THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

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THE NEW ZEALAND MEDICAL JOURNAL
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This Issue in the Journal

The cost of paediatric and perianal Crohn's disease in Canterbury, New Zealand
Michaela Lion, Richard B Gearry, Andrew S Day, Tim Eglinton

We wanted to determine the direct and indirect costs of Crohn's disease (CD) in paediatric (infants, children, and adolescents) and perianal (surrounding the anus) patients in Canterbury in 1 year. Interviews were conducted over 3 months to obtain information regarding demographic, socioeconomic factors, and indirect costs. Hospital clinical notes were reviewed to determine direct health care utilisation and costs. In 1 year the total costs per patient for paediatric CD were $14,375 with direct and indirect costs comprising $12,583 and $1,792, respectively. The total costs per patient for perianal CD were $20,366 with direct and indirect costs comprising $18,261 and $2,105, respectively. Estimating these data across New Zealand, the total cost of paediatric and perianal CD in 1 year in New Zealand is approximately $25.9 million and $36.7 million, respectively. Paediatric and perianal CD are high-cost diseases with significant costs borne by patients and their families. Expensive pharmaceuticals comprise a significant proportion of the costs; increased access to these drugs might decrease hospital admissions and prevent work absenteeism and loss of carer productivity.

Screening for *Mycobacterium tuberculosis* infection among healthcare workers in New Zealand: prospective comparison between the tuberculin skin test and the QuantiFERON-TB Gold In-Tube® assay
Joshua T Freeman, Roger J Marshall, Sandie Newton, Paul Austin, Susan Taylor, Tony C Chew, Siobhan Gavaghan, Sally A Roberts

The QuantiFERON GOLD in tube test (QFT-GIT) has been approved by the FDA as an alternative to the traditional skin test for TB infection commonly known as the "Mantoux" (TST). The QFT-GIT has several advantages over the TST including: 1) the requirement for only a single visit compared to the need for at least one follow up visit with the TST; 2) less subjectivity in interpreting results; 3) less cross reactivity with the TB vaccination known as "BCG". However, the performance of the QFT-GIT as a pre-employment screening tool among healthcare workers in NZ had not been assessed prior to this study. Our findings suggest that the high rate of BCG vaccination among healthcare workers in NZ makes the QFT-GIT a desirable alternative to the TST as a pre-employment screening tool. The increased up front cost of the QFT-GIT to healthcare providers may be largely offset by the reduction in the number of unnecessary chest X-rays ordered on the basis of a "false positive" TST. With any screening program however, care must be taken to interpret results in light of the "pre-test probability". In other words, positive tests in very low risk persons should be interpreted cautiously, as should negative results in persons at very high risk of TB infection.

Audit of stroke thrombolysis in Wellington, New Zealand: disparity between in-hours and out-of-hours treatment time
Katie Thorne, Lai-Kin Wong, Gerard McGonigal

Stroke is a very common cause of disability in New Zealanders. Thrombolysis or 'clot-busting' treatment for stroke has been recommended for over 15 years as it can prevent and reduce disability and dependency. Despite overwhelming evidence, many New Zealand hospitals have not made this treatment available to those suffering from stroke. This paper further highlights an important disadvantage in treatment times for those admitted with stroke outside normal working hours in Wellington. Wellington has addressed this. It is time for all New Zealand hospitals to make this treatment available and ensure access is equitable no matter where or the time of day a stroke happens.

Training medical students in Pacific health through an immersion programme in New Zealand
Faafetai Sopoaga, Jennie L Connor, John D Dockerty, John Adams, Lynley Anderson

This paper describes the development of a Pacific Immersion Programme that provided medical students at the Dunedin School of Medicine the opportunity to learn about Pacific health in a new way. Students, staff and the local community agreed teaching in the community context provided a useful learning environment.

Insomnia treatment in New Zealand
Karyn M O'Keeffe, Philippa H Gander, W Guy Scott, Helen M Scott

Insomnia is perhaps the most common sleep disorder and is defined as having difficulty initiating or maintaining sleep, or non-restorative sleep, together with impaired waking function that has been present for at least one month. Based on a national survey of insomnia symptoms, we have estimated that 13% of New Zealanders aged 20-59 years have insomnia. Using the international literature, along with structured interviews with 21 insomnia treatment providers in New Zealand, we have described insomnia treatment in New Zealand and estimated the annual societal costs of insomnia among New Zealanders aged 20-59 years. This study has highlighted a number of significant issues; in particular, the unstructured nature of insomnia treatment in New Zealand. However, effective insomnia treatment would result in substantial saving to the healthcare system. The net annual saving for treating insomniacs aged between 20-59 yrs was estimated at $21.8 million.

Evaluation of New Zealand's bicycle helmet law
Colin F Clarke

This evaluation of New Zealand's bicycle helmet law finds it discouraged cycling usage by 51%. It results in more than 5000 of fines (for not wearing a helmet) per year, plus it may promote aspects of discrimination in accident compensation cases. Road safety and cyclist's safety should be improved by coherent policies, which support health, the environment, and without the legal requirement to wear a helmet.

Sun protection policies and practices in New Zealand primary schools
Anthony I Reeder, Janet A Jopson, Andrew Gray

Randomly selected primary schools were assessed for whether they met the 12 SunSmart Schools Accreditation criteria for sun protection policy, provision of information, hat wearing, 'play in the shade' rules, sunscreen, clothing, role modelling, curriculum content, planning, rescheduling of outdoor activities, shade provision and policy review. There remains considerable room for improvement, with fewer than 4% of schools fully meeting the 12 accreditation criteria. Schools were least likely to meet the criteria for clothing protection (42%), curriculum delivery and provision of adequate shade (each 54%). Ongoing efforts will be required to consolidate gains and encourage comprehensive sun protection through policies, practices, environment and curriculum. There will be a need to further assist schools, particularly regarding sun protective clothing, curriculum delivery and environmental shade.

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A call for collaboration on inflammatory bowel disease in New Zealand

Russell Walmsley

In this edition of the *Journal* the first attempt at calculating the financial burden of inflammatory bowel disease (IBD) in either New Zealand or Australia is described.[1] The Christchurch group of Tim Eglinton and his team—building on their reputation for sturdy epidemiological studies—have chosen just two categories of Crohn's disease (CD) patients where the burden to healthcare providers and patients and their families are perhaps the highest, namely paediatric and perineal disease.

The combined indirect and direct costs of one year of care for a paediatric case of Crohn's disease in 2009 was calculated to be NZ$14,375 and for a patient with perineal disease to be NZ$20,366.

The paper by Eglinton et al is timely in a number of ways. The contract for provision of Humira™ (Abbott) as the preferred anti-TNF agent with Pharmaceutical Management Agency in New Zealand (PHARMAC) is to be renegotiated this year; the move for PHARMAC to take over hospital pharmacies is underway, with anxiety over the consequent ability for clinicians and health authorities to decide on the use of other anti-TNF agents and emerging 'biologicals'.[2] Thirdly, and not least, the patient support group Crohn's and Colitis New Zealand has reorganised and relaunched themselves last year with renewed vigour to help promote IBD patients' care.[3]

Tim Eglinton and his team have extrapolated their calculations to suggest that the total cost (in 2009) of paediatric Crohn's disease to the nation was NZ$25.9 million, and for perineal disease was NZ$36.7 million.[1]

These conclusions should be taken with caution, however. The patients were seen at the tertiary IBD clinics at Christchurch Hospital, which serves an official population of 502,000 in the South Island of New Zealand. The cases, therefore, are likely to be the extreme end of the spectrum. It is not clear how they were chosen—e.g. whether they were consecutive cases, or what the response rate was from those contacted to fill in the indirect cost questionnaires.

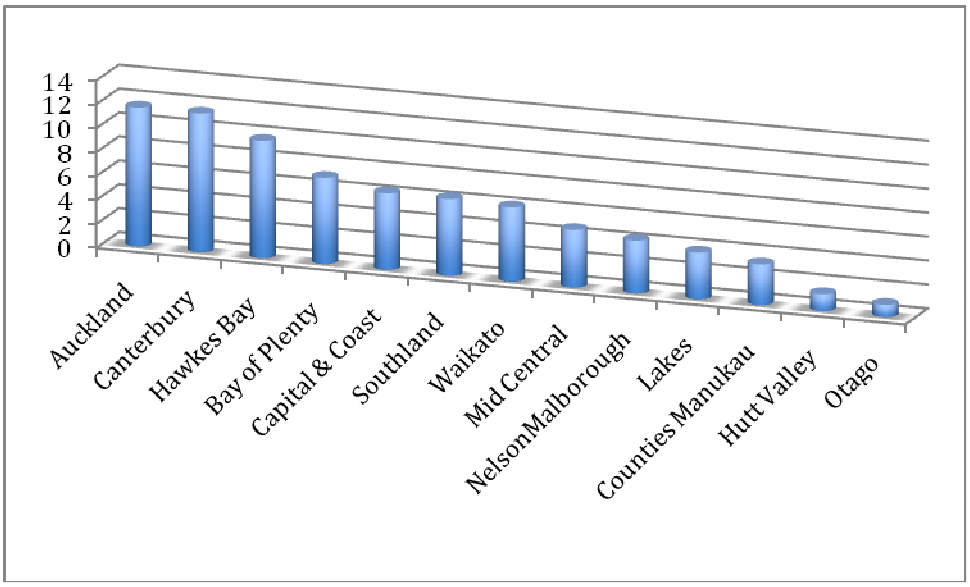
Retrospective recall of hours of work and income lost is bound to be fragile data. It should be noted, however, that the indirect costs at 8% and 9.7% of total costs for paediatric and perineal disease respectively is much lower than results from previous studies attempting the same calculation, as the authors point out.

When one looks closely at where the money is being spent you will see that 68% of outpatient costs and 46% of hospital-associated costs for perineal Crohn's disease was due to pharmaceuticals, and that 61% of the cost of this was due to anti-TNF therapies. We have good data based on 2008 prices in Europe that show that healthcare costs incurred in the first year of diagnosis of CD can vary by 30% between the UK and the rest of Europe where the use of biological agents is acknowledged to be quite different.[4]

If we are to extrapolate these findings to help inform the Ministry of Health on what the burden of cost to the nation is likely to be then we also have to realise that the prescribing of these agents is also far from uniform across New Zealand.

From a survey of members of the New Zealand Society of Gastroenterology, also undertaken in 2009, but before the subsidised access to Humira, Canterbury had the highest use of anti-TNF agents per capita (Figure 1).[5]

Figure 1. Prescription of biological agents for IBD, per 100,000 for the New Zealand District Health Boards serving populations of over 125,000 for the year ending June 2009.



Note: No data available for Northland and Waitemata. Full survey reported at the Annual Scientific Meeting of the New Zealand Society of Gastroenterology, 2009.

There is not space to debate the economics of using these treatments, but it is argued that they result in less hospital admissions and maybe less surgical episodes, so in the long run are economically 'viable'.[6,7] Could it be that the indirect costs are higher but pharmaceutical costs are less in DHBs such as Hutt Valley, where the use of biological agents is 10 times less? Additional information on the outcomes of the use of these expensive treatments in New Zealand, using our access criteria, is now required to help plan how best they are to be used from here on.

Another missing piece of the national IBD jigsaw that makes further extrapolation of the Christchurch information difficult is the lack of data on the prevalence of Crohn's disease in the rest of the country. The excellent epidemiological data from Canterbury for 2004–5 found an incidence of 16.5 per 10,[5] which is amongst the highest recently reported anywhere in the world, with only Canada (20.2) and the Australian state of Victoria reporting higher (29.3).[8] Compared to the previous New Zealand data published in 1986 this is a near 7-fold increase, and similar to trends seen around the world.

Crohn's disease predominantly affects people from Northern Europe as well as Jewish people, particularly those of Ashkenazi (Eastern European) descent. According to the 2006 New Zealand Census, 77.4% of people in Canterbury belonged to the European (mainly British and Irish) ethnic grouping, compared to 67.6% for New Zealand as a whole.[9]

If 20% of Crohn's patients have significant enough perineal disease to be treated in the same way that Christchurch treats these 26 examined cases, and the prevalence of Crohn's is taken as 140 per 10^5 (10% less than Canterbury data) with a population now nearer 4.43 million, then the annual cost to at 2009 prices might be nearer $25 million.

This paper puts a figure on the personal and national economic burden of a proportion of the growing number of predominantly young, potentially economically vital, people who have Crohn's disease. Attempting to extrapolate the findings to the rest of New Zealand highlights the deficiencies in epidemiological data, information on clinical outcomes and access of patients to expensive but internationally recognised treatments.

Surely now is the time for all interested parties, including the Ministry of Health, PHARMAC, the New Zealand Society of Gastroenterology and a revitalised Crohn's and Colitis New Zealand and the pharmaceutical companies to come together to fill these gaps in our knowledge of IBD in NZ and begin to grasp the problems of this increasingly relevant group of chronic diseases.

I feel 2012 could be an interesting year.

Competing interests: Member of the advisory boards for Humira (Abbott) and Remicade (Janssen). Member of PHARMAC Gastrointestinal sub-Committee.

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References:

1. Lion M, Gearry RB, Day AS, Eglinton T. The cost of paediatric and perineal Crohn's disease in Canterbury, New Zealand. N Z J Med 2012;125(1349). http://journal.nzma.org.nz/journal/125-1349/5055
2. The Pharmaceutical Management Agency. http://www.pharmac.govt.nz/2009/06/30/2009-06-30%20PHARMAC%20notification%20of%20decisions%20involving%20multiple%20pharmaceuticals%20including%20TNF%20inhibitors.pdf accessed 30 January 2012.
3. http://www.crohnsandcolitis.org.nz/home accessed 30 January 2012.
4. Buchanan J, Wordsworth S, Ahmad T, et al. Managing the long term care of inflammatory bowel disease patients: The cost to European health care providers. Journal of Crohn's and Colitis 2011;5:301-316.
5. Griffiths B, Wyeth JW. Poster presentation—Use of Biologic Therapy in IBD: a survey of New Zealand DHBs. New Zealand Society of Gastroenterology Annual Scientific meeting, Wellington, 2009.

6. Nguyen GC, Nugent Z, Shaw S, Bernstein CN. Outcomes of patients with Crohn's disease improved from 1988 to 2008 and were associated with increased specialist care. Gastroenterology 2011;141:90-97.
7. Jones DW, Finlayson SRG. Trends in surgery for Crohn's disease in the era of Infliximab. Annals of Surgery 2010;252(2):307-311.
8. Molodecky NA, Soon IS, Rabi DM, et al. Increasing Incidence and Prevalence of Inflammatory Bowel Disease with Time, based on systematic review. Gastroenterology 2012;142(1):46-54.
9. http://www.stats.govt.nz accessed 30 January 2012.

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The cost of paediatric and perianal Crohn's disease in Canterbury, New Zealand

Michaela Lion, Richard B Gearry, Andrew S Day, Tim W Eglinton

Abstract

Aims The aim of this study was to determine the direct and indirect costs of Crohn's disease (CD) in paediatric and perianal patients in Canterbury in one year.

Methods A retrospective cross-sectional analysis was performed. Paediatric CD patients and adult patients with perianal CD were recruited over a three month period. Interviews were conducted to obtain information regarding demographic, socioeconomic factors, and indirect costs. Hospital clinical notes were reviewed to determine direct health care utilisation and costs.

Results Forty-nine patients (24 paediatric and 25 perianal CD) were enrolled. In one year the total costs per patient for paediatric CD were $14,375 with direct and indirect costs comprising $12,583 and $1,792, respectively. The total costs per patient for perianal CD were $20,366 with direct and indirect costs comprising $18,261 and $2,105, respectively. Extrapolating these data across New Zealand, the total cost of paediatric and perianal CD in one year is approximately $25.9 million and $36.7 million, respectively.

Conclusions Paediatric and perianal CD are high-cost diseases with significant costs borne by patients and their families. Expensive pharmaceuticals comprise a significant proportion of the costs: increased access to these drugs might decrease hospital admissions and prevent work absenteeism and loss of carer productivity.

Crohn's disease (CD) is a chronic inflammatory bowel disease characterised by transmural segmental inflammation of the gastrointestinal tract. The incidence of CD is increasing worldwide[1] and the peak age of onset is between 15 and 35 years.[2] CD remains incurable and a proportion of patients will endure recurrent and prolonged periods of illness requiring extensive medical and surgical interventions during what would otherwise be a highly productive time of life. Hence CD presents an increasingly significant health problem not only in terms of morbidity, but also in cost to the individual patient and society.

Previous studies have indicated that the majority of the total cost associated with this disease relates to "extensive interventions required by a small proportion of severely affected individuals".[3] Several clinical markers of disease severity have been documented including diagnosis at a young age and the presence of perianal disease.[4] In addition to being independent markers of a more severe disease course, paediatric patients have a longer temporal exposure to CD-related complications and perianal disease patients develop local perianal complications requiring frequent intervention[5] hence, both these patient groups are more likely to use significant health resources and incur the largest costs as a result of the disease.

A number of international studies have considered the cost of inflammatory bowel disease overall,[6–15] however, to the authors' knowledge, there are no data documenting the average patient cost of IBD in New Zealand or Australia and no previous work specifically investigating the paediatric and perianal CD groups.

Evidence is accumulating that newer therapies, such as anti-tumour necrosis factor alpha (anti-TNFα) antibodies, have significant efficacy in inducing and maintaining remission and have particular roles in complicated CD, such as perianal CD.[16,17] However, these agents are expensive and predicting which patients will benefit from their early use remains a challenge.[4] The introduction of these modern biological treatments has created a need for government agencies to consider the economic impact of therapeutic alternatives. To achieve this, the cost of CD and its societal burden requires further study.

This is a cross-sectional retrospective study, which aimed to estimate the direct and indirect costs of paediatric and perianal CD in one year in Canterbury using patient-based data. The costs of CD are borne not only by the taxpayer through government funded healthcare but the patient and their family, therefore the cost perspective is approached from a societal point of view.

Methods

This study was performed in Christchurch Hospital, a tertiary university hospital serving a population of around 500,000. Ethical approval for the study was obtained from the regional ethics committee. All patients with CD according to previously documented diagnostic criteria[18] presenting to the institution during the period November 2009 to February 2010 were eligible for entry into the study.

Paediatric patients were defined as those aged 16 years or less. Perianal disease was defined as any symptomatic perianal lesion included in the American Gastroenterological Association classification.[19] Patients were recruited through Gastroenterology, Colorectal surgical and Paediatric outpatient clinics and hospital admissions.

After giving written informed consent, patients or their parents were submitted to a structured interview to obtain information regarding demographic and socioeconomic factors, work and school absenteeism, alternative health resource use and other related data for the preceding twelve month period. Participants were also offered the opportunity to nominate other costs that were not mentioned in the interview. Following the structured interview, hospital clinical notes were reviewed to determine direct health care utilization. This included hospital inpatient and outpatient visits and prescription drug use.

For the purposes of this analysis the costs were classified as direct or indirect. Direct costs included hospital (Emergency Department visits, laboratory tests, radiological investigations, endoscopy, pharmaceuticals, inpatient care and operating theatre costs) and outpatient (General Practice visits, specialist clinic visits, alternative health professional visits, non-prescription medications, pharmaceuticals, laboratory tests, District Nurse and Social Work services) associated costs. Indirect costs included; lost productivity, travel, carers, tutors and additional phone or internet requirements.

The costs of hospital resources were determined through the Costing Department of the Canterbury District Health Board (CDHB) and the Ministry of Health. Hospital costs were calculated using DRG codes assigned to the patient each time they visited the hospital. A different code is given for each service required during each visit. Based on the quantity used during the visit the cost is calculated for each service used in a given visit.

The cost department for the CDHB supplied the authors with all the codes and costs accrued by the patients during the study period. For primary care cost calculation, it was assumed that all patients were enrolled in a primary health organisation (PHO). The New Zealand Government provides subsidies to lower the cost of general practitioner (GP) visits for eligible people enrolled in a PHO. The cost of GP services was estimated using the average cost of an appointment by age as obtained from Pegasus Health PHO and the 2010 yearly capitation rates provided by the Government. The capitation rates took into account whether or not the patient had a high user health card (HUHC).

Pharmaceutical costs were calculated from the cost to the Pharmaceutical Management Agency in New Zealand (PHARMAC) provided by their pharmaceutical schedule accessed 1 December 2009. Additionally a 4% mark-up was added to pharmaceutical costs plus a $5.80 dispensing fee attributable to pharmacists. The co-payments paid by the patient are a transfer not a cost therefore these were not included.

The human capital method as described by Drummond et al[20] was employed in calculating indirect costs. Patients were asked the number of days they had off work as either unpaid or annual leave related to CD. This was transferred into hours off work and was multiplied by their gross hourly wage. For those patients not in work their indirect costs are discussed descriptively as monetary values were not able to be estimated. Government welfare payments received by patients not in work were not included as they represent a transfer rather than a cost.

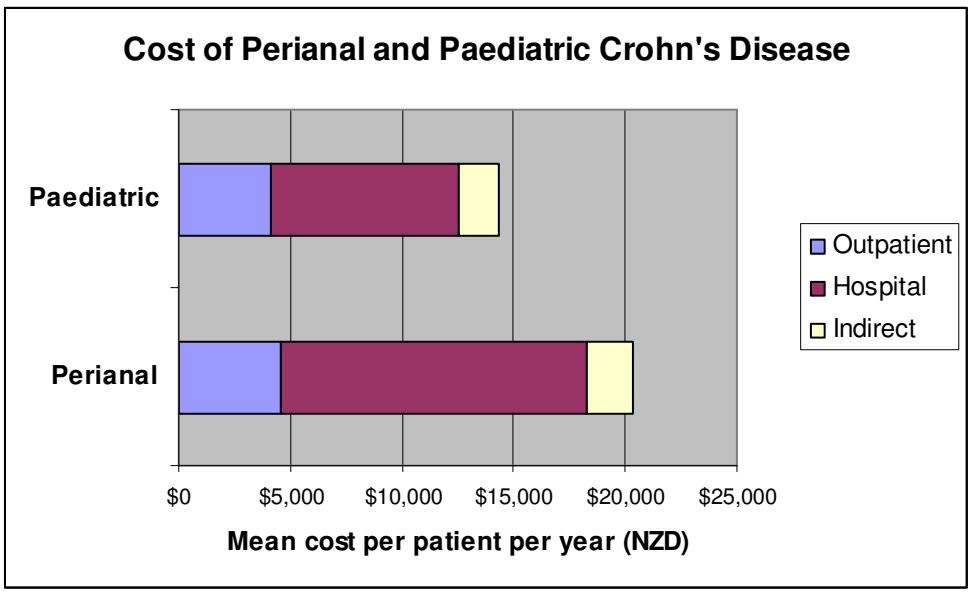
It is conservatively estimated that there are approximately 9000 individuals with IBD in NZ, with perianal and paediatric patients consisting of about 20% each. This estimation was used to extrapolate the data to obtain values for the cost to society in New Zealand of perianal and paediatric CD in one year.

Results

In total 49 patients were entered into the study; 24 paediatric CD patients (mean age 12 years, range 4 to 15 years) and 25 adult patients with perianal CD (mean age 33 years, range17 to 73 years). The paediatric group contained 16 males and 21 of the patients were of New Zealand European ethnicity.

The perianal group contained 15 males; 21 declared New Zealand European ethnicity. All of the paediatric patients were attending school except for one who was at kindergarten part-time. In the perianal sample, 18 patients were in some form of employment, three were in the education system, three were not participating in any work or education activities and one was retired. In one year, the average total cost per patient for paediatric CD was $14,375 with direct and indirect costs comprising $12,583 and $1792, respectively (Figure 1).

Figure 1. Overall cost of perianal and paediatric CD divided into outpatient, hospital and indirect costs



The most significant direct costs were inpatient costs (Figure 2) followed by pharmaceutical costs (Figure 3 and 4). Foregone productivity as a result of parental absenteeism from work was the greatest indirect cost (Figure 5). The children had an average of 21 days off school during the year.

Figure 2. Outpatient cost of perianal and paediatric CD showing the components that made up the total cost

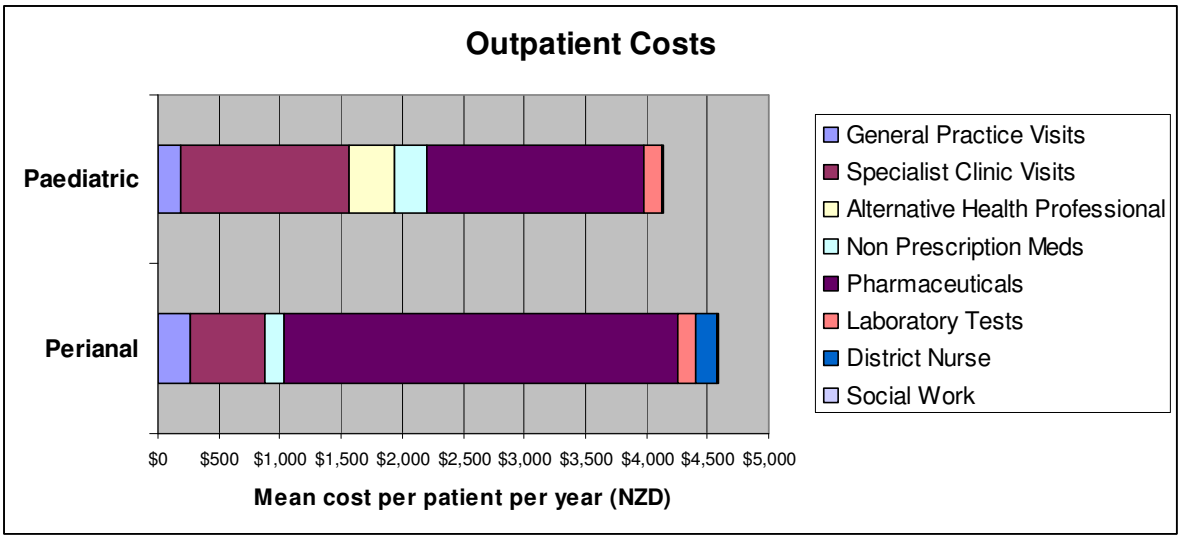


Figure 3. Hospital cost of perianal and paediatric CD showing the components that made up the total cost

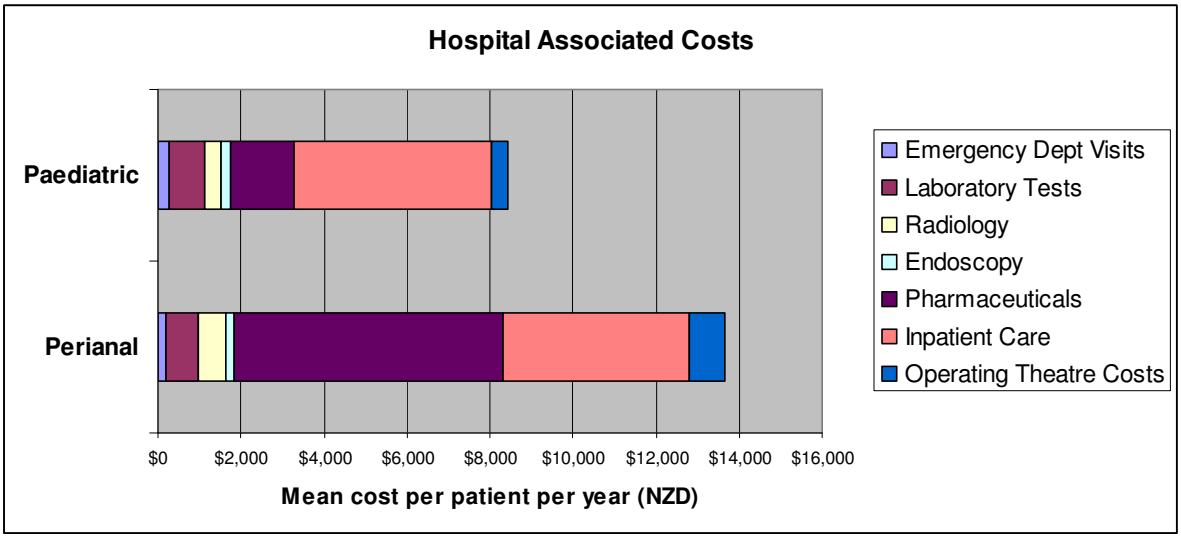


Figure 4. Cost of perianal and paediatric CD with comparison of hospital and pharmaceutical cost

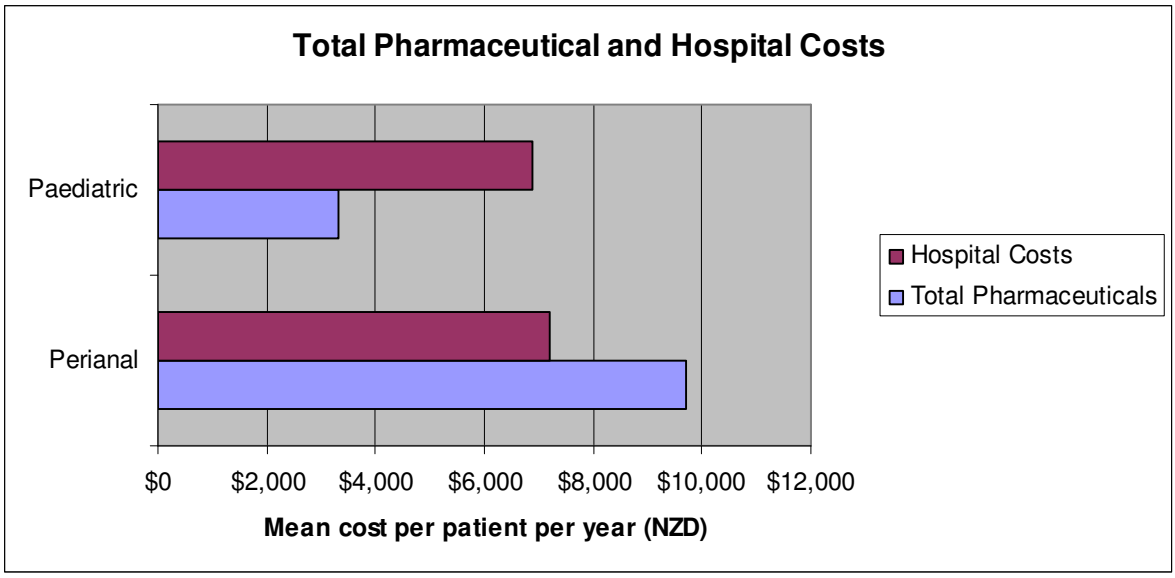
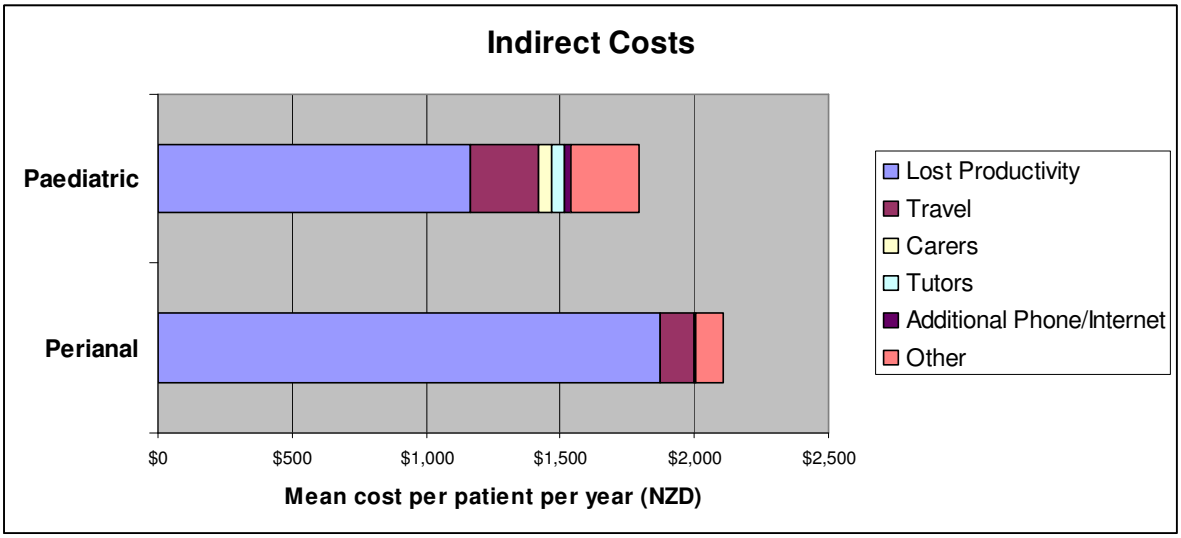


Figure 5. Indirect costs of perianal and paediatric CD showing the components that made up the total cost



The average total costs per patient for perianal CD were $20,366 with direct and indirect costs comprising $18,261 and $2,105, respectively (Figure 1). Eight of the 25 perianal CD patients (32%) received anti-TNF therapy during the study period, The most significant costs were pharmaceuticals followed by inpatient costs. This was highlighted when the total pharmaceutical bill was compared to other hospital costs (Figure 4).

Anti TNFα medication made up 61% of the pharmaceutical costs for perianal patients. The greatest indirect cost was patient and immediate family absenteeism from work (Figure 5).

Extrapolating these data across New Zealand, the total cost of paediatric and perianal CD in one year is estimated to be at least $25.9 million and $36.7 million, respectively.

Discussion

These results demonstrate that both paediatric and perianal Crohn's disease are high-cost disorders. No previous studies in New Zealand or internationally have documented the costs created specifically by these specific groups of patients.

Several studies that considered IBD overall found that CD was associated with greater cost than ulcerative colitis.[6,13,14] A number of studies have also looked specifically at the cost of CD overall.[8,9,11,12,15] The heterogeneous nature of these studies in terms of methodology, varying costs in different health care systems and the differential effects of inflation since the period of the study makes direct comparison with the present data difficult.

Juan et al reported the annual cost per patient in a Spanish cohort in 2003 was €6,808 with €2,104 from direct costs and €4,704 from indirect costs.[12] Extrapolating data reported in 2009 by Mesterton et al the average annual cost of CD in Swedish patients was €9,400[15] (approximately 16,240 New Zealand dollars at current exchange rates). Hence this group found comparable figures and also noted that increased cost was predicted by increased severity as measured by the Harvey-Bradshaw index.

While the patients in the present study were not stratified for severity, perianal disease itself is a predictor of severe disease and this may explain the slightly higher direct costs reported here. This is supported by the previous finding that the presence of fistulae doubled the costs of care.[15] Patients recruited from tertiary referral centres, as in the present study, also tend to have more severe disease and this too could have contributed to the higher direct costs reported in this study. This fact and the small sample size in the present study mean some caution should be exercised in interpreting the results of this study when extrapolated to the perianal and paediatric CD population across New Zealand.

In contrast to the present analysis, both the previous European studies determined a higher relative contribution from indirect compared with direct costs. This may reflect the method of estimation of indirect costs. As the patients (or carers) in the present study were asked to estimate the absenteeism over the previous year, an element of recall bias may be present. In addition, no allowance for lost productivity while at work due to the disease (presenteeism) was made in this study.

In some cases parents of CD patients admitted to the use of flexible work or leave arrangements, for example working from home or being able work late to make up time off. There were several cases where parents were only able to work part-time and some not at all that were not included as lost productivity in this analysis as the lost productivity was only partly attributable to CD. Other reasons contributing to the reduced productivity included having more than one child with a sickness and less requirement to work due to their spouse having adequate income.

The patients were not asked if they were working part-time as a result of their disease. This potentially could have increase the value of their lost productivity. There was an economic recession for the majority of 2009 that could have contributed to the reduced work hours; therefore it does not seem appropriate to assume working part-time was a result of their illness. Altogether, these factors likely underestimate the indirect costs associated with CD in the current study.

These issues highlight the fact that there is some controversy in the literature in regards to the best approach to use when estimating indirect costs. The human capital-cost method is recommended over other approaches by Liljas[21] and Johannesson[22] for cost studies from a societal perspective.

These authors propose it is the most consistent with economic theory, therefore, indirect costs were estimated using the human capital method in this study, consistent with the approach taken in other recent studies.[14,15] Despite this, the lower proportion of indirect costs found here could indicate the total cost has been underestimated in the present study hence these cost figures should be considered as a minimum.

Studies of this nature do not capture other effects of a chronic disease on productivity because the estimation of indirect costs is calculated based on the patients current gross wage rate. However, this does not take into account the wage rate the patient could have realised had they not been diagnosed with CD. Given that many patients with CD are diagnosed before or during the second or third decades of life, their disease may have potential life-long impact on educational achievement, career prospects and earning potential consequent to disrupted education and work.

Paediatric CD patients in the present study had an average of 21 days absent from school in the previous year consistent with levels of absenteeism documented in two previous case control studies.[23,24] Of note while the cases in these two studies showed significantly greater absenteeism than controls, decreased ability to present for exams and some degree of discrimination from teachers, there was no difference in level of educational achievement between cases and controls. Despite this, forty seven percent of respondents to a survey conducted in Germany felt that CD had interfered with their career prospects.[14] A separate study revealed 30% of CD patients concealed their diagnosis from their employers.[24] Hence more subtle loss of productivity costs are likely to exist which have not been measured in this study.

In addition to both the direct and indirect costs discussed above, this study has not included intangible elements associated with the burden of disease, or loss of wellbeing, associated with CD. A more global assessment of the economic impact of CD would include such costs. Recent economic theory has allowed integration of these concepts into cost calculations.

Using willingness to pay measures of mortality and morbidity associated with disease, economists have developed estimates of the Value of a Statistical Life. This can be used to attach a monetary value to the non-financially derived Disability Adjusted Life Year concept and thereby derive a financial cost associated with the burden of disease.[25] This was not attempted in the present study where the aim was to produce estimates for future cost-benefit analysis that will utilise the direct costs associated with CD.

The principal direct costs incurred were for inpatient care and pharmaceuticals. In the adult group with perianal disease, the total pharmaceutical bill was greater than the other hospital associated costs (Figure 4). In several early studies, hospital costs were found to make up a higher proportion of the total cost of care for IBD than pharmaceutical costs.[7,9,26] However, these three studies were performed prior to the widespread use of anti-TNFα agents.

As the present study was performed in the era of biologic treatments, the proportionally increased pharmaceutical costs are likely to represent a genuine finding and are consistent with the other most recent cost study from Sweden.[15] Anti-TNFα therapy is expensive but it may prove cost-effective if it leads to a reduction in hospitalisation and the high costs associated with this.

This study was not designed to assess this. However, Hay and Hay[27] have previously created a model for the cost-effectiveness of expensive drug therapy in CD. Their model demonstrated that if a new drug reduced other costs such as hospitalisation by 20% then, despite a doubling of the pharmaceutical bill, the overall cost of care would reduce by 13%.[29]

Surgery was another significant direct cost associated with CD in the present study. As patients undergoing surgery tend to more severely affected, direct comparisons of efficacy with medical treatments for similar clinical states are limited. However, Silverstein et al[8] found in a Markov analysis that despite higher costs for surgery, post surgical remission was longer than for patients treated medically. These authors concluded that surgery may therefore be a more cost-effective option in selected cases.[8]

Laparoscopic surgery is now increasingly used in the treatment of IBD. Short term advantages of laparoscopic surgery include improved pulmonary function, decreased ileus, shorter hospital stay and improved cosmesis.[28] In the longer term, there does not appear to be any difference in recurrence rates compared with open surgery.[29] While operative time and intraoperative expenses are increased, total hospital costs are reduced with the decreased length of stay.[28] Hence, any future cost-benefit analysis will need to allow for the impact of laparoscopic surgery.

This study has confirmed that paediatric and perianal CD patients consume significant health resources. Prior to this, no studies in New Zealand have estimated the cost of IBD and no international studies have estimated the indirect costs of paediatric Crohn's disease. With the advent of increasingly costly and effective medical therapies and evolving surgical treatment, this research will provide valuable information for future cost-effectiveness studies.

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References:

1. Loftus EV, Jr. Clinical epidemiology of inflammatory bowel disease: Incidence, prevalence, and environmental influences. Gastroenterology 2004;126(6):1504-17.
2. Farrokhyar F, Swarbrick ET, Irvine EJ. A critical review of epidemiological studies in inflammatory bowel disease. Scand J Gastroenterol 2001;36(1):2-15.
3. Bodger K. Cost of illness of Crohn's disease. Pharmacoeconomics 2002;20(10):639-52.
4. Eglinton TW, Gearry RB. Clinical factors predicting disease course in Crohn's disease. Expert Rev Clin Immunol 2010;6(1):41-5.
5. Eglinton T, Reilly M, Chang C, et al. Ileal disease is associated with surgery for perianal disease in a population-based Crohn's disease cohort. Br J Surg 2010;97(7):1103-9.
6. Odes S, Vardi H, Friger M, et al. Cost analysis and cost determinants in a European inflammatory bowel disease inception cohort with 10 years of follow-up evaluation. Gastroenterology 2006;131(3):719-28.
7. Blomqvist P, Ekbom A. Inflammatory bowel diseases: health care and costs in Sweden in 1994. Scand J Gastroenterol 1997;32(11):1134-9.
8. Silverstein MD, Loftus EV, Sandborn WJ, et al. Clinical course and costs of care for Crohn's disease: Markov model analysis of a population-based cohort. Gastroenterology 1999;117(1):49-57.
9. Feagan BG, Vreeland MG, Larson LR, Bala MV. Annual cost of care for Crohn's disease: a payor perspective. American Journal of Gastroenterology 2000;95(8):1955-60.
10. Bernstein CN, Papineau N, Zajaczkowski J, et al. Direct hospital costs for patients with inflammatory bowel disease in a Canadian tertiary care university hospital. American Journal of Gastroenterology 2000;95(3):677-83.
11. Cohen RD, Larson LR, Roth JM, et al. The cost of hospitalization in Crohn's disease. American Journal of Gastroenterology 2000;95(2):524-30.
12. Juan J, Estiarte R, Colome E, et al. Burden of illness of Crohn's disease in Spain. Digestive & Liver Disease 2003;35(12):853-61.
13. Bassi A, Dodd S, Williamson P, Bodger K. Cost of illness of inflammatory bowel disease in the UK: a single centre retrospective study. Gut 2004;53(10):1471-8.
14. Stark R, Konig HH, Leidl R. Costs of inflammatory bowel disease in Germany. Pharmacoeconomics 2006;24(8):797-814.

15. Mesterton J, Jonsson L, Almer SH, et al. Resource use and societal costs for Crohn's disease in Sweden. Inflammatory Bowel Diseases 2009;15(12):1882-90.
16. Present DH, Rutgeerts P, Targan S, et al. Infliximab for the treatment of fistulas in patients with Crohn's disease. New England Journal of Medicine 1999;340(18):1398-405.
17. Hanauer SB, Feagan BG, Lichtenstein GR, et al. Maintenance infliximab for Crohn's disease: the ACCENT I randomised trial. Lancet 2002;359(9317):1541-9.
18. Lennard-Jones JE. Classification of inflammatory bowel disease. Scand J Gastroenterol Suppl 1989;170:2-6; discussion 16-9.
19. American Gastroenterological Association Clinical Practice C. American Gastroenterological Association medical position statement: perianal Crohn's disease. Gastroenterology 2003;125(5):1503-7.
20. Drummond MF. Methods for the economic evaluation of health care programmes. 2nd ed. Oxford ; New York: Oxford University Press, 1997.
21. Liljas B. How to calculate indirect costs in economic evaluations. Pharmacoeconomics 1998;13(1 Pt 1):1-7.
22. Johannesson M. Avoiding double-counting in pharmacoeconomic studies. Pharmacoeconomics 1997;11(5):385-8.
23. Mayberry MK, Probert C, Srivastava E, et al. Perceived discrimination in education and employment by people with Crohn's disease: a case control study of educational achievement and employment. Gut 1992;33(3):312-4.
24. Ferguson A, Sedgwick DM, Drummond J. Morbidity of juvenile onset inflammatory bowel disease: effects on education and employment in early adult life. Gut 1994;35(5):665-8.
25. Access Economics. The Economic Cost of Crohn's Disease and Ulcerative Colitis, 2007.
26. Hay JW, Hay AR. Inflammatory bowel disease: costs-of-illness. Journal of Clinical Gastroenterology 1992;14(4):309-17.
27. Hay AR, Hay JW. Inflammatory bowel disease: medical cost algorithms. Journal of Clinical Gastroenterology 1992;14(4):318-27.
28. Rosenthal RJ, Bashankaev B, Wexner SD. Laparoscopic management of inflammatory bowel disease. Dig Dis 2009;27(4):560-4.
29. Eshuis EJ, Slors JF, Stokkers PC, et al. Long-term outcomes following laparoscopically assisted versus open ileocolic resection for Crohn's disease. Br J Surg 2010;97(4):563-8.

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Screening for *Mycobacterium tuberculosis* infection among healthcare workers in New Zealand: prospective comparison between the tuberculin skin test and the QuantiFERON-TB Gold In-Tube® assay

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Abstract

A cross-sectional study was used to compare the "QuantiFERON-TB Gold In-tube" assay® (QFT-GIT) to the Mantoux tuberculin skin test (TST) as a test for *Mycobacterium tuberculosis* (TB) infection among healthcare workers in Auckland, New Zealand (NZ). New employees who underwent pre-employment interviews between 1 May 2007 and 18 March 2008 were recruited. Participants completed a detailed questionnaire to assess their risk of TB.

All participants were tested by the QFT-GIT and TST. Multiple logistic regression analysis was used to correlate positive results with risk factors for TB and previous BCG. None of the 325 participants were found to have active TB. Approximately 67% had been BCG vaccinated. Positive results for each test were associated with residence in a high incidence country [odds ratio (OR)=6.77; p=0.0005 and 4.48; p<0.0001 for QFT-GIT and TST respectively]. Although positive TST results were associated with "high-risk occupational exposure" to TB [OR=4.13; p=0.016], they were also associated with previous BCG vaccination (OR=5.10; p=0.003).

Both tests were associated with at least one well described risk factor for TB infection. The association between positive TST and BCG implies that a high percentage of positive TST results occurred due to non-specific cross-reactivity with BCG. Our findings suggest that among low TB prevalence populations with a high rate of BCG vaccination, the QFT-GIT is more specific than the TST although the superior specificity may be at the expense of some sensitivity.

As healthcare workers (HCW) are at increased risk of *Mycobacterium tuberculosis* (TB) infection,[1] many hospitals routinely screen HCW for TB infection prior to employment as a strategy to reduce the risk of nosocomial transmission of TB.

The traditional test used to screen HCW for latent TB infection (LTBI) has been the tuberculin skin test (TST). Its limitations include the need for return visits, subjectivity in reading results and false positive reactions due to Bacille Calmette-Guerin (BCG) vaccination.[2] As a consequence, when the TST is used as a pre-employment screening tool among populations of HCW with high rates of BCG, the positive predictive value of the TST may be low, particularly in countries that also have a low prevalence of TB.[3]

New tests for LTBI require a single blood sample only. These tests measure interferon-gamma (IFN-γ) release by T-lymphocytes in response to incubation with

TB-specific antigens. IGRA are more specific for LTBI than TST because they include specific antigens present in *Mycobacterium tuberculosis* but absent from BCG.[4]

We compared the performance of the QuantiFERON-TB Gold In-Tube® assay (QFT-GIT) to the performance of the TST as a pre-employment screening tool for TB infection among HCWs in Auckland, New Zealand (NZ).

Methods

Recruitment—Participants were healthcare workers who were recruited from two tertiary hospitals within Auckland, NZ: Middlemore Hospital (720 beds) and Auckland City Hospital (900 beds). Both sites routinely screen new workers for TB infection prior to employment using a two-step Mantoux test (TST). Between 1 May 2007 and 18 March 2008, employees were invited to participate in this study at their pre-employment screening interview. Exclusion criteria included pregnancy, a previously positive TST and / or a history of tuberculosis. The study was approved by the local ethics committee. All participants provided written consent. Funding for the study was provided by the Ministry of Health, Wellington NZ.

Assessment of risk factors for TB infection—A standardised questionnaire was completed by each study participant to assess their risk of TB infection. We defined "high-risk countries" as those with an incidence of TB ≥100/100 000 population; "intermediate risk countries" as those with an incidence of ≥20/100 000 population but less than 100/100,000 population; and "low risk countries" as those with an incidence of <20/100,000 population.[5]

The following information was collected: country of birth; duration of previous residence or travel to a "high-risk" or "intermediate risk" country; duration of previous employment in healthcare; history of healthcare work in intermediate or high-risk countries; history of previous employment in a TB ward, clinic or microbiology laboratory; known previous exposure to infectious TB and previous BCG vaccination (Table 1).

Demographic data included age, gender, job category and ethnicity. Participants were also asked about medications and medical conditions that might affect their immune system. "High-risk occupational exposure" was defined as previous work in an intermediate or high-risk country in one or more of the following settings: a microbiology laboratory; a respiratory ward or TB ward; a respiratory outpatient clinic or a bronchoscopy suite. TST measurements and QFT-GIT results were recorded for each participant. Phlebotomists reading TST reactions were blinded to QFT-GIT results, as were laboratory scientists to the TST results.

All chest X-rays performed in response to positive TST were assessed for evidence of TB. Referrals to the respiratory physician were also recorded as was the prescribing of TB prophylaxis or treatment. Although patients were not formally followed up as part of the study, the authors of this study were members of Infection Prevention and Control teams and would have been routinely notified in the event that any staff member developed TB.

Mantoux test (TST)—The Mantoux test was performed and interpreted as recommended by the 2003 "NZ guide for the Control of Tuberculosis".[6] Trained phlebotomy staff administered five tuberculin units of purified protein derivative by intra-dermal injection. Results were read by the same staff after 48 to 72 hours Individuals with a TST that was initially negative underwent a second TST one week later. The second test was performed and interpreted using the same cut offs that were applied to the first test.

Quantiferon-TB Gold In-Tube test (QFT-GIT)—Whole blood was collected for the QFT-GIT immediately prior to inoculation of PPD for the TST. One ml of blood was collected into each of three tubes provided by the manufacturer. Following inoculation, each tube was mixed thoroughly.

The tubes were transported within 12 hours of collection to the testing laboratory at room temperature and were incubated for 24 hours at 37˚ C. Following incubation, the tubes were refrigerated at 4˚C for up to two weeks before being tested in batches. IFN-γ concentrations were measured by ELISA using the Trituris® automated platform. Test results were interpreted according to the manufacturer's instructions.[7]

Statistical analyses—Statistical analyses were performed using Stata software. Concordance between the TST and QFT-GIT were calculated using the kappa statistic. Univariate and stepwise logistic regression analyses were used to examine the association between positive results of either test, and risk factors for LTBI / previous BCG vaccination. The multivariate models were done separately for positive TST and QFT as the outcome variables and included age; gender; birth in high-risk country; "high-risk occupational exposure" and "residence in a high-risk country for >10 years" as predictors. Stepwise procedures were done to determine the key predictors. Mean TST diameters were compared between groups using the unpaired t-test. P values of < 0.05 were considered statistically significant.

Results

A total of 1839 HCWs underwent pre-employment screening during the study period and 343 (19%) were recruited. Data on the reason for non-participation were available on a sample of 194 out of the 1496 non-participants. All 194 were ineligible for inclusion in the study because they had a previously positive TST.

Of the 343 recruits, 18 [5%] were excluded due to one or more of the following: (1) not being tested by TST, (2) not being tested by QFT-GIT or (3) not filling in the questionnaire. This left a total of 325 HCW: 192 HCW [59.1%] from Auckland City Hospital and 133 HCW [40.9%] from Middlemore Hospital. Further analysis was carried out on this remaining group of 325.

A number of participants were uncertain of their previous BCG vaccination status (31/325, 9.5%). Of the 294 with known BCG status, 217 had been previously vaccinated (73.8%, see Table 2). Other characteristics of study participants are detailed in Table 2.

Overall, there was 80% agreement between the two tests (κ=0.315, 95% confidence interval: 0.153-0.476). Table 1 shows the breakdown of results. Concordant results occurred less frequently among BCG vaccinated HCW than unvaccinated HCW (168/217 [77.4%] vs 64/70 [91.4%]; p=0.0087).

Subjects with positive QFT-GIT results had a significantly higher mean TST diameter than subjects with negative QFT-GIT results (difference between the two means=8.69mm [95% CI 6.39–10.98]; p<0.0001). Nine participants (2.8%) had a positive QFT-GIT but a negative TST. Eight (2.5%) had indeterminate QFT-GIT results. One had an 18mm TST and had been born in a high incidence country and had worked on a respiratory ward in that country before immigrating to New Zealand. The other seven HCW had negative TST. Only one HCW with an indeterminate result was immune-compromised.

Both tests were strongly associated with numerous risk factors for TB infection by univariate analysis (Table 3). Following stepwise logistic regression, only previous residence in a high-risk country for > 10 years was independently associated with positive results for both of the two tests, whereas positive TST results were additionally associated with high-risk occupational exposure and previous BCG (Table 4).

Of the 28 subjects with positive QFT-GIT results, 19 subjects also had a positive Mantoux result and were thus referred for chest X-ray. Approximately 16% (3/19) had chest X-ray changes that warranted referral to a respiratory physician. Of the 68 subjects with positive TST results, 62 had a follow up chest X-ray.

Four of these subjects (6.5%) had chest X-ray changes that prompted referral. Three of these four subjects also had a positive QFT-GIT. No study subjects were treated for either latent or active TB and no study subjects were known to have developed suspected or confirmed active TB during the 18 months following completion of the study.

Table 1. Agreement between results of the QFT-GIT and TST

Variables	Positive TST[b] (%)	Negative TST[b] (%)	Total (%)
Positive QFT-GIT[a]	19 (5.8)	9 (2.8)	28 (8.6)
Negative QFT-GIT[a]	48 (14.8)	241 (74.2)	289 (88.9)
Indeterminate QFT-GIT[a]	1 (0.3)	7 (2.2)	8 (2.5)
Total	**68 (21)**	**257 (79.1)**	**325 (100)**

[a] QFT-GIT=QuantiFERON-TB Gold In Tube test; [b] TST=Tuberculin Skin Test.

Table 2. Characteristics of study participants

Characteristic	Number of patients (%)
Age 18–30 years	160 (49.2)
Age 31–50 years	136 (41.8)
Age 51–60 years	29 (8.9)
Male	55 (16.9)
Female	270 (83.1)
Nurse or allied health professional	247 (76.0)
Doctor	29 (8.9)
Clerical	6 (1.8)
Laboratory worker	7 (2.2)
Healthcare assistant	36 (11.1)
BCG vaccinated	217 (66.8)
BCG unvacccinated	77 (23.7)
BCG vaccination status unknown	31 (9.5)
Born in New Zealand or other low risk country	211 (64.9)
Born in medium risk country*	27 (8.3)
Born in high-risk country*	78 (24)
Place of birth unknown	9 (2.8)
No previous travel to / residence in high-risk country	240 (73.8)
<1 year travel / residence in high-risk country	29 (8.9)
1–10 years residence in high-risk country	13 (4.0)
>10 years residence in a high-risk country	43 (13.2)
No previous healthcare work in high-risk country	289 (88.9)
<1 year previous healthcare work in high-risk country	14 (4.3)
>1 year previous healthcare work in high-risk country	22 (6.8)
Previous household exposure to confirmed pulmonary TB	5 (1.5)
NO previous household exposure to confirmed pulmonary TB	320 (98.5)

Source: Global Tuberculosis Database 2006. * Risk categories defined in text using figures provided by the World Health Organization.

Table 3. Association between positive results by the TST and QFT-GIT and risk factors for TB infection (univariate analysis)

Risk factor	Positive QFT-GIT[a] OR[c] (95% CI)	Positive TST[b] OR[c] (95% CI)
Age>50 years	0.33 (0.10–1.05) p=0.07	0.24 (0.10–0.604) p=0.001
Male gender	1.72 (0.71–4.18) p=0.29	0.93 (0.46–1.91) p=1.0
Birth in a high-risk country	5.96 (2.69–13.17) p<0.0001	4.29 (2.41–7.64) p<0.0001
Lived in high-risk country >10 years	8.30 (3.64–18.97) p<0.0001	6.79 (3.41–13.49) p<0.0001
Previous healthcare work in high-risk country >1 year	8.71 (3.32–23.00) p<0.0001	7.36 (2.98–18.16) p<0.0001
Previous BCG	4.12 (1.05–16.09) p=0.05	7.14 (2.60–19.54) p<0.0001
Worked over 10 years in healthcare	1.93 (0.87–4.27) p=0.12	1.57 (0.92–2.68) p=0.10
"High-risk occupational exposure"	5.02 (1.70–14.96) p=0.011	8.07 (2.96–21.94) p<0.0001

[a] QFT-GIT=QuantiFERON-TB Gold In Tube test; [b] TST=Tuberculin Skin Test; [c] OR=Odds ratios

Table 4 Association between risk factors for TB infection with positive results for the TST and QFT-GIT (multivariable analysis)

Risk factor	Positive QFT-GIT[a] OR[c] (95% CI)	Positive TST[b] OR[c] (95% CI)
Previous BCG	2.53 (0.56–11.51) p=0.23	5.10 (1.75–14.84) p=0.003
Lived in high-risk country >10 years	6.77 (2.70–16.90) p=0.0005	4.48 (2.11–9.49) p<0.0001
High-risk occupational exposure	2.24 (0.63,8.01) p=.214	4.13 (1.30–13.06) p=0.016

[a] QFT-GIT=QuantiFERON-TB Gold In Tube test; [b] TST=Tuberculin Skin Test; [c] OR=Odds ratios.

Discussion

Whilst a large proportion of HCW in our study were born in high incidence ("high-risk") countries (approximately 30%), NZ itself has a relatively low incidence of TB (<10/100 000). Among all participants, only four (1.2%) had an abnormal chest X-ray and none received either prophylaxis or treatment for TB. Of note, approximately 9% of those who had a positive Mantoux and 32% of those with a positive QFT-GIT failed to get a follow up chest X-ray. The reasons for this are uncertain although it is reassuring that no HCW at either study site developed active TB either during the study period or during the 18 months following study completion.

Although our HCW population had a low incidence of TB infection, the rate of BCG-vaccination was high (~70%). BCG vaccination is known to cause false positive TST reactions.[8] We predicted therefore that the TST would have poor positive predictive value in our population of HCW. This hypothesis is supported by the two major findings of our study. More specifically, we found that positive TST results were twice as common as positive QFT-GIT results (20.9% vs 8.6%; p<0.0001).

Secondly, we found that, positive TST results (but not positive QFT-GIT results) were independently associated with previous BCG vaccination. These findings are consistent with a number of previously published studies.[8-13] For example an Australian study of 481 HCW with 78% BCG coverage found that positive TST results were five times more common than positive QFT-GIT.[9] Similar findings have also been reported in studies of HCW in countries such as Denmark, Germany and Italy.[10-12]

On the other hand, some studies have yielded slightly different results from our study and the studies above.[14-17] For example, a study of 332 HCW in Japan failed to detect an association between positive TST results and risk factors for TB.[16] One possible explanation for this inconsistency is the relatively *high* rate of BCG vaccination (95%) and relatively *low* rate of TB infection in Japan compared to NZ. Thus, a relatively high proportion of positive TST results would have been caused by BCG vaccination in the Japanese study compared to ours.

Slightly different findings from ours were also obtained from a study performed on HCW in rural India. [17] In that study, despite 71% of HCW being previously vaccinated with BCG, no association was observed between previous BCG and positive TST results. The inconsistency between the finding of this study and others can be explained by the exceptionally high prevalence of TB infection among HCW in rural India: If the sensitivity of TST and QFT-GIT are assumed to be similar, the tests will demonstrate higher levels of agreement as the number of "true positive" cases of TB infection increases. The apparent inconsistencies between these studies and ours illustrate how the characteristics of a HCW population can affect the predictive values of both the QFT-GIT and TST.

Yet other studies have yielded similar findings to ours but have drawn quite different conclusions.[18] For example in a study of American HCW, 93% of HCW had been vaccinated with BCG and positive TST results were approximately 5 times more common than positive QFT-G. However, the authors of that study suggest that the primary reason for the discordance between QFT-G and TST results was *not* superior specificity of the QFT-G but the superior sensitivity of the TST. If the QFT-GIT does indeed have a lower sensitivity than the TST then this could explain why only positive TST results were associated with "high-risk occupational exposure to TB" in our study.

Thus, our study findings can be interpreted as supporting the notion that the QFT-GIT is *both* more specific *and* less sensitive than the TST. This notion has also been supported by a recent study that used a statistical model to define LTBI.[19] Furthermore a meta-analysis of studies using culture-proven active TB as a gold standard found that the TST had a "pooled sensitivity" of 78% (95% confidence interval: 73-82%) whereas the pooled sensitivity of the QFT-GIT was slightly lower at 70% (95% confidence interval: 63-78%).[20]

We agree therefore with the recommendation of the CDC that "each QFT-G result and its interpretation should be considered in conjunction with other epidemiologic, historic, physical and diagnostic findings".[21] Thus for HCW at low to medium risk of TB, a positive QFT-GIT should prompt further investigation for TB infection whereas for those at very high-risk of TB, investigations should be carried out regardless whether or not the QFT-GIT is positive. Although we did not specifically assess rates of negative or positive QFT-GIT results following results that were initially indeterminate, our local experience suggests that approximately two thirds will have a positive or negative result on repeat testing.

Our study had several limitations. Firstly, only 19% of all those who underwent pre-employment screening participated in the study. However, most of the non-participants had a previously positive TST and were therefore not eligible for inclusion in the study. Thus the participation rate of 19% would be far higher if only

eligible HCW were considered. Nevertheless, a relatively low recruitment rate may have meant that our cohort was not a representative sample of new HCW across the two hospitals. Secondly, BCG vaccination status was not known for 9.5% of our study subjects. Thirdly, our study design did not include systematic, active follow-up study subjects in order to determine the rate of progression to active TB. Finally, our study did not directly compare the costs of a screening protocol based on QFT-GIT with one based on the TST. However our data does suggest that there are many downstream costs associated with false positive TST results including a large number of unnecessary chest-X-rays and follow-up appointments.

Moreover, it is likely that many more unnecessary X-rays were carried out during the study period than we recorded due to the many HCW with previously positive Mantoux results who did not participate in the study. Avoidance of the expense associated with unnecessary chest X-rays and clinic appointments will offset the increased "up front" cost of the QFT-GIT compared to the TST, although the extent to which this is true remains to be determined in future studies.

In conclusion we found that among HCW in NZ, the QFT-GIT produced significantly fewer positive results than the TST. While it is possible that this finding may be partly explained by the QFT-GIT having a lower sensitivity than the TST, the strong independent association between positive TST and previous BCG provides additional evidence that the QFT-GIT is markedly more specific than the TST. Thus the findings of our study provide further evidence that the QFT-GIT is a useful tool that can be used to screen for TB in populations of HCW with high rates of BCG vaccination.

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References:

1. Sepkowitz KA. Tuberculosis and the health care worker: a historical perspective. Ann Intern Med 1994;120:71-79.
2. Snider, DE Jr. Bacille Calmette-Guerin vaccination and tuberculin skin tests. JAMA 1985;253:3438-3439.
3. Edwards PQ, Edwards LB. Story of the tuberculin skin test from an epidemiologic viewpoint. Am Rev Respir Dis 1960;81:1-47.
4. Mori T, Sakatani M, Yamagishi F et al. Specific detection of tuberculosis infection: An interferon-γ-based assay using new antigens. Am J Respir Crit Care Med 2004;170:59-64. doi:10.1164/rccm 200402-1790C.
5. The World Health Organization: Global Tuberculosis Database 2006.
6. The New Zealand Ministry of Health: Guidelines for Tuberculosis Control in New Zealand 2003.
7. Cellestis. QuantiFERON-TB Gold IT method and technical Instructions. Melbourne, Australia: Cellestis 2007.
8. Tissot F, Zanetti G, Francioli P, Zellweger J, Zysset F. Influence of Bacille Calmette-Guerin vaccination on size of tuberculin skin test reaction: to what size? Clin Infect Dis 2005;40:211-217. doi:10.1086/426434.
9. Ozdemir, D, Annakaya A, Tarhan G et al. Comparison of the Tuberculin Skin Test and the Quantiferon test for latent Mycobacterium tuberculosis infections in health care workers in Turkey. Jpn J Infect Dis 2007;60:102-105.
10. Vinton P, Mihrshahi S, Johnson P et al. Comparison of QuantiFERON-TB Gold In-Tube Test and Tuberculin Skin Test for identification of latent Mycobacterium tuberculosis infection in healthcare staff and association between positive test results and known risk factors for infection. Infect Control Hosp Epidemiol 2009;30:215-221. doi: 10.1086/595695.
11. Soborg B, Andersen A, Larsen H et al. Detecting a low prevalence of latent tuberculosis among health care workers in Denmark detected by M. tuberculosis specific IFN-γ whole-blood test. Scand J Infect Dis 2007;39:554-559. doi: 10.1080/00365540601148483.
12. Neinhaus A, Schablon A, Le Bacle C, Siano B, Diel R. Evaluation of the interferon-γ release assay in healthcare workers. Int Arch Occup Environ Health 2008;81:295-300. doi:10.1007/s00420-007-0212-1.
13. Carvalho A, Crotti N, Crippa M et al. QuantiFERON-TB Gold test for healthcare workers. J Hosp Infect 2008;69:91-92. doi:10.106/j.jhin.2008.01.032.
14. Alvarez-Leon E, Espinosa-vega E, Santana-Rodiguez E et al. Screening for Tuberculosis infection in spanish healthcare workers: Comparison of the QuantiFERON-TB Gold In-Tube Test with the Tuberculin Skin Test. Infect Control Hosp Epidemiol 2009;30:876-883. doi:10.1086/598519.
15. Cummings K, Smith T, Shogren E et al. Prospective comparison of Tuberculin Skin Test and QuantiFERON-TB Gold In-Tube assay for the detection of latent Tuberculosis infection among healthcare workers in a low-incidence setting. Infect Control Hosp Epidemiol 2009;30:1123-1126. doi: 10.1086/644754.
16. Harada N, Nakajima Y, Higuchi K, Sekiya Y, Rothel J, Mori T. Screening for Tuberculosis infection using whole-blood interferon-γ and Mantoux testing Among Japanese healthcare workers. Infect Control Hosp Epidemiol 2006;27:442-448.
17. Pai M, Gokhale K, Joshi R et al. Mycobacterium tuberculosis infection in health care workers in rural India: Comparison of a whole-blood interferon-γ assay with Tuberculin Skin Testing. JAMA 2005;293:2746-2755.
18. Pollock N, Campos-Neto A, Kashino S et al. Discordant QuantiFERON-TB Gold test results among US healthcare workers with increased risk of latent Tuberculosis infection: A problem or solution? Infect Control Hosp Epidemiol 2008;29:878-886. doi:1086/590262.

19. Girardi E, Angeletti C, Puro V et al. Estimating diagnostic accuracy of tests for latent Tuberculosis infection without a gold standard among healthcare workers. Euro Surveill 2009;14.
20. Pai M, Zwerling A, Menzies D. Systematic review: T-Cell-based assays for the diagnosis of latent Tuberculosis infection: An update. Ann Intern Med 2008;149:177-184.
21. Mazurek G, Jereb J, Iademarco M, Metchock B, Vernon A. Guidelines for using the QuantiFERON-TB Gold test for detecting Mycobacterium tuberculosis infection, United States [erratum appears in MMWR Morb Wkly Rep 2005;54:1288]. MMWR Recomm Rep 2005;54:49-55.

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Clinical audit of stroke thrombolysis in Wellington, New Zealand: disparity between in-hours and out-of-hours treatment time

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Abstract

Aims To report on the safety and efficiency of a comprehensive stroke thrombolysis service and look for evidence of disparity between in-hours and out-of-hours treatment times.

Method Clinical audit of patients treated with tissue plasminogen activator, alteplase (rt-PA) for stroke at Wellington Hospital between 1 November 2009 and 31 October 2010.

Results Thirty-one patients were treated with rt-PA. All were treated within agreed clinical eligibility criteria. The median NIHSS score pre-treatment was 10; post treatment 5. Two patients died, both from intracranial haemorrhage. Overall the average time to treatment from symptom onset was 168 minutes. Those treated out-of-hours had an additional delay of 33 minutes compared to in-hours treatment (p=0.03).

Conclusions Patients admitted out-of-hours had significantly longer delays to rt-PA treatment. Those planning Stroke Services should ensure this source of inequity is addressed within their localities.

Stroke is a common cause of death, disability and institutionalisation in New Zealand. Around one half of those that suffer a stroke will remain permanently disabled.[1] For 16 years evidence has accumulated that confirms that recombinant tissue plasminogen activator, alteplase (rt-PA) can reduce the combined outcome of death and disability if given within three hours of stroke onset.[2]

Recently published New Zealand Guidelines recommend the use of rt-PA for selected individuals up to four and a half hours from stroke onset.[3] It has been demonstrated that rt-PA can be safely delivered within the New Zealand Health system.[4] Despite these evidence-based guidelines, the burden of disability from stroke and the evidence that rt-PA can be safely administered, there remains disparity in access to thrombolysis treatment for stroke between hospitals in New Zealand.[5]

A twenty-four hour service to deliver thrombolysis treatment for stroke was introduced at Wellington Regional Hospital in November 2009. This clinical audit reports the safety and efficiency of this model and examines whether there is a disparity between in-hours and out-of-hours treatment times. The purpose is to inform and stimulate discussion around the development of equitable, comprehensive stroke thrombolysis service models within New Zealand.

Method

The medical records of all patients treated with rt-PA between 1 November 2009 and 31 October 2010 were examined. Data collected included time of stroke onset to ambulance call; arrival in the emergency department (ED); ED assessment; CT scan and administration of rt-PA.

The delivery of rt-PA is driven by a strict protocol and clinical eligibility criteria agreed by the Stroke Physician Leads at Capital and Coast, Hutt and Wairarapa District Health Boards (Table 1). All patients undergo CT scan prior to treatment and have a further scan between 24 and 36 hours after treatment. The National Institute of Health Stroke Scale (NIHSS) scores are calculated pre-treatment and 24 hours post treatment. Final place of discharge is recorded for all patients.

Symptomatic intracranial haemorrhage is classified as any blood on the second CT scan and deterioration in NIHSS of greater or equal to 4 or leading to death. Asymptomatic haemorrhage is classified as any blood on the second CT scan with or without a deterioration in NIHSS score <4.[6]

In-hours care is considered as weekdays Monday to Friday 08:00 to 16:00, excluding public holidays, during which period rt-PA is delivered by the onsite stroke team comprising of a stroke specialist nurse, stroke registrar and stroke physician. All other periods are categorised as out-of-hours and rt-PA is delivered by the oncall medical team comprising two medical registrars, a house surgeon and an offsite stroke specialist.

Statistical analysis was performed using unpaired two-tailed Student t-tests for parametric data. A p value of <0.05 was considered significant.

Table 1. Clinical eligibility criteria for treatment with alteplase (rt-PA)

All patients must have: – Clinical diagnosis of ischaemic stroke with measurable deficit – Onset of symptoms well established to be <4.0 hours
Absolute clinical contraindications to treatment: – Profound stroke with obtundation, fixed eye deviation AND complete hemiplegia – Clinical presentation consistent with SAH, even if CT normal – Any evidence of haemorrhage on initial CT – CT/MRI – infarct >1/3 MCA territory – Active internal bleeding – Stroke, intracranial surgery or head trauma (last 3 months) – Any history of intracranial haemorrhage, AVM, aneurysm – Recent major surgery or organ biopsy (14 days) – Recent transmural MI or post-MI pericarditis (3 weeks) – Platelet count $<100\times10^9$/L – INR >1.5 (if patient not on warfarin, treatment can begin pending INR result) – IV heparin AND have an APTT >40 sec – Any LMWH in last 48 hours – Pregnancy or parturition last 30 days – Despite treatment, systolic BP >185 mmHg or Diastolic BP >110 mmHg
Relative contraindications (presence of two or more absolutely contraindicate treatment): – Minor or rapidly improving symptoms – Recent lumbar puncture or arterial puncture at non-compressible site (14 days) – Blood sugar <3 or >22 mmol/L – INR between 1.3–1.5 – History of GI/GU or other internal bleeding within 21 days – Any seizure – Obvious small vessel disease on initial CT – Age > 80 years – Not ambulatory and/or dependent on others for ADLs and/or frailty

Results

Thirty-one people received rt-PA over the 12-month period, 16 of whom received treatment in-hours and 23 of whom were male. The average age of patients was 69 years, with range 43–87 years. Demographic details are shown in Table 2.

Table 2. Demographic details for those treated in-hours and out-of-hours with alteplase (rt-PA)

Variables	In-hours (n=16)	Out-of-hours (n=15)	Overall
Male, number	11	12	23
Average age, years (range)	68 (43–87)	69 (44–86)	69 (43–87)
Pre-treatment NIHSS, median (range)	12 (4–26)	10 (4–22)	10 (4–26)
Post-treatment NIHSS,median (range)	6 (0–23)	3 (0–14)	5 (0–23)

There was no significant difference in age, gender or NIHSS scores between those who presented in-hours compared with out-of-hours.

Treatment times for the overall group are presented in Table 3.

Table 3. Treatment times for all those given alteplase (rt-PA)

Variables	Average time in minutes (range)
Symptom onset to ambulance	21 (0–83)
Ambulance to ED	43 (0–89)
ED Assessment	20 (0–51)
ED to CT scan	35 (1–53)
CT scan to rt-PA	48 (7–150)
Average time to treatment	168 (88–240)

Table 4 compares treatment times in-hours versus out-of-hours.

Table 4. Comparison between treatment times in-hours and out-of-hours for those given alteplase (rt-PA)

Variables	Average time in minutes (range) in-hours (n=16)	Average time in minutes (range) out-of-hours (n=15)	Significance (p value)
Symptom onset to ambulance	22 (0–83)	20 (0–68)	0.77 NS
Ambulance to ED	42 (0–82)	43 (9–71)	0.89 NS
ED assessment	21 (0–50)	19 (2–51)	0.72 NS
ED to CT scan	31 (1–63)	38 (17–59)	0.26 NS
CT scan to rt-PA	34 (8–71)	62 (7–150)	0.01** S
Average time to treatment	152 (88–225)	185 (115–240)	0.03** S

Safety—Two of the 31 patients died (6.5%), both from intracranial haemorrhage. Both suffered haemorrhagic transformations within large embolic infarcts; one in anterior circulation and the other middle cerebral artery territories. There were no other symptomatic intracranial haemorrhages.

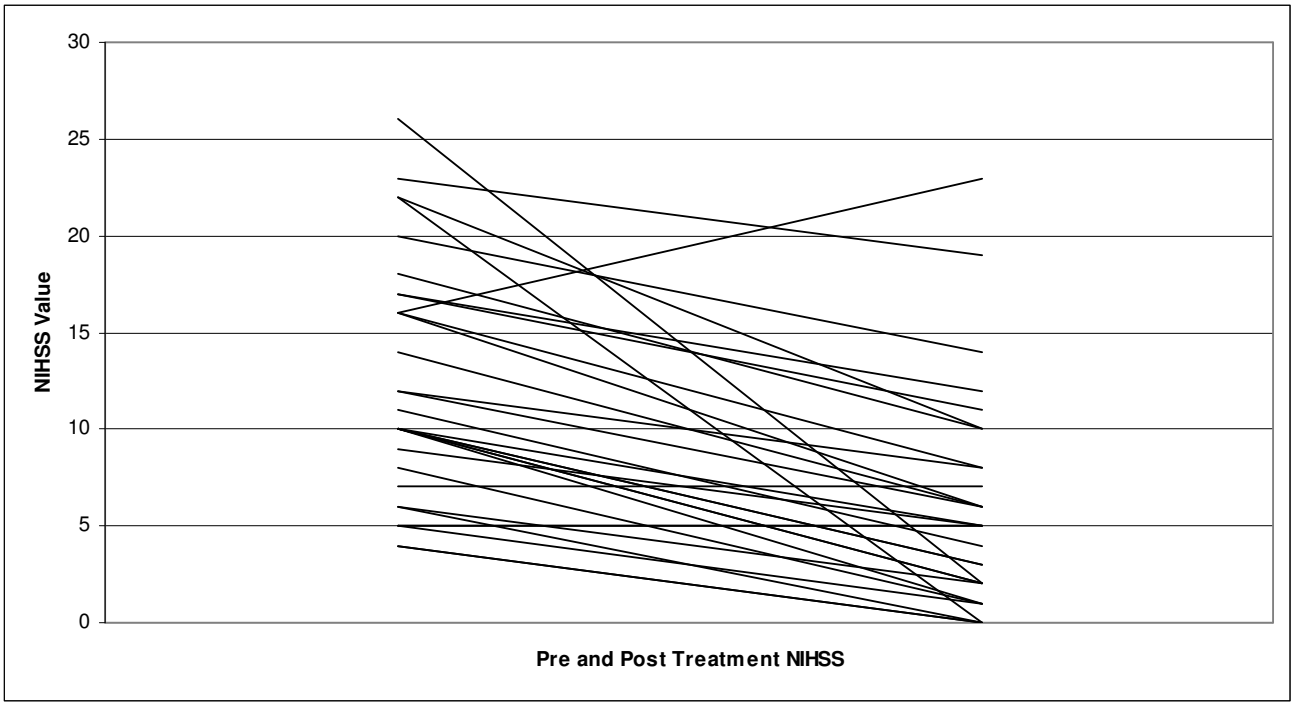
Three patients had small petechial intracranial haemorrhage without any deterioration in NIHSS score.

Three further patients had minor gastrointestinal bleeds that required no specific treatment.

All patients were treated within the clinical eligibility criteria with no protocol violations.

Outcome—Median NIHSS pre-treatment was 10 (range: 4–26). Median post treatment NIHSS was 5 (range: 0–23) (Figure 1); 24 patients were able to return to their own homes, 4 patients went to live with relatives, 1 patient needed nursing home care and 2 died.

Figure 1. Individual changes in NIHSS scores pre- and post-treatment with alteplase (rt-PA)



Note: Two patients had NIHSS change 4 to 0; two patients 10 to 2; two patients 10 to 3 and two 5 to 5.

Discussion

Wellington delivers rt-PA efficiently and within agreed clinical eligibility criteria. The symptomatic intracranial haemorrhage rate (6.5%) is consistent with other published data from other centres in New Zealand and internationally though the

absolute numbers of events are too low to draw robust conclusions.[2,4,7] To assist those not familiar with the NIHSS scale a score <5 could be considered a 'minor' stroke, 5–15 a 'moderate to severe' stroke and >15 a 'severe to devastating' stroke. Of interest is the dramatic improvement in some individuals who presented with NIHSS scores >23 whom may have been assessed previously as not eligible for thrombolysis (Figure 1).[7,8]

There is growing evidence that rt-PA can be safely administered to people who would have been excluded from treatment in the original trials, for example those aged over 80 years.[8] Stroke Physicians from Wellington, Hutt and Wairarapa District Health Boards met to review the current evidence and developed clinical eligibility criteria that allow discretion in decision-making (Table 1). The criteria are reviewed annually. There are other examples of centres using this approach which supports treatment within a changing evidence base.[8,9]

This collaborative approach drove the development of stroke thrombolysis services across the three District Health Boards. We acknowledge a lack of conformity in eligibility criteria for thrombolysis across New Zealand. Based on our experience a national process for review and revision of rt-PA eligibility criteria could become a catalyst to ensure this treatment is made available throughout the country. It is an approach we would recommend.

Overall assessment and treatment times were similar to those reported previously by Christchurch Hospital.[4] Although comparisons between hospitals can be problematic, Christchurch reported a time from stroke onset to rt-PA bolus of 150 minutes for those treated in-hours which is remarkably consistent with our experience of 152 minutes (Table 4). In both centres potential candidates for rt-PA are given priority within the ambulance, ED and CT scan services and results show this process works well. The surprising result in Wellington is the prolonged time taken from CT scan to rt-PA treatment particularly out-of-hours.

The audit demonstrated an important disadvantage in treatment times for rt-PA given out-of-hours (Table 4). This is of relevance to those planning stroke service models of care. The number needed to benefit for one additional patient to have a good outcome for rt-PA is 3.6 if treated within 90 minutes, 4.3 if treated within 90–180 minutes and 5.9 if treated within 180–270 minutes.[10] Our numbers treated were too small to show differences in outcome, however the evidence is that thirty minutes delay has notable implications and so this disparity in time to treatment is clinically significant.

Internationally there is evidence to show increased mortality amongst those admitted as medical emergencies out-of-hours and at weekends.[11,12] Disadvantage in outcome has also been found specifically in those admitted with stroke at weekends and out-of-hours.[13-15] Possible explanations such as reduced clinical staffing, availability of on-site senior medical officers and access to diagnostics have been suggested.[11-15]

This audit demonstrates that treatment delay after CT scan is a highly relevant consideration and accounted for the disparity in treatment times. Our delay was not due to CT scan reporting rather the availability of medical staff. Out-of-hours the medical registrar was more likely to be diverted to other tasks rather than accompany the patient to the CT scan. Subsequently when the patient returned to ED for

treatment the registrar had to be re-called. In-hours the stroke registrar remains with the patient from ED to CT scan to rt-PA treatment.

To address this disadvantage we propose that when a stroke patient arrives in ED out-of-hours a doctor or senior nurse remain with the patient until either the drug is administered or deemed contraindicated. This clinician will drive the speed of the process and alert the stroke consultant as soon as the scan is arranged. The stroke consultant can review the CT scan immediately and make the treatment decision. This should reduce time from CT scan to rt-PA treatment. As this time period accounts for the disadvantage in out-of-hours care this should be reduced by this recommendation.

Stroke is common and rt-PA treatment substantially reduces disability. rt-PA should be available to all who suffer a stroke and fulfil eligibility criteria. This is currently not the situation in New Zealand. This clinical audit has demonstrated an inequitable delivery of treatment out-of-hours. We will address this disadvantage in Wellington and hope other Stroke services are inspired to do the same.

Competing interests: None declared.

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References:

1. Warlow C, Sudlow C, Dennis M, et al. Stroke. Lancet. 2003;362:1211-1224.
2. Ingall TJ, O'Fallon WM, Asplund K, et al. Findings from the Reanalysis of the NINDS Tissue Plasminogen Activator for Acute Ischaemic Stroke Trial. Stroke. 2004;35(10):2418-2424.
3. Stroke Foundation of New Zealand and New Zealand Guidelines Group. Clinical Guidelines for Stroke Management 2010. Stroke Foundation of New Zealand. 2010.
4. Fink J. Twelve months experience of acute stroke thrombolysis in Christchurch, New Zealand: emergency department screening and acute stroke service treatment. N Z Med J. 2005;118(1214): http://www.nzma.org.nz/journal/118-1214/1430/
5. Stroke Foundation of New Zealand. National Acute Stroke Audit 2009. Stroke Foundation of New Zealand. 2010.
6. Hacke W, Kaste M, Bluhmki E, et al. Thrombolysis with Alteplase 3 to 4.5 hours after ischaemic stroke. N Eng J Med. 2008;359:1317-1329.
7. The National Institute of Neurological Disorders and Stroke rt-PA Study Group. Tissue Plasminogen Activator for acute ischaemic stroke. N Eng J Med. 1995;333:1581-1587.
8. Meretga A, Putaala J, Tatlisumak T, et al. Off-label Thrombolysis is not associated with poor outcome in patients with stroke. Stroke. 2010;415:1450-1458.
9. The Brain Attack Coalition. TPA study Group Guidelines. http://www.stroke-site.org/guidelines/tpa_guidelines.html
10. Lansberg MG, Schrooten M, Bluhmki E, et al. Treatment Time Specific Number Needed to Treat Estimates for Tissue Plasminogen Activator Therapy in Acute Stroke Based on Shifts over the Entire Range of Modified Rankin Scale. Stroke. 2009;40(6):2079–2084.
11. Maggs F, Mallet M. Mortality in out-of-hours emergency medical admissions–more than just a weekend effect. J R Coll Physicians Edinb. 2010;40(2):115-118.

12. Aylin P, Yunnis A, Bottle A, et al. Weekend mortality for emergency admissions. A large multi-centre study. Qual Saf Health Care. 2010;19:213-17.
13. Hasegawa Y, Yoneda Y, Okuda S, et al. The effect of weekends and holidays on stroke outcome in acute stroke units. Cerebrovasc Dis. 2005;20(5):325-31.
14. Fang J, Saposnik G, Silver FL, Kapral MK. Association between weekend hospital presentation and stroke fatality. Neurology. 2010;75(18):1589-96.
15. Saposnik G, Baibergenova A, Bayer N , Hachinski V. Weekends: A Dangerous Time for Having a Stroke? Stroke. 2007;38:1211-1215.

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Training medical students in Pacific health through an immersion programme in New Zealand

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Abstract

Aims Medical schools are required to adequately prepare students to work in increasingly diverse and multi-ethnic societies. Students need to be able to integrate clinical knowledge with an understanding of the society they live in. Pacific peoples are a disadvantaged migrant minority ethnic group in New Zealand. This paper discusses the development of, and lessons learnt from a Pacific Immersion Programme for medical students at the University of Otago, New Zealand.

Methods A cultural programme was developed where fourth-year medical students spent a week-end with a local Pacific family in Dunedin. Students were invited as part of the programme evaluation to provide feedback on their experiences and lessons learnt. Student evaluations were analysed and are reported here in summary form.

Results Medical students were able to learn from observations, participation in activities and stories shared by families about issues that influenced the health of the community. This provided insight about factors that are important to consider, when working with Pacific peoples in New Zealand. The programme also provided positive benefits for the local community.

Conclusions This cultural immersion programme provided important learning opportunities for medical students. It is important to value and empower communities when developing cultural teaching programmes. The incorporation of the programme as part of the curriculum, and its implications for overall assessment and performance of students, makes it a valued part of learning.

The importance of ensuring that medical students are adequately prepared to work in increasingly diverse and multi-ethnic societies is well recognised from international research.[1-5] This has resulted in efforts to incorporate the teaching of culture and cultural competencies in medical schools in the US,[3] Canada,[4] Sweden,[6] and New Zealand.[7] These efforts include language training, lectures, workshops, electives, rotations and cultural immersion programmes.[7–13]

Medical schools are challenged to teach students how to integrate clinical knowledge and expertise with an understanding of the society they live in, so they are better able to respond to needs in the community.[14] The significant implications for students, staff and the community make culture and cultural competence an important subject in medical education.[14–16] Some studies have reported positive changes from the inclusion of "culture and medicine" in the curriculum.[13,17] For example, the evaluation of knowledge, attitude and skills in cultural competence appeared to improve following cultural teaching for medical students.[15]

The literature identifies the importance of having a longitudinal thread of "culture and medicine" teaching in the curriculum.[18,19] Isolated or disconnected lectures may not enable students to acquire adequate knowledge and skill. Some research however found cultural training failed to show a change in the knowledge and attitudes of medical students.[16] There is caution about continuing disparities if physicians are not able to engage effectively with at-risk communities.[18,20]

The proper evaluation of education in "culture and medicine" and assessment of students' competencies require investment of time and adequate resources. The areas that can be assessed in cross-cultural education are students' attitudes, knowledge and skills.[1] Assessment of knowledge and skills is relatively easy to do but assessing students' attitudes remains a challenge.[9] For example, quantitative tools based on social cognitive theory used by social psychologists to assess attitudes are long and cumbersome.[21]

Students may also elect to answer questions in a socially desirable way. A review of the literature on cross cultural experiences and their benefits identified 42 studies,[22] and articulated the need for more rigorous methods to assess outcomes. The development of appropriate assessment and evaluation methods on how "culture and medicine" teaching impacts on students' learning was recommended.

Pacific peoples: a migrant population in New Zealand

New Zealand has had a unique relationship and influence as a colonial power in the Pacific Islands over many years.[23] In the 1950s, many Pacific workers were actively recruited from the Pacific Islands to provide the workforce New Zealand needed for many industries and manufacturing sectors.[24,25] This was during economic prosperous times after World War 2.

The New Zealand Government changed its attitude towards Pacific workers during the economic recession in the 1970s.[26] Previously, the government and employers ignored Pacific workers' requirements to have appropriate work permits or renewal of these permits when they expired. During the recession, the government's solution to addressing economic problems was to expel Pacific overstayers through the unfortunate "dawn raids". This affected the health and wellbeing of all Pacific peoples living in New Zealand, and stimulated the organisation of Pacific peoples as a political group. Pacific peoples now living in New Zealand make up approximately 7% of the total New Zealand population, and are a diverse minority group.[27]

In contrast to the total population, the Pacific population has a younger age structure (median age 21 compared to 36 in the total population), and is predicted to grow to 9.6% of the total population by 2026.[27]. Pacific peoples suffer disproportionately from poor health and other socioeconomic disadvantages compared to the total population.[28]

Only 1% of all doctors working in New Zealand have Pacific ethnicity,[29] therefore most Pacific peoples who access health services will be cared for by non-Pacific health professionals. It is very important to provide opportunities for all medical students to learn about Pacific peoples, their cultures and factors that influences their health.

The New Zealand Medical Council recently published a resource booklet for health professionals outlining practice implications when working with Pacific peoples.[30] The Ministry of Health has guidelines on Pacific cultural competencies,[31] and a national Pacific plan to assist health professionals working with Pacific communities.[32]

University of Otago and community engagement

Engaging at-risk communities in the work of training institutions is seen as important in making a difference for these communities.[33] For example, Duke University and the city of Durham developed a set of "Principles of Community Engagement" to assist in the training of health professionals.

The University of Otago had already worked successfully with a Maori community to teach medical students about indigenous health through an immersion programme.[7] A similar approach had not previously been explored for teaching involving migrant communities in New Zealand. A Pacific Immersion Programme was developed where students would spend time living with a local Pacific family. Funds for the programme were made available through the Dean's Office.

Faculty of Medicine training

School leavers who wish to train in Medicine at the University are required to do a prescribed competitive Health Science First Year course.[34] Some students are admitted as competitive graduates, and others as "Other category" students. The Faculty of Medicine currently accepts 270 students into the second year of training. Those who are successful enter the Early Learning in Medicine programme (which lasts 2 years). Students then progress to the Advanced Learning in Medicine programme, and are distributed to either Christchurch, Dunedin or Wellington.

The development of a new medical curriculum at the Faculty of Medicine in 2008 provided the opportunity to introduce a longitudinal approach to teaching Pacific Health. Teaching of Pacific Health in the new Early Learning in Medicine programme (Years 2 and 3 of a 6-year programme) was delivered through whole class lecture series. Following this, the Pacific Immersion Programme was incorporated into Year 4 at the Dunedin Campus (one of the three campuses which run the Advanced Learning in Medicine (Years 4, 5 and 6) in 2010. Teaching in Pacific Health in Year 5 was incorporated into the curriculum as a whole class lecture series. Students were encouraged to consider conducting their medical electives in the final year (sixth year) of training in one of the Pacific Islands.

Pacific Immersion Programme development

The Pacific Immersion Programme was part of the Public Health attachment at the Department of Preventive and Social Medicine. The Department has a senior lecturer position in Pacific Health. This assisted discussions about developing a Pacific Immersion Programme within the local community.

The objectives of the attachment for students were to:

- Experience Pacific family life in NZ
- Observe how culture, religion and socioeconomic environment influence health
- Practise and observe cross cultural communication
- Provide opportunities for the community to teach students about their health, and how best to engage with them in the clinical setting
- Determine from observations and information shared, what could be useful for them in the future

Students were well prepared prior to engagement with the local community. Year 4 students have had training in confidentiality issues and professional development. Students were ambassadors for the University, and a high level of professionalism was expected.

There were four rotations during the year. Year 4 students attended in groups of approximately 20. Christianity plays a large part in the lives of many Pacific peoples and a weekend attachment enabled students to observe how religion influenced their lives. Three options were given to students : “Whole weekend stay”, “Day stay only” and “Opt out”. Those who opted out, were required to register a reason for doing so.

Information was provided to students about the programme objectives, guidance on learning opportunities, cultural protocols and processes specific to the Pacific community involved. The information provided also assisted students in writing a reflective essay about what they learnt.

Leaders from the Pacific community met students at the University the day before each attachment. This meeting provided opportunities for students to ask questions. The community welcomed students and staff in line with usual cultural protocols at the beginning of the programme. These included a traditional kava ceremony (a ceremonial welcome drink), singing, dancing and gifting of leis (traditional flower necklaces). Students were then introduced to their host families.

Everyone shared a morning tea together before students left with their host families. The wider community (including families not involved in the programme) gathered again the following day (Sunday) after church services for a combined celebration meal before the farewells. A debrief meeting with University staff the following day (Monday) gave students the opportunity to discuss observations, issues and experiences from the weekend. The students were also invited to complete a feedback form.

Community consultations

Consultation with the wider Pacific community was initiated in August 2009 when funding for the Pacific Immersion Programme was approved. The Pacific Immersion Programme catered for four groups of students (attachments) through the year. The three largest Pacific ethnic groups in New Zealand were Samoans, Cook Islands and Tongans, and it was agreed the first attachment was with the Samoan group, followed by the Cook Islands and Tongan groups.

The last attachment was allocated to combined smaller Pacific ethnic groups. Each group was involved once only during the year. The objectives and expectations of the programme was translated for Pacific families who wished to participate. Each community group nominated a coordinator to liaise closely with the University on all aspects of programme development.

These coordinators were leaders within their own communities, and responsible for matching students to host families. Coordinators also ensured excellent communication between the University and the local Pacific community. Community coordinators met with University staff after each attachment for a debrief and feedback meeting. Feedback from the community about the programme was well received.

Programme feedback

There were 77 fourth-year medical students enrolled at the Dunedin School of Medicine in 2010, and 57 (74 %) participated in the programme. Of the 20 who opted out, one was unwell and others had prior commitments. All student participants reported they enjoyed the programme, felt welcomed by the local community and were comfortable in the host environment.

Most students felt the programme helped their understanding of Pacific cultures, and gave them confidence to work across cultures (Table 1). Approximately, three quarters reported their upbringing was different from that of their hosts, however most felt there were able to communicate with them.

Table 1. Feedback from medical students in the Pacific Immersion Programme

Question	Yes n(%)	No n(%)	Don't Know n(%)
Do you feel this programme helped your understanding of a Pacific Islands culture?	55(96.5)	1(1.8)	1(1.8)
Were there any differences in your upbringing compared to what you observed?	42(73.7)	10(17.5)	3(5.3)
Did you feel you were able to communicate with people?	54(94.7)	1(1.8)	2(3.2)
Did you feel there were any barriers with respect to communication?	10(17.5)	37(64.9)	8(14)
Did this experience give you confidence to work within a different culture?	47(82.5)	2(3.5)	6(10.5)

Community coordinators reported their community felt the medical students had a positive influence on their young people inspiring some to consider further education after high school. They also felt empowered through the opportunity to shape the training of future doctors.

The Dean of the Dunedin School of Medicine and senior staff members attended some of the attachments. The community appreciated the level of engagement and commitment from the University.

Lessons learned

Make the programme a required component of learning—The programme was offered as an optional part of medical training. Twenty students did not take part in the programme. Many who missed out requested for another opportunity to take part in the programme. Unfortunately, this could not be arranged. The cultural immersion programme was a unique opportunity to learn about the health of an underserved community in New Zealand. In 2011, all students will be expected to undertake the programme. This requirement has been endorsed by the Dean of the Dunedin School of Medicine.

Value cultural training in health—The teaching of "culture and health" should be seen to be valued by the institutional leadership.[18] Support from the Dean and senior staff provided endorsement at this level. The literature in this area suggested a number of core components were required in the teaching of "culture and health" in undergraduate medical education.[18] One of these was community participation as the "expert teacher". Subjects critical to students' education should be clearly defined, articulated and examinable.[10] Students were motivated to learn subjects that were assessed.[35] Incorporating Pacific Health as part of the examinable component of medical education will ensure students value it as an important part of the medical curriculum.

Clear communication and transparent processes are essential—The work and commitment of Pacific community coordinators were critical in ensuring the Programme's success through excellent communication between the community and the University. They also assisted to ensure University processes were transparent and clear to the local community. This ensured the success of the programme.

Empowering community—The Pacific community was empowered by the experience, because they felt what they had to share was valued. All families who participated wanted to be involved in future programmes. Many felt they could influence the care they receive in the future by teaching future doctors about how best to engage with them. Others believed they were also helping the wider Pacific community and society in New Zealand by contributing to the training of future health professionals.

Looking ahead :

Extension of the programme—The Pacific Immersion Programme at the University of Otago medical programme could be explored as a method for teaching Pacific health in other campuses and universities in New Zealand. It could also provide a template for the engagement of other minority community groups in the training of health professionals.

Developing relationships—The development of good relationships through these types of programmes could lead to establishing excellent networks for service, research and teaching. This will benefit not only tertiary education institutions but also underserved communities.

Conclusion

The Pacific Immersion Programme was explored as a way to dramatically enhance Pacific Health learning for medical students at the University of Otago, New Zealand. Students and staff felt it was an effective way of teaching students about Pacific health and engaging the community in the work of the University. Community relationships and networks developed formed a basis for further work and collaborations in the future. Lessons learnt from developing this programme may be useful for other health training institutions.

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References:

1. Seeleman C, Suurmond J, Stronks K. Cultural competence: a conceptual framework for teaching and learning. Med Educ. 2009;43:229-237.
2. Rapp DE. Integrating cultural competency into the undergraduate medical curriculum. Med Educ. 2006;40:704-710.
3. Dolhun EP, Munoz C, Grumbach K. Cross-cultural education in US medical schools: Development of an assessment tool. Acad Med. 2003;78:615-622.
4. Taylor JS. Confronting "culture" in medicine's "culture of no culture". Acad Med. 2003;78:555-559.
5. Dogra N, Reitmanova S, Carter-Pokras O. Twelve tips for teaching diversity and embedding it in the medical curriculum. Med Teach. 2009;31:990-993.
6. Wachtler C, Troein M. A hidden curriculum: mapping cultural competency in a medical programme. Med Educ. 2003;37:861-868.
7. Dowell A, Crampton P, Parkin C. The first sunrise: an experience of cultural immersion and community health needs assessment by undergraduate medical students in New Zealand. Med Educ. 2001;35:242-249.
8. Gupta AR DT, Johnston MA. Incorporating multiculturalism into a doctor-patient course. Acad Med. 1997;72:428.
9. Mao C BC, Harway EC, Khalsa SK. A workshop on ethnic and cultural awareness for second-year students. J Med Educ. 1988;63:624-628.
10. Esfandiari A DC, Wilerson L, Gill G. An international health/tropical medicine elective. Acad Med. 2001;76:516.
11. Takayama JI CC, Pearl DB. A one-month cultural competency rotation for pediatrics residents. Acad Med. 2001;76:514-515.
12. Godkin M WL. A pathway on serving multicultural and underserved population. Acad Med. 2001;76:513-514.

13. Dogra N. The development and evaluation of a programme to teach cultural diversity to medical undergraduate students. Med Educ. 2001;35:232-241.
14. Durey A. Reducing racism in Aboriginal Australia: where does cultural education fit. Aust N Z J Public Health, 2010; S87-S92.
15. Roberts JH, Sanders T, Wass V. Students' perceptions of race, ethnicity and culture at two UK medical schools: a qualitative study. Med Educ. 2008;42:45-52.
16. Crampton P, Dowell A, Parkin C, Thompson C. Combating effects of racism through a cultural immersion medical education program. Acad Med. 2003;78:595-598.
17. Crandall SJ, George G, Marion GS, Davis S. Applying theory to the design of cultural competency training for medical students: a case study. Acad Med. 2003;78:588-594.
18. Tervalon M. Components of culture in health for medical students' education. Acad Med. 2003;78:570-576.
19. Wear D. Insurgent multiculturalism: Rethinking how and why we teach culture in medical education. Acad Med. 2003;78:549-554.
20. Beagan BL. Teaching social and cultural awareness to medical students: "it's all very nice to talk about it in theory, but ultimately it makes no difference". Acad Med. 2003;78:605-614.
21. Bandura A. Social Cognitive Theory: An agentive perspective. Annual Review of Psychology. 2001;52:1-26.
22. Mutchnick IS, Stern DT. Expanding the Boundary of Medical Education : Evidence for Cross-cultural Exchanges. Acad Med. 2003;78:S1-S5.
23. Denoon D. The Cambridge History of the Pacific Islands. Cambridge Cambridge University Press; 1997.
24. Spoonley P. A contemporary political economy of labour migration in New Zealand. Tijdschrift voor Economische en Sociale Georgrafie. 2006;97:17-25.
25. Robyn R, Iredale CH, Castles S. Migration in the Asia Pacific : population, settlement and citizenship issues. Edward Elgar Publishing Inc; 2003.
26. Ministry for Culture and Education NZ. History and Migration. The Encyclopedia of New Zealand, Wellington, 2005-2011.
27. Statistics New Zealand. Demographics of New Zealand's Pacific Population. 2006. http://www.stats.govt.nz/browse_for_stats/people_and_communities/pacific_peoples/pacific-progress-demography.aspx [Accessed 22 November 2010.]
28. Ministry of Health. A Portrait of Health. Key results of the 2006/7 New Zealand Health Survey. 2008. http://www.moh.govt.nz/moh.nsf/0/6910156be95e706e4c2568800002e403?OpenDocument [Accessed 23 November 2010.]
29. Ministry of Health. Pacific Health and Disability Workforce Development Plan. 2004. http://www.moh.govt.nz/moh.nsf/pagesmh/3681 [Accessed 17 November 2010]
30. New Zealand Medical Council. Best Outcomes for Pacific Peoples : Practice Implications. 2010. http://www.mcnz.org.nz/portals/0/publications/Best%20health%20outcomes%20for%20Pacific%20Peoples.pdf [Accessed November 17, 2010]
31. Tiatia J. Pacific Cultural Competencies : A Literature Review. 2008. http://www.moh.govt.nz/moh.nsf/indexmh/pacific-cultural-competencies-literature-review [Accessed 29 November.]
32. Ministry of Health and Ministry of Pacific Island Affairs. 'Ala Mo'ui : Pathways to Pacific Health and Wellbeing 2010-2014. 2010. http://www.moh.govt.nz/moh.nsf/pagesmh/10007/$File/ala-moui-pathways-to-pacific-health-wellbeing2010-2014.pdf [Accessed 29 November 2010]
33. Michener JL, Yaggy S, Lyn M, et al. Improving the Health of the Community: Duke's Experience with Community Engagement. Acad Med. 2008;83:408-413.

34. University of Otago. University of Otago Calendar. Applications for admission to medicine. 2010. http://www.healthsci.otago.ac.nz/admissions/micn2011.html [Accessed 22 November 2010.]
35. Newble D. Assessment. Medical Education in the Millenium. 1998:131-142.

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Insomnia treatment in New Zealand

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Abstract

Aim To describe insomnia treatment in New Zealand and estimate the annual societal costs of insomnia among New Zealanders aged 20–59 years.

Method Twenty-one interviews were conducted with insomnia treatment providers in New Zealand using a snowballing recruitment method. Information from the interviews and the international literature was used to estimate treatment profiles, availability, uptake and costs, as the basis for a decision analytic model with micro costing of each potential outcome. Sensitivity analyses were conducted with 10,000 Monte Carlo simulations randomly varying between each model parameter between minimum and maximum estimates.

Results The treatment provider interviews highlighted the unstructured nature of insomnia treatment in New Zealand. The net cost of treating a person with insomnia was estimated to be –$482. The net annual benefit (saving) for treating insomniacs aged between 20–59 yrs was estimated at $21.8 million.

Conclusion The estimated total societal costs per QALY gained by treating insomnia is substantially lower than the average QALY cost-effectiveness threshold ($6,865) of PHARMAC funding decisions for new pharmaceuticals. Thus, these analyses strongly support the cost-effectiveness of insomnia treatment.

Insomnia is defined as having difficulty initiating or maintaining sleep, or non-restorative sleep, together with impaired waking function that has been present for at least one month. These complaints occur despite having adequate time and opportunity for sleep.[1,2] Insomnia may occur secondary to other conditions including medical and psychological conditions, substance abuse and other sleep disorders, or it may occur as primary insomnia.

Based on a national survey of insomnia symptoms,[3,4] we have estimated that 13.0% of New Zealanders aged 20–59 yrs are affected by at least one symptom of insomnia often/always, together with excessive daytime sleepiness. Māori are affected disproportionately (prevalence in the study population: Māori 19.1%, non-Māori 8.9%). The risk of reporting a chronic sleep problem (lasting longer than six months) increased with increasing socioeconomic deprivation and increasing age, but ethnicity and sex were not significant independent risk factors. These figures are similar to international population prevalence estimates, in which approximately 30% of individuals report symptoms of insomnia, 15–20% report insomnia symptoms with daytime impairment and 5–10% meet a diagnosis of insomnia according to standardised criteria.[1,2,5]

A number of health factors such as poor physical health, poor mental health, and symptoms of anxiety or depression are associated with insomnia.[6] Individuals with insomnia are more likely to develop symptoms of depression at a later assessment,

and persistent insomnia symptoms may increase the likelihood of developing a mental health disorder at a later date.[7] It has been shown that insomnia symptoms precede the onset of depression and that depressed older individuals with persistent insomnia are more likely to remain depressed (OR 1.8) than those who do not suffer from insomnia.[8,9]

Few studies have investigated whether there is a causal relationship between insomnia and poor physical wellbeing, however it is known that short sleep duration is a risk factor for increased body mass, metabolic dysfunction, type 2 diabetes and hypertension.[10] Individuals with insomnia are more likely to take more medications, use more healthcare resources, be absent from work due to illness more often, and have more work-related and motor vehicle accidents.[11,12] Recent studies controlling for anxiety, depression and various medical comorbidities have shown that poor sleep can independently impair health-related quality of life.[13]

Despite significant advancement in pharmacological and non-pharmacological insomnia treatment internationally, including practice parameters for the behavioural and psychological treatment of insomnia,[14,15] there is currently no standardised approach to the diagnosis and treatment of insomnia in New Zealand. There is also no formal training of health care professionals working in this area and no current requirement for treatment providers to be legally registered.

This study aimed to:

- Investigate insomnia treatment in New Zealand based on interviews with a snowball sample of different providers; and
- Estimate the societal costs of insomnia among New Zealanders aged 20–59 yrs.

The economic modelling and analysis followed the approach we used in estimating the societal costs of obstructive sleep apnoea syndrome in New Zealand.[16]

Method

Interview—Structured interviews of insomnia treatment providers were conducted in person and by telephone between October 2007 and March 2008. The interviews were used to collect information on the profile of patients seen, the diagnostic and treatment practices being used, length of treatment regimes, patient outcomes in terms of treatment success, and the costs of treatment. In addition to open interview questions relating to their diagnosis and treatment practices, interviewees were shown or read a list of treatment options sourced from the literature, but not limited to those validated in the literature.[14,15] Interviewees were also given the opportunity to comment on any issue pertaining to insomnia treatment that they felt had not been adequately discussed within the structured interview.

Initial interviews were conducted with sleep physicians known to the researchers and currently working in established clinics, as well as other health practitioners known to specialise in insomnia treatment. A snowballing method was used during the interview process to build a small database of practitioners to interview that would canvas the range of treatment options currently available.

In order to obtain a reasonable representation of the different insomnia treatment practices available in New Zealand, providers were categorised as: *specialist physicians* (appropriately qualified physician working in specialty medical practice other than general practice); *general practitioners* (GP); *psychologists*; *pharmacists; health practitioners* (a medically-trained GP or other qualified health practitioner who has taken an interest, or undergone some training, in sleep); and *alternative health practitioners* (a practitioner with any level of training in alternative medicine, practising insomnia treatment). An equal number of treatment providers were sought from each category for interview.

A scoring system for comparing providers was developed based on: category of practitioner; being registered to practise with an appropriate New Zealand Registration Board or Council under the Health Practitioners Competence Assurance Act (2003); apparent knowledge of sleep terminology and medicine (and in particular, insomnia); diagnostic and treatment approaches used; practising within their scope of practice and competency guidelines; and provision of patient follow-up. Knowledge of sleep terminology and sleep medicine was rated against a four-point scale ranging from poor (lacked any knowledge or understanding of sleep terminology or medicine) to excellent (full understanding of sleep terminology and medicine).

Economic analysis—This study is a retrospective, prevalence-based, cost utility analysis (CUA) where net treatment costs were compared with quality of life years (QALYs) gained. The impact of treated and untreated insomnia on health resource utilisation and quality of life were evaluated to estimate the total costs of all cases in a one year period. As a one year time frame was used, discounting of costs was not necessary.

The interviews and a review of the literature were used to develop a treatment cost decision tree which was used as a basis for the health economic costing model. The decision tree took into account potential pathways for patients with insomnia, the population prevalence of insomnia, and the direct medical and non-medical costs associated with diagnosis of insomnia. To maintain a manageable level of complexity, the final version of the model represented a simplified version of all potential pathways. Pathways with low probability were excluded and the model was limited to one level of on-referral. On-referral pathways were also limited to those identified by the interviewees.

Patient pathways—The final decision tree is shown in Figure 1. In the first instance, a patient with insomnia could choose to seek treatment or not seek treatment. The treatment provider interviews were used to estimate the total number of patients who would seek treatment, and the proportions who would seek treatment from each category of provider. Since patients cannot self-refer to a specialist physician, this option only occurs in subsequent branches of the model. In each case, the probability of a confirmed diagnosis of insomnia, successful treatment of insomnia, and on-referral to other treatment providers were estimated.

For example, we assumed that approximately 40% of patients first approached their GP for treatment of insomnia. Of these, 65% received a diagnosis of insomnia and treatment was initiated. It was estimated that for 87.5% of these patients treatment was successful, with the remaining 12.5% were on-referred to either a psychologist, specialist physician or health practitioner. Similar pathways were constructed for other treatment providers. To account for uncertainty in estimates of the proportion of patients following each pathway, high and low probabilities were calculated as ±25% of the base case.

Prevalence and QALYs—The prevalence of insomnia was estimated as 13.0%, with a high probability of 16.2% and a low probability of 9.7%. Quality of life years (QALYs) gained with successful treatment were estimated from international literature for the base case values, and 0 was used as the low value.[18–20] Two treatment providers retrospectively completed a EuroQoL 5D (EQ–5D) questionnaire relating to successful treatment of insomnia in their patients, which were compared against the international literature. The EQ–5D score of one treatment provider was used for the high case value for QALYs gained in the decision tree. The score from the remaining practitioner was determined to be a high outlier and was disregarded.

Resource utilisations—At each node in the decision tree, events take place and resources are consumed. For example, a person with insomnia may consult a pharmacist and be recommended an over-the-counter medication. A purchase is made and transport costs are incurred. The resource utilisation estimates are summarised in Table 1.

Table 1. Resource utilisations by event

Events	Units of resource utilised by each event									
	General Practitioner Consult	Specialist Physician Initial Consult	Specialist Physician Follow Up Consult	Psychologist Consult	Health Practitioner Consult	Alternative Health Practitioner Consult	Prescription Medicine	Non Prescription Medicine	Transport For Treatment (Round Trip)	Increase In Total Health Cost Per Capita
Do not seek treatment										1.0
Seek treatment										
1 Pharmacist								1.0	1.0	
1.1 Refer General Practitioner	1.0						1.0		1.0	
1.2 Success										
2 General Practitioner	1.0								1.0	
2.1 No further action										
2.2 Treat							1.0			
2.2.1 Success										
2.2.2 Refer										
2.2.2.1 Psychologist				4.0					4.0	
2.2.2.2 Specialist Physician		1.0	1.0				1.0		2.0	
2.2.2.3 Health Practitioner					2.0				2.0	
3 Health Practitioner					1.0				1.0	
3.1 No further action										
3.2 Treat							0.5			
3.2.1 Success										
3.2.2 Refer										
3.2.2.1 Psychologist				4.0					4.0	
3.2.2.2 Specialist Physician		1.0	1.0				1.0		2.0	
3.2.2.3 General Practitioner	1.0						1.0		1.0	
3.2.2.4 Other Health Practitioner					3.0		0.5		3.0	
4 Psychologist				1.0					1.0	
4.1 No further action										
4.2 Treat										
4.2.1 Success										
4.2.2 Refer/ no further action										
4.2.2.1 Other Psychologist				4.0					4.0	
4.2.2.2 Specialist Physician		1.0	1.0				1.0		2.0	
4.2.2.3 General Practitioner	1.0						1.0		1.0	
4.2.2.4 No further action										
5 Alternative Health Practitioner						1.0			1.0	
5.1 No further action										
5.2 Treat						2.0			2.0	
5.2.2 Success										
5.2.3 Refer/ no further action										
5.2.3.1 Other Alternative Health Practitioner						2.0			2.0	
5.2.3.2 General Practitioner									1.0	
5.2.3.3 No Further Action										

Cost estimates—Costs were categorised as direct medical and direct non-medical. This study assumed the cost of any conservative, behavioural and/or psychological therapy was included in the cost of the consultation. Only incremental costs were included. That is, if a cost would have been incurred regardless of whether an event occurred or not, it was not included in the analysis. High and low values for each cost were calculated as ±25% of the base case value, and all costs were exclusive of GST.

Table 2. Unit resource cost estimates for insomnia treatment in 2009 New Zealand dollars

Resource	Base Case	Source
Direct Medical		
General practitioner	$48.89	Average adult consultation fee[21]
Specialist physician initial	$222.22	Initial adult consultation fee for medical practitioner band III[21]
Specialist physician follow up	$99.56	Follow-up adult consultation fee for medical practitioner band III[21]
Psychologist	$88.89	Initial adult consultation fee[21]
Health practitioner	$120.00	Average adult consultation fee for medical practitioner band II;[21] high case, interviews
Alternative health practitioner	$75.56	Base case and range, interviews
Prescription medicine	$6.42	Zopiclone,[22,23] interviews; base case, 7.5mg @30 days plus prescription dispensing fees[24]
Non prescription medicine	$16.00	Blackmores Valerian Forte,[25] interviews
Increase in cost per capita for individuals with and without insomnia	$627.52	Difference in total health costs of individuals with and without insomnia, derived from population prevalence estimates[3,4,26–28]
Direct Non-Medical		
Transport for treatment (round trip)	$16.71	Average reimbursement of $0.63/km for round trip to hospital (average 29.83km)[29]

Sensitivity analysis—To account for uncertainty in prevalence and cost estimates, 10,000 Monte Carlo simulations were conducted using randomly generated variables between the low and high estimates for each model parameter.[30] Multiple linear regression was then used to evaluate the effects of each model parameter on the total direct and indirect costs, and the total costs calculated by the model.

Results

Insomnia treatment providers—Of 31 providers approached, 18 agreed to complete an interview. Three specialist physicians, two GPs, one pharmacist, five psychologists, three health practitioners and four alternative health practitioners completed a full interview. Three pharmacists were not able to commit the time required to complete a full interview and agreed to complete a shortened version so that costing information and patient treatment pathways could be determined. Data from all 21 interviews were used in the analyses.

Insomnia patients were referred to treatment providers via a number of pathways. Self-referral and GP were the most common modes of referral, with occasional referrals from psychologists, psychiatrists, nurses, occupational health physicians and sleep physicians. Those interviewed reported that insomnia patients have often consulted several other insomnia treatment providers before seeking their services.

Among interviewees, 81% stated that their patients had consulted a GP at some stage and 81% stated that their patients had consulted alternative health practitioners in the

past. Psychologists, psychiatrists, occupational health physicians and sleep physicians were also occasionally consulted. Four of the 21 interviewees did not hold any registration to practise under the Health Practitioners Competence Assurance Act (2003), three because their field of work is not covered by the Act and one whose registration had lapsed.

Providers indicated treating individuals aged 3 months to 90 years for insomnia, of which the majority were middle-aged and Caucasian. The number of patients seen by each practitioner varied greatly, ranging from 15–110 patients per year for specialist physicians to 1000–5200 per year for pharmacists.

The majority of interviewees (62%) had poor/fair knowledge of the different types of insomnia. This included all the pharmacists, alternative health practitioners, and GPs interviewed. These providers were also the most likely to confirm a diagnosis of insomnia, were the least likely to use structured interviews, validated questionnaires, or supplementary tools for diagnosis, had the poorest understanding of standard sleep terminology, and offered the most limited range of treatment options. All psychologists and specialist physicians, and the majority of health practitioners, considered supplementary diagnostic tools in their every day practice. Sleep diaries were the most commonly employed adjunct diagnostic tool.

Table 3 shows the treatment options offered to insomnia patients by different providers. Sleep hygiene education was the most popular option being offered by 61.9%. Approximately half the providers (52.4%) also considered pharmacological management of insomnia. Specialist physicians, GPs, health practitioners and psychologists always used validated treatment options as per the American Academy of Sleep Medicine guidelines.

A quarter of alternative health practitioners used validated treatment options, while the remainder of the interviewees (38%) used forms of treatment lacking any evidence base for successful insomnia treatment. None of the pharmacists interviewed used validated treatment options. Specialist physicians, GPs, and psychologists always implemented treatment according to best practice guidelines, as did three of four alternative health practitioners. However, while health practitioners reported using validated treatment options, two of the three deviated from best practice guidelines for these treatments.

Table 3. Treatment options offered to insomnia patients by provider type

Treatment options	Specialist Physician	Pharmacist	GP	Psychologist	Health Practitioner	Alternative Health Practitioner
N	*3*	*4*	*2*	*5*	*3*	*4*
Pharmacological management	100%	100%	100%	20%	33%	0%
Herbal remedies	0%	100%	0%	0%	0%	50%
Stimulus control	66.7%	0%	0%	40%	66.7%	0%
Temporal control therapy	33.3%	0%	0%	0%	33.3%	0%
Relaxation training	33.3%	0%	50%	40%	66.7%	75%
Imagery training	0%	0%	0%	20%	33.3%	0%
Sleep restriction therapy	100%	0%	0%	20%	66.7%	0%
Paradoxical intention	0%	0%	0%	0%	33.3%	0%
Cognitive therapy	100%	0%	0%	20%	33.3%	0%
Cognitive behavioural therapy	66.7%	0%	0%	100%	66.7%	0%
Multi-component therapy	33.3%	0%	0%	0%	33.3%	0%
Biofeedback	100%	0%	0%	0%	33.3%	0%
Sleep hygiene education	100%	50%	50%	60%	66.7%	50%
Exercise	0%	0%	0%	0%	66.7%	25%
Light therapy	33.3%	0%	0%	0%	66.7%	0%
Other treatment options	33.3%	0%	50%	60%	66.7%	100%

Note: The numbers of treatment providers interviewed in each group are shown in row labelled N.

Only 57.1% of those interviewed officially assessed treatment effectiveness as part of their insomnia treatment plan. Table 4 outlines the number of consultations and the estimated treatment success rate by treatment provider type. Unsuccessfully treated patients may continue to see a treatment provider for up to 180 days (up to 12 consultations) before being offered a referral to an alternative provider or discontinuing consultation.

Table 4. Estimated number of consultations, success rate and length of treatment regime for insomnia by provider type

	Specialist Physician	Pharmacist	GP	Psychologist	Health Practitioner	Alternative Health Practitioner
N	*3*	*4*	*2*	*5*	*3*	*4*
Number of consultations	1–2	1–2	2–3	2–6	3–4	3–5
Treatment success rate (%)	*	95	85–90	65–100	75–80	90–100
Average time for successful treatment(days)	90–365	3–7	14–30	14–56	21–90	14–126

* It was common for specialist physician to confirm diagnosis and treatment plan before referring back to another treatment provider/GP for implementation and ongoing management.

Most providers (94%) were of the opinion that there was an unmet need for insomnia treatment in New Zealand. Many felt that patients did not have sufficient validated treatment options available to them, and that patients were not currently provided with accurate information when seeking treatment for insomnia. A proportion of treatment providers commented that they felt overwhelmed by the number of patients approaching them for insomnia treatment, and either could not meet that demand themselves, or were not confident in their ability to provide effective insomnia treatment.

Costs—Figure 1 depicts the final decision tree used to model treatment pathways and costs.

Figure 1. Treatment cost decision tree (base case values)

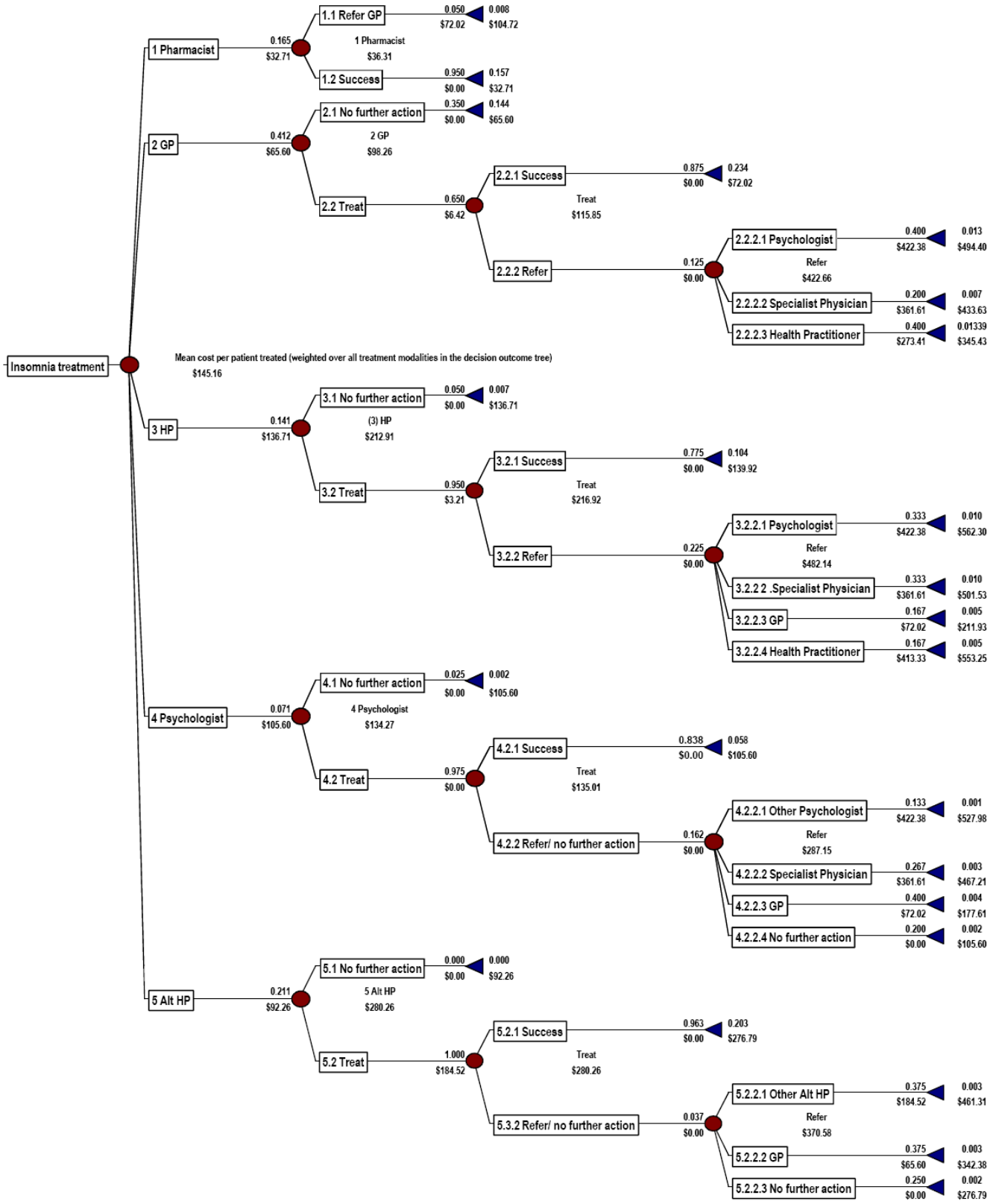

Table 5 summarises the estimated total societal costs of insomnia in New Zealand. The mean treatment cost across all treatment provider types was $145 per patient. The net cost of treating a person with insomnia was estimated to be –$482 ($145 less health costs avoided of $628), and 90% of the Monte Carlo simulations fell between –$679 and –$41 (a negative cost is a benefit).

Table 5. Total societal costs of insomnia treatment in New Zealand

Variables	Per Person Treated	NZ Total Million
At risk population (20–59 years, at June 2008)[31]		2.317
Prevalence of insomnia 13.0%[24]		0.300
Proportion seeking treatment 15%		0.045
Costs incurred	$145.00	$6.52
Costs avoided	*–$628.00*	*–$28.4*
Net cost	*–$482.00*	*–$21.8*
QALYs gained (#)	0.157	0.007
Net cost per QALY gained	*–$3,072*	*–$21.8*

The net annual benefit (saving) for treating insomniacs aged between 20–59 yrs was estimated at $21.8 million. The cost per QALY gained was estimated to be –$3072, and 90% of the Monte Carlo simulations fell between –$8102 and –$240.

The sensitivity analyses indicated that the three key determinants of the cost utility ratio were (in order of importance); costs avoided (difference in increase in health costs per capita for those untreated patients compared with successfully treated patients); the number of QALYs gained from successfully treating a patient; and costs incurred (treatment costs).

Discussion

The interviews undertaken for this study have highlighted the diversity of treatment services being offered, and the relatively small proportion of providers who have any awareness of international best practice standards for insomnia diagnosis and treatment. Insomnia treatment providers are not required to be registered or accredited, so there is no way of assessing how representative the 21 interviewees are of services being offered nationwide. Nevertheless, the snapshot provided does not indicate effective, efficient, or equitable provision of treatment services.

The cost utility analysis, comparing net treatment costs to QALYs gained, is based on a different approach to that used in previously published estimates of the societal costs of insomnia. Despite this different approach, the results of this study remain aligned with previous research[31,32] and indicate that effective treatment of insomnia saves money.

In 2009, the net annual societal cost of treating a person with insomnia was estimated to be –$482 per person (a saving). The estimated annual savings associated with

effective treatment of all people with insomnia aged 20–59 yrs was $21.8 million. The cost per QALY gained was estimated to be –$3072, and 90% of the Monte Carlo simulations fell between –$8102 and –$240. By way of comparison, the cost-effectiveness threshold of PHARMAC funding decisions for new medicines between 1999 and 2005 was $6,865 per QALY gained.[24]

The economic analysis has a number of limitations. The sensitivity analysis indicated that the number of QALYs gained from successfully treating a patient was a significant determinant of the cost utility ratio. In the absence of New Zealand data, QALY estimates were taken from the international literature. The published studies have used the SF-36 (a general health profile not specifically designed for economic evaluation). Two of the interviewees completed an EQ–5D (an economics tool designed for estimating QALYs), but this was retrospective and provided a general indication only of the quality of life improvements assumed by New Zealand practitioners.

An accurate assessment of QALYs gained would require prospective completion of the EQ–5D by individuals undergoing insomnia treatment. It was also not possible to include the costs of accidents and decreased productivity for people with untreated insomnia, or the extended impact on their family and friends.

The cost estimates in this study are based on the international literature and the treatment provider interviews. They have the limitation that we do not know how representative the treatment providers are. In addition, self-rated on-referral and treatment rates were used to determine event probabilities in the treatment cost decision tree. These values were not able to be independently verified and treatment providers did not provide an exact definition of 'success'. However, a conservative approach was taken in all calculations.

With the exception of 'other' alternative health techniques, most self-rated treatment success rates aligned reasonably well with the international literature and tended towards conservative estimates.[15,33] Although treatment success may not be a true reflection of patient outcomes, treatment success influenced how a treatment provider interacted with a patient (that is, on-referral, continuing treatment or discharge).

Population prevalence is a key factor in determining the total societal costs of insomnia. The international literature estimates insomnia prevalence in the range of 9–30%[5] and previously published New Zealand data indicated that 25% of people aged 20–59 yrs had a current sleeping problem.[3,4] The present study used a conservative population prevalence estimate calculated from the national survey dataset, requiring at least one insomnia symptom along with daytime impairment (ESS >10), with a population prevalence of 13%.[27]

The international literature suggests that insomnia is associated with a range of other medical conditions. However, the causal relationship between these conditions and insomnia is not well established. It is also not known whether insomnia treatment alone would improve associated medical conditions, such as anxiety or depression. Therefore, the study took the conservative approach of estimating costs only from total health care utilisation. In general, research addressing the health and safety consequences of untreated insomnia is less developed than for sleep-related breathing

disorders, where knowledge has been greatly enhanced by a number of large longitudinal cohort studies.

The providers interviewed have sought to address patient needs in the absence of a structured approach to insomnia treatment in the healthcare system. A 2010 survey of sleep services provided by District Health Boards found that none had dedicated funding for the treatment of insomnia (Kanchana Pathirana, personal communication).

The dilemma of a high burden of disease and lack of appropriate diagnosis and treatment services for insomnia is not unique to New Zealand.[6,17] In part, this probably reflects the diverse aetiology of insomnia and the range of treatment options, which should be linked to differential diagnosis. Nevertheless, the conservative economic analysis presented here indicates that a more systematic approach to treatment would be highly cost-effective for the New Zealand healthcare system.

This study highlights a number of significant issues. New Zealand would greatly benefit from a standardised approach to insomnia diagnosis and treatment. In particular, the multiple aetiologies of insomnia require differential diagnosis and a systematic approach to treatment that is implemented by trained individuals across a range of disciplines.

New Zealand is fortunate to have a well characterised population in terms of sleep disorder prevalence. However, a more accurate assessment of the economic burden of insomnia would also require prospective measurement of quality of life, resource utilisation, treatment pathways, and incidence of adverse health and safety outcomes in those undergoing treatment for insomnia.

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References:

1. American Academy of Sleep Medicine. The international classification of sleep disorders: Diagnostic and coding manual (ICSD–2). 2nd ed. Westchester, IL: American Academy of Sleep Medicine; 2005.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM–IV–TR). 4th ed. Washington, DC: American Psychiatric Association; 2000.
3. Paine SJ, Gander PH, Harris R, Reid P. Who reports insomnia? Relationships with age, sex, ethnicity, and socioeconomic deprivation. Sleep. 2004;27(6):1163–9.

4. Paine SJ, Gander PH, Harris RB, Reid P. Prevalence and consequences of insomnia in New Zealand: disparities between Maori and non–Maori. Aust N Z J Public Health. 2005;29(1):22–8.
5. Ohayon MM. Epidemiology of insomnia: What we know and what we still need to learn. Sleep Med Rev. 2002;6(2):97–111.
6. Morin CM, LeBlanc M, Daley M, et al. Epidemiology of insomnia: Prevalence, self–help treatments, consultations, and determinants of help–seeking behaviors. Sleep Med. 2006;7(2):123–30.
7. Fong SY, Wing YK. Longitudinal follow up of primary insomnia patients in a psychiatric clinic. Aust N Z J Psychiatry. 2007;41(7):611–7.
8. Johnson EO, Roth T, Breslau N. The association of insomnia with anxiety disorders and depression: Exploration of the direction of risk. J Psychiatr Res. 2006;40(8):700–8.
9. Pigeon WR, Hegel M, Unutzer J, et al. Is insomnia a perpetuating factor for late–life depression in the IMPACT cohort? Sleep. 2008;31(4):481–8.
10. Grandner MA, Patel NP, Gehrman PR, et al. Problems associated with short sleep: Bridging the gap between laboratory and epidemiological studies. Sleep Med Rev. 2010;14(4):239–47.
11. Leger D, Guilleminault C, Bader G, et al. Medical and socio-professional impact of insomnia. Sleep. 2002;25(6):625–9.
12. Kapur VK, Redline S, Nieto FJ, et al. The relationship between chronically disrupted sleep and healthcare use. Sleep. 2002;25(3):289–96.
13. Kyle S, Morgan K, Espie C. Insomnia and health-related quality of life. Sleep Med Rev. 2010;14(1):69–82.
14. Morgenthaler T, Kramer M, Alessi C, et al. Practice parameters for the psychological and behavioral treatment of insomnia: an update. An American Academy of Sleep Medicine report. Sleep. 2006;29(11):1415–9.
15. Morin CM, Bootzin RR, Buysse DJ, et al. Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998–2004). Sleep. 2006;29(11):1398–414.
16. Gander P, Scott G, Mihaere K, Scott H. Societal costs of obstructive sleep apnoea syndrome. N Z Med J. 2010;123(1321):13–23.
17. Stinson K, Tang NK, Harvey AG. Barriers to treatment seeking in primary insomnia in the United Kingdom: A cross–sectional perspective. Sleep. 2006;29(12):1643–6.
18. Hatoum HT, Kong SX, Kania CM, et al. Insomnia, health–related quality of life and healthcare resource consumption. A study of managed–care organisation enrollees. Pharmacoeconomics. 1998;14(6):629–37.
19. Leger D, Scheuermaier K, Philip P, et al. SF–36: evaluation of quality of life in severe and mild insomniacs compared with good sleepers. Psychosom Med. 2001;63(1):49–55.
20. Katz DA, McHorney CA. The relationship between insomnia and health–related quality of life in patients with chronic illness. J Fam Pract. 2002;51(3):229–35.
21. Southern Cross Medical Care Society. Ultra Care Plan. Retrieved 16 March 2009 from http://www.southerncross.co.nz/sx_internet/library/v14120_3.pdf
22. Pharmaceutical Management Agency of New Zealand. Annual review 2007. Wellington: Pharmaceutical Management Agency Limited; 2008.
23. Pharmaceutical Management Agency of New Zealand. New Zealand Pharmaceutical Schedule Update. Wellington: Pharmaceutical Management Agency of New Zealand; 2009.
24. Pharmaceutical Management Agency of New Zealand. Prescription for pharmacoeconomic analysis: Methods for cost–utility analysis. Wellington: Pharmaceutical Management Agency of New Zealand; 2007.
25. Pharmacy Direct. Blackmores Valerian Forte 2000mg Tablets 30. Retrieved 16 March 2009 from http://www.pharmacydirect.co.nz/blackmores–valerian–forte–2000mg–tablets–30.html?&partner=sli&bid=3

26. New Zealand Ministry of Health. Health expenditure trends in New Zealand 1998–2008. Retrieved 2011 from http://www.moh.govt.nz/moh.nsf/indexmh/health–expenditure–trends?Open
27. Sleep/Wake Research Centre. Data on file. 2008.
28. Statistics New Zealand. National population estimates: December 2010 quarter. Retrieved 2011 from http://www.stats.govt.nz/browse_for_stats/population/estimates_and_projections/nationalpopulationestimates_hotpdec10qtr.aspx
29. New Zealand Automobile Association. Fuel & car running costs, petrol compact 1500cc–2000cc. Wellington: New Zealand Automobile Association; 2009. Retrieved 16 March 2009 from http://www.aa.co.nz/SiteCollectionDocuments/pdf/car_running_costs/petrol_08.pdf
30. Palisade Corporation. @ Risk version 4.5 Risk analysis and simulation add–in for Microsoft Excel. Newfield, New York; 2004.
31. Ozminkowski RJ, Wang S, Walsh JK. The direct and indirect costs of untreated insomnia in adults in the United States. Sleep. 2007;30(3):263–73.
32. Daley M, Morin CM, LeBlanc M, et al. The economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. Sleep. 2009;32(1):55–64.
33. Riemann D, Perlis ML. The treatments of chronic insomnia: A review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev. 2009;13(3):205–14.

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Evaluation of New Zealand's bicycle helmet law

Colin F Clarke

Abstract

The New Zealand helmet law (all ages) came into effect on 1 January 1994. It followed Australian helmet laws, introduced in 1990–1992. Pre-law (in 1990) cyclist deaths were nearly a quarter of pedestrians in number, but in 2006–09, the equivalent figure was near to 50% when adjusted for changes to hours cycled and walked. From 1988–91 to 2003–07, cyclists' overall injury rate per hour increased by 20%. Dr Hillman, from the UK's Policy Studies Institute, calculated that life years gained by cycling outweighed life years lost in accidents by 20 times. For the period 1989–1990 to 2006–2009, New Zealand survey data showed that average hours cycled per person reduced by 51%. This evaluation finds the helmet law has failed in aspects of promoting cycling, safety, health, accident compensation, environmental issues and civil liberties.

New Zealand (NZ) helmet law (all ages) came into effect from 1 January 1994. It followed Australian helmet laws, introduced in 1990–1992. Survey data from Australia indicated legislation was a poor approach as it discouraged cycling—e.g. child cycle use fell 44% by the second year of the helmet law in New South Wales, Australia.[1]

A NZ report from 1985 by Sage et al[2] detailed that out of 20 bicycle riders fatally injured in Auckland, between 1974 and 1984, 16 died (80%) of injury to multiple organ systems and suggested that not many lives could be saved by wearing helmets.

The aim of the study was therefore to review the efficacy of the New Zealand's bicycle helmet law in terms of safety, health, law enforcement, accident compensation, environmental issues and civil liberties.

Method

This evaluation reviews publically available data and analyses[3–7,9] to assess the outcome for cycling activity levels, safety, health, law enforcement, accident compensation, environmental issues and civil liberties. The data compares cyclists to pedestrians and evaluates changes to population and road safety trends. A summary and conclusions draw together the findings and suggests the best way forward.

Results and Assessments

Changes in walking and cycling activity—Consideration of both cycling and walking may provide a clearer indication of overall changes in physical activity.

Table 1 provides survey information on hours walked and cycled for four time periods.[3,4] Estimates for the NZ population are shown for each period.

Table 1. Comparison (from 1989–90 to 2006–09) of average time per person walking and cycling in NZ

Period **Estimated NZ population**	**1989–1990** **3,407,000**	**1997–1998** **3,770,000**	**2003–2006** **4,080,000**	**2006–2009** **4,250,000**
Hours spent walking per year (million hours)	191	215	199	212
Hours spent cycling per year (million hours)	39	26	22	24
Average hours walked per person (% change from 89–90 period)	56	57 (+2%)	49 (-12%)	50 (-11%)
Average hours cycled per person (% change from 89–90 period)	**11.4**	**6.9 (-40%)**	**5.4 (-53%)**	**5.6 (-51%)**

Sources: 1989–2006 data: Sustainable and safe land transport trends and indicators[3];
2006–2009 data: Cycling for transport: Ongoing New Zealand Household Travel Survey 2006–2009[4]

As shown in Table 1, from the period 1989–1990 to 2006–2009, the number of hours cycling reduced—from 39 million to 24 million. The average hours walked and cycled per person reduced by 11% and 51% respectively.

The NZ Ministry of Transport stated 'The travel surveys show that from 1989/90 to 2005/08, the average time spent cycling per week decreased from 28 minutes to 8 minutes among those aged 5–12 years and from 52 minutes to 12 minutes among those aged 13–17 years.'[5] Averaging data for the two age groups implies a 75% reduction for children aged 5-17 from 40 minutes to 10 minutes per person per week.

In addition, concerns were expressed about the safety outcome 'Of particular concern are children and adolescents who have experienced the greatest increase in the risk of cycling injuries despite a substantial decline in the amount of cycling over the past two decades'.[6]

Tin Tin et al also reported 'In New Zealand, the overall travel mode share for cycling declined steadily from 4% in 1989 to 1% in 2006'.[6]

If people cycle less and this in turn reduces their overall fitness it could contribute to them walking less as well. The survey information 1989/90–2003/06 suggests a drop of 53% and indicates that the helmet law discouraged cycling to a significant extent.

Fatality comparison, cyclist vs pedestrians (1989–2009)—The fatality data[7] shows a significant reduction for both cyclists and pedestrians over the past two decades.

Table 2. Annual NZ fatalities of cyclists compared to pedestrians (1989–2009)

Year	89	90	91	92	93	94	95	96	97	98	99
Pedestrians (n)	81	104	88	76	74	54	71	63	54	71	63
Cyclists (n)	20	27	22	17	17	15	15	13	12	16	8
Cyclists / Pedestrians (%)	25	26	25	22	23	28	21	21	22	22	13
Year	**00**	**01**	**02**	**03**	**04**	**05**	**06**	**07**	**08**	**09**	
Pedestrians	35	52	45	58	38	31	44	45	31	31	
Cyclists	19	10	14	6	7	12	9	12	10	8	
Cyclists / Pedestrians (%)	54	19	31	10	18	39	18	27	32	26	

Source: http://www.transport.govt.nz/research/Documents/Motor-Vehicle-Crashes-2010-Historical.pdf

A simple calculation, from the data in Table 2, shows that, for the 5-year period 1989 to 1993, the 103 cyclist deaths represented 24% of the number (423) for pedestrians. For the 4-year period 2006–2009, cyclist deaths were 41 compared to 151 for pedestrians or 27%.

Increasing the totals to equate pre law levels of cycling and walking (average hours walked and cycling reduced by 11% and 51%), would give totals of 83 and 170. This indicates that cyclist safety, compared to pedestrians, has reduced appreciably from 24% to the equivalent of 49% (83/170). In 1990 cyclist deaths were nearly a quarter of pedestrians in number but by the 2006–09 period, the equivalent figure was near to 50% when adjusted for changes to hours cycled and walked.

The information from Table 1 on average hours walked and cycled, together with fatality data from Table 2 allows rates to be calculated relative to the time spent walking or cycling, and the relative risk for pedestrians and cyclists to be compared for the different time periods (Table 3).

Table 3. Relative risk of cycling versus walking: average pedestrian and cyclist deaths per year compared to average hours walked and cycled

Period	1989–1990	1997–1998	2003–2006	2006–2009
Pedestrian deaths / average per year	185 / 92.5	125 / 62.5	171 / 42.75	151 / 37.75
Average hours walked per person	56	57	49	50
Pedestrian, deaths / average hours walked	1.65	1.10	0.87	0.75
Cyclist deaths / average per year	47 / 23.5	28 / 14	34 / 8.5	41 /10.25
Average hours cycled per person	11.4	6.9	5.4	5.6
Cyclist, deaths / average hours cycled	2.05	2.03	1.57	1.83
Risk ratio, cyclist / pedestrian	1.24	1.85	1.80	2.44

Source: Calculations from the data in Tables 1 and 2.

Sage et al[2] stated “This study indicates that compulsory wearing of suitable safety helmets by cyclists is unlikely to lead to a great reduction in fatal injuries, despite their enthusiastic advocacy". The details provided show about 46% of cyclists’ deaths (in Auckland 1974–1984) were children aged 6–15 years.

Collins et al reported that 39% of all cyclist fatalities in NZ occurred to those aged 5–14 years for the period 1979/88.[8] For the age group 5–17 years they may have

traditionally incurred about 45% or more of cyclist fatalities and they had a reduction in cycling of about 75%.

Injury assessment—Selected data in Table 4 below is from a recent study by Tin Tin et al[6] plus additional data showing the percentage change (**bold**) from 1988–91.

Table 4. Annual numbers and rates of traffic injuries on NZ roads that resulted in death or hospital inpatient treatment

Mode of travel	Annual number of injuries			Annual number of injuries per million hours spent travelling (change relative to 1988–91)		
	1988–91	1996–99	2003–07	1988–91	1996–99	2003–07
Overall						
Cyclists	941	512	682	25.61	21.38 **(-17)**	30.74 **(+20)**
Car/van driver	2081	2051	1714	4.24	3.22 **(-24)**	2.10 **(-50)**
Car/van passenger	1568	1428	1086	5.64	4.67 **(-17)**	2.89 **(-49)**
Motorcyclist	1655	895	784	185.14	161.77 **(-13)**	107.64 **(-42)**
Pedestrian	743	638	471	4.29	3.40 **(-21)**	2.38 **(-45)**
Serious injuries (AIS≥3)						
Cyclists	377	117	138	10.27	4.86 **(-53)**	6.24 **(-39)**
Car/van driver	886	774	629	1.81	1.22 **(-37)**	0.77 **(-47)**
Car/van passenger	643	516	367	2.31	1.69 **(-27)**	0.98 **(-57)**
Motorcyclist	483	273	190	54.05	49.24 **(-9)**	26.11 **(-51)**
Pedestrian	362	254	187	2.09	1.36 **(-35)**	0.95 **(-45)**

Source:. Tin Tin S. Injuries to pedal cyclists on New Zealand roads, 1988-2007.

Table 5 below, calculated from the data in Table 4, shows the ratio of cyclist to pedestrian injuries per million hours of travel from 1988-91 to 2003–07. Cyclist's overall injuries more than doubled compared with pedestrians, 5.97 to 12.91, indicating a major reduction in safety.

Table 5. Ratio of cyclist to pedestrian injuries in NZ per million hours of activity

Period	1988–91	1996–99	2003–07
Overall	5.97	6.28	12.91
Serious injuries	4.9	3.52	6.57

Source: Derived from Table 4.

As shown in Table 4, by 2003–07, cyclists had a 20% higher accident rate compared with pre law. In comparison all other road users had reductions of 42% to 50%. . Serious injuries reduced for cyclists by 39% compared to 45% to 57% for other road

users. For those aged 13–17 years cycling activity reduced from 52 minutes to 12 minutes per week [6], a reduction of 77%. This age group tends to incur more serious accidents involving motor vehicles than younger cyclists.

Erke and Elvik (Norwegian researchers) 2007[10] stated: 'There is evidence of increased accident risk per cycling-km for cyclists wearing a helmet. In Australia and NZ, the increase is estimated to be around 14 percent.' It appears probable that the 14% figure is a low estimate compared to more recent data.

Injury data[11] for 2006–09 compared to 1998/90 shows an average reduction of approximately 18.5% (858/1052), compared to cycling reducing by 38.5% (24/39 million hours). The approximate risk per million hours cycling therefore increased from 27 to 35.7 or by 32%.

Clarke[12] details a number of reports indicating a higher accident rate associated with helmet use and provides details of why this may occur. The increased risk probably relates to a combination of factors, 'Safety in Numbers',[13] risk compensation[14–16] and balance and riding stability aspects may also play a part.

Head injuries—Collins et al[8] reported accident data for 1988, 'Fifty-one percent of those hospitalised were aged 5–14, and males accounted for 70% of all admissions. Thirty-four percent involved a collision with a motor vehicle. Intracranial injuries and skull fractures accounted for 46% of hospital admissions, and had the highest scores on the abbreviated injury scale (AIS).' For the age group 5–17 it is possible that this group account for about 65% of head injury admissions pre law. Estimating a 75% reduction as per survey information on 65% would mean an estimated 48% reduction in head injuries. In addition road safety has improved resulting in far fewer deaths, e.g. 754 deaths in 1989 and 393 in 2006.[7]

Serious injuries per million hours of travel to pedestrians and motor vehicle users reduced by between 45% and 57%, (Table 4) suggesting that head injuries also reduced. Data from Canada also show head injuries reducing over a decade of change.[17] Robinson showed that there had been no reduction in head injuries to cyclists over and above the general trend experienced by the population as a whole.[18]

Overall safety assessment—In 1989/90, road deaths in NZ were approximately 217 per million population and by 2006/09 period about 93 per million population, a reduction of 57%. Cyclist deaths per hour of cycling fell by about 11% compared to a fall of 55% for pedestrians. Injuries to cyclists per hour of cycling increased by 20% (Table 4) compared to a reduction of 45% for pedestrians. This indicates a net reduction in cyclist's safety of 65% (-20% to +45) compared to pedestrians. Cyclist safety has been reduced due to the helmet requirement and law.

Injuries to cyclists per hour of cycling have increased by 32% based on *Motor Vehicle Crashes in New Zealand 2009.*[11]

Health assessment—Moderate cycling has many physical and mental benefits (BMA 1992[19]) by reducing the risk of developing heart disease,[20] diabetes, high blood pressure, colon cancer and depression, and helping to control weight and increase fitness. Dr Hillman from the UK's Policy Studies Institute calculated the life years gained by cycling outweigh life years lost in accidents by a factor of 20 to 1.[21] For NZ the average hours cycled per person reduced from 11.4 to 5.6 and assuming this is due

to the helmet requirements an approximate calculation based on the World Health Organization (WHO) assessment method, "Quantifying the positive health effects of cycling and walking"[22] can be made.

Data assumed:

Hours of cycling lost per year 5.8 × 4.0 million = 23.2 million hours per year

Distanced cycled at 12 km/hr = 278 million km

Assume 190,000 trips per day × 4 km × 365 = 277 million km

Based on this data an estimate that the law making helmets compulsory for cyclists has resulted in an overall *increase* in approximately 53 premature deaths per year.

There are concerns in NZ about the weight gain by children, for example; 'Obesity in New Zealand children: a weighty issue' discusses some of the issues.[23] Making cycling less convenient and with the potential for parents to incur a fine if their child is not wearing a helmet could add to the discouraging effects due to legislation. The helmet law actually reduced public health.

Law enforcement—Police figures show 9618 tickets were issued in 2010 for not wearing a helmet. The annual tally has generally tended upwards since 2000, when 5550 tickets were issued.[24] Not wearing one risks a $55 fine. The time spent by the police on cyclists could be used to enforce road safety in general thus lowering road deaths from the 375 in 2010. Enforcement is at a reasonable level but the outcome is reduced cycling levels.

Accident compensation assessment—From 2006 to 2009 there were 1565 road fatalities, including 42 cyclists—about 37 people killed for each cyclist—many will have incurred head injuries. Approximately four times more pedestrians and many more motor vehicle occupants suffer lethal head injuries than cyclists.

Great Britain accident data[25] for 2009 include the proportion of road casualties with injury to head/face. For the age group 0–15 years, pedestrians 53%, car occupants 46%, pedal cyclists 40%. For all ages, pedestrians 46%, car occupants 32%, pedal cyclists 37%.

Discrimination can occur in accident compensation cases where a cyclist was not wearing a helmet, compared to pedestrians or indeed motor vehicle occupants who received head injuries. The helmet laws result in unfair compensation and a biased legal process.

Environmental issues—Bicycles use the least energy (kilojoules [kJ] per person per kilometer) for general transport[26] and have average kJ values of:

Cyclist	**150**
Pedestrian	230
Tram	2000
Motorcyclist	2100
Bus	2500
Car (driver only)	5000

Source: Victorian Bicycling Strategy; Vic Roads, Australia 1990.

Transport is responsible for 44% of NZ's carbon dioxide emissions, and around 16% of total greenhouse gas emissions.[9] Vehicle exhaust emissions are a major source of air pollution in some areas, particularly around busy road corridors. Pollutants include carbon monoxide (CO), nitrogen dioxide (NO2), benzene, and particulate matter.

Vehicle emissions affect people's health. A recent study estimated that 399 people will die prematurely each year due to vehicle air pollution.[9,34] Vehicles also emit carbon dioxide (CO_2), which is a greenhouse gas (GHG) and that has increased by 28% from 1990 to 2001.[27]

After enteric fermentation (methane emissions from domestic livestock), land transport is the largest source of GHG emissions in NZ. It is also the fastest growing, accounting for 18% of the growth of GHG emissions over the 1990–2001 period.[28] The bicycle helmet law directly contributes to environmental pollution by discouraging cycling and using plastics in the production of helmets.

Civil liberties—The UK's National Children's Bureau (NCB) provided a detailed review in 2005[29] stating "the case for helmets is far from sound", "the benefits of helmets need further investigation before even a policy supporting promotion can be unequivocally supported" and "the case has not yet been convincingly made for compulsory use or promotion of cycle helmets."

The benefits of helmets are overstated and the costs of chronic health conditions, including obesity, among children and youth are massive. The ECF (European Cycling Federation) stated "the evidence from Australia and NZ suggests that the wearing of helmets might even make cycling more dangerous,"[30] indicating safety was actually reduced. It is not certain that helmets actually improve safety and data for children shows their safety has been reduced.

Curnow[31] reporting on Australia concluded, "Compulsion to wear a bicycle helmet is detrimental to public health". The UK consumer magazine *Which?*[32] independently tested 24 helmets and reported that only 9 passed all tests and therefore even new helmets may not be reliable.

Where a reasonable doubt exists about any product providing a net benefit then the consumer should have the right not to use it. It is simply, but importantly, respecting human rights by allowing the individual to decide. Insufficient respect for human rights is shown across the world and unless the individual is allowed to exercise their rights then this opens the way for devaluing human rights in general.

Voluntary helmet wearing rates in NZ prior to legislation were about 56% for teenagers and 86% for younger children.[33] It is therefore possible that a good proportion wear helmets without legislation. About 50%+ were wearing helmets prior to the law and about 10% may not wear them after the law. From 100 cyclists pre law, about 50 did not wear one and survey information shows about 50% stopped cycling, so therefore the law has failed to appreciably increase the number wearing helmets but instead appears to have just put people off cycling.

Summary

The following trends were observed following the introduction of New Zealand's helmet law:

- Cycling usage reduced by 51%.
- Cyclist's injury risk per hour increased by 20–32%.
- Estimated to have contributed to 53 premature deaths per year (due to reluctance to cycle and hence people not exercising).
- Thousands of fines are issued annually for not wearing a helmet.
- May contribute to discrimination in accident compensation and the legal processes.
- Could have contributed to environmental pollution and environmental harm (due to use of vehicles in place of cycles).
- Possibly diminishes civil liberties and human rights (by imposing a requirement to wear a helmet when several reports raise serious doubts whether they improve safety overall).

Is a mandatory cycle helmet requirement the best approach to promoting health and safety for the nation?

Conclusions

This evaluation of NZ's bicycle helmet law finds it has failed in aspects of promoting cycling, safety, health, accident compensation, environmental issues and civil liberties. It is estimated to cost about 53 lives per year in premature deaths and result in thousands of fines plus legal aspects of discrimination in accident compensation cases. Road safety and cyclist's safety should be improved by coherent policies, which support health, the environment, and without the legal requirement to wear a helmet. Additional information is available via web sites at http://www.cycle-helmets.com/zealand_helmets.html and http://www.cyclehelmets.org

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References:

1. Smith NC, Milthorpe FW. An observational survey of law compliance and helmet wearing by bicyclists in NSW. Roads and Traffic Authority of NSW. 1993.
2. Sage MD, Cairns FJ, Toeimeyer TD, Sweeton WMI. Fatal injuries to bicycle riders in Auckland. N Z Med J. 1985 Dec 25;98(793):1073-4.

3. Sustainable and safe land transport trends and indicators – Trend 2, Land Transport Safety Authority. http://www.cycle-helmets.com/nz-ltsa-2006.pdf
4. Cycling for transport: Ongoing New Zealand Household Travel Survey 2006–2009, Land Transport Safety Authority. http://www.transport.govt.nz/research/Documents/Cycling_for_transport_2009.pdf
5. Ministry of Transport: How New Zealanders travel: trends in New Zealand household travel 1989-2008. Wellington: Ministry of Transport; 2009. http://www.transport.govt.nz/research/Documents/How%20New%20Zealanders%20travel%20web.pdf
6. Tin Tin S. Injuries to pedal cyclists on New Zealand roads, 1988-2007. BMC Public Health 2010;10:655. http://www.biomedcentral.com/1471-2458/10/655
7. Crashes in New Zealand 2010 http://www.transport.govt.nz/research/Documents/Motor-Vehicle-Crashes-in-New-Zealand-2010.pdf
8. Collins BA, Langley JD, Marshall SW. Injuries to pedal cyclists resulting in death and hospitalisation., N Z Med J. 1993 Dec 8;106(969):514-6. http://www.ncbi.nlm.nih.gov/pubmed/8183490
9. Environmental effects http://www.mfe.govt.nz/issues/transport/environmental.html
10. Erke A, Elvik R. Making Vision Zero real: Preventing Pedestrian Accidents And Making Them Less Severe, Oslo June 2007. page 28. https://www.toi.no/getfile.php/Publikasjoner/T%d8I%20rapporter/2007/889-2007/889-2007-nett.pdf
11. Motor Vehicle Crashes in New Zealand 2009, Table 36, Pedal Cyclist Casualties and Population Statistics Historical. http://www.transport.govt.nz/research/Documents/Motor-Vehicle-Crashes-2009-Pedal-cyclist-casualties-and-crashes.pdf
12. Clarke CF, The Case against bicycle helmets and legislation, VeloCity Munich, 2007. http://www.ta.org.br/site/Banco/7manuais/colin_clarke_cycle_helmet.pdf
13. Jacobsen PL. Safety in numbers: more walkers and bicyclists, safer walking and bicycling. Inj Prevent 2003;9:205-209.
14. Phillips, RO, Fyhri A, Sagberg F. Risk Compensation and Bicycle Helmets. Risk Analysis (epub published ahead of print); 2011.
15. Walker I. Drivers overtaking bicyclists: Objective data on the effects of riding position, helmet use, vehicle type and apparent gender. Accident Analysis and Prevention. 2007;39:417-425.
16. Morrongiello BA, Walpole B, Lasenby J. Understanding children's injury-risk behavior: Wearing safety gear can lead to increased risk taking. Accid Anal Prev 2007;39:618-623.
17. Head Injuries in Canada, A Decade of Change, Canadian Institute of Health Information, August 2006.
18. Robinson DL. Costs and benefits of the NZ helmet law. http://cyclehelmets.org/papers/c2019.pdf
19. British Medical Association; Cycling towards Health and Safety, Oxford University Press, 1992.
20. Kennedy A. Exercise and heart disease: cardiac findings in fatal cycle accidents. B J of Sport Medicine 1997;31(4):328-331.
21. Hillman M. Cycling and the promotion of health, Policy Studies Vol 14, Policy Studies Institute, London.
22. Quantifying the positive health effects of cycling and walking http://www.euro.who.int/transport/policy/20070503_1v
23. Taylor RW. Obesity in New Zealand children: a weighty issue [editorial]. N Z Med J. 2007;120(1260). http://www.nzma.org.nz/journal/120-1260/2689/
24. Benefit of cycle helmet downgraded by study. nz.herald.co.uk http://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=10718438

25. Reported Road Casualties Great Britain: 2009, http://www.dft.gov.uk/pgr/statistics/datatablespublications/accidents/casualtiesgbar/rrcgb2009
26. Victorian Bicycling Strategy; Vic Roads, Australia 1990.
27. Climate Change http://www.mfe.govt.nz/publications/climate/nir-apr03/nir-apr03.pdf
28. Sustainable Transportation in New Zealand , http://www.ipenz.org.nz/ipenz/forms/pdfs/Sustainable_Transportation.pdf
29. Gill T, Cycling and Children and Young People – A review, National Children's Bureau, 2005. http://www.cycle-helmets.com/cyclingreport_timgill.pdf
30. European Cycling Federation. 'Improving bicycle safety without making helmet use compulsory; Brussels, Belgium. 1998. http://www.fiab-onlus.it/andare/helm_gb.doc
31. Curnow WJ. Bicycle helmets and public health in Australia, Health Promotion Journal of Australia, 2008 Apr;19(1):10-15.
32. Consumers Association (a British consumer protection organisation), Which?; Get a head start, p 28–31, October, UK, 1998. http://www.which.co.uk/about-which/
33. Helmet laws: New Zealand, Bicycle Helmet Research Foundation. http://www.cyclehelmets.org/1008.html
34. Health effects of vehicle emissions, http://www.transport.govt.nz/research/HealthEffectsofVehicleEmissions/

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Sun protection policies and practices in New Zealand primary schools

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Abstract

Aim For schools with primary age students, to report the percentages meeting specific requirements of the New Zealand SunSmart Schools Accreditation Programme (SSAP).

Methods Schools were randomly selected, within geographic regions, from the Ministry of Education schools database. A questionnaire, mailed to school principals, assessed schools regarding 12 criteria for accreditation: policy, information, hats, 'play in the shade', sunscreen, clothing, role modelling, curriculum, planning, rescheduling, shade provision and review. Post-stratification weights (for achieving each criterion) were used to compensate for oversampling within some regions and differential response rates between regions, using the number of schools per region.

Results 388 schools (representative in socioeconomic decile, size and type) participated. Less than 4% fully met accreditation criteria. Clothing (42%), curriculum delivery and shade (each 54%) requirements were met by the fewest schools. Staff role modelling (92%) was the most commonly met. Schools with uniforms tended to have more protective clothing expectations.

Conclusions Ongoing promotion is needed to consolidate gains and encourage comprehensive sun protection through policies, practices, environment and curriculum. Staff role modelling requirements may be strengthened by implementing existing occupational guidelines for mitigating UVR hazards. There is a need to further assist schools, particularly regarding sun protective clothing, curriculum delivery and environmental shade.

Skin cancer is a concern in New Zealand (NZ) where cutaneous malignant melanoma incidence is among the world's highest: 43.0 and 37.4 per 100,000 (age standardised to World Health Organization [WHO] world population) for men and women, respectively, and 371 deaths in 2008.[1] For each person dying from skin cancer an estimated average 15.5 potential years of life is lost, and skin cancers place a substantial burden on direct health system costs, estimated at $NZ57.1 M per annum, with total annual economic costs estimated at $NZ123 M.[2]

Yet most skin cancers are potentially preventable, as excessive solar ultraviolet radiation (UVR) exposure plays a key role in development,[3] causing as much as 65% of melanoma worldwide (95% in high exposure contexts like Australia) and 99% of basal cell and squamous cell carcinomas.[4] NZ UVR levels are ~40% higher than those at similar northern hemisphere latitudes in summer,[5] and experienced by a largely European population more susceptible to negative effects than groups indigenous to areas of comparably high UVR.

Excessive childhood solar UVR exposure increases the risk of skin cancers.[6,7] Since 'sun exposure in the first 10 years of life determines to a substantial degree the lifetime potential for skin cancer', there is 'a very strong case on epidemiological grounds for giving priority to the control of early life sun exposure.'[4] School settings are an identified priority,[8] since students can spend extended periods outdoors during school hours in organised and discretionary activities.

A recent review concluded there was 'sufficient' evidence that education and policy approaches can be effective for increasing sun protective behaviours in primary school settings.[9] An economic evaluation of the US 'SunWise' programme, a school-based programme similar to the SSAP, concluded that for every dollar invested, between $2 and $4 in medical care costs and productivity losses were saved.[10]

The WHO recommends as 'best practice' a comprehensive approach to school sun protection policy and practices, classroom teaching, and the education of parents and caregivers, with an award system to acknowledge effort,[11] similar to the Australian SSAP.[12] The NZ SSAP is modelled on the Australian programme, with administration and resource distribution coordinated through a comprehensive website, and support from Cancer Society of New Zealand (CSNZ) health promotion staff.

Schools must meet 12 criteria for accreditation: policy, information, hats, 'play in the shade', sunscreen, clothing, role modelling, curriculum, planning, rescheduling, shade provision and review. The present paper reports the 2009 distributions for each of these components among randomly selected NZ primary schools.

Methods

Sample—Two 10% samples of state or state integrated schools (representing 99% of primary-age children) were randomly selected, within geographical regions corresponding to CSNZ Divisions and Centres, from the Ministry of Education national schools database. The first included 200 from 1,999 then eligible schools (March 2005), with additional schools randomly selected to reach a minimum of 16 within each CSNZ centre, producing 242 participants. Subsequent re-organisation grouped CSNZ centres into six Divisions, listed from North to South in Table 1.

Replacements for non-responding schools were randomly selected within regions.[13] In 2009, 189 of these 242 schools agreed to participate again, supplemented with an additional, similarly selected sample (199 of 1,973 eligible schools) to strengthen analyses (Figure 1). This sampling process allowed the proportion of schools which reported following any particular guideline to be estimated using 95% confidence intervals (CI's) ± 25% within centres and ± 5.1% overall when looking at all 388 schools. Three institution types were represented: Full Primary (Years 1–8; age ~5 to 13 years), Contributing (Years 1–6; age 5–11 years) and Composite/Area (Years 1–13; age 5–18 years) schools.

Instrument—The survey instrument to assess sun protection policy, practice, curriculum and environment was adapted from Australian precedent[14] in consultation with CSNZ staff developing SSAP application forms. Minimum criteria and requirement(s) are directly related to these forms (Table 2). The CSNZ proposed no weighting for programme components and, since non-subjective weighting was considered difficult to achieve and justify, each criterion was treated as of equal weight. The CSNZ proposed that, although all criteria needed to be met in order to achieve accreditation, no arbitrary level of compliance was required to register—the goals being to facilitate participation, reduce barriers and monitor progress.

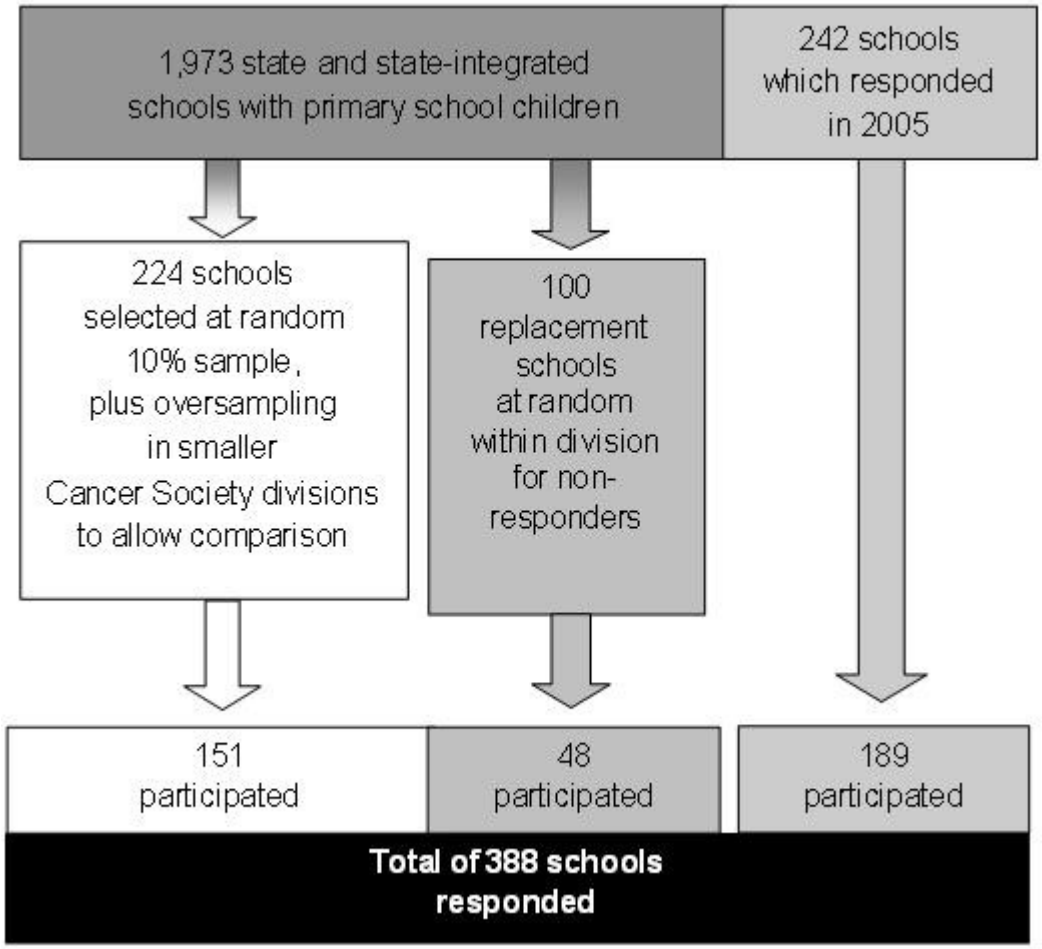
Procedures—The survey was mailed (2 September 2009) to school principals, with a Freepost, pre-addressed envelope enclosed and a request to return completed questionnaires and related policy documents. Scheduled follow-ups were by email (23 September) and post (23 October—which included an extra questionnaire in case the original was misplaced.

Further email and telephone reminders followed and, where possible, the principal was contacted directly and urged to complete the survey in order to facilitate a representative summary of the situation in primary schools.

All participants were asked to respond to questions in relation to primary students (Y1–6) and practices in Terms 1 and 4, when solar UVR can reach 'very high' to 'extreme' levels[15] and sun protection is recommended. Ethical approval was obtained at the Departmental level (20 August 2009), following University of Otago procedures.

Analysis—Responses to specific survey questions determined the attainment of each criterion and were analysed with Stata v11.1 statistical software. Most measures used fixed response options, but some included an 'other' option, allowing alternatives to be recorded, collated and coded as discrete responses. Sampling and post-stratification weights, using the number of schools per region in 2009, were used to estimate percentages giving particular responses and achieving each criterion, compensating for oversampling within some regions and differential response rates between regions.

Figure 1. Sample selection and response to the 2009 survey of NZ schools with students of primary school age



Results

Of the 242 schools in the baseline survey, 78% participated at follow-up. In 2009, 199 of the 324 (61%) additional randomly selected schools participated.

Overall, the 388 participating schools were comparable with schools nationally in socioeconomic decile distribution (level 1 being the 10% of schools with the highest proportions of students enrolled from low socioeconomic communities) and institution type (Table 1).

Participating schools were representative of the national distribution of primary school size. Because of the 'boosting' to a minimum number in the smaller centres, the geographical distribution of participating schools was somewhat different to the national distribution, with higher representation from divisions which contained smaller centres in the original sampling.

Adjustments were made to correct the effect of this oversampling. Overall, the responses of the participating schools are likely to provide a representative and comprehensive picture of sun protection practices in NZ primary schools.

Table 1. Characteristics of all eligible New Zealand schools with primary school age children, and those schools participating in the 2009 survey

School characteristic	All eligible NZ schools with primary age children (n=1999)	All schools participating in 2009 survey (n=388)	
Integration status	**%**	**%**	**n**
State	87	86	333
State-integrated	13	14	55
Socioeconomic decile			
1*(lowest)*–3	31	30	114
4–7	38	41	160
8–10 *(highest)*	31	29	114
Institution type			
Full primary	55	53	205
Contributing	40	43	167
Composite	5	4	16
CSNZ divisions			
Auckland	24	24	92
Waikato / Bay of Plenty	19	18	71
Central Districts	20	22	84
Wellington	13	16	61
Canterbury	14	12	45
Otago / Southland	10	9	35
School roll size*			
<50	19	18	70
51–100	14	17	66
101–150	13	11	42
151–200	12	16	61
201–300	16	17	64
301–400	10	7	28
>401	16	15	56

* Roll size was not available for one school in the national database so percentages are based on 387 schools.

Table 2. Minimum criteria for SSAP accreditation, and percentages of schools attaining each of 12 criteria, based on survey responses

	Minimum criteria	Requirement(s) to meet*	Attained %
Policy	Sun protection policy is implemented during terms 1 and 4, when UVR levels most intense.	Either a Sun Protection Policy or a sun protection section in the Health and Safety Policy is in place Copy of policy returned with survey	58
Information	All staff, students and parents / caregivers are to be informed of the skin protection policy and its intended practices.	Some information given to parents/caregivers at enrolment At least three methods used to convey general sun protection messages at school	87
Hats	All students wear a broad brimmed (minimum 7.5cm brim), legionnaire or bucket hat (minimum 6cm brim, deep crown) when outside.	Hat wearing enforced Broad-brimmed, legionnaires or bucket hats ONLY used at school	74
Play in shade	Students not wearing a hat are required to play in allocated shade areas	Hat wearing enforced Consequences for students not wearing hats	87
Sunscreen	The use of SPF 30+ broad spectrum sunscreen is encouraged.	Students 'actively encouraged' to wear sunscreen SPF 30+ sunscreen available at school	77
Clothing	The use of sun protective clothing is encouraged (e.g. sleeves and collars).	Students encouraged to wear shirts with collars and longer sleeves One of the following is true: a uniform schools had sun protective options b non-uniform schools require midriff covered and ban singlets/spaghetti strap tops	42
Role model	Staff are encouraged to act as role models by practising SunSmart behaviours.	Staff encouraged to wear broad-brimmed, bucket or legionnaire hat	92
Curriculum	SunSmart education programmes are included in the curriculum at all levels every year.	Extended teaching on sun protection taught at all levels every year	54

Planning	The sun protection policy is reflected in the planning of all outdoor events (e.g. camps, excursions, sporting events).	One of the following is true: a. sunscreen is available for student use on specific occasions b. sports days are held before 11am or after school hours c. outdoor excursions are scheduled early where possible	76
Rescheduling	Outdoor activities are rescheduled, whenever possible, to minimise time outdoors between 11 am and 4 pm.	At least two of these eight are true: a. assemblies held indoors, under shade or before 11am b. lunch is eaten in shaded areas or indoors c. teachers asked to use shade for outdoor classes after 11am d. PE classes held before 11am e. outdoor excursions scheduled early in the day where possible f. children can stay indoors on fine days for breaks g. sports days before 11am or after school h. extended morning tea break / short lunch break	91
Shade	The school has sufficient shade or is working towards increasing the number of trees and shade structures so as to provide adequate shade in the school grounds.	One of the following is true: a. substantial shade for active and passive activities b. definite plans to increase shade in next 12 months.	54
Review	The Board of Trustees and Principal review the sun protection policy regularly, including making suggestions or improvements at least once every three years.	Sun Protection Policy or section of policy is in place Copy of policy returned with survey	58

*Schools to meet each point listed, with some sub-points, as outlined.

Note: The time period requirement for Planning and Rescheduling has subsequently been extended to cover the period from 10am to 4pm.

The percentages of schools attaining each of the 12 SSAP criteria are presented in Table 2. Details of responses to specific survey questions are reported for each criterion.

Policy—The written sun protection policies provided included 10% with a section in their Health and Safety Policy. Additionally, 9% had a policy 'under development'.

Information—Schools were asked to indicate, from a list, which things parents/caregivers were informed about, during enrolment, regarding sun protection at school—multiple responses were allowed. Information was most commonly provided about the requirement for children to wear hats when outside (92%), the need for parents 'to supply their children with sunscreen to take to school' (49%), encouragement 'to wear clothing that protects the skin from the sun' (46%), and for adults 'to practice sun protection behaviours when involved with the school' (45%). Two percent indicated that no sun protection information was given. Respondents were also asked to indicate from a list which method(s) their school used to convey messages about sun protection (Table 3).

Table 3. Methods of conveying sun protection messages at school

Method	Schools reporting %
Reminders about sun protection are given:	
in class regularly during Terms 1 and 4	83
in assemblies regularly during Terms 1 and 4	71
in newsletters regularly in Terms 1 and 4	71
in assemblies at the beginning of Terms 1 and 4	67
at staff meetings	49
once or twice a year	41
Posters about sun protection are displayed around the school	41
The maximum predicted clear sky UVI for the day is displayed	1

Hats—Hat wearing was 'enforced' in 87% of schools and 'encouraged' in the remainder. With respect to the types of hats worn by students, multiple responses were permitted. In 64% of schools, students wore either 'broad-brimmed hats (at least 7.5 cm brim)', legionnaire hats, or bucket hats (at least 6 cm brim and deep crown).

The remaining 23% of schools may have chosen one of those protective options, but additionally chose an option unacceptable for accreditation: 'any hat', 'bucket hats (less than 6 cm brim)', or a free response, such as 'caps'. When enforcement was re-examined in relation to hat type, a more realistic picture emerged. Overall, 60% reported enforcing the wearing of broad-brimmed, legionnaire, or big bucket hats.

Play in the shade—The most common consequence for not wearing a hat when outside was that students 'must play in the shade' (81%). Other consequences selected from a list of four options included that 'they must play indoors' (13%) or 'they must wear a hat from school spare hats' (30%). Write-in responses included punitive measures (such as 'time out', exclusion from physical education (PE) class or sitting in shade with *no play* allowed), encouragement to wear a hat 'next time', or measures 'differing in different conditions'.

In 6% of schools there was 'no restriction or consequence'. Although not penalised a second time for the type of hats worn, respondents were required to indicate that hat wearing was enforced, not simply encouraged.

Sunscreen—Schools reported that: 'students are actively encouraged to use sunscreen' (87%); 'parents are encouraged to provide sunscreen' (49%); sunscreen is available 'in all classrooms' (61%); and available 'at various points around the school' (38%).

Sunscreen was not supplied in 7% of schools, including 5% which neither supplied nor encouraged parents to provide sunscreen. SPF30+ was the most common type of sunscreen available at school (88%), with an additional 9% having at least SPF 15.

Clothing—Opportunities to choose sun protective clothing were explored through three questions about uniform or dress codes. Overall, 48% of schools had a uniform and most of these (98%) had a polo type shirt for summer use, whereas 30% had an option that included elbow length sleeves or longer. However, in 46% of these schools, male students were required to 'wear shorts that fall above the knee' and in 43% female students were required to 'wear shorts or skirts that fall above the knee', indicating that uniforms did not necessarily include sun protective options.

For non-uniform schools, we assumed there would be no restriction on wearing protective clothing, but for both uniform and non-uniform schools we were interested to know about protective expectations. There were substantial differences in some clothing expectations between uniform and non-uniform schools (Table 4).

Table 4. Percentages of uniformed and non-uniformed schools meeting various clothing expectations or dress code options in 2009*

Clothing guidelines at school	All %	Uniform %	Non-uniform %
Students encouraged to wear shirts with collars & longer sleeves	39	40	39
Wearing of singlets or 'spaghetti-strap' tops is forbidden	40	60	20
Students allowed to wear sunglasses	64	61	67
Students must not show midriffs	47	64	29
Students must wear shirts for PE/outdoor activities	56	67	44
None of the above	9	4	14

* Calculations based on 367 schools with complete data for clothing questions.

Role modelling—In most schools (92%) staff were 'encouraged to wear a sun protective hat during school outdoor activities and breaks in Terms 1 and 4.'

Curriculum—An accreditation question specifically asked if an 'extended' session on sun protection was taught as part of either the Science or Health/PE curriculum 'at all levels throughout the school every year', in order to distinguish schools which simply gave reminders about protection (covered by the 'information' criterion). Overall, 46% attained this criterion through the Health / PE curriculum, and an additional 7% through the Science curriculum.

Planning—Although there were no specific questions (in the survey or SSAP application form) about planning outdoor events, several questions addressed related

issues. To meet this criterion, respondents needed to indicate at least one way in which sun protection was considered.

Rescheduling—Although there was potential overlap between the 'planning' and 'rescheduling' criteria, the latter focused on practices by means of which time spent outside between 11am and 4pm was minimised (Table 5).

Table 5. Percentages of schools reporting rescheduling practices to minimise time spent outside, 11am–4pm, Terms 1 and 4

Variables	%
Assemblies are either held indoors, or, if outdoors, are held under shade or before 11am	88
Lunch is eaten in shaded areas *	87
Teachers are requested to use shade for outdoor classes after 11am	27
PE classes are held before 11am	19
Lunch is eaten indoors *	19
Outdoor excursions are scheduled early in the day where possible	16
There is an extended morning tea break & a shortened lunch break	15
Sports days are held before 11am or after school	13
Children are allowed to stay indoors during breaks on fine days	10

* For accreditation, these two categories were combined (as on SSAP application form), so schools needed to indicate an additional practice in order to meet the rescheduling criterion. In 5% of schools, lunch was eaten neither indoors nor in shade.

Environmental shade—When indicating the 'situation at your school with respect to shade', 13% of respondents selected the most protective option: 'substantial shade available for both passive and active activities.' Most respondents indicated that there was 'sufficient existing shade for most students to sit under for passive activity' (52%), including areas for eating lunch and outdoor classroom activities. An additional 31% indicated that there was 'some useful shade, but insufficient for most activities', whereas 4% agreed that there was 'inadequate shade for students to use for any activity.'

Among those without 'substantial shade', 21% had 'definite plans to increase shade in the next 12 months' and 26% in 1–3 years. However, 23% reported that 'providing shade is not currently a priority area' and 38% indicated that increasing shade 'poses funding concerns.' Formal shade assessment is not required for accreditation and for 67% of all schools there was 'no documented assessment of shade provision.' Few schools carried out a 'formal shade inventory or shade audit' (5%), although in 23% there had been a 'less formal, but written, assessment of shade provision.'

Review—In response to the question: 'Do your Board of Trustees and Principal review the sun protection policy or guidelines at least every 3 years?', 82% responded affirmatively with an additional 12% indicating that was the intention, but the policy or guidelines were less than 3 years old.

Table 6. Percentages of schools achieving total accreditation scores 12–1; weighted to correct for oversampling in some areas

Total score	Schools achieving (%)
12	3.9
11	14.7
10	18.0
9	15.9
8	16.7
7	14.0
6	8.4
5	2.5
4	3.4
3	2.0
2	0.5
1	0.0

Discussion

In 2009, most NZ primary schools only partially addressed sun protection, with 4% meeting all 12 criteria for accreditation. An additional 15% met 11 criteria and could potentially achieve accreditation with relatively limited changes. However, 52% of Australian schools surveyed in 2005 had attained SunSmart status.[16]

Furthermore, whereas 58% of NZ schools reported having a written school sun protection policy, 80% of Australian schools had such a policy in 2005. Nevertheless, Australian and NZ levels are both substantially higher than those generally reported for the US,[17–19] other than California and Colorado, where elementary school policies are controlled at district level.[20] Higher level administrative procedures may exert a positive influence, so stronger support for sun protection through the National Administrative Guidelines (NAGs) for health and safety in NZ schools may provide more sun safe environments more quickly.

The NZ SSAP criteria least likely to be met relate to clothing (42%), shade and the curriculum (54% each). With respect to clothing, there is a need to ensure that attractive, suitably protective and affordable products are readily available and, if not, to work with suppliers to achieve that. The provision of adequate shade can be costly and requires professional guidance to achieve optimal placement at the required time. Although a substantial NZ manual is available,[21] relatively few professionals seem to be engaged and there are limited training opportunities for architects and planners. Considerable improvement would probably be achieved if shade was required to be considered in all school building plans.

With respect to the curriculum, some suitable and attractive components are available,[22] but these deserve greater promotion to all levels of the education sector. Development of further suitable curricular materials should also be a priority.

The ‘role modelling’ criterion may be too lax, and could be strengthened as part of occupational UVR hazard management.[23] Rescheduling of outdoor excursions and sports days (Table 5) could receive greater attention, and the high UVR period

immediately after school hours (3–4pm), which falls outside of school jurisdiction, deserves greater attention as a time when harmful UVR exposure may occur.

The SSAP currently only applies to primary and intermediate level students, with a lack of continuity through the secondary level. Earlier research found that few secondary schools had a written sun protection policy, there was little evidence of related curriculum content and students often lacked knowledge and protective attitudes.[24]

Study limitations—The present study was based on reports from school staff, which may overestimate positive practices. However, as an adjunct to this study, on-site visits were conducted at 22 primary schools,[25] and there was broad agreement between observation and practices reported as survey data. Measures in the survey instrument were based on Australian precedent and not specifically tested for validity or reliability in NZ, but were similar to the SSAP application form. Statistical predictors of accreditation scores, changes over time, and regional differences will be addressed elsewhere.

Conclusions

A review of interventions for the primary prevention of skin cancer in children and adolescents concluded that:

- The most effective interventions used multi-component curricula administered over extended time periods;
- Multi-unit or multi-component programmes demonstrated greater success in achieving improvement in sun protection knowledge, awareness, behaviours and attitudes and should replace short-term, single-faceted programmes.[26]

The SSAP takes such an approach, but requires consolidation. Although progress has been made towards making NZ primary schools safer for students with regard to sun protection, there remains considerable room for improvement. Schools struggled most in the areas of curriculum delivery, clothing protection and provision of adequate shade. There is a need to further assist developments in these areas.

Incomplete implementation of the SSAP potentially leaves a substantial number of NZ primary school children at risk of harmful UVR exposure at school and less than fully informed about the rationale and need for sun protection in other contexts.

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References:

1. Ministry of Health. Cancer: New registrations and deaths 2008. Wellington: Ministry of Health; 2011.
2. O'Dea D. The costs of skin cancer to New Zealand. A report to the Cancer Society of New Zealand Inc. Wellington: University of Otago; 2009. http://www.cancernz.org.nz/reducing-your-cancer risk/sunsmart/about-skin-cancer/costs-of-skin-cancer/
3. Lucas R, McMichael T, Smith W, Armstrong B. Solar ultraviolet radiation. Global burden of disease from solar ultraviolet radiation. Environmental burden of disease Series No. 13. Geneva: WHO; 2006.
4. Armstrong B. How sun exposure causes skin cancer: an epidemiological perspective. In: Hill D, Elwood JM, English DR, editors. Dordrecht: Kluwer Academic Publishers 2004, p89-116.
5. McKenzie R, Bodeker G, Scott G, et al. Geographical differences in erythemally-weighted UV measured at mid-latitude USDA sites. Photochem Photobiol Sci. 2006;5:343-52.
6. Oliveria SA, Saraiya M, Geller AC, et al. Sun exposure and risk of melanoma. Arch Dis Childhood. 2006;91:131-8.
7. Marks R. Epidemiology of non-melanoma skin cancer and solar keratoses in Australia: a tale of self-immolation in Elysian fields. Australas J Dermatol. 1997;38(S1):S26-9.
8. Ministry of Health. The New Zealand Cancer Control Strategy Action Plan 2005-2010. Wellington: Ministry of Health and New Zealand Cancer Control Trust; 2005.
9. Saraiya M, Glanz K, Briss PA, et al. Interventions to prevent skin cancer by reducing exposure to ultraviolet radiation: a systematic review. Am J Prev Med. 2004;27:422-66.
10. Kyle JW, Hammitt JK, Lim HW et al. Economic evaluation of the US Environmental Protection Agency's SunWise program: sun protection education for young children. Pediatr Rev. 2008;12:e1074-84.
11. World Health Organization. Sun protection and schools. How to make a difference. Geneva: WHO; 2003. http://www.who.int/uv/publications/en/sunprotschools.pdf
12. Dobbinson S, Peipers A, Reading D, Sinclair C. A national approach to skin cancer prevention: the National SunSmart Schools Program. Med J Aust 1998;169:513-4.
13. Reeder AI, Jopson JA, Gray A. Baseline survey of sun protection policies and practices in primary school settings in New Zealand. Health Educ Res. 2009. 24:778-87.
14. Anti-Cancer Council of Victoria. SunSmart Program 2003-2006. Melbourne: Anti-Cancer Foundation of Victoria; 2002.
15. World Health Organization, World Meteorological Organization, United Nations Environment Programme, International Commission on Non-Ionizing Radiation Protection. Global solar UV index: a practical guide. Geneva: World Health Organization; 2002.
16. Jones SBW, Beckmann K, Rayner J. Australian primary schools' sun protection policy and practice: evaluating the impact of the National SunSmart Program. Health Prom J Austral. 2008;19:86-90.
17. Buller DB, Geller, AC, Cantor M, et al. Sun protection policies and environmental features in US elementary schools. Arch Dermatol. 2002;138:771-4.
18. Eakin P, Maddock J, Techur-Pedro A, et al. Sun protection policy in elementary schools in Hawaii. Prev Chronic Dis. 2004;1:1-10.
19. Geller AC, Zwirn J, Rutsch L, et al. Multiple levels of influence in the adoption of sun protection policies in elementary schools in Massachusetts. Arch Dermatol. 2008;144:491-6.

20. Buller DB, Klein Buller M, Ashley J. Sun Safe Schools: Baseline survey in a campaign to motivate adoption of sun protection policies by public school districts in California and Colorado. San Diego USA: American Public Health Association 136th Annual Meeting and Expo. http://apha.confex.com/apha/136am/webprogram/Paper180541.html
21. Greenwood JS, Soulos GP, Thomas ND. Under Cover: Guidelines for shade planning and design. Wellington: Cancer Society of New Zealand. 2000.
22. Cancer Society of New Zealand Inc. The SunSmart Learning Programme Wellington: CSNZ. http://www.sunsmartschools.co.nz/teachers/curriculum-resources
23. Health and Technical Services, Occupational Safety and Health Service, Department of Labour. Guidance notes for the protection of workers from solar ultraviolet radiation. Wellington: Occupational Safety and Health Service; 1997.
24. Jopson JA, Reeder AI. Sun protection in New Zealand secondary schools: Obstacles and opportunities. Technical Report UR 8. Dunedin: University of Otago, Cancer Society Social & Behavioural Research Unit; 2004.
25. Jopson JA, Reeder AI. SunSmart voices: Results from site visits to 22 primary schools throughout New Zealand. Technical Report UR20. Dunedin: University of Otago, Cancer Society Social & Behavioural Research Unit; 2011.
26. Hart KM, Demarco RF. Primary prevention of skin cancer in children and adolescents: a review of the literature. J Pediatr Oncol Nurs. 2008;25:67-78.

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Should measurement of vitamin D and treatment of vitamin D insufficiency be routine in New Zealand?

Mark J Bolland, Andrew Grey, James S Davidson, Tim Cundy, Ian R Reid

Abstract

Epidemiological studies have reported associations between lower vitamin D levels and a great variety of diseases, prompting calls for widespread treatment of individuals with low vitamin D levels.

Most of New Zealand's population have vitamin D levels for at least part of the year that are considered insufficient (25-hydroxyvitamin D <50–80 nmol/L). However, evidence for benefits of vitamin D supplementation in such populations is controversial and there is some evidence of harmful effects. Until adequately powered, randomised, controlled trials of vitamin D supplementation demonstrate safe improvements in health, clinicians should not focus on detecting/treating individuals with vitamin D insufficiency, instead treating those at high risk of vitamin D deficiency (25-hydroxyvitamin D <25 nmol/L), such as the frail elderly, and those with specific clinical indications. Treatment for such individuals does not require vitamin D measurements.

Requests for vitamin D measurements in Auckland have nearly quadrupled in the past decade, from 8500 in the year 2000 to 32,800 in 2010, with substantial increases in cost. Vitamin D measurement is often inaccurate and imprecise, and the vast majority of tests performed currently do not reveal vitamin D deficiency. Therefore, a move away from routine vitamin D measurements seems sensible, though they are still indicated when investigating suspected metabolic bone disease or hypocalcaemia.

Background

Vitamin D deficiency causes rickets in children and myopathy, osteoporosis, secondary hyperparathyroidism, and osteomalacia in adults.[1] The precise vitamin D level at which these conditions occur is not known and may be influenced by other factors such as dietary calcium intake, but a widely accepted definition of vitamin D deficiency is serum 25-hydroxyvitamin D (25OHD) <25 nmol/L.[1]

More recently, vitamin D insufficiency, variously defined as 25OHD <50-80 nmol/L,[2] has been associated in epidemiological studies with a very wide variety of diseases, including cancer, neurological disorders, vascular diseases, infectious conditions, autoimmune diseases, osteoporosis, type 2 diabetes mellitus and obesity.[3] These findings have prompted a substantial increase in requests for vitamin D measurements and led to calls for widespread treatment of individuals with "insufficient" vitamin D levels.[3]

Defining vitamin D insufficiency as 25OHD <80 nmol/L classifies most of New Zealand's population as vitamin D insufficient. In the 1997 National Nutrition survey, the mean 25OHD level was 50 nmol/L and only 25% of individuals had levels >70

nmol/L.[4] Thus, the implication of recommending treatment of vitamin D insufficiency is that most of New Zealand's population would be treated, at least in the winter and spring months when 25OHD levels are lowest.

A policy of such widespread use of vitamin D supplements should only be implemented in a context of rigorous evidence of the benefits and safety of vitamin D supplements in populations with vitamin D insufficiency. Here, we present 10 reasons why such a policy should not currently be implemented, and discuss a strategy for sensible policy on measurement of vitamin D.

Evidence for treating vitamin D insufficiency

Defining vitamin D insufficiency: many methods, much inconsistency, no consensus—Vitamin D insufficiency can be defined using surrogate endpoints such as intestinal calcium absorption, parathyroid hormone (PTH) levels, bone density, or clinical endpoints such as falls, fractures or cardiovascular events, and the definition can be based on data from randomised control trials (RCTs) or observational (non-experimental) studies.[2]

Most definitions are based on surrogate skeletal endpoints such as PTH, although there is little, if any, evidence that such surrogate endpoints are valid predictors of key clinical endpoints such as fracture, and it is not clear what surrogate endpoints should be used for non-skeletal clinical events. Moreover, definitions based upon data from observational studies vary substantially from those based upon RCTs, and definitions derived using different surrogate endpoints are also inconsistent.[2] Consequently, definitions of vitamin D insufficiency range from as low as 30 nmol/L to >100 nmol/L,[2] with some experts now recommending a threshold of 50 nmol/L and others 75-80 nmol/L.[2]

The Institute of Medicine recently concluded that a level of 40 nmol/L represents the median population requirement.[5] None of these definitions states how seasonal variation of 25OHD should be dealt with: the month of vitamin D measurement is the strongest determinant of 25OHD levels in populations distant from the equator.[6,7] Presumably these thresholds refer to the lowest 25OHD level throughout the year, which occurs in late winter or early spring. If this is the case, summertime 25OHD levels much higher than these thresholds are required to ensure year round vitamin D sufficiency. For example summertime 25OHD levels of 90-120 nmol/L can be required to ensure wintertime 25OHD >80 nmol/L.[8]

Measuring vitamin D is difficult and expensive—25OHD is the metabolite that best reflects overall vitamin D status, but measuring it is technically challenging, and there is substantial variation in results between assays, and between laboratories reporting 25OHD values.[9-11] The assays that are generally accepted as the most accurate [liquid chromatography tandem mass spectrometry (LC-MS/MS)] are not widely available.

Typical 25OHD immunoassays may give results differing by up to 40% from the LC-MSMS result,[10] meaning that clinicians cannot be certain that measurements of 25OHD in the insufficient range truly indicate low vitamin D status. A further drawback is that measurement of 25OHD is expensive, and a single measurement can cost substantially more than the annual cost of vitamin D supplements for an individual.

Interpreting observational studies- correlation is not causality—Almost all of the recent data linking vitamin D insufficiency with non-skeletal endpoints comes from observational studies, but by design, observational studies can only ever show associations between variables and cannot prove a causal relationship.[12]

Observational studies usually divide the cohort into various subgroups by 25OHD levels, and compare outcomes between the subgroups. However, the baseline characteristics of the subgroups vary- vitamin D insufficient subgroups are older and heavier, exercise less, have more co-morbidities and are more frail/less healthy than vitamin D sufficient subgroups.[13] Researchers usually try to account for these differences by adjusting for the variables which differ between the subgroups in their models. However, adjusting for healthiness is difficult, and such adjustments may not include all relevant variables in the model, and/or may not account for the differences in health between the groups.[14]

A further bias rarely considered is the constant risk fallacy. Adjusting for covariates assumes that the relationship between the covariate and the outcome is consistent for all values of the covariate.[15] However, this assumption is rarely tested and often untrue. For example, there has been a consistent secular trend for increasing life expectancy over recent decades, thus a cohort aged 60y will have a lower mortality rate than a cohort aged 70y even taking account of the 10y age difference between the groups. Similar differences may apply to other variables relevant to vitamin D insufficiency, such as obesity.

Interpreting observational studies- seasonal issues specific to vitamin D—In countries distant from the equator, 25OHD varies substantially throughout the year in a non-linear manner. This can lead to misclassification, for example when a 25OHD measurement in summer is classified as "sufficient" although, because of seasonal variation, a 25OHD measurement from the same subject in winter would be classified as "insufficient".[8] Some studies do not account for seasonal variation, others group individuals by season of measurement, ignoring the substantial changes in 25OHD that occur within each season, and others adjust for season using linear techniques even though the seasonal variation in 25OHD is non-linear. The effects of such misclassification have not been well studied.

Interpreting observational studies-conflicting results—While numerous observational studies report associations between vitamin D insufficiency and clinical endpoints, a substantial number do not. For example, while some studies have reported increases in fracture incidence with lower 25OHD levels,[16-19] others have not.[13,20-22] The *lack* of association between vitamin D insufficiency and clinical endpoints in such studies is rarely acknowledged, and often dismissed, by enthusiasts for vitamin D supplementation, and raises the possibility of publication bias in favour of studies reporting associations between vitamin D insufficiency and negative health outcomes.

Interpreting RCTs- calcium and vitamin D are not interchangeable—Many RCTs have tested the intervention of co-administered calcium and vitamin D supplements, yet positive results are attributed to vitamin D. For example, a study that is commonly cited as evidence for benefits of vitamin D on cancer incidence, was actually a comparison of 3 treatments: co-administered calcium and vitamin D, calcium alone,

or placebo.[23] Co-administered calcium and vitamin D had a relative risk of 0.4 for cancer incidence compared with the placebo group.

To determine whether there was an independent effect of Vitamin D, the appropriate comparison is to compare the co-administered calcium and vitamin D arm with the calcium alone arm. For this comparison, there was no significant between-groups difference in the risk of cancer. Similarly, in a meta-analysis of fracture outcomes in 17 trials of calcium with or without vitamin D supplementation, the relative risk of fracture was 0.9 with calcium alone, and was 0.87 with co-administered calcium and vitamin D.[24]

In this meta-analysis, all of the trials studied vitamin D in daily doses of ≤800 IU/day. Similarly, a meta-analysis of 18 trials of vitamin D supplements reported a 7% reduction in mortality.[25] However, 13/18 trials were of co-administered calcium and vitamin D, and vitamin D supplements by themselves did not reduce mortality in this or subsequent meta-analyses.[26,27]

These analyses suggest the addition of vitamin D to calcium supplements does not substantially impact upon either cancer incidence, or fracture incidence, and possible mortality benefits might require co-administered calcium and vitamin D supplements.

RCT evidence for benefit of vitamin D is inconsistent—Results from RCTs of vitamin D supplements for skeletal endpoints in community-dwelling populations with vitamin D insufficiency have been largely negative. Meta-analyses of these studies report no benefit of vitamin D supplements on hip or total fracture when vitamin D alone is the intervention or when calcium and vitamin D are co-administered.[26]

In contrast, in two studies of vitamin D deficient, institutionalised, elderly women at high risk of fracture, co-administered calcium and vitamin D supplements reduced hip and non-vertebral fracture incidence.[28,29] There are a number of RCTs that have reported data on non-skeletal endpoints but few have been adequately powered. While a number of RCTs have reported positive benefits from vitamin D, generally there are more studies, often larger in size and longer in duration, that report no effects. For example, systematic reviews have concluded there is no existing evidence that vitamin D supplements prevent cardiovascular disease,[27,30,31] type 2 diabetes,[27,31] or cancer,[32] or impact upon blood pressure, blood glucose or cholesterol.[27]

Individual RCTs have shown no benefits of vitamin D supplements on body weight,[33] and conflicting results for incidence of respiratory infections.[34,35]

Interpreting meta-analyses: proceed with caution—By mid 2009, there were at least 9 meta-analyses of the 17 RCTs of vitamin D and falls, and at least 14 meta-analyses of the 22 RCTs of vitamin D and fractures.[36] The conclusions vary substantially between analyses, with most reporting no effect, but some reporting strongly positive findings. These conflicting conclusions have generated substantial confusion. The differences between analyses mainly arise from the heterogeneous patterns of results from RCTs, so that methodological decisions regarding inclusion criteria and sub-grouping of studies in the meta-analysis largely determines the results obtained.[36]

Potential for harms from vitamin D supplementation—Discussion on harms from vitamin D supplements has focused almost exclusively on hypercalcaemia, which only occurs as a result of administration of very high doses of vitamin D. However, a recent placebo-controlled RCT of 2256 older women with median baseline 25OHD of 49 nmol/L reported that an annual dose of 500,000 IU vitamin D increased the risks of fractures and falls by 26% and 15%, respectively.[37]

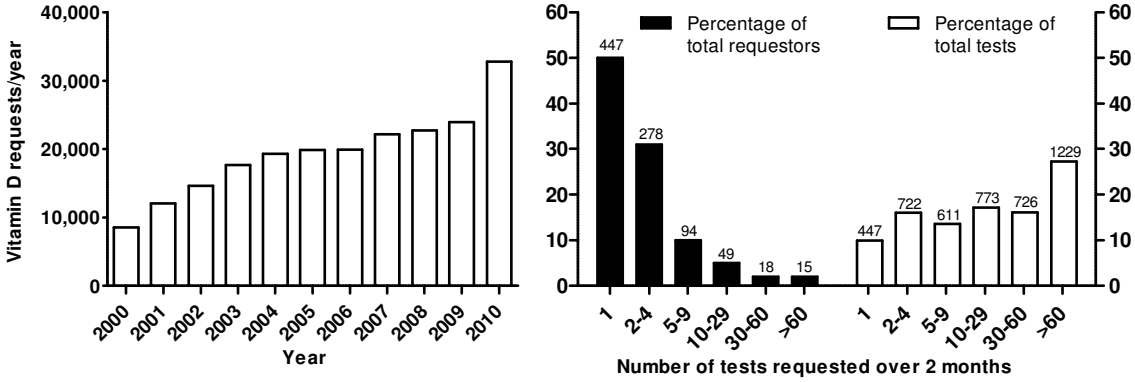
Consistent with these results, another recent RCT of 173 people following hip fracture with mean 25OHD of ~30 nmol/L, compared daily 2000 IU vs. 800 IU vitamin D and reported a 28% (P=0.06, not significant) increase in falls with higher dose vitamin D.[38] Meta-analyses of RCTs of vitamin D used without co-administered calcium report an increase in risk of hip fracture of 15% (95% confidence interval -1% to 33%) of borderline statistical significance.[26]

Some observational studies have reported increased mortality in individuals with higher 25OHD levels compared with intermediate levels,[39,40] although there is no evidence of increased mortality with vitamin D supplementation in meta-analyses of RCTs,[25-27] in which the typical dose studied was 400–800 IU/day. The narrow focus on hypercalcaemia as the only possible and relevant harm of vitamin D supplements was never appropriate and can no longer be defended.

Recent history–déjà vu all over again—Finally, in the very recent past there has been similar enthusiasm for hormone replacement therapy, antioxidants, and folic acid/B vitamins for cardiovascular disease or cancer prevention, based upon promising results from observational studies. When the definitive RCTs have been carried out, harms[41–43] or no benefits[44–46] of these agents were observed.

Implications of vitamin D enthusiasm for health costs—In the Auckland region, there has been an increase of 380% in the number of requests for measurement of 25OHD in the past decade, from 8,500 in 2000 to 32,800 in 2010 (Figure 1). A large proportion of the requests come from a small number of individual requestors. In May and June 2010, 4508 vitamin D measurements were requested by 901 individuals. 50% of individuals requested one measurement over these 2 months, and 80% <5 measurements. However, these 80% of individual requestors only accounted for 26% of the total measurements. 2% of individuals requested 27% of the total measurements, and 9% and 19% requested 61% and 74% respectively of the total measurements (Figure 1).

Figure 1. Number of vitamin D requests per year and by individual requestors over a 2-month period



The left panel shows the total number of requests for vitamin D measurements between 2000 and 2010. The right panel shows the relationships between percentage of total requestors (dark bars) or percentage of total tests (open bars) and number of tests per requestor, over a two month period (May, June 2010). The numbers above each bar represent total number of individual requestors (dark bars) or the total number of vitamin D requests (open bars).

The current total cost of a single measurement of 25OHD is $31.10. Thus, the total cost of vitamin D measurement in 2010 in the Auckland region exceeded $1 million. In a previous analysis of >21,000 consecutive results in adults, 25OHD was <25 nmol/L in only 15%, and was ≥50 nmol/L in 52%,[47] suggesting that the considerable majority of tests are carried out in individuals at low risk of vitamin D deficiency.

There was considerable variation in vitamin D status by season: the proportion with vitamin D deficiency was 8%, 10%, 20%, and 21% in summer, autumn, winter, and spring, respectively. Six measurements were needed to detect 1 case with vitamin D deficiency, and the cost of these 6 tests was $186. In the summer months, 13 measurements were needed to detect 1 case, costing $404. In context, a treatment course of vitamin D 50,000 IU monthly for 1 year costs <$10 per patient.

Given that measurement of 25OHD is expensive and mostly identifies people with vitamin D levels in the range for which there is no compelling evidence of benefit from routine supplementation, the utility of 25OHD measurements must be questioned. Furthermore, as most of the 25OHD assays currently in use in New Zealand do not have high precision or accuracy, particularly at lower levels of 25OHD (Christchurch is the only centre that uses a LC-MS/MS assay), clinicians cannot be confident that the result of a single measurement of 25OHD accurately reflects their patient's vitamin D status. Thus, it can reasonably be argued that vitamin D measurements should only be requested when the result is likely to change patient management.

There are a few clear cut examples: investigation of rickets or osteomalacia and other uncommon metabolic bone diseases, and hypocalcaemia. For most individuals who are at high risk of vitamin D deficiency, which includes those with deeply pigmented skin, the frail elderly, and those who actively avoid the sun for cultural or medical

reasons, treatment with vitamin D supplements or encouraging sensible sunshine exposure is reasonable without the need for vitamin D testing.

ACC also recommends that people living in residential care do not require vitamin D testing before or during treatment.[48] For active, community-dwelling New Zealanders with regular sunlight exposure, no testing of vitamin D should be undertaken, and vitamin D supplements are not necessary. With the substantial increase in requests for vitamin D tests over the last decade, in the absence of a clear rationale for the increase in the requirement for these tests, it is likely that voluntary restrictions and education campaigns may be insufficient to halt the increase in vitamin D test requests.

The approach of enforced restriction of vitamin D tests has already occurred elsewhere- for example in several provinces in Canada where the tests are no longer publicly funded. One potential advantage of such restrictions is that the number of requests for 25OHD measurement would be likely reduced so that one or two sites in New Zealand with the most accurate assay (LC-MS/MS) could carry out all testing.

Conclusions

There are considerable uncertainties regarding diagnosis and definition of vitamin D insufficiency, and an absence of rigorous evidence that vitamin D supplementation improves health in vitamin D-insufficient populations. Several large RCTs are underway and hopefully will provide strong evidence of benefits or otherwise of vitamin D supplements.

Unless adequately powered RCTs do provide evidence of health improvement, clinicians should not routinely measure vitamin D or routinely prescribe vitamin D supplements in low-risk populations. However, routine treatment of individuals at high risk of vitamin D deficiency (frail elderly, deeply pigmented, veiled) is reasonable without measurement of 25OHD. Measurement of vitamin D is costly, inaccurate and imprecise, and the majority of tests do not reveal vitamin D deficiency. Therefore, vitamin D testing should be limited to the investigation of suspected metabolic bone disease and hypocalcaemia.

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References:

1. Lips P. Vitamin D deficiency and secondary hyperparathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. Endocr Rev. 2001;22:477-501.
2. Dawson-Hughes B, Heaney RP, Holick MF, et al. Estimates of optimal vitamin D status. Osteoporos Int. 2005;16:713-6.

3. Pearce SH, Cheetham TD. Diagnosis and management of vitamin D deficiency. BMJ. 2010;340:b5664.
4. Rockell JE, Skeaff CM, Williams SM, Green TJ. Serum 25-hydroxyvitamin D concentrations of New Zealanders aged 15 years and older. Osteoporos Int. 2006;17:1382-9.
5. IOM (Institute of Medicine). Dietary Reference Intakes for Calcium and Vitamin D. Washington, DC: The National Academies Press; 2011.
6. Bolland MJ, Grey AB, Ames RW, et al. Determinants of vitamin D status in older men living in a subtropical climate. Osteoporos Int. 2006;17:1742-8.
7. Lucas JA, Bolland MJ, Grey AB, et al. Determinants of vitamin D status in older women living in a subtropical climate. Osteoporos Int. 2005;16:1641-8.
8. Bolland MJ, Grey AB, Ames RW, et al. The effects of seasonal variation of 25-hydroxyvitamin D and fat mass on a diagnosis of vitamin D sufficiency. Am J Clin Nutr. 2007;86:959-64.
9. Binkley N, Krueger D, Gemar D, Drezner MK. Correlation among 25-hydroxy-vitamin D assays. J Clin Endocrinol Metab. 2008;93:1804-8.
10. Roth HJ, Schmidt-Gayk H, Weber H, Niederau C. Accuracy and clinical implications of seven 25-hydroxyvitamin D methods compared with liquid chromatography-tandem mass spectrometry as a reference. Ann Clin Biochem. 2008;45:153-9.
11. Carter GD. 25-Hydroxyvitamin D assays: the quest for accuracy. Clin Chem. 2009;55:1300-2.
12. Grey A, Bolland M. Vitamin D: a place in the sun? Arch Intern Med. 2010;170:1099-100.
13. Bolland MJ, Bacon CJ, Horne AM, et al. Vitamin D insufficiency and health outcomes over 5 y in older women. Am J Clin Nutr. 2010;91:82-9.
14. Christenfeld NJ, Sloan RP, Carroll D, Greenland S. Risk factors, confounding, and the illusion of statistical control. Psychosom Med. 2004;66:868-75.
15. Nicholl J. Case-mix adjustment in non-randomised observational evaluations: the constant risk fallacy. J Epidemiol Community Health. 2007;61:1010-3.
16. Gerdhem P, Ringsberg KA, Obrant KJ, Akesson K. Association between 25-hydroxy vitamin D levels, physical activity, muscle strength and fractures in the prospective population-based OPRA Study of Elderly Women. Osteoporos Int. 2005;16:1425-31.
17. Cauley JA, Lacroix AZ, Wu L, et al. Serum 25-hydroxyvitamin D concentrations and risk for hip fractures. Ann Intern Med. 2008;149:242-50.
18. Looker AC, Mussolino ME. Serum 25-hydroxyvitamin D and hip fracture risk in older U.S. white adults. J Bone Miner Res. 2008;23:143-50.
19. Cauley JA, Parimi N, Ensrud KE, et al. Serum 25-hydroxyvitamin D and the risk of hip and nonspine fractures in older men. J Bone Miner Res. 2010;25:545-53.
20. Cummings SR, Browner WS, Bauer D, et al. Endogenous hormones and the risk of hip and vertebral fractures among older women. Study of Osteoporotic Fractures Research Group. N Engl J Med. 1998;339:733-8.
21. Jackson RD, LaCroix AZ, Gass M, et al. Calcium plus vitamin D supplementation and the risk of fractures. N Engl J Med. 2006;354:669-83.
22. Garnero P, Munoz F, Sornay-Rendu E, Delmas PD. Associations of vitamin D status with bone mineral density, bone turnover, bone loss and fracture risk in healthy postmenopausal women. The OFELY study. Bone. 2007;40:716-22.
23. Lappe JM, Travers-Gustafson D, Davies KM, et al. Vitamin D and calcium supplementation reduces cancer risk: results of a randomized trial. Am J Clin Nutr. 2007;85:1586-91.
24. Tang BM, Eslick GD, Nowson C, et al. Use of calcium or calcium in combination with vitamin D supplementation to prevent fractures and bone loss in people aged 50 years and older: a meta-analysis. Lancet. 2007;370:657-66.
25. Autier P, Gandini S. Vitamin D Supplementation and Total Mortality: A Meta-analysis of Randomized Controlled Trials. Arch Intern Med. 2007;167:1730-7.

26. Avenell A, Gillespie WJ, Gillespie LD, O'Connell D. Vitamin D and vitamin D analogues for preventing fractures associated with involutional and post-menopausal osteoporosis. Cochrane Database Syst Rev. 2009:CD000227.
27. Elamin MB, Abu Elnour NO, Elamin KB, et al. Vitamin D and cardiovascular outcomes: a systematic review and meta-analysis. J Clin Endocrinol Metab. 2011;96:1931-42.
28. Chapuy MC, Arlot ME, Duboeuf F, et al. Vitamin D3 and calcium to prevent hip fractures in the elderly women. N Engl J Med. 1992;327:1637-42.
29. Chapuy MC, Pamphile R, Paris E, et al. Combined calcium and vitamin D3 supplementation in elderly women: confirmation of reversal of secondary hyperparathyroidism and hip fracture risk: the Decalyos II study. Osteoporos Int. 2002;13:257-64.
30. Wang L, Manson JE, Song Y, Sesso HD. Systematic review: Vitamin D and calcium supplementation in prevention of cardiovascular events. Ann Intern Med. 2010;152:315-23.
31. Shapses SA, Manson JE. Vitamin D and prevention of cardiovascular disease and diabetes: why the evidence falls short. JAMA. 2011;305:2565-6.
32. Manson JE, Mayne ST, Clinton SK. Vitamin D and prevention of cancer--ready for prime time? N Engl J Med. 2011;364:1385-7.
33. Sneve M, Figenschau Y, Jorde R. Supplementation with cholecalciferol does not result in weight reduction in overweight and obese subjects. Eur J Endocrinol. 2008;159:675-84.
34. Li-Ng M, Aloia JF, Pollack S, et al. A randomized controlled trial of vitamin D3 supplementation for the prevention of symptomatic upper respiratory tract infections. Epidemiol Infect. 2009:1-9.
35. Urashima M, Segawa T, Okazaki M, et al. Randomized trial of vitamin D supplementation to prevent seasonal influenza A in schoolchildren. Am J Clin Nutr. 2010;91:1255-60.
36. Bolland MJ, Grey A, Reid IR. Time for a moratorium on vitamin D meta-analyses? BMJ. 2009;339:b4394.
37. Sanders KM, Stuart AL, Williamson EJ, et al. Annual high-dose oral vitamin D and falls and fractures in older women: a randomized controlled trial. JAMA. 2010;303:1815-22.
38. Bischoff-Ferrari HA, Dawson-Hughes B, Platz A, et al. Effect of high-dosage cholecalciferol and extended physiotherapy on complications after hip fracture: a randomized controlled trial. Arch Intern Med. 2010;170:813-20.
39. Melamed ML, Michos ED, Post W, Astor B. 25-hydroxyvitamin D levels and the risk of mortality in the general population. Arch Intern Med. 2008;168:1629-37.
40. Michaelsson K, Baron JA, Snellman G, et al. Plasma vitamin D and mortality in older men: a community-based prospective cohort study. Am J Clin Nutr. 2010;92:841-8.
41. Omenn GS, Goodman GE, Thornquist MD, et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. N Engl J Med. 1996;334:1150-5.
42. Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/progestin Replacement Study (HERS) Research Group. JAMA. 1998;280:605-13.
43. Rossouw JE, Anderson GL, Prentice RL, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results From the Women's Health Initiative randomized controlled trial. JAMA. 2002;288:321-33.
44. Yusuf S, Dagenais G, Pogue J, et al. Vitamin E supplementation and cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. N Engl J Med. 2000;342:154-60.
45. Bonaa KH, Njolstad I, Ueland PM, et al. Homocysteine lowering and cardiovascular events after acute myocardial infarction. N Engl J Med. 2006;354:1578-88.
46. Lonn E, Yusuf S, Arnold MJ, et al. Homocysteine lowering with folic acid and B vitamins in vascular disease. N Engl J Med. 2006;354:1567-77.
47. Bolland MJ, Chiu WW, Davidson JS, et al. The effects of seasonal variation of 25-hydroxyvitamin D on diagnosis of vitamin D insufficiency. N Z Med J. 2008;121:63-74.
48. ACC. Vitamin D Prescribing Criteria. ACC 4697. 2008.

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A case of yellow fever vaccine-associated disease

Heather Isenman, Andrew Burns

Yellow fever is a mosquito-borne flavi-virus endemic and epidemic in South America, and Sub-Saharan Africa.[1] Wild type disease incidence is between 100 to 1200 cases per annum worldwide,[2] with mortality 20–50%. Whilst South America has instituted mass immunisation campaigns, the highest incidence is reported in Africa, where vaccination coverage is poor.

All vaccines currently in use are a live attenuated form of the virus prepared from the 17D strain of the virus, which induces seroconversion in over 95% of recipients.[3] It is contraindicated in the immunocompromised, infants under 6 months, and those with known egg allergy. It is not contraindicated in pregnant or lactating women, but caution should be exercised.

Given the severity of disease, certain endemic countries mandate yellow fever vaccination as a condition of entry. Current CDC guidelines for travellers recommend vaccination in at risk travellers and in those requiring entry into a country with mandatory vaccination[4]. Since vaccination was introduced, there have been only 10 recorded cases of naturally acquired yellow fever recorded in travellers.[4]

Vaccine-derived yellow fever is a multisystem illness that was first described in 2001.[5] There are two syndromes: vaccine associated neurotropic (YEL-AND) and viscerotropic (YEL-AVD) disease.[5,6] To date, there are 57 described cases, most commonly described in older age groups[5] and those with underlying immunocompromise.

YEL-AVD appears to occur after the first dose of yellow fever vaccine, rather than with booster doses. The onset of YEL-AVD is 1–8 days after vaccination and reported case-fatality ratio is 65%. The incidence of YEL-AVD in the United States is 0.4 cases per 100,000 doses of vaccine administered. The rate is highest in those older than 60 years, with a rate of 1.0 per 100,000 doses in people aged 60–69 years and 2.3 per 100,000 doses in those older.[7]

Here we describe a case of YEL-AVD, with discussion of guidelines and cautions for vaccination in light of the risks of vaccine-derived disease.

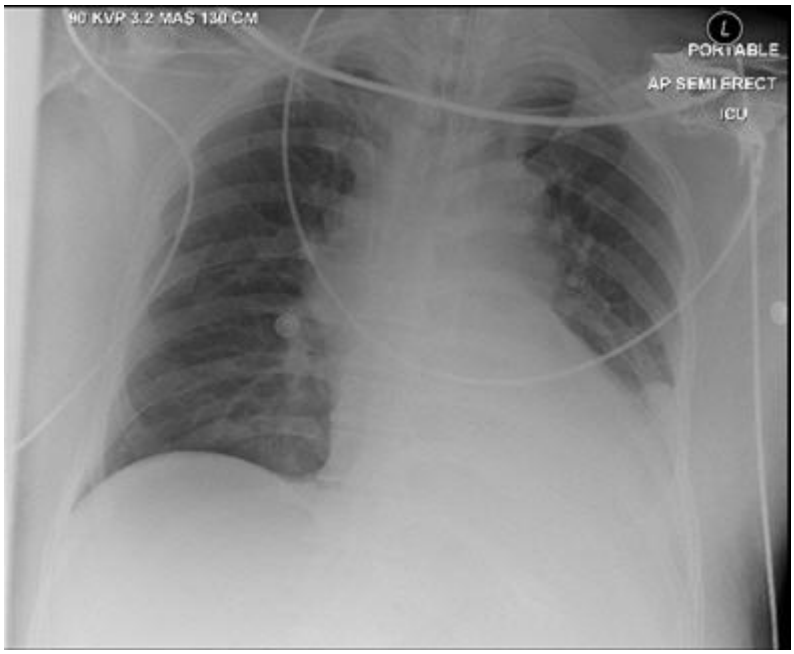
Case report

A 66-year-old retired male administrative worker, presented to hospital with a 1-day history of diarrhoea, fevers and epistaxis . Seven days prior he received a yellow fever vaccination in anticipation of a trip to South America.

Initial examination revealed a diaphoretic, man, with a BP of 90/50 mmHg, HR 130, atrial fibrillation, temperature 38.5°C and oxygen saturations of 98%.

Chest auscultation revealed bibasal inspiratory crackles, and abdominal and neurological examination were unremarkable.

Figure 1. Chest X-ray on admission



Chest X-ray (CXR) demonstrated an opacity in the upper mediastinum. A computerised tomography (CT) scan of the thorax confirmed an 11 cm ×10 cm × 6 cm anterior mediastinal mass with associated hilar and mediastinal lymphadenopathy.

Admission laboratory testing showed a normal WCC, thrombocytopaenia (platelets 17×10^9L), hepatitis (ALT 214 U/L, ALP 58 U/L, bilirubin 38 umol/L), coagulopathy (INR 1.4, APTT 36 seconds), renal impairment (creatinine 117 mmol/L), and a CRP of 245 mg/L.

He was resuscitated with IV fluids and antibiotics. On day 2 of admission , he had persistent tachycardia , hypotension, low urine output and increasing oxygen requirements. Bleeding from phlebotomy sites and macroscopic haematuria were noted.

On day 3 he developed respiratory compromise. Bedside echocardiography demonstrated a small left ventricle with good systolic function. CXR suggested acute respiratory distress syndrome (ARDS), and renal failure ensued (creatinine 239 mmol/L).

He was transferred back to the High Dependency Unit (HDU) with multiorgan failure. He developed a DIC picture (APTT > 180, INR 1.6, fibrinogen 1.5). Despite maximal inotropic support and haemofiltration multiorgan failure progressed, and on day 5 support was withdrawn. He died shortly thereafter.

Blood, sputum and urine cultures were sterile and *Legionella*, leptospiral, hepatitis A, B and C serology negative. Serum tested positive for yellow fever IgM, and a blood sample (tested by CDC, Atlanta, USA) detected yellow fever viral RNA.

Scientists at the Institute of Environmental Science and Research (ESR) , New Zealand amplified by PCR the envelope protein of the patient's YF strain and confirmed a 100% match with the 17D vaccine strain.

Our patient had a multisystem illness with clinical and biochemical features mirroring those of wild type yellow fever virus infection. The illness was temporally related to his yellow fever vaccine, with positive serology and PCR for yellow fever, suggesting a causal link between vaccination and his fatal illness. A post mortem examination was not undertaken, but it is likely the mediastinal findings represented a thymic mass.

Discussion

In a previous case series of 24 cases of vaccine derived yellow fever, 4 had previous thymectomies or thymic disease.[8] The possibility of thymic disease coupled with advancing age put our case at higher risk of vaccine related disease—from current literature the risk in the over-60 age group is increased seven-fold[5].

Whilst yellow fever vaccine adverse events are extremely rare,[9] and the vaccine is highly effective at inducing immunity to an often fatal disease, we suggest that patients especially over 60 years of age should be adequately counselled prior to vaccination.

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Acknoweledgement: We thank Dr Richard Hall, research scientist at the Institute of Environmental Science and Research (ESR), New Zealand for viral molecular sequencing.

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References:

1. Barnett ED. Yellow fever: epidemiology and prevention. Clin Infect Dis. 2007 ;44:850-6.
2. Jentes ES, Poumerol G, Gershman MD, et al. The revised global yellow fever risk map and recommendations for vaccination, 2010: consensus of the Informal WHO Working Group on Geographic Risk for Yellow Fever. Lancet Infect Dis. 2011; 11: 622-32.
3. Monath TP, Nichols R, Archambault WT, et al. Comparative safety and immunogenicity of two yellow fever 17D vaccines (ARILVAX and YF-VAX) in a phase III multicenter, double-blind clinical trial. Am J Trop Med Hyg. 2002;66:533-41.
4. www.cdc.gov/travel (accessed on 06/06/2011) archived at http://www.webcitation.org/61hyhJ1AK
5. CDC 2010 Yellow Fever Vaccine, Recommendations of the Advisory Committee on Immunization Practices (ACIP), MMWR 59(RR07);1-27.
6. Martin M, Tsai TF, Cropp B et al. Fever and multisystem organ failure associated with 17D-204 yellow fever vaccination: a report of four cases. Lancet. 2001 14;358:98-104.
7. Khromava AY, Eidex RB, Weld LH, et al; Yellow Fever Vaccine Safety Working Group. Yellow fever vaccine: an updated assessment of advanced age as a risk factor for serious adverse events.Vaccine. 2005 9;23:3256-63.

8. Barwick R; Eidex for the Yellow Fever Vaccine Safety Working Group. History of thymoma and yellow fever vaccination. Lancet. 2004 11-17;364:936.
9. Thomas RE, Lorenzetti DL, Spragins W, et al. VActive and passive surveillance of yellow fever vaccine 17D or 17DD-associated serious adverse events: systematic review. Vaccine. 2011 Jun 20;29:4544-55.

THE NEW ZEALAND MEDICAL JOURNAL
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An unusual cause of carotid sinus hypersensitivity/syndrome

Donny Wong, Joey Yeoh

Syncope is extremely common, especially in the elderly population. Although neurocardiogenic (vasovagal) syncope accounts for ≥35% of cases, carotid sinus hypersensitivity (CSH) is a notable, albeit rarer, cause of reflex syncope.

We present an unusual case of CSH resulting from malignancy, representing a diagnostic and therapeutic challenge.

Case report

A 78-year-old gentleman presented acutely via ambulance following an unprovoked and unwitnessed cardinal episode of syncope whilst driving. He had no known medical conditions and was not on any regular medications. He was highly functioning and still worked as a freelance accountant. He had a 50-year history of daily pipe smoking. In retrospect, he reported a mild difficulty in swallowing and a 5-kg weight loss over the preceding 6 weeks.

Physical examination was unremarkable except for palpable cervical lymph nodes. Preliminary electrolytes, thyroid function, resting electrocardiography and echocardiography were all normal. He had no orthostatic hypotension. He experienced another unprovoked syncopal episode in hospital associated with bradycardia (and preceded by 6 second pause) and hypotension. He had a positive controlled carotid sinus massage (CSM) test.

He also had computed tomography imaging of his head and neck, which showed an invasive malignancy in the right tonsilllar fossa associated with significant necrotic lymphadenopathy affecting the neck bilaterally (Figures 1A and 1B). The nodes were directly anterior to, and encroaching on, the carotid sheath at multiple levels including the carotid sinus. There were also multiple pulmonary nodules but no other metastatic spread. Histology from a subsequent core biopsy confirmed squamous cell carcinoma.

A Medtronic dual chamber (VVI) pacemaker was implanted. Due to the extent of his malignancy (Stage T4a N2c M1), he was only amenable to palliative radiotherapy with 30 Gy administered in 10 fractions with small clinical improvement in his lymphadenopathy.

Despite significant reduction in syncope frequency, he still experiences rare events associated with hypotension but no bradycardia.

Figure 1A. Coronal view showing the relationship of necrotic cervical lymph nodes (white arrows) in relation to bilateral external and internal carotid arteries (black arrows)

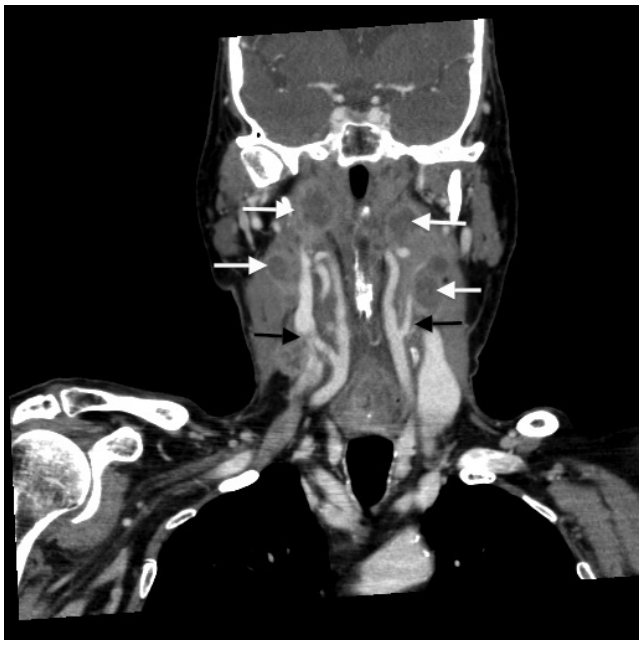
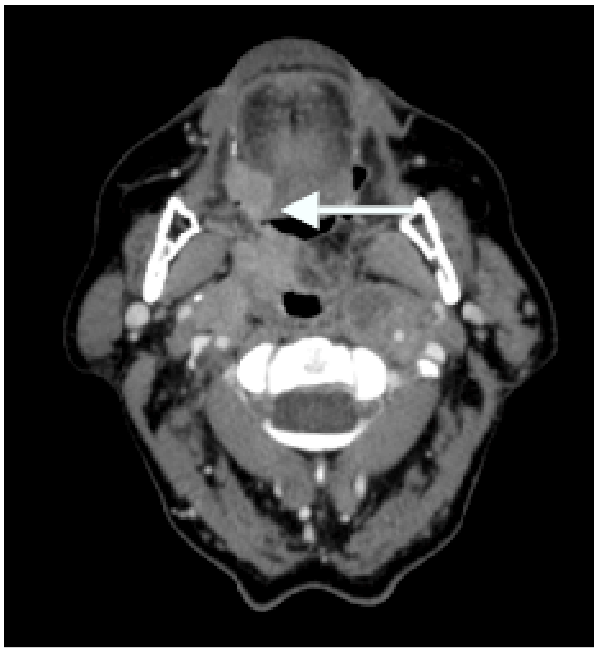


Figure 1B. Transverse view of the primary mass in the right tonsillar fossa (white arrow) and involvement of the palate



Discussion

CSH results from exaggerated responses to carotid sinus stimulation; cardioinhibitory resulting in bradycardia (70–75%), vasodepressor with reduction in vasomotor tone (5–10%), or a mixture of both (20–25%).[1,2] Regardless, they all culminate in reduction of cardiac output.[1,2]

CSH is more common in males and accounts for 1–5% of recurrent syncope (uncommon <50 years, incidence increases with age).[3] CSH from primary or metastatic malignancies (direct carotid sinus pressure) is uncommon.[4–6]

Diagnosis requires exclusion of all other causes of syncope in addition to a positive CSM test.[2-3] A pause (asystole) of >3 seconds (cardioinhibitory), systolic blood pressure drop of ≥50 mmHg (vasodepressor) or both (mixed) constitutes a positive test.[2,3] It is recommended that CSM is done in all patients >40 years presenting with recurrent syncope (and without absolute contraindications).[3]

Pacemaker implantation (DDD, DDI or VVI) is effective in reducing syncope frequency in cardioinhibitory and mixed CSH in most cases, but intuitively, not in vasodepressor mediated CSH.[7,8] The vasodepressor effect may well be difficult to manage though epinephrine has been trialled with limited success[4] Midodrine, fludrocortisone and sertraline were previously trialled by Sharma et al without any success.[6]

This case highlights the importance of high clinical suspicion and thorough clinical examination in the diagnostic workup of unexplained syncope, especially in the elderly, as not all cases are due to vasovagal or orthostatic causes. Additionally, this case highlights the fact that there is no curative therapy for CSH since syncopal episodes may still occur following pacemaker implantation. This is especially true in incurable malignancy-related CSH (especially with predominant vasodepressor response), representing an intractable and difficult therapeutic challenge.

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References:

1. Weiss S, Baker JP. The carotid sinus reflex in health and disease: its role in the causation of fainting and convulsions. Medicine 1933;12:297–354.
2. McIntosh SJ, Lawson J, Kenny RA. Clinical characteristics of vasodepressor, cardioinhibitory, and mixed carotid sinus syndrome in the elderly. Am J Med 1993;95:203–8.
3. Moya A, Sutton R et al. Guidelines for the diagnosis and management of syncope (version 2009). Eur Heart 2009: Nov; 30(21):2631–71.
4. Patel AK, Yap VU, Fields J, Thomsen JH. Carotid sinus syncope induced by malignant tumors in the neck. Emergence of vasodepressor manifestations following pacemaker therapy. Arch Intern Med. 1979 Nov;139(11):1281–4.
5. Campbell S, Walker D, Lanzon-Miller S, Gurr P. Nasal carcinoma--a rare indication for a permanent pacemaker. Clin Med. 2004 Mar-Apr;4(2):165–7.
6. Sharma J, Dougherty AH. Recurrent syncope in a cancer patient: a case report and review of the literature. Cardiol Res Pract. 2011 Apr 4; 2011:678237.
7. Claesson JE, Kristensson BE, Edvardsson N, Währborg P. Less syncope and milder symptoms in patients treated with pacing for induced cardioinhibitory carotid sinus syndrome: a randomized study. Europace. 2007 Oct;9(10):932-6. Epub 2007 Sep.
8. Sutton R, Brignole M, Menozzi C, et al. Dual-chamber pacing in the treatment of neurally mediated tilt-positive cardioinhibitory syncope: pacemaker versus no therapy: a multicenter randomized study. The Vasovagal Syncope International Study (VASIS) Investigators. Circulation. 2000 Jul 18;102(3):294–9.

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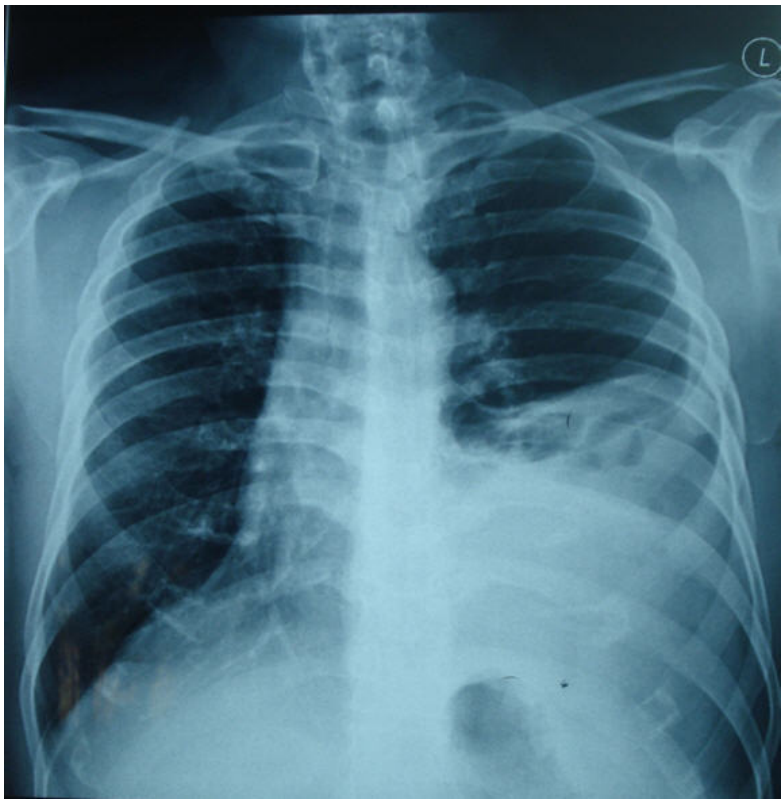
Antero-lateral diaphragmatic hernia presenting in sixth decade

Emmanuel Bhaskar, Karthik Vishnu, Krishnan Vasanthan, Mani Rajkumar

Clinical—A 52-year-old male presented to our outpatient department with complaints of abdominal distension for 6 months and breathlessness on exertion for 3 months. The symptoms minimally interfered with his daily activities . To the best of his memory there was no past or recent history of trauma. He never had a chest X-ray imaged in the past.

Although on examination he appeared comfortable he had dyspnoea after going up four flights of stairs. His vitals were stable. Respiratory system examination had features suggestive of left moderate pleural effusion. Examination of the abdomen was unremarkable. 'Chest X-ray PA view (Figure 1) showed homogenous opacity over the left lower zone with circumscribed internal air translucencies, a finding consistent with left lower lobe parenchymal pathology possibly cavities associated with para-pneumonic effusion.

Figure 1. Chest X-ray PA view with homogenous opacity over the left lower zone with circumscribed internal air translucencies



Since his primary symptom was abdominal distension, a CT-thorax with abdominal screening (Figures 2 and 3) was requested which showed a 4 cm × 4 cm diaphragmatic defect in the antero-lateral region and a 1 cm × 1 cm defect in the postero-lateral region of the left diaphragm with herniation of bowel loops into the thorax . The bowel loops could be visualised up to the level corresponding to tracheal bifurcation along with hypoplasia of left lower lobe (Figure 4).

Figure 2. Lung window of CT-thorax showing the larger antero-lateral defect and smaller postero-lateral defect in the left diaphragm

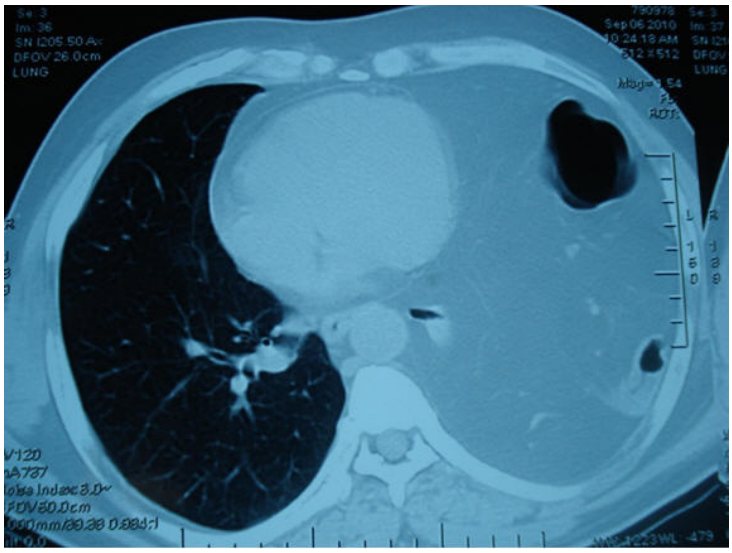


Figure 3. Lung window of CT-thorax showing the herniated bowel loops in the thorax

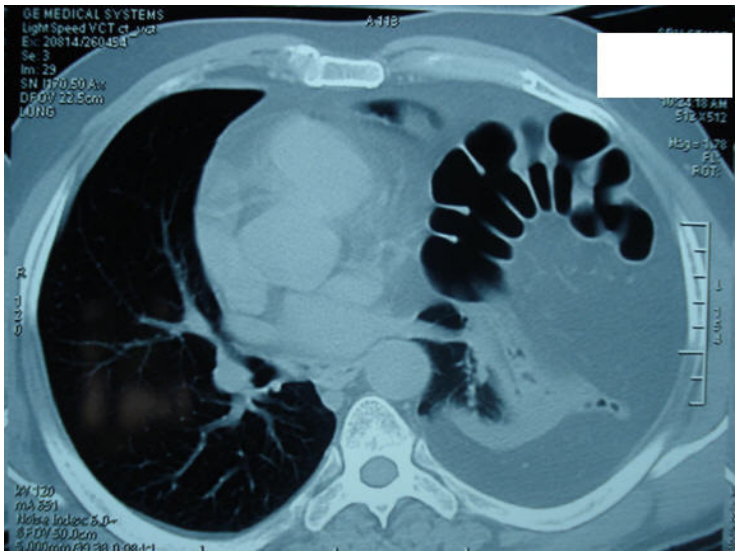
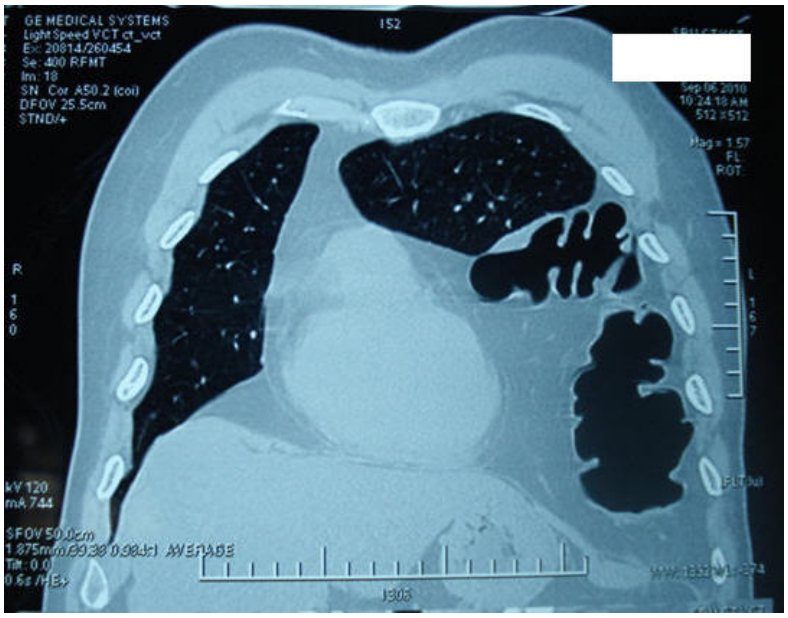


Figure 4. Reconstructed film of CT-thorax showing the extent of bowel herniation into thorax



He underwent surgical reduction of the abdominal contents which herniated through the anterolateral defect (no herniation were found through the posteriolateral defect) followed by closure of the anterolateral and posterolateral defect in the left diaphragm with a prolene mesh. Peritoneal covering observed over the diaphragmatic defect and lack of post-inflammatory features in the defect indicated the hernia to be of congenital nature.

Discussion—Diaphragmatic hernia may be congenital or acquired. The incidence of congenital diaphragmatic hernia (CDH) range from 1:2000 to 1:5000 live births.[1] Bochdalek hernia(defect in the postero-lateral part of the diaphragm) and Morgagni hernia (defect in anterior part of diaphragm close to midline) account for more than 90% of congenital diaphragmatic hernias.[2] Though CDH is often diagnosed in the antenatal or immediate postnatal period, about 10% of them can present later in life at an age from 32 days to 15 years.[2]

Reports of antero-lateral diaphragmatic hernia are rare.[3,4] The prolonged asymptomatic period in our patient could possibly be due to minimal herniation of bowel loops into thorax earlier in life with a recent increase in herniation making him symptomatic. The age of our patient, asymptomatic status for 5 decades, and antero-lateral herniation is unusual.

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References:

1. Adelman S, Benson CD. Bochdalek hernias in infant: Factors determining mortality. J Pediatr Surg 1976;11:569-573.
2. Mei-Zahav M, Solomon M, Trachsel D, Langer JC. Bochdalek diaphragmatic hernia: not only a neonatal disease. Arch Dis Child 2003;88:532–535.
3. Steiner Z, Mares AJ. Anterolateral diaphragmatic hernia: is it a Morgagni hernia? Eur J Pediatr Surg 1993;3:112-4.
4. Mouroux J, Pop D, Guiraudet P, et al. A new type of diaphragmatic hernia: anterolateral hernia. Thorac Cardiovasc Surg 2008;135:955-6.

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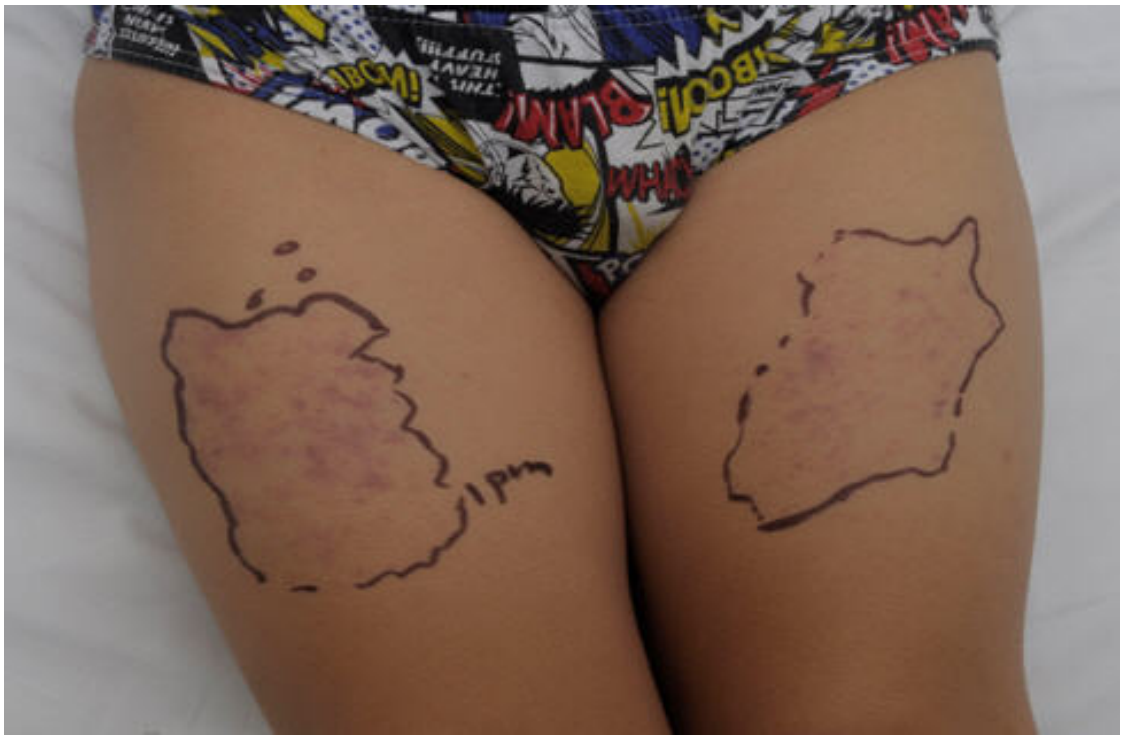
Bruising—unusual aetiology

Tilak de Almeida, Kewa Mascelle

An 8-year-old boy was referred to the Emergency Department with a history of bilateral bruising to the anterior aspect of each upper thigh. He was afebrile and well with no other bruises. After an initial blood work-up, the paediatrician was contacted.



What is the aetiology of the bruising?

Answer

The paediatrician noted the symmetrical and localised nature of the bruising and questioned the possibility of an injury to the front of legs. The boy promptly replied that he had been performing 'the haka' at home. The boy's mother confirmed he performs a haka daily in the shower when bathing. The paediatrician also elicited a history of excessive post-partum bleeding in mum and frequent nasal bleeding in a sibling.

Laboratory tests for coagulation defects including Von Willebrand disease and platelet function abnormalities were normal.

To our knowledge this is the first report of *haka-induced bruising* in a child.

Discussion

A *haka* is a traditional dance of challenge and welcome from the Māori people of New Zealand. Ka Mate ('the haka') is the most widely known haka because it is performed by the All Blacks (New Zealand's national rugby union team) before every game. It involves vigorous movements including stamping of the feet and beating of the thighs and chest.

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Initial experience with dabigatran etexilate at Auckland City Hospital

We write with reference to the inclusion of dabigatran etexilate (Pradaxa)—a pro-drug of dabigatran—on the pharmaceutical schedule in New Zealand without restriction on 1 July 2011.[1]

Dabigatran is a reversible thrombin inhibitor licensed for the use of stroke prevention in atrial fibrillation (AF) granted on the basis of data from the RE-LY study[2] and is an alternative to anticoagulation with warfarin. Due to its novelty, the place of dabigatran in the management of AF is not fully established and, importantly, there are no specific antagonists to dabigatran-induced bleeding.

Warnings at the end of 2011 from the Therapeutic Goods Administration,[3] Food and Drugs Administration,[4] and Medicines and Healthcare products Regulatory Agency[5] all highlight at risk patients of drug-induced bleeding (patients >75 years and those with renal impairment). Patients given drugs in everyday practice may differ from the demographic of patients recruited for drug trials upon which licensing decisions are based.

We sought to determine whether the initial cohort of patients admitted to Auckland City Hospital (ACH) on dabigatran, for 6 months July to December, 2011, mirrored the demographic of the RE-LY study participants.

The notes of all patients admitted through the Adult Emergency Department and the Admission and Planning Unit of ACH while taking dabigatran were reviewed and their details recorded for the six month time frame. The information was recorded irrespective as to whether the dabigatran was the cause of admission or just an association.

In all cases patient demographics were recorded (that were available) in an anonymised fashion, including age; weight (kg); renal function (creatinine clearance calculated by Cockroft-Gault formula, CrCl, ml/min); indication for dabigatran use and dosage used; presence of haemorrhage; and concomitant drug use. Data are presented as mean values ± standard error and shown against the RE-LY figures.

Seventy nine patients (33 female) taking dabigatran were admitted through AED/APU at Auckland City Hospital between July and December 2011. All patients had AF as the indication for dabigatran therapy. The mean age of patients was 76 ± 1.7 (range: 19–93, median 84) years; weight 77 ± 2.3 (range: 38-126, median 73) kg; CrCl 67 ± 5.9 (range: 12-137, median 61) ml/min. The mean RE-LY equivalents are: age 71 years; weight 82.5 kg; CrCl not stipulated in article (patients with CrCl <30 ml/min were excluded from the trial).

The ACH patients were on a mean 6.0 ± 0.8 other medications in addition to their dabigatran. Seven of the 79 patients were on an antiplatelet agent (aspirin or clopidogrel) in addition to dabigatran and one patient was on warfarin plus dabigatran plus aspirin. Sixteen patients were admitted with dabigatran-induced bleeding (11

gastrointestinal bleed; 4 haematuria; one haematoma). Three patients had their emergency surgery delayed (2 fractures; one ischaemic limb) because of inability to reverse dabigatran-bleeding.

These initial data show that dabigatran is being prescribed to patients who differ from those enrolled in the original trial. This may have consequences for drug pharmacokinetics and the incidence of adverse effects, including bleeding the management of which remains uncertain.

Dabigatran is predominantly excreted renally and patients with renal impairment are at risk of enhanced bleeding.[6] Patients with CrCl < 30 were excluded from the RE-LY study (six of our patients had this degree of impairment) and recent warnings[3,5] suggest that the drug should be used with caution if CrCl is between 30-50 ml/min (22 of our patients). Our patients were generally less heavy than those in RE-LY. The effect of weight on dabigatran kinetics is not known.[7]

Dabigatran is not metabolised by the cytochrome P450 system but is excreted by p-glycoproteins and inhibitors of this system (e.g. amiodarone) can increase its bioavailability.[7] Dabigatran was coprescribed with a mean 6 other medications and future experience will inform us of important drug interactions. Seven of our patients were prescribed an antiplatelet agent in addition to dabigatran. Such concurrent use markedly enhances bleeding tendency in an expected pharmacodynamic manner[8] without good evidence of enhanced patient outcomes.

In conclusion, we have found that in its first 6 months of use, in the catchment area of ACH, dabigatran has been used in a group of patients that differs from those recruited in studies. Our patients were older, of lower weight and with a lower CrCl than the RE-LY-study patients. Dabigatran was also used in patients on multiple other drugs, including antiplatelet agents, and clinicians should be vigilant for potential drug interactions (whether pharmacokinetic or pharmacodynamic) and report any concerns to the Centre for Adverse Reactions Monitoring. Dabigatran use resulted in the delay of emergency surgery in three patients.

While it may not be surprising that drugs are used outside of their evidence-base, we urge caution in the future prescribing of this novel anticoagulant (that has no antagonist or means of monitoring) in patient groups that may be at enhanced risk of drug-induced haemorrhage.

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References:

1. PHARMAC. Dabigatran notification letter; 2011. http://www.pharmac.govt.nz/2011/06/13/Dabigatran%20notification%20letter.pdf
2. Connolly SJ, Ezekowitz MD, Yusuf S, et al. Dabigatran versus warfarin in patients with atrial fibrillation. N Engl J Med. 2009;361:1139-51.
3. Dabigatran (Pradaxa): risk of bleeding relating to use. http://www.tga.gov.au/safety/alerts-medicine-dabigatran-111103.htm
4. Pradaxa (dabigatran etexilate mesylate): Drug Safety Communication - Safety Review of Post-Market Reports of Serious Bleeding Events. http://www.fda.gov/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/ucm282820.htm
5. Dabigatran (Pradaxa®): risk of serious haemorrhage—need for renal function testing. http://www.mhra.gov.uk/Safetyinformation/DrugSafetyUpdate/CON137771
6. Douketis JD. Pharmacologic properties of the new oral anticoagulants: A clinician-oriented review with a focus on perioperative management. Current Pharmaceutical Design. 2010;16:3436-3441.
7. Stangier J, Clemens A. Pharmacology, pharmacokinetics, and pharmacodynamics of dabigatrane etexilate, an oral direct thrombin inhibitor. Clin Appl Thromb Hemost. 2009;15(1S):9S-16S.
8. PHARMAC. Dabigatran – NZ datasheet; 2011. http://www.pharmac.govt.nz/2011/07/04/New%20Zealand%20Datasheet.pdf

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Support for Associate Professor Barendregt's stance against PSA testing of asymptomatic men

We strongly support Associate Professor Barendregt's suggestion of a more evidence-based approach to PSA screening in New Zealand (i.e. the use of PSA testing in asymptomatic men).[1]

Screen-detected disease is often different to clinical disease: the simplistic idea that early detection by screening must save lives has been shown repeatedly to be illusory. The putative benefits of any screening test must be weighed against the serious harms screening can cause. The continued selective use of a minority of results from the six randomised studies of PSA testing, in order to prop up a position on the value of PSA testing in asymptomatic men by its advocates,[2] is inappropriate.

Lamb et al[2] also refer to the Health Select Committee report for guidance in recommending what general practitioners should do in the care of their patients. This elevates the judgement of members of a parliamentary select committee to specialist medical opinion, which would be a novel approach for medical practice in New Zealand.

Systematic evidence-based approaches continue to be unsupportive of PSA testing for asymptomatic men due to both the conflicting evidence regarding reduced prostate cancer mortality and the considerable iatrogenic illness caused from overdiagnosis and, therefore, overtreatment.

Such overtreatment and its side effects represent a considerable waste of healthcare resources. Until results from additional well-conducted studies are forthcoming, more analysis of the available studies is most unlikely to resolve the issue.

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References:

1. Barendregt JJ, Screening for prostate cancer is not recommended [letter]. N Z Med J. 2011;124(1346):99-100. http://journal.nzma.org.nz:8080/journal/124-1346/4983/content.pdf
2. Lamb DS, Delahunt B, Nacey JN. Revised status of PSA testing in the early detection and treatment of prostate cancer [editorial]. N Z Med J. 2011;124(1345):13-5. http://journal.nzma.org.nz:8080/journal/124-1345/4933/content.pdf

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More on BPAC

Since the New Zealand Medical Association has no opinion about the proposals about to be thrust upon the doctors with a "general" registration by the Medical Council, I crave space in order to take a closer look at them. We can feel certain that the proposals are unnecessary, and very expensive in terms of both time and money. Who, then, is behind them? Well, a thing called bpac^{nz}, for one.

"Welcome to bpac^{nz}. We provide evidence-based, educational material for primary healthcare professionals in New Zealand."

The bpac^{nz} site reveals that this "not-for-profit" organisation, already distributing materials to doctors, has five shareholders, who, one charitably presumes, have no interest in making money, but who may indeed be spending it. It is all a bit strange. The shareholders are:

- Procare Health
- South Link Health
- General Practice NZ (GPNZ)
- Pegasus Health Limited. (the word "limited" stands for "limited liability," and is used by all bodies anxious to protect themselves if they go broke.)
- University of Otago.

The University of Otago is in Dunedin. Dunedin is a small town, but there is plenty of room there for academics, and we quickly find that the University has a Department of General Practice and Rural Health. This Department has a Head of Department; it has academic and research staff numbering twenty, and they include two Professors, and two Associate Professors. There are nine other people described as Support Staff, and four Research Postgraduate students. The website provides no financial information, but it offers spadefuls of higher education.

Turning to Wellington, we can uncover "Our People in the Department of Primary Health Care and General Practice." Listed are 29 people with miscellaneous tasks. Auckland is, naturally, bigger still. It boasts a PhD student studying sleep.

The involvement in bpac^{nz} of a body entitled General Practice NZ led me to another body with a cluttered website. It seems to have rounded up all the GPs, but the funding of General Practice NZ is not divulged. That visit led on to the Royal New Zealand College of General Practitioners, which is loosely affiliated to it. From its inception, the College has wanted to get all the GPs on board. It now claims to have 95%, but that figure cannot possibly include all doctors who work as locums. It has grown into a big business, rumoured to be insolvent. Right from the start, it fished for privileges for its members, and, when these were denied, it cast a wider net. Years ago, the Professors of General Practice did not have a particularly easy time, but now it is harder for dissenters to argue with the numbers that their publicly-funded

departments control, (shown above), and with the links that the academics are shrewdly forging with the issuers of the licenses.

My research left me in no doubt on one thing. It is time for an audit. We have to be told just how much it now costs to become a GP, and just how much of that sum the government puts up one way or another, and who collects what once you've arrived. What are the ongoing, endless, and increasing costs of being a general practitioner? Once you have spent the day surfing the net, as I have, and caught a glimpse of the people who are fleecing the doctors, the patients, and the taxpayers in one way or another, you begin to feel like a cow that has wandered into a swamp in the middle of an information blackout, and who fears that it will never get out.

Milton Friedman pointed out in 1979 that, "as government has come to play a larger role in medicine, and to finance a larger share of medical costs, the power of the American Medical Association has declined." It has happened here, too, and we can now begin to understand what happens when the power to license the doctors shifts, unopposed, to several expanding bureaucracies. They never see the poorer patients scuttling past the medical centres on their way to the Casualty Departments.

Roger M Ridley-Smith
Retired GP
Wellington

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Greater Auckland Integrated Health Network (GAIHN) response to Tim Parke's 'Diversion of emergency acute workload to primary care: attractive private sector alternative to public hospital emergency departments?'

Tim Parke[1] refers to the need for a "real focus on truly determining "right patient, right place, right time" in the provision of acute services. The successful roll out of the GAIHN Community based management of Ambulance transported patients' project is a step towards achieving of this goal and gives a broader range of options to better deal with acute events in the community.

The major goal of the GAIHN programme is to achieve a reduction of avoidable hospitalisations.

GAIHN in conjunction with the Auckland Regional Primary Options for Acute Care (POAC) Service has been working closely with the four Auckland Emergency Departments (ED), St John and Accident & Medical (A&M) facilities to establish a new pathway for patients and make community based care more accessible.

Although this is not a new process for St John, charges to patients have in the past meant patients, who could otherwise have been safely and efficiently treated in a primary care setting, have chosen to go to a hospital emergency department. The POAC service will fund the cost of treatment in a community setting.

In order to ensure that potential pitfalls are minimised and to really focus on "right patient, right place, right time", the Auckland region (including St John, the EDs and the A&M Facilities) has agreed a set of clinical guidelines for the paramedics to work with.

In addition, local agreements under a Memorandum of Understanding (MoU) with the participating A&M Facilities ensure that good communication and re-engagement with the patients Medical Home is prioritised in order to facilitate better ongoing management of their health needs. Stage 2 of this project, anticipated to take place in March, includes transporting patients to their medical home, further strengthening the appropriate primary care relationships.

The new arrangements are being monitored closely to ensure that only appropriate patients are utilising this option.

Clinical leadership, collaboration and engagement across services and professional groups have been key in the success and roll out of this project. However, as noted by Tim Parke, this is only one of a series of initiatives necessary to address the unsustainable rising demand in acute admissions to hospital.

Reporting have indicated that the 'GAIHN Community based management of Ambulance transported patients project' in the first four weeks transported more than 160 patients to A&Ms.

Background information on the project and the GAIHN work programme is located in the 'Work Programme' section of the GAIHN website www.gaihn.health.nz

Campbell Brebner
GAIHN Clinical Lead
Penrose, Auckland

Reference:

1. Parke T. Diversion of emergency acute workload to primary care: an attractive private sector alternative to public hospital emergency departments? N Z Med J. 16 December 2011;124(1347):6-9. http://journal.nzma.org.nz/journal/124-1347/5002/content.pdf

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Top selling new cars in New Zealand: recent trends in air pollution and greenhouse gas ratings

Car emissions have significant implications for public health in terms of local air pollution but also in terms of greenhouse gas emissions (a major threat to international health). Car fuel efficiency also has implications for domestic living costs and energy security at a national level. Here we add new data to previous work in 2005 to describe recent trends in the New Zealand setting.

Methods—We obtained a list of the top 10 selling new car *models* in 2011 for New Zealand (personal communication with Perry Kerr, Chief Executive Officer of the Motor Industry Association, 2012) and for the United Kingdom.[1] Data on fuel efficiency, greenhouse ratings and pollution ratings were obtained from the same official Australian Government website (Green Vehicle Guide: http://www.greenvehicleguide.gov.au/GVGPublicUI/home.aspx) used in a previous study[2] in which one of us (NW) was involved. To maximise comparability with the previous study, we decided against using a similar website that has since become available for the New Zealand setting (http://rightcar.govt.nz). Neither database differentiates between model variants (e.g., the Holden Commodore is available in 3.0L and 6.0L engine size variants), so the variant with the median fuel economy was used in the analysis. Three of the top 10 selling models in the United Kingdom were missing from the Australian database, and were omitted from the analysis.

Results & Discussion—The top 10 selling new car models for New Zealand between the years 2005 and 2011 improved from a health perspective with respect to: (i) greenhouse ratings (based on CO_2 emissions), from 5.4/10 to 6.2/10; (ii) air pollution ratings, from 5.6/10 to 6.8/10; and (iii) fuel efficiency, from 10.0L/100km to 8.5L/100km. Mean engine size decreased from 2.80L to 2.49L (see Table 1).

These trends may be partly explained by impacts of the global economic crisis in New Zealand along with higher fuel prices (with the petrol price increasing in New Zealand from ~$1.50/L in 2005 to $2.06/L in December 2011). Other contributing factors may be: (i) increased awareness by car buyers of issues around climate change and air pollution; (ii) changing demographics with smaller families and households (favouring smaller cars); (iii) growing parking constraints in cities (possibly favouring smaller cars); and (iv) and fashions relating to car size.

But despite these improvements, the top 10 selling new car models in 2011 for New Zealand were somewhat behind (in efficiency and pollution rankings) compared to the equivalent models for the United Kingdom (see Table 1). Similarly, when compared to the winner of the "2010 AA Energywise Rally" held in New Zealand (see: http://uat.aa.co.nz/about/events/AAenergywiserally/Pages/default.aspx). To accelerate progress towards low pollution levels and fuel efficiency, the New Zealand Government could consider following the lead of other countries to have tighter fuel efficiency standards for vehicles and to adopt standards on CO_2 emission levels (such as those forthcoming in the European Union[3] and the United States[4]). Higher

registration fees or taxes on inefficient vehicles with larger engines are another approach.

Table 1. Mean performance and pollution ratings of the top selling cars in New Zealand and comparisons over time and with selected other cars

Car performance features*	Advertised cars (NZ magazines 2001-2005)[2]	Top 10 selling cars in NZ in 2005[2]	Top 10 selling cars in NZ in 2011	Top selling cars in UK in 2011†	AA Energywise Rally Winner (2010)
Mean CO_2 produced (per km travelled)	–	–	202.4	156.7	109.0
Mean "Greenhouse ratings" (score of 10 is "best")	5.3/10	5.4/10	6.2/10	7.4/10	8.5/10
Mean "Air pollution ratings" (score of 10 is "best")	5.4/10	5.6/10	6.8/10	7.1/10	8.5/10
Mean engine size (L)	2.98	2.80	2.49	1.73	1.30
Mean fuel efficiency‡ (L/100 km)	9.8	10.0	8.5	6.7	4.6

Notes: See the full dataset at: http://www.scribd.com/doc/79802639/Car-Efficiency-Data-for-NZ-and-UK-2011

* Ratings were based on those in an Australian Government website "the Green Vehicle Guide" (http://www.greenvehicleguide.gov.au/GVGPublicUI/home.aspx).

† Three of the top 10 selling models in the United Kingdom were missing from the Australian database, and were omitted from the analysis present here.

‡ Using the "combined" consumption of urban and extra-urban driving (see calculation details at: http://www.fuelsaver.govt.nz/explain.html).

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References:

1. The Telegraph. Top 10 best selling cars of 2011, 2012. http://www.telegraph.co.uk/motoring/picturegalleries/8997492/Top-10-best-selling-cars-of-2011.html
2. Wilson N, Maher A, Thomson G, Keall M. Vehicle emissions and consumer information in car advertisements. Environ Health 2008;7:14.
3. EUROPA. Euro 5 and Euro 6 standards: reduction of pollutant emissions from light vehicles, 2010. http://europa.eu/legislation_summaries/environment/air_pollution/l28186_en.htm
4. U.S. Environmental Protection Agency. EPA and NHTSA Finalize Historic National Program to Reduce Greenhouse Gases and Improve Fuel Economy for Cars and Trucks. US EPA, 2010. http://www.epa.gov/otaq/climate/regulations/420f10014.htm

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

On the cases of plague admitted to the Auckland Hospital: from March 20th to May 3rd, 1911

Excerpt from an article by J. Campbell Macdiarmid, M.B., Ch.B. (Edin.) and J. Howard Lawry, M.B., Ch.B. (Edin.) and published in NZMJ 1911Aug;10(39):1–19.

The first case of plague of the series was admitted to the Auckland Hospital on the 20th March, 1911; it was that of a female, and of the severe bubonic type. The second case, that of a man, the husband of the preceding, was of a mild type in which the bubo did not suppurate. The third case likewise was a male, and worked in other two persons' shop.

On admission he showed no glandular enlargement, but four days afterwards he developed definite signs of pneumonic plague, and died early on the morning of the 29th. On the following day a lad of 19, who did not live in the same district as the above, was admitted to the Hospital as a case of Typhoid, but a careful examination revealed the fact that he was suffering from typical bubonic plague, there being a large bubo in the left groin. His case was not of oo severe a type.

We must now refer to the case of the nurse who contracted pneumonic plague from the man who died of it. Her recovery was as remarkable as it was unexpected, when one considers the gravity of her condition, complicated as it was, and the high mortality of the disease.

This was the last case of plague until May 3rd, when two more bubonic cases were admitted. In regard to the first four cases, we may note that they had lived in places where a high mortality amongst rats had been noticed.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Symptoms of myocardial ischaemia—any differences between men and women?

Previous studies have suggested that there are age differences in the syptomatology of myocardial ischaemia. If this were true, failure to efficiently recognise ischaemic symptoms in women could partly explain delays in presentation, diagnosis, and lower rate of treatment to guidelines observed in women.

The researchers studied 305 patients (39.7% female) undergoing elective non-urgent percutaneous coronary intervention (PCI). Transient coronary occlusion during PCI was correlated with the subjects' symptoms. They report no statistically significant differences in women's and men's rates of chest and other typical symptoms during ischaemia, although women were more likely to experience throat and jaw discomfort.

European Heart Journal 2011;32:3107–14.

Advanced chronic kidney disease, cardiovascular events and the effects of diabetes

The authors of this report point out that the incidence of cardiovascular (CV) events in patients with end-stage kidney disease is significantly greater than that of healthy, age-matched controls. They also note that CV disease accounts for approximately 50% of the mortality of the dialysis population. The aim of their study was to examine whether diabetes is independently associated with an increased risk of major cardiovascular events and death in patients with advanced chronic kidney disease (CKD). They have done this with a post hoc analysis of data from a trial which showed that folic acid supplementation did not reduce CV events in patients with CKD.

Their conclusion was that diabetes significantly increases the risk of major CV events, particularly peripheral vascular events in patients with advanced CKD.

Internal Medicine Journal 2011;12:825–32.

Antipsychotics and the risk of myocardial infarction

The authors of this study note that patients that are prescribed antipsychotics have higher mortality rates than those of a representative healthy comparison population. This excess mortality in these patient groups can partly be attributed to a two-fold higher cardiovascular death rate. On the other hand, patients requiring antipsychotic agents may be at a higher risk of myocardial infarction (MI) regardless of any effect of antipsychotic medication. An unfavourable risk profile for MI is associated with various lifestyle factors and comorbidities that are more prevalent in patients with a mental illness.

The aim of their systematic review was to ascertain whether the antipsychotic agents were associated with the increased risk. They discovered only five relevant studies. Four studies with small numbers of events reported a moderate to strong effect of typical antipsychotic agents on the risk of myocardial infarction. However, the largest study involving over 21,000 subjects (approximately ×10 the number of the other four combined) reported no association between the risk of myocardial infarction for either typical or atypical antipsychotic agents.

Br J Clin Pharmacol 2011;72(6):871–8.

Blood transfusion after hip surgery in elderly patients with cardiovascular risk factors

The haemoglobin threshold at which postoperative red-cell transfusion is warranted is controversial. This report concerns a randomised trial to determine whether a higher threshold for blood transfusion would improve recovery in patients who had undergone surgery for hip fracture. They enrolled 2016 patients who were 50 years of age or older, mean age 81.6 years, who had either a history or risk factors for cardiovascular disease, and whose haemoglobin level was below 10 g per decilitre after hip-fracture surgery.

Patients were randomly assigned to a liberal transfusion strategy (a haemoglobin threshold of 10 g per decilitre) or a restrictive transfusion strategy (symptoms of anaemia or at physician discretion for a haemoglobin level of <8 g per decilitre).

They found that the liberal transfusion strategy conferred no benefit over the restrictive strategy in terms of mortality or fitness to walk at 60 days nor did it reduce in-hospital morbidity. An editorial notes the results with interest and agrees that such a restrictive strategy would save blood and reduce transfusion reactions. However, it cautions that transfusion should be used on clinical grounds rather than laboratory finding alone.

N Engl J Med 2011;365:2453–63 and 25323.

Low-molecular-weight heparin in acutely ill medical patients

Pharmacologic thromboprophylaxis has been proved to reduce the incidence of venous thromboembolism in both surgical patients and acutely ill medical patients. Such treatments have been shown to reduce the death rate from pulmonary embolism and all causes in surgical patients. However, in acutely ill medical patients, although embolism rates are improved there is concern that overall death rates may not. This study evaluates this matter.

Over 8000 acutely ill medical patients were randomised to have subcutaneous enoxaparin 40 mg daily for 10 ± 4 days or placebo. Both cohorts also wore elastic stockings with graduated compression. The results were that there were no difference in the rate of death from any cause at 30 days. A somewhat counterintuitive finding which underlines the fact that acutely ill medical patients have more problems that the risk of embolism.

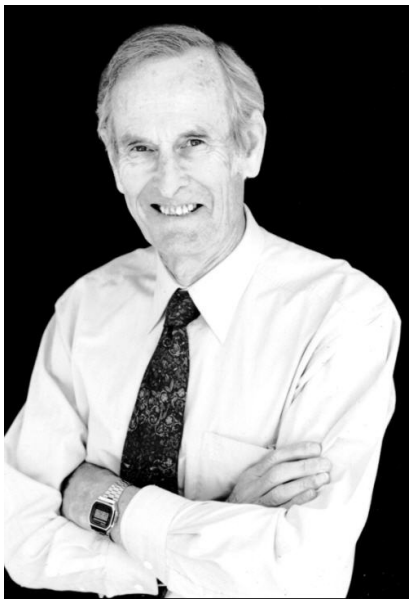
N Engl J Med. 2011;365:2463–72.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

John Murray Neutze

MBChB MD, FRACP, ONZM (1934 – 2011)

A leading heart specialist, medical researcher, and teacher, Professor John Neutze trained and mentored several generations of cardiologists and paediatricians. He is remembered as a man with a common touch, sharp wit and quixotic sense of humour.



John was born in Geraldine and grew up in rural New Zealand during the 1930s Depression and Second World War years.

A humble man, he was suspicious of those who presumed authority, and in his later years set to exposing the intellectual shallowness of the ideologically-driven health reforms of the day.

His role as a health advocate was grounded in a selfless and caring personality that made him popular with patients, families and staff.

A meticulous approach to medical research, superb clinical skills, and an untiring capacity for hard work placed him at the forefront of advances in the treatment of congenital heart defects in the 1960s and 70s.

John graduated MB ChB from Otago University in 1958. He trained at Green Lane Hospital under Dr James Lowe before joining a leading fetal and infant cardiac research group in the United States. At that time most babies with severe heart defects died shortly after birth.

While in America John realised that the team in New Zealand had the ability to make a major contribution to this field. He also witnessed first-hand the problems inherent in privately funded medicine, an experience that contributed to his lifelong support for a strong public health system.

John returned to New Zealand in 1967, and with surgeon Sir Brian Barratt-Boyes and cardiac radiologist Dr Peter Brandt, further developed the world leading Cardiac Unit at Green Lane Hospital.

These were exciting times. Cardiac surgery using the heart-lung bypass machine was in its early days, and infant cardiac surgery was just around the corner. For it to become a reality babies had to be stabilised prior to surgery. John was a leading innovator and was instrumental in a number of firsts in this field.

Shortly after returning to Green Lane Hospital, he performed the first balloon septostomy in New Zealand—a life-saving procedure that involved passing a balloon from a vein in the leg across the top chambers of the heart. Using X-ray guidance, the balloon was inflated, pulled across the foramen ovale, and a larger atrial septal defect

created. This procedure heralded the advent of interventional cardiology, an area in which John was active throughout his career.

Perhaps most notably John was a central figure in the first use of prostaglandin in the world, an event that was recorded in *The Lancet* in 1975. Prior to this, there was no way of stabilising babies with duct-dependent congenital heart disease. Diagnosis was frequently made post-mortem, and in those who survived to surgery, acidosis and multiorgan failure complicated treatment, contributing to a high mortality rate. By maintaining patency of the arterial duct, prostaglandin has revolutionised the treatment of newborn babies with heart conditions. It remains in use today and has allowed countless blue babies to survive so that cardiac surgery can be undertaken with low risk.

With help from physiologists and technical staff at Green Lane Hospital, John developed a method to measure oxygen uptake, which enabled accurate assessment of systemic and pulmonary blood flow. Detailed and precise measurements of pulmonary vascular resistance, accumulated over many years, resulted in an understanding of pulmonary haemodynamics that guides operability to this day.

For several decades John and Dr Louise Calder were the only two paediatric cardiologists at Green Lane Hospital and covered the call roster 3 months about. Despite this rigorous workload, John found time to discuss cricket and politics, seeking and valuing the opinions not only of colleagues, but also of patients and families. He maintained a keen interest in music and gardening and was a proud supporter of his sons' sporting endeavours. Always leading by example, John is fondly remembered for his antique commuter bicycle, upon which he negotiated Auckland's increasingly chaotic traffic wearing a series of old suits and a trademark bow tie.

Dr John Neutze was a world expert in the care of children with rheumatic heart disease and was a member of the WHO advisory group on Rheumatic Fever and Rheumatic Heart Disease. He was Chair of the Department of Cardiology at Green Lane Hospital for 18 years, and received an honorary professorship from the University of Auckland in 1994. His contribution to cardiology was further recognised in 2000 when he was made an Officer of the New Zealand Order of Merit.

A prolific author, his publications included 6 textbooks, 13 book chapters and over 100 medical articles. John was a strong supporter of a number of professional organisations. He served terms on Council for the Asian-Pacific Society of Cardiology, and the Cardiac Society of Australia and New Zealand, and sat on the scientific committee of the National Heart Foundation of New Zealand. Retirement did not curtail these activities, and he was elected for a term to the Medical Council of New Zealand.

An extraordinary New Zealander who touched the lives of countless families, John Neutze set the standard to which his colleagues aspire. He is survived by Beverly (his wife of 54 years), three sons and six grandchildren.

Tom Gentles (Paediatric Cardiologist) and Green Lane Hospital cardiology colleagues wrote this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

John Butler MacGibbon

Born Christchurch NZ 24 November 1921; died Baltimore US 24 December 2011

A much-loved Christchurch doctor, who lived for more than 60 years in Baltimore, Maryland, in the United States, where he practised and lectured in medicine, died on Christmas Eve, 2011. Dr John MacGibbon was 90. He died from the effects of a stroke and a fall. His ashes were to be scattered off the coast of his home town, Christchurch, where he was born and grew up.

MacGibbon was the eldest of four siblings in a prominent sporting family. His late brother, Tony, represented New Zealand at cricket, as a fast bowler. His late sister, Jean, was a New Zealand women's tennis champion.

His parents encouraged the children to be achievers. For MacGibbon, who was not a notable sportsman, this meant applying his considerable academic ability to the study of medicine. From Fendalton Primary School, he won a scholarship to Christ's College.

He then attended the University of Otago Medical School at Dunedin, where he graduated in 1948. Two years later, he won a Fulbright Scholarship, which took him to the US. He felt a strong attraction to Baltimore and settled there.

MacGibbon was a specialist in internal medicine and infectious diseases. He served in the US Navy in the 1950s as a medical officer with the rank of Lieutenant Commander. He was a keen sailor and enjoyed regular yachting excursions at Annapolis, on Chesapeake Bay.

His early medical career in Baltimore was spent at Wyman Park, where he provided outpatient and inpatient medical care for the crews of ships which used the Baltimore harbour when this was a very active port. In 1982, the Wyman Park facility was closed and MacGibbon moved his practice to the nearby Church Home Hospital.

MacGibbon was a legendary figure to those who knew him at this time. His knowledge of infectious diseases was extraordinary and a great asset in the medical care of shipping crews from around the world, most of whom were poor and had no health insurance.

He was a gifted linguist, with the mastery of several languages and the ability to converse in several more. His ease of learning languages assisted him in his profession as he treated sailors of many cultures. He was able to discuss with sailors the basic questions pertaining to their health issues, in their own languages.

The Baltimore port had a marked reduction in activity in the late 20th Century, and Church Home Hospital was closed in 2000. MacGibbon then provided general medical care in the city, with a special interest in his speciality of infectious diseases.

He was also a noted medical educationist. He was awarded a post-doctoral fellowship at Johns Hopkins University School of Medicine from 1954 to 1957 and was an instructor on the faculty of the Department of Medicine from 1960 to 2007. He regularly attended the weekly infectious disease conference. Colleagues say he was an exemplary physician, who was adored by his patients. He was a quiet, modest man considered by his peers to be a brilliant doctor.

MacGibbon remained passionate about anything relating to his home country. This was reflected in his never having relinquished his New Zealand passport in favour of a US one. He travelled widely and visited New Zealand regularly. He made his last trip here last February, at the age of 89. He arrived in Christchurch on February 22, just one hour before the magnitude-6.3 earthquake struck.

MacGibbon was a renaissance man, an avid reader and an accomplished watercolour painter. He enjoyed a wide range of music and loved the cinema, particularly art-house movies and comedies. He was an excellent cook and from his travels developed a taste for ethnic cuisines. He rode a motorcycle well into his later years.

He was a philanthropist, donating regularly and generously to a range of charities. He maintained close links with the University of Otago through the branch of its alumni in Baltimore.

MacGibbon never married, but devoted his life to his profession. Even after his retirement, he kept abreast of the latest developments in medicine through reading professional journals, researching online and attending lectures and seminars. He served as a member of the Baltimore City Medical Society board of directors and MedChi, the Maryland State Medical Society, for nearly 60 years. Through his deep involvement, he was interwoven into the fabric of Baltimore, its waterfront community and Johns Hopkins University.

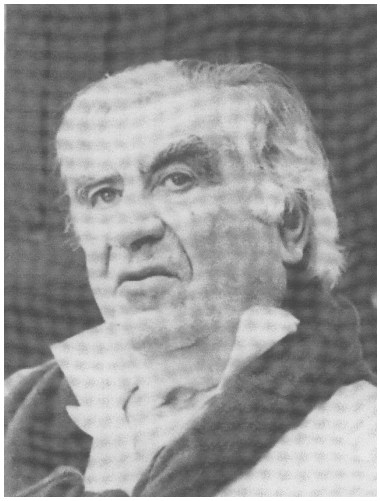
He remained close to his wider family and is sadly missed by his five nieces and two nephews, as well as many friends, colleagues and patients.

This obituary entitled *Brilliant doctor found success in Baltimore* originally appeared in *The Press* newspaper (Christchurch) and was written by Mike Crean.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Stuart Wendon Agnew

I am Stuart's wife, Hannelore. I met Stuart in 1981 in the Whitsundays (Queensland). I had come from Germany for holidays while Stuart had come from New Zealand for holidays too. Stuart was born in June 1922 in Dunedin, the first child of Elisabeth and David Agnew. His two sisters are Peggy in Oamaru and Frances in Geraldine.



Stuart studied at the Medical School in Otago (Dunedin). During World War 2 he volunteered for service in the New Zealand Army.

He served with the Medical Corps in Egypt and Italy and fought against the "Horrible Germans", and here in April 1987 in Auckland, Stuart married a German girl—me.

After atomic bombs were dropped on Hiroshima and Nagasaki, Stuart's battalion went to Japan as part of the Occupation Force. The poor boys, they surely received a dose of radiation. I think in 1945 people did not yet realise what the nuclear fallout did to their health.

After Japan, Stuart went back to Dunedin and worked for a while in his father's import business, but that was not the right thing for him. He wanted to continue his medical studies so he went to Ireland (his Mum and Dad originated from Ireland).

In 1956 at Queen's University in Belfast Stuart was admitted to the degrees of Bachelor of Medicine, Bachelor of Surgery and Bachelor of Obstetrics.

In 1959 he came back to Dunedin as his father was dying of terminal cancer. In 1960 Stuart took up practice in Oamaru where he worked for 4 years. He also was Medical Officer at Otematata at the Benmore Hydro Dam. Though very busy he still could take up his passion for glider flying.

In 1965 Stuart came to Auckland, worked first in his own practice and then joined a group practice on the North Shore. He also acted as Coroner. Here in Auckland in his spare time he now could follow his great interest—sailing. Stuart loved the sea very much and I remember with pleasure when I crewed for him on his sailing boat "Safari".

Stuart passed away on 28 December 2011 (aged 89 years and 6 months).

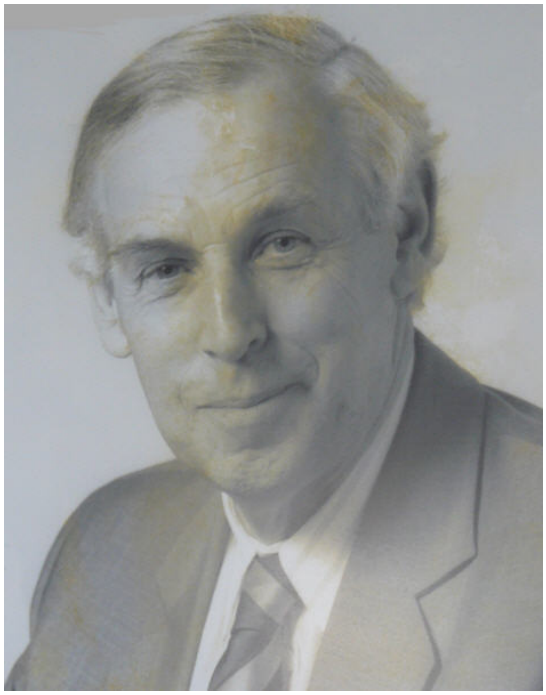
Goodbye Stuart, we miss you. From your family, many many friends and colleagues.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Liam Hugh Wright

December 1926 – August 2011

Mr Liam Wright was an obstetrician and gynaecologist in Auckland with a particular interest in gynaecological malignancy.



Liam was born and raised in Taranaki.

His mother Stella Hickey had been Headmistress of Opunake School and his father William John Wright had returned from the War in 1920. They married and settled on a farm purchased by Liam's grandfather in the 1870s.

Schooling commenced at the Rahotu School then Opunake School with Liam matriculating from St Patrick's College, Silverstream in 1942 at the age of 16. Liam's older brother Ralston was with the Airforce and Liam spent the years 1943–46 working on the farm.

Liam completed his Medical Intermediate in Wellington and then went to Dunedin in 1948.

Accomodation in Dunedin then would not be contemplated by students now: a shared bedroom and facilities in a family home with another student, however Liam greatly enjoyed his time in Dunedin, completing his MB ChB and a BMed Science in Anatomy, subsequently lecturing in Anatomy. He also played rugby and was socially active, being elected President of the Otago University Students Association in 1951.

Liam met Barbara Brosnan in Wellington but it was in Dunedin, where Barbara was studying physiotherapy, that they become close and married in January 1952. Liam had a bonded Health Department grant and after two house officer years in Palmerston North, he and Barbara with two small children moved to Mangakino where Liam became the sole general practioner.

In those years Mangakino was the centre for the Waikato Dam construction project and was very busy however Liam studied for and passed his surgical primary being one of the first to do so in New Zealand.

The path to obstetrics and gynaecology reflected his experience as a general practioner in rural New Zealand in the 1950s where acute obstetrics could be a major challenge. Liam applied for a position as Registrar at National Womens Hospital (NWH), at that time based in an ex-army hospital in Cornwall Park, moving into a flat in the hospital grounds with Barbara and now three young children. It was here that he

met Pat Dunn with whom he worked closely for many years. Liam was also influenced and guided by Professor Harvey Carey, Chair of Obstetrics and Gynaecology.

In 1959, Liam, Barbara and four children aged 6 weeks to 6 years took the *Athenic* (a passenger cargo ship) to the UK. Liam worked for a year in London, then in Oxford and finally as Senior Registrar in General Surgery in Nottinghamshire. He gained his MRCOG in 1959 (FRCOG in 1976), FRCS and FRCS(Ed) in 1961 and subsequently FRACS.

Later he and others realised that the combination of geographical separation and different approach meant that the direct links with and supervision by the RCOG was not ideal and he supported an independent college. Although he became the first president of the RNZCOG in 1982 he always believed that Australia and New Zealand should stand together and was gratified when they combined to form the RANZCOG in 1998.

The family returned to Auckland in 1962 where Liam joined the staff of NWH, became one of the founding members of the Middlemore Hospital Obstetric Unit and commenced private practice. With Pat Dunn he ran an obstetric unit at The Mater Misericordiae (now Mercy) Hospital; this service closing in the 1970s because of funding issues.

Liam also developed his interest in the management of patients with gynaecological malignancy particularly cervical cancer. During the 1970s and 80s he was involved with much of the radical pelvic surgery indicated for this disease. It was probably with some relief that Liam was able to hand on this demanding surgery to younger colleagues.

It is not surprising that Liam was required to give evidence to The Cervical Cancer Inquiry, the report being published in 1988. He had rare insight into the personalities involved and great knowledge of the clinical issues. At that time he did not have full insight into the political issues, however one of those present described him as "refreshingly honest" but then went on to describe him as "detached". It may be that this detachment in a stressful situation was one factor in being a good surgeon. In reality with his patient (as with all others) he had great empathy and sympathy in both public and private settings.

Liam continued with his general gynaecology and obstetrics, and became the Chair of the first Medical Advisory Committee to The Auckland Hospital Board (now divided into three District Health Boards). This committee reviewed all senior appointments and advised on significant and sometimes controversial service changes—for example the transfer of general surgical services from Greenlane to North Shore Hospital. He also joined the administrative staff of The Mercy Hospital as Medical Supervisor, being Acting CEO for a period. Here, as with all his appointments, he demonstrated his ability to calm troubled waters, manage pressure without apparent stress and see the best in all others.

The clinical practice of obstetrics and gynaecology remained a source of enjoyment for Liam who undertook his last delivery at the age of 64 and his last operation at the age of 69, ages which he had selected as appropriate years previously. He continued to consult and to work on the staff at The Mercy until he retired at the age of 74.

Apart from his professional life, Liam's greatest pleasure and joy was his family and extended family. He never completely recovered from the death of his oldest son Christopher in a motorcycle accident in 1977 but his overall optimism and positive outlook remained . Liam remained closely in contact with his Taranaki origins throughout his life and was proud of his parents and his siblings and their achievements.

Liam's greatest relaxation came from his annual camping holiday which he and Barbara undertook each January at Bland Bay, a quiet Northland Beach. He did not miss a year in the last 40. In Auckland he derived great pleasure from his regular bridge and even during the last few months managed the crossword from the *Herald* and the *Guardian*.

Liam is survived by his wife Barbara, and by Peter and Mark, respectively a barrister and surgeon in Auckland, also by Virginia a documentary producer in Christchurch. He is sadly missed.

Mark Wright (a son of Liam) wrote this obituary.

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Reviewers for the New Zealand Medical Journal in 2011

The Editorial Board (T Buckenham, J Reid, R Mulder, J Connor, R Beasley) and Editorial Team thank all those who generously gave their time and expertise in reviewing papers for the *NZMJ* in 2011. (We apologise to anyone whose name has been inadvertently omitted from the following list.)

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