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This Issue in the Journal

Assessing Māori/non-Māori differences in cardiovascular disease risk and risk management in routine primary care practice using web-based clinical decision support: (PREDICT CVD-2)
T Riddell, R Jackson, S Wells, J Broad, L Bannink

Current New Zealand guidelines recommend cardiovascular disease (CVD) risk assessment be conducted for men over the age of 45 years, and women over the age of 55 years. CVD risk assessment for Māori is recommended a decade earlier—i.e. at ages 35 years for Māori men, and 45 years for Māori women. A computerised decision support programme, PREDICT, has been developed to assist general practitioners and practice nurses assess and manage CVD risk in these target groups. This paper describes the CVD risk factor status and risk management of Māori patients compared to non-Māori patients (European and other, Pacific, Indian, and other Asian) who were assessed using this tool in a large Auckland primary healthcare organisation. It demonstrates that PREDICT can be used to systematically generate CVD risk burden and risk management data for Māori and non-Māori populations in routine clinical practice to inform health services and improve care.

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R Harris, B Robson, E Curtis, G Purdie, D Cormack, P Reid

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C Hanger, V Fletcher, J Fink, A Sidwell, A Roche

Stroke units save lives, reduce dependency, and increase the chance of returning home. A 15-bed Acute Stroke Unit (ASU) was opened at Christchurch Hospital to complement an established Stroke Rehabilitation Unit (SRU) at the Princess Margaret Hospital. The aim of this study was to address whether patient care was improved with the establishment of the ASU. A before and after design was utilised to audit the processes of care (PoC) using an internationally validated stroke audit tool. 648 patients were admitted to the ASU in the first year. A subset of these patients were audited. The "after" cohort had more severe strokes (greater incontinence at 1 week and worse level of consciousness). Despite this length of stay, domicile on discharge and mortality outcomes were similar for the two cohorts. Processes of care improved in the "after" cohort in 27 of the 43 domains audited. Adding an ASU to complement an existing SRU can give major improvements in PoC across many different facets of stroke care. We believe this is one step closer to both the ideals of an overall coordinated stroke service and better overall care for patients with stroke.

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Monoamine oxidase, addiction, and the "warrior" gene hypothesis

Rod Lea, Geoffrey Chambers

In August 2006, news broke that a "warrior" gene was linked to risk-taking, aggression, and criminality in Māori. The story sparked widespread controversy in New Zealand—with journalists, politicians, academics, scientists, and the general community all scrambling to publicly express their views on the matter. However, much of the controversy was unjustified because it stemmed from a combination of misquotes and misunderstandings printed in the original article released by the Australian Press Association.

Despite our sincere efforts to set the story straight through subsequent high-profile media interviews, the critical commentary continues in this issue of the *Journal*. We therefore welcome this opportunity to present the scientific rationale behind our monoamine oxidase gene research—including our findings to date and the relevance to medicine, ethics, and Māori.

The monoamine oxidase gene and behavioural traits

Monoamine oxidases (MAOs) are enzymes responsible for breaking down the neurotransmitters—serotonin, dopamine, and adrenalin—and are therefore capable of affecting mood. Indeed, MAO inhibitors (e.g. moclobemide) can effectively treat symptoms of depression and tobacco dependence. The activity of MAO enzymes can vary among individuals and is influenced by inherited genetic factors.[1] Understanding the genetic variability of MAO activity and the linkage to drug response traits should assist in the design of more effective treatment options for certain clinical disorders.

The MAO genes are located on the X chromosome, thus males inherit only a single maternal copy. In 1997, Sabol et al reported that the MAO-A subtype contains a 30bp repeat polymorphism (MAO-A30bp-rpt) that is associated with transcriptional regulation (i.e. gene function).[2] Hundreds of epidemiological studies of the MAO-A30bp-rpt variant have since been conducted and associations reported with psychiatric disorders including depression, anxiety, and addiction (e.g. tobacco dependence and alcoholism). Studies have also implicated the 3-repeat allele of MAO-A30bp-rpt, postulated to correspond to lower MAO-A activity and higher dopamine levels, with risk-taking[3] and aggressive behaviour traits (see Merriman and Cameron's article—*Risk-taking: behind the warrior gene story*—http://www.nzma.org.nz/journal/120-1250/2440). For the latter reason, Gibbons (2004) dubbed it a "warrior" gene.[4]

Since most neuropsychiatric and behavioural conditions are aetiologically complex (or multifactorial) it is not surprising that some of the reported associations to MAO-A30bp-rpt are significantly modified when considered in combination with non-genetic (environmental) factors.

In this issue of the *Journal*, Merriman and Cameron review the topic of MAO gene-by-environment interactions with relevance to aggressive behaviour. We note, however, that this diverges from our research agenda, which does not involve investigation of aggressive traits in Māori or any other population.

Ethnic differences in MAO-A allele frequencies

Ethnic variation in allele frequency (called population stratification by geneticists) is notorious for confounding genetic association studies and leading to false positive results. Therefore, it is sound research practice to identify and attempt to control for these effects prior to commencement of such studies, especially in ethnically and genetically mixed populations such as New Zealand.

MAO-A30bp-rpt allele frequencies appear to vary substantially between different worldwide ethnic groups (Table 1). For our studies of alcohol response traits in males, we estimated the population prevalence of the MAO-A30bp-rpt alleles for Māori by genotyping 46 unrelated male individuals. We found that the 3-repeat or "low activity" allele was present at a frequency of 56% (Table 1). Although the modest sample size places uncertainty around this statistic (95% CI:42–70), the frequency is almost two-fold higher then the Caucasian frequencies reported by Caspi et al, 2002[5] (P-value for Yates corrected χ^2 test=0.002) and is consistent with the Pacific Islander data from Sabol et al (1997).[2] Note that the highest frequency of the 3-repeat allele was observed in Chinese males (77%).[6]

Table 1. Estimates of MAO-A30bp-rpt (3-repeat) allele frequencies among ethnic groups

Ethnic Group	Allele frequency (%)		N (chromosomes)	Reference
	3-repeat	95% CI		
Caucasian (males)	34	32–36	2382	Caspi et al, 2002
Chinese (males)	77	66–88	55	Lu et al, 2002
African (male + female)	59	46–72	52	Sabol et al, 1998
Hispanic (male + female)	29	12–46	27	
Pacific Islander (male + female)	61	47–75	50	
Māori males (at least 1 Māori parent)*	56	42–70	46	Lea et al, 2005

CI=confidence interval; *Individuals were recruited from the general Wellington population and were affiliated with multiple iwi (tribes) and hapū (subtribes). Therefore we considered this to be a "fairly" random, albeit small, sample of the Māori population. All participants were informed about the nature of the research (to the best of our ability) and gave consent to participate in Dr Chamber's studies of genetic markers and alcoholism at Victoria University (current ethics approval no. WEC 04/06/040).

The difference in MAO-A30bp-rpt allele frequency we observed for Māori males compared to Caucasians raises some scientific and medically relevant questions:

- Do historical population genetic forces in Polynesia explain the differences?
- Do these differences contribute to the differential patterns of alcohol and tobacco use seen between these groups?

  and, more importantly…

- Can this information be utilised for developing more appropriate treatments (e.g. smoking and drinking cessation) and lead to better health outcomes for Māori?

Positive selection at the MAO-A gene

In a high-profile study, Gilad et al (2002) re-sequenced the entire MAO-A gene from globally diverse groups of males and found additional polymorphisms spanning the entire 90kb of MAO-A DNA sequence.[7] Analysis of this data provided evidence that MAO allele frequencies were influenced by positive selection perhaps acting on behavioural traits. The authors concluded by saying:

> "This finding should motivate further studies of this region as a candidate in genetic association studies"

The findings of Gilad and colleagues, coupled with the unusual migratory history of the Māori population, prompted us to investigate the MAO-A locus further before testing it as a candidate for alcohol and tobacco-use traits.

To date, we have characterised and published associations among polymorphisms spanning the entire MAO-A gene (including MAO-A30bp-rpt) and identified two additional polymorphisms that are suitable for scoring the most common haplotype (AGCCG).[8] This haplotype was present in 70% of the Māori we tested (n=46) compared to 40% of the global (non-Māori) sample tested by Gilad et al.[7] In a sub-sample of 17 Māori males (selected because they had 8 Māori great grandparents and thus reduced European admixture), the AGCCG haplotype frequency was increased in carriers of the "functional" 3-repeat allele compared to non-Māori carriers ($p<0.014$).[7,8]

This finding in itself is evidence of positive (natural) selection acting at the MAO-A gene. It suggests to us that Polynesian males who embark on long, dangerous canoe voyages and engaged in (and survived) war with other islander tribes carried the AGCCG haplotype, coupled with the 3-repeat allele of MAO-A30bp-rpt, to Aotearoa (New Zealand) where they both increased in frequency due to rapid population growth. More importantly, these results emphasise that researchers conducting case-control studies of MAO-A gene variants and drug response or disease traits in New Zealand cohorts need to exercise extreme caution when interpreting allele frequencies so as not to declare false positive associations.

The "warrior" gene hypothesis and Māori

The Māori population of Aotearoa (New Zealand) represents the final link in a long chain of island-hopping voyages stretching across the South Pacific—"the last of the great human migrations."[9]

> "Kupe had monumental courage and a huge sense of adventure, to go where no man had ever gone before" (From Alan Duff's *Māori Heroes*)

It is well recognised that historically Māori were fearless warriors. Indeed, reverence for the "warrior" tradition remains a key part of Māori cultural structure today and one that many New Zealanders take an obvious pride in, especially in the sporting context.

In an effort to explain the significance of our research findings we reason that the MAO-A gene may have conferred some selective advantage during the canoe voyages

and inter-tribal wars that occurred during the Polynesian migrations and may have influenced the development of a substantial and sophisticated culture in Aotearoa (New Zealand).

It is important that the incidental formation of this "warrior gene hypothesis" is interpreted for what it is—a retrospective, yet scientifically plausible explanation of the evolutionary forces that have shaped the unique MAO-A gene patterns that our empirical data are indicating for the Māori population.

As alluded to by Merriman and Cameron, the extrapolation and negative twisting of this notion by journalists or politicians to try and explain non-medical antisocial issues like criminality need to be recognised as having no scientific support whatsoever and should be ignored.

Final comments

In summary, our research involves analysis of the MAO-A gene as a genetic marker for alcohol and tobacco response traits with a view to improving the health of New Zealanders. In this article we have provided statistically significant evidence that allele frequencies of the "functional" variant (MAO-A30bp-rpt) are different in Māori compared to Caucasian. We have provided further evidence supporting the notion that MAO-A in Māori has been shaped by ancient episodes of positive selection and genetic bottlenecks, and we suggest this was due to both environmental pressures during the migrations and behavioural characteristics of Polynesian voyagers. Through studying the evolutionary history of MAO-A we have gained valuable knowledge for conducting large-scale, robust association studies of drug response traits in New Zealanders with the aim of developing more personalised disease treatments based on MAO-A genotype.

In this issue of the *Journal*, Crampton and Parkin (*Warrior genes and risk-taking science*; http://www.nzma.org.nz/journal/120-1250/2439) convey their ethical concerns surrounding the "warrior gene" story. In terms of our research, we assure readers that we have taken all reasonable steps over the years, including extensive consultation with Māori and ethics committees, to ensure that our genetic studies comply with the expectations of participants.

We do not see how our revealing evidence of positive selection at the MAO gene in Māori (and our suggested reasons for why this might have occurred) as transcending ethical boundaries, but rather as logical scientific interpretation. With this in mind, it was surely our obligation as ethical researchers to disseminate findings to key stakeholders including the general public through media engagement. Of course, when engaging the media, scientists can only do their best to convey (often technical) findings and hope that journalists accurately report the scientific interpretations. If the media distorts the story, as was the case here, then the investigators have an extended social responsibility to engage in subsequent debate and try to ensure that correct interpretation prevails.

The publicity surrounding the "warrior gene" story has taught us some valuable lessons and has led to the establishment of an ESR policy working group, which is comprised of Māori academics, iwi members, researchers, and scientists. The goal of the group is to develop best practice procedures for genetic research involving Māori including informing participants, use of data, and dissemination of findings.[10] We

expect the group's developments will also be helpful to other researchers and ethics committees regarding genetic studies involving Māori.

In conclusion, the "warrior gene" controversy, although largely based on negative media hype and misconception, has catalysed important social and scientific debate about genetic screening in human populations. It has highlighted the point that complex human characteristics (such as behavioural traits) mostly confer potential rather than setting an inescapable fate. We feel that continued debate and public education on this topic is a positive move forward that should ultimately lead to informed and sensible policies for the appropriate use of genetic information, thus minimising risk of data misuse for mischievous ends.

There is mounting evidence that individuals respond differently to environmental exposures, including medicines, based partly on differences at key genes. There is also evidence that people with Māori ancestry inherit such genes (e.g. MAO-A) with a different likelihood compared to their European counterparts.[9]

It is important that biomedical and public health researchers, clinicians, drug companies acknowledge genetic differences in Māori since some of these may contribute to drug response differences and/or disease disparities in New Zealand. Indeed, ignoring evolutionary forces (such as gene selection), and assuming that all population subgroups have the same genetic background when designing diagnostic, prevention, and treatment regimes, is both unscientific and unethical and destined to do minority groups such as Māori a disservice in terms of health care.

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Author information: Rod A Lea, Genetic Epidemiologist, Institute of Environmental Science and Research Ltd, Porirua; and Adjunct Research Associate, Victoria University of Wellington; Geoffrey K Chambers, Reader, School of Biological Sciences, Victoria University of Wellington, Wellington.

Correspondence: Dr Rod A Lea, Environmental Science and Research Ltd, 34 Kenepuru Drive, PO Box 50-348, Porirua. Email: Rod.Lea@esr.cri.nz

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Improving Māori health outcomes with decision support

Phil Hider

For some time, a major challenge in New Zealand has been the pressing need to redress differing health outcomes between Māori and non-Māori. Much of the difference in mortality rates between Māori and non-Māori lies in the comparatively rapid decline in cardiovascular death among non-Māori which has not been shared by Māori.[1]

Underpinning the continuing epidemic of cardiovascular mortality among Māori are sustained high levels of major risk factors,[2] along with relatively lower intervention rates[3] for Māori than would be expected for their clinical need. Despite the results from some promising initiatives,[4] improvements in health outcomes for Māori have been more anticipated than realised. Alarmingly now, after decades of disparity, the differences in mortality between Māori and non-Māori appear to be widening.[1]

To address the challenge of improving health outcomes for Māori, a spectrum of activity has been advocated.[5] Critical among them is the pressing need to ensure that all Māori patients are able to have an evaluation of their risk factors in primary care and receive the best available preventive treatment. To support this activity, evidence-based guidelines have been developed in New Zealand to guide risk assessment and treatment for both Māori and non-Māori patients.[6]

If fully implemented, the potential of these guidelines to improve health outcomes is immense—some 55% of future cardiovascular disease events could be prevented.[6] One of the key benefits from the guidelines would come from their ability to close the gap between optimal treatment and actual prescribing rates in primary care where research suggests that about two-thirds of patients with vascular disease may not be receiving the medications that can effectively improve their survival.[7]

To help realise the full benefit for Māori, the guidelines recommend that their risk assessment should be provided a decade earlier than non-Māori.[6] The decision support software studied in this issue of the *Journal* provides the ability to undertake risk assessment with patients in primary care and then also present them with specific management advice that directly employs the recommendations from the cardiovascular guidelines.[8]

Evidence-based medicine (EBM)[9] and information technology[10] have separately been attributed with great potential to improve the quality of health care. Electronic decision support now fuses the promise of evidence-based material with the possibilities of information technology. The ability of information technology to link complex sets of information within the electronic health record gives decision support its unique platform.

By enabling EBM to operate at a systems level, decision support also maximises the opportunity for all practitioners to put into practice the best available evidence. Decision support bypasses many of the problems associated with paper-based guidelines. No longer will the use of guidelines depend on necessity that practitioners

will take the extra time required to locate them in a busy consultation or possess the additional skills needed to effectively apply them.[11]

For many GPs, the issue has been simply one of information overload—they have felt overwhelmed by the amount of information available to them. Decision support ensures that the best evidence can be automatically available at the time of clinical decision making. Indeed, making the best evidence available at the time of clinical decision making has been shown to be effective at improving practitioner performance across a whole spectrum of clinical activity including disease prevention, diagnosis, management, and detailed aspects of prescribing.[12]

Just expecting a decision support tool though to improve practitioner performance and patient outcomes is not enough. Like any intervention, it needs to be subjected to rigorous evaluation to assess its uptake and impact on practitioner performance and patient care—adverse findings have been recorded from the use of some decision support tools, especially among those systems that have been unpopular with clinical staff.[12]

The results from the current study give promise to the potential of decision support to improve cardiovascular health for both Māori and non-Māori. The report follows on from previous data that suggests that decision support is an acceptable tool that can improve practitioner performance.[13] Decision support is as likely to increase the documentation of cardiovascular disease risk assessment and risk factors for Māori as non-Māori.[14]

With the current report, information is now available about one of the largest cohorts of Māori and non-Māori patients ever assembled in New Zealand. In addition to assisting with direct patient care, the large size of the database now suggests that information from the risk assessments (when fed back to a central repository and combined with mortality and morbidity information from other sources) could be used to customise the risk assessment information to generate a New Zealand-specific profile of cardiovascular prognosis among both Māori and non-Māori populations.

New developments such as decision support offer an opportunity to bridge the quality chasm and narrow the disparity in ethnic outcomes. Risk assessment and management is a key linchpin at the interface between personal and population health care.

However, before the promise offered by this tool may be fully realised, several potential obstacles at different levels of the health system should be addressed:

- The work plan laid out in the WAVE report has to be advanced;[15]
- Clinical information systems in primary care need to be able to communicate with each other and then in turn share relevant clinical data with other parts of the health system. (The development of shared electronic information systems across primary care offers decision support its best platform to widely inform patient care and gather the full information about risk profiles.);
- Decision support tools need to be distributed across the country and made available not only in traditional general practices but also in other more Māori-specific models of primary care.

- In addition to decision support, electronic tools that track patients who have been identified at high risk (but who are slow to present) need to be put in place so that everyone is given the opportunity for regular follow-up.
- Opportunities for increased intervention revealed by the decision support tools need to be addressed;
- More support for Māori-specific lifestyle interventions needs to be available.
- Rates of secondary prevention among Māori patients with known cardiovascular disease still need to be optimised such that all patients (unless contraindicated) are receiving anti-platelet or anticoagulant, antihypertensive, and lipid-lowering medications.
- To support these activities, adequate funding needs to be extended to primary care practices and other providers who manage and monitor Māori patients and their whānau (extended family).
- Downstream from primary care, better access to secondary care procedures including revascularisation needs to be available for Māori patients.

To coordinate and maximise all these efforts, both primary and secondary care organisations (especially District Health Boards) need to facilitate quality improvement plans that educate providers and patients to the importance of addressing risk factors, while promoting systems that assess risk for every patient and identify those patients in need of effective treatments.

The widespread adoption of decision support tools across the whole country would create a world class database of information about risk and management that would be New Zealand-specific. In particular, the universal delivery of evidence-based information to all patients (especially identifying those most at risk) would advance the pressing need to bridge disparities in mortality between Māori and non-Māori.

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Author information: Phil Hider, Senior Lecturer in Clinical Epidemiology, University of Otago, Christchurch

Correspondence: Dr Phil Hider, Senior Lecturer in Clinical Epidemiology, University of Otago, PO Box 4345, Christchurch. Fax (03) 364 3697; email phil.hider@chmeds.ac.nz

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Assessing Māori/non-Māori differences in cardiovascular disease risk and risk management in routine primary care practice using web-based clinical decision support: (PREDICT CVD-2)

Tania Riddell, Rod Jackson, Susan Wells, Joanna Broad, Lot Bannink

Abstract

Aim To describe the cardiovascular disease risk factor status and risk management of Māori compared with non-Māori patients opportunistically assessed in routine practice using PREDICT-CVD, an electronic clinical decision support programme.

Methods In August 2002, a primary healthcare organisation, ProCare, implemented PREDICT-CVD as an opportunistic cardiovascular risk assessment and management programme. Between 2002 and February 2006, over 20,000 cardiovascular risk assessments were undertaken on Māori and non-Māori patients. Odds ratios and mean differences in cardiovascular risk factors and risk management for Māori compared to non-Māori (European and other, Pacific, Indian, and other Asian) patients were calculated.

Results Baseline risk assessments were completed for 1450 (7%) Māori patients and 19, 164 (93%) non-Māori patients. On average, Māori were risk assessed 3 years younger than non-Māori. Māori patients were three times more likely to be smokers, had higher blood pressure and TC/HDL levels ,and twice the prevalence of diabetes and history of cardiovascular disease as non-Māori. Among patients with a personal history of cardiovascular disease, Māori were more likely than non-Māori to receive anticoagulants, blood pressure-lowering and lipid-lowering medications. However, of those patients with a history of ischaemic heart disease, Māori were only half as likely as non-Māori to have had a revascularisation procedure.

Conclusion An electronic decision support programme can be used to systematically generate cardiovascular disease risk burden and risk management data for Māori and non-Māori populations in routine clinical practice in real-time. Moreover, the PREDICT-CVD programme has established one of the largest cohorts of Māori and non-Māori ever assembled in New Zealand. Initial findings suggest that Māori are more likely than non-Māori to receive drug-based cardiovascular risk management if they have a personal history of cardiovascular disease. In contrast, among the subgroup of patients with a history of ischaemic heart disease, Māori appear to receive significantly fewer revascularisations than non-Māori.

The gap in life expectancy between Māori and non-Māori in New Zealand increased over the period 1980–1999. Most notably, the slow decline in Māori cardiovascular disease (CVD) mortality rates over this period contrasted with the rapid decline in non-Māori rates.[1] As a consequence, CVD remains the major contributing cause to the widening life expectancy disparity between Māori and non-Māori people in New Zealand.

The most recent New Zealand guideline on CVD risk management[2] recommends a systematic evidence-based approach to assessment and management based on a patient's absolute 5-year risk.

The target population for CVD risk assessment is all men over the age of 45 years, and all women over the age of 55 years. However, at any given age, Māori have an increased prevalence of CVD compared to non-Māori. Therefore, risk assessment is recommended a decade earlier for Māori patients—i.e. at ages 35 years for Māori men, and 45 years for Māori women.

PREDICT-CVD is a web-based clinical decision support programme for CVD risk assessment and management. Its development has been described elsewhere.[3] PREDICT-CVD has been shown to be an effective tool for increasing CVD risk assessment in routine primary care practice. Indeed, in a before-after evaluation, investigators found its use produced a four-fold increase in risk assessment and risk factor documentation for both Māori and non-Māori.[4,5]

This paper reports on differences at baseline between Māori and non-Māori CVD risk assessments and management in the first 3½ operational years of PREDICT-CVD in ProCare a large primary health organisation in Auckland, New Zealand.

Methods

This report describes a pre-planned analysis of Māori/non-Māori differences in a study examining CVD risk and risk management in primary care in Auckland, New Zealand. A full description of the study methods and data definitions can be found in the paper by Bannink et al.[3]

A web-based clinical decision support programme, PREDICT-CVD, was integrated with the patient management system MedTech for primary care practitioners in ProCare, a large primary health organisation (patient population approximately 650,000) in Auckland, New Zealand.

Whenever a ProCare health practitioner used PREDICT-CVD, an anonymous electronic patient profile was generated and stored on the PREDICT server. PREDICT-CVD generates either one or two datasets; the first dataset is generated on all patients assessed and includes a CVD risk factor profile and estimate of 5-year CVD risk.

A second extended dataset that includes risk assessment and risk management is generated if the practitioner uses PREDICT to provide individualised management advice based on a patient's CVD risk. This is typically requested for high-risk patients. This latter dataset includes more extensive information on CVD risk factors and current drug and non-drug management of CVD risk. The data from these two patient data sets formed the basis for this study.

When clinicians use PREDICT, ethnicity data are automatically transferred from the patient management system. The New Zealand Health Information Service recommend the categorisations of Statistics New Zealand for self-reported ethnicity.[6] For the purpose of these analyses, ethnicity data from PREDICT were categorised into two groups: Māori and non-Māori (European and other, Pacific, Indian, and other Asian peoples).

All data were analysed using SAS 9.1 statistical software. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for Māori compared with non-Māori using a logistic regression model that adjusted for age group and sex. For continuous data, mean differences and 95% CIs were calculated using a general linear model that adjusted for age group and sex.

Subgroup analysis was undertaken for those patients with a personal history of CVD and the smaller subgroup of those with a history of ischaemic heart disease (IHD). A logistic regression model adjusting for age group and sex was used to test the association of Māori compared to non-Māori of having had a revascularisation procedure (percoronary intervention such as a stent or angioplasty, or coronary artery bypass graft—PCI or CABG).

Ethical approval: The PREDICT project was approved by the Auckland Ethics Committee (AKY/03/12/314).

Results

Between 2002 and February 2006, PREDICT-CVD collected and stored 20,614 CVD risk assessments. Of these, 1450 (7%) were for Māori patients and 19,164 (93%) for non-Māori patients. The mean age of Māori patients was 53.2 years for Māori and 46% were female. For non-Māori the mean age was 56.5 years and 44% were female.

Table 1 presents ORs and mean differences with 95% CIs for CVD risk factors for all Māori and all non-Māori patients adjusted by age group and sex.

Table 1. CVD risk factor profiles for all Māori compared to all non-Māori

CVD risk factor	Māori (n=1450) n (%)	non-Māori (n=19,164) n (%)	Odds ratio or mean difference ORs (95% CI)
Smoking	471 (32)	2200 (11)	3.51 (3.12–3.96)
Diabetes mellitus	353 (24)	2642 (14)	2.23 (1.96–2.53)
History CVD	215 (15)	2126 (11)	1.88 (1.61–2.21)
Ischaemic heart disease	152 (10)	1575 (8)	1.75 (1.45–2.10)
Stroke/Transient ischaemic attack	67 (5)	495 (3)	2.36 (1.80–3.08)
Peripheral vascular disease	37 (3)	291 (1)	2.22 (1.56–3.16)
Family history CVD	484 (33)	5077 (26)	1.31 (1.16–1.46)
			RDs (95% CI)
Mean systolic blood pressure (mmHg)	138	135	2.30 (1.34–3.26)
Mean diastolic blood pressure (mmHg)	84	81	2.81 (2.25–3.36)
Mean total cholesterol/HDL ratio	4.3	4.1	0.22 (0.15–0.28)

Odds ratios and mean differences adjusted for age group and sex.

Within this cohort of ProCare patients, Māori were three times more likely to be smokers than non-Māori. Māori patients were also twice as likely to have diabetes and a personal history of CVD (IHD, stroke or transient ischaemic attack, and peripheral vascular disease) as non-Māori.

The reporting of a family history of CVD was 30% higher for Māori compared to non-Māori. On average, body mass index, systolic and diastolic blood pressures, and total cholesterol/HDL ratios were higher for Māori compared to non-Māori.

Of the 20,614 patients screened, 2341 (11%) had a history of CVD. Of this group of high-risk patients, 215 (9%) were Māori and 2126 (91%) non-Māori. Management data (i.e. the second PREDICT dataset described in the Methods section) was available for 1363 (58%) of these patients, with a greater proportion of Māori (66%) than non-Māori (57%) patients having both risk assessment and risk management datasets completed.

Table 2 presents ORs and mean differences with 95% CIs for CVD risk factors and management variables for Māori compared to non-Māori with a history of known CVD. Within this group of patients, Māori were more likely than non-Māori to be smokers, to have diabetes, a family history of CVD and a high BMI. However, Māori were more likely to be prescribed antiplatelet or anticoagulant, antihypertensive, and lipid-lowering medications. As a result, Māori and non-Māori in this subgroup had comparable blood pressure and serum lipid measurements.

Table 2. CVD risk factor and management profiles for Māori compared to non-Māori with a personal history of CVD

CVD risk assessment or risk management variable	Māori (n=215)	non-Māori (n=2126)	Odds ratio or mean difference (95% CI)
Risk assessment variable	**n (%)**	**n (%)**	
Smoking	69 (32)	264 (12)	2.96 (2.14–4.10)
Diabetes mellitus	95 (44)	550 (26)	2.26 (1.69–3.02)
Family history CVD	88 (41)	656 (31)	1.17 (1.04–1.31)
Mean systolic blood pressure (mmHg)	135	135	0.15 (-2.57–2.86)
Mean diastolic blood pressure (mmHg)	81	80	1.26 (-0.18–2.70)
Mean total cholesterol/HDL ratio	4.2	3.9	0.32 (0.15–0.49)
PREDICT risk management module used in consultation*	143 (66)	1220 (57)	1.41 (1.05–1.91)
Risk management variable	**n (%)**	**n (%)**	
Anticoagulants	106 (74)	861 (71)	1.43 (0.96–2.17)
Antihypertensives	111 (78)	857 (70)	1.60 (1.06–2.48)
Lipid-lowering medications	88 (61)	746 (61)	1.04 (0.72–1.50)
Triple pharmacotherapy	69 (48)	539 (44)	1.24 (0.87–1.77)
Mean body mass index	32	29	2.83 (1.75–3.90)
Mean total cholesterol (mmol/L)	5.2	5.2	-0.03 (-0.24–0.17)
Mean HDL cholesterol (mmol/L)	1.4	1.4	-0.08 (-0.17–0.00)
Mean LDL cholesterol (mmol/L)	3.0	3.0	-0.07 (-0.26–0.11)
Mean triglycerides (mmol/L)	2.1	1.9	0.24 (0.05–0.43)

Odds ratios and mean differences adjusted for age group and sex; *PREDICT includes both risk assessment and risk management modules (see Methods); The risk management module is typically used for high risk patients

A further subset analysis of those with a history of ischaemic heart disease (IHD) (n=1727) was undertaken to determine access to revascularisation procedures. Only 18% of Māori (27/152) compared with 30% of non-Māori (473/1575) patients with a history of IHD had undergone a revascularisation procedure. Māori had approximately half (OR=0.46, 95%CI: 0.34-0.83) the revascularisation rate as non-Māori in this cohort of patients.

Discussion

This paper has reported differences between Māori and non-Māori CVD risk assessments undertaken opportunistically in the first 3½ years of a large pilot of a web-based clinical decision support programme in routine general practice.

Baseline CVD risk assessments were completed for over 20,000 patients aged 35 years and older from ProCare, a large Auckland-based primary care organisation, establishing one of the largest cohorts of Māori and non-Māori ever assembled in New Zealand. Of these assessments, 7% (1450) were from Māori patients.

The mean age of Māori participants was 53 years while non-Māori participants were on average 3 years older, complying toward (but not meeting) current guideline recommendations and the higher age specific CVD risk of Māori. This indicates that more can be done to risk assess Māori at a younger age. The higher prevalence of CVD risk factors among Māori in this study was similar to previous studies.

Tobacco consumption is more prevalent among Māori than non-Māori, and has declined less for Māori over the last 20 years.[7] Diabetes prevalence among Māori is three and five times higher for males and females respectively compared to New Zealand Europeans.[8]

The observation of greater CVD risk associated with increased body mass index, blood pressure, and cholesterol levels for Māori compared to non-Māori is also consistent with other New Zealand cross-sectional surveys.[9-14]

Robust epidemiological and clinical trial evidence supports the use of anticoagulant, antihypertensive, and lipid-lowering medications for the secondary prevention of CVD.[15-18] Encouragingly, for those patients with a history of CVD, Māori were more likely than non-Māori to be receiving (and taking) these pharmacotherapies given the comparable systolic blood pressure; and total, HDL, and LDL cholesterols.

This may indicate that primary healthcare practitioners are intensifying their efforts given the highlighted Māori health disparities. However, (unless contraindicated) all those with known CVD should be receiving antiplatelet or anticoagulant, antihypertensive, and lipid-lowering treatment in the secondary prevention of coronary heart disease.

Disappointingly, we found that only 58% of those with known CVD had risk management data available, and of those less than half were receiving all three drug therapies described above. This represents a significant gap between evidence-based secondary prevention and the clinical reality in primary care. Therefore, targeting risk assessment to those groups most in need is not enough.

Systematic risk management must follow risk assessment and be subject to a quality-driven implementation programme.[5]

Of concern, this study suggests that Māori with known IHD receive significantly fewer revascularisation procedures than non-Māori. Indeed, this is consistent with the findings of other studies.[19-22]

After controlling for differences in age, sex, and deprivation, one study found that CABG and PCI intervention rates for Māori patients were about half those of non-Māori.[23] It is unacceptable that Māori have greater exposure to CVD health risks and less access to high-quality secondary healthcare services in New Zealand.

Differential access to health services is a likely contributor to Māori CVD inequalities.[24] The health sector has a statutory responsibility and key role to ensure that access to healthcare for Māori is equitable.[25]

This study has highlighted that there are a number of opportunities to optimise management for Māori with CVD. They include the revision of coronary scoring and surgical prioritisation methods; providing full funding for drug therapies, including plant sterol and stanol-fortified spreads;[26] and more intensive management and monitoring of risk factors at the whānau (extended family) level.

In conjunction with drug treatment, non-drug interventions for Māori at high risk should include intensive lifestyle advice about cardioprotective diets and physical activity as well as ready access to smoking cessation programmes that are Māori specific. These opportunities could become realities with increased funding for

healthcare practitioners who actively manage and monitor high risk Māori patients and their whānau.

In addition, cardiovascular health research investment that is weighted to Māori and aimed at improving Māori CVD inequalities is needed.

Possible misclassification bias associated with ethnicity data collected by PREDICT-CVD was not addressed in these analyses. An ethnicity validation substudy is currently being analysed (A. Lindsay, personal communication, 2006). This cross-sectional analysis will compare patient ethnicity data recorded in PREDICT-CVD to self-reported ethnicity recorded via a postal questionnaire (using the standard ethnicity question recommended by the 2004 report on Ethnicity Data Protocols for the Health and Disability Sector[27]).

Over time, it will be possible to link this large and continually expanding PREDICT dataset with national data on hospital admissions and deaths. Linkage will enable us to generate New Zealand population CVD-risk prediction equations.

Māori-specific equations will replace the Framingham equations based on a white, largely middle-class, North American population. New Zealand will then have a world class CVD and diabetes data repository from which information on the burden and management of these chronic conditions can be generated and acted upon.

Note: PREDICT-CVD was developed by the University of Auckland and Enigma Publishing Ltd in collaboration with ProCare Health Ltd, Counties Manukau District Health Board, Ministry of Health, National Heart Foundation, New Zealand Guidelines Group, and MedTech Global Ltd.

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Author information: Tania Riddell, Senior Lecturer; Rod T Jackson, Professor of Epidemiology; Sue Wells, Senior Lecturer; Joanna Broad, Research Fellow; Lot Bannick, Visiting Scholar; Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland

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Correspondence: Dr Tania Riddell, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Private Bag 92019, Auckland**.** Fax: (09) 3737 624**;** email: t.riddell@auckland.ac.nz

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Māori and non-Māori differences in caesarean section rates: a national review

Ricci Harris, Bridget Robson, Elana Curtis, Gordon Purdie, Donna Cormack, Papaarangi Reid

Abstract

Aim To investigate the relationship between caesarean section (CS), deprivation, and ethnicity; and to examine Māori/non-Māori differences in CS after controlling for possible confounding factors.

Method Total, acute, and elective CS rates (as proportions of women giving birth in New Zealand hospitals) during 1997–2001 were examined by ethnicity and area deprivation. Logistic regression was used to adjust for age, deprivation, some clinical factors, and District Health Board (DHB).

Results Total, acute, and elective CS rates were significantly higher among non-Māori compared to Māori women (total CS, 21% vs 13%, ratio 1.59, p<0.0001). CS rates decreased with increasing levels of deprivation. After controlling for deprivation and age, differences between Māori (M) and non-Māori (nM) remained (total CS odds ratio nM:M 1.43, 95% confidence interval 1.39–1.48; elective OR 1.44 (1.36–1.52); acute OR 1.38 (1.33–1.43)). Differences also remained after controlling for other factors including a limited number of clinical factors.

Conclusion Results suggest that non-clinical factors may be contributing to ethnic differences in CS in New Zealand. While deprivation contributes to this difference it does not fully explain it. Further research is needed to investigate whether ethnic differences in CS impact on birth outcomes, and which factors, other than those clinically indicated, contribute to ethnic differences in caesarean section in New Zealand.

Caesarean section (CS) births have become increasingly common in New Zealand; they have risen from 9.6% in 1983/84 to 22.1% in 2001.[1–4] Within the context of rising CS rates, concerns over disparities by ethnicity have also been raised with the lowest rates among Māori women compared with other groups.[2,3,5–7]

In 2001, of women giving birth in hospital, the CS rate for Māori was 15.2% compared with 24.5% among European women.[4] Pacific women had a CS rate between that of Māori and European women at 18%.

Lower rates of other obstetric interventions have also been documented among Māori, including instrumental vaginal delivery, inductions, epidural analgesia, and episiotomy.[2–4] It has been suggested that, given higher risk pregnancies and more health problems among Māori women, higher rates of obstetric intervention, including caesarean section, might be expected.[2] This raises questions about why there is an apparent inverse relationship.

While differences persist after considering maternal age, clinical factors (such as parity,[5,7] and non-clinical factors) have been raised as possible contributors to ethnic differences.[2,7]

One New Zealand study[5] undertaken at National Women's Hospital (NWH) examined associations between ethnicity and obstetric intervention (including caesarean section) after controlling for parity and obstetric risk in more detail. Among 43,367 singleton, cephalic deliveries, not preceded by caesarean section between 1992–1999, results showed that rates of pre-labour caesarean remained lower for Māori and Pacific women than for all other ethnicities (after controlling for age, parity, and multiple clinical risk factors). For caesarean delivery rates overall, however, adjusted analyses were not significantly different for Māori or Pacific Island women compared to 'Other' ethnic groups.

Differences in CS by deprivation have also been documented in New Zealand with lower rates of CS at increasing levels of area deprivation.[2] As with ethnicity, the authors suggest that given the likely higher clinical need for intervention among women from more deprived areas, one might expect higher rates of intervention.

Ethnic disparities in deprivation (with the skewed distribution of the Māori population towards the most deprived areas)[8] may therefore contribute to differences in CS between Māori and non-Māori women. The impact of socioeconomic position on ethnic disparities in CS has not been considered in New Zealand. The interplay between ethnicity and deprivation requires further consideration in order to contribute to our understanding of ethnic disparities in caesarean section.

This study aimed to investigate the relationship between caesarean sections, deprivation, and ethnicity. It also aimed to examine Māori/non-Māori disparities in caesarean section after controlling for possible confounding factors using national hospital information.

Method

National hospital data with any diagnosis of ICD-9-CM V27, the code for outcome of delivery, were obtained from the New Zealand Health Information Service (NZHIS). This includes deliveries in public and private hospitals. Coding of caesarean sections as elective and acute was introduced in 1996.[9] Therefore, data analyses were restricted to the period 1 January 1997 to 30 June 2001. For any women having a hospital birth during this period, all admissions up to one year prior to birth, and any previous hospital births after 1 January 1988 were also obtained.

Total, elective, and acute caesarean sections (numerator) were analysed by ethnicity and deprivation among all admissions of women having hospital births (denominator).

Total CS were coded as any ICD-9-CM procedure beginning with 74.0 or 74.1.

ICD-9-CM defines an *elective caesarean* as a caesarean section carried out as a planned procedure before the onset of labour or following the onset of labour, when the decision was made before labour (ICD-9-CM codes 7401, 7411).

An *acute/emergency caesarean* is defined as a caesarean required because of an emergency situation—e.g. obstructed labour, fetal distress. It is best described as "when the caesarean section is performed having not been considered necessary previously" (ICD-9-CM 7402, 7412). Other types of caesarean sections were excluded from the analysis (n=32).

In this study it was assumed that there was likely undercounting of Māori in hospital data.[10] Therefore, any record that included Māori in either the event or National Health Index (NHI) ethnicity fields was identified as being of Māori ethnicity. Where individuals with multiple admissions were 'ever' recorded as Māori in any admission record, the ethnicity for all records for that individual was

identified as Māori. All remaining records (including those with no ethnicity specified) were classified as 'non-Māori'.

A New Zealand Deprivation Index (NZDep96) scale from 1 to 10 was assigned using women's NHI meshblock. NZDep96 is an area-based measure of socioeconomic deprivation that combines (by principal component analysis) nine variables from the 1996 Census, reflecting eight domains of deprivation.[11] Each variable was calculated as the proportion of people with the specified deprivation characteristic in each meshblock in New Zealand. The ordinal scale ranges from 1 to 10 whereby 1 is assigned to the least deprived 10% of areas and 10 to the most deprived 10% of areas.

All statistical analyses were conducted using SAS version 9.1 (SAS Institute Inc, Cary, North Carolina). Caesarean section rates were calculated as proportions of all admissions of women having hospital births. Univariate associations with ethnicity and deprivation decile were examined. Data with insufficient information available to assign a deprivation decile was excluded from deprivation analyses. Chi-squared (χ^2) tests were used to test for differences in proportions. Mantel-Haenszel Chi-squared tests were used to test for significant trends.

Age is an important confounding factor as CS increases with increasing age, and the age distribution for women having babies is different for Māori and non-Māori (i.e. Māori women tend to be younger). Logistic regression models were run to examine the relationship between CS and ethnicity after adjusting for age, as well as age and deprivation. Ethnicity was entered into the models as a dichotomous variable (non-Māori vs Māori). Age and NZDep96 were included as continuous variables.

Additional models were run on a selected group of women—adjusted for maternal age, deprivation and other potential confounding factors. To control for parity and previous caesarean, this analysis was restricted to women having their first baby—i.e. no hospital delivery since 1988, and no coding of previous caesarean (ICD-9-CM 654.2).

Other available clinical variables coded on the admission with delivery included:

- Fetal presentation (malpresentation ICD-9-CM 652);
- Gestation at delivery (pre-term delivery <37 weeks gestation ICD-9-CM 644.2;
- Post-term delivery >42 weeks gestation ICD-9-CM 645);
- Multiple births (singleton ICD-9-CM V270-271 vs multiple ICD-9-CM 272-279);
- Maternal hypertension (HT, ICD-9-CM 401, 642, 796.2);
- Maternal diabetes (DM, ICD-9-CM 250, 648.0, 648.8); and
- Antepartum haemorrhage (APH, ICD-9-CM 641).

In addition, District Health Board (DHB) of women's residence was also entered into models to assess the impact of regional differences in CS on ethnic disparities.

An interaction term (ethnicity × NZDep96) was added to the models to test for any differences in the relationship between deprivation and CS for Māori and non-Māori women. Odds ratios (OR) and 95% confidence intervals (CI) are presented for non-Māori compared to Māori and for each increasing NZDep96 decile.

All odds ratios presented are for each category of caesarean section versus all other types of birth—e.g. total CS vs all else, acute CS vs all else and elective CS vs all else. For acute CS the comparison group also includes women with an elective CS birth. Models were also run estimating the odds of acute CS vs no CS. These showed similar results and are therefore not presented.

Results

From January 1997 to June 2001, there were a total of 243,539 admissions for women having hospital births (21% Māori and 79% non-Māori). The most common age group (in 5-year bands) for Māori women to give birth was 20–24 years, and for non-Māori it was 30–34 years. Thirty-two of these births involved other types of CS and are excluded from the analysis. The CS rate was 19.5% (n=47,363).

Large disparities in area deprivation were evident between Māori and non-Māori women, although both groups were slightly more deprived than Māori and non-Māori in the total population.

Caesarean section rates, both elective and acute, were significantly higher among non-Māori women compared with Māori women (Table 1). Among women who had a CS, Māori women were significantly more likely (p<0.0001) to have an acute CS (71% of all caesareans) compared to non-Māori women (64%).

Table 1. Caesarean section by ethnicity, Jan 1997–June 2001 (number, percentage of deliveries, and non-Maori to Maori ratio)

Caesareans	Māori (M) (N=51,106)		non-Māori (nM) (N=192,401)		Ratio nM vs M (95% CI)	P values
	n	% of deliveries	n	% of deliveries		
Acute	4777	9.35	25,912	13.47	1.44 (1.40–1.48)	<0.0001
Elective	1989	3.89	14,685	7.63	1.96 (1.87–2.05)	<0.0001
Total	6766	13.24	40,597	21.10	1.59 (1.56–1.63)	<0.0001

There was a significant relationship between CS rates and deprivation (Table 2), with CS rates decreasing with increasing area deprivation for both acute and elective CS (p<0.0001, Mantel-Haenzel chi-square test for trend).

Table 2. Caesarean section by deprivation, Jan 1997–June 2001 (number and percent of deliveries)

Caesareans		NZDep96 decile										P values for trend
		1	2	3	4	5	6	7	8	9	10	
Acute	n	2557	2526	2489	2431	2361	2518	2665	2819	2864	3342	
	%	13.6	14.0	13.9	13.3	12.8	13.0	13.1	12.6	11.7	10.9	<0.0001
Elective	n	1756	1533	1489	1451	1355	1344	1307	1296	1328	1410	
	%	9.3	8.5	8.3	8.0	7.4	6.9	6.4	5.8	5.4	4.6	<0.0001
Total	n	4313	4059	3978	3882	3716	3862	3972	4115	4192	4752	
	%	22.9	22.5	22.3	21.3	20.2	19.9	19.5	18.4	17.1	15.5	<0.0001

Logistic regression modelling showed that after adjusting for age, non-Māori women were significantly more likely to have a CS birth than Māori for total, elective, and acute caesareans (Table 3). After adjusting for age and deprivation, the odds of CS comparing non-Māori to Māori reduced slightly overall and was mostly due to the stronger association between deprivation and elective CS. Adjusting for deprivation had little effect on the association between ethnicity and acute CS.

For total and elective CS there was a significant gradient by deprivation after adjusting for age and ethnicity, with a decreasing chance of CS with increasing deprivation (Table 3). This relationship was not significantly different for Māori and non-Māori.

Table 3. Logistic regression models—odds ratios of caesarean section among all admissions of women for delivery, January 1997–June 2001

Caesareans	Variables	Age adjusted model OR (95% CI)	Age and NZDep96 adjusted model OR (95% CI)	Age and ethnicity adjusted model OR per decile (95% CI)†
Acute CS	Non-Māori:Māori	1.39 (1.34–1.44)	1.38 (1.33–1.43)	
	NZDep96 (more deprived: less deprived)			0.996 (0.992–1.001)*
Elective CS	Non-Māori:Māori	1.54 (1.46–1.62)	1.44 (1.36–1.52)	
	NZDep96 (more deprived: less deprived)			0.970 (0.964–0.976)
Total CS	Non-Māori:Māori	1.47 (1.43–1.52)	1.43 (1.39–1.48)	
	NZDep96 (more deprived: less deprived)			0.985 (0.981–0.989)

208806 observations, 34701 excluded with missing NZDep96; *Significant interaction; †linear fit from decile 1 (least deprived) to decile 10 (most deprived).

For acute CS, the results did not demonstrate a significant relationship with deprivation after adjusting for age and ethnicity (Table 3). However, modelling with inclusion of the interaction term indicated that the relationship by deprivation is significantly different for Māori and non-Māori (χ^2=6.17, DF=1, p=0.013).

Among Māori women there was a significant relationship between increasing deprivation and less likelihood of an acute CS (OR at each level of deprivation=0.981, 95%CI=0.968-0.994, p=0.0036). Among non-Māori women, there was no significant relationship between deprivation and acute CS (OR at each level of deprivation=0.998, 95%CI=0.993-1.003, p=0.49).

The association between ethnicity and CS may be confounded by other factors such as parity and clinical risk, or differential access to services. Table 4 presents analyses restricted to women having their first baby (no previous admissions since 1988) with no previous CS. There were 108,636 admissions (16% Māori, 84% non-Māori).

Among Māori women, the CS rate was 15% compared with 24% among non-Māori. The elective CS rate among Māori was 1.9% compared with 4.6% among non-Māori. The acute CS rate among Māori was 14% compared with 19% among non-Māori.

Among women having their first baby in hospital, non-Māori women were more likely to have a CS than Māori, after adjusting for age. After adjusting for age, deprivation, and other clinical factors, the OR for non-Māori compared to Māori is reduced towards one for total, elective, and acute CS—but non-Māori are still significantly more likely to have a CS birth than Māori, especially for elective CS.

Table 4. Logistic regression models—odds of caesarean section (non-Māori [nM]: Māori [M]) among women having their first baby, with no previous CS; January 1997–June 2001

Caesareans		Age adjusted model OR (95% CI)	Age, deprivation and clinical factors adjusted model‡ OR (95% CI)	Age, deprivation, clinical factors and DHB adjusted model‡‡ OR (95% CI)
Acute CS	nM:M	1.17 (1.11–1.23)	1.14 (1.07–1.20)	1.13 (1.06–1.19)
Elective CS	nM:M	1.73 (1.52–1.96)	1.47 (1.28–1.69)	1.36 (1.18–1.56)
Total CS	nM:M	1.25 (1.19–1.32)	1.19 (1.13–1.26)	1.16 (1.10–1.23)

93374 observations, 15262 excluded with missing NZDep96 or other variables; ‡Model adjusted for age, deprivation, multiple births, fetal presentation, gestation at delivery, HT, APH, DM; ‡‡Model adjusted for age, deprivation, multiple births, fetal presentation, gestation at delivery, HT, APH, DM, DHB.

There were no significant interactions between ethnicity and deprivation detected in these analyses.

With the addition of the DHB variable into the models, the odds of CS for non-Māori compared to Māori are further reduced towards one, particularly for elective CS. This suggests that the differences in CS seen across different DHBs may be contributing to the ethnic differences in CS seen at a national level.

Discussion

Our study shows significant differences between Māori and non-Māori for total, elective, and acute CS after controlling for deprivation, with non-Māori women more likely to have a CS than Māori women. This suggests that while deprivation may explain some of the disparity between Māori and non-Māori in CS, it does not explain it all. In addition, lower rates of CS among Māori women persist after also controlling for available clinical factors. Among women having their first baby with no previous CS, differences between Māori and non-Māori are greatest for elective CS.

Strengths of our study are that it explores ethnic disparities in CS among women having hospital births nationally, and considers the role of socioeconomic position (as measured by NZDep96) as well as other potential confounders. However, there are a number of limitations that should be considered in the interpretation of our findings.

It is likely that there is some degree of misclassification of variables. We used administrative data, which introduces the possibility of coding errors with regards to ICD-9-CM classification. Despite our attempts to minimise the undercount of Māori hospitalisations by the 'ever Māori' method of categorisation, subsequent studies using this method suggest that it improves but does not fully account for this undercount.[12] Such misclassification applies to both numerators and denominators in this study, and would therefore tend to bias disparities to the null.

Misclassification may also occur where record linkage was used to determine variable classification—e.g. parity. Duplicate or incorrect NHI numbers have been identified as affecting data quality[3] and may lead us to underestimate parity if individuals are not accurately linked to having previous births. Whether this is different for Māori and non-Māori women is unclear.

There may also be residual confounding of the disparities in CS between Māori and non-Māori. For example, the impact of socioeconomic position on ethnic inequalities in CS may well be underestimated in our study as we only used NZDep96. This is only one measure and will not fully capture all dimensions of socioeconomic position.[13,14]

In addition, we were limited by the data available in the NZHIS dataset. For example, we could not adjust for other potential clinical risk factors such as maternal weight, smoking, and other comorbidities. Health service information such as time at booking, and maternity carer was not available. Nor could we link mother's records to those of the baby. Thus, factors such as type of carer and baby's birth weight were unable to be measured.

However, smoking, obesity, small for gestational age, and a number of other health status measures are closely correlated with deprivation[8,15–17] and their unobserved effect may be partially captured by the inclusion of deprivation. The NWH study[5] was able to control for a wider range of clinical variables. The addition of obstetric risk factors to the model in the NWH study tended to reduce the odds of Māori having a caesarean section compared to non-Maori. Therefore, the addition of other such risk factors to the current analyses may not reduce the disparity between Māori and non-Māori.

Our results are similar to those found at NWH for ethnic inequalities in elective caesarean.[5] However, we found that Māori women also had lower adjusted rates of acute CS, which was not the case at NWH. The differences in these findings may result from the use of different methods and adjustors or perhaps reflect differences at a regional compared with a national level.

Internationally, differences in CS have been examined between various ethnic groups in different countries including the United States,[18-21] Canada,[22] Brazil,[23] South Africa,[24] Norway,[25] and Australia.[26] While there is variation in the magnitude and direction of ethnic disparities in these studies, in most studies[18–20,22,24,25] ethnic differences persist after adjusting for clinical and socioeconomic factors, thus suggesting the influence of non-clinical factors.

Even taking into consideration the limitations of our study, our findings—which show ethnic differences in CS after adjusting for socioeconomic position and various clinical factors—raise the possibility that non-clinical factors may be operating.

Possible non-clinical explanations that may influence ethnic inequalities in CS have previously been suggested. These include: patient factors such as maternal request, and patient preferences and expectations;[2,7,27] provider practice[6] and the patient provider interaction;[2] and, differential access to information and care, and differential management.[2,7]

The reasons for ethnic disparities in CS are likely to be complex and multifactorial, occurring across the continuum of care and associated with wider determinants of health and inequality. We would argue, that to address any 'inequities' between Māori and non-Māori, it is important to take a broad perspective to the investigation of potential explanations.

To focus primarily on patient and Māori 'cultural' explanations risks 'victim blaming'[28] and fails to acknowledge dominant 'cultural' explanations, the role of

providers, and structural influences of the healthcare system.[29] In addition, it does not consider wider determinants of health and inequality that influence access to care and individual risk.[30,31]

Our study shows that disparities in deprivation may partially contribute to ethnic disparities in CS. However, as a potential risk factor, ethnic disparities in socioeconomic position alone are limited as they fail to incorporate factors that lead to the unequal distribution of socioeconomic resources by ethnicity in the first place. Further research directly examining potential non-clinical reasons for ethnic disparities in CS is required within this wider context.

Finally, we note that our study does not determine appropriate CS rates for Māori and non-Māori, or whether ethnic disparities in type of delivery contribute to ethnic disparities in birth outcomes for mothers and babies. Further research is required to assess this.

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Author information: Ricci Harris, Public Health Physician, Te Rōpū Rangahau Hauora a Eru Pōmare, Department of Public Health, Wellington School of Medicine and Health Sciences, Otago University, Wellington; Bridget Robson, Director, Te Rōpū Rangahau Hauora a Eru Pōmare, Department of Public Health, Wellington School of Medicine and Health Sciences, Otago University, Wellington; Elana Curtis, Public Health Physician, Senior Lecturer Medical, Te Kupenga Hauora Māori, Department of Māori Health, University of Auckland, Auckland; Gordon Purdie, Biostatistician, Department of Public Health, Wellington School of Medicine and Health Sciences, Otago University, Wellington; Donna Cormack, Research Fellow, Te Rōpū Rangahau Hauora a Eru Pōmare, Department of Public Health, Wellington School of Medicine and Health Sciences, Otago University, Wellington; Papaarangi Reid, Tumuaki, Faculty of Medical and Health Sciences, University of Auckland, Auckland

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Correspondence: Dr Ricci Harris, Te Rōpū Rangahau Hauora a Eru Pōmare, Department of Public Health**,** Wellington School of Medicine and Health Sciences**,** University of Otago**,** PO Box 7343, Wellington**.** Fax: (04) 385 5924; email Ricci.Harris@otago.ac.nz

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Is it possible to distribute a scarce resource equitably? Access to invasive procedures for patients with acute myocardial infarction

Phillip Insull, Ritwik Kejriwal, Hitesh Patel , Jonathan Christiansen, Anthony Scott, Hamish Hart, Colin Edwards, Guy Armstrong

Abstract

Aims To compare waiting times for inpatient cardiac catheterisation between a hospital with on-site cardiac catherisation facility (Auckland City Hospital, ACH) and one of its referring hospitals (North Shore Hospital, NSH).

Methods Patients were included if they were admitted ACH or NSH with a myocardial infarction, and subsequently underwent inpatient coronary angiography.

Results 853 patients were identified from NSH and 600 from ACH. Patients from NSH waited significantly longer for coronary angiography (median delay 6 versus 3 days, p<0.0009) and fewer underwent this procedure within 48 hours of admission (11% versus 36%, p<0.0009). Delays in percutaneous coronary intervention were significantly longer for NSH patients (6 versus 3 days, p<0.0009), and fewer NSH patients underwent this procedure within 48 hours (12% versus 41%, p<0.0009).

Conclusions Inpatients with myocardial infarction waited longer for coronary angiography and percutaneous coronary intervention at a hospital without invasive facility than similar patients at the regional referral hospital with on-site invasive facility.

Randomised trials have shown that prompt revascularisation for patients with acute myocardial infarction (MI) will reduce mortality and reinfarction.[1] Reinfarction rates may be further reduced if angiography and appropriate revascularisation is carried out within hours rather than delaying for 3 days.[2]

New Zealand patients with acute coronary syndromes have differential access to cardiac catheterisation, depending on whether their hospital has such invasive facilities on site.[3]

We assessed waiting times for diagnostic cardiac catheterisation and coronary revascularisation for acute MI patients in two large metropolitan hospitals. North Shore Hospital (NSH) has the largest catchment population in New Zealand, approaching 500,000. It does not have on-site invasive cardiac services and transfers all patients for cardiac catheterisation to Auckland City Hospital (ACH). The 13-kilometre ambulance journey takes 40–70 minutes (including 10 minutes loading and unloading).

Methods

All patients admitted with myocardial infarction who subsequently underwent inpatient coronary angiography between November 2002 and January 2005 from ACH and NSH were identified from Cardiac Catheterisation and Chemical Pathology Laboratory databases. To exclude transfers to ACH

from other referring hospitals, patients were only included if they were domiciled within either Waitemata or Auckland District Health Board catchments. Hospital discharge codes were used to determine baseline characteristics and rates of percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG).

Myocardial infarction was defined as an elevation above 0.03 µg/L for troponin T (TnT) on an Elecsys E170 module (Roche Diagnostics, Basel, Switzerland) at Auckland Hospital and above 0.2 µg/L for troponin I (TnI) on an Advia Centaur at North Shore Hospital (Bayer Immunodiagnostics, NY).[4–6]

To maximise case ascertainment, the numbers of PCI and CABG procedures done in the private sector were obtained from the sole private provider in the Auckland region (MercyAscot Hospital).

Major endpoints were the waiting time from hospital admission to coronary angiography and PCI, and the proportion of patients undergoing these procedures within 48 hours of admission, as suggested by local and international guidelines.[7,8] We also assessed waiting time for CABG.

Data analysis—Binary variables are presented as frequency (percentage) and continuous variables are presented as mean ± standard deviation or median (interquartile range). Differences between groups were evaluated with Chi-squared (χ^2) or Fisher's exact test for categorical variables and Mann-Whitney test for continuous variables. SPSS (v13.0) statistical software was used for analysis and a two-sided $p<0.05$ was considered significant.

Results

Patients with unstable angina or MI numbered 4417 at NSH and 2597 at ACH (during the 26-month study period clinicians adhered variably to the new definition MI, obscuring the coding distinction between unstable angina and MI). A similar proportion from each hospital underwent inpatient coronary angiography after fulfilling criteria for myocardial infarction; 853 (19%) from NSH and 600 (23%) from ACH, and these formed our study group.

Table 1. Baseline characteristics

Variables	ACH N=600	NSH N=853	P value
Age (mean±SD)	64±13	63±12	P=0.1
Female	200 (33.3%)	246 (28.7%)	P=0.07
Diabetes	160 (26.7%)	211 (24.6%)	P=0.4
Hypertension	383 (63.8%)	379 (44.2%)	P<0.0009
Hypercholesterolaemia	187 (31.2%)	359 (41.9%)	P<0.0009
Chronic renal failure and dialysis	73 (12.2%)	48 (5.6%)	P<0.0009
PVD	52 (8.7%)	37 (4.3%)	P=0.001
Ever Smoked	37 (63.2%)	485 (56.6%)	P=0.013
Prior CHF	20 (3.3%)	42 (4.9%)	P=0.15
Prior Angina	25 (4.2%)	86 (10.0%)	P<0.0009
Prior MI	19 (3.2%)	86 (10.0%)	P<0.0009
Prior CABG	68 (11.3%)	41 (4.8%)	P<0.0009
Prior PCI	106 (17.7%)	37 (4.3%)	P<0.0009

PVD=peripheral vascular disease; CHF=congestive heart failure; MI=myocardial infarction; CABG=coronary artery bypass grafting; PCI=percutaneous coronary intervention; ACH=Auckland City Hospital; NSH=North Shore Hospital.

Patients from ACH appeared to more comorbid with more hypertension, chronic renal failure and dialysis, peripheral vascular disease, prior PCI, and prior CABG (Table 1). The regional renal and peripheral vascular services are located at ACH, so increased

incidence of these conditions is expected. Patients from NSH had more hypercholesterolaemia, historical angina, and prior MI.

Patients from NSH waited significantly longer for coronary angiography (median delay 6 versus 3 days, p<0.0009) and fewer underwent this procedure within 48 hours of admission (11% versus 36%, p<0.0009) (Table 2, Figure 1).

Table 2. Variable comparison

Variables	ACH N=600	NSH N=853	P value
Angio delay (days)	3.0 (2–5)	6.0 (4–8)	P<0.0009
Angiography within 48 hours	219 (36.5%)	94 (11.0%)	P<0.0009
Ongoing or recurrent chest symptoms or ongoing labile ECG changes	7 (1.2%)	19 (2.2%)	P=0.13
CHF in index admission	29 (4.8%)	50 (5.8%)	P=0.48
Private revascularisation	6 (1.0%)	18 (2.1%)	P=0.1

Angio delay=the inpatient waiting time for coronary angiography, presented as median (interquartile range); CHF=congestive heart failure; ACH=Auckland City Hospital; NSH=North Shore Hospital.

Delays in PCI were significantly longer for NSH patients (6 versus 3 days, p<0.0009) and fewer NSH patients underwent this procedure within 48 hours (12% versus 41%, p<0.0009) (Table 3, Figure 2). There was no difference in the proportion of PCI done on the same day as coronary angiography (95.8% for NSH versus 96.3% for ACH patients, p=0.6).

Table 3. Time to PCI

Variables	ACH	NSH	P value
PCI	271 (45.2%)	403 (47.2%)	P=0.4
PCI delay (days)	3.0 (2-6)	6.0 (4-9)	P<0.0009
PCI within 48 hours	111 (41.0%)	48 (11.9%)	P<0.0009

PCI=percutaneous coronary intervention, presented as median (interquartile range). ACH=Auckland City Hospital; NSH=North Shore Hospital.

Longer waits for CABG were experienced by NSH patients (16 versus 10 days, p<0.0009) and fewer patients from NSH underwent this procedure during their index admission (13% versus 45%, p<0.0009) (Table 4, Figure 3).

Table 4. Time to CABG

Variables	ACH	NSH	P value
CABG	100 (16.7%)	145 (17.0%)	P=0.4
CABG delay (days)	10.0 (7-17)	16.0 (11-26)	P<0.0009
CABG in index admission	45 (45.0%)	19 (13.1%)	P<0.0009
CABG within one week	28 (28%)	12 (8%)	P<0.0009

CABG=coronary artery bypass grafting, presented as median (interquartile range). ACH=Auckland City Hospital; NSH=North Shore Hospital.

Discussion

This study found that acute MI inpatients from a hospital (NSH) without cardiac catheterisation facility had significantly greater waiting time for coronary angiography and PCI than patients from the referral centre with on site cardiac catheterisation facilities (ACH). Fewer NSH patients achieved the guideline recommendation of angiography within 48 hours (11% versus 37%) or PCI within 48 hours (12% versus 41%).

The baseline differences in patients between NSH and ACH invalidated a comparison of clinical outcomes. However, these delays are may result in detrimental clinical sequelae including reinfarction and increased mortality. We are currently investigating the clinical significance of these delays.

Geographic isolation makes some inequity of access inevitable, despite affirmative action by ACH in assigning dedicated lists to NSH patients, and free access for urgent cases. After canvassing colleagues at both NSH and ACH, the perceived reasons for delayed treatment included delays in the referral process (faxed rather than verbal referrals) and unanticipated openings in the schedule that can only be realistically filled in-house, given the logistics of transportation and travel time. Bed shortages at ACH influence the scheduling priority for patients at that hospital, but bed shortages at NSH do not influence scheduling at ACH.

In addition to issues related to geographic isolation' differences in models of service provision are likely to be germane. All patients with acute coronary syndromes are admitted directly to cardiology at ACH, whereas at NSH, a minority are admitted under cardiology (directly to the coronary care unit), with most admitted under general medicine. This causes delays due to 'double handling', whereby general medicine refers to cardiology, who decide whether to refer to ACH for cardiac catheterisation.

The disparities are exaggerated by the extreme scarcity of the invasive resource, which makes equitable distribution much harder. During the time period under study, the cardiac catheterisation facilities and staff were over-stretched, with lists routinely finishing at 7–8 pm. The current study includes the period when acute cardiac services transferred from Greenlane Hospital to Auckland City Hospital, during which time service provision was hampered.

More resources have been assigned to the Auckland region with the third regional hospital (Middlemore Hospital) opening a cardiac catheterisation laboratory at the end

of 2005, and one planned to open at NSH at the end of 2007. In the future, we plan to repeat the study to assess the effect of these extra resources.

The general principle of equitable access to cardiac services is particularly topical with ongoing regional discussions regarding drug-eluting stents, non-contact mapping for arrhythmia ablation, and computed tomography coronary angiography.

Inequities in cardiovascular health outcomes within New Zealand have been recognised previously, particularly socioeconomic differences in cardiovascular mortality, and ethnic differences in coronary mortality, heart failure mortality, and admissions.[9] There are previous report of differential rates of coronary angiography and revascularisation between hospitals with cardiologists and cardiac catheterisation facilities compared with hospitals with general physicians and no cardiac catheterisation facilities.[3,10]

Conclusion

Inpatients with myocardial infarction waited longer for coronary angiography and percutaneous coronary intervention at a hospital without invasive facility than at the regional referral hospital with on-site invasive facility. This occurred despite affirmative action to facilitate access for off site patients.

Some extra waiting time is inevitable due to geographic isolation, but this may be exacerbated by extreme scarcity of the invasive resource.

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Author information: Phillip Insull, PGY1 House Officer, Whakatane Hospital, Whakatane; Ritwik Kejrewal, PGY1 House Officer, Middlemore Hospital, Otahuhu, Auckland; Hitesh Patel, Jonathan Christiansen, Anthony Scott, Hamish Hart, Colin Edwards, Guy Armstrong, Cardiologists, North Shore Hospital, Takapuna, Auckland

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Correspondence: Dr Guy Armstrong, Ward 3, North Shore Hospital, Private Bag 93-503, Takapuna, Auckland 0620. Fax: (09) 523 6890; Guy.Armstrong@waitematadhb.govt.nz

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Improving care to stroke patients: adding an acute stroke unit helps

Carl Hanger, Valerie Fletcher, John Fink, Andrew Sidwell, Anne Roche

Abstract

Background Stroke units save lives, reduce dependency, and increase the chance of returning home. A 15-bed Acute Stroke Unit (ASU) was opened on the acute hospital campus to complement an established Stroke Rehabilitation Unit (SRU) on a distant campus. The aim of this study was to address whether patient care was improved with the establishment of the ASU.

Methods Retrospective case-note review of a sample of patients admitted with an acute stroke to Christchurch Hospital. A before and after design was utilised to audit the processes of care (PoC) using the Royal College of Physicians (London) stroke audit tool.

Results 648 patients were admitted to the Acute Stroke Unit in the first year. The retrospective audit included 119 and 72 patients in the "before" and "after" cohorts respectively. The "after" cohort had more severe strokes (greater incontinence at one week, [p=0.03], and worse level of consciousness [p=0.008]). Length of stay, domicile on discharge, and mortality outcomes were similar for the two cohorts. Processes of care improved in the "after" cohort in 27 of the 43 domains audited.

Conclusion Adding an ASU to complement an existing SRU can give major improvements in PoC across many different facets of stroke care. We believe this is one step closer to both the ideals of an overall coordinated stroke service and better overall care for patients with stroke.

Patients with stroke have better outcomes if they are admitted to a stroke unit (SU);[1–3] they have greater chance of being alive and independent and less likely to require long term institutional care at one year.[1] Guidelines for the management of stroke advocate for the establishment of stroke units as well as organised stroke services throughout New Zealand.[4]

Christchurch Hospital is the acute hospital for Christchurch and surrounding North Canterbury and serves a catchment population of approximately 450,000 people.[5] It is a university teaching hospital and has the regional neurosurgical unit. Approximately 800 people are admitted each year with a diagnosis of an acute stroke.

In Christchurch, a stroke rehabilitation unit (SRU) for older patients was opened in 2001 with documented benefits both for patients and the District Health Board (DHB).[6,7]

The Christchurch SRU was always envisaged as being one part of a larger integrated stroke service, with an acute stroke unit (ASU) and a community based specialist rehabilitation team planned from the outset.[8]

As the SRU is on a separate, geographically distant campus from the acute hospital, a combined acute and rehabilitation unit was deemed not feasible. Instead an ASU was established on the acute hospital site in October 2004 with an emphasis on making the transitions to other parts of stroke care as seamless as possible.

Prior to the establishment of the ASU, patients admitted with an acute stroke to Christchurch Hospital were treated on any one of six different medical wards or one neurology ward. These patients were under the care of general physicians (12 teams, with 2 teams admitting on any given day) or neurologists in approximately 80:20 ratio. The decision as to whether the admitting team was neurologist or general physician was based predominantly on an age cut-off (<65 years) but also comorbidities and presence of neurological complications. There were no formal protocols for the management of stroke used consistently across all areas, with the exception of thrombolysis.

Thrombolysis for selected stroke patients started in April 2002.[9] These patients were admitted to the neurology high dependency area for post thrombolysis monitoring and care. A Clinical Nurse Specialist in Stroke (CNSS) position was established in 2002. Referrals to allied health professionals (AHPs) were made on individual basis by each clinical team.

Patients requiring further inpatient rehabilitation were transferred either to the SRU, or to the Brain Injury Rehabilitation Service (BIRS) for patients younger than 65 years. Both of these rehabilitation services are 5 kilometres away (in Cashmere and Burwood respectively) from the acute hospital (Central Christchurch city), but on different campuses. Outpatient rehabilitation services were also provided from these distant sites.

The ASU was established on 4 October 2004. This is a 15-bed unit embedded in one of the general medical wards (30 beds total). The ASU aims to take all stroke patients, irrespective of age, gender, and stroke severity.[10] The patients are admitted under a general physician or neurologist as before, with the exception that only one (of the two) general medical teams on take each day admitted strokes—this reduced the number of general medical teams involved to six.

To develop and maintain consistency of care between the many different treating teams, and to across different geographic sites within the city, common protocols (e.g. hypertension, use of urinary catheters, early mobilisation), educational strategies, and documentation were developed. These were developed jointly by members of the multidisciplinary teams (MDT) in the ASU, SRU, and BIRS.

Thrombolysis for selected patients continues, but is now provided in the ASU. All stroke patients are admitted using a mutually agreed proforma which acts as a prompt, but is not a formal clinical pathway.[11] The aim is to minimise duplication of information collection, whilst ensuring important information is not omitted.

Pre-stroke functioning is recorded only once in a joint “Life before Stroke” section and each professional group of the MDT supplements this with their own initial assessment proforma. Following these initial assessments, all of the MDT record ongoing progress in the same part of the notes. When a patient transfers to the SRU, this common documentation is continued as a contiguous, single set of notes.

Staff numbers were increased for AHP (physiotherapy by 1 full-time equivalent [FTE] position, occupational therapy by 1 FTE, social worker by 0.5 FTE, and speech language therapist by 0.6 FTE). Nursing or medical staff were not increased. The CNSS position predated the ASU, but it was always envisaged as an integral part of its establishment.

Each discipline is expected to see every patient within one working (Monday–Friday) day, thus negating the need for written referrals. Whilst acute stroke treatments are an important component of the ASU work, the ethos is also to provide consistent, skilled nursing and allied health professional care and to begin the rehabilitation and education processes early[12]—in line with the 5 key components of stroke units (SU).[13]

This emphasis on SU care has greater potential community benefit than a pure medical model.[14] An interdisciplinary approach for each patient is fostered with regular team meetings. To facilitate nursing involvement in rehabilitation, therapy is performed on the ward where possible.

All staff were given the opportunity to update their stroke skills. In particular, nurses were trained to screen for dysphagia and are involved in a programme of continuing professional development coordinated by the CNSS. Close linkages with the SRU and BIRS were emphasised, and the stroke specific documentation is common to all three units.

Following the introduction of the ASU, we wished to assess whether it gave additional benefit over and above the gains accrued by the SRU.[6,7] Benefit may be assessed in two main ways. One is to look at overall patient outcomes such as function, domicile, and survival, whereas the other is to look at consistency of key clinical care processes.[15]

As consistency of care (particularly in the acute phase) was thought to be poor, we have chosen to focus on the latter approach in this study. Using an internationally recognised stroke audit tool[16], this paper addresses the question—"Does the addition of an ASU give improvements in stroke care over and above the benefits already accrued by a SRU?"

Methods

The Royal College of Physicians of London stroke audit tool (RCPLSA) was used to assess the process stroke care.[16] This was designed for retrospective case note review and has been shown to have high inter-rater reliability.[17] It has two main sections: (1) casemix indicators and (2) 48 clinical process audit questions covering 12 broad areas of care.

As the last section covered five aspects of care after hospital discharge, which are not recorded in the hospital clinical record, we did not collect data on these five areas. The response to each standard is recorded as either Y (meets standard), N (does not meet standard), or not appropriate. Criteria for the latter category are tightly defined. Overall compliance (%) for each standard is defined as Y*100/(Y+N), thus excluding those not appropriate.

Prior to ASU implementation, function was only assessed in the two rehabilitation hospitals (using the Functional Independence Measure [FIM]). Since 4 October 2004, functional abilities are also routinely measured at CH using the FIM. As the two questions on function in the casemix section of the audit require a Barthel Index (BI), and not the FIM, these two questions were also omitted.

Discharge coding data (ICD-10 codes I61-I64, I67.2, and I67.5-9) were used to identify both the "before" and "after" cohorts. All patients admitted to Christchurch Hospital (CH) between 1 December 2003 and 29 February 2004, formed the "before" group. The "after" group consisted of a selection of patients (two-thirds selection, by omitting every third patient) admitted to CH between 1 February 2005 and 31 March 2005. Both groups included some patients transferred to Princess Margaret Hospital

(PMH). As the audit aimed to assess stroke care throughout all three hospitals, admission to ASU was not a prerequisite in the "after" cohort.

Categorical and continuous casemix variables were compared using Chi-squared (χ^2) analysis and Student's t-test respectively.

The study was approved by the Upper South A Regional Ethics Committee (URA/05/02/004).

Results

During the 12-month period from 4 October 2004—3 March 2005, 648 patients with an acute stroke were admitted to the ASU, with a mean (median) length of stay (LOS) of 8 (6) days. 305/648 (47.0%) were transferred to PMH or BIRS for further inpatient rehabilitation. During the same period, 735 patients were discharged from CH with a diagnosis of acute stroke. Thus approximately 88% of all strokes were admitted to ASU.

Casemix variables for the two cohorts that were audited using RCPLSA are shown in Table 1. Patients in the post-ASU cohort were significantly more likely to spend the majority of their hospital stay in a SU and less likely to have multiple ward transfers.

This group had a higher proportion of women, and had more disabling strokes as indicated by a lower level of consciousness on admission, and a worse continence status at 1 week. There were no differences in discharge destinations between the cohorts.

The results from the audit of process of care variables are shown in Table 2. Significant improvements in process of care were shown for the post-ASU cohort (compared to pre-ASU) in 27 of the 43 areas recorded.

Table 1. Casemix of the two audited groups

Variables	Pre-ASU	Post-ASU	P value
N=	119	72	
Mean age (years) [range]	72.7 [34–95]	73.5 [44–91]	0.73
Male:Female (%)	65:35	43:57	**0.006**
Spent >50% of stay in a Stroke Unit	20 (17%)	50 (69%)	**<0.001**
Changed wards more than twice	7 (6%)	0 (0%)	0.04
Type of stroke			
Ischaemic	91 (77%)	63 (88%)	
Haemorrhagic	15 (13%)	8 (11%)	0.14
Unknown	9 (8%)	1 (1%)	
Required transfer to an inpatient rehabilitation ward	37 (31%)	28 (39%)	0.27
Total length of stay (ASU and rehab. combined) –mean number days [median]	12.0 [6]	15.7 [8]	0.14
Died within 30 days (%)	28 (24%)	18 (25%)	0.94
Accommodation pre stroke (%)			
Own Home	102 (86%)	61(85%)	
Retirement unit	4 (3%)	2 (3%)	0.98
Rest home	14 (12%)	9 (13%)	
Long term Hospital	0 (0%)	0 (0%)	
Accommodation post stroke (of survivors)			
Own home	71 (76%)	37 (73%)	
Retirement unit	1 (1%)	2 (4%)	0.78
Rest home	9 (10%)	5 (10%)	
Long-term hospital	13 (14%)	7 (14%)	
Urinary incontinence at 1 week	13 (13%)	17 (27%)	0.03
Worst level of consciousness			
Fully conscious	92 (79%)	47 (65%)	
Drowsy	4 (3%)	13 (18%)	**0.008**
Semi conscious	4 (3%)	3 (4%)	
Unconscious/dead	16 (14%)	9 (13%)	

Table 2. Results of RCPL audits in pre-ASU and post-ASU cohorts

COMPLIANCE WITH STANDARD (%)				
Audit Question Number	**Standard – Audit Question**	**Pre-ASU**	**Post-ASU**	**P value**
Initial assessment				
17	Conscious level recorded in first 24 hours	117 (98%)	70 (97%)	0.61
	Eye movements recorded in first 24 hours	90 (76%)	62 (86%)	0.08
	Limb movements recorded in first 24 hours	113 (97%)	72 (100%)	0.05
18	Screening for swallowing disorders	55 (52%)	46 (75%)	0.003
	Communication	60 (59%)	50 (83%)	0.002
	Trunk control or gait	57 (56%)	51 (86%)	0.01
19	Formal mental test	16 (20%)	4 (8%)	0.04*
	Assessment of visual fields	69 (76%)	50 (88%)	0.08
	Assessment of visual inattention	29 (32%)	24 (46%)	0.10
	Sensory testing	71 (79%)	48 (89%)	0.13
Clinical diagnosis				
20	Clear diagnostic description of likely site of cerebral lesion	62 (52%)	55 (76%)	0.001
	Clear diagnostic description of type of lesion	63 (53%)	65 (90%)	<0.000
21	Brain scan carried out within 24 hours	101 (88%)	67 (96%)	0.07
Screening and functional assessment				
28	Patient weighed at least once during admission	70 (69%)	50 (79%)	0.13
29	Assessment of nutritional needs	37 (36%)	49 (82%)	<0.000
30	Pre-stroke function recorded	28 (27%)	56 (88%)	<0.000
	Function at discharge recorded	28 (28%)	52 (88%)	<0.000
32	Evidence that patient's mood have been assessed	21 (21%)	36 (61%)	<0.000
33	Patient had standardised cognitive assessment (e.g. of perception, concentration, memory, etc)	24 (26%)	27 (51%)	0.002
Multidisciplinary involvement				
16	Patient came under responsibility of a specialist multidisciplinary team of clinicians specialised in stroke within 7 days of stroke	12 (21%)	51 (82%)	<0.000

22	Swallowing assessed by speech and language therapist within 72 hours of admission	47 (51%)	33 (72%)	0.02
24	Initial assessment of communication problems by speech and language therapist within 7 days of stroke	41 (61%)	34 (85%)	0.009
23	Patient assessed by physiotherapist within 72 hours of admission	64 (69%)	55 (96%)	<0.000
25	Patient assessed by occupational therapist within 7 days of admission	61 (84%)	52 (91%)	0.20
31	Social work assessment within 7 days of referral	48 (63%)	40 (85%)	0.009
Management planning				
34	Rehabilitation goals agreed by the multi disciplinary team	27 (44%)	45 (88%)	<0.000
35	Individual goals include reference to areas of higher level of function	11 (11%)	37 (65%)	<0.000
37	Plan for mood disturbance	9 (11%)	8 (24%)	0.09
38	Positioning and handling Risk assessment of pressure sores Hydration Prevention of deep vein thrombosis Prevention of faecal impaction	14 (22%) 13 (11%) 24 (20%) 12 (10%) 20 (17%)	29 (88%) 42 (58%) 56 (78%) 42 (58%) 44 (61%)	<0.000 <0.000 <0.000 <0.000 <0.000
44	Evaluation of patient's progress on target dates for the team rehabilitation plan	28 (28%)	19 (37%)	0.30
Continence management				
26	Patient incontinent at 7 days post admission – 24 hours of documented monitoring of the patterns of incontinence	3 (4%)	4 (40%)	†
27	If patient catheterised – documentation that simpler methods of incontinence management have been tried	0 (0%)	1 (33%)	†
36	Management plan to promote urinary continence	17 (57%)	9 (75%)	0.27
Secondary prevention				
40	Treatment plan for the management of hypertension if blood pressure diagnosed above normal	29 (46%)	23 (74%)	0.01
41	Aspirin prescribed for prevention of further cerebral infarction	73 (91%)	51 (94%)	0.37
43	Other risk factors discussed with patient or carer (e.g. smoking, alcohol consumption)	33 (38%)	45 (88%)	<0.000

Information giving				
39	Discussion with patient about diagnosis and prognosis	58 (53%)	58 (89%)	<0.000
	Discussion with patient about post stroke complications	29 (30%)	45 (75%)	<0.000
	Discussion with patient about therapy goals	24 (24%)	58 (97%)	<0.000
	Discussion with patient about changes in therapy	24 (24%)	52 (87%)	<0.000
	Discussion with patient about discharge planning	54 (54%)	58 (98%)	<0.000
51	Patient/carer knows the plans for follow up	39 (41%)	56 (97%)	<0.000
52	A named contact given to patient/carer on discharge	33 (34%)	45 (80%)	<0.000
	Patient/carer given on written discharge summary on discharge	95 (97%)	53 (96%)	0.85
	Information on statutory agencies given to the patient/carer on discharge	15 (17%)	21 (45%)	0.001
	Information on voluntary agencies given to patient/carer on discharge	3 (4%)	19 (40%)	<0.000
	On discharge patient/carer given information on who will contact them after discharge and when	25 (29%)	35 (69%)	<0.000
53	Copy of community care plan from social services in the team, medical or nursing notes	9 (19%)	17 (59%)	<0.000
Discharge planning				
45	Stairs/ground floor/lift at home	46 (55%)	42 (89%)	<0.000
	Access to toilet at home	46 (55%)	38 (83%)	0.001
46	Home visit performed	24 (69%)	20 (87%)	0.11
47	Discharge to nursing home – patient had opportunity to both visit and to decide on the accommodation before discharge	9 (82%)	7 (88%)	0.78
48	Discharge to nursing home within 4 weeks of stroke – documented evidence that there is no potential for further functional improvement	5 (83%)	4 (67%)	†
	Discharge to nursing home within 4 weeks of stroke – documented evidence that other discharge options have been explored and found unsuitable	6 (100%)	5 (71%)	†
	Discharge to nursing home within 4 weeks of stroke – documented evidence that there is a review planned within 2 months of discharge	3 (30%)	2 (29%)	

Communication with GP				
49	GP informed of patient's discharge/death by day of discharge (or day following death)	50 (42%)	57 (79%)	<0.000
50	Discharge summary to GP includes diagnosis	96 (94%)	57 (97%)	0.36
	Discharge summary to GP includes medication	93 (95%)	68 (98%)	0.28
	Discharge summary to GP includes level of functional ability at discharge	31 (32%)	58 (69%)	<0.000
	Discharge summary to GP includes plans for future health care and management	67 (69%)	40 (88%)	0.007
	Discharge summary to GP includes community services organised	13 (33%)	52 (53%)	0.09
Communication with carer				
54	Carer's needs for support assessed separately	30 (31%)	34 (68%)	<0.000
55	Record of explanations to carer about disabilities that the patient has	33 (33%)	42 (81%)	<0.000
56	Evidence of carer participation in setting goals	11 (12%)	41 (82%)	<0.000
57	Evidence that the carer was consulted during the first half of the patient's stay about planning for discharge	29 (31%)	37 (74%)	<0.000
58	Evidence that the carer's needs for skill training were assessed	7 (37%)	10 (71%)	0.05
59	Evidence that the skills required were taught	2 (13%)	8 (62%)	0.008

*Agreed at time of opening ASU that formal assessment of cognition within 24 hours (as required by RCPLSA tool) was inappropriate, so compliance discouraged.

†Based on very small numbers so statistical comparisons not valid.

Discussion

This audit has shown that important PoC for stroke patients improved following the introduction of an ASU. These benefits were additional to those already gained by having a SRU.[6,7] These results also indicate that changes made at the "front end" of stroke care can improve a wide range of processes throughout the whole inpatient stay. The gains were not limited to just one professional group's work but were across all disciplines.

By using shared protocols and documentation, together with close linkages between the units, this ASU complements the existing SRU and goes some way towards the goal of an overall coordinated stroke service.[4,18]

Previous studies from both New Zealand and overseas have shown significant deficits in stroke care.[19–22, 37] with inadequate assessment or treatment in acute phase, and poor attention to secondary prevention strategies on discharge. More recent studies have shown improvements in care processes,[23] clinician attitudes,[24] and (in the last 5 years) development of SUs in some District Health Boards (DHBs).[6,23,25,26]

This paper shows that with the development of an overall inpatient stroke service (with most patients under the care of a stroke specialist team), these PoC can be further improved.

Whilst the initial assessments have greatly improved (with exception of visual attention), further improvements are still required. There was a documented plan to manage hypertension in the longer term in only 75%, despite recent studies and guidelines advocating more aggressive blood pressure management [4,27]. Others have noted similar deficits in hypertension plans, although overaggressive management in some older frail patients may cause postural hypotension later.[23]

Whilst screening for swallowing difficulties improved between the cohorts, only ¾ of the after sample were screened within 24 hours of admission. Some of those who were not screened were not admitted to ASU. Thus we need to have alternative options to assess swallowing when patients are cared for in non-SU settings. Some processes for important, yet non-life-threatening consequences of stroke (continence, cognition, mood, and carer needs) have improved, but there remains considerable room for further improvement. Attention in these areas may improve quality of life for stroke patients.

The gains above have been achieved without extending length of stay (LOS) in hospital. The mean LOS in this study is similar, or lower, than comparable studies.[15,22,23,28–30] This is in keeping with international SU literature, where better quality outcomes are achieved with similar LOS.[1] The return home rate is also comparable or better.[23,25,31]

Our reported 30-day mortality (24–25%) appears high compared to New Zealand studies, but these have reported in-hospital mortality only.[15,22,23,25] Our 30-day figures are comparable to those reported in the Auckland Stroke Study.[32]

There is not uniform agreement whether an improvement in documented PoC translates into better patient outcomes. McNaughton et al [22] found that there is a relationship between PoC and outcomes, but that this relationship is weak and complex. We agree with their comments on complexity. However their conclusions

on the strength of relationship were derived from audits using the older RCPL stroke audit tool.[20]This was heavily biased towards the medical management of stroke.

The newer version (used in this study)[16] is much broader and looks at many multidisciplinary processes over 12 key clinical domains. Each process was chosen because it was sensitive to variations in quality of care; was thought to be relevant to clinical outcomes; and (where possible) were based on the UK guidelines for stroke.[33]

Kwan[11] found that improvements in quality of documentation and PoC were associated with fewer complications. In two recent studies, greater adherence to quality PoCs were associated with improved patient outcomes (reduced complications or mortality, and trend to improved independence at home).[34,35] Similarly better performance on process measures was strongly associated with better survival amongst community dwelling older adults.[36]

Some secondary prevention PoC (such as antiplatelet therapy and blood pressure management) have long-term beneficial outcomes,[27] but these are hidden when measuring short term functional or domicile outcomes. Thus we believe that the improved PoC demonstrated here will have a very positive longer term impact on the quality and consistency of care given to our stroke patients.

The development of an ASU at Christchurch Hospital occurred within a larger context, with a plan to develop an overall stroke service.[4] A pilot community-based stroke rehabilitation team commenced in March 2006, and is the third (and equally important) component of such a service.

ASUs should not occur in isolation and need close links with rehabilitation, community, vascular surgical, and general medical services. This will ensure the patient gets the appropriate care at the right time and transitions through the various stages of their illness with minimal disruption.[1,18]

What has made the difference? Was it just better documentation? This may be a partial answer as each discipline has initial assessment proforma, which may have prompted better recording. Consistency of care given by staff (with an interest and expertise in stroke) in a SU is another critical component.

During the planning and implementation of the ASU, considerable emphasis was placed on developing all of the key elements of a successful SU.[13,37] These elements include acute assessment procedures; early management policies (not just medical treatment); rehabilitation (including coordination of multidisciplinary team (MDT) care, regular carer involvement, and MDT meetings); and building staff expertise with regular education and training.

Patients with stroke were previously managed on many different wards, with varying degrees of expertise and interest. Our inclusive admission policy has increased the numbers of patients who were cared for in a SU environment (RCPLSA data 69%, but approximately 88% from discharge coding data).

During the implementation phase there was some pressure to develop a clinical care pathway for patients with acute stroke. This was resisted, as evidence for a strict pathway is equivocal.[11,30,38,39] The team also resisted the concept of a mobile team of stroke experts—as this has been shown to be an inferior model to a geographically distinct SU.[3]

Instead, our team focused on a geographically distinct SU concept together with development of a stroke proforma, shared documentation (and protocols) across disciplines and units, and ensuring that all the key elements of a SU were developed.[12,13,35] Such an approach allows the staff to use their expertise and experience to make judgements, without the tight confines of a care pathway.

Using a historical control group in a before and after study has limitations. Many different processes may have changed, rather than just the intervention being tested. In this case, the intervention is not just the setting up of an ASU, but also the change in documentation and ethos of all stroke care. The magnitude of the changes found, suggests that the ASU and associated changes in PoC have made a significant impact over and above other background changes.

Whilst the audits were done at a similar time of year for both cohorts, the pre-ASU audit did include the Christmas holiday break, which could potentially have reduced therapist availability for this cohort. However any negative effect from this is likely to be mitigated by the larger number included in this cohort (N=119). The assumption that what is documented reflects the PoC delivered has already been discussed earlier.

It is disappointing that we have not been able to show improved patient outcomes in this study. Like the profitability of any new business, we expect these improved PoCs to translate into better outcomes over time. However, the finding of similar clinical outcomes despite the post-ASU cohort having more severe strokes (and hence more challenging to rehabilitate and successfully discharge) is encouraging.

The reason for this change in casemix is not clear. Whilst the sampling was non random, sample sizes were considerably larger than those used for each trust in the UK National Sentinel Audit.[33] A trend to increased ambulatory care for patients with TIA or minor stroke may be another factor with primary care access to DHB funded CT scanning for stroke now available in Canterbury. It is feasible that subtle drowsiness is better recognised in the ASU and hence lower levels of consciousness are now being recorded. This might account for an apparent worsening in casemix, but a similar explanation would not account for the worsened continence status.

Setting up the ASU was challenging. One problem was the perceived heaviness or dependency of acute stroke patients and hence the ASU was not seen as a desirable place for nurses to work.[25] A counter to this was giving nurses greater roles and a chance to specialise, as well as the strong sense of teamwork that developed. Other clinical staff held the view of "Why do we need a SU? We all look after stroke patients and do it well".

The pre-ASU audit shows that PoCs were not done well and needed to be improved. Furthermore the literature is clear that SU care is superior to general ward care,[1,2,4] and patients prefer SUs.[40]

Changes in service delivery in a large organisation are slow and difficult to achieve. This ASU is no exception to that, taking at least 5 years from conception to fruition.[8] Fiscal restrictions were real, but the set-up costs are small compared to the much larger costs of poor stroke outcomes such as institutional care.[41].

In summary, adding an ASU to complement an existing SRU gave major improvements in PoC across many different facets of stroke care. We believe this is

one step closer to the ideals of an overall coordinated stroke service, as recommended in stroke guidelines, and better care for patients with stroke.

Conflict of interest statement: All authors were involved in the set up of, and/or continue to work in, the Acute Stroke Unit.

Author information: H Carl Hanger[1]; Valerie Fletcher[1,2]; John Fink[3]; Andrew Sidwell[1,2]; Anne Roche[1,2]

1. Geriatrician, Older Persons Health, The Princess Margaret Hospital, Christchurch.
2. Physician, Department of General Medicine, Christchurch Hospital, Christchurch
3. Neurologist/Senior Lecturer, Christchurch School of Medicine and Health Sciences, University of Otago, Christchurch.

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Correspondence: Dr H Carl Hanger, Older Persons Health, The Princess Margaret Hospital, PO Box 800, Christchurch. Fax: (03) 3377823; email: carl.hanger@cdhb.govt.nz

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Snakes alive! Caput medusae due to cerebral venous angioma

Hu Liang Low, Brian Simpson

The increasing sophistication and availability of modern cranial imaging techniques has resulted in the detection of incidental lesions in greater numbers. Caution needs to be exercised when deciding whether these lesions are responsible for the patient's symptoms and whether treatment is merited. We present a case where two coincident brain lesions led to a referral for possible neurological surgery.

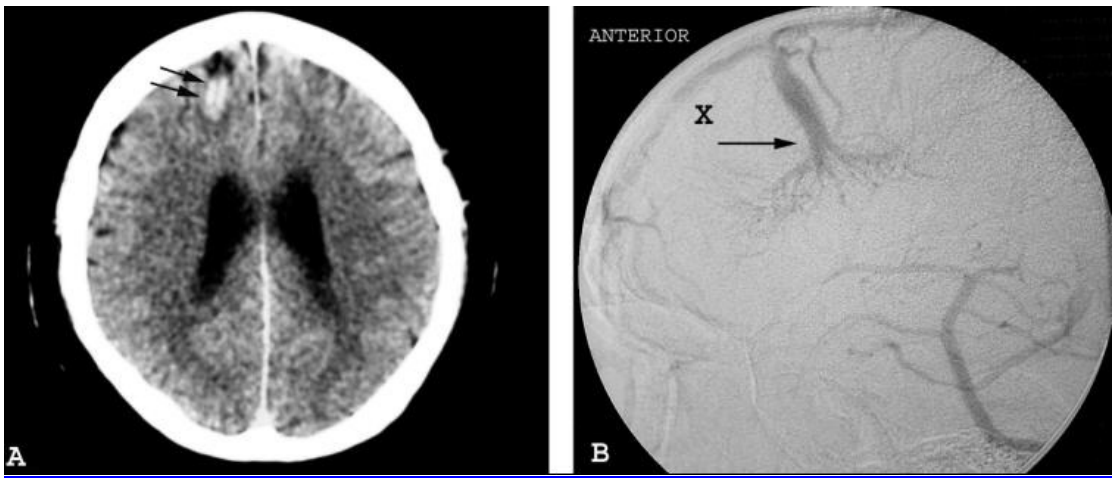
Case report

A 45-year-old man presented to the emergency department of a Welsh district hospital with a 1-week history of persisting headaches and dizziness after hitting the top of the right side of his head forcefully against an overhead lamp. On examination, no abnormal neurological findings were found.

In view of his lingering symptoms, an un-enhanced computer tomographic (CT) scan of his brain was performed. This showed a small, superficial right-frontal haematoma (Figure 1). He underwent a magnetic resonance imaging (MRI) scan which was unremarkable except for the haematoma. The patient subsequently had a digital subtraction cerebral angiogram which revealed a leash of vessels converging onto a dilated central vessel which drained into the superior sagittal sinus (Figure 2).

Figure 1. Un-enhanced computed tomographic scan of brain showing the superficial right-frontal haematoma.

Figure 2. Digital subtraction cerebral angiogram during the venous phase. The arrow points to the 'caput medusa' of the cerebral venous angioma. The 'X' marks the position of the intracerebral haematoma relative to the venous anomaly.



At this stage, the patient was referred to the neurosurgical team for consideration of surgery. When we reviewed the angiograms we found that the vascular lesion was consistent with a cerebral venous angioma. Moreover, this anomaly was distal to the location of the haematoma. There were no other vascular abnormalities. The frontal haematoma was a contusion secondary to the patient's head injury. He was treated conservatively and made a full recovery.

Discussion

The actual prevalence of cerebral venous angiomas (CVA) is probably higher than the quoted 3% of the population[1]—as this figure has been derived from brain imaging and autopsy studies.

CVAs are developmental anomalies composed entirely of venous structures interspersed with normal brain parenchyma and are found mainly in the posterior fossa, cerebral cortex, and the deep white matter.[2] The pathogenesis of CVAs is unknown but may represent attempts at establishing a collateral venous circulation following an ischaemic insult during the development of medullary veins and their tributaries.[3] CVAs therefore drain normal brain tissue.

CVAs are usually discovered incidentally during investigations for unrelated neurological disorders. They have a characteristic angiographic appearance which has been likened to the head of the Medusa (caput medusae).[2] They appear as stellate structures on contrast-enhanced MRI scans unless obscured by the presence of haematoma.[2] The incidence of second cerebrovascular malformations in patients with known CVAs is as high as 19%.[4]

The majority of these coincident vascular malformations are cavernous haemangiomas and there is a possibility that the development of both these malformations may be related.[5] CVAs had previously been associated with various neurological symptoms and haemorrhage. However, there is increasing evidence to suggest that venous angiomas rarely, if ever, bleed.[2,4]

Most recent studies suggest that neurological symptoms attributed to CVAs (especially haemorrhage) are probably due to associated cavernous malformations, arteriovenous fistulas, or unidentified vascular malformations.[2,4]

Patients found to have a CVA should ideally have at least an MRI to exclude concurrent vascular malformations as these are more likely to give rise to symptoms. Due to the low morbidity associated with bleeding (if it was actually due to a CVA) and the high complication rates when these lesions are treated with either surgery or radiosurgery, the majority of neurosurgeons now treat cerebral venous angiomas conservatively.[2,4]

Author information: Hu Liang Low, Fellow in Stereotactic and Functional Neurosurgery, University of British Columbia; Vancouver, Canada; Brian A Simpson, Consultant Neurosurgeon, University Hospital of Wales, Cardiff, UK

Correspondence: Dr HL Low, Surgical Centre for Movement Disorders, 8105-2775 Laurel Street, Vancouver, British Columbia V5Z 1M9, Canada. Fax: +1 604 8754882; email: lowhli@hotmail.com

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Beware of infants with respiratory distress, rash, and hepatomegaly at birth: a case of congenital syphilis

Malcolm Battin, Lesley Voss

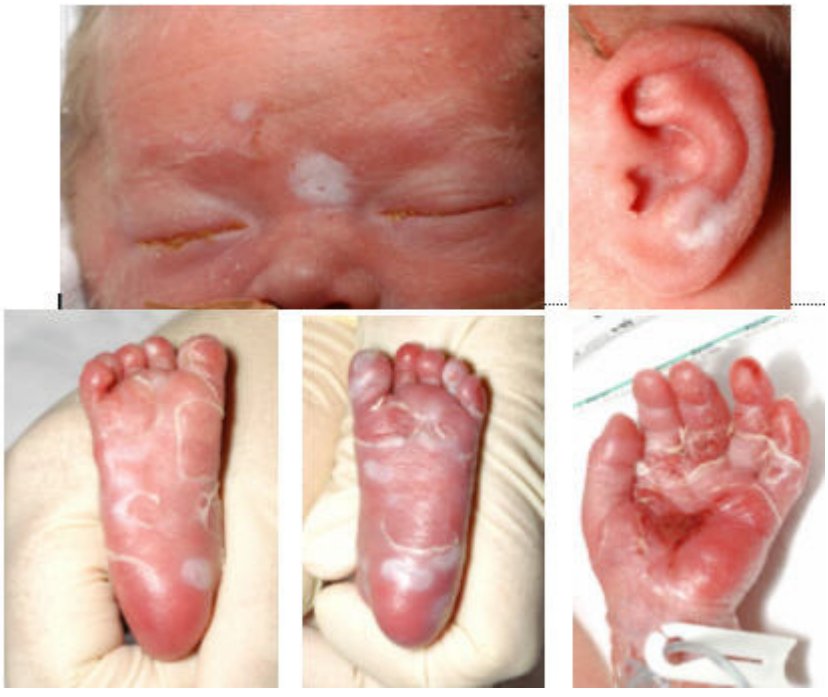
Pregnant women with syphilis may be asymptomatic, hence identification is dependent on serological screening. Despite New Zealand (NZ) having a longstanding policy of such screening at pregnancy booking, congenital syphilis still occurs.

Case report

An 1800-gram infant was born to a 21-year-old NZ European woman, with an unrecognised pregnancy who had no antenatal care. This was her second pregnancy; the first resulted in a live spontaneous vaginal birth at term. Spontaneous onset of labour occurred at 32 weeks' gestation, based on approximate last menstrual period (LMP), with rapid delivery shortly thereafter.

A live male infant was born in the ambulance, with an initial cry and some respiratory effort. He deteriorated quickly despite receiving bag-mask positive-pressure ventilation and chest compressions in the ambulance. On arrival at the hospital he was cold and limp with a heart rate of 40 bpm—but responded to intubation, positive-pressure ventilation, and cardiac massage. Once stabilised, he was transferred to the neonatal unit.

On admission examination, there were multiple, punched out, pale, blistered lesions mainly on peripheries but also on ears and bridge of nose and associated desquamation of palms and plantar surfaces of the feet (Figure).



Additionally, there was meconium staining of the skin and marked hepatomegaly. The initial chest radiograph was consistent with a congenital pneumonia and the diagnosis of congenital sepsis with pneumonia was made. Antibiotics were commenced with amoxycillin and gentamicin in standard neonatal doses. Blood cultures were negative as were viral cultures of both stool and nasopharyngeal aspirate. Swabs from the ear, lesions, gastric contents and placenta did not reveal any pathogen.

However, the diagnosis of congenital syphilis was confirmed with positive venereal disease research laboratory (VDRL) screen and reactive rapid plasma regain (RPR) and *Treponema pallidum* particle agglutination assay (TPPA) tests. Maternal serology was also positive, with RPR and TPPA reactive. Examination of the infant's cerebrospinal fluid (CSF) revealed VDRL was reactive, red cells 244×10^6/L, white cells 17×10^6/L (polymorphs 5% lymphocytes 58% monocytes 37%), elevated protein 1.47 g/L (0.15–0.45 g/L), glucose: 3.1 mmol/L (2.8–4.4 mmol/L), with no growth on culture. Subsequent cerebral ultrasound scans were within normal limits. He completed 12 days of parenteral penicillin G.

The neonatal course included pulmonary hypertension treated with positive pressure ventilation, two doses of surfactant, and nitric oxide for 3 days— the infant recovered well. The family were referred to the adult infectious diseases and sexual health teams, and the infant to the paediatric infectious diseases (ID) team for follow-up.

Discussion

A resurgence in syphilis has been documented in a number of developed countries in recent years.[1–3] In NZ, a recent paper suggests an increase in the number of people being diagnosed with syphilis at sexual health services.[4] An apparent increase was seen in heterosexual cases including two women found to be positive on antenatal screening.

Antenatal screening for syphilis is an effective method of identifying women for treatment to prevent the birth of an infected child. However, it does have some limitations. Firstly, it will not identify women who acquire syphilis during later stages of pregnancy after the antenatal screening has been performed or those incubating disease at initial testing.[5] Secondly, as in our case, when the woman does not have antenatal bloods performed, the potential for treatment in pregnancy is lost. Thirdly, the clinician may have difficulties in interpreting abnormal syphilis serology resulting in either lack of follow up or suboptimal treatment.[6–8]

Although the VDRL/RPR is used as a screening test for syphilis, it is a nonspecific test and biological false positives do occur in a number of other conditions, including pregnancy. In the past, yaws has also caused confusion and difficulty interpreting syphilis serology, particularly in women from the Pacific. Therefore for any women with a positive syphilis screen in pregnancy, a specific syphilis test—TPPA/FTA-ABS (fluorescent treponemal antibody absorption test)—should be performed and appropriate treatment given if syphilis confirmed. If concerns remain at the time of delivery, then the neonate should be investigated.

Congenital syphilis can present with protean manifestations and lead to delayed diagnosis. Characteristic features include, rash (maculopapular or vesicular),

mucousal lesions, nasal discharge, hepatomegaly, bony tenderness, or eye lesions. The Hutchinson's triad, first described in 1858, describes the late findings of congenital syphilis of notched incisor teeth, interstitial keratitis, and eighth cranial nerve deafness. However children can be born with no obvious clinical manifestations initially, and the disease can present much later.

In the current era of rising rates of sexually transmitted diseases in NZ, it is important to check maternal serology and consider this disease in any child with suspicious clinical findings, particularly if antenatal screening has not occurred, but even if the initial pregnancy screening on the mother has been negative.

Author information: Malcolm Battin, Senior Lecturer (Neonatology), University of Auckland and National Women's Health, Auckland; Lesley Voss, Paediatrician, Infectious Diseases Services, Starship Children's Hospital, Auckland

Correspondence: Dr Malcolm Battin, Newborn Service, 9th Floor Support Building, Auckland City Hospital, Private Bag 92189, Auckland. Email: malcolmb@adhb.govt.nz

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Risk-taking: behind the warrior gene story

Tony Merriman, Vicky Cameron

Abstract

In 2006, the monoamine oxidase-A gene was widely reported in the media as being associated with risk-taking and aggressive behaviour in Māori. We examine the scientific evidence underlying this claim. Whilst there is credible evidence for a contribution of a monoamine oxidase-A genetic variant to antisocial behaviour in Caucasians, there is no direct evidence to support such an association in Māori. Insufficient rigour in interpreting and applying the relevant literature, and in generating new data, has (in conjunction with a lack of scientific investigative journalism) done science and Māori a disservice.

In the second week of August 2006, the media were gripped with the announcement that Māori men were genetically predisposed to "violence, criminal acts, and risky behaviour." (*Christchurch Press*, 9 August 2006). The basis for this release was the report of a study that purported to show that a genetic variant of the monoamine oxidase *(MAO)-A* gene, dubbed the "warrior gene" by the media, had previously been "strongly associated with risk taking and aggressive behaviour" and was "strikingly over-represented" in Māori men.[1]

Because these findings were released at an international conference by a government-funded researcher, there has generally been an assumption that the link between the *MAO-A* variant and aggression in Māori is based on robust scientific evidence. Here we place the scientific evidence under closer scrutiny.

The regulatory region of the human *MAO-A* gene has a genetic variation consisting of a small stretch of DNA repeated a variable number of times.[2] The 3-repeat form of this 'variable number of tandem repeats (VTNR)' polymorphism (present on 33% of Caucasian chromosomes) exhibits up to 10-fold less activity than the 4-repeat form (present in 65% of Caucasian), and has been commonly termed the 'low-activity' variant.[2]

The MAO-A gene controls the production of the MAO-A enzyme, which is involved in the breakdown of several neurotransmitters in the brain, such as dopamine and serotonin. It is believed that this enzyme is important in preventing the build-up of an excess of these transmitters. In *MAO-A* deficient mice that lack the enzyme altogether, increased levels of neurotransmitters were documented and the mice were observed to exhibit aggressive behaviour.[3]

In a Dutch family, a dysfunctional MAO-A gene has been linked to antisocial behaviour and increased aggression amongst humans.[4] Based on this observation, the *MAO-A VNTR* has been tested for association with antisocial behaviour in population-based male cohorts. The three largest studies (all in groups of Caucasian ethnicity) published remarkably consistent findings; no main effect was found for the relationship between the *MAO-A VNTR* genotype and antisocial behaviour, but the high-activity form was associated with buffering males who were abused and

neglected in childhood from increased risk of anti-social behaviour later in life.[5–7] That is, there was evidence for a gene-environment interaction where the effect of the *MAO-A* low-activity variant was dependent on the environment—analysed in isolation the gene variant could predict nothing about aggression in male carriers.

This effect was, however, not evident in a cohort of American non-whites.[7] A possible biological explanation of the genetic findings is that the higher-activity variant produces more MAO-A enzyme and is better able to mop up excess neurotransmitters, such as dopamine and serotonin, in the brain.

It is clear from the genetic data that *MAO-A* itself is not associated with aggression (contrary to the interpretation by Lea and colleagues of "strong" association with aggressive behaviour[1]). More generally, it is important to emphasise that the strong, clear, and direct causal relationship implied by the phrase of "a gene for…" does not exist for psychiatric disorders.[8]

The MAO-A gene was termed the "warrior gene" on the basis of experiments in Rhesus macaque monkeys.[9] These primates have a comparable VNTR upstream of the MAO-A gene with the 5- and 6-repeat forms having 1.3-fold greater activity than the longer 7-repeat form.[9] However, in contrast to the human gene, in the Rhesus macaque, fewer repeats means more MAO-A enzyme is produced.

When tested for association with aggressive behaviour in 45 male macaque monkeys there was no main effect of genotype on aggression. There was, however, evidence for a gene-environment interaction; in peer-reared monkeys, the higher-activity allele was associated with aggression, whereas in mother-reared monkeys the lower-activity variant was associated with aggression.[9]

This gene-environment interaction superficially conflicts with the human data, although there are numerous reasons (including species difference) that could account for this apparent conflict.[9] Nevertheless, on the basis of one un-replicated experiment on only 45 animals, the *MAO-A* gene was (dubiously) termed the "warrior gene" at the 2004 Annual Meeting of the American Association of Physical Anthropologists by a scientific journalist.[10]

As far as we are aware, the term "warrior gene" has not been applied to humans in previous studies of *MAO-A* and aggression. Dr Lea and colleagues reported that the frequency of the 3-repeat short form of the *MAO-A VNTR* was just over 60% in a very small sample of 17 Māori males.[1] Based on these data they concluded that "positive selection of *MAO-A* associated with risk taking and aggressive behaviour has occurred during the Polynesian migrations" and termed the low-activity variant the "warrior allele". The *MAO-A* gene was then linked to antisocial behaviour in contemporary Māori males in the publicity surrounding the conference presentation.

We believe that this conclusion is based on science with insufficient investigative rigor, both in interpreting and applying the relevant literature, and in generating new data.

Central to the argument of Dr Lea and colleagues[1] is the assumption that the low-activity *MAO-A* allele is associated with aggression in Māori males. This assumption cannot be made without the appropriate genetic epidemiological experiments being done to test for an association between *MAO-A* and aggression. However no such study has ever been reported.

The lack of evidence for involvement of this variant with violence and antisocial behaviour in American non-whites[7] demonstrates that extreme caution is needed when translating *MAO-A* genetic findings between racial groups. A central tenet of complex phenotype genetics is that genetic associations are likely to vary between racial groups.

There is no direct evidence to support the claim that the *MAO-A* gene confers "warrior" qualities on Māori males, either modern or ancestral. Furthermore, the assumption that a genetic association in Caucasian applies in Māori; the use of the "warrior gene" label in the context of human *MAO-A* aggression studies; generalising from a sample of 17 individuals not representative of the general Māori population; and the lack of scientific investigative journalism have combined to do science and Māori a disservice.

Conflict of interest statement: There are no conflicts of interest.

Author information: Tony Merriman, Senior Research Fellow, Department of Biochemistry, Otago School of Medical Sciences, University of Otago, Dunedin; Vicky Cameron, Research Associate Professor, Christchurch Cardioendocrine Research Group, Christchurch School of Medicine and Health Sciences (University of Otago), Christchurch

Correspondence: Dr Tony Merriman, Department of Biochemistry, Otago School of Medical Sciences, University of Otago, PO Box 56, Dunedin. Fax: (03) 479 7866; email: tony.merriman@stonebow.otago.ac.nz

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Warrior genes and risk-taking science

Peter Crampton, Chris Parkin

Abstract

This article provides a summary of our ethical concerns regarding the so-called "warrior gene" line of research. Prompted by recent claims that there is a genetic explanation for negative social and health statistics for Māori, the article discusses issues related to informed consent of research participants, the validity of the underlying science related to the "warrior gene", and scientifically unfounded speculation regarding the causality of complex social issues. We conclude that in all science, and particularly where there is a highly charged social and political setting, the scientist has a responsibility for the way in which findings are disseminated and for ensuring a clear public understanding of the limitations of the work.

This viewpoint article was prompted by recent claims by Dr Rod Lea that there is a genetic explanation for negative social and health statistics for Māori. Dr Lea's comments were widely reported in the New Zealand media including, for example, that the monoamine oxidase gene "goes a long way to explaining some of the problems Māori have. Obviously, this means they are going to be more aggressive and violent and more likely to get involved in risk-taking behaviour like gambling."[1]

The claims were made on the back of a small-scale, as yet unpublished research project. We have concerns about the veracity and ethics of such extravagant speculation regarding the causality of complex social issues. In this article, we provide a summary of our ethical concerns regarding the "warrior gene" line of research.

Decades of medical research have yielded embarrassingly recurrent examples of unethical science with various populations, for example: prisoners, POWs, children, the elderly, the intellectually handicapped, and, not least, many ethnic minorities.[2]

A widely accepted antidote has been the development of stringent independent ethical review, given its most powerful impetus in New Zealand by the Cartwright Report.[3] Ethical principles governing research involving human subjects bear largely on empowering individual participants and protecting them from risk. However, they also draw attention to the importance of ensuring that the science is good, that harm is minimised, and that the research is carried through with cultural and social responsibility.[4]

A critically important way in which individual research participants are protected is through the provision of informed consent: before taking part in research individuals are made fully aware of the purpose of the research and its potential harms and benefits. We are seeking reassurance from Dr Lea that the participants in the research, and the research ethics committee, were aware that the research included the exploration of hypotheses linking the "warrior gene" with violent and antisocial behaviour, and that generalisations were to be made from the research participants to

the entire Māori population despite the lack of evidence for association between the two (see below).

Good science conforms with canons of rigorous enquiry in striving to make non-trivial additions to knowledge, and hence becomes "risk-taking" when its authority is given to views with questionable credentials. New truths are often hard won. Their validity turns on the extent and the quality of the empirical data garnered to support them and on the coherence of the theoretical framework used to make sense of them.

The empirical data thus far indicate the presence of the MAO-A gene in just over 60% of a (presumably non-random) sample of N=17. Simply to extrapolate from that not just to a contemporary Māori population of several hundred thousand but also to past generations of Māori back to the migrations, is risky in the extreme.

A missing keystone in Dr Lea's case for involvement of the MAO-A gene in antisocial behaviour in Māori is the lack of evidence for association between the two (see accompanying article by Merriman and Cameron; *Risk-taking: behind the warrior gene story*; http://www.nzma.org.nz/journal/120-1250/2440).

In Caucasian (but not non-white Americans) the gene is associated with antisocial behaviour in males only against a background of prior maltreatment. However no association is apparent when the gene is examined in isolation. Even if the MAO-A data in Caucasian were to be replicated in Māori males, that does not make the gene the cause of the behaviour. Its presence might predispose towards such behaviour but only in the context of a disadvantageous environment. To make the causal claim on the evidence of association alone is naïve. In this case, the naïvety is masked by the positive public stereotype of the cutting edge scientist reporting a breakthrough.

Of special concern is the fact that Dr Lea has made extravagant claims concerning the "warrior gene" in Māori despite having himself at times cautioned against risking the naïve leap to a simplistic causal connection.

On National Radio's *Morning Report* he observed:

> "This gene has been linked to different anti-social and risk-taking behaviours, but the link has usually been quite weak, and often is only present in association with non-genetic factors—that is, other factors such as upbringing, socioeconomic circumstances, other lifestyle factors."[5]

Putting the spotlight on the "warrior gene", whose presence by implication we cannot do much about, is to that extent a harm which can and ought to be minimised. It is harmful because it risks diverting attention from social and economic conditions which, by contrast, are amenable to change, no matter how challenging the processes of policy development needed to effect sustained improvement.

The popular rhetoric of a "warrior gene" offers what appears to be a 'simple' explanation, almost certainly to be seized upon as such, irrespective of its proponents' caveats. This harm is likely to have been amplified by the very high level of media interest following the death of the Kahui twins and the generally negative portrayal of Māori in the media.

In such highly charged social and political settings, the scientist has a particular responsibility for the way in which findings are disseminated and for ensuring a clear public understanding of the limitations of the work to date.

> A wise man, therefore, proportions his belief to the evidence.[6]

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Author information: Peter Crampton, Head of Department, Department of Public Health**;** Chris Parkin, Senior Teaching Fellow (Medical Ethics), Department of Primary Health Care and General Practice; Wellington School of Medicine and Health Sciences, University of Otago, Wellington

Correspondence: Professor Peter Crampton, Head of Department, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, Wellington. Fax (04) 389 5319**;** email: peter.crampton@otago.ac.nz

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The Health and Safety in Employment Act and the influenza vaccination of healthcare workers

Stuart McLennan, Leo Anthony Celi, Paul Roth

Abstract

Despite studies demonstrating that the annual influenza vaccination of healthcare workers has a statistically significant reduction of morbidity and mortality among the patients they care for, and District Health Boards (DHBs) establishing voluntary programs to provide the influenza vaccine to healthcare workers free of charge, vaccination rates among healthcare workers are dismal, with only about 20%–40% coverage rates being achieved.

With these low rates posing a serious health threat to the vulnerable patient populations that are entrusted into healthcare workers' care, and the current voluntary programmes clearly failing to adequately address this issue, we believe the time has come for the annual influenza vaccination to be made a mandatory requirement for all healthcare workers with direct patient contact unless a medical contraindication exists.

Indeed, a compelling case may be made that the duties imposed on DHBs and healthcare workers under the Health and Safety in Employment Act 1992 requires making the annual influenza vaccination an occupational requirement.

The Health and Safety in Employment Act 1992

The objective of the Health and Safety in Employment Act 1992 (hereafter "the HSE Act") is to promote the prevention of harm to all people at work, and others in, or in the vicinity of, places of work. The HSE Act applies to all New Zealand workplaces, and places duties on employers, employees, and others who are in a position to manage or control hazards.

The emphasis of the HSE Act is on the systematic management of health and safety at work. It requires employers and others to maintain safe working environments, and to implement sound practice.

The HSE Act imposes a general duty on employers to take all practicable steps to ensure the safety of employees while at work. It also imposes specific duties on them to take all practicable steps to eliminate every "significant hazard", which is defined in section 2 of the HSE Act as meaning a hazard that is an actual or potential cause or source of, inter alia, "serious harm", which in turn is defined in the First Schedule to the HSE Act as including "communicable disease".

Employers also have duties to people who are not employees under section 15 of the HSE Act, which states, "every employer shall take all practicable steps to ensure that no action or inaction of any employee while at work harms any other person."

Employees themselves have similar duties under section 19, which states "every employee shall take all practicable steps to ensure…(b) that no action or inaction of the employee while at work causes harm to any other person".

Influenza

Influenza is a serious health issue worldwide, causing significant morbidity and mortality annually.[1] However, the implications of an influenza infection are not the same for everyone. Influenza infection is particularly dangerous for the vulnerable patient populations congregated in healthcare facilities—notably the elderly, the immunocompromised, the critically ill, and young children.[2–5] Influenza infection in these populations can often result in severe, prolonged, devastating illness, and death.[4]

Healthcare workers are at risk for occupational exposure to (and subsequent illness from) influenza due to the close contact with patients who may have influenza. Healthcare workers may also act as potential vectors for nosocomial transmission of influenza to their vulnerable patients whom the disease would most jeopardise.[6,7]

Nosocomial spread of influenza involving healthcare workers has been shown in many healthcare facilities, including long-term care facilities, oncology units, transplant units, neonatal intensive care units, general medical and paediatric wards, emergency departments, and other facilities.[5,8,9]

The risk of nosocomial spread of influenza to patients and co-workers is compounded by the tendency of healthcare workers to continue working despite being ill with influenza.[10–12] At any rate, the influenza virus can be transmitted to patients and co-workers by both symptomatic and asymptomatic healthcare workers, hence, simply staying home from work is an insufficient strategy for preventing nosocomial transmission of influenza.[13,14]

The influenza virus may be shed for at least 1 day prior to significant symptomatic illness.[15] Moreover, only an average of 50% of people develop classic symptoms of the illness, yet can shed the virus for 5–10 days.[16,17]

Influenza vaccine

The most efficient means of preventing a significant number of influenza infections, and the resulting morbidity and mortality, is by using an annual pre-exposure vaccination. Inactivated influenza vaccine is effective in healthy people under 65 years of age, and provides 70%–90% protection against influenza infection.[10,18] However, the immunologic response to the influenza vaccine is not as effective among the older population, particularly those with chronic illness,[19] and not everyone can be vaccinated.

These populations need the additional protection provided by the well-established concept of ring vaccination, which is the basis for the recommendation to vaccinate household members and caregivers of these populations.

The influenza vaccination is also safe, with the only common side effect being minor injection site soreness for 1–2 days.[7] Systemic effects are no more common than they are with placebos.[20]

Severe reactions are very rare. In 1967, the swine influenza vaccine was associated with a slight increase in Guillain-Barré syndrome; however, studies during subsequent

years have not documented a clear increase in incidence associated with receipt of the influenza vaccine.[16]

Despite studies demonstrating that the annual influenza vaccination of healthcare workers has a statistically significant reduction of morbidity and mortality among the patients they care for,[21,22] and New Zealand District Health Boards establishing voluntary programs to provide the influenza vaccine to healthcare workers free of charge, vaccination rates among New Zealand healthcare workers are dismal, with only about 20%-40% coverage rates being achieved.[23]

A survey of the district health boards in 2006 revealed an uptake of 19–30% among nurses, 26-49% among doctors, and 27-52% among allied health personnel (Jo Stodart, Infection Control Practitioner, Otago District Health Board, Personal Communication; 2006).

Multiple studies have been conducted worldwide to examine the reasons why healthcare workers do not currently receive the influenza vaccination. These include concerns about adverse reactions, perceived lack of susceptibility, and alleged lack of vaccine effectiveness.[6,11,24–26] Anecdotal reports in New Zealand suggest that these findings also hold in the New Zealand context.[23] These concerns of healthcare workers show insufficient knowledge about influenza and the influenza vaccination, and act as a significant barrier to greater vaccination rates.[6,27]

Discussion

The current low rates of influenza vaccination among healthcare workers pose a serious health threat to the vulnerable patient populations that are entrusted into their care. It is clear that the current voluntary programs established by DHBs have failed to adequately address this issue. While some have advocated educational strategies to improve these rates,[23] we believe the time has come for the annual influenza vaccination to be made a mandatory requirement for all healthcare workers with direct patient contact unless a medical contraindication exists.

Indeed, a compelling case may be made that the HSE Act requires making the annual influenza vaccination an occupational requirement. DHBs have a duty under the HSE Act to take all practicable steps to eliminate every significant hazard, and a duty to take all practicable steps to ensure that no action or inaction of any employee while at work harms any other person. Likewise, healthcare workers have a duty to take all practicable steps to ensure that no action or inaction by them while at work causes harm to any other person.

The fact that the majority of healthcare workers fail to be vaccinated against influenza constitutes a “significant hazard” under the HSE Act. This action by healthcare workers—of exposing themselves in an unvaccinated state to patients, co-workers, and others in the workplace—also threatens to harm the vulnerable patient populations in healthcare facilities.

DHBs making the annual influenza vaccination an occupation requirement—in order to eliminate this “significant hazard”, and to minimise the chance of the vulnerable patient populations being harmed by nosocomial influenza—is a reasonably practicable step for DHBs to take in the circumstances.

At present, there is no case law directly on point. However, a close analogy can be drawn with employee compliance with drug testing regimes in the workplace where safety is involved. In *NZ Amalgamated Engineering ect Union Inc v Air New Zealand* (AC 22/04, 13 April 2004), the Employment Court considered the legality of a proposal by an employer to introduce a drug testing regime into safety sensitive areas of its operation. The employer sought to justify this proposal in part on the employer's duty to provide and maintain a safe workplace in terms of the HSE Act. In relation to employee obligations under the HSE Act, the Court observed that:

> … [Section] 19 of the Act, as amended in 2002, provides that every employee must take all practicable steps to ensure the employee's safety while at work, and that no action or inaction of the employee while at work causes harm to any other person. This strongly suggests, although it is a question of degree, that employees in occupations which impinge upon the safety of other persons, must see to it that they come to work substantially (perhaps, depending on the work, completely) free from the influence of alcohol or drugs. Because of this duty, they must expect to co-operate with the employer's attempts to monitor the situation. It is no different in principle to the statutory duty imposed on employees to wear protective clothing and to use protective equipment as well as a duty on employers to ensure that they do. (para 45)

Adapting this reasoning to influenza vaccinations, employees should have a legal obligation not to come to work where they pose a potential but easily remediable health hazard to others. Just as workers in safety sensitive occupations have an obligation not to come to work substantially impaired by drugs or alcohol, so too do workers in the health sector have a positive obligation to get vaccinated against influenza so that by coming to work, they do not pose a health hazard to other people, particularly those whose state of health has already been weakened.

Conflict of interest statement: There are no conflicts of interest.

Author information: Stuart McLennan, Masters Student, Bioethics Centre, Dunedin School of Medicine, University of Otago, Dunedin; Leo Anthony Celi, Specialist in Infectious Diseases and Intensive Care, Department of Medicine, Dunedin Hospital, Dunedin; Paul Roth, Professor of Law, Faculty of Law, University of Otago, Dunedin

Correspondence: Stuart McLennan, Bioethics Centre, Dunedin School of Medicine, University of Otago, PO Box 913, Dunedin. Email: stumclennan@hotmail.com

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Presidential Address: Richard John Seddon tribute and fake doctors under fire

Published in the N Z Med J 1907;5(22):43–57.

Since our last annual meeting, death has taken from our midst two dominant personalities whom this colony could ill afford to lose—The Hon. Richard John Seddon, whose zealous and self-sacrificing work for the welfare of the people included much that was of interest to the medical profession, and Dr. Duncan McGregor, the late, Inspector of Hospitals, in whom the highest scholastic attainments were combined in uncommon degree with shrewd practical knowledge of men and things. The medical profession lament the loss of these strong, brave men, and sincerely sympathise with their sorrowiug relatives.

The title of my address this evening is—
THE MEDICAL SERVICE OF NEW ZEALAND.

Being a review and criticism of the way in which the people of this colony are catered for, in regard to the prevention and cure of the various ills that flesh is heir to.

The duly qualified physicians and surgeons at present on the New Zealand Register, and who are actually practising in the colony, number roughly 600. The population of New Zealand is in round numbers 960,000, so that we have here one doctor to about every 1,600 of the inhabitants. A glance through the Register of the practitioners, will indicate that New Zealand now possesses a medical service decidedly ample in quantity, and on the whole, sound in quality.

By a recent act of Parliament, the portals of registration in New Zealand have been very properly narrowed. No one can now register as a medical practitioner who has not received the diploma of a school giving a complete modern medical education.

The curriculum of a modern medical school necessitates a minimum of five years of diligent study of the theory and practice of the various departments of medicine, and a large proportion of' the practitioners spend six or seven years in thoroughly equipping themselves for their profession. It is fair then, that unregistered practitioners, men with no proper medical education, men without genuine diplomas, or with diplomas granted for financial consideration by some irresponsible body of charlatans, or puesdo [sic] scientists, is it fair that such men should be permitted to call themselves doctors, to delude the public into thinking that they are medical doctors, and to enter unjustly into competition with registered medical practitioners who have been obliged to spend so much time and so much money on education.

I am not speaking just at this moment of the ordinary quack. He comes under a different category, and I shall refer to him presently. He does little or no harm to the duly qualified medical practitioner. But the man who poses before the public as a qualified medical practitioner, when he is absolutely unregisterable as such, enters unfairly into competition with doctors; and the medical profession should demand that so monstrous a wrong to them and to the public should no longer be tolerated.

In the present state of the law there is no protection from this sort of thing. Actually the names of unqualified, unregistered individuals are printed on the telephone subscribers list, on the first page, amongst the doctors. The Council of the New Zealand Branch recently called the attention of the Postmaster-general and Minister of Telegraphs to this impropriety, and politely requested that the names of unregistered men should not be printed on the first page of the telephone list with the regular qualified practitioners. The Postmaster-general, after due consideration, replied that he is not prepared at present, to exclude the names of unregistered men from the telephone list of medical practitioners.

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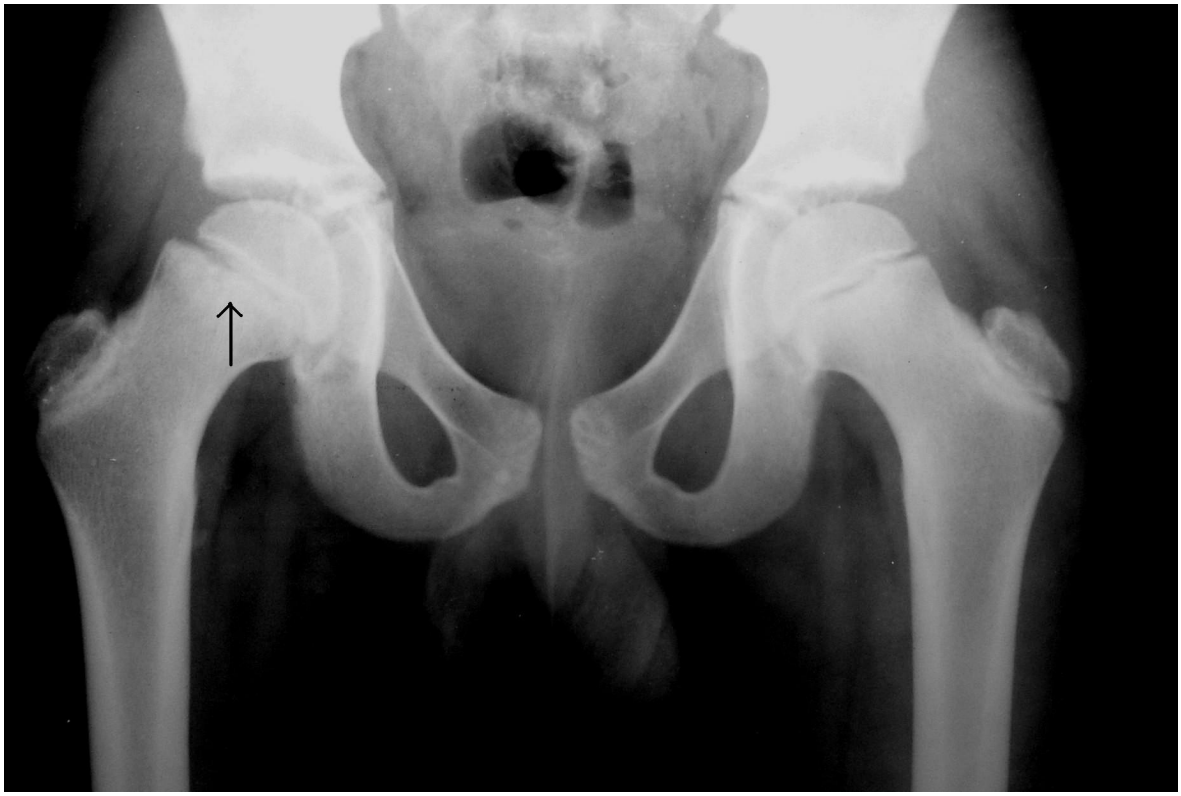
A boy with a limp

Jubbin Jacob, Thomas Paul

A 15-year-old boy was evaluated for short stature and increasing weight gain. On the basis of an elevated thyroid stimulating hormone (TSH) value, palpable thyroid gland, and presence of thyroid antibodies in significant titres, he was diagnosed to have juvenile hypothyroidism secondary to autoimmune thyroiditis. He was started on replacement levothyroxine therapy at a dose of 700 mcg/week/m^2 of the calculated body surface area.

Three months later, the boy was brought to the hospital with complaints of a limp associated with pain on bearing weight on the right hip. On re-examining his blood, he had normal TSH values, and he was compliant with his medications. An X-ray of the hip was obtained. (Figure 1).

Figure 1



What is the diagnosis?

Diagnosis and discussion

Analysis of anteroposterior radiographs demonstrates a medioinferior slipping of the capital femoral epiphyses and an increase in the width of the growth plate and articular cartilage. This is consistent with the diagnosis of a *slipped capital femoral epiphysis* (SCFE).

SCFE is defined as a posterior and inferior slippage of the proximal femoral epiphysis on the metaphysis (femoral neck), occurring through the physeal plate during the early adolescent growth spurt. It is the most common hip disorder in adolescents and the aetiology is thought to be a combination of endocrine and biomechanical factors.[1] The diagnosis may be missed because occasionally the pain is localised to the knee or the groin. This delay results in a less favourable long-term prognosis.[2]

The diagnosis of SCFE should always be considered when a physician is assessing the causes of limp and/or hip, thigh, and knee pain in children.

There are many theories as to the cause of SCFE. Most likely, SCFE is caused by multiple factors including local trauma, obesity overcoming the physeal plate (growth plate), inflammatory factors, and possible endocrine abnormalities (increased incidence seen in hypothyroidism, panhypopituitarism, renal osteodystrophy).

Obesity seems to be the strongest risk factor for SCFE, and in this case it is believed that the child's increased weight caused excessive mechanical stress on the physis (growth plate).[3]

Author information: Jubbin J Jacob, Senior Registrar; Thomas V Paul, Consultant; Department of Endocrinology, Christian Medical College and Hospital, Vellore, Tamil Nadu, India

Correspondence: Jubbin Jagan Jacob, Senior Registrar, Department of Endocrinology, Christian Medical College Vellore, Tamil Nadu, India 632004. Fax: +91 461 5203570; email: jjacob@cmcvellore.ac.in

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George Bernard Shaw and "the doctor's dilemma"

George Bernard Shaw in the preface to this play savagely attacked the medical profession for its direct personal and pecuniary interest in the treatment of patients and argued that doctors could not be trusted to act in their patients' best interests.

In an interesting editorial, Martin Van Der Weyden, the editor of the *Medical Journal of Australia*, speculates on what George Bernard Shaw would think of doctors now. As a prominent Fabian, Shaw would enthusiastically endorse Australia's Medicare scheme and other similar national health services. He would be interested to know that Australia spends 9.8% of its gross domestic product (GDP) each year on health—much more than New Zealand and much less than the United States. And there are other points to ponder, evidence-based medicine, the impact of the pharmaceutical companies, etc, etc.

The problems may be different but doctors still have dilemmas.

Med J Aust 2006;185:585–6

Intravenous versus oral antibiotics in severe community acquired pneumonia

When patients are first admitted to hospital with community acquired pneumonia, antibiotics are usually given intravenously to provide optimal concentrations in the tissues. The question then arises—when is it appropriate to change to the oral route?

In this trial from Holland, patients were given 3 days of treatment with intravenous antibiotics, followed (when clinically stable) by oral antibiotics or by 7 days of intravenous antibiotics. Their conclusion was that early switch from intravenous to oral antibiotics in patients with community acquired pneumonia is safe and decreases length of hospital stay by 2 days. Patients excluded were those sick enough to be admitted to Intensive Care Units.

The accompanying editorial (written by two New Zealand clinicians) applauds the study but noted that the trial used overly conservative discharge practices, and it would have been safe to shorten length of stay further for most patients. One would have to say also that 7 days of intravenous antibiotics as the reference arm is rather heavy-handed.

BMJ 2006;333:1181–2 & 1193–5

Hip fracture and long-term proton pump inhibitor therapy?

Clinicians are aware that the use of proton pump inhibitors (PPIs) has revolutionized the management of acid-related diseases such as gastroesophageal reflux disease (GERD)—or GORD as we prefer to call it.

Apparently there is some evidence that PPI therapy may decrease insoluble calcium absorption or bone density. On the other hand, there is evidence that omeprazole may decrease bone resorption by inhibiting osteoclastic activity.

Hence this nested case-control study which evaluated the incidence of hip fracture in subjects older than 50 years in relationship to their usage or non-usage of PPI therapy. They report that the adjusted odds ratio (AOR) for hip fracture associated with more than 1 year of PPI therapy was 1.44 rising to 1.59 after 4 years of treatment. In view of their findings they recommend clinicians to use the lowest effective dose for patients with appropriate indications in the elderly.

JAMA 2006;296:2947–53

Persistent middle-ear effusion and developmental impairment?

Developmental impairment in children have been attributed to persistent middle-ear effusion in their early years of life.

Myringotomy with insertion of tympanostomy tubes in order to clear the effusion and restore hearing acuity to a normal level has been claimed to be the solution; this view has been disputed by a collaboration of otolaryngologists in the USA who have published on the topic over the last 5 years.

In their latest study of a cohort of 6350 infants, they noted that before 3 years of age, 429 children with persistent effusion were randomly assigned to undergo the insertion of tympanostomy tubes either promptly or up to 9 months later if effusion persisted. Subsequently, using 48 developmental measures, they assessed literacy, attention, social skills, and academic achievement in 391 of these children at 9 to 11 years of age. The group treated early did no better. So they recommend, in otherwise healthy children, that watchful waiting for at least 6 additional months when effusion is bilateral (and for at least 9 additional months when effusion is unilateral) is the preferred management option. An editorial commentator endorses their opinion.

N Engl Med 2007;356:248–61 & 300–2

Variation in gastrointestinal endoscopy reports

Gastrointestinal endoscopy has proven to be of great use in the diagnosis of upper and lower gastrointestinal disease. Non-gastroenterologists regard endoscopy reports as the gold standard, but how do gastroenterologists regard them?

In this study, all gastroscopies and colonoscopies performed in two UK teaching hospitals over a period of one year were audited to investigate whether endoscopic reporting of endoscopies and colonoscopies by different endoscopists is consistent.

Endoscopic videos of 1814 colonoscopies and 2127 gastroscopies were reviewed. Somewhat disconcertingly the frequency of reporting common diagnoses was variable and the differences between specialist endoscopists were highly significant, including for important conditions such as peptic ulceration (range 2–10%, $p=0.001$) and colonic polyps (16–45%, $p<0.0001$). And their solution—more emphasis on interpretation in training endoscopists.

Clin Med 2007;7:23–7

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Talking health, doing harm: New Zealand Government investment in industries that kill

The New Zealand Government's Superannuation Fund invests in companies that manufacture nuclear weapons and cluster bombs.[1] This situation brings New Zealand's contribution to international health into disrepute, and is completely incompatible with this country taking a leadership role in nuclear disarmament and opposition to weapons that cause indiscriminate harm to civilians (i.e. cluster bombs).

While New Zealand Disarmament and Arms Control Minister, Phil Goff, is asking for 'legally binding controls on the use of cluster munitions',[2] and while New Zealand is sending army personnel to Lebanon to help remove cluster bombs, profiting from their manufacture appears to erode the credibility of the Government, and our reputation as a peacekeeping nation.

Furthermore, the New Zealand Government also invests in tobacco companies via the NZ Superannuation Fund, the Government Superannuation Fund, the National Provident Fund, and The Earthquake Commission. There appears to be no real progress on this issue since it was revealed in December 2005,[3] except for the decision of the Accident Compensation Corporation to not invest in tobacco companies.[4]

New Zealand has ratified its membership of the Framework Convention on Tobacco Control. This requires the government to 'act to protect [public health] policies from commercial and other vested interests of the tobacco industry' (Article 5 (3)). We suggest that investing in the tobacco industry would appear to contradict this requirement.

For some commercial areas there may be difficulties in where exactly to draw the line with ethical investment, but the approaches of the Norway Pension Fund provide a model (i.e. it has blacklisted various companies for ethical reasons[5]). However, the issues of nuclear weapons and tobacco are reasonably straightforward, as these are well defined areas that successive New Zealand Governments have had policies against.

Legislation is urgently needed to address this situation, possibly based on strengthening an existing proposal by Maryan Street MP and upgrading it from the lottery of private member's bills. Until this is done, New Zealand's status as a contributor to international health and being a responsible global citizen will remain in doubt.

Nick Wilson, George Thomson
Department of Public Health
Wellington School of Medicine & Health Sciences, University of Otago
Wellington
(nick.wilson@otago.ac.nz)

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Vasectomy procedures

The Disciplinary Tribunal's findings relating to vasectomies performed by Dr Wilson are reported in *NZMJ* 26 January 2007;120(1248) *Professional Misconduct – Vasectomy Procedures*; http://www.nzma.org.nz/journal/120-1248/2406. I wholeheartedly agree with the bulk of the Tribunal's findings, particularly relating to the importance of postoperative seminal fluid analysis and excellent communication with the patient. However I disagree with two of the Tribunal's findings.

"Histological Examination of Sample"—I dispute the value of this investigation as a routine. For $NZ68.67(GST inclusive) per patient all that is established is that the material submitted is indeed vas deferens. With "No Scalpel" technique, where a single midline scrotal puncture is used, it is perfectly possible to secure the same vas twice—and the left and right vas are histologically identical!

There may be occasions where there is clinical concern about the nature of the resected material when histology could be useful. The objective is to achieve permanent azospermia—not to submit pieces of vas for histology. In Australia, histology of excised vasectomy specimens is not funded and unlikely to be performed. I am not aware their results are inferior to New Zealand's.

By not submitting some 7000 patients' vasa deferentia for histology, I have saved the taxpayer approaching $NZ500,000 (GST inclusive). There are better uses for health dollars.

"Failure to Cauterise and Ligate the Severed Ends of the Vas"—I would suggest that this should read "Failure to Cauterise and/or Ligate the Abdominal End of the Severed Vas".

"Open Ended" vasectomy where the testicular end is deliberately left unobstructed is considered by some to lessen postoperative pain, and is an established technique. A ligature is liable to create a foreign body reaction and problems such as some of those experienced by Dr Wilson's patient Mr L.

After obtaining informed consent, my own practice is to use no foreign material. A loop of one vas is delivered through a midline scrotal puncture and a 1cm length removed; the lumen and severed ends (both testicular and abdominal) are coagulated with cautery. The testicular end is slipped into a fascial pocket and the mouth coagulated with cautery. Both ends are returned through the puncture and the other vas is delivered through the same puncture, and an identical procedure performed.

The patient is advised (and signs consent to the effect) that he must not abandon other contraception until azospermia has been confirmed at least 3 and 4 months postoperatively.

I perform around 600 vasectomies a year—and each year, one or two men will have persistent live sperms in their seminal fluid and require to be redone. Out of over 7300 cases, I know of two pregnancies after the man has had azospermia at 3 and 4 months. In one case, further seminal fluid analysis still showed azospermia—and in the other,

a pregnancy occurred 6 years after the vasectomy and confirmation of azospermia. Seminal fluid showed a small number of sperms—many of which were motile. He wishes to have the vasectomy redone.

Phil Airey
GP, Merivale Medical Practice
Christchurch

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PHARMAC and statins—correction is needed

Last year Dr. Chris Ellis and Professor Harvey White discussed past aspects of PHARMAC's operations relating to the provision of statins to improve cardiovascular risk.[1] We made a brief response at the time (http://www.nzma.org.nz/journal/119-1238/2092/),[2] and we are now providing additional information.

While welcoming different perspectives, a number of comments were made that PHARMAC considers to be factually incorrect. Our main focus is to correct these misperceptions, which we do in Table 1 (below). It is important that PHARMAC does this—not just in terms of responding to criticism, but because of the important role of statins in the management of cardiovascular disease in New Zealand.

Evidence underpinning our views is on public record in the Journal—both for statins[2–4] and wider.[5–24] Previous access restrictions for statins related to their prohibitive prices; simvastatin is now 1/20th the price of what it was 13 years ago. There is a continual need to balance priorities in order to maximise health gain across the population.

PHARMAC welcomes close scrutiny of its decisions and constructive suggestions for improvement. Ongoing improvement in processes and system—as for all organisations—is critical to best practice and keeping well-prepared for future challenges. PHARMAC's recent consultations on its cost-utility analysis framework[25] and how best to fund high cost medicines (http://www.pharmac.govt.nz/highcostmeds.asp) are examples of valuable opportunities to contribute. The Government's medicines strategy work (http://www.moh.govt.nz/moh.nsf/pagesmh/5633/$File/towards-newzealand-medicines-strategy-consult.pdf) is also an opportunity to look at potential improvements across the whole medicines system—from research and registration, through to whether medicines are optimally used, and important parts in-between like PHARMAC's role and prescribing decisions.

We believe that PHARMAC's approach for the funding of statins has, over time, successfully targeted access to give long-term health gains. While readers will draw their own conclusions,[1–5] PHARMAC is satisfied that its work has been careful and, in our view, robust, mindful of responsibilities to all patient groups across the population—as with all medicines.

Scott Metcalfe
Chief Advisor Population Medicine

Peter Moodie
Medical Director

PHARMAC
Wellington

Table 1. Specific claims with PHARMAC responses

Topic/claim	PHARMAC response	Comment
Population coverage of statins	NZ's statin usage is comparable internationally and now equals Australia's, at 1/3rd the cost	Material on the British Medical Journal website (http://bmj.bmjjournals.com/cgi/eletters/328/7436/385#52963) shows the use of statins has been comparable to other countries. New Zealand's per-capita use of statins is now the same as that of Australia[2]—despite the per-capita nominal costs in New Zealand being 1/3rd that of Australia (Australia spent AUS$886 million on statins in 2005).[26]
Patient access to atorvastatin is less than for simvastatin	The known differences in potency between simvastatin and atorvastatin are probably overstated Access to simvastatin is now unrestricted, and atorvastatin remains available as a second-line agent A recent British Medical Journal editorial[27] advocates reference pricing atorvastatin to generic simvastatin in the United Kingdom, as happens in Germany	The authors cite an incomplete published meta-analysis (Law et al BMJ 2003[28]) that did not include head-to-head trials. Up to April 2004, we had identified 20 additional published head-to-head randomised parallel group or cross-over studies directly comparing simvastatin and atorvastatin for LDL-C lowering, with 8,855 patients. Overall it seems that simvastatin is around 80% as potent at reducing LDL-C as atorvastatin at lower nominal daily doses (10–40mg) and 90% as potent at 80mg/day. These differences are less than those indicated by Law meta-analysis,[28] which was of largely less-comparable non-head-to-head studies. The Law meta-analysis also did not include studies published after 2001—e.g. STELLAR,*[29] Karalis et al 2002[30]—and only two of the 164 studies it used directly compared atorvastatin and simvastatin in the same group of patients (with 290 patients studied).[31,32] The Pharmacology and Therapeutics Committee (PTAC) has previously noted that measures such as LDL-cholesterol reduction (the commonest way to compare between statins) are ultimately surrogate markers, not clinically relevant outcomes. There is no head-to-head dose-equivalent outcomes evidence comparing atorvastatin with simvastatin, and hence no firm basis for stating that either is better than the other. In terms of surrogate markers, simvastatin appears to be more effective than atorvastatin for other important surrogate measures such as raising HDL-C.[33,34] PTAC in February 2006 reviewed the evidence for the current dose equivalence between atorvastatin and simvastatin, and considered that 1:2 dose equivalence provides a rough guide, and that in clinical practice the dose of any statin should be adjusted according to the individual patient's response [in light of absolute cardiovascular risk].
Cholesterol target levels	The New Zealand recommendations seem reasonable	PTAC in February 2006 noted the focus on greater reductions in LDL cholesterol. It noted that LDL targets have been progressively lowered, and also that by increasing doses to lower LDL levels the risk of side-effects also increased, and that this can occur with limited additional clinical gains. PTAC recommended that the target LDL levels remain unchanged, but noted that it may reassess them if the New Zealand Guidelines Group (NZGG) recommended changes to target LDL levels. A recent paper in the BMJ[35] (http://bmj.bmjjournals.com/cgi/content/full/332/7555/1419) suggests that adopting the approach recommended by the

		NZGG's cardiovascular guidelines[36] may be highly efficient. Modelling the theoretical impacts of various international guidelines on the Canadian population, the researchers estimated that the New Zealand guideline was the most efficient of all the guidelines, potentially avoiding nearly as many deaths as predicted by applying the Australian and British guidelines while recommending treatment to the fewest number of people (12.9% of people *v* 17.3% with the Australian and British guidelines)—important when treating some patients unnecessarily means not funding other health priorities. If their 'optional' recommendations are included, the use of the US guidelines' recommendations would mean treating about twice as many people as the New Zealand guidelines (24.5% of the population, an additional 1.4 million people) with almost no increase in the number of deaths avoided.
PHARMAC responsiveness to new evidence, as exemplified by issues around statin switching in 1997.	Access was extended to more patients, using a satisfactory if not the best statin. PHARMAC has already acknowledged that in hindsight the implementation process was imperfect.[37] PHARMAC subsequently fully funded simvastatin for patients who met defined criteria by January 1998, and atorvastatin later that year. There is a continuum of belief for class effects between evidence from a single clinical trial and mortality-based studies at doses used in clinical trials in similar populations.[38] There has been no good evidence of any harm that resulted from the switch from simvastatin to fluvastatin, and nor of increased mortality as a result of the application of reference pricing.	In 1997, PHARMAC widened access to statins by subsidising fluvastatin and reference pricing all available statins to it. This meant for the 12,000 existing patients either a change in medicine or an additional surcharge, but it also offered access for some 112,500 new patients. PHARMAC had to consider how fluvastatin compared to simvastatin and the possible risks of reference pricing. Fluvastatin did have limited outcome data[39,40] although no significant mortality data. The lipid-lowering effect of fluvastatin was expected to be 85% that of simvastatin's at equipotent doses,[41] which needed to be weighed against the potential to give benefit to many more patients within available resources.[42] There has been no good evidence of any harm that resulted from the switch from simvastatin to fluvastatin, and nor of increased mortality as a result of the application of reference pricing. The Dunedin paper was criticised internationally[44–46] for the following limitations: 1. Comparable mortality data were not collected—patients treated on simvastatin before the switch would have had to survive to remain in the cohort, and since no such restriction occurred after switching to fluvastatin, deaths after the switch logically should have been excluded. 2. Because it was an uncontrolled before-and-after study, potential bias was introduced by the unmasking of clinicians who admitted and then assessed patients, and of the evaluators who extracted and assessed the data. 3. The data before the switch were obtained from the hospital computer system (not fully reliable), whereas the data after the switch appeared to have been collected systematically and with care. 4. The analysis tabulated but failed to comment on a key possible reason behind the reported increase in cholesterol concentrations—being the possible subtherapeutic dosing of patients with the substituted drug (fluvastatin).[46,47]

		Both the New Zealand and Canadian experience has suggested that switching of other cardiovascular medicines such as DHP CCBs and ACE inhibitors—associated with reference pricing policies—has not been associated with clear evidence of worsening population health outcomes.[48–50] We are not aware of any similar population-based observational analyses for statins. We do acknowledge here the known biases with such analyses, which need to be interpreted in light of their methodological limitations versus the costs and availability of more robust evidence.
Atorvastatin 2004—PHARMAC response to consultation concerns over proposed switch to simvastatin for patients using 40mg atorvastatin	PHARMAC takes consultation seriously. The supplier would have withdrawn for other reasons.	It is not clear to PHARMAC that there is any causative link between pharmaceutical sales in a country and the commercial evaluation of where to locate research programmes.
Safety of the 80mg simvastatin dose	PTAC considers that statin adverse effects are a class effect	PTAC's February 2006 meeting examined claims by the supplier that the safety of high-dose atorvastatin was superior to that of high dose simvastatin. PTAC noted that the risk of adverse effects were dose-related, and considered that risk factors (dose and potency) were the important issue and that the increase in adverse effects with increased doses was a class effect and could occur with an increase in dose of any statin. PTAC also considered the use of atorvastatin 80 mg, and noted that in comparison to moderate doses or 80mg simvastatin, treatment with atorvastatin 80 mg results in a small additional decrease in LDL cholesterol but may be associated with the potential for an increase in the risk of adverse effects. Members considered that if atorvastatin 80 mg was listed in the Pharmaceutical Schedule there would be a risk that patients would begin atorvastatin at the 80 mg daily dose.
Need to protect patients	This argument goes both ways	The authors cite Begg et al[51] on the need to advocate for patients. PHARMAC's response at the time (http://www.nzma.org.nz/journal/116-1170/361/)[3] was that patients' needs extend beyond individuals presenting in limited clinical settings, and the need for a population approach. Historically, patients at highest overall cardiovascular risk have tended not to receive statin treatment, particularly Māori and Pacific men. That is why PHARMAC is working with DHBs and communities with its One Heart Many Lives programme attempting to redress this. It is PHARMAC's role to represent the public interest. PHARMAC's staff are very mindful of their responsibilities to all patients across all disease and disability groups—to achieve the best health gains for the New Zealand population within the funding available.

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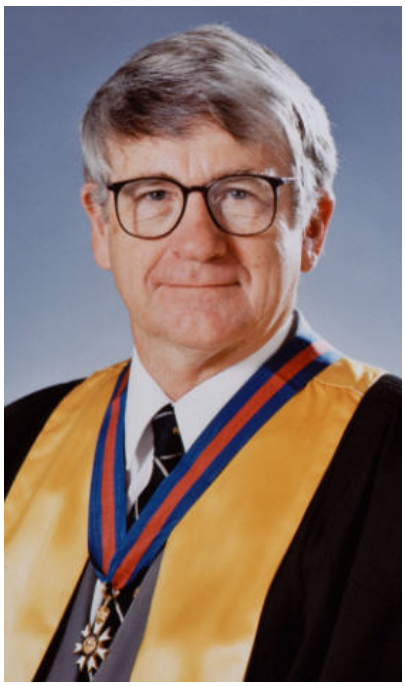
Alan Maxwell Clarke

12 December 1932–21 January 2007

Alan Clarke died following heart surgery at Christchurch after a rich life of 3 careers: Dunedin surgeon; Dean of the Christchurch School of Medicine; and customer and crusader for the Burwood Spinal Injuries Unit. He was farewelled by his close friend, the Rev Bruce Hansen, at a packed Knox Church in Merivale, Christchurch.



Alan was born in New Plymouth where father, John Maxwell (Marcus) Clarke, was a general surgeon and mother had been a theatre nurse.

When Marcus went abroad to war, mother took him and young brother John to her brother's Manawatu farm.

His uncle acquainted him with the usual rural childhood skills—milking, animal care, shooting, and so forth—thus engendering a love of nature, especially ornithology.

At the local sole-teacher primary school his early interest in science was strongly encouraged.

The nearby Ohakea Airbase was a magnet for every young boy, and after gazing at exotic military planes, of course he was going to be an aviator!

His father, Marcus, returned after 6 years. Alan's memories were of someone who "suddenly turned up from nowhere and told me what to do". The family moved to Auckland where Marcus took a surgical post at Middlemore Hospital.

Alan attended King's College where, through the Air Training Corps, he was awarded a 2-year RNZAF flying scholarship. Father with stern good sense insisted that becoming a doctor ruled out flying. Medical intermediate was at Auckland University. Later student years were at Knox College in Dunedin, graduating MBChB in 1956.

On the way he picked up 3 academic awards and Otago and New Zealand University Blues for athletics. He was 1956 Australasian champion hammer thrower. Through the Student Christian Movement he became engaged to arts student Jane Malloch of Waikouiti who became his constant companion and support.

Alan chose surgery. Initial postgraduate training (including neurosurgery) was in Auckland and Dunedin. Later he and Jane travelled to Scotland. En route, while the ship was held stationary off Canary Islands, he performed an emergency craniotomy to relieve a crew member's subdural haematoma using instruments hastily fashioned by the ship's engineers.

At Glasgow's Western Infirmary he trained under Sir Charles Illingworth. Alan abhorred British bullying and had frequent occasion to address the contempt of junior staff shown by seniors. His FRACS followed in 1961 and he practiced general and vascular surgery in Dunedin as Senior Lecturer in Surgery. In 1970 he was appointed Ralph Barnett Chair of Surgery. His research included intestinal absorption, liver regeneration, and peptic ulcers.

David Stewart (a Knox College roommate) said Alan, Robin Irvine, John Hunter, and Tom O'Donnell emerged as the "Young Turks" of the School of Medicine which at the time was experiencing a severe crisis of chronic under-resourcing. This group lobbied long, hard, and successfully for a major review of the School. The resulting Christie Report proposed staff and curriculum restructuring and establishment of clinical schools at Christchurch and Wellington (Clarke and O'Donnell later became Deans of these schools, respectively). Alan, in particular, was developing radical curriculum ideas.

In 1979 he was diagnosed with bladder cancer. Intensive radiotherapy was followed by major surgery but with extremely severe complications. His recovery was largely an outcome of his determination. The episode reminded him of life's caprices so he promptly fulfilled his early dream by gaining his private pilot licence and his own light aeroplane. He was troubled by the inconsistency of cancer treatment throughout New Zealand, leading to the far-reaching document "Cancer Consensus Manual" in 1984.

He was appointed Dean of the Christchurch School of Medicine in 1986, a post he held during 8 years of challenges. He found that clinical science course lectures were poorly attended yet students achieved well in exams. This supported a notion of a decade earlier that lectures have more tradition than value. His solution was the abolition of lectures, favouring instead self-directed learning in small groups within clinical situations. This radical change required the courage to outflank traditionalists, to gain support of liberal colleagues, and to inspire confidence among apprehensive students. It was all fuelled by Alan's immense energy.

These were the turbulent times of the health reforms and the re-forging of relationships between universities and new crown health enterprises. He described his task as "educating" each of the new and often short-lived managers. Equally the University sector was being restructured. Through all of this he remained a cheerful advocate for his staff, students, and researchers. He fostered close links with the Canterbury Medical Research Foundation and other research funding bodies.

Meanwhile he and various colleagues commuted to and from Dunedin in his aeroplane, Cessna ZK-EXE, and he found time to drive and edit the multi author book "Understanding Cancer" aimed to help patients understand and cope with their disease.

In 1991 he fell from his porch roof. He suffered a paralysing spinal injury. As a result of this experience he expressed a vehement dislike of clinical practices which excluded the patient from the therapeutic team. He objected to the impersonal nature and the lack of privacy of the ward rounds. Typically he demanded their abolition. Now wheelchair-bound, he returned—in record time, according to Allan Bean—to his role as Dean. His new trick was "wheelies", precariously balancing his wheelchair on only its two big wheels for many minutes at a time but he became despondent about

his plane which required simultaneous operation of throttle, rudder pedals, and wheel. Old friend Tim Wallis told him not to be silly, and together they imported a simple T-bar connecting his wrist to the pedals. Alan was aloft, happily commuting as before.

His contributions to medicine were recognised publicly in 1995 when he was bestowed Companion of the Most Excellent Order of St Michael and St George.

He became Director of the Spinal Injuries Unit in 1994. He was an avid advocate for patients to manage their treatment and rehabilitation with doctors as advisors. He encouraged patients to understand their own condition. Of course this required that they were self-directed and motivated, traits not shared by all. His infectious energy and motivation meant that he could never accept the status quo. He vigorously changed the external environment of the Spinal Unit to positively foster rehabilitation. Among his efforts was planting, with a Polish forester, a *Robinia* nursery at Burwood to occupy clients and provide future funds for the Unit.

Alan was aware that the spinal injury specialty was regarded as lacklustre so he set about raising its profile to attract attention and funding. An early outcome was the Burwood Spinal Trust which evolved to the New Zealand Spinal Trust as a foundation for spinal research by professional researchers and patients. He conceived and set up the Allan Bean Centre for Research and Learning, with a major emphasis on the provision of knowledge on spinal injuries. Alan never shirked at inveigling others to freely contribute their skills to the Unit.

As if this was not sufficient he then set up the Burwood Academy of Independent Living to fund and encourage the wider aspects of rehabilitation regardless of cause (be it physical, psychological, or circumstantial).

Alan described himself as a "surgical consumer and survivor". He was characterised by good humour, a keen sense of fairness for all, and the ability to be seized by a passion. Not only did he have energy to see these passions realised but also the facility to enthuse and harness the expertise and energy of others. He was a keen family man, who enjoyed his family's support in return.

Alan Maxwell Clarke's outlook on life is epitomised in the last paragraph of his CV:

> In December 1991, 8 months after my accident, an old lady said to me "At your age you are very lucky to become a paraplegic—you can start your life all over again."

Alan is survived by his wife Jane; sons Richard, John, and Alistair; and daughter Fiona.

Max Abernethy (one-time Academic Secretary at the Christchurch School of Medicine) prepared this obituary. He acknowledges Jane Clarke, David Stewart, Jim Clayton, Bruce Hansen, Allan Bean, Andrew Hall, and several others for supplying first-hand information about Alan as well as the Cotter Medical History Trust for providing records.

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Erratum

Kimberley Thomson, Ralph Pinnock, Lochie Teague, Rachel Johnson, Noel Mannikam, Ross Drake. *Vincristine for the treatment of Kasabach-Merritt syndrome: recent New Zealand case experience.* N Z Med J 2007(February 16);120(1249). URL: http://www.nzma.org.nz/journal/120-1249/2418

The surname of the fifth author (NM), as supplied to the NZMJ, was previously spelt incorrectly. The fifth author's surname is Manikkam (not Manikkan).

Please refer to the above URL to view the corrected copy of the article.

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Examination Paediatrics: a guide to paediatric training (3rd edition)

Wayne Harris. Published by Churchill Livingstone (Elsevier), 2006.
ISBN 9780729537728. Contains 400 pages. Price AUD$82.50 (GST included)

Examination Paediatrics is well established as essential reading for those sitting the RACP clinical examination in Paediatrics. This is based on the book's readability, comprehensiveness, and relevance to the Australasian examination. The third edition relies on the same successful formula and layout, while expanding and updating some of the clinical information.

The structure of the book is related to the format of the exam, i.e. long and short cases. The chapters cover either a system (cardiology, endocrinology, gastroenterology, haematology, nephrology, neurology, the respiratory system, rheumatology), or an area of paediatrics (behavioural and developmental paediatrics, genetics and dysmorphology, neonatology, oncology).

The first four chapters give a general introduction to the exam, including one chapter dedicated to the psychology of sitting and passing. Each subsequent chapter then discusses a number of different long and short cases, with the long case discussions tending to include some background clinical information.

Wayne Harris is now the sole author of Examination Paediatrics. A reasonable amount of new information has been added, filling some of the gaps in the previous edition. It does, however, remain a book primarily dealing with what should be looked for in performing an examination, rather than a text on how to examine children.

Detail on how to illicit some signs may need to be found in other texts, or demonstrated by colleagues; preferably a Consultant who has been an examiner. The background information in each of the sections is often very practical, but as the author explains, this is not a comprehensive textbook of paediatrics.

As final testament to the quality of this text, reading the third edition, within a year of passing the exam myself, did not result in any DSM-IV classifiable state as one might predict. I fear the same may not be true of Nelson's.

Examination Paediatrics is an important tool for passing the RACP clinical examination. As long as upcoming changes to the exam format can be incorporated into future editions, this will continue to be the case for some time.

John Garrett
Paediatric Registrar
Christchurch Hospital
Christchurch