical evidence that the social outcomes for the mother, child and family are likely to be worse than for planned pregnancies, this study suggests that more needs to be done to provide contraceptive advice and services to Pacific women. Here, too, culture and acculturation are important. Higher education, being born in New Zealand, and living longer in New Zealand (indicators of increasing cultural capital) are all predictive of a planned over an unplanned pregnancy. Contraceptive choices and behaviour are about more than just culture, though – for example, and most obviously, gender is also a major factor. Park et al, in their research on sexuality and reproduction, found that in the Samoan community children were seen as God's gifts and a blessing.[6] For the younger men contraception was a part of God's way of teaching them to plan, but for the older men the suggestion that they could not provide for a large family would insult their masculinity. An important point to take from this finding is that the notion of God's blessing can be assimilated into quite different behaviours.

Two other papers in this issue consider Maori health from an epidemiological perspective.[7,8] Ellison-Loschmann et al add to the evidence base on Maori/non-Maori differences in asthma by reporting that asthma hospitalisation rates among Maori are higher than among non-Maori,[7] despite most other studies finding a similar prevalence of asthma by ethnicity. At a further level of detail, the elevated rates of asthma among Maori were most pronounced in rural areas. (However, one has to be cautious at this level of analysis – undercounting of Maori deaths (although not hospitalisations) has been shown to be less common in rural areas compared with urban areas.[9]) Assuming the pattern is real, the results again point to the importance of accessibility to health services. However, it is not clear which aspect of health services is important. Is it the access to primary care, quality of primary care delivered, preferences of the caregivers/whanau, or even simply the patient's distance from secondary health services? Given that rural Maori communities are often long distances from hospitals, a lower threshold for overnight hospitalisation may be a desirable and prudent measure in case symptoms flare up again overnight.

The final paper on Maori road crash injuries (fatal and non-fatal) by Sargent et al provides a descriptive account of demographic, crash and clinical characteristics of nearly 10 000 Maori injured in or by motor vehicles from 1980 to 1994.[8] Whilst not an analytical study (ie, there is no comparison group), the absence of ethnicity data on police crash reports means that this linked police–health data set provides hitherto unknown information. As the authors state, meaningful comparisons of risk characteristics over time were not possible due to varying data quality over time. This inability to make comparisons over time is most unfortunate, as it prevents a deeper understanding of why and how Maori road-traffic-crash fatality rates remained the same (or even moderately increased) during the 1980s, then decreased during the 1990s.[1]

The explanations for ethnic differences in health are many and multi-layered. A range of research from a variety of perspectives will assist in finding significant intervention

 © NZMA

points in terms of both aetiology and policy. Growing cadres of Maori and Pacific researchers are critical to this journey of understanding. In raising the debate about the role of culture it could be easy to fall back on the colonialist and imperialist project of assimilation. More considered approaches would explore the ways in which culture acts as a resource, and the ways in which cultural capital can be built without the negative aspects of assimilation. Ellison-Loschmann et al offer a way forward here, when they point to findings that asthma self-management programmes have been successful when planned, established and maintained through active Maori involvement.[7] An ownership model may offer a way of building cultural capital, maintaining shared understandings and resisting deficit models of culture.

Author information: Tony Blakely, Senior Research Fellow; Kevin Dew, Senior Lecturer, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, Wellington

Correspondence: Dr Tony Blakely, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, PO Box 7343, Wellington. Fax: (04) 389 5319; email: tblakely@wnmeds.ac.nz

References:

1. Ajwani S, Blakely T, Robson B, et al. Decades of disparity: ethnic mortality trends in New Zealand 1980–1999. Wellington: Ministry of Health; 2003. Available online. URL: http://www.wnmeds.ac.nz/academic/dph/research/nzcms/Ethnic_Mortality_Trends070703.pdf Accessed January 2004.
2. Barnes L, Moss-Morris R, Kaufusi M. Illness beliefs and adherence in diabetes mellitus: a comparison between Tongan and European patients. NZ Med J. 2003;117(1188). URL: http://www.nzma.org.nz/journal/117-1188/743/
3. Paterson J, Tumama Cowley E, Percival T, Williams M. Pregnancy planning by mothers of Pacific infants recently delivered at Middlemore Hospital. NZ Med J. 2003;117(1188). URL: http://www.nzma.org.nz/journal/117-1188/744/
4. MacDonald J. The culture of health and illness. In: Davis P, Dew K, editors. Health and society in Aotearoa New Zealand. Melbourne: Oxford University Press; 1999. p. 111–27.
5. McLennan G, Ryan A, Spoonley P. Exploring society. Auckland: Pearson Education; 2000.
6. Park J, Suaalii-Sauni T, Anae M, et al. Late twentieth-century Auckland perspectives on Samoan masculinities. In: Worth H, Paris A, Allen L, editors. The life of Brian: masculinities, sexualities and health in New Zealand. Dunedin: Univeristy of Otago; 2002. p. 43–55.
7. Ellison-Loschmann L, King R, Pearce N. Regional variations in asthma hospitalisations among Maori and non-Maori. NZ Med J. 2003;117(1188). URL: http://www.nzma.org.nz/journal/117-1188/745/
8. Sargent M, Begg D, Broughton J, Stephenson S, Wright C, Baxter J. Motor vehicle traffic crashes involving Maori. NZ Med J. 2003;117(1188). URL: http://www.nzma.org.nz/journal/117-1188/746/
9. Ajwani S, Blakely T, Robson B, et al. Unlocking the numerator-denominator bias III: Adjustment ratios by ethnicity for 1981–1999 mortality data. NZ Med J. 2003;116(1175). URL: http://www.nzma.org.nz/journal/116-1175/456/

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Illness beliefs and adherence in diabetes mellitus: a comparison between Tongan and European patients

Lucy Barnes, Rona Moss-Morris and Mele Kaufusi

Abstract

Aims The aim of this study was to determine whether there are cultural differences in the way in which Tongan and European people with Type 2 diabetes conceptualise their illness and treatment. The relationships between patients' illness and treatment perceptions and their adherence to self-care regimens were also assessed.

Methods Participants completed either a Tongan or English version of a questionnaire, which included standardised measures of personal beliefs about diabetes and medication, and self-reported adherence. Information about the severity of patients' diabetes was obtained from patients' notes.

Results Comparisons of glycosylated haemoglobin levels showed that Tongan patients had significantly poorer control over their diabetes than did European patients. They were also significantly more likely than European patients to perceive their diabetes as acute and cyclical in nature, uncontrollable, and caused by factors such as God's will, pollution in the environment, and poor medical care in the past. Tongan patients perceived less necessity for medication, and exhibited higher emotional distress related to their diabetes. The beliefs that characterised the Tongan patients tended to be associated with poorer adherence to diet and medication taking.

Conclusions This study highlights the need for assessment of patients' personal and cultural beliefs about their illness. Understanding patients' perceptions may provide an avenue for improving adherence to self-care regimens.

Diabetes mellitus is a major health problem in New Zealand. There are approximately 115 000 people known to have diabetes and an estimated 40 000–60 000 undiagnosed cases.[1] The Ministry of Health predicts that diabetes rates will increase by 78% in the next ten years. Maori and Pacific Island people are particularly affected. Rates of diabetes are three times more common in these groups and are forecast to rise by 130–150% by 2011 compared with 58% in European people.[1] Diabetes-related mortality rates are also 10 times higher in Maori and Pacific Island people than in Europeans, and Pacific Island people have higher admission rates to hospital for diabetes than any other ethnic group.[1]

These dramatic statistics suggest that more needs to be done to understand diabetes-related morbidity, particularly in high-risk groups. Studies have shown that barriers such as cost, lack of knowledge, and limited access to and utilisation of healthcare all contribute to the high incidence of diabetes-related morbidity in Pacific Island people.[2–4] The increased rates of the illness itself are believed to be due in part to a shift from a traditional physical environment to an urbanised and westernised lifestyle.[5] A shift towards a diet high in protein, sugar, salt and animal fat has led to an increase in blood sugar, obesity, and cholesterol levels.[6] Clearly more needs to be done to encourage people to alter their diets. However, Pacific Island people's cultural

© NZMA

beliefs may play an important role here. For instance, traditional Tongan culture accepts that there is a cure for every illness and that healers and spirituality can effect cures.[7] Therefore, Tongan people may focus on spiritual aspects of treatment and on recommendations presented by traditional healers, rather than on medical advice to alter diet, exercise and take medication.

Research based on psychological theory has highlighted the importance of patients' personal beliefs about their illness and treatment in their self-management of a range of chronic illnesses.[8,9] Work by Leventhal and colleagues has shown that patients' beliefs about their illness cluster around five dimensions: (1) Identity: including the disease label and associated beliefs about the symptoms of the disease; (2) Timeline: beliefs about the course and duration of the disease; (3) Consequences: the effects of the disease; (4) Cause: the perceived cause(s) of the disease; (5) Cure/control: including beliefs about recovery from the illness or controllability of an existing condition.[8] Patients' beliefs regarding these dimensions are often different to those held by their healthcare practitioner, and unless specifically asked patients may not reveal the details of their personal beliefs. However, these beliefs have been shown to be important in understanding the ways in which patients choose to cope with their illness. For instance, research on illness beliefs and diabetes has indicated that patients who believe they have control over their illness are more likely to seek treatment and engage in healthcare behaviours.[10,11]

In addition to holding their own personal model of their illness, patients have their own ideas about the necessity of taking their prescribed medication and concerns about the possible long-term effects of medication regimens. Research on a variety of illness groups has suggested that these beliefs are strong predictors of medication adherence.[12,13]

Adherence to prescribed treatment is essential for the avoidance of complications and for quality of life in diabetes.[14,15] The management of diabetes lies predominantly in the hands of the patient. A lack of knowledge and barriers to care have been identified as key factors contributing to poor management of the disease among Pacific Island people.[2,4,16] However, little research has been conducted looking specifically at the different dimensions of these patients' illness and medication beliefs. The aim of the present study was to identify cultural differences in illness perceptions and medication beliefs among European and Tongan people with diabetes. The relationships between these beliefs and adherence to diabetes self-care regimens were also examined.

Methods

Sample Tongan and European patients with diabetes were recruited from the Auckland Diabetes Centre and affiliated satellite clinics over a six-month period. Patients were invited to participate in the study while waiting for clinic appointments. The study and its rationale were described to the subjects by the researcher or if necessary a translator or by the Tongan nurse. One hundred and ninety people were asked to participate in the study. These included 72 Tongan patients and 118 European patients. One hundred and thirteen people consented to participate and returned the questionnaire – a response rate of 59%. Patients' clinic records were then accessed to confirm that subjects fitted the inclusion criteria: patients who had Type 2 diabetes, who were of either Tongan or New Zealand European decent and who were at least 18 years of age. Seventeen of the European patients had Type 1 diabetes and 14 failed to return the consent form giving their personal details, so their information could not be accessed from the files. These patients were excluded from the analysis. The final sample included 43 Tongan patients and 39 European patients.

© NZMA

Measures The demographic section of the questionnaire included questions relating to age, ethnic group, unemployment due to health, financial compensation due to diabetes, duration of diabetes, hospital admissions, and feet condition. Diabetes severity was measured by gathering the most recent total glycosylated haemoglobin (HbA1c) information from clinic records. HbA1c levels were grouped according to the Gill criteria as non-diabetic range (4–5.9%), excellent control (6–6.9%), good control (7–7.9%), indifferent control (8–8.9), poor control (9–9.9%), and exceptionally poor control (>10%).[17] For the purpose of this study the participants who fell within the non-diabetic range were still classified as having diabetes as they had been diagnosed as having Type 2 diabetes and were being treated for the condition.

Illness perceptions were assessed using the Revised Illness Perceptions Questionnaire (IPQ-R),[18] which is a psychometrically sound, standard measure of patients' perceptions of their condition. In accordance with Leventhal's model,[8] the IPQ-R measures beliefs surrounding illness identity, timeline, consequences, personal and treatment control, and cause. It also measures the degree to which patients feel they have a coherent understanding of their condition (illness coherence) and their distress in relation to their illness (emotional representations). Two causal items relevant to this population group 'God's will' and 'punishment' were added in accordance with the Tongan researcher's recommendations.

The Beliefs about Medications Questionnaire (BMQ)[12] was used to measure beliefs about diabetes medication. It consists of two subscales labelled 'need' and 'concern'. The former measures patients' beliefs about the necessity for taking medication and the latter patients' concerns about the negative effects of taking medication.

Adherence to diet was measured using the dietary subscale of the Summary of Diabetes Self-Care Questionnaire (SDSCA),[19] which is a brief self-report scale of eating habits over the past week. The Medication Adherence Representation Questionnaire (MARS)[20] was used to measure adherence to diabetes medication. This scale consists of statements of adherent and non-adherent behaviour. Two items relevant to the Tongan group were added to this questionnaire, including questions pertaining to taking traditional medicines and behaviour relating to religious beliefs. The internal reliability of this modified scale measured by Cronbach Alpha was 0.79.

A Tongan translation of the questionnaire was available for those who required it, which all Tongan participants utilised. This version was translated through the Middlemore translation service.

Statistical methods Data were analysed using SPSS version 11.0. Chi-square and t tests were used to assess whether differences existed in the demographic and illness-severity data between the two groups. Differences in illness and medication perceptions and self-reported adherence across the groups were assessed utilising independent samples t tests and Mann-Whitney U tests. The data from the IPQ-R identity subscale had to be dropped from the study as most of the Tongan patients failed to fill in this part of the questionnaire. It appears that the translated version of the identity subscale was confusing for these patients.

Pearson and Spearman correlations were used to establish the relationship between illness perceptions and self-reported adherence in the patient groups. All the data met the basic assumptions necessary for parametric statistics except for the MARS scale measuring medication adherence. Consequently, non-parametric tests were used in the analysis of the MARS data.

Results

Demographic and diabetes-severity data Table 1 presents the demographic and diabetes-severity data of the two patient groups. Tongan patients had significantly higher unemployment due to their health, higher rates of hospital admissions, and higher HbA1c levels. There were no significant differences between the groups with regards to duration of diabetes, feet condition (sores or black spots on the feet), and smoking.

© NZMA

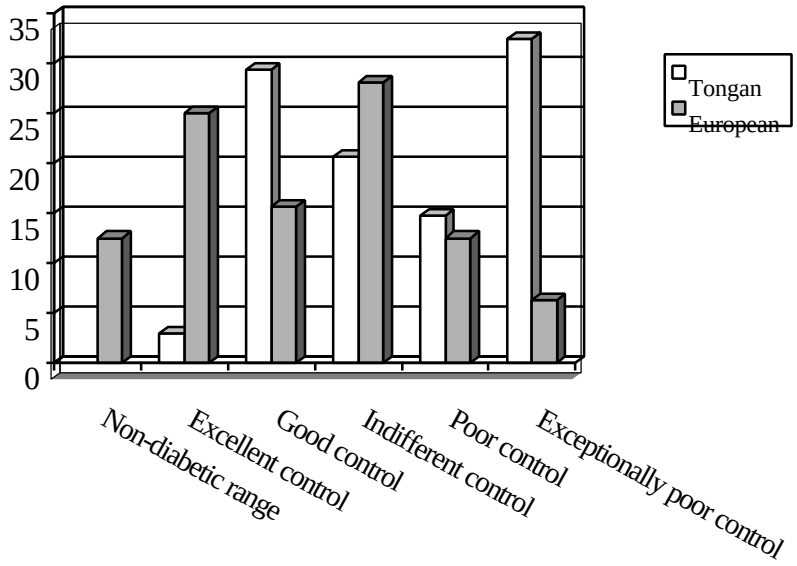
Table 1. Demographic and illness-severity features across groups

	Tongan	European	
Age, mean years (SD)	59.2 (11.2)	59.6 (12.7)	t = -0.16 p = 0.87
Duration of diabetes, mean years (SD)	8.0 (7.4)	6.3 (5.7)	t = 1.11 p = 0.27
Sores and ulcers on feet (yes)	14.0%	5.1%	χ^2 =1.81 p = 0.18
Admitted into hospital due to diabetes (yes)	59.5%	17.9%	χ^2 =15.36 p = 0.004*
Cigarette smoker (yes)	46.5%	64.1%	χ^2 = 2.56 p = 0.11
HBA1c, mean (SD)	9.2 (2.0)	7.8 (2.0)	t = 2.77 p = 0.01*
Unemployment due to health (yes)	66.7%	23.1%	χ^2 = 15.48 p =0.00†

*p <0.01; †p <0.001.

Figure 1 shows the HbA1c data across the patient groups as a function of control category. Of the European patients, 37.5% fell into the categories of non-diabetic range or excellent control compared with only 2.9% of the Tongan patients. Of the Tongan patients, 32.4% had exceptionally poor diabetes control compared with 6.3% of the European patients.

Figure 1. Percentage means for HbA1c control categories across ethnic groups (n = 66)



Group differences in illness perceptions, medication beliefs and adherence Table 2 shows the results of the t tests and Mann-Whitney U tests for the group differences

© NZMA

in perceptions of illness, medication beliefs, and adherence. Significant differences were found within several of the illness perception IPQ-R dimensions. With regards to timeline, Tongan patients had a more acute perception of their illness and perceived their disease to be more cyclical in nature than did European patients. Tongan patients were significantly more emotionally distressed by their illness and were less likely to believe that their treatment could control their diabetes. Three of the 19 causal items of the IPQ-R were different between the groups at the level of $p < 0.01$ and are included in Table 2. Tongan patients were more likely to believe in external causes for their diabetes including 'poor medical care in the past', 'environmental pollution', and 'God's will'. The two groups did not differ in their perceptions of the severity of the consequences of diabetes, beliefs about personal control, and perceptions of how much they understand their illness.

Table 2. Comparisons between the groups of illness perceptions, medication beliefs and adherence

	Tongan Mean (SD)	European Mean (SD)	t value
IPQ-R			
Timeline (acute/chronic)	18.8 (3.0)	21.0 (4.6)	-2.44*
Timeline (cyclical)	13.8 (3.2)	11.2 (3.4)	3.48†
Consequences	18.1 (5.5)	17.7 (4.5)	0.39
Personal control	21.9 (4.5)	22.4 (3.8)	-0.56
Treatment control	17.2 (3.7)	15.7 (2.9)	2.08*
Illness coherence	17.0 (4.2)	15.9 (4.6)	1.13
Emotional representations	20.4 (5.0)	15.7 (5.0)	4.17†
Causes (poor medical care in the past)	3.3 (1.3)	1.9 (0.8)	5.44†
Causes (environmental pollution)	2.7 (1.4)	1.9 (0.9)	2.80‡
Causes (God's will)	2.9 (1.5)	2.0 (1.2)	2.89‡
BMQ			
Need	8.4 (3.0)	14.3 (6.2)	-5.17†
Concern	16.8 (3.7)	19.0 (4.3)	-2.44*
Medication adherence§	26.9	42.3	-3.25† (Z)
Diet adherence	-1.6 (3.7)	0.8 (3.3)	-3.08‡

IPQ-R = Revised Illness Perceptions Questionnaire;[18] BMQ = Beliefs about Medications Questionnaire[12]

*$p < 0.05$; †$p < 0.001$; ‡$p < 0.01$

§data analysed using Mann-Whitney U test

With regards to beliefs about medication, Table 2 shows that Tongan patients exhibited less concern about the effects of medication (BMQ, Concern) than European patients and were less likely to see the need for medication (BMQ, Need) than the European patients. Tongan patients also reported significantly lower levels of adherence to diet recommendations and their medication regimen than did European patients.

The correlations between illness and medication beliefs and adherence are presented in Table 3. The beliefs that diabetes is a cyclical illness and that the illness was caused by poor medical care in the past were correlated with poorer adherence to diet recommendations. Adherence to medication was correlated with high scores on the BMQ Need subscale. Non-adherence to medication was associated with the beliefs

© NZMA

that environmental pollution, God's will and poor medical care in the past were causes of diabetes. A perception of a cyclical timeline and that the consequences of diabetes were serious were also correlated with lower medication adherence.

Table 3. Correlations between illness and medication perceptions and adherence

	Adherence to diet	Adherence to medication (r_s)
IPQ-R		
Timeline (acute/chronic)	-0.05	0.03
Timeline (cyclical)	-0.23*	-0.27*
Consequences	-0.11	-0.28*
Personal control	-0.11	0.06
Treatment control	-0.14	-0.03
Illness coherence	-0.06	0.01
Emotional representation	-0.14	-0.13
Causes (poor medical care in the past)	-0.31†	-0.29*
Causes (environmental pollution)	-0.20	-0.33†
Causes (God's will)	-0.17	-0.40‡
BMQ		
Need	0.05	0.36†
Concern	-0.07	0.14

*p <0.05, †p <0.01, ‡p <0.001

Discussion

The findings from this study suggest that perceptions of diabetes differ between the ethnic groups included in it, and that the perceptions that characterise Tongan patients tend to be associated with lower adherence to dietary and medication recommendations. Tongan patients believed their diabetes to be a more cyclical, acute illness than did European patients, who tended to view their illness as chronic. Tongan patients were also more likely to attribute their illness to external factors including the beliefs that poor medical care in the past, environmental pollution and God's will caused their diabetes. They were more emotionally distressed by their diabetes and had less confidence in the ability of their treatment to control their illness. Finally, Tongan patients saw less necessity for diabetes medication than did European patients.

Of particular significance was the finding that one third of the Tongan group had exceptionally poor control over their diabetes compared with only 6% of European patients. Tongan patients were also more likely to be hospitalised for their diabetes, to be less adherent to dietary and medication regimens, and to be unemployed because of their condition. These data highlight the importance of effective clinical interventions for these patients. Exploring patients' illness beliefs may be one way of addressing this problem. In this study, patients who saw the necessity for medications were more likely to adhere to their medication regimen. On the other hand, those who believed that their diabetes was cyclical, and caused by external factors such as pollution, God's will, and poor medical care in the past were less likely to adhere to medication and diet recommendations.

© NZMA

The data from this study are in accordance with previous work emphasising the importance of addressing diabetic patients' illness perceptions. In previous studies accurate beliefs about the effectiveness of treatment, the necessity for medication, and disease course have been found to be predictive of adherence.[10,12] Accurate knowledge of causal beliefs has also been shown to be predictive of both better adjustment and adherence in diabetes.[10,21,22] The finding that Tongan patients had a stronger belief in external causes is also consistent with previous research, which has shown that that Pacific Island people are strongly influenced by 'powerful others' including spirituality and family.[23] These beliefs may preclude patients from feeling a sense of personal control over their illness.

It is interesting to note that although the Tongan patients held less accurate beliefs about the causes and time course of diabetes, there was no difference between Tongan and European patients' perceptions of the degree to which they understood their condition. Perceptions of understanding were generally unrelated to adherence behaviour. This emphasises the importance of uncovering specific beliefs rather than just asking patients whether they feel they have a good knowledge or understanding of their diabetes.

Certain limitations of this study should be noted. Although the self-reported measures of adherence in this study are validated questionnaires they may not always provide accurate data.[24] There may be a retrospective bias as patients are asked to report their adherence over the last seven days. Social desirability may also influence patients' responses as they may wish to appear more adherent to the researcher. The questionnaire did, however, clearly state that the information that the patient provided would be confidential and would not be seen by staff at the clinic. Comparing data from the questionnaire translated into Tongan with the original English version may have revealed some discrepancies in the way in which questions were phrased. As mentioned earlier, the identity subscale of the IPQ-R did not appear to translate well and Tongan patients largely left this section unanswered.

Despite these limitations this is one of the first studies to provide a quantitative comparison of illness beliefs in Tongan and European patients. The data confirm that Tongan patients have less accurate medical perceptions of their condition. Some of these beliefs may be culturally determined, such as the beliefs that illness is largely acute and curable, and that spiritual factors may play a role in causing the illness itself. Asking patients about their beliefs may provide medical practitioners with an avenue to address poor adherence to self-care. Explanations can be offered that build on rather than contradict existing beliefs. For instance, it can be acknowledged that 'God's will' may be a factor in the illness, but that other aspects, such as taking appropriate medication or changing one's diet, are also important and can be addressed. Studies have shown that interventions that target patients' illness beliefs are effective in improving self-management behaviours in diabetes.[25]

This study has focused on illness and medication beliefs and how these may contribute to adherence behaviours. However, there are other factors that are likely to be important. People's expectations about their ability to engage in exercise and dietary change have also been shown to play an important role in adherence and these expectations may differ across cultural groups.[26] Future research should focus on expectations of personal competence in changing behaviours, and on social aspects that may affect adherence, such as ease of access to clinics.

© NZMA

Author information: Lucy Barnes, Health Psychology Intern; Rona Moss-Morris, Senior Lecturer in Health Psychology, Health Psychology, The University of Auckland; Mele Kaufusi, Diabetes Nurse Specialist, The Auckland Diabetes Centre, Auckland Hospital, Auckland

Acknowledgements: We thank the Auckland Diabetes Centre for their support of the study and the participants for taking the time to complete the questionnaire. This study was supported in part by a grant from Diabetes New Zealand.

Correspondence: Rona Moss-Morris, Health Psychology, Faculty of Medical and Health Sciences, The University of Auckland, Private Bag 92019, Auckland. Fax: (09) 373 7013; email: r.moss-morris@auckland.ac.nz

References:

1. Ministry of Health. Modelling diabetes: a summary. The Public Health Group. Wellington: New Zealand; 2002.
2. Simmons D, Weblemoe T, Voyle J, et al. Personal barriers to diabetes care: lessons from a multi-ethnic community in New Zealand. Diabet Med. 1998;15:958–64.
3. Simmons D, Voyle J. Psychological and behavioural aspects of NDDM among Pacific Islanders in South Auckland. Pacific Health Dialog. 1995;3:100–6.
4. Simmons D, Shaw L, Kenealy T, et al. Ethnic differences in diabetes knowledge and education: the South Auckland Diabetes Survey. NZ Med J. 1994;107:197–200.
5. Zimmet P, Dowse G, Finch C, et al. The epidemiology and natural history of NIDDM – lessons from the South Pacific. Diabetes Metab Rev. 1990;6:91–124.
6. Bathgate M, Alexander D, Mitikulena A, et al. The health of Pacific Islands people in New Zealand. Wellington: Public Health Commission; 1994.
7. Moata'ane L, Muimui-Heata S, Guthrie B. Tongan perceptions of diet and diabetes mellitus. J NZ Diet Assoc. 1996;50:52–6.
8. Leventhal H, Meyer D, Nerenz D. The common sense representation of illness danger. In: Rachman S, editor. Contributions to medical psychology. Oxford: Pergamon Press; 1980. p. 7–30.
9. Petrie KJ, Weinman J, Sharpe N, Buckley J. Role of patients' view of their illness in predicting return to work and functioning after myocardial infarction: longitudinal study. BMJ. 1996;312:1191–4.
10. Hampson SE, Glasgow RE, Foster, LS. Personal models of diabetes among older adults: relationship to self-management and other variables. Diabetes Educ. 1995;21:300–7.
11. Kavanagh DJ, Gooley S, Wilson PH. Prediction of adherence and control in diabetes. J Behav Med. 1993;16:509–22.
12. Horne R, Weinman J, Hankins M. The beliefs about medications questionnaire: the development of a new method for assessing the cognitive representation of medication. Psychol Health. 1999;14:1–24.
13. Horne R. Treatment perceptions and self-regulation. In: Cameron LD, Leventhal H, editors. The self-regulation of health and illness behaviour. London: Routledge; 2003. p. 138–154.
14. Cox DJ, Gonder-Frederick L. Major developments in behavioral diabetes research. J Consult Clin Psychol. 1992;60:628–38.
15. Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 1993;329:977–86.

 © NZMA

16. Mitikulena A, Smith RB. Views of Pacific Islands people with noninsulin dependent diabetes: A Wellington survey. NZ Med J. 1996;109:467–9.

17. Gill M, editor. Diagnostic handbook: a handbook for the interpretation of laboratory tests. Auckland: Adis International; 2001.

18. Moss-Morris R, Weinman J, Petrie KJ, et al. The Revised Illness Perception Questionnaire (IPQ-R). Psychol Health. 2002;17:1–6.

19. Toobert DJ, Glasgow RE. Assessing diabetes self-management: the summary of diabetes self-care activities questionnaire. In: Bradley C, editor. Handbook of pscyhology and diabetes: a guide to psychological measurement in diabetes research and practice. Switzerland: Harwood Academic Publishers; 1994. p. 351–73.

20. Horne R, Weinman, J. Patients' beliefs about prescribed medicines and their role in adherence to treatment in chronic physical illness. J Psychosom Res. 1999;47:555–67.

21. Bradley C, Lewis KS, Jennings AM, Ward JD. Scales to measure perceived control developed specifically for people with tablet-treated diabetes. Diabet Med. 1990;7:685–94.

22. Hampson SE, Glasgow RE, Toobert DJ. Personal models of diabetes and their relations to self-care activities. Health Psychol. 1990;9:632–46.

23. Simmons D. Diabetes and its complications in New Zealand: an epidemiological perspective. NZ Med J. 1996;109:245–7.

24. Johnson SB. Methodological issues in diabetes research. Measuring adherence. Diabetes Care. 1992;15:1658–67.

25. Petrie KJ, Broadbent E, Meechan G. Self-regulatory interventions for improving the management of chronic illness. In Cameron LD, Leventhal H, editors. The self-regulation of health and illness behaviour. London: Routledge; 2003. p. 257–77.

26. Williams KE, Bond MJ. The roles of self-efficacy, outcome and expectancies and social support in the self-care behaviours of diabetics. Psychol Health Med. 2002;7:127–41.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Pregnancy planning by mothers of Pacific infants recently delivered at Middlemore Hospital

Janis Paterson, Esther Tumama Cowley, Teuila Percival and Maynard Williams

Abstract

Aim To describe pregnancy planning by mothers of Pacific infants recently delivered at Middlemore Hospital.

Methods The data were gathered as part of the Pacific Islands Families: First Two Years of Life (PIF) Study in which 1365 birth mothers were interviewed six weeks after the birth about the planning of their pregnancy. Mothers were asked if the pregnancy was planned, if the pregnancy was unplanned, the form of contraception used, or, if not used, their main reasons for not using contraception.

Results Forty per cent of the mothers reported that they had planned their pregnancy. Of the 60% of mothers who had not planned their pregnancies, 70.8% were not using contraception when they conceived. The main reasons given by mothers for not using contraception were that they never thought about contraception (46.8%), did not like using contraception (42.5%), decided to take a chance (39.4%), did not want to risk the associated weight gain (30.4%), and did not think they could have a baby (17.3%). Factors significantly associated ($p < 0.05$) with non-use of contraception by birth mothers who did not plan their pregnancy were lack of post-school qualifications and strong alignment with Pacific culture.

Conclusions The findings showed that many women in this cohort did not avail themselves of the various contraceptive services available to them. An investigation into the accessibility and acceptability of family planning services needs to be undertaken to ensure that services are delivered in a way that maximises choices regarding the use of contraceptives.

The rapidly growing population of Pacific peoples living in New Zealand is partly due to immigration levels but more significantly to the high fertility rate of Pacific women.[1] High Pacific birth rates have also been described as due to low utilisation of contraception and a cultural preference for large families.[2,3] There is evidence that Pacific women in New Zealand have the highest rate of unplanned pregnancies and abortions across all age groups, with a high number of hospitalisations of Pacific women due to pregnancy, childbirth and health services relating to reproduction.[2]

Ethnic differences in the planning of pregnancy may be due to women in many communities having aspirations about reproduction and contraception different from their partners.[4,5] A study carried out in Tonga found that contraception was described as a female responsibility, with limited knowledge and usage among males, and little discussion between partners.[5] There are few studies of contraceptive practices and values among Pacific families in New Zealand; however, it is thought that the male partner has the main role in determining family size.[6] Within Pacific communities, cultural mores are likely to be the biggest obstacle to effective family planning.[7] Asiasiga concluded that Pacific people regard sexuality and contraception as subjects

© NZMA

not spoken of in the home.[7] There is no word for sex in some Pacific languages,[8] a very strong message of no sex before marriage,[6,8] and women are not encouraged to use contraception.[9]

Some Pacific women, particularly older women, may use abortion as a form of contraception,[10,11] which is partly due to the traditional expectations of their spouses.[6] In a recent study tubal ligation was found to be the preferred contraceptive option for the wives of the older Pacific men, and older Pacific men were found to be less likely to have vasectomies than their Palagi (European) counterparts, some claiming 'traditional cultural reasons'. However, younger, New Zealand-born Pacific males described contraception as part of a strategy on which both partners can work together for their future. It has been suggested that this shift in attitude may be due to the younger males being better informed, better educated and more acculturated into New Zealand society.[6]

The purpose of this paper is to describe pregnancy planning by mothers of Pacific infants and to examine the maternal and socio-demographic factors associated with non-use of contraception (unplanned pregnancy).

Methods

Data were collected as part of the Pacific Islands Families: First Two Years of Life (PIF) Study. The PIF Study is a longitudinal investigation of a cohort of 1398 infants born at Middlemore Hospital, South Auckland, during the year 2000. Middlemore Hospital was chosen as the site for recruitment of the cohort as it has the largest number of Pacific births in New Zealand and is representative of the major Pacific ethnicities. All potential child participants were selected from live births at Middlemore Hospital where the child had at least one parent who identified as being of a Pacific Island ethnicity and also a New Zealand permanent resident. Recruitment procedures occurred through the Birthing Unit in conjunction with the Pacific Islands Cultural Resource Unit, which provided a daily list of Pacific admissions.

Approximately six weeks after the birth of their child, mothers were visited in their homes by Pacific interviewers fluent in both English and a Pacific language. Once eligibility criteria were established and informed consent was gained, mothers participated in one-hour interviews concerning the health and development of the child and family functioning. Each interview was carried out in the preferred language of the mother. All procedures and interview protocols had ethical approval from the National Ethics Committee. Detailed information about the cohort and procedures is described elsewhere.[12] Birth mothers were interviewed about the planning of their pregnancy. Mothers were asked if their pregnancy was planned, if the pregnancy was unplanned, the form of contraception used, or, if not used, their main reasons for not using contraception. Maternal and socio-demographic factors that may be associated with not planning a pregnancy and not using contraception were assessed by univariate and multivariate procedures.

Results

Ninety six per cent (n =1590) of potentially eligible mothers of Pacific infants who had been born between 15 March and 17 December 2000 gave consent to be visited in their homes when the infant was six weeks old. Of the 1477 mothers contacted and who met the eligibility criteria, 1376 (93.2%) agreed to participate in the study. A more conservative recruitment rate of 87.1% would include mothers who consented to contact and were (a) confirmed eligible, or (b) of indeterminable eligibility due to inability to trace.

© NZMA

Table 1. Numbers (row percentages) and univariate odds ratios for non-use of contraception by mothers following unplanned pregnancies by selected variables

Variable	Non-use of contraception		Univariate odds ratio (95% CI)	
Age (years)				
<20	65	(74.7)	1.00	
20–29	320	(72.9)	0.91	(0.54–1.54)
30–39	178	(65.7)	0.65	(0.38–1.12)
40+	18	(75.0)	1.02	(0.36–2.88)
Ethnicity				
Samoan	270	(72.8)	1.00	
Cook Island Maori	106	(67.9)	0.79	(0.53–1.19)
Niuean	31	(67.4)	0.77	(0.40–1.49)
Tongan	106	(72.1)	0.97	(0.63–1.48)
Other Pacific*	24	(66.7)	0.75	(0.36–1.55)
Non-Pacific	44	(67.7)	0.78	(0.44–1.38)
Social marital status				
Partnered	424	(69.5)	1.00	
Non-partnered	157	(74.4)	1.27	(0.89–1.82)
Education				
Post-school qualification	142	(62.0)	1.00	
Secondary school qualification	191	(71.5)	1.54	(1.06–2.24)†
No formal qualifications	248	(76.3)	1.97	(1.36–2.86)‡
English fluency				
Yes	370	(67.9)	1.00	
No	211	(76.4)	1.54	(1.10–2.14)§
Parity				
1	167	(75.6)	1.00	
2–4	317	(68.6)	0.71	(0.49–1.02)
5+	91	(70.5)	0.77	(0.48–1.26)
Born in NZ				
Yes	207	(66.6)	1.00	
No	374	(73.3)	1.38	(1.02–1.88)§
Religion				
Yes	529	(71.2)	1.00	
No	52	(66.7)	0.81	(0.49–1.33)
Cultural alignment				
High NZ, low Pacific Island	191	(64.7)	1.00	
Low NZ, high Pacific Island	183	(77.2)	1.85	(1.25–2.72)†
High NZ, high Pacific Island	105	(77.2)	1.84	(1.16–2.94)§
Low NZ, low Pacific Island	100	(67.6)	1.13	(0.75–1.73)
Years lived in NZ				
0–5	105	(77.8)	1.00	
6–10	56	(71.8)	0.73	(0.38–1.38)
>10	420	(69.1)	0.64	(0.41–0.99)
Household income				
<$20 000	229	(73.9)	1.00	
$20 001–$40 000	267	(69.0)	0.79	(0.56–1.10)
>$40 000	64	(67.4)	0.73	(0.44–1.20)
Unknown	21	(72.4)	0.93	(0.40–2.18)

*includes mothers identifying equally with two or more Pacific Island groups, equally with Pacific Island and non-Pacific Island groups, or with Pacific Island groups other than Tongan, Samoan, Cook Island Maori or Niuean

†$p < 0.01$; ‡$p < 0.001$; §$p < 0.05$

© NZMA

Of the 1376 mothers in the cohort (1.7% gave birth to twins), nine adoptive mothers and two foster mothers were eliminated. Of the 1365 remaining birth mothers, 47.2% self-identified their major ethnic group as Samoan, 21% as Tongan, 16.9% as Cook Islands Maori, 4.3% as Niuean, 3.4% as Other Pacific, and 7.2% as Non-Pacific. The Other Pacific group includes mothers identifying equally with the Pacific and Non-Pacific groups, or with Pacific groups other than Samoan, Tongan, Cook Island Maori or Niuean. The Non-Pacific group refers to mothers of infants fathered by Pacific men. The mean (SD) age of mothers was 27 (6.2) years; 80.5% were married or in de facto partnerships; 33.0% of mothers were born in New Zealand; and 27.4% had post-school qualifications.

Forty per cent of the mothers reported that they had planned their pregnancies. Of the 60% of mothers who had not planned their pregnancy, 70.8% were not using contraception when they conceived. The main reasons given by mothers for not using contraception were that they never thought about contraception (46.8%), did not like using contraception (42.5%), decided to take a chance (39.4%), did not want to risk the associated weight gain (30.4%), and did not think they could have a baby (17.3%). For mothers who were using contraception when they conceived (29.2%), the main methods of contraception used were the pill (15.0%), condoms (6.7%), and the contraceptive injection (6.1%). There were no maternal reports of vasectomy in male partners and only three mothers reported having a tubal ligation.

Table 1 lists the variables examined for potential association with non-use of contraception for birth mothers who had unplanned pregnancies. For the categories within each variable the numbers and percentages of mothers who did not use contraception are given, along with the associated odds ratios. Mothers with no post-school qualifications, who were not born in New Zealand, who were strongly aligned with Pacific way of life and customs, and who were not fluent in English were significantly ($p < 0.05$) more likely not to have used contraception. The variables of maternal ethnic group, age, parity, religion, social marital status and years lived in New Zealand did not reach significance in association with the non-use of contraception.

When controlling for the effects of all the variables in Table 1 in a multiple regression model, factors that remained significantly associated ($p < 0.05$) with non-use of contraception by birth mothers who did not plan their pregnancy were lack of post-school qualifications and reporting a strong alignment with Pacific way of life and customs.

Discussion

A woman's ability to space or limit the number of her births has a direct impact on her health and wellbeing. Effective family planning not only affects the social and economic circumstances of women and their families, but can also improve the chance of healthy pregnancy outcomes.[13] The high rate of unplanned pregnancies (60%), the majority of which were due to not using contraception (70.8%), reported by this cohort of Pacific mothers in New Zealand is in line with other reports.[2,3] In New Zealand, overall contraceptive use by married women of child-bearing age is reported to be 75%, compared with 34% in Samoa and 63% in the Cook Islands.[14] Although the present study is not a Pacific contraceptive-use prevalence study it does highlight that a large number of women in this cohort did not plan their pregnancy

© NZMA

and did not avail themselves of the various contraceptive services available to them. The poor access to, or use of, family planning indicated by these findings adds to the general picture of poor access to effective healthcare for Pacific peoples.[15–17]

The overcoming of cultural barriers towards the use of contraception was described a decade ago as a major hurdle facing Pacific-health workers.[9] The taboo around talking about sexuality and contraception within Pacific families has been well documented by Pacific writers.[6–8] Such barriers appear to persist, with significant numbers of mothers reporting that they did not think about contraception, did not like using contraception, and did not think they could have a baby. The reasons for not using contraception appear to be linked to lack of information or awareness about family planning services or methods.

Although the findings of the PIF Study demonstrate that information about effective family planning has not reached a number of Pacific women, it is suggested that with the increasing number of Pacific people born and educated in New Zealand contraception use and planned pregnancies may increase in the future. The factors significantly associated with non-use of contraception in unplanned pregnancies were not having a postgraduate education and reporting a strong alignment with Pacific way of life and customs. Other variables, although significant only at univariate level, were non-fluency in English and being born in a Pacific Island. Taken together, these findings reflect a similar profile to that suggested by Anae et al,[6] and show that mothers who have more resources to support their lives in New Zealand (eg, higher education) are more likely to use contraception. The findings showed that the women in this cohort did not avail themselves of the various contraceptive services available to them. It is likely that the importance of family planning is low in Pacific communities. An investigation into the dynamics of contraceptive use and the accessibility and acceptability of family planning services needs to be undertaken to ensure that services are delivered in a way that maximises fertility choices.

Author information: Janis Paterson, Associate Professor, Co-Director; Esther Tumama Cowley, Pacific Development Manager, Pacific Islands Families: First Two Years of Life Study, Auckland University of Technology; Teuila Percival, Co-Director, Pacific Islands Families: First Two Years of Life Study, Auckland University of Technology and Kidz First Children's Hospital and Community Services, South Auckland Health; Maynard Williams, Senior Research Fellow and Statistician, Faculty of Health Studies, Auckland University of Technology, Auckland

Acknowledgments: The PIF Study is supported by grants awarded from the Foundation for Research, Science, and Technology, the Health Research Council of New Zealand, and the Maurice and Phyllis Paykel Trust. We thank the families who have participated in the study, the Pacific Peoples Advisory Board, and other members of the research team.

Correspondence: Dr Janis Paterson, Faculty of Health Studies, Auckland University of Technology, Private Bag 92006, Auckland. Fax: (09) 917 9877; email: janis.paterson@aut.ac.nz

References:

1. Ministry of Health. Making a Pacific difference in health policy. Wellington: Ministry of Health; 1998.

 © NZMA

2. Ministry of Health. Making a Pacific difference. Strategic initiatives for the health of Pacific people in New Zealand. Wellington: Ministry of Health; 1997.
3. Bathgate M, Alexander D, Mitikulena A, et al. The health of Pacific people in New Zealand. Wellington: Public Health Commission; 1994.
4. Burbank V, Chisholm J. Gender differences in the perception of ideal family size in an Australian aboriginal community. In: Hewlett B, editor. Father-child relations: cultural and biosocial contexts. New York: Aldine de Gruyter; 1995.
5. Ivarature H. Family planning and contraception in Tongoleka Village, Kingdom of Tonga. Pacific Health Dialog. 1997;4:43–50.
6. Anae M, Fuamata N, Lima I, et al. The roles and responsibilities of some Samoan men in reproduction. Auckland: Pacific Health Research Centre, University of Auckland; 2000.
7. Asiasiga L. Abortion and Pacific Islands women. A pilot study for the New Zealand Family Planning Association. Wellington: New Zealand Planning Association (NZFPA); 1994.
8. Fuemana D. A brown girl's perception of sex and sexuality. GP Weekly. 1996:24–5.
9. Glasgow J. Pacific nurses face hurdle. J Gen Prac. 1990;12.
10. Young LK, Farquhar CM, McCowan LM, et al. The contraceptive practices of women seeking termination of pregnancy in an Auckland clinic. NZ Med J. 1994;107:189–92.
11. Donald H. Abortion and Pacific women in New Zealand. Pacific Health Dialog. 1997;4:80–3.
12. Paterson J, Tukuitonga C, Abbott M, et al. The Pacific Islands Families Study. Technical Report Number 1. Auckland: Auckland University of Technology; 2002.
13. United Nations Population Division. World contraceptive use, 2001. Available online. URL: http://www.un.org/esa/population/publications/contraceptive2001/contraception01.htm Accessed October 2002.
14. United Nations Department of Economic and Social Affairs. Demographic, social and housing statistics indicators on child bearing. Available online. URL: http://unstats.un.org/unsd/demographic/social/childbr.htm Accessed January 2004.
15. Ministry of Health. Taking the pulse: 1996/97 New Zealand Health Survey. Wellington: Ministry of Health; 1999.
16. Mitchell EA. Racial inequalities in childhood asthma. Soc Sci Med. 1991;32:831–6.
17. Young N. Pacificans access to primary health care in New Zealand: a review. Pacific Health Dialog. 1997;4:189–91.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Regional variations in asthma hospitalisations among Maori and non-Maori

Lis Ellison-Loschmann, Ron King and Neil Pearce

Abstract

Aim To examine regional patterns of asthma hospitalisations in Maori and non-Maori.

Methods We studied asthma hospitalisations in Maori and non-Maori during 1994–2000. Hospitalisation rates for Maori and non-Maori were calculated for ages 5–34 years in each of the 74 territorial authorities (TAs), of which 15 are urban and 59 predominantly rural. The data were also analysed separately for Maori and non-Maori in the age groups 0–4, 5–14, 15–34 and 35–74 years.

Results For Maori, the highest hospitalisation rates were in Tauranga, Invercargill, Wanganui, South Wairarapa and Gisborne; the lowest rates were in Rodney, Tasman, Franklin, Waitaki and North Shore City. The rate of asthma hospitalisation was higher in Maori than non-Maori in each age-group: 0–4 years relative risk (RR) = 1.43; 5–14 years RR = 1.08; 15–34 years RR = 1.31; 35–74 years RR = 2.97. The differences were higher in rural areas (RR 1.65, 1.17, 1.34 and 3.13 respectively) than in urban areas (RR 1.25, 1.00, 1.22, 2.79 respectively).

Conclusions These analyses confirm previous evidence that asthma hospitalisation rates are higher in Maori than in non-Maori, despite the fact that asthma prevalence is similar in Maori and non-Maori children. They also indicate that this excess of hospitalisations is higher in rural than in urban areas, although the difference is not large.

Although the prevalence of asthma is similar in Maori and non-Maori children, Maori children and adults experience excess morbidity and higher hospitalisation rates than non-Maori.[1,2] The Maori Asthma Review concluded that asthma was more severe and that hospitalisation and mortality rates for Maori exceeded those of non-Maori primarily due to inadequate access to appropriate healthcare and asthma education.[3,4] It was suggested that these problems of access might be particularly acute in rural areas. More generally, there is widespread interest in regional differences and a common belief that asthma prevalence may be higher in rural areas.[5]

In fact, few studies have examined regional differences in asthma prevalence or severity in New Zealand, and those that have been conducted have found little evidence of regional differences. The New Zealand arm of the International Study of Asthma and Allergies in Childhood (ISAAC) was conducted in six New Zealand centres in 1992–1993.[6] Three of these were urban (Auckland, Wellington, Christchurch) and three were 'provincial' (Bay of Plenty, Hawke's Bay, Nelson). In general, there were no regional differences in prevalence rates of asthma symptoms with the possible exception of Nelson, which had slightly lower prevalence rates in the 6–7 years age group.

© NZMA

Similar analyses for adults were conducted as part of the New Zealand component of the European Community Respiratory Health Survey (ECRHS).[7,8] The 12-month-period prevalence of asthma (defined as woken by shortness of breath, or an attack of asthma in the past year, or current asthma medication) was 15.2% overall, but was 22.1% in Maori, 20.6% in Pacific people and 14.3% in 'others' (this study in adults therefore found a difference in asthma symptom prevalence between Maori and non-Maori in contrast with previous studies in children, which have generally found asthma prevalence to be similar in Maori and non-Maori)[1]. The regional findings were not presented separately for Maori and non-Maori. However, overall there was no urban/rural difference in adult asthma prevalence: the prevalence of asthma was 15.5 % in urban areas, 14.7 % in provincial areas, and 13.8 % in rural areas. In North Island electorates, the highest age- and ethnicity-standardised prevalences were found in some of the electorates in the Auckland and Wellington urban regions, although prevalence was also high in some rural electorates including Raglan (18.0%), Horowhenua (18.4%) and Wairarapa (18.4%); the lowest prevalences were found in other rural electorates including King Country (5.5%), Matamata (10.1%) and Rotorua (10.3%). In South Island electorates, the highest prevalences were found in the Christchurch and Dunedin urban areas, and the lowest prevalences were again found in rural electorates including Clutha (11.3%), Rangiora (9.5%), and Wallace (9.4%).

Thus, previous studies of asthma prevalence in New Zealand children and adults show little evidence of systematic urban/rural differences in asthma prevalence. The small differences that may exist involve slightly lower prevalence in rural areas. However, these findings were not reported separately for Maori and non-Maori, and were related to asthma prevalence rather than severity. While they are imperfect measures of asthma morbidity, hospitalisation and mortality rates do nonetheless provide important information as markers of asthma severity, although it is possible that the level of severity may still be underestimated. While these analyses examine regional and ethnic differences in asthma hospitalisation rates we acknowledge that trends in asthma admission rates are difficult to interpret, being dependent on asthma severity, access to healthcare and individual patterns of medical practice.[2]

Methods

Calculation of Maori and non-Maori rates There are considerable problems in the calculation of Maori health statistics, particularly when examining time trends, because of changes in both the numerator and denominator information. These issues have been reviewed in depth elsewhere,[2,9–12] but will be considered briefly here. Prior to 1986, both deaths and census data were based on a biological definition of Maori; from the 1986 Census the question became one of self-identification, and for the 1986 and 1991 censuses the 'sole Maori' definition is the most appropriate in calculating mortality and hospitalisation rates because this provides reasonable consistency over time.[9,10] Changes to ethnicity recording for death certificates in 1995, and further modification of the ethnicity question in the 1996 Census mean that, for both numerator and denominator data, the 'Maori ethnic group' definition is most appropriate from 1996 onwards.[11,12] These problems are less acute when calculating hospitalisation rates, as in this study, since the methods of recording ethnicity in hospitalisation data have not changed markedly during the period under consideration. Furthermore, we are not examining time trends during this period, but rather we are comparing hospitalisation rates in different regions during this time period as a whole. Thus, problems in the classification of ethnicity are likely to result in an underestimate of the overall relative risk between Maori and non-Maori, but are less likely to affect the regional comparisons within Maori and within non-Maori data.

Hospitalisation data We studied asthma hospitalisations (defined here as the primary diagnosis, ICD-9 code 493) in Maori and non-Maori during 1994–2000 using the Ministry of Health filtered, publicly

 © NZMA

funded discharge data set (excludes hospital transfers and duplicate records). For the reasons previously discussed, census data using the 'sole Maori' definition have been used in the calculation of the population totals and hospitalisation rates for 1994 and 1995, while the 'Maori ethnic group' definition is used for the 1996–2000 data set. There were only a small number of hospitalisations for Pacific people in many areas, and therefore the Pacific data were excluded from the analyses. Hence, the term 'other' refers to hospitalisation rates for non-Maori/non-Pacific people.

Data analysis We calculated hospitalisation rates for Maori and non-Maori in each of the 74 territorial local/land authorities (TLAs), commonly referred to as territorial authorities (TAs). TAs were chosen as the area unit for analysis because these provided reasonable numbers of hospitalisations, and it was possible to classify them as urban or rural. The boundaries of TAs are defined according to 'community of interest' considerations including the relevance of the community components to each other and the ability of the unit to effectively service its community.[13] The 74 TAs comprise 15 cities and 59 districts. This classification provides a useful proxy for urban versus rural populations. The term 'urban' relates to the city authorities whose populations are predominantly urban; the term 'rural' relates to those district authorities that have the greatest proportion of their population residing in rural and smaller urban areas.

The TA-based hospitalisation rates are calculated using the spatially aggregated hospitalisation records, which contain the patients' resident domicile codes (alternatively known as census area units). However, there still remain some TAs where hospitalisation or denominator numbers for particular age groups are too small to reliably calculate. Any TAs with fewer than 15 counts for the 1994–2000 period are designated on the maps (Figures 1 and 2) as 'insufficient data'. The 15-count cut-off is due to the large increase in relative standard error (>25% RSE) below this.

Analyses of deaths typically focus on the 5–34 age range, because of the difficulty in confirming asthma diagnoses for deaths outside of these years.[14] While the data are reasonably accurate for asthma hospitalisations[15] we have focused on the 5–34 age group in presenting the findings although other age groupings (0–4, 5–14, 15–34 and 35–74 years) were also analysed and have been included. The software used for the mapping was Environmental Systems Research Institute ArcView 3.2 desktop Geographic Information System. The data were extracted from the NZHIS National Minimum Dataset using SAS 8 for Windows.

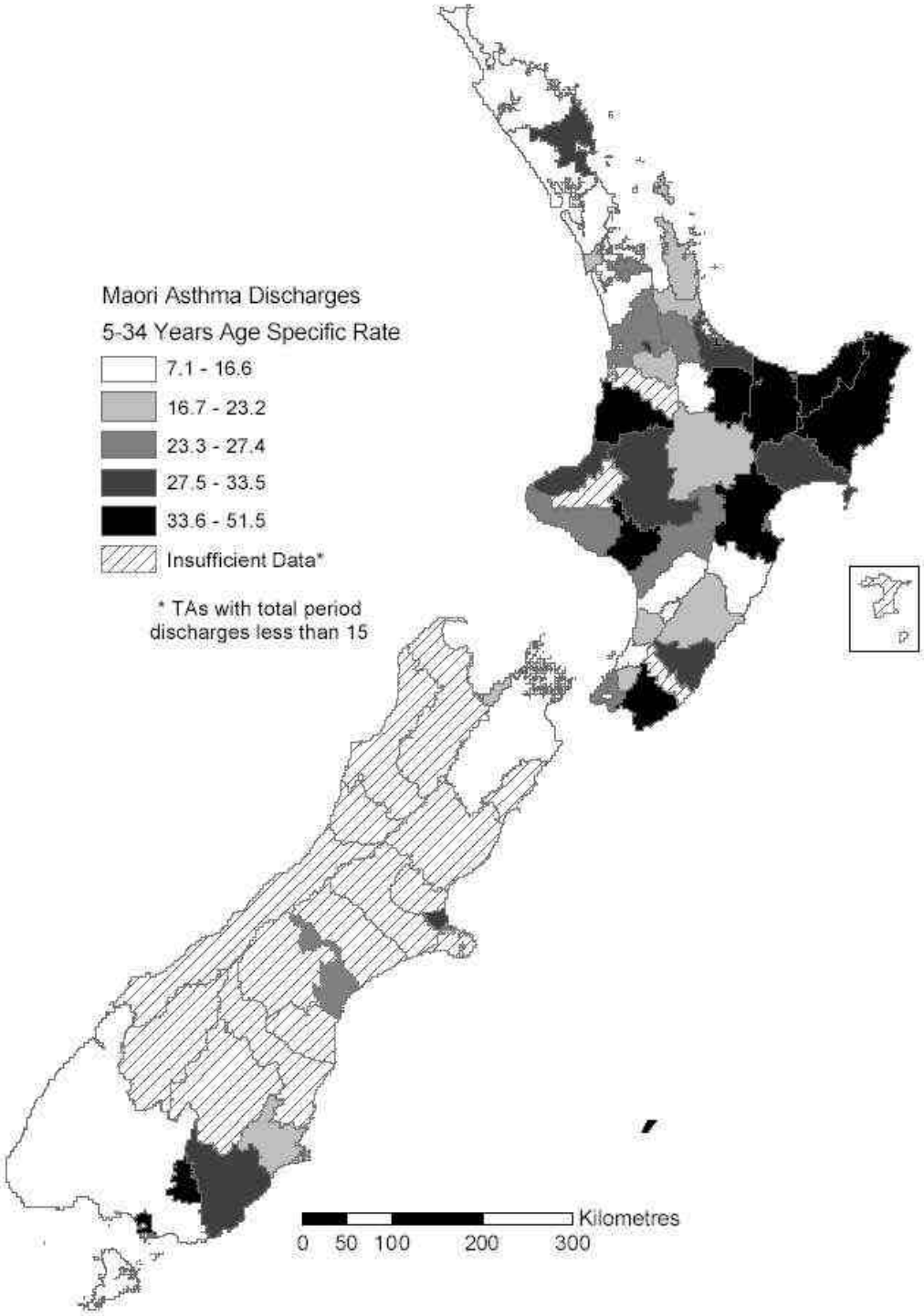
Results

Each of the TAs were ranked based on asthma hospitalisation rates for Maori and non-Maori. Figure 1 shows the map of 5–34 year age-specific discharge rates (per 10 000 per year) for Maori by TA. The highest rates were in Tauranga, Invercargill, Wanganui, South Wairarapa and Gisborne; the lowest rates were in Rodney, Tasman, Franklin, Waitaki and North Shore City. Figure 2 shows the corresponding patterns for non-Maori. Table 1 presents the number of admissions and hospitalisation rates for Maori and non-Maori with the total rate, ranked from highest to lowest, for each TA.

We also conducted regional analyses separately in Maori and non-Maori for the 0–4, 5–14, 15–34 and 35–74 age groups (not shown in the figures). For Maori the highest rates in the 0–4 age group (per 10 000 per year) were in Invercargill (18.08), Tauranga (17.91), Hastings (17.51), Masterton (17.49) and New Plymouth (15.84). In the 5–14 years age group, Invercargill (5.76), Wanganui (4.96), Queenstown Lakes (4.55) and Gisborne (4.43) recorded the highest hospitalisation rates; for 15- to 34-year-olds, South Wairarapa (3.39), Kaikoura (3.27) and Tauranga (3.25) had the highest rates; while Stratford (6.48), Central Hawke's Bay (6.31) and South Taranaki (5.21) had the highest asthma hospitalisation rates for the 35–74 years age group.

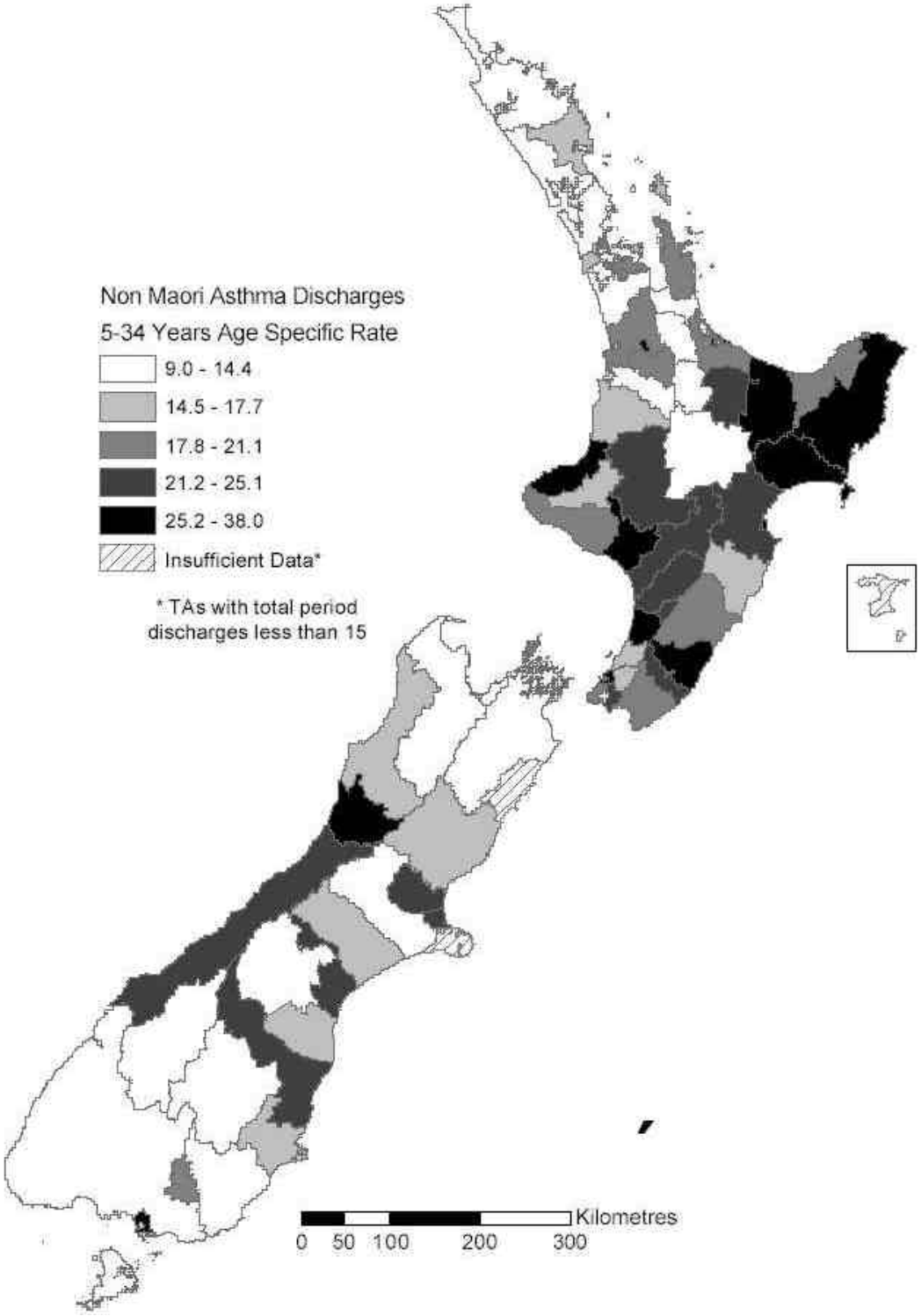
 © NZMA

Figure 1. Maori 5–34 year age-specific asthma discharge rates by territorial authority (1994–2000)



© NZMA

Figure 2. Non-Maori 5–34 year age-specific asthma discharge rates by territorial authority (1994–2000)



© NZMA

Table 1. Maori/non-Maori hospitalisation rates for ages 5–34 years by territorial authority 1994–2000

Territorial authority	Maori		Non-Maori		Total	
	Number	Rate*	Number	Rate*	Number	Rate*
Wanganui District	161	47.4	376	36.9	537	39.5
Gisborne District	341	45.5	233	31.9	574	38.8
Whakatane District	193	36.8	209	38.0	402	37.4
Tauranga District	259	51.5	560	32.6	819	36.8
New Plymouth District	119	32.2	623	36.2	742	35.5
Masterton District	47	30.0	177	33.9	224	33.0
Rotorua District	389	42.9	315	25.1	704	32.6
Grey District	8	19.1	130	33.9	138	32.4
Invercargill City	133	49.0	405	28.5	538	31.8
Opotiki District	70	37.4	22	20.8	92	31.4
Kawerau District	63	33.5	24	25.6	87	30.9
Wairoa District	65	31.7	31	26.7	96	29.9
Napier City	92	26.0	379	30.7	471	29.7
Hastings District	253	41.5	355	24.0	608	29.1
Hamilton City	248	29.5	847	28.0	1095	28.3
Far North District	265	32.1	190	24.1	455	28.2
Porirua City	99	26.7	346	28.1	445	27.8
Ruapehu District	79	31.9	75	21.9	154	26.1
Christchurch City	291	31.3	2211	24.7	2502	25.3
South Wairarapa District	24	46.8	39	19.3	63	24.9
Lower Hutt City	162	27.3	612	24.3	774	24.8
Horowhenua District	45	20.0	167	26.5	212	24.8
Timaru District	23	23.5	267	24.3	290	24.2
Rangitikei District	37	23.3	89	24.3	126	24.0
Waitomo District	48	36.1	27	15.0	75	24.0
Westland District	6	15.8	54	25.0	60	23.6
Papakura District	102	27.3	201	20.9	303	22.7
Carterton District	6	20.9	38	22.8	44	22.5
Gore District	19	34.8	69	19.8	88	21.8
Manukau City	468	25.7	1427	20.4	1895	21.5
Palmerston North C	69	16.5	499	22.2	568	21.3
Waimakariri District	12	13.9	192	22.0	204	21.2
Waitaki District	4	9.7	120	22.0	124	21.1
Thames-Coromandel District	29	20.8	99	21.1	128	21.0
Western Bay of Plenty District	70	28.7	135	18.4	205	21.0
Manawatu District	22	15.1	166	21.9	188	20.8
Whangarei District	185	28.7	236	17.0	421	20.7
South Taranaki District	54	25.8	139	18.8	193	20.3
Waikato District	101	24.3	153	17.8	254	19.9
Tararua District	26	19.4	85	18.6	111	18.8
Wellington City	139	26.6	888	17.9	1027	18.7
Waipa District	47	19.4	184	18.5	231	18.7
Upper Hutt City	36	18.0	176	17.7	212	17.8
North Shore City	66	12.7	884	18.2	950	17.6
Auckland City	304	23.2	1727	16.9	2031	17.6
Hurunui District	4	18.1	42	17.5	46	17.6
Marlborough District	26	16.4	167	17.7	193	17.5
Dunedin City	59	19.3	640	17.4	699	17.5
Waimate District	2	19.8	33	17.2	35	17.3
Kapiti Coast District	27	15.9	142	17.5	169	17.2
Buller District	6	17.3	45	16.7	51	16.8

© NZMA

Central Hawke's Bay District	19	16.6	48	16.5	67	16.5
Waitakere City	166	19.3	701	15.8	867	16.4
Ashburton District	6	13.3	106	16.0	112	15.8
Stratford District	9	21.3	39	14.8	48	15.7
Clutha District	20	31.5	64	13.2	84	15.4
Taupo District	67	17.9	78	12.8	145	14.8
Mackenzie District	2	16.7	17	14.4	19	14.6
Nelson City	31	22.9	148	13.4	179	14.4
Southland District	17	15.0	122	14.3	139	14.4
Rodney District	16	7.1	245	15.3	261	14.3
Matamata-Piako District	42	27.4	91	11.6	133	14.2
Kaikoura District	6	30.1	8	10.0	14	14.0
Kaipara District	25	16.6	47	12.7	72	13.8
Hauraki District	23	18.1	49	12.2	72	13.6
South Waikato District	49	15.9	61	11.0	110	12.7
Selwyn District	3	4.7	94	12.5	97	11.9
Central Otago District	2	4.6	46	12.5	48	11.7
Otorohanga District	5	4.8	30	13.8	35	10.9
Tasman District	10	9.4	98	9.6	108	9.6
Queenstown-Lakes District	6	16.5	40	9.0	46	9.5
Franklin District	30	9.4	108	9.0	138	9.1
Chatham Islands District	1	6.6	1	11.3	2	8.4
Banks Peninsula District	4	17.3	12	6.9	16	8.1
All New Zealand*	**6036**	**27.9**	**19829**	**21.0**	**25865**	**22.3**

***5–34 years age-specific rate per 10 000

Table 2 shows the findings (as age-specific rates per 10 000) grouped into urban and rural areas. In each age group the relative risk of hospitalisation for Maori was higher in rural TAs than in urban TAs, whereas for non-Maori overall the relative risk of hospitalisation was higher in urban than in rural TAs.

Table 2. Hospitalisation rates by age and ethnicity for rural/urban territorial authorities (per 10 000)

Age group	Maori	Other	Relative risk (RR) Maori vs other	95% CI
0–4 years				
Rural	1108.7	673.0	1.65	1.58–1.71
Urban	1003.8	805.3	1.25	1.20–1.30
Total	**1064.7**	**745.0**	**1.43**	**1.39–1.47**
5–14 years				
Rural	262.0	222.9	1.17	1.11–1.24
Urban	241.9	245.7	1.00	0.92–1.05
Total	**254.1**	**234.8**	**1.08**	**1.04–1.13**
15–34 years				
Rural	173.3	129.2	1.34	1.27–1.42
Urban	140.0	114.5	1.22	1.15–1.30
Total	**157.7**	**120.1**	**1.31**	**1.26–1.37**
35–74 years				
Rural	259.2	82.8	3.13	2.98–3.29
Urban	244.4	87.7	2.79	2.63–2.95
Total	**253.4**	**85.4**	**2.97**	**2.86–3.08**

© NZMA

Discussion

We have examined regional patterns of asthma hospitalisations in Maori and non-Maori. There are some limitations to the data that should be noted. First, as with our earlier paper examining time trends in hospitalisation and mortality rates,[2] the lack of standardised ethnicity data means that the monitoring of Maori hospitalisation trends is not straightforward, although these problems are unlikely to be of major concern when making regional comparisons during the same time period. Second, the hospitalisation data represent episodes of care and may include people who have been hospitalised on more than one occasion. Similarly, many admissions in the age groups 0–4 years and 35 years or more that are classified as asthma will be due to viral infections and chronic obstructive pulmonary disease respectively. However, this factor is unlikely to have significantly affected the regional patterns presented here.

These analyses confirm previous evidence that asthma hospitalisation rates are higher in Maori than in non-Maori,[2] despite the fact that asthma prevalence is similar in Maori and non-Maori children.[1] They further indicate that this excess of hospitalisations is higher in rural than in urban areas, although the difference in hospitalisation rates is not large. We found non-Maori hospitalisation rates were generally higher in urban areas in contrast with the pattern for higher rates in rural areas seen in the Maori population.

These findings are in contrast with previously published data on regional differences in asthma prevalence.[6,7] The earlier studies showed little or no urban/rural difference, and the small differences that did exist appeared to involve slightly lower prevalences in rural areas. Thus, differences in prevalence are unlikely to account for the higher asthma hospitalisation rates for Maori in rural areas. This suggests that what we have observed reflects differences in asthma exacerbation and disease severity. It is possible that there may be some real differences in asthma prevalence between some TAs; however, the evidence from this review indicates that there does not seem to be any systematic urban/rural difference.

The Maori Asthma Review concluded that asthma was more severe and that hospitalisation and mortality rates for Maori exceeded those of non-Maori primarily because of inadequate access to appropriate healthcare and asthma education.[3,4] It was also reported that these problems may be particularly severe in rural areas. At the individual level, cost was a major factor related to access identified in the Maori Asthma Review.[3] Costs included travel to the doctor's surgery, doctor's fees and prescription charges. These costs might be further exacerbated for those living in isolated rural communities. There was also strong support expressed in the Review for low-cost health clinics, but only as a 'second best' option to the provision of free primary healthcare. The introduction in 1997 of free consultations for patients under six years old may have relieved some of the financial burden associated with visits to GPs for this age group.[16] However, prescription costs for medications remain a major issue and, obviously, there are significant numbers of people with asthma who have been outside of the qualifying age parameter. Similarly, in a 1998 study of 401 low-income households around New Zealand, 56% of participants had not visited a doctor in the previous year because of cost and 17% identified asthma as a condition that had gone untreated as a result of this.[17] GP consultation fees and prescription charges have been under review as part of the Primary Health Care Strategy.[18]

 © NZMA

A study of access and utilisation of primary healthcare amongst Maori and low-income New Zealanders, using data collected during 1994–1995, found cost to be a significant barrier in both population groups, together with poor access to public transport and isolated populations in rural settings.[19] Overseas studies have found that inaccessibility of acute hospital services may increase the risk of asthma mortality.[20] Geographic isolation and limited public transport were documented in the Maori Asthma Review as being significant factors for Maori in their decision making about accessing health services. There was also the additional factor of cost associated with transport for those living in rural or isolated areas, with very limited options available in terms of public transport.[3] The recently announced funding for rural services by the Ministry of Health[21] will concentrate resources on supporting and retaining primary healthcare teams currently working in rural areas as well as provide for some national initiatives to be undertaken for encouraging the recruitment of primary healthcare workers to rural areas on both a short- and long-term basis.

In addition to issues of cost and geographic isolation, the Maori Asthma Review identified differential management of asthma and inadequate access to appropriate healthcare and asthma education as contributing to the high asthma morbidity rate amongst Maori.[3] One study found that Maori were less likely to have an action plan and less likely to use a peak flow meter. Relative to the severity of their asthma, Maori lost more time from work or school and needed more hospital services.[22] A further study in Auckland found that 33% of Polynesian children were not receiving any asthma drugs in the 24 hours prior to a hospital admission compared with 14% of European children. It also found that fewer Maori children were taking preventive medications compared with European children (13% vs 25%). The study concluded that rates of acute, severe asthma, resulting in higher admission rates for Maori and Pacific Islanders, were primarily due to differences in medical management. Issues such as compliance and utilisation of services have been shown to be contributing factors, but the major influence was that of the prescribing patterns of medical practitioners.[23] Similar conclusions have been reached in subsequent studies, which propose that differences in asthma morbidity, between Maori and non-Maori, are most likely related to differences in access to, and delivery of, asthma care.[1] It is not clear whether these problems of asthma management are particularly acute in rural areas. However, where Maori have been actively involved in the planning, establishment and maintenance of rural, community-led asthma self-management programmes, improved access to health services and reduced asthma morbidity was seen.[24,25]

Passive exposure to tobacco smoke may contribute to the increased hospital admissions seen in Maori children, although it is unlikely to entirely explain the level of greater asthma severity reflected in hospital admission rates.[26] One New Zealand study of adult asthma prevalence suggests that the increased frequency of symptoms amongst adult Maori may in part be a reflection of greater non-allergenic bronchial symptoms related to increased exposure to tobacco, both actively and passively, compared with non-Maori.[27]

In summary, while we have found that there are rural/urban differences in Maori and non-Maori asthma hospitalisation rates, these differences are not large, and there are Maori/non-Maori differences within urban areas as well as within rural areas. However, it is likely that the higher asthma hospitalisation rates among Maori that we have observed reflect differences in asthma exacerbation and disease severity as a

 © NZMA

result of reduced access to asthma health services, which may be particularly acute for those people living in rural areas.

Author information: Lis Ellison-Loschmann, HRC Maori Health Research Training Fellow, Centre for Public Health Research, Massey University; Ron King, Public Health Intelligence, Ministry of Health, University of Auckland; Neil Pearce, Professor, Centre for Public Health Research, Massey University, Wellington

Acknowledgements: Lis Ellison-Loschmann is funded by a Training Fellowship in Maori Health Research, and the Centre for Public Health Research is supported by a Core Programme Grant from the Health Research Council. Thanks to Craig Wright at the Ministry of Health for assisting with collating the data used in these analyses.

Correspondence: Lis Ellison-Loschmann, Centre for Public Health Research, Massey University Wellington Campus, Private Box 756, Wellington. Fax: (04) 380 0600; email: l.ellison-loschmann@massey.ac.nz

References:

1. Ellison-Loschmann L, Pearce NE. He Mate Huango: an update on Maori asthma. Pac Health Dialog. 2000;7:82–93.
2. Ellison-Loschmann L, Cheng S, Pearce N. Time trends and seasonal patterns of asthma deaths and hospitalisations among Maori and non-Maori. NZ Med J. 2002;115:6–9.
3. Pomare EW, Tutengaehe H, Hight M, et al. He Mate Huango: Maori Asthma Review. Wellington: Huia Publications; 1991.
4. Pomare E, Tutengaehe H, Ramsden I, et al. Asthma in Maori people. NZ Med J. 1992;105:469–70.
5. Holt S, Pearce N. Asthma in New Zealand: myths and realities. NZ Med J. 2000;113:39–41.
6. Asher MI, Barry D, Clayton T, et al. The burden of symptoms of asthma, allergic rhinoconjunctivitis and atopic eczema in children and adolescents in six New Zealand centres: ISAAC Phase One. NZ Med J. 2001;114:114–20.
7. Lewis S, Hales S, Slater T, et al. Geographical variation in the prevalence of asthma symptoms in New Zealand. NZ Med J. 1997;110:286–9.
8. D'Souza W, Lewis S, Cheng S, et al. The prevalence of asthma symptoms, bronchial hyperresponsiveness and atopy in New Zealand adults. NZ Med J. 1999;112:198–202.
9. Pomare E, Keefe-Ormsby V, Ormsby C, et al. Hauora. Maori Standards of Health III. A study of the years 1970–1991. Wellington: Eru Pomare Maori Health Research Centre; 1995.
10. Te Roopu Rangahau Hauora a Eru Pomare. Counting for nothing: understanding the issues in monitoring disparities in health. Social Policy Journal of New Zealand. 2000;14:1–16
11. Sporle A, Pearce N. Impact of changes in the death registration process upon Maori mortality statistics. NZ Med J. 1999;112:411–2.
12. Sporle A, Pearce N, Davis P. Social class mortality differences in Maori and non-Maori men aged 15–64 during the last two decades. NZ Med J. 2002;115:127–31.
13. New Zealand Standard Areas Classification Manual 1992, Catalogue No.19.035.0092. Wellington: Department of Statistics; 1992.
14. Sears MR, Rea HH, de Boer G, et al. Accuracy of certification of deaths due to asthma. A national study. Am J Epidemiol. 1986;124:1004–11.

 © NZMA

15. Pearce N, Beasley R, Burgess C, Crane J. Asthma epidemiology: principles and methods. New York: Oxford University Press; 1998.

16. Newton-Howes G, Patel H, Frizzell C, et al. Free visits for under sixes – has it made a difference? NZ Fam Physician. 1998;25:60–1.

17. Waldegrave C, King P, Stuart S. The monetary constraints and consumer behaviour in New Zealand low income households. Wellington: The Family Centre Social Policy Research Unit; 1999.

18. Ministry of Health. The Primary Health Care Strategy. Wellington: Ministry of Health; 2001.

19. Barnett JR, Coyle P. Social inequality and general practitioner utilisation: assessing the effects of financial barriers on the use of care by low income groups. NZ Med J. 1998;111:66–70.

20. Jones AP, Bentham G, Horwell C. Health service accessibility and deaths from asthma. Int J Epidemiol. 1999;28:101–5.

21. Ministry of Health. Funding for rural primary health care workforce retention and recruitment. Available online. URL: http://www.moh.govt.nz/moh.nsf/wpg_index/News+and+Issues-Funding+for+rural+primary+health+care+workforce+retention+and+recruitment Accessed January 2004.

22. Garrett JE, Mulder J, Wong-Toi H. Reasons for racial differences in A&E attendance rates for asthma. NZ Med J. 1989;102:121–4.

23. Mitchell EA. Racial inequalities in childhood asthma. Soc Sci Med. 1991;32:831–6.

24. D'Souza W, Crane J, Burgess C, et al. Community-based asthma care: trial of a "credit card" asthma self-management plan. Eur Respir J. 1994;7:1260–5.

25. D'Souza WJ, Te Karu H, Fox C, et al. Long-term reduction in asthma morbidity following an asthma self-management programme. Eur Respir J. 1998;11:611–6.

26. Shaw R, Woodman K, Crane J, et al. Risk factors for asthma symptoms in Kawerau children. NZ Med J. 1994;107:387–91.

27. Crane J, Lewis S, Slater T, et al. The self reported prevalence of asthma symptoms amongst adult New Zealanders. NZ Med J. 1994;107:417–21.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Motor vehicle traffic crashes involving Maori

Melanie Sargent, Dorothy Begg, John Broughton, Shaun Stephenson, Craig Wright and Joanne Baxter

Abstract

Aims To provide a descriptive epidemiology of fatal and non-fatal motor vehicle traffic (MVT) crashes involving Maori (1980–1994 inclusive) and to describe factors associated with those crashes.

Methods A data set for 1980–1994 was created by linking: (1) the New Zealand Police traffic crash reports (providing crash details), and (2) the New Zealand Health Information Services (NZHIS) hospital inpatient and mortality files (providing ethnicity, other demographic and injury details).

Results The linked data set contained 8178 MVT crash events involving 8273 vehicles/drivers in which there were 9288 Maori casualties. Findings showed 82% of Maori casualties were aged less than 34 years and 70% were male. Nearly 30% were car drivers, 32% passengers, 15% motorcyclists and 12% pedestrians. Most crashes occurred in fine weather (82%), on a sealed (98%), two-way (97%) road, in or near an urban area (62%). The largest proportion (44%) of crashes occurred between 5pm and midnight and the most common days of the week on which they occurred were Saturday (21%) and Friday (19%). The most common type of crash was 'loss of control' on a corner (27%) or a straight road (13%).

Conclusions Motor vehicle traffic crashes are a major cause of mortality and morbidity for Maori. Future research, and the development of strategies to prevent traffic-related injury among Maori, would be facilitated by the inclusion of an ethnicity indicator on the traffic crash reports, and the collection of more comprehensive crash data on safety measures, such as occupant protection and driver-licence status.

Motor vehicle traffic (MVT) crashes are a leading cause of injury mortality and hospitalisation among Maori, particularly young Maori.[1,2] Despite this, there has been little research identifying factors associated with crashes involving Maori with a subsequent paucity of information to develop effective strategies aimed at reducing Maori mortality and morbidity related to motor vehicle crashes.

An important factor contributing to this lack of information is difficulty in obtaining data by ethnicity. Police traffic crash reports incorporate details about the crash and are required for all crashes involving injury, but these reports have not included ethnicity. The New Zealand Health Information Services (NZHIS) mortality and hospitalisation databases do record ethnicity. Thus, the linking of records on the traffic crash report database (crash details), to the NZHIS fatality and hospitalisation records for those people with injuries related to motor vehicle crashes (termed 'casualties') provides one means of identifying crashes by ethnicity. Variables common to both databases are the names and some personal details (eg, date of birth) of crash victims. The feasibility of using these variables to electronically link the

© NZMA

NZHIS records and the police traffic crash reports has been investigated, and a satisfactory level of linkage achieved.[3] This procedure has been used for creating a database for other research studies,[4] and was used for the present study.

The aim of this research was to undertake a Maori-focused analysis of factors associated with MVT crashes involving Maori. This paper describes selected demographic characteristics of Maori injured in MVT crashes (between 1980 and 1994), details of injuries sustained, and environmental factors associated with the crashes.

Methods

The database used for this research was created by linking the New Zealand Police traffic crash reports (TCRs) to the New Zealand Health Information Services (NZHIS) hospital inpatient and mortality files for the years 1980 to 1994. MVT crash casualties were identified from the NZHIS data using the External Cause of Injury codes, E810–819.[5] Maori were identified using the ethnicity indicator contained on this file. The NZHIS records and TCRs were linked using Automatch.[6] A full description of the linkage procedure has been provided elsewhere.[3]

To examine whether the linked database was representative of the original NZHIS data, several comparisons were made. For the fatalities a comparison of the number of linked records with the number of original NZHIS fatality records showed that the linkage rate ranged from 75–100%. Linkage of the non-fatal crashes was not expected to be as high as for fatalities because of the under-reporting of non-fatal crashes to the police.[3] Also, in the earlier years of this study, the names of casualties were not recorded on TCRs thus making linkage very difficult. An inspection of the linkage rate for non-fatal casualties showed that for some of the earlier years as few as 10% of the hospital records were linked to a TCR. For the later years, when full names were recorded, this increased to 56%. Despite the low linkage rates, the distributions by gender and road-user status of the cases included in the linked database were very similar to those in the original hospital database. For example: 68% of the original file and 69% on the linked file were male; 24% versus 28% were vehicle drivers; 32% versus 32% passengers; 17% versus 15% motorcycle drivers; 3% versus 2% were pillion passengers; and 11% versus 11% were pedestrians.

The NZHIS data included an ethnicity indicator, other demographic data (age, gender, domicile) and information on the nature and type of injury sustained by non-fatal casualties. The severity of the primary injury was scored according to the Abbreviated Injury Scale (AIS),[7] which is a six-point scale of anatomical threat to life ranging from minor (1) to virtually unsurvivable (6). Scores were derived for the period 1988–1994 using ICDMAP-90.[8] Prior to 1988 injury diagnosis coding was not in a format that allowed mapping to AIS.

Information on occupant protection (eg, child restraints, seatbelts, and helmets) was available for the years 1980–87 only, because the recording of these data on the TCRs was discontinued after 1987. Deprivation scores were calculated from the domicile information (place of residence) contained on the NZHIS file, which was matched to the NZDep91.[9] The NZDep91 is an index of deprivation calculated by combining census data including income, transport, living space, home ownership and employment. Deprivation scores were calculated for casualties between 1989 and 1993 only (two years either side of the index year of 1991).

In this study ‘casualties’ refers to both fatal and non-fatal injury outcomes, and the casualty factors examined were considered separately for fatal and non-fatal crash victims.

Results

From 1980 to 1994, there were 8178 crash events involving 9288 Maori casualties, 1240 (13%) of whom were fatally injured. The distribution of the casualties by age and gender, by crash outcome, is given in Table 1, and shows the highest proportion of casualties (both fatal and non-fatal) were aged 15–24 years and almost 70% were male.

© NZMA

Table 1. Demographic characteristics of fatal and non-fatal Maori casualties in motor vehicle traffic crashes, 1980–94

	Fatal		Non-fatal		Both	
	n	**%**	**n**	**%**	**n**	**%**
Age						
0–14	1315	16.3	139	11.2	1454	15.7
15–24	3633	45.1	477	38.5	4110	44.3
25–34	1784	22.2	288	23.2	2072	22.3
35–44	669	8.3	140	11.3	809	8.7
45–54	347	4.3	83	6.7	430	4.6
55–64	206	2.6	64	5.2	270	2.9
65–74	76	1.0	31	2.5	107	1.2
75+	18	0.2	18	1.5	36	0.4
Total	**8048**	**100.0**	**1240**	**100.0**	**9288**	**100.0**
Gender						
Female	2462	30.6	353	28.5	2815	30.3
Male	5586	69.4	887	71.5	6473	69.7
Total	**8048**	**100.0**	**1240**	**100.0**	**9288**	**100.0**

The relative deprivation distribution (the deprivation scores (deciles) based on the NZDep91 for the casualties in the years 1989–1993) is shown in Table 2. There was a higher proportion of casualties living in areas of higher relative deprivation, with 44% of the non-fatal and 40% of the fatal casualties in the group having a decile score of 9–10.

Table 2. Distribution of deprivation scores (NZDep91) for fatal and non-fatal Maori casualties in motor vehicle traffic crashes, 1989–1993*

NZDep91 decile score	**Fatal**		**Non-fatal**		**Both**	
	n	**%**	**n**	**%**	**n**	**%**
0 (missing)	27	0.8	2	0.5	29	0.7
1–2	130	3.7	10	2.2	140	3.4
3–4	354	9.9	42	9.4	396	9.8
5–6	570	15.9	85	19	655	16.3
7–8	937	26.2	131	29.3	1068	26.6
9–10	1559	43.6	177	39.6	1736	43.1
Total	**3577**	**100.0**	**447**	**100.0**	**4024**	**100.0**

*two years either side of the index year of 1991

The injury diagnoses for the primary injury, sustained by those with non-fatal injuries, are given in Table 3. Head injuries were the most common injury (35%). The severity of the injuries assessed using the Abbreviated Injury Scale (AIS) showed that 46% were serious/moderate and, in the acute phase of treatment, 28% of casualties with non-fatal injuries spent more than a week in hospital.

© NZMA

Table 3. Primary injury diagnosis, injury severity and length of stay in hospital for Maori casualties in motor vehicle traffic crashes, non-fatal injuries only

Diagnosis	**n**	**%**
Intracranial injury	2187	27.2
Skull fracture	620	7.7
Fracture – lower limb	1328	16.5
Fracture – neck/trunk	735	9.1
Fracture – upper limb	494	6.1
Open wound – neck, head, trunk	756	9.4
Open wound – lower/upper limb	376	4.7
Internal – chest, abdomen, pelvis	505	6.3
Contusions	464	5.8
Other	583	7.2
Total	**8048**	**100.0**
Injury severity (AIS scores)	**n**	**%**
1 Minor	1200	24.4
2 Moderate	1687	34.3
3 Serious	570	11.6
4 Severe	90	1.8
5 Critical	20	0.4
6 Maximum	5	0.1
9 Unknown	1346	27.4
Total	***4918**	**100.0**
Number of days in hospital	**Frequency**	**%**
0	656	8.2
1–2	3054	37.9
3–4	1165	14.4
5–7	917	11.4
8–14	999	12.4
15–21	402	5.0
22+	855	10.6
Total	**8048**	**99.9**

*includes injuries from 1988–1994

An examination of the distribution of casualties by road-user status (Table 4) shows that of the fatal casualties 31% were passengers, 37% drivers, 12% motorcycle drivers, and 14% pedestrians. The distribution was similar for non-fatal casualties.

Table 4. Road-user status of Maori casualties in motor vehicle traffic crashes, 1980–94, by crash severity

Road-user status	**Non-fatal**		**Fatal**		**Both**	
	n	**%**	**n**	**%**	**n**	**%**
Passenger in motor vehicle*	2547	31.6	386	31.1	2933	31.6
Driver of motor vehicle*	2273	28.2	463	37.3	2736	29.5
Motorcyclist (driver)	1240	15.4	148	11.9	1388	14.9
Motorcyclist (pillion)	188	2.3	19	1.5	207	2.2
Pedestrian	904	11.2	173	14.0	1077	11.6
Bicyclist	252	3.1	35	2.8	287	3.1
Unspecified/other	644	8.0	16	1.3	660	7.0
Total	**8048**	**100.0**	**1240**	**100.0**	**9288**	**100.0**

*other than a motorcycle

© NZMA

Safety protection data for the period 1980–87 showed that protection (a seatbelt or crash helmet) was used by 17% of the fatally injured and 35% of the non-fatally injured casualties, but these data were missing for 45% and 37% of the fatal and non-fatal casualties, respectively.

The environmental conditions associated with the crash events are summarised in Table 5, and show that 82% occurred in fine weather, 98% on a sealed road, and 62% in or near an urban area.

Table 5. Environmental conditions associated with motor vehicle traffic crash events involving at least one Maori casualty, 1980–94

	*n	%
Weather conditions		
Fine	6684	81.7
Heavy rain	267	3.3
Light rain	1072	13.1
Other (eg, mist, snow)	155	1.9
Light conditions		
Bright sun	2249	27.5
Dark	3793	46.4
Overcast	1742	21.3
Twilight	369	4.5
Missing	25	0.3
Road surface		
Sealed	7975	97.5
Unsealed	203	2.5
Road lanes		
One way	169	2.1
Two way	7903	96.6
Other	106	1.3
Road characteristics		
Flat road	6003	73.4
Hill road	1767	21.6
Bridge/bridge approach	270	3.3
Railway crossing	31	0.4
Other	107	1.3
Road markings		
Centre line	5370	65.7
Centre island	734	9.0
No passing line	514	6.3
Pedestrian crossing	100	1.2
None	1427	17.5
Other	33	0.4
Speed limit		
Urban (50–60 kph)	4051	49.5
Open road (100 kph)	3064	37.5
City outskirts (70–80 kph)	986	12.1
Road works (30 kph)	73	0.9
Other	4	0.1

*total n = 8178

© NZMA

The distribution of crash events by month of the year was relatively even, ranging from 7.3% crashes occurring in February to 9.7% in December. This suggests very little seasonal influence on crash occurrence. Table 6 presents the distribution of crashes by day of the week and time of day. Most crashes occurred between Thursday and Sunday, and the most common times of day in which crashes occurred were between 5pm and midnight (44%), and midday and 5pm (25%).

Table 6. Time of day and day of week for motor vehicle traffic crash events involving Maori, 1980–94

	n	%
Day of week		
Sunday	1247	15
Monday	711	9
Tuesday	821	10
Wednesday	867	11
Thursday	1305	16
Friday	1519	19
Saturday	1682	21
Total	***8152**	**100**
Time of day		
12.00–4.59am	1131	14
5.00–11.59am	1382	17
12.00–4.59pm	2031	25
5.00–11.59pm	3608	44
Total	***8152**	**100**

*data missing for 26 crash events

Table 7 shows that 40% of crashes were due to loss of control; 27% occurred while cornering and 13% on a straight road. Approximately 11% were head-on collisions and nearly 12% involved a pedestrian.

Table 7. Crash classification based on the movement of the vehicles involved in the crash event, for motor vehicle traffic crashes involving Maori, 1980–94

Movement classification	**n**	**%**
Loss of control – cornering	2214	27.1
Loss of control – straight road	1051	12.9
Pedestrian	949	11.6
Head on	933	11.4
Right turn against traffic	443	5.4
Straight ahead, other vehicle turning	379	4.6
Vehicles crossing – no turns	433	5.3
Vehicles crossing – vehicle turning	376	4.6
Collision with an obstruction	388	4.7
Overtaking and lane change	341	4.2
Rear end	216	2.6
Merging or manoeuvring	341	4.2
Other	114	1.4
Total	**8178**	**100.0**

© NZMA

Discussion

This research provides a profile of Maori casualties (ie, Maori who were hospitalised or who died) as a result of motor vehicle injury, as drivers, passengers, bicyclists, or pedestrians. Until recently much of the available information on traffic crashes involving Maori has been based on anecdotal evidence or unpublished data[10] because it has not been possible to identify Maori in the official New Zealand traffic crash database. This research aimed to fill some gaps in our knowledge about motor vehicle crashes involving Maori.

Young age was an important risk factor with two thirds of Maori casualties aged between 15 and 34 years. Rangatahi (young adults) in the 15–24 age group were particularly over-represented (45% of the non-fatal casualties and 39% of the fatalities). Given this age group makes up approximately 21% of the Maori population,[11] the results of this study emphasise the significant burden to the health of young Maori created by death and injury due to motor vehicle crashes.

This research showed that head injuries were incurred by around 35% of the Maori casualties admitted to hospital, followed by fractures of the lower limb, neck and trunk. Long-term disability resulting from injuries not only impacts on the individual but also on carers and whanau, and the short- and long-term consequences of injury for those who survive provide further important motivation for prevention. Durie has commented that the effect of injury to rangatahi is accentuated by the loss of the benefits that can flow from competent, healthy and skilled whanau members.[12] Thus, the provision of appropriate and accessible rehabilitation services, and the ongoing monitoring and evaluation of their effectiveness, is an important part of achieving Maori health gains in injury, alongside effective preventive measures.

Demographically, the over-representation of males is consistent with other research.[13] Alongside being young and male, a high proportion (70%) of Maori casualties were from areas with high levels of deprivation (deciles 7–10). This finding is consistent with international research,[14,15] and highlights the need to further explore the association between the social and economic determinants of health in relation to injury and motor vehicle traffic crashes.

The use of seatbelts in cars and helmets by motorcyclists has been shown to be associated with decreased levels of injury.[16] This was not fully described in the present research as from 1987 onwards these data were no longer recorded in the traffic crash reports. The monitoring of restraint use is undertaken by way of observational surveys. These surveys are limited as certain characteristics of the occupants, including ethnicity, can not be determined by observation alone. This has left an important gap in information about safety protection that would be helpful for setting road safety targets relevant for Maori.

Analysis of the environmental issues highlights the fact that the majority of crashes involving Maori occurred on two-way, sealed roads that were either in a city or on the outskirts of a city. Given that by 1996 more than 80% of Maori lived in urban settings,[11] it would be expected that much of the driving done by Maori would be in or near a city and these results support this. The finding that many of the crashes occurred at night-time during the weekend is consistent with crashes involving young

 © NZMA

people. Also, many of the crashes occurred because the driver lost control of the vehicle, 27% on a corner and 13% on a straight road. This too is consistent with crashes involving young, male drivers. Without further investigation of the factors associated with crashes involving rangatahi it is possible only to speculate about the likely relationship between many of these factors.

Limitations must be considered. First, limitations in this study relate to the accuracy of the ethnicity data within the NZHIS data set. There is the potential to underestimate Maori casualties due to under-reporting of Maori ethnicity on the hospital and mortality databases in the years for which these data were collected. Previous research has shown that Maori ethnicity has been underestimated in hospital inpatients by around 30%.[17] Similarly, under-reporting of Maori in the mortality database has led to underestimation of Maori deaths.[18] The issue of recording ethnicity data on the traffic crash reports has been raised in the 2010 Road Safety Strategy consultation document, with the proposal that guidelines would be developed to standardise the collection of these data.[19]

A further limitation of using the linked data set was that trends in crash rates over time could not be examined. In this study the variation in linkage rates from the earlier to the later years meant that the number of cases per year varied greatly over time and this may have been due solely to the linkage process and not fluctuations in the incidence of traffic crashes.

Despite these limitations, this research provides useful information for developing strategies for prevention of road traffic injury among Maori. It reinforces a need for research into rehabilitation and support services for Maori injured as the result of motor vehicle crashes. This paper also highlights the importance of road traffic crashes as a health issue for young Maori males, and reinforces the need for consistent and accurate ethnicity data across all relevant databases.

Author information: Melanie Sargent, Junior Research Fellow, Ngai Tahu Maori Health Research Unit and Injury Prevention Research Unit; Dorothy Begg, Senior Research Fellow, Injury Prevention Research Unit; John Broughton, Associate Professor, Ngai Tahu Maori Health Research Unit; Shaun Stephenson, Research Fellow (Biostatistician), Injury Prevention Research Unit; Craig Wright, Data Manager, Injury Prevention Research Unit; Joanne Baxter, Senior Research Fellow, Ngai Tahu Maori Health Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin

Acknowledgements: This research was funded by a project grant from the Health Research Council of New Zealand (HRC) and by the Accident Compensation Corporation (ACC). The Ngai Tahu Maori Health Research Unit is a health initiative of the Ngai Tahu Development Corporation in partnership with the Dunedin School of Medicine, University of Otago. The Injury Prevention Research Unit is funded by the HRC and ACC. We thank Professor John Langley and Jason Hope of the Injury Prevention Research Unit for their contribution to this research, Mrs Christine Rimene of the Ngai Tahu Maori Health Research Unit for her input and advice in relation to Maori health research, and the Land Transport Safety Authority for providing the traffic crash data.

 © NZMA

Correspondence: Dr Dorothy Begg, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, PO Box 913, Dunedin. Fax: (03) 479 8337; email: dorothy.begg@ipru.otago.ac.nz

References:

1. Langley J, Broughton J. Injury to Maori. I: Fatalities. NZ Med J. 2000;113:508–10.
2. Broughton J, Langley J. Injury to Maori. II: Serious injury. NZ Med J. 2000;113:511–3.
3. Alsop J, Langley J. Under-reporting of motor vehicle traffic crash victims in New Zealand. Accid Anal Prev. 2001;33:353–9.
4. Begg DJ, Stephenson S, Alsop J, Langley J. Impact of graduated driver licensing restrictions on crashes involving young drivers in New Zealand. Inj Prev. 2001;7:292–6.
5. World Health Organization. International classification of diseases. Geneva: World Health Organization; 1978.
6. Match Ware Technologies Inc. AUTOMATCH Generalized Record Linkage System. Silver Spring, MD, USA; 1995.
7. American Association for Automotive Medicine (AAAM). The abbreviated injury scale, 1985 revision. Arlington Heights, Illinois: AAAM; 1985.
8. MacKenzie E, Sacco W. ICDMAP-90: A user's guide. Baltimore, MD: The Johns Hopkins University School of Public Health and Tri-Analytics, Inc; 1997.
9. Crampton P, Salmond C, Sutton F. NZDEP91 Index of deprivation. Wellington: Health Services Research Centre; 1997 May, 1997.
10. Land Transport Safety Authority. Background information on Maori in road crashes. Wellington: Land Transport Safety Authority; 2000.
11. Statistics New Zealand Te Tari Tatau. 1996 New Zealand census of population and dwellings: Maori. Wellington: Statistics New Zealand Te Tari Tatau; 1997.
12. Durie M. Mauri Ora: The dynamics of Maori health. Auckland: Oxford University Press; 2001.
13. Tavris DR, Kuhn EM, Layde PM. Age and gender patterns in motor vehicle crash injuries: importance of type of crash and occupant role. Accid Anal Prev. 2001;33:167–72.
14. Hasselberg M, Laflamme L, Weitoft GR. Socioeconomic differences in road traffic injuries during childhood and youth: a closer look at different kinds of road user. J Epidemiol Community Health. 2001;55:858–62.
15. Engstrom K, Diderichsen F, Laflamme L. Socioeconomic differences in injury risks in childhood and adolescence: a nation-wide study of intentional and unintentional injuries in Sweden. Inj Prev. 2002;8:137–42.
16. Evans L. Traffic safety and the driver. New York: Van Nostrand Reinhold; 1991.
17. Harris R, Robson B, Reid P, Keefe V. CHE cultural reviews: ethnicity data collection – error rates and training effectiveness. Wellington: Eru Pomare Maori Health Research Centre; 1997.
18. Reid P. Nga mahi whakahaehae a te tangata tiriti. In: Davis P, Dew K, editors in association with Te Ropu Rangahau Hauora A Eru Pomare. Health and society in Aotearoa New Zealand. Auckland: Oxford University Press; 1999. p. 83–93.
19. National Road Safety Committee. Road Safety Strategy 2010: a consultation document. Wellington: Land Transport Safety Authority; 2000.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Representative case series from public hospital admissions 1998 I: drug and related therapeutic adverse events

Robin Briant, Wasan Ali, Roy Lay-Yee and Peter Davis

Abstract

Aims To examine a representative case series of drug and related therapeutic adverse events in New Zealand public hospitals with a view to assessing their occurrence, causation, patient impact and preventability.

Methods An analysis was carried out on 192 drug and related therapeutic adverse events classified by reviewing physicians. These were identified from among 850 adverse events determined by two-stage retrospective review of a representative sample of 6579 medical records drawn from 13 public hospitals in 1998.

Results One fifth of all adverse events were in the combined group, comprising drug (15.4%) and related therapeutic (7.3%) incidents. In comparison with other adverse events these incidents appeared to be more common among older patients, were less likely to have occurred in hospital, and had slightly less impact on patients. Cardiovascular drugs accounted for nearly half of drug events; poor infection management, by contrast, was the dominant factor among related therapeutic events, with problems in cardiovascular management also important. Inadequate monitoring and follow up of medication was identified as the most common reason for failure to prevent both drug and related therapeutic adverse events.

Conclusions This study shows that morbidity related to medications is extensive. Drug-related adverse events are frequent and many are preventable. Better monitoring and more appropriate medication choice for individuals are the most common prevention strategies identified.

The recently completed New Zealand Quality of Healthcare Study (NZQHS) examined 6579 medical records using two-stage retrospective review applied to a representative sample of hospital admissions for the calendar year 1998. The sample was drawn by systematic list selection, after the exclusion of specialist institutions, from 13 public hospitals providing acute care and with over 100 beds. The main aim was to quantify the impact of adverse outcomes of healthcare management in the New Zealand public hospital system.[1]

The NZQHS reported that 12.9% of public hospital admissions were associated with an adverse event,[1] a rate that is similar to those recorded for Australia (16.6%) and the United Kingdom (10.8%) in comparable studies.[2,3] Half of these events in New Zealand were preventable and occurred inside hospital and, of these, 7.5% were associated with pharmacological treatment and 10.7% with therapy-related incidents.[4]

The United States (US) report on the quality of care by the Institute of Medicine has raised international awareness of the incidence and effects of adverse drug events in hospitals, concluding that, although not all adverse drug events result in actual harm, those that do are costly.[5] Therefore, the principal objective of this paper is to assess

© NZMA

the situation in New Zealand by 'getting behind' such aggregate figures and examining in greater detail this important subset of adverse events. Hence, these events are presented as a representative case series and analysed according to their occurrence, causation and preventability, as well as their patient impact and implications for improved patient care.

Methods

The method of data collection used in the NZQHS, as reported elsewhere,[6] was based on that used in the Harvard Medical Practice Study (HMPS)[7,8] and the Quality of Australian Health Care Study (QAHCS).[2] It involved structured, implicit review of randomly selected hospital records, seeking evidence of potential harm to patients attributable to healthcare management.

An adverse event (AE) was operationally defined as: (1) an unintended injury; (2) resulting in disability; and (3) caused by healthcare management rather than the underlying disease process.

Patient impact was measured by disability defined as temporary, lasting up to a year, or permanent impairment of function, or death. Attributable bed days refer to those extra days associated with an AE that were spent in the study hospital during one or more admissions.

Preventability was assessed as an error in healthcare management due to failure to follow accepted practice at an individual or system level.

Figure 1. Examples of drug and related therapeutic adverse events (AEs)

Examples of drug AEs

64-year-old woman, on metoprolol and diuretic for hypertension. Developed upper respiratory tract infection, prescribed co-amoxyclav. On Day 2 developed vomiting and diarrhoea, and continued all medicines. Admitted to hospital Day 4, with severe dehydration. Given 3 litres of intravenous normal saline in a few hours and developed pulmonary oedema, requiring frusemide.
Sequential AEs – medication-related diarrhoea and dehydration, then iatrogenic fluid overload.
High preventability: initial prescription of antibiotic probably not indicated; unmonitored use of IV saline.

71-year-old man given ibuprofen for acute gout. He was on eight other medications at the time. Had a large haemorrhage from the upper GI tract, and was admitted to hospital shocked. He was resuscitated successfully with blood and IV fluids, but later developed infection at IV access site. Treatment for this precipitated diarrhoea with *C. difficile* toxin in stools.
Sequential AEs: medication-related GI bleed, IV line infection, antibiotic-induced colitis.
Moderate preventability: high-risk patient not given gastrointestinal protection.

Examples of drug-related therapeutic AEs

29-year-old woman presents with second thromboembolic episode in nine months. After the first she had been shown to have activated protein C resistance, but warfarin was given for six months only; DVT recurred after warfarin discontinuation.
AE related to non-use of indicated treatment.
High preventability: long-term warfarin use indicated.

64-year-old man admitted intoxicated and with a fractured ankle. Despite the history of heavy, prolonged alcohol use, no withdrawal prophylaxis was given. After open reduction of his fracture, he developed delirium tremens and required additional days in hospital.
AE related to incomplete assessment of risks and non-use of indicated therapy.
Moderate preventability: patient at high risk of withdrawal syndrome.

© NZMA

AEs were classified by study reviewers into a number of clinical areas, including 'operative', 'system', 'drug', 'therapy', 'diagnosis', 'procedure', and 'other'.[6] Those AEs attributed to the 'drug' and 'therapy' areas are further analysed here, using the data provided by the medical reviewer applying the study instrument Review Form 2 (RF2). Each AE was identified from patient records and then detailed in the clinical summary portion of the RF2. An AE was classified as drug related where medication was implicated in patient harm as shown in the upper panel of Figure 1. Therapeutic AEs, as shown in the lower panel of Figure 1, involved correct diagnosis but inappropriate or delayed treatment. However, for the purposes of the current analysis, only a subset of this otherwise relatively heterogeneous category is included. These are management problems involving medications and fluids; other events involving surgical management problems will be reported elsewhere.

A judgement on the degree to which an individual AE was preventable was also made. The reviewer scored causation and preventability on a scale of 1 to 6, where 1 represents no evidence of causation or preventability, 6 represents virtually certain evidence, and 3 and 4 are 'close calls' either side of a 50:50 likelihood.[6]

The analysis in this paper focuses on medication-related AEs. Therefore, where a common variable for both drug and related therapeutic AEs existed, it has been analysed for the combined group and compared with other AEs.

Results

There were 131 (15.4%) 'drug' AEs (the second most numerous clinical area after 'operative') from a total of 850 AEs. There were another 89 AEs classified as 'therapeutic', 61 of which were related to overall medical management and are included in this series. This gave a subtotal of 192, 22.6% of the total 850, for this analysis of drug and related therapeutic AEs. The patients in this combined group were older than those from the other AE groups, had slightly less additional bed stay, and slightly less disability due to the AE (Table 1). Drug and related therapeutic AEs were more likely to occur outside hospital; 40.1% compared with 13.7% for the other AE group.

Table 1. Distribution of combined drug and related therapeutic adverse events (AEs) and other AEs by mean age of patient, patient impact and location

	All AEs	Drug and related therapeutic AEs	All other AEs
Number	850	192	658
Mean age of patient (years)	51.5	58.6	49.4
Mean added bed days	9.3	7.5	9.8
Patient impact (%)			
Disability <1 month	61.6	71.9	58.6
Disability 1–12 months	19.1	12.5	21.0
Permanent disability	10.1	8.3	10.8
Death	4.5	3.1	4.8
Unable to determine from medical record	4.7	4.2	4.8
Total	**100**	**100**	**100**
Location, n (%)			
Occurred out of hospital	167 (19.6%)	77 (40.1%)	90 (13.7%)
Occurred in hospital	683 (80.4%)	115 (59.9%)	568 (86.3)

In Table 2 the two classes of AE are broken down into their major groups or categories. For medication-related AEs, cardiovascular drugs were by far the largest contributor, making up nearly half the total. Cardiovascular drug problems were

© NZMA

dominated by angiotensin-converting enzyme inhibitors (ACE inhibitors) and/or diuretics, which produced 24 AEs, all involving elderly patients who experienced dehydration, hypotension, syncope or renal failure. Warfarin caused similar numbers of problems to low-dose aspirin (seven and eight respectively), nearly all haemorrhage for both. The centrally acting drugs (responsible for 23 AEs, 17.5%) were a diverse group, with sedation and CNS depression the most common consequences of their use. Eight of these reports concerned opiates, five relating to opiate-induced respiratory depression or coma. Nonsteroidal anti-inflammatory drugs (NSAIDs) were responsible for nine incidents, involving upper gastrointestinal (GI) bleeds or ulcers (6) or renal failure (2). Antibiotics accounted for 18 incidents, the most common being related to cephalosporins (9) and penicillins (5). There were nine instances of antibiotic-induced diarrhoea. Three of these cases (all involving cephalosporins) had definite *Clostridium difficile* superinfection, but in most of the others this toxin was not sought. Seven hypersensitivity reactions to antibiotics were recorded, but on further review two were excluded as being of very dubious hypersensitivity. Of the five with definite allergic reactions, four involved beta-lactam antibiotics; of these two had previously documented penicillin allergy, one had an identical reaction to cefuroxime, and one was given penicillin and flucloxacillin causing a serious systemic reaction.

Table 2. Distribution of adverse events among main drug groups and therapeutic categories

Main drug groups	n	%
Cardiovascular	**59**	**45.1**
ACE inhibitor and/or diuretic	24	
Low-dose aspirin alone	8	
Warfarin alone	7	
Combination of anticoagulants	4	
Amiodarone	4	
Beta-receptor antagonist	4	
Calcium antagonist	4	
Digoxin	2	
Lipid lowering	2	
Central acting	**23**	**17.5**
Opiates	8	
Other	15	
Antibiotics	**18**	**13.7**
Cephalosporins	9	
Penicillins	5	
Other	4	
Nonsteroidal anti-inflammatory	**9**	**6.9**
Other	**22**	**16.8**
Total	**131**	**100**
Main therapeutic categories		
Poor infection management	31	50.8
Poor cardiovascular management	15	24.6
Poor fluid management	7	11.5
Other	8	13.1
Total	**61**	**100**

© NZMA

Of the 61 therapeutic AEs in this analysis there were 31 cases in which poor infection management was at the basis of the AE. In 22 of these cases (71%) the antibiotic choice was inappropriate for the known or likely bacteria present, or the course was too short, or it was given too late. In a further four cases, both poor drainage or wound care and inadequate antibiotic treatment were noted. All resulted in prolonged hospital stay or readmission with unresolved or recurrent infection.

There were 15 instances in which cardiovascular medication use was considered to be incorrect – too much, too little, inappropriately discontinued, or poorly monitored – and at the basis of the AE. These incidents involved the use of warfarin (4 instances), rate-control agents being inadvertently withdrawn (3), and four instances in which treatment for heart failure was inadequately monitored. There were seven cases of poor fluid management, four where excess intravenous fluid was given with consequent pulmonary oedema, and three patients excessively dehydrated. Of the eight miscellaneous cases, two adult patients with depression overdosed on medication supplied to them on their initial admission, and one child overdosed on adult medicine.

Where preventability was identified, the reviewer was required to indicate what steps might have been taken to secure prevention of an event; for the combined drug and related therapeutic group the reasons for failure to prevent AEs are shown in Table 3. Failure to prevent the AE was most commonly seen as a consequence of inadequate monitoring of medication (32.9%), and inappropriate choice of medication (17.5%). Other data show that, for 35 of the drug AEs, system factors were also implicated in causation. Analysis of these shows that, again, failure to monitor patients adequately was the most common problem.

Table 3. Distribution of preventable drug and related therapeutic adverse events (AEs) by reason for failure to prevent

Reasons	**n**	**%**
Inadequate monitoring, follow up	47	32.9
Inappropriate, outmoded therapy	25	17.5
Failure to act on results	16	11.2
Doctor/other practising outside expertise	10	7.0
Failure to perform indicated test	7	4.9
Failure to prevent accidental injury	7	4.9
Avoidable delay in treatment	4	2.8
Failure to check drugs/equipment	4	2.8
Other	23	16.0
Total	**143**	**100**

It should be noted that, in data not presented here, there were four deaths attributed to drug AEs. Two patients were frail and elderly, and suffered consequences of the administration of ACE inhibitors/diuretics and sedating agents that began the spiral of events till death. The other two had antibiotic-related problems: one elderly patient with *C. difficile* diarrhoea, and one young patient with superinfection and multiple organ failure in the context of multiple antibiotic use. The deaths were all considered highly related to the use of the drug concerned (high scores for both causation and preventability).

© NZMA

Discussion

More than 20% of all adverse events identified in New Zealand public hospitals were drug or related therapeutic adverse events, with the former alone accounting for 15%. The prevalence of drug AEs in this sample is comparable to that in the QAHCS (15.4% v 10.8%), and the predominant medications implicated in these events are strikingly similar.[2]

Patients suffering drug or related therapeutic AEs were on average older than those from other AE groups (Table 1). This result is similar to the American study, where both drug-related and therapeutic-related adverse events increased with age.[8] Among all medications involved, cardiovascular drugs were those most often implicated (Table 2) and, of those, ACE inhibitors and diuretics caused the most morbidity. Antibiotic diarrhoea and hypersensitivity were common, and antibiotic choice was often inappropriate for the organisms involved, thus leading to the prolongation or recurrence of infection.

The most common prevention strategies identified were better monitoring of medications with a high likelihood of causing side-effects, and more appropriate medication choice for individuals (Table 3).

The strength of this study is its nationally representative sample of New Zealand hospitalised patients and its internationally based methodology. Furthermore, there is a degree of consistency of certain findings with those of the Australian[2] and American[8] studies.

This method of retrospective record review for identifying AEs has limitations, including only moderate reliability between reviewers,[9] but remains one of the better means so far developed to gauge the adverse impact of medical care on patients. The method does not identify those situations where, despite inappropriate medication, there are no measurable unwanted effects. Thus this type of review does not highlight potential problems and near misses.

Adverse effects of medicines are frequent and often severe.[10] This study suggests that AEs due to medications have a high potential for preventability if proper knowledge and thought goes into the prescription, and proper monitoring of the patient is undertaken. In a recent study of prevalence of medication errors in the US it was found that 56% of adverse drug events detected were due to prescribing errors and 44% to the drug's administration.[10]

Many trials have demonstrated that morbidity and mortality can be reduced if cardiovascular medications are given to patients with established heart disease or hypertension (eg, beta-blockers, ACE inhibitors, A II blockers, spironolactone, statins, aspirin, and warfarin).[11] But the trial patients are carefully selected and seldom match the frailty and the comorbidities of our hospitalised elderly.[12] It is essential that the evidence base of practice includes a realistic assessment of the probability of doing good rather than harm with medications, and that monitoring and early discontinuation are high on the agenda.

However, there are also an important number of patients for whom the AE was related to the non-prescription of indicated medication; whether or not such treatment would have prevented the situation that led to the admission remains speculative, but is assumed in the determination of AEs.

© NZMA

Good prescribing of medication is a challenge that requires correct diagnosis and correct choice of agent, plus the exercise of judgement as to the suitability of the treatment for a given patient. When prescribing for the frail elderly in particular, but also for the chronically impaired or the otherwise healthy individual, the questions to ask are: 'What am I trying to achieve for this patient with this medication?' and 'What is the likely balance of benefit to harm?' If the answer to either of these lacks a clear rationale, the prescription likewise may not be justified and should not be issued. The use of any one, and particularly all three, of the ACE inhibitor, diuretic, and NSAID in the frail elderly is particularly hazardous. These medications should be prescribed with great caution and for good reason, and their use should be monitored carefully and discontinued early.[13]

Computer software to assist the prescriber is now available and its use should improve patient safety, by identifying other drugs or coexisting conditions that would make a new prescription particularly hazardous. Such programmes promise much in decisions about the best drug, the best dose and the safe combinations, and in some situations have been shown to reduce harm from the use of medications.[14–16]

A major finding of this study is the amount of morbidity associated with antibiotics, and resulting from the use of an inappropriate antibiotic or inadequate duration of antibiotics for severe infections. Antibiotic prescriptions are among the most common, both in and out of hospital, and antibiotic resistance is an increasing problem worldwide, requiring optimal targeting of antibiotic to organism. Pestotnik reports on the value of a computer-assisted decision support system with local antibiotic guidelines embedded, to achieve more appropriate antibiotic use, at lesser cost, despite greater overall antibiotic use.[17]

In summary, this analysis of a representative case series of drug-related AEs in New Zealand public hospitals shows that there is substantial morbidity related to medications; this morbidity is worse for older individuals and it causes a significant extra load on inpatient beds. Commonly used drugs cause the most frequent problems; there is some predictability and a moderate degree of preventability about these events. New approaches to drug prescribing and monitoring are needed.

Author information: Robin Briant, Clinical Director, Division of Community Health, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Wasan Ali, Assistant Research Fellow, New Zealand Health Technology Assessment, Department of Public Health and General Practice, Christchurch School of Medicine and Health Sciences, University of Otago, Christchurch; Roy Lay-Yee, Assistant Research Fellow, Centre for Health Services Research and Policy, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Peter Davis, Professor, Department of Public Health and General Practice, Christchurch School of Medicine and Health Sciences, University of Otago, Christchurch

Acknowledgements: Work on this study was funded by the Health Research Council of New Zealand. We thank the 13 New Zealand hospitals that participated in the study and Dr David Richmond (Chair) and members of the study's Advisory and Monitoring Committee. We also thank Professor Stephan Schug, Sandra Johnson and Wendy Bingley, our medical review and data processing teams, and hospital records staff. Professor Alastair Scott was a co-investigator on the study.

 © NZMA

Correspondence: Professor Peter Davis, Department of Public Health and General Practice, Christchurch School of Medicine, University of Otago, PO Box 4345, Christchurch. Fax: (03) 364 0425; email: peter.davis@chmeds.ac.nz

References:

1. Davis P, Lay-Yee R, Briant R, et al. Adverse events in New Zealand public hospitals I: occurrence and impact. . NZ Med J. 2002;115(1167). URL: http://www.nzma.org.nz/journal/115-1167/271/
2. Wilson RM, Runciman WB, Gibberd RW, et al. The Quality in Australian Health Care Study. Med J Aust. 1995;163:458–71.
3. Vincent C, Neale G, Woloshynowych M. Adverse events in British hospitals: preliminary retrospective record review. BMJ. 2001;322:517–9.
4. Davis P, Lay-Yee R, Briant R, et al. Adverse events in New Zealand public hospitals II: preventability and clinical context. NZ Med J. 2003;116(1183). URL: http://www.nzma.org.nz/journal/116-1183/624/
5. Kohn LT, Corrigan JM, Donaldson MS, editors. To err is human. Building a safer health system. Washington, DC: National Academy Press; 1999. Available online. URL: http://books.nap.edu/openbook/0309068371/html/index.html Accessed January 2004.
6. Davis P, Lay-Yee R, Briant R, et al. Adverse events in New Zealand public hospitals: principal findings from a national survey. Occasional Paper No 3. Wellington: Ministry of Health; 2001. Available online. URL: http://www.moh.govt.nz/publications/adverseevents Accessed January 2004.
7. Brennan TA, Leape LL, Laird NM, et al. Incidence of adverse events and negligence in hospitalized patients. Results of the Harvard Medical Practice Study I. N Engl J Med. 1991;324:370–6.
8. Leape LL, Brennan TA, Laird N, et al. The nature of adverse in hospitalized patients. Results of the Harvard Medical Practice Study II. N Engl J Med. 1991;324:377–84.
9. Thomas EJ, Lipsitz SR, Studdert DM, Brennan TA. The reliability of medical record review for estimating adverse event rates. Ann Intern Med. 2002;136:812–6.
10. Barker KN, Flynn EA, Pepper GA, et al. Medication errors observed in 36 health care facilities. Arch Intern Med. 2002;162:1897–903.
11. Williams CM. Using medications appropriately in older adults. Am Fam Physician. 2002;66:1917–24.
12. Davis P, Lay-Yee R, Fitzjohn J, et al. Co-morbidity and health outcomes in three Auckland hospitals. NZ Med J. 2002;115:211–5.
13. Savage R. A dangerous trio. Prescriber Update. 2002;23:20.
14. Raschke RA, Gollihare B, Wunderlich TA, et al. A computer alert system to prevent injury from adverse drug events: development and evaluation in a community teaching hospital. JAMA. 1998;280:1317–20.
15. Bates DW, Pappius EM, Kuperman GJ, et al. Measuring and improving quality using information systems. Medinfo. 1998;9:814–8.
16. Teich JM, Merchia PR, Schmiz JL, et al. Effects of computerized physician order entry on prescribing practices. Arch Intern Med. 2000;160:2741–7.
17. Pestotnik SL, Classen DC, Evans RS, Burke JP. Implementing antibiotic practice guidelines through computer-assisted decision support: clinical and financial outcomes. Ann Intern Med. 1996;124:884–90.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

White-tailed spider bites – arachnophobic fallout?

Jonathan Banks, Phil Sirvid and Cor Vink

Abstract

Aim To investigate if public concern regarding the toxic effects of the bites from white-tailed spiders, *Lampona cylindrata* and *L. murina*, is reflected in the case histories of patients admitted to Christchurch Hospital with a diagnosis of spider bite.

Methods The case histories of patients admitted to Christchurch Hospital with a diagnosis of 'contact with venomous spiders' were examined for evidence that the patients developed necrotising arachnidism.

Results Ten patients were admitted to Christchurch Hospital between January 2001 and January 2003 with a diagnosis of 'contact with venomous spiders'. We found no evidence that patients developed necrotising arachnidism. No patients admitted to Christchurch Hospital required re-admission to treat sequelae of the putative spider bite. Support for a spider bite as the causative agent was not robust and alternative agents could have been the cause.

Conclusions The public's fear of bites from white-tailed spiders is likely misplaced and, if the spider was not caught in the act of biting the patient, alternative diagnoses should be considered before assuming a white-tailed spider was responsible for the patient's symptoms.

Australian white-tailed spiders (*Lampona cylindrata* (L. Koch) and *L. murina* (L. Koch)) are common throughout much of New Zealand and are often associated with human dwellings. *Lampona cylindrata* was first recorded in Nelson in 1913, while *L. murina* (Figure 1) has been known in the North Island for at least 100 years.[1] In their native Australia, *L. cylindrata* and *L. murina* are part of a complex of 57 species,[1] but in New Zealand these two species are the only representatives of their family (Lamponidae) and are distinct visually from other spiders that occur here. Note that the distinction between *L. cylindrata* and *L. murina* was not formalised until Platnick's taxonomic revision in 2000.[1] Consequently, literature prior to this typically refers to a single species, *L. cylindrata*.

Recently, considerable media attention in New Zealand has focused on the adverse effects attributed to bites of white-tailed spiders. New Zealand press headlines and statements, such as 'Fears of biting spider plague',[2] 'Spider suspect in death mystery',[3] and 'Doctors believed the wound was caused by a white-tailed spider',[4] have done much to foster public anxiety about these spiders. These accounts, and others[5] warned of sequelae such as severe skin damage, pain, inflammation and loss of quality of life persisting for several months after alleged white-tailed spider bites. Reports from Australia have suggested that white-tailed spider bites have left their victims with headaches, liver problems, gastrointestinal complaints, and immune system disorders, and that patients are at risk of amputation following the development of gangrene.[6] The term 'necrotising arachnidism' has been used to describe a range of symptoms, from the very general 'potential cutaneous reaction to

© NZMA

spider bite venom',[7] to the more specific 'skin blistering, ulceration and necrosis after spider bite'.[8]

Figure 1. White-tailed spider, *Lampona murina* (scale bar intervals are 1 mm)
© Norm Heke, Te Papa

Understandably there is considerable public concern in New Zealand surrounding the presence of white-tailed spiders. Questions regarding white-tailed spiders made up 22% of all spider inquiries to the Museum of New Zealand Te Papa Tongarewa web site, 15% of spider inquiries to Otago Museum, and 21% of spider inquiries to Auckland Museum and Landcare Research, Auckland (Phil Sirvid, unpublished data for 1995–1997). We examined the records of Christchurch Hospital for patients admitted with a diagnosis of 'contact with venomous spiders' to investigate whether the concern regarding white-tailed spider bites and the sequelae of the spider bites is reflected in admissions to a major regional hospital.

Methods

Patients with reported spider bites were identified from Christchurch Hospital records and their notes examined to investigate the sequelae of white-tailed spider bites and any concurrent medical conditions that may have contributed to the development of adverse reactions to the bites.

© NZMA

Results

Ten patients admitted to Christchurch Hospital between January 2001 and January 2003 were diagnosed with 'contact with venomous spiders'. We reviewed the medical records of nine patients; the records of one patient were unavailable. Patient ages ranged from 15 to 80 years with a mean of 37.6 years. Patients were admitted to the hospital for an average of 3.2 days.

No patient reported capturing or observing a white-tailed spider in the act of biting. Generally the wounds were attributed to white-tailed spiders because of their presence in the patient's environment. Typically the records stated 'thinks was bitten while getting into sleeping bag' or 'has killed several white-tailed spiders over the last few weeks'.

Four of the nine patients had asthma and another patient reported allergies to eggs and the influenza vaccine. Two other patients had multiple medical problems. Six patients had microbiological swabs taken. One patient's swab was negative; one grew group G *Streptococcus* and four grew *Staphylococcus aureus*.

Eight of the nine patients were treated with antibiotics while in hospital. The most commonly prescribed antibiotic was flucloxacillin (six patients), either alone or in combination with a second antibiotic. Other antibiotics used were amoxicillin, penicillin, amoxicillin/clavulanate or cephalexin. No patients required re-admission to the hospital to treat the sequelae of their putative spider bites.

Discussion

Given the media coverage devoted to alleged bite cases and the large number of inquiries to New Zealand museums and similar institutions, surprisingly few people were admitted to Christchurch Hospital with a diagnosis of spider bite. The evidence supporting the diagnosis of bites from white-tailed spiders as the cause of the patients' wounds in the nine patient histories we examined was extremely weak, as no patient reported observing a spider bite them.

The bacteria grown from skin cultures of the Christchurch Hospital patients were unremarkable. *Staphylococcus aureus* is a well-known, transient part of human skin flora, can survive indefinitely in the nostrils and is often one of the pathogens responsible for causing cellulitis.[9] Group G streptococci are also often one of the constituents of the normal skin flora of humans and can produce necrotising soft-tissue infections in patients with underlying medical problems.[10] These infections can require surgical debridement and treatment with antibiotics.[10]

Others have noted that the symptoms described in patients with a putative spider bite can be mistakenly diagnosed as necrotising arachnidism. Other diagnoses of the symptoms that should be excluded before diagnosing necrotising arachnidism include ecthyma, pyoderma gangrenosum, ecthyma gangrenosum, focal vasculitis, foreign body, herpes zoster, purpura fulminans and staphylococcal infections.[7,11]

Despite the well-documented long-term presence[12–14] and widespread distribution throughout New Zealand of white-tailed spiders, as well as their close contact with humans and their distinctive appearance, it is interesting to note that the first New Zealand account of verified white-tailed spider bites does not appear until 1980.[15] A report on the medical impact of insects and arachnids for 1967–1976[16] made no

© NZMA

mention of white-tailed spider bites other than to cite Sunde's paper.[15] Accounts of white-tailed spider bites are also absent from earlier works discussing poisonous spiders.[17,18]

Widespread public concern about white-tailed spiders in New Zealand appears to have started in 1991, when Denis Welch, political writer for the widely read *NZ Listener* was unable to produce his regular column because of an alleged white-tailed spider bite. Since then, there has been a dramatic surge in inquiries about the spider made to institutions such as museums (personal communication, RL Palma, 2003), reflected in the inquiry statistics cited earlier. The scarcity of reports before this date suggests the public perception of these spiders as dangerous may be misplaced.

Many of the case reports from Australia associating necrotising arachnidism with white-tailed spider bites have been drawn from similarly tenuous evidence and there has been considerable debate as to whether white-tailed spiders are responsible for necrotising arachnidism.[11,19–23] Often it is only after problems develop that symptoms are attributed to white-tailed spiders. For example, a case history typical of many of the Australian reports was of an elderly gentleman who presented with painful swelling of his right leg. Three days earlier he had been gardening and noticed the onset of pain in the knee later that evening. He was diagnosed with right ileofemoral venous thrombosis in association with superficial spreading cellulitis. Despite treatment with antibiotics and heparin, the patient eventually required several skin grafts and was discharged after two and half months of hospital care. Partly based on a nurse's experience in Vietnam, the cause was attributed to a spider bite and it was speculated that the spider responsible was a white-tailed spider; all this despite no spider having been seen.[24]

As well as the weakness of the evidence identifying white-tailed spiders as the cause of these necrotic wounds, there is also debate as to whether white-tailed spider venom is toxic to humans. The venom of white-tailed spiders had little effect on mouse skin in vivo and little effect on cultured mouse and human skin.[25] White-tailed spider venom has no sphingomyelinase activity, which is thought to be the enzyme responsible for many of the necrotic effects of the bites of the brown recluse spiders, *Loxosceles rufescens* (Dufour), of North America.[26]

Microorganisms such as *Mycobacterium ulcerans* have also been proposed as a cause of the necrotic skin lesions following putative spider bites.[20,27] However, *M. ulcerans* was discounted as a cause of necrotising arachnidism as the organism does not survive in and will not colonise the midgut of a spider. As *M. ulcerans* survives only briefly on exposed surfaces, inoculation would have to occur simultaneously with a spider bite for a person receiving a bite to be infected.[28] Additionally there is no correlation between areas in Australia where *M. ulcerans* is endemic and the areas from which necrotic arachnidism has been reported.[28] While not yet recorded in association with white-tailed spider bites, the fungal disease sporotrichosis has been documented with bites and stings of other terrestrial arthropods.[29]

A review of 14 Australian cases of suspected white-tailed spider bites found that the spider was positively identified as a white-tailed spider in only three cases.[30] All three patients developed a red, erythematous, itchy rash that formed skin ulcers. In two of the patients the ulcers healed then broke down again and eventually healed.[30] The

 © NZMA

other patient had multiple episodes of shallow lesions that healed but then recurred with a gradual decrease in frequency.[30]

In nine more Australian cases where white-tailed spiders were positively identified as responsible, the bites were all described as painful or severely painful and the bites all occurred indoors.[31] The severity of the pain experienced when bitten suggests to us that those patients who develop lesions overnight, without waking, as occurred with one patient admitted to Christchurch Hospital, are unlikely to have been bitten by a spider.

The practice of blaming spiders for idiopathic necrotic wounds is not restricted to Australia and New Zealand. In the United States, many necrotic wounds are attributed to brown recluse spiders, often despite no record of the presence of brown recluse spiders in the patient's environment.[32] One factor that may pressure New Zealand physicians into attributing idiopathic wounds to white-tailed spiders is New Zealand's system of personal medical insurance, provided by the Accident Compensation Corporation (ACC). The ACC requires an external force to be identified before subsidising medical care and paying benefits to people unable to work. If an external force is not identified, the ACC will not cover the costs associated with the injury.

There were no reports of sequelae from the spider bites severe enough to require re-admission to Christchurch Hospital in the patient histories we examined. Five of the nine Christchurch Hospital patients had a previous history of allergy or asthma and two of the four patients without a history of allergy or asthma had multiple medical problems. It is possible that these concurrent medical conditions contributed to the symptoms experienced by the patients.

Given the weakness of the evidence associating white-tailed spiders with necrotic arachnidism we believe that much of the fear that surrounds these spiders is unwarranted. For example, more people were admitted to New Zealand hospitals as the result of fly bites between 1967 and 1976 than were admitted because of spider bites.[16] We found no evidence that the patients admitted to Christchurch Hospital developed necrotising arachnidism.

Author information: Jonathan C Banks, Drug Information Pharmacist, Clinical Pharmacology, Christchurch Hospital, Christchurch, New Zealand; Phil Sirvid, Arachnologist, Entomology Section, Museum of New Zealand Te Papa Tongarewa, Wellington, New Zealand; Cor J Vink, Arachnologist, Department of Biology, San Diego State University, San Diego, California, USA

Acknowledgements: We thank Professor Evan Begg and the two anonymous NZMJ reviewers for their comments on this paper.

Correspondence: Dr Jonathan Banks, Entomology, 320 Morrill Hall, 505 S Goodwin Ave, University of Illinois, Champaign Urbana, IL 61801, USA. Fax: +1 217 244 3499; email: jbanks@life.uiuc.edu

References:

1. Platnick NI. A relimitation and revision of the Australasian ground spider family Lamponidae (Araneae: Gnaphosoidea). Bull Am Mus Nat Hist. 2000;245:1–330.
2. Boland MJ. Fears of biting spider plague. Evening Post. 2000 Apr 3.
3. Betts M. Spider suspect in death mystery. Dominion Post. 2003 Mar 5.

 © NZMA

4. Price A. Venomous spiders back on the bite. The Mail. 2001Feb 22.
5. McCarthy J. Spider bite painful experience. The Press. 2003 Mar 13.
6. Messenger S. The Spider Research Project. Available online. URL: http://www.bulkmsm.com/research/spider/Australia Accessed January 2004.
7. Sams HH, Dunnick CA, Smith ML, King LE Jr. Necrotic arachnidism. J Am Acad Dermatol. 2001;44:561–73.
8. Hawdon GM, Winkel KD. Spider bite. A rational approach. Aust Fam Physician. 1997;26:1380–5.
9. Failla DM, Pankey GA. Optimum outpatient therapy of skin and skin structure infections. Drugs. 1994;48:172–8.
10. Humar D, Datta V, Bast DJ, et al. Streptolysin S and necrotising infections produced by group G streptococcus. Lancet. 2002;359:124–9.
11. Isbister GK. Spider mythology across the world. West J Med. 2001;175:86–7.
12. Urquhart AT. On new species of Araneida. Trans NZ Inst. 1887;19:72–118.
13. Dalmas CD. Spiders of New Zealand. Ann Soc Ent Fr. 1917;86:317–430.
14. Forster RR. The spiders of New Zealand part V. Cycloctenidae, Gnaphosidae, Clubionidae. Otago Museum Bulletin. 1979;5:1–94.
15. Sunde RG. Lampona cylindrata (Araneae: Gnaphosidae): a note. NZ Entomol. 1980;7:175–6.
16. Brock RL. Medical impact of insects and arachnids in New Zealand 1967–1976. Wellington: NZ Deparment of Health; 1982.
17. Chamberlain G. Arachnoidism as applied to New Zealand spiders. A preliminary note. Rec Auck Inst Mus. 1947;3:157–9.
18. Forster RR, Forster LM. New Zealand spiders: an introduction. Auckland: Collins; 1973.
19. Hodgson WC. Pharmacological action of Australian animal venoms. Clin Exp Pharmacol Physiol. 1997;24:10–7.
20. Harvey MS, Raven RJ. Necrotising arachnidism in Australia: a simple case of misidentification. Med J Aust. 1991;154:856.
21. Sutherland SK. Watch out, Miss Muffet! Med J Aust. 1987;147:531.
22. Sutherland SK. Spider bites in Australia. There are still some mysteries. Med J Aust. 1983;2:597.
23. White J. Necrotising arachnidism. Med J Aust. 1999;171:98.
24. Spring WJ. A probable case of necrotizing arachnidism. Med J Aust 1987;147:605–7.
25. Atkinson RK, Wright LG. Studies of the necrotic actions of the venoms of several Australian spiders. Comp Biochem Physiol C. 1991;98:441–4.
26. Young AR, Pincus SJ. Comparison of enzymatic activity from three species of necrotising arachnids in Australia: Loxosceles rufescens, Badumna insignis and Lampona cylindrata. Toxicon. 2001;39:391–400.
27. Hayman JA, Flemming HB, Monash DA, Miller IM. Mycobacteriun ulcerans infection in paradise. Med J Aust. 1991;155:130.
28. Atkinson RK, Farrell DJ, Leis AP. Evidence against the involvement of Mycobacterium ulcerans in most cases of necrotic arachnidism. Pathology. 1995;27:53–7.
29. Moaven LD, Altman SA, Newnham AR. Sporotrichosis mimicking necrotising arachnidism. Med J Aust. 1999;171:685–6.
30. Pincus SJ, Winkel KD, Hawdon GM, Sutherland SK. Acute and recurrent skin ulceration after spider bite. Med J Aust. 1999;171:99–102.

 © NZMA

31. Isbister G, Gray M. Acute and recurrent skin ulceration after spider bite. Med J Aust. 2000;172:303–4.

32. Vetter RS. Myth: idiopathic wounds are often due to brown recluse or other spider bites throughout the United States. West J Med. 2000;173:357–8.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Gouty tenosynovitis – more common than we think?

David Townshend and Vasu Pai

We report a case of acute, gouty flexor tenosynovitis in a Maori farmer, initially diagnosed as infective tenosynovitis. There was no previous history of gout and the serum urate was normal. Suppurative tenosynovitis caused by bacterial infection is a common condition. Tenosynovitis mimicking bacterial infection may be caused by gout, calcific tendonitis, rheumatoid arthritis and amyloidosis.[1] Gouty tenosynovitis has been reported to mimic tuberculous tenosynovitis,[2] and has been implicated in tendon rupture.[3] Only one case of gouty tenosynovitis mimicking bacterial infection has been previously reported.[4] It is important to recognise gout as a differential diagnosis of infective tenosynovitis, particularly where no organism is found. This is especially important in New Zealand where a high prevalence of gout has been reported in Maori men and men from the South Pacific Islands.[5,6]

Case report

A 52-year-old, Maori male farmer presented with a two-day history of fusiform swelling of the right ring finger. This had become increasingly painful, with difficulty in grip. On examination he had a temperature of 37.2 °C, and a pulse rate of 82/min. His finger was hot, red and swollen, with a tense effusion of the flexor sheath. The finger was held in a slightly flexed position and any attempts at passive extension were extremely painful. He also complained of some pain around the metacarpo-phalangeal (MCP) joint of the middle finger, which on examination showed moderate soft-tissue swelling over the dorsal aspect. There was no history of trauma and the patient was in good health with no history of rheumatoid arthritis, diabetes or alcoholism. He was not on any regular medication and had a normal diet. C-reactive protein was raised at 30 mg/l (normal <9 mg/l) but other laboratory tests were normal: white cell count 9.8, neutrophils 62%, erythrocyte sedimentation rate 10 mm/hour. The blood urate level was also normal at 0.35 mmol/l (normal 0.20 to 0.40 mmol/l). Radiological examination of the hand was unremarkable.

Clinical signs were suggestive of an infective tenosynovitis and so the flexor sheath was explored under general anaesthetic. A classical double-incision technique was used. There was inflammation of the synovium and a small amount of straw-coloured fluid but no other abnormality was seen. The MCP joint of the middle finger was aspirated and was found to contain similar straw-coloured fluid. The joint was opened through a dorsal incision and exploration revealed some chalky material in the joint. This raised a suspicion of gout and aspirates from both the tendon sheath and the joint were sent for polarising microscopy as well as Gram's stain and culture. Aspirates were sent in both plain and EDTA tubes.

Gram's stain did not reveal any organisms and results of blood cultures and culture from the tendon sheath were negative. The aspirates of both the tendon sheath and MCP joint were, however, positive for uric acid crystals. Antibiotics were therefore stopped and the patient was commenced on indomethacin and then allopurinol. The patient received hand physiotherapy and four months later had 80% range of

© NZMA

movement and returned to work as a farmer. Serum urate was 0.41 mmol/l (normal 0.20 to 0.40 mmol/l) at eight weeks.

Discussion

It is not uncommon to see a patient with a diagnosis of 'infective' tenosynovitis where no organism is found in the aspirate. Three such cases of 'sterile flexor tenosynovitis' have been reported,[7] but the incidence in wider practice is uncertain. Strong suspicion is required if cases of gouty flexor tenosynovitis are not to be missed. It is important to differentiate gout from infection although they may coexist.[8] Diagnosis is made by synovial fluid examination for urate crystals and it is recommended that specimens are fixed in alcohol. In 5–15% of cases of gouty arthritis, crystals may be absent in the synovial fluid aspiration and a synovial biopsy may be needed to confirm the diagnosis.[9] Gouty tenosynovitis in the hand can be present without visible tophi or previous involvement of the upper limb,[1] and is difficult for the clinician to differentiate from infective tenosynovitis. Serum urate is often normal at the time of an acute attack of gout. In this patient there was no history of gout, serum urate was normal at presentation and the diagnosis of gouty tenosynovitis would have been missed without the finding of chalky material on exploration of the MCP joint.

The exact incidence of gouty tenosynovitis is unknown. We believe that gout mimicking infective tenosynovitis may be more common than is currently recognised and should be considered in the differential diagnosis. This is of particular note in our New Zealand population where the prevalence of gout is high.

Author information: David Townshend, Honorary Clinical Lecturer, Department of Orthopaedics, Dunedin School of Medicine, University of Otago, Dunedin; Vasu Pai, Registrar, Department of Orthopaedics, Dunedin Hospital, Dunedin

Acknowledgements: We thank Professor John Highton for his assistance in preparing this case report.

Correspondence: Dr Vasu Pai, 207 Musselburgh Road, Dunedin. Email: vasu_chitra@slingshot.co.nz

References:

1. Wolfe SW. Tenosynovitis. In: Green DP, Hotchkiss RN, Pederson WC, editors. Green's operative hand surgery. 4th edition. Philadelphia: Churchill Livingstone; 1999. p. 2022–44.
2. Kostman JR, Rush P, Reginato AJ. Granulomatous tophaceous gout mimicking tuberculous tenosynovitis: report of two cases. Clin Infect Dis. 1995;21:217–9.
3. Hankin FM, Mayhew DE, Coapman RA, et al. Gouty infiltration of a flexor tendon simulating rupture. Clin Orthop. 1985;194:172–5.
4. Abrahamsson SO. Gouty tenosynovitis simulating an infection. A case report. Acta Orthop Scand. 1987;58:282–3.
5. Klemp P, Stansfield SA, Castle B, Robertson MC. Gout is on the increase in New Zealand. Ann Rheum Dis. 1997;56:22–6.
6. Darmawan J, Valkenburg HA, Muirden KD, Wigley, RD. The epidemiology of gout and hyperuricemia in a rural population of Java. J Rheumatol. 1992;19:1595–9.
7. Zubowicz VN, Raine TR. Sterile flexor tenosynovitis of the hand: a report of three cases. J Hand Surg. 1986;11:140–2.

 © NZMA

8. Blyth P, Pai VS. Recurrence of gout after total knee arthroplasty. J Arthroplasty. 1999;14:380–2.
9. Williamson SC, Roger DJ, Petrera P, Glockner F. Acute gouty arthropathy after total knee arthroplasty. A case report. J Bone Joint Surg Am. 1994;76:126–8.

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Thymoma and immunodeficiency

Michela Di Renzo, Anna Pasqui, Fulvio Bruni, Luca Voltolini, Paola Rottoli, Grazia Perali and Alberto Auteri

Good syndrome (GS) was first described in 1954 by Good, who reported hypogammaglobulinaemia in a small percentage of patients with thymoma.[1] GS is a rare, adult-onset, immunodeficiency disease characterised by hypogammaglobulinaemia, low or absent B cells in the peripheral blood and, variably, defects in cell-mediated immunity.[2] GS was often considered a subset of common variable immunodeficiency (CVID) with thymoma, whereas nowadays it is regarded as a distinct clinical entity whose pathogenesis is still uncertain.[3] A bone marrow defect impairing B cell maturation due to an aggression towards B cell precursors has been suggested,[4] and deficiencies in other cell lineages with eosinopenia, pure red cell aplasia or neutropenia are often reported.[5]

Here we report the case of a male patient who developed recurrent respiratory tract infections for two years before being diagnosed with GS.

Case report

A 61-year-old male presented in March 1996 with a two-year history of recurrent respiratory infections including otitis, bronchitis, sinusitis and two episodes of pneumonia. Chest X-ray and chest CT scan showed a mediastinal mass, biopsy of which revealed lymphoid cells.

The mass was resected in June 1996 and histopathologic examination showed a mixed epithelial and lymphocytic thymoma with infiltration of the capsule. The patient had low serum levels of IgG (24 mg/dl, normal >700), IgM (5 mg/dl, normal >40) and IgA (5 md/dl, normal >70). The total number of lymphocytes was normal, but the CD4+ T cell count was 420 per ul, with an inverted CD4/CD8 ratio of 0.39 and absent peripheral B cells. PPD skin reactivity was positive. A serological test for HIV was negative.

Two months after thymectomy, he developed pneumonia due to *Serratia marcescens*, which resolved with antibiotics. Monthly intravenous immunoglobulin (IVIG) infusions were started at a low dosage (150 mg/kg) and the patient improved slightly. In August 1998 he presented with oral thrush, odynophagia, and a weight loss of 7 kg. Upper gastrointestinal endoscopy revealed *Candida oesophagitis*, which was treated with itraconazole. Cough and sputum production persisted. In April 2000 *Haemophilus influenzae* type B was isolated from sputum and the patient was treated with a 15-day course of ceftriaxone. The dosage of IVIG infusion was increased (400 mg/kg every three weeks) and the patient experienced a gradual improvement in his condition, with decreased sputum and cough and fewer febrile episodes. A recent chest CT scan did not show bronchiectasis.

He continues to take daily itraconazole in spite of which he occasionally develops oral candidiasis, which resolves with local nystatin treatment. In 2000 and 2001 immunological evaluation was repeated: CD4+ T cells were still reduced with an

© NZMA

inverted CD4/CD8 ratio. In vitro lymphocyte proliferation was normal. Intracellular IL2 and IFN-γ cytokine production was normal. He also has mild neutropenia (1200/mm^3).

Discussion

GS was one of the first immunodeficiency diseases to be classified.[1] Patients with GS are usually middle-aged or elderly when they develop recurrent infections, most frequently of the respiratory tract. It is usually the infections rather than the local symptoms due to the mediastinal mass that call attention to the possibility of thymoma. Only 3–6% of patients with thymoma are, however, hypogammaglobulinaemic and the relationship between thymoma and the immune dysfunction is not clear;[6] thymoma does not seem to induce hypogammaglobulinaemia because after it is surgically removed the immune impairment persists, as in our patient who, after thymectomy, continued to be hypogammaglobulinaemic.

The deficient humoral immunity in GS is the main cause of the recurrent respiratory infections, with an overall spectrum of manifestations and pathogens similar to other hypogammaglobulinaemic conditions, such as CVID.[7] However, in GS opportunistic infections, such as mucocutaneous candidiasis (as seen in our patient), herpes zoster, *Pneumocystis carinii* pneumonia and recurrent herpes simplex virus infections develop more frequently than in CVID.[8] The occurrence of opportunistic infections in GS seems to be due to defects in cell-mediated immunity, which have been found in several patients.[9] In our patient, CD4+ T cells were reduced with an inverted CD4/CD8 ratio but he also had a mild neutropenia, which may also contribute to the recurrence of mycotic infections.

Several haematological disorders have been reported in GS, including pure red cell aplasia, pancytopenia, and autoimmune haemolytic anaemia.[5] The target in many of these disorders appears to be the stem cell committed to a haemopoietic lineage with loss of that lineage, even if the role of autoantibody-mediated mechanisms in the destruction of the lineage cannot be excluded.[9]

Finally, our case report confirms the importance of IVIG replacement in GS at appropriate doses in order to improve the control of infections and perhaps prevent the development of bronchiectasis,[10] which to date has not been identified in our patient.

Author information: Michela Di Renzo, Internal Medicine Physician; Anna L Pasqui, Cardiologist; Fulvio Bruni, Cardiologist, Department of Internal Medicine and Immunological Sciences; Luca Voltolini, Thoracic Surgeon, Department of Thoracic Surgery; Paola Rottoli, Respiratory Physician; Grazia Perali, Biologist, Department of Respiratory Diseases; Alberto Auteri, Immunologist, Department of Internal Medicine and Immunological Sciences, University of Siena, Siena, Italy

Correspondence: Dr Michela Di Renzo, Dipartimento di Medicina Interna e Scienze Immunologiche, Policlinico Le Scotte, 53100 Siena, Italy. Fax: +3 905 774 4114; email: cd28apo@hotmail.com

 © NZMA

References:

1. Good RA. Agammaglobulinaemia – a provocative experiment of nature. Bull Univ Minn. 1954;26:1–19.
2. Asherson GL, Webster ADB. Thymoma and immunodeficiency. In: Asherson GL, Webster ADB, edtiors. Diagnosis and treatment of immunodeficiency diseases. Oxford: Blackwell; 1980. p. 78–98.
3. Primary immunodeficiency diseases. Report of an IUIS Scientific Committee. International Union of Immunological Societies. Clin Exp Immunol. 1999;118(Suppl 1):1–28.
4. Masci AM, Palmieri G, Vitiello L, et al. Clonal expansion of CD8+ BV8 T lymphocytes in bone marrow characterizes thymoma-associated B lymphopenia. Blood. 2003;101:3106–8.
5. Mitchell EB, Platts-Mills TA, Pereira RS, et al. Acquired basophil and eosinophil deficiency in a patient with hypogammaglobulinaemia associated with thymoma. Clin Lab Haematol. 1983;5:253–7.
6. Asherson GL, Johnson S, Platts-Mills TA, Webster AD. Pathogenesis of hypogammaglobulinaemia with thymoma and late-onset hypogammaglobulinaemia. J Clin Pathol Suppl (R Coll Pathol). 1979;13:5–9.
7. Strober W, Chua K. Common variable immunodeficiency. Clin Rev Allergy Immunol. 2000;19:157–81.
8. Tarr PE, Sneller MC, Mechanic LJ, et al. Infections in patients with immunodeficiency with thymoma (Good syndrome). Report of 5 cases and review of the literature. Medicine (Baltimore) 2001;80:123–3.
9. Yip D, Rasko JE, Lee C, et al. Thymoma and agranulocytosis: two case reports and literature review. Br J Haematol. 1996;95:52–6.
10. Stadtmauer G, Cunningham-Rundles C. Outcome analysis and cost assessment in immunologic disorders. JAMA. 1997;278:2018–23.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

More lust for operation

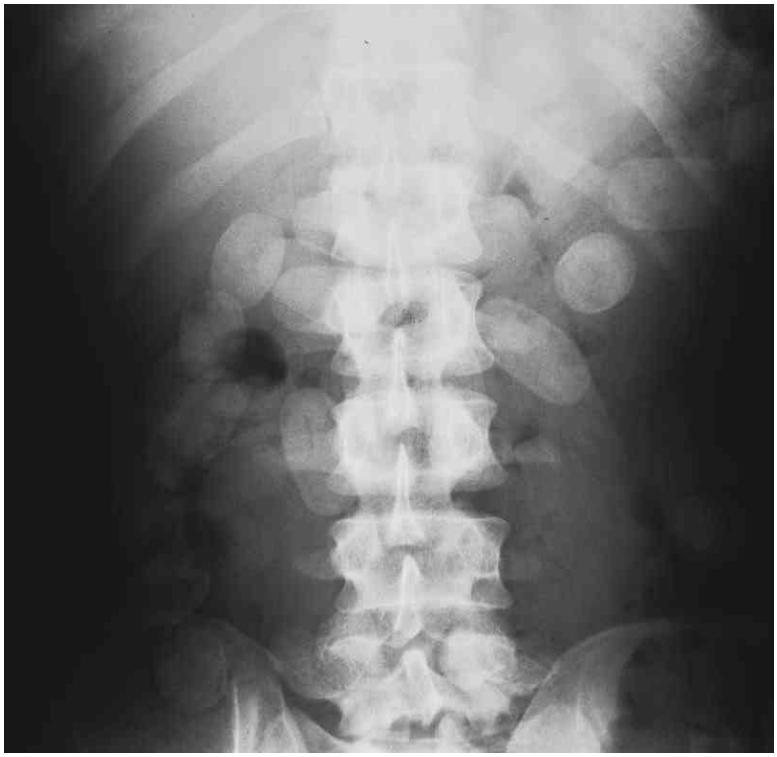
This extract is taken from an article by Dr Hatherley, Wanganui, published in the New Zealand Medical Journal 1903, Volume 3 (10), p247–54

The old school of surgeons, who regarded operative treatment as a last rather than as a first resource, seems to be slowly yet surely dying out. A later school is springing up in our midst who are extending the scope of operative procedure in every direction, and appear to be never quite happy unless they are using their one sovereign remedy for all the ills that flesh is heir to – the knife. The fact that with a careful antiseptic or aseptic technique the human body will survive a marvellous amount of mutilation has created a demand and a supply of operating surgeons who are ever on the *qui vive* for something to find which will in their opinion necessitate an operation. They seem to be guided by the principle that a diligent search may often disclose some slight lesion which can either be cut out or stitched up, rather than seek to discover some method of treatment which does not necessarily involve an operation. I use the word “lust” advisedly, because with some practitioners the desire to operate is so fierce as to be well-nigh insatiable. They have their own private hospitals, their own staff of nurses, and their own assistants; they studiously ignore the usual medical attendant, who, if consulted, might possibly be in favour of milder measures; and grave surgical operations are being performed almost daily without any of the safeguards which are strictly enforced in our public hospitals. I believe the rule in all our public hospitals is that no important operation should take place without a consultation being held, at which every member of the staff is entitled to assist. It may happen that the staff is divided in opinion as to the necessity of immediate operation, in which case the operation is usually deferred; and I can quote many instances in which the postponement of an operation has given the patients an opportunity of making an excellent recovery without it.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Bodypacker



The image above is an abdominal radiograph of a young woman recently apprehended in Bermuda and suspected of being a drug courier. The standard investigation of smugglers suspected of drug ingestion is a plain radiograph, as the air trapped in the receptacles (often condoms) gives a distinct appearance as shown in the figure above. Retrieval of the drugs is a less appealing procedure!

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Antibiotic development pipeline runs dry

For some time, infectious-disease experts worldwide have warned that just as 'super bugs' are becoming a greater threat, many pharmaceutical companies are curtailing their antibacterial research and development programmes, and, in some cases, pulling out of the market altogether.

The reason why drug companies are getting out of the business is not complex. It costs the same to develop an antibiotic as it does other drugs. But these drugs are given for 1–2 weeks compared to many drugs such as lipid-lowering agents, which are lifelong. Hence the returns are lower.

It is estimated that it takes about $900 million to bring a drug to market, and pharmaceutical companies are finding it more difficult to recoup their costs, says John Bartlett, chair of the Antimicrobial Availability Task Force of the IDSA (Infectious Disease Society of America). The high cost of drug development 'has had more of an effect on antibacterials, as compared to other anti-infective agents. There's been four new antiretroviral agents this year alone, but only two new antibiotics. Last year, there were none.' Right now, says Bartlett, there are about 400 drugs in the pipeline that are likely to be approved in the near future, but only five are antibacterial agents.

Lancet. 2003;362:1726–7

Health tourists?

In the United Kingdom, National Health Service patients with foreign-sounding names and accents may be required to produce passports, payslips and other records to show they are entitled to free treatment. The measures are necessary, ministers say, to prevent valuable resources being used by so-called health tourists.

That view is hotly contested by opposition parties, who say the Government is unable to justify its assertion that the NHS is spending hundreds of millions of pounds on patients not entitled to receive care. Doctors, too, have been infuriated by the proposed identity checks. The British Medical Association said it wanted to work with the Government to minimise fraud, but stated: 'It is not the role of doctors to be the agents of the state in policing eligibility for healthcare.'

Under the new rules free treatment will be withheld from visiting spouses and relatives of overseas residents in the UK. John Hutton, a junior health minister said: 'The NHS is there to provide free treatment for those who live here, not those who don't. It is a national health service, not an international service.'

Guardian Weekly, 8–14 January 2004

© NZMA

Because testicular cancer is highly curable there is a need to minimise long-term toxic effects of therapy without jeopardising effectiveness

Patients who present with cancer confined to the testis have a cure rate of almost 100% and even patients with metastatic disease can expect a cure rate of 80%. This success story, although gratifying, has highlighted important issues in the treatment of testicular cancer such as patient choice, long-term treatment side-effects, and quality of life. In a review, Jones and Vasey discuss current gold standards for management of the disease including surgical options for patients with early-stage tumours and appropriate chemotherapy protocols for patients with advanced disease.

Lancet Oncol. 2003;4:730–37, 738–47

Could CT scanning in infancy affect later learning?

Receiving low doses of ionising radiation to the head in infancy may impair the developing brain and affect intellectual development. Hall and colleagues studied 3094 men from Sweden who had received radiotherapy for cutaneous haemangioma before the age of 18 months.

They analysed military records reporting the men's intellectual capacity at age 18 or 19 and found that exposure to doses of radiation greater than 100 mGy, the equivalent of a computed tomography scan, was negatively correlated with high school attendance and learning ability assessed by cognitive tests. The authors call for re-evaluating the use of computed tomography for minor head injuries in infants.

BMJ. 2004;328:19–21

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Buteyko pseudoscience

Asthma is an important health problem in New Zealand, causing a great deal of morbidity and an economic burden estimated to be $825 million per year. As with many chronic illnesses, patients with asthma often use alternative therapies and it is important that these therapies are researched in high-quality trials, as with any other medicine or health intervention.

Although the intervention may be alternative, the study methodology and reporting should be of the standard required for its non-alternative counterparts. Unfortunately, the trial by McHugh et al on the Buteyko Breathing Technique published recently in the NZMJ (http://www.nzma.org.nz/journal/116-1187/710/)[1] falls below that required standard.

The main concern is that the design of this trial would inevitably lead to a positive result. The dose of inhaled steroid was reduced if the participants were using less reliever medication. It can be assumed that the Buteyko group were encouraged to do breathing exercises if they felt asthma symptoms and to reduce reliever medication use.[2] Therefore, it is not surprising that the Buteyko group used less reliever and were instructed to use less inhaled steroid.

There are numerous other concerns regarding the trial design and reporting, including the following:

- To state that medication dosage was reviewed and appropriate advice given is simply not good enough for a trial whose main endpoint is medication dose.
- Symptom score data and other indices of ventilatory function were collected but not reported.
- It is not clear if smokers were excluded.
- The pairing of subjects is not adequately explained.
- It is not stated from which group(s) the four participants left the study.
- The abstract states that there were no adverse events, yet five patients required prednisone for exacerbations.

For the only objective outcome, % predicted FEV_1, there was no difference between the two groups.

Given the inadequacies in the reporting and methodology of the study, the authors' conclusion that Buteyko is efficacious in asthma management is not justified. A well-designed study with objective outcome measures is needed.

Shaun Holt
Director
P3 Research, Wellington

Richard Beasley
Director
Medical Research Institute of New Zealand, Wellington

© NZMA

References:

1. McHugh P, Aitcheson F, Duncan B, Houghton F. Buteyko Breathing Technique for asthma: an effective intervention. NZ Med J. 2003;116(1187). URL: http://www.nzma.org.nz/journal/116-1187/710/
2. Buteyko Asthma Management web site. URL: http://www.buteyko.co.nz Accessed January 2004.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

The Green Prescription

Undoubtedly, there is a need to increase the physical activity of New Zealanders and a need for people with a knowledge of the pathophysiology of disease, the musculoskeletal system, and the psychological factors that affect a person's adherence to a programme to be involved. It was, therefore, pleasing to see the article written by Phil Handock and Carolyn Jenkins, published recently in the NZMJ (http://www.nzma.org.nz/journal/116-1187/713/).[1] Disappointingly, it failed to mention the role that physiotherapists play. Physical activity is at the core of physiotherapy.

Physiotherapy is a four-year degree course leading to an excellent understanding of biomechanics, physiology and neurophysiology. Physiotherapy research is also very focused on methods to promote adherence to an exercise regime, as we are aware it remains 'the greatest obstacle to effective physical activity'.[1]

The New Zealand Society of Physiotherapists (NZSP) continues to promote the role of physiotherapists in the prescription of exercise for injury prevention and for the promotion of fitness in many chronic diseases. This was highlighted by a recent article in Physiotherapy.[2]

To achieve this, NZSP works closely with various organisations (eg, Diabetes New Zealand, Osteoporosis New Zealand, and the Asthma and Respiratory Foundation of New Zealand) in promoting physical activity and educating the public on its importance as a means of reducing disease risk.

A major change for health professionals in New Zealand is the Health Practitioners Competence Assurance Act (HPCA), which was passed into law on 18 September 2003. Its main purpose is to protect the health and safety of the public by providing ways to ensure that health practitioners are competent and fit to practise their professions. The Physiotherapy Board of New Zealand is now working with physiotherapists in New Zealand to establish scopes of practice.

Preliminary work on establishing a generic scope of practice (the result of which is a consultation document that stakeholders have recently been invited to comment upon) focuses on the role physiotherapists play 'in the promotion, maintenance and restoration of health and in the diagnosis, prevention and treatment of health associated problems throughout the life span', and their ability 'to promote mobility, health and independence', 'rehabilitate after injury or illness' and 'maximise potential for activity'. This language – to reiterate – emphasises that activity is the core of physiotherapy.

A targeted exercise programme instigated by a physiotherapist as part of a rehabilitation programme involves a full assessment of the patient, including identification of specific areas of weakness, and of movements that cause pain. An individual programme can then be developed. As registered health professionals, physiotherapists do not need a referral to see patients. However, they work in close collaboration with GPs and other health professionals, and often with other physical-activity providers.

© NZMA

The benefits of a tailored, community-based exercise programme for the prevention of falls in the elderly has been well publicised following research at the Otago Medical School.[3] This research was sponsored by ACC, who have recently updated and published an information booklet following the regime shown to be effective in the Dunedin trials. This booklet will be distributed by health professionals to appropriate patients. NZSP is working closely with ACC and the programme developers to promote the implementation of this programme.

There is well-documented research showing the effectiveness of pulmonary rehabilitation programmes in patients with asthma and chronic obstructive respiratory disease, and physiotherapists are very involved in their implementation. These programmes typically involve strength and endurance training for the upper and lower body, plus counselling and education. They have been shown to result in an increase in walking distance, and an improvement in health-related quality of life.[4]

These are examples of the compelling evidence that individually tailored exercise programmes supervised by physiotherapists have a beneficial effect on pain, function, general wellbeing and quality of life.

It is important that the public is aware of the health professionals qualified in this area. We must all recognise one another's skills, and work together to combat one of the major health risks of the 21st century.

Janet Copeland
Research Liaison Officer
NZSP

References:

1. Handcock P, Jenkins C. The Green Prescription: a field of dreams? NZ Med J. 2003;116(1187). URL: http://www.nzma.org.nz/journal/116-1187/713/
2. Hussey J, Wilson F. Measurement of activity levels is an important part of physiotherapy assessment. Physiotherapy. 2003;89:585–593.
3. Campbell AJ, Robertson MC, Gardner MM, et al. Falls prevention over 2 years: a randomized controlled trial in women 80 years and older. Age Ageing. 1999;28:513–8.
4. Herbert RD, Maher CG, Moseley AM, Sherrington C. Effective physiotherapy. BMJ. 2001;323:788–90.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Chronic idiopathic singultus: is there life after cisapride?

It was a pleasure reading in the Journal the report by Porzio et al about their success in the treatment of hiccup using gabapentin (http://www.nzma.org.nz/journal/116-1182/605/).[1] Their experience with this structurally GABA-related substance is similar to ours: occasionally patients respond to it dramatically.[2,3]

Unfortunately, more often than not, therapy for chronic idiopathic singultus (CIS) is less straightforward, requiring the use of a combination of drugs. The efficacy of several drug combinations (cisapride, omeprazole and baclofen (COB) with or without gabapentin, or gabapentin alone) has been assessed in several studies.[4,5]

For practical purposes, idiopathic hiccup can be assumed to have its origin either in the viscera (gastrointestinal tract) or in the central nervous system. Cisapride and omeprazole – through facilitation of gastric emptying and reduction of gastric acid production, respectively – are thought to reduce an assumed afferent input from the periphery to a putative supraspinal hiccup center. Baclofen (a centrally acting GABA-B receptor agonist) and gabapentin (acting mainly via the alpha$_2$-delta subunit of the calcium channel) are thought to reduce excitability and depress reflex hiccup activity.

COB, a 'broadband therapy' for this condition, was considered by our group – until the withdrawal of cisapride from the market – to be the empirical therapy of choice, gabapentin being an excellent add-on drug or in some cases replacement drug for baclofen.

While, as always in polypharmacy, it is impossible to assess the exact role of a particular component of the drug combination used, it is fair to say that at least in some cases the selective serotonin 5-HT$_4$-receptor agonist cisapride had an important role. This has led to the search for a replacement gastrokinetic substance to be used, at least in those cases where delayed gastric emptying is felt to be contributory.

The best-known available alternative, the benzamide compound metoclopramide (Reglan), is a mixed dopamine receptor antagonist, 5-HT$_4$-receptor agonist, and cholinesterase inhibitor. It has a long tradition in the treatment of hiccups, going back to the late 1960s.[6] However, although adverse reactions are rare, a potential exists for extrapyramidal side-effects to occur, and therefore we prefer to avoid its long-term use. Similar concerns apply to itapride (Ganaton). Mosapride (Gasmotin) is also undesirable because, like cisapride, it prolongs the QT interval.

Two newer drugs deserve mention and consideration as potential gastrokinetic agents in hiccup patients.

Tegaserod (Zelnorm) is a 5-HT$_4$-receptor partial agonist, recently introduced for treatment of constipation in women with irritable bowel syndrome.[7] The substance might offer advantages similar to those of cisapride, without the danger of torsade de pointes. Interestingly enough, recent research has revealed that the breathing centre in the brain stem is (at least in part) under serotoninergic control via a subtype of 5-HT$_4$-receptors, opening the possibility that 5-HT$_4$ agonists might also influence hiccupping by activating the rhythm-generating respiratory neurons.[8]

© NZMA

The ‘afil’ class of selective phosphodiesterase inhibitors (sildenafil, vardenafil, and tadalafil), while certainly not needing any introduction as therapy for erectile dysfunction, have additional useful effects that are less known. Recently, a gastric prokinetic effect of sildenafil was reported, offering a rationale for its inclusion in the group of potential hiccup treatments.[9] Even if the gastrokinetic effect of ‘afils’ turns out to be irrelevant, a case report by Peleg and Peleg, entitled ‘Sexual intercourse as potential treatment for intractable hiccups’, gives reason for hope of a cure.[10]

We certainly look forward to case reports describing the effect (if any) of these types of drugs on chronic hiccup.

Georg A Petroianu
Department of Pharmacology and Therapeutics, UAE University
United Arab Emirates

References:

1. Porzio G, Aielli F, Narducci F, et al. Hiccup in patients with advanced cancer successfully treated with gabapentin: report of three cases. N Z Med J. 2003;116(1182). URL: http://www.nzma.org.nz/journal/116-1182/605/
2. Gee NS, Brown JP, Dissanayake VU, et al. The novel anticonvulsant drug, gabapentin (Neurontin), binds to the alpha2delta subunit of a calcium channel. J Biol Chem. 1996;271:5768–76.
3. Petroianu G, Hein G, Stegmeier-Petroianu A, et al. Gabapentin “add-on therapy” for idiopathic chronic hiccup (ICH). J Clin Gastroenterol. 2000;30:321–4.
4. Petroianu G, Hein G, Petroianu A, et al. ETICS Study: Empirical therapy of idiopathic chronic singultus. Z Gastroenterol. 1998;36:559–66.
5. Petroianu G, Hein G, Petroianu A, et al. Idiopathic chronic hiccup: combination therapy with cisapride, omeprazole, and baclofen. Clin Ther. 1997;19:1031–8.
6. Frosali L. On the treatment of intra-operative and postoperative hiccup with metoclopramide (Plasil). Acta Anaesthesiol. 1968;19:1245–56.
7. Appel-Dingemanse S. Clinical pharmacokinetics of tegaserod, a serotonin 5-HT(4) receptor partial agonist with promotile activity. Clin Pharmacokinet. 2002;41:1021–42.
8. Manzke T, Guenther U, Ponimaskin EG, et al. 5-HT4(a) receptors avert opioid-induced breathing depression without loss of analgesia. Science. 2003;301:226–9.
9. Bianco A, Pitocco D, Valenza V, et al. Effect of sildenafil on diabetic gastropathy. Diabetes Care. 2002;25:1888–9.
10. Peleg R, Peleg A. Case report: sexual intercourse as potential treatment for intractable hiccups. Can Fam Physician. 2000;46:1631–2.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Medical discipline – professional misconduct

Charge: The Director of Proceedings charged that Dr Ian Lindsay Breeze was guilty of disgraceful conduct or in the alternative professional misconduct.

The particulars of the charge were as follows:

1. On or about 16 December 1999, Dr Breeze failed to ensure the adequate preparation of the patient's bowel prior to surgery in that he failed to ensure that adequate corrective bowel preparation agents were administered to the patient on becoming aware that the patient had broken his fast; and/or
2. He failed to adequately assess the patient postoperatively; before 1200 hrs on 18 December 1999; and/or
3. He failed to adequately and appropriately respond to the patient's clinical presentation in that he failed to re-operate, at any time after 0700 hrs and between 2400 hrs on 17 December 1999; and/or
4. Between 1100 hrs on 17 December 1900 and 1200 hrs on 18 December 1999 he failed to consult with and/or transfer care of the patient to an appropriately qualified specialist surgeon in a timely manner; and/or
5. He failed to adequately, and in a timely fashion, document in the clinical notes [his] operative and/or postoperative care in relation to the patient.

Background: The patient was a 65-year-old with diet-controlled diabetes. The patient was referred to Dr Breeze with a history of passing blood with his faeces. Dr Breeze saw the patient on 6 December 1999 when it was revealed the patient had a polypoidal cancer of the rectum.

The patient had a colonoscopy on 15 December prior to scheduled bowel surgery on 16 December, which did not reveal any other tumour. It would appear that the nurse discharging the patient on 15 December advised the patient to have a sandwich before leaving the hospital and a light meal that evening.

When the patient arrived at hospital on 16 December he told the admitting nurse that he had eaten a light meal the previous evening. The nurse telephoned Dr Breeze's rooms but was unable to speak to him directly. The message relayed to the nurse from Dr Breeze was that the patient should receive a Microlax enema. A Microlax enema only cleans the rectum, not the colon or caecum. The fact that only a Microlax enema was administered is clearly documented in the nursing notes. Dr Breeze maintained that he instructed that the patient be given a Fleet enema as it is a fast-acting colonic laxative.

The procedure followed by Dr Breeze involved a lower anterior resection and the formation of a loop ileostomy. The rectum was divided below and above the tumour and rejoined. The joined section of the rectum is called an anastomosis. It is critical that the surgeon ensure the anastamosis is intact. If the anastamosis is not complete and leakage occurs there is a real risk of infection and complications. Part of the technique involves a simultaneous slicing of a small portion of the ends of the rectum

© NZMA

that are joined. These cut portions are called ‘donuts’. Each donut is about the size of a 1 cent coin and the surgeon should check the donuts to ensure they are intact. A donut that is not complete indicates that the anastamosis may not be intact and may leak.

Dr Cooke, who was the anaethetist for the surgery, was concerned that one of the donuts was equivocal. Dr Breeze was standing approximately four feet from Dr Cooke. Dr Breeze suggested he could see the donuts and was satisfied they were intact. Dr Breeze also said he examined the donuts at the end of the operation when they were in a container. His handwritten operation note was extremely brief and comprised just ten words.

The patient was transferred to the ward at 1900 hrs. The duty nurse telephoned Dr Breeze at 2210 hrs concerning pain in the left side of the patient’s abdomen and shivering. Dr Breeze gave no new instructions.

Dr Breeze visited the patient at 0700 hrs the next day before going to Tauranga Hospital to perform two operations. There is no record of any clinical observations or assessments made by Dr Breeze at 0700 hrs. The records do however show Dr Breeze prescribed some medication including further antibiotics (Flagyl and Gentamicin).

At approximately 1115 hrs Dr Breeze was telephoned by a nurse concerning the patient’s laboratory blood test results. The laboratory reported that the blood tests showed a very toxic-looking picture which needed triple antibiotic cover. It showed the white blood cell count had fallen dramatically and indicated neutrophil toxic changes. Dr Breeze telephoned back at 1150 hrs when he was made aware of the lab results and the patient’s low blood pressure. He told the nurse that the patient’s bowel preparation was not very good so it was likely there was some contamination.

Dr Cooke visited the patient at 1250 hrs and was told there could be faecal material in the pelvic drain. Dr Cooke assessed the patient’s condition and determined an immediate transfer to the ICU at Tauranga Hospital was necessary.

Dr Cooke telephoned Dr Breeze at approximately 1330 hrs. Dr Cooke stressed to Dr Breeze that his patient was very sick. Dr Breeze explained he wished to follow a ‘conservative’ approach in treating the patient, that is to say, he did not want to re-operate but chose instead to deal with the infection by drainage and antibiotics. The patient was transferred to the Tauranga Hospital ICU at approximately 1500 hrs on 17 December.

After the patient was admitted, a doctor at Tauranga Hospital, Dr Jackson, telephoned Dr Breeze who by this stage was approximately 10 minutes’ drive from Tauranga Hospital. Dr Jackson told the Tribunal that when he spoke to Dr Breeze he told Dr Breeze of his assessment and diagnosis. Dr Jackson was in no doubt Dr Breeze was made aware of the patient’s deteriorating condition and in particular:

- The profound septic shock that would necessitate inotropic support;
- That respiratory failure would likely require artificial ventilation in the near future;
- The depressed white blood cell count; and
- The extreme (500 mls) faecal material that had been drained.

 © NZMA

Dr Jackson said he asked Dr Breeze about an exploratory laparotomy and washout but was told this was definitely not indicated.

During the course of this telephone conversation Dr Breeze indicated he would visit the patient the next day and that he would contact the surgical registrar on call who in turn would be expected to contact Dr Breeze if there was any deterioration in the patient's condition.

Dr Breeze believed he telephoned the acute general surgical registrar on call, Dr Martin, and that together they planned to trial conservative management. Dr Martin gave evidence in which he was adamant Dr Breeze did not contact him. Dr Martin relied on the fact that there is no record in the clinical notes of his speaking to Dr Breeze and he is certain that he would have made an entry in the notes if he had been asked to participate in the patient's management.

Dr Breeze went home soon after 1700 hrs and he went out to an end-of-year function that evening.

During the night of 17 December the patient's condition continued to deteriorate. At 0245 hrs on 18 December the patient required resuscitation but his condition was so bad that Dr Jackson decided no further resuscitation attempts would be made.

Dr Breeze visited the patient on 18 December but by this time the patient's fate was sealed. The patient's life was maintained in the Tauranga ICU until the morning of 21 December when he passed away.

A post mortem was carried out on 22 December. The pathologist's report noted that:

> *'In the region of the rectum, there is an 18 mm defect, which has surrounding staples. There are fibrous adhesions in the lower abdominal cavity. Approximately 150 mls of brown stained fluid and admixed faecal material are present in the abdominal cavity.'*

Finding: The Tribunal found Dr Breeze guilty of professional misconduct.

The Tribunal was not satisfied Particular 1 of the charge was established. The Tribunal was satisfied that Dr Breeze was justified in asking that the patient be given a Fleet enema after he learned his patient had broken his fast. The Tribunal was concerned Dr Breeze appeared not to have appreciated the patient was in fact administered a Microlax enema. It would appear Dr Breeze did not read or properly read the nurses' notes before operating on his patient. The Tribunal was of the view that this oversight did not in itself justify a disciplinary finding against Dr Breeze.

The Tribunal was very satisfied Particular 2 was established and that his failing in this regard amounted to professional misconduct. The Tribunal considered Dr Breeze should have personally attended upon and assessed the patient well before he visited his patient on the morning of 18 December. The Tribunal considered Dr Breeze had a responsibility to personally assess and monitor his patient's progress, particularly as Dr Breeze had resolved to pursue a conservative course of management. The Tribunal found Dr Breeze should have attended upon, examined and carefully assessed his patient as soon as his operation commitments finished on 17 December 1999, and was very concerned he gave priority to an end-of-year function over attending to his critically ill patient. The Tribunal did not believe Dr Breeze spoke to the surgical

 © NZMA

registrar on call on 17 December. However, even if he did, the Tribunal considered Dr Breeze needed to fully assess and monitor his patient's condition.

The Tribunal believed Dr Breeze's failure to personally attend and assess his patient for at least 26 hours from 0700 hrs on 17 December was a serious abrogation of his duties.

When considering Particular 3 the Tribunal was in no doubt that Dr Breeze failed to adequately and appropriately respond to the patient's clinical presentation between 0700 hrs and 2400 hrs on 17 December 1999. However, the Tribunal considered Particular 3 was based on the belief that re-operation was mandatory between 0700 hrs and 2400 hrs on 17 December. Whilst the Tribunal strongly suspected that re-operation was necessary, the Tribunal also accepted that ultimately the decision to re-operate or not had to be a clinical judgment and that accordingly the Tribunal should not conclude re-operation was mandatory. It considered Dr Breeze's critical error was he failed to give adequate consideration to re-operating, not that he failed to re-operate. In these circumstances the Tribunal could not make an adverse finding against Dr Breeze in relation to Particular 3.

When considering Particular 4, the Tribunal was satisfied Dr Breeze failed to consult with, and/or transfer the care of the patient to an appropriately qualified specialist surgeon. The Tribunal had no hesitation in concluding that if Dr Breeze was unable to attend to and personally assess his patient then he had a duty to ensure his care was transferred to another consultant surgeon.

The Tribunal was unanimous in its finding that the Director of Proceedings had established Dr Breeze failed to consult with and/or transfer the care of the patient to an appropriately qualified specialist when he should have done so. However, only a majority of the Tribunal considered that Dr Breeze's breaches of duty as established in Particular 4 justified a disciplinary finding against him. The majority was satisfied that in relation to Particular 4 Dr Breeze was guilty of professional misconduct.

The Tribunal was unanimously of the view that Particular 5 had been established. The Tribunal considered Dr Breeze's records were grossly inadequate. It considered Dr Breeze's lack of professionalism in this regard justified a finding of professional misconduct.

The Tribunal carefully considered whether the cumulative effect of its findings in relation to Particulars 2, 4 and 5 constituted disgraceful conduct in a professional respect. It concluded that Dr Breeze's shortcomings, even when viewed cumulatively, fell short of disgraceful conduct in a professional respect.

Penalty: The Tribunal ordered Dr Breeze pay a fine of $12 500 and costs of $37 825.94. It further ordered publication of the Tribunal's findings in the New Zealand Medical Journal.

The full decisions relating to the case can be found on the Tribunal web site at www.mpdt.org.nz Reference No: 03/99D.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Medical discipline – not guilty

Charge: The Director of Proceedings charged the Doctor with professional misconduct. The particulars of the charge were as follows:

1. On or about 13 November 1998, or at any time after that, the Doctor failed to adequately inform the patient of the possible consequences for her baby were her uterus to rupture during trial of labour/scar; and/or
2. On 8 June 1999, between 0800 hours and 0900 hours, or thereabouts, as on-call xx for the delivery suite at xx Hospital, the Doctor failed to:

   (a) adequately assess the patient; or

   (b) ensure that the patient was adequately assessed by a medical practitioner.

Background: In November 1998, when the patient was 12 weeks pregnant she consulted her general practitioner about a 'trial of labour' for her third pregnancy. The patient wanted to know if she would be able to deliver her baby vaginally as she had had a Caesarean section in March 1997. Her general practitioner referred the patient to the Doctor.

The patient met the Doctor on 13 November 1998. The Doctor explained that because the patient had had a Caesarean section there was a risk that her uterus could rupture. The patient was certain the Doctor did not tell her about the potential consequences of a ruptured uterus for her baby. This was a crucial issue during the case. The Doctor was adamant that he did explain to the patient the risks for her baby if the patient's uterus ruptured. After meeting with the Doctor the patient decided to proceed with a 'trial of labour'.

The patient chose two lead maternity carers (LMCs). For her pregnancy, the patient chose to be managed by Dr A, a general practitioner, and during the delivery of her baby she chose to be managed by Mrs B, a midwife.

On 20 May 1999, when the patient was 38½ weeks pregnant she had an antenatal consultation with Dr A. During that appointment Dr A told the patient that the baby had not descended into the pelvis. The patient was subsequently advised by Dr A, that Dr A and the Doctor had discussed her case and that the Doctor had agreed to a trial of labour. When the Doctor gave his evidence he explained that the circumstances under which this telephone conference took place meant that the Doctor would not necessarily connect this patient with previous or subsequent consultations.

The patient started to experience contractions on 7 June 1999 at 2330 hours. At 0020 hours on 8 June 1999 the patient and her husband went to the delivery suite at the Hospital where they were met by Mrs B and another hospital midwife.

The patient had an epidural for pain relief at 0316 hours and at 0330 hours Mrs B ruptured the patient's membranes. At 0545 hours the patient's cervix was 5–6 cm dilated and the baby's head was at station –2 cm. At 0730 hours Mrs B advised Dr B, the obstetrician on call, of the patient's progress.

© NZMA

The Respondent Doctor started duty at approximately 0800 hours. He was certain he visited the patient at about 0800 hours while she was asleep. The patient was equally certain the Doctor did not see her. There is a record of Mrs B contacting the Doctor at 0810 hours. The Doctor explained that he used this telephone conversation to request a vaginal examination by Mrs B.

Dr A visited the patient at about 0830 hours. Whilst Dr A was visiting the patient Mrs B performed the vaginal examination. The patient's cervix was 5–6 cm dilated and the baby's head was still at station –2. The notes also record a telephone conversation between Mrs B and the Doctor at 0830 hours during which the Doctor approved the administration of syntocinon to augment the labour process. Syntocinon was commenced at 0852 hours.

Mrs B reassessed the cervix at 1030 hours when it was noted the cervix was still 5–6 cm dilated. The augmentation continued and at 1300 hours Mrs B noted the patient's cervix was fully dilated. The baby's head was however still at station –2. Mrs B increased the syntocinon and topped up the epidural. At about 1430 hours Dr A returned. A vaginal examination performed at about that time showed the baby's head was still at station –2. Soon thereafter (between 1435 and 1445) the Doctor was told of the lack of progress and asked to attend.

At 1455 hours the patient suffered severe abdominal pain. The fetal heart monitor recorded sudden fetal bradycardia. The Doctor was summoned urgently. He promptly arrived and expedited the Caesarean section. Baby L was delivered at 1518 hours and was found to have an Apgar score of 0 at 1, 5 and 10 minutes. The patient had suffered a significant rupture of her uterus displacing the baby into the abdominal cavity.

The baby was flown to another hospital on 10 June. He was unable to survive without artificial support and died on 10 June.

Finding: The Tribunal found the Doctor not guilty of professional misconduct.

The Tribunal was satisfied Particular 1 was not established. It was confident that when the Doctor met the patient on 13 November 1998 he explained to her that there was a remote possibility her baby could be injured or even die if the patient's uterus were to rupture during a trial of labour.

The Doctor said that he told the patient the risk of fetal death following uterine rupture was 1:10 000. The patient also referred to the Doctor talking about a 1:10 000 risk. However, the patient thought the Doctor was referring to the risk of uterine rupture, not the risk of death to her baby. The Tribunal was told by the Doctor that the figure 1:10 000 is the figure he always mentions and relies upon when explaining the risk of fetal death in the event of a uterine rupture during a trial of labour. The Doctor advised the Tribunal that the risk of uterine rupture is reported in the literature to be 0.5–1% in pregnant women.

Both the patient and the Doctor recalled the Doctor referring to a 1:10 000 risk. The Tribunal was satisfied that when the Doctor referred to that statistic he was referring to the risk of fetal death following uterine rupture. In addition, as the Doctor had had the misfortune to have experienced this type of tragic outcome on two previous occasions, the Tribunal considered it unlikely the Doctor would overlook warning a woman of the risk of fetal death following uterine rupture.

© NZMA

The Tribunal was satisfied Particular 2 was not established. Mrs B was the LMC from the time the patient arrived at hospital until the Doctor took over her care for the purposes of performing the Caesarean section. Mrs B became the LMC at approximately 0020 hours on 8 June and remained the LMC until approximately 1445 hours that day. At no time prior to 1445 hours did Mrs B seek to transfer the patient's care to the secondary services.

The Tribunal was satisfied when the Doctor examined the patient's notes at approximately 0800 hours on 8 June he did not take over the patient's care. The Doctor was simply familiarising himself with cases in the maternity suite who might require specialist care and assistance. Similarly, when the Doctor agreed to the patient receiving syntocinon in accordance with the hospital guidelines, he was not being asked by Mrs B to accept responsibility for the patient's care. The Tribunal considered the Doctor did not have a professional responsibility to personally examine the patient, or arrange another doctor to examine her between 0800 and 0900 hours.

The Tribunal believed that it may have been appropriate for the Doctor to have insisted that he be allowed to assess the patient's progress after syntocinon had commenced. In the Tribunal's view the Doctor should have arranged, as part of his 0830 telephone consultation, a formal follow up approximately two hours after the introduction of syntocinon. This would have allowed him to assess the effect of syntocinon and to ensure satisfactory progress was being achieved. Ideally this follow up should have been by a personal assessment and examination.

The Tribunal considered that the second particular of the charge could not be stretched to encompass the Doctor's failure to personally assess the patient approximately two hours after syntocinon had commenced. The Tribunal wished to emphasise that in making these observations it was not suggesting that it would have necessarily found the Doctor guilty of professional misconduct if the second particular of the charge had been framed in broader terms. The Tribunal also stressed that even if there had been an examination of the patient during the course of the morning by the Doctor the ultimate outcome may not have been any different.

The Tribunal directed publication of a summary of the Tribunal's findings in the New Zealand Medical Journal.

The full decisions relating to the case can be found on the Tribunal web site at www.mpdt.org.nz Reference No: 03/104D.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Medical discipline – charge struck out

Charge: On 26 May 2003, a Complaints Assessment Committee charged the Doctor with disgraceful conduct in a professional respect. The particulars of the charge alleged that on 6 January 1983 the Doctor:

1. …conducted a vaginal examination of the complainant…without wearing gloves; and
2. In the course of the examination the Doctor inappropriately manually stimulated the complainant's clitoris and then later in the examination when placing the speculum in the vagina slid the instrument in and out of the vagina and manually stimulated her clitoris, without any clinical or medical justification; and
3. In the course of the examination the Doctor inappropriately asked the complainant if what he was doing 'felt good' or words to that effect.

An application was filed on behalf of the Doctor to have the charge struck out or stayed. The application was opposed by the CAC.

Finding: The Tribunal ordered that the first particular be struck out on the grounds of general prejudice, and that the second and third particulars be struck out on the grounds of specific prejudice.

Specific prejudice

In the Tribunal's view there was one crucial matter which strongly suggested the Doctor would suffer specific prejudice in trying to defend the second and third particulars of the charge. The event which raised a serious issue of specific prejudice was the fact the complainant's mother was dead and could no longer provide crucial evidence about key matters.

The Tribunal considered the fact the Doctor and four other witnesses may have difficulty in recalling events said to have occurred 20 years ago were not factors which satisfied the criteria for specific prejudice when considering an interlocutory application to strike out/stay the charge. Before the Tribunal could be satisfied that the witnesses in question do not have an ability to recall specific matters the witnesses would need to give evidence to the Tribunal. The Tribunal would then be able to assess the extent (if any) to which the witnesses' memory have been impaired. The Tribunal considered the recollection of the complainant's mother of what the complainant said immediately after the consultation on 6 January 1983, and her understanding of the discussions held with another doctor on 7 January 1983 would be crucial in assisting the Tribunal determining the second and third particulars of the charge. Similarly, the Tribunal considered her evidence, if it could have been obtained, may have been very significant in assisting the Doctor defend the charge. As she was no longer alive, the Tribunal was satisfied there was a serious risk of prejudice to the Doctor.

General prejudice

© NZMA

The Tribunal believed that the delays on the part of the complainant created general prejudice in relation to the Doctor's ability to defend the first particular of the charge.

The first particular was not struck out by the Tribunal on the grounds of specific prejudice because there was no suggestion the complainant's mother could have provided any assistance in determining whether or not the Doctor conducted the vaginal examination on 6 January 1983 without wearing gloves.

In striking out the first particular of the charge on the grounds of general prejudice the Tribunal was conscious that it should be extremely cautious before invoking its jurisdiction to strike out a charge on this ground. The reasons why the Tribunal invoked its jurisdiction to strike out the first particular of the charge on the grounds of general prejudice were:

- The complainant's long delay in bringing this issue to the attention of the appropriate authorities created serious difficulties for the Doctor in defending the charge. The Tribunal considered it would be almost impossible for the Doctor to recall whether he did or did not wear gloves during the examination he performed on 6 January 1983.
- It would appear questions first arose about whether or not the Doctor wore gloves when conducting the vaginal examination when the complainant was interviewed by the CAC. The complainant did not appear to have specifically raised this issue in her letter of complaint. While this fact was not in itself determinative, it was a consideration the Tribunal took into account in assessing the overall prejudice which the Doctor would suffer.
- Whilst the Tribunal considered it was very important that medical practitioners wear gloves when conducting vaginal examinations, if the Doctor did not do so on this occasion then his omission would definitely not constitute disgraceful conduct in a professional respect.

Reasons for delay

The Tribunal believed it important to convey to the complainant that it understood the reasons why she delayed complaining to the Police and the Medical Council. The Tribunal sympathised with the complainant and appreciated that it is not an easy matter for any woman to raise issues of alleged sexual abuse. The Tribunal wished to make it clear the fact the charge was struck out should not be construed as a criticism of the complainant. The charge was struck out without the Tribunal reaching any conclusions about the facts.

The full decisions relating to the case can be found on the Tribunal web site at www.mpdt.org.nz Reference No: 03/107C.

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Alister Douglas George Bowron

Alister was born in New Brighton, Christchurch in 1923 and died in Tahunanui, Nelson, on 10 July 2002.

He was educated at Fendalton Primary School, Christchurch Boys High School and the Teachers Training College (briefly) before joining the RNZAF in World War II and serving nearly four years in Fiji, Guardalcanal and the New Hebrides. He was seconded to the US Marine Intelligence and was frequently under fire.

He returned to New Zealand and entered the Otago Medical School in 1946, graduating in 1952. He served as a house officer at Christchurch, Greymouth and Hokitika hospitals and he became the Special Area Medical Practitioner at Murchison in 1955. He was to remain until 1970.

He married Lindsay Brown, a registered general and maternity nurse, in 1960.

The Murchison Special Area was a large, sparsely populated area ranging from Glenhope and Nelson Lakes National Park to the north and east, Lyell in the Buller Gorge to the west, and Warwick Junction, Maruia, to the South. It was an hour's drive to the boundaries in any direction on rough, winding, unsealed roads. If a patient asked for a visit Alister always responded, taking medicines and suture material.

The manual telephone exchange could always find the doctor! Initially it closed from midnight to 6 am, connecting the doctor and hospital directly for that period, but eventually progress had the exchange working 24 hours, so no respite from calls – not that there were many. An answer service was almost necessary with the advent of the automatic exchange – the friendly operators were sorely missed. Such is progress! The population still had a wartime mentality – you only drove to town once a month and could not be expected to leave home if you or your child was sick.

For periods there was no chemist in the are so Alister did the dispensing of medicines. Following the Inangahua Earthquake a disastrous and lethal concoction of numerous chemicals, medicines, homebrew and broken glass covered the floor of the surgery.

The nearest colleagues were at Reefton or Wakefield, three hours away, with the base hospital at Nelson, four hours by ambulance. The two Wakefield doctors, John Davies and Ted Bassett, were called upon occasionally to cover as locums were very sparse. They would drive the fifty miles to see patients or deliver the unexpected baby at all hours, returning to cope with their own busy practices – assistance that was very much appreciated.

The Cottage Hospital had four beds for maternity patients and an outpatient theatre/room where many accident victims were attended, with the assistance of the one registered nurse–midwife, Sister Allan. She had coped during the war years when there was often no doctor in the area, so was a great support.

© NZMA

Wild pigs and deer abounded and Alister joined with RSA mates in many a hunt. He was frequently required to suture pig dogs, there being no vet available then. A bach at Lake Roroiti provided brief respites from the strain of being on call all day, every day. Only 24 hours away was allowed ‘without the express consent of the Minister’!

In 1971 the family moved to Nelson and set up a general practice with a large maternity content. Alister was the second police surgeon, sharing the work with Humphrey Belton and on call on alternate nights and weekends. In 1975 the family purchased a cottage at Tennyson Inlet, Pelorus Sound, and most free weekends were spent there. Alister retired in 1985.

His interests were centred round his home and family, a productive orchard and vegetable garden. He grew grapes and hops, made passable wine and beer, and latterly grew and pickled gherkins. He enjoyed experimenting with tomatoes and heritage vegetables.

While in Murchison he joined the Masonic Lodge and on moving to Nelson occupied senior posts in the organisation.

Alister is survived by his wife Lindsay, son Greg, daughter Nicki, and five grandchildren.

We are grateful to Lindsay Bowron for this obituary

 © NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

The Hawke's Bay Medical Research Foundation Inc: Medical Research and Study Grants

Applicants seeking funding for medical research or postgraduate study grants are invited to apply to this Foundation.

Information regarding criteria, together with the application form, can be obtained from the Secretary, PO Box 596, Napier.

Applications need to be in the Secretary's office by 31 March 2004.

Research or study with an emphasis on Hawke's Bay will receive some preference.

For further information, telephone 06 879 9199 day/evening or visit our web site: www.hawkesbaymedicalresearch.org.nz

J M Baxter
(Secretary)

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Multiple sclerosis: immunology, pathology, and pathophysiology

Robert M Henderson (ed). Published by Demos Medical Publishing, Inc, 2003. ISBN 1-888799-62-5. Contains 256 pages. Price US$89.95

This is a multi-authored textbook, which covers an extremely complex topic with a style that is both comprehensive and accessible. The contents are divided into four sections: (1) morphologic substrates of demyelination; (2) experimental aspects of demyelination; (3) cytokines, chemokines and interferons; and (4) pathology. The sections each comprise 3–6 short chapters. There is a remarkable consistency of format and style throughout the book. Each chapter is presented in a style reminiscent of a journal review article and includes the very welcome feature of a clearly presented ‘conclusion’. The chapters are extremely well referenced and complemented with black-and-white reproductions of exceptional quality of photomicrographs, and clear and helpful diagrams and tables. The glossy paper, printing and binding are all first-rate.

The book does have some limitations. Within such a short volume the Editor was not able to address some important areas of current MS research, such as genetics and epidemiology. Like all textbooks summarising a rapidly evolving field, it will become outdated relatively quickly. However, this book does succeed extremely well in its aim to provide an overview of the basic sciences as they relate to MS. It should be considered essential reading for any postgraduate student or neuroscientist joining one of the labs in New Zealand that are pursuing MS research.

John N Fink
Consultant Neurologist/Senior Lecturer in Medicine
Christchurch School of Medicine and Health Sciences

© NZMA

THE NEW ZEALAND MEDICAL JOURNAL
Vol 117 No 1188 ISSN 1175 8716

Life on cripple creek: essays on living with multiple sclerosis

Dean Kramer. Published by Demos Medical Publishing, Inc, 2003. ISBN 1-888799-68-4. Contains 160 pages. Price US$18.95

Dean Kramer is a monthly columnist for *MS World, Inc* and this book is her first collection of these essays. Her essays have an entertaining, light-hearted and humorous style reminiscent of many syndicated columnists (such as Bill Bryson), complete with the obligatory ironic twist in the final sentence. They highlight the day-to-day challenges that face a person living with MS as she manages to laugh a little at her failings and celebrate each of her small victories.

Many of the issues she addresses will be common to many people with chronic disabilities and are not challenges unique to people with MS. These include dealing with the prejudices of the able-bodied community, and negotiating one's way through medical systems. Her desire to shun sterile 'political correctness' in favour of simple, matter-of-fact observation is reflected in the book's title and her willingness to refer to herself as 'crippled'. She takes the same unembarrassed approach to other topics, including her own symptoms of cognitive impairment – too often a taboo subject.

Ms Kramer's experience of life is particularly well encapsulated in her essay 'Chicken Little and the Terminator', which describes a somewhat 'schizophrenic' existence that can find her one day inconsolably anxious about the significance of the smallest intensification of her symptoms, but another day portraying the hero capable of anything despite adversity. Between these two extremes, she tries to settle on the character of the Little Engine that 'sometimes could and other times could not'.

Although written by someone living in rural Pennsylvania, USA, this book would be appreciated by a New Zealand readership wanting an entertaining but thoughtful insight into life with a disability – particularly a readership that already owned all of Bill Bryson's books on travel.

John N Fink
Consultant Neurologist/Senior Lecturer in Medicine
Christchurch School of Medicine and Health Sciences

© NZMA