In this issue:
- The high health burden from alcohol in New Zealand and the need for an appropriate government response
- Euthanasia and physician-assisted dying
- Prevalence predictions for age-related macular degeneration in New Zealand have implications for provision of healthcare services

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SUMMARIES

The burden of disease and injury attributable to alcohol in New Zealanders under 80 years of age: marked disparities by ethnicity and sex
Jennie Connor, Robyn Kydd, Kevin Shield, Jürgen Rehm

This study summarises major effects of current drinking patterns on health of the NZ population, although there are additional health and social harms not included in the study. Men are more affected than women, and Maori are more affected than non-Maori. These differences are largely due to higher rates of injury in these groups, due to more frequent intoxication. The leading cause of alcohol-related death in women is breast cancer, and alcohol also increases the risk of other common cancers. Improvements in health, and reduction in disparities, will require reduction in consumption across the population using types of regulation known to be effective for doing this.

Motor neurone disease in the greater Wellington region: an observational study
Viswas Dayal, Ian Rosemergy, Janet Turnbull

Motor neurone disease (MND) is a degenerative condition affecting the nervous system and leads to progressive disability due to weakness involving the limbs, muscles involved in swallowing and speech, and breathing. MND in the Wellington region was found to be prevalent at a rate of 8.5 per 100,000 people over a 12 month period. The average age of onset of MND was 66.2 years, and the median survival time for these patients was 29 months (2.4 years). The disease most commonly manifested as a combination of limb and bulbar (swallowing and speech) muscle weakness, and less commonly as weakness of respiratory (breathing) muscles. The Wellington region provides comprehensive healthcare services to these patients with input from medical specialists, dietitians, speech language therapists, and palliative care providers.

Does Pūkawakawa (the regional-rural programme at the University of Auckland) influence workforce choice?
Christina Matthews, Warwick Bagg, Jill Yielder, Vernon Mogol, Phillippa Poole

University of Auckland medical students spending a year in Northland greatly value the experience. In the early years after graduation they are likely to work in regional and rural areas of New Zealand. In addition, these graduates show an intention to work in general practice. Together these outcomes may help to address workforce shortages and misdistribution in New Zealand. Funding of regional and rural medical student learning opportunities ought to be a high priority.

Prevalence predictions for age-related macular degeneration in New Zealand have implications for provision of healthcare services
David Worsley, Andrew Worsley

Age-related macular degeneration (AMD) is a very common eye condition and is associated with ageing. AMD causes approximately 50% of blindness in New Zealand. As New Zealand has a rapidly ageing population, the prevalence of AMD can be expected to also increase markedly in the next two decades. In this study we predict the prevalence of AMD in New Zealand from 2014 through to 2026. The prevalence of any AMD in New Zealand for the 45–85 year age group is estimated to be 184,400 in 2014 (10.3% of this age group) and increase 12.9% to 208,200 (9.9% of this age group) in 2026. For 2014 and 2026 respectively, early disease is estimated to be 167,500 and increase to 189,200 and late disease is estimated to be 7,600 and increase to 8,600. AMD prevalence predictions for the very elderly (over 85 year age group) could not be included in this study, but may add approximately 15% to these figures.

The expected increase in prevalence of AMD is likely to be a major healthcare burden for which New Zealand is not well prepared. By international standards New Zealand has a low level of investment in AMD healthcare; the lowest public funding of anti-VEGF treatment in the OECD, no specific funding for prevention strategies and a relatively small ophthalmic workforce and public infrastructure. As such, there is an urgent need to plan for an increasing demand for AMD treatment, prevention strategies and associated ophthalmic services. The alternative is risking a major increase in preventable blindness.
The dissolution of the Alcohol Advisory Council: a blow for public health ((viewpoint article))
Kypros Kypri, Jennie Connor, Doug Sellman

Alcohol consumption is a leading cause of premature death and disability in New Zealand. These harms are suffered disproportionately by men and Māori but there are also harms from others’ drinking (e.g., domestic violence) that are less well documented and more often suffered by women and children. The Alcohol Advisory Council (ALAC), set up in 1976 and funded with a hypothecated (i.e., earmarked) tax on alcohol, was dissolved by the Government in 2012 and replaced with the Health Promotion Agency (HPA). While still receiving the hypothecated tax revenue, the HPA is less able to express views that might offend government and to undertake or fund research evaluating alcohol policies. It is also compromised by having a leading alcohol industry figure on its Board. We propose that the tax proceeds which fund HPA’s research functions go directly to the Health Research Council to fund policy-relevant alcohol research. We are pessimistic about the likelihood that HPA will contribute robustly to reducing the heavy burden of alcohol-related harm in NZ.
The high health burden from alcohol in New Zealand and the need for an appropriate government response

Nick Wilson, Tony Blakely

This issue of the Journal features two articles on alcohol. The article by Connor et al details the large health burden from alcohol in New Zealand at an estimated 5.4% of all deaths under 80 years old (around 800 premature deaths annually). They also estimated 6.5% of all healthy life lost among 0–79 year olds in 2004 was attributable to alcohol (a loss of around 28,000 disability-adjusted life-years [DALYs]—although balanced against this was around 7000 DALYs averted). Furthermore, the analysis provides additional evidence that alcohol use contributes to health inequalities between Māori and non-Māori and between men and women.

Should New Zealand policymakers consider these estimates credible? We think they should—while still being aware of the limitations of this type of analysis as outlined by Connor et al themselves. Indeed, the key methods used are well accepted by public health scientists—as witnessed by the publication of the Global Burden of Disease 2010 (GBD2010) study result in top journals such as the Lancet. Work by the New Zealand Ministry of Health has also used such methods in the recent New Zealand Burden of Disease Study (NZBDS).

If anything though, the methods used by Connor et al seem conservative and so probably underestimate the net health harms of alcohol use in New Zealand. For example, they mention a particularly important Mendelian randomisation study casting doubt on cardioprotective benefits of alcohol use (that we have previously commented on), but there is also now another relevant study just published in the BMJ. This recent study also suggests the limited nature of any cardioprotective effects from alcohol (i.e., largely limited to women drinkers aged 65+ years). Furthermore, if the long-term trend of declining cardiovascular disease continues in New Zealand, it is likely that any such cardioprotective benefits will decline further (but so too will some of the cardiovascular disease harm from high alcohol intake, e.g., haemorrhagic stroke).

Of course from a total societal cost perspective the impact of alcohol presented by Connor et al is also a marked underestimate of the burden of harm. As the authors note there are various other harms “such as crime, public disorder, disruption of families, or loss of employment” that were out of scope in their analysis—but which national level policymakers need to consider.

While benefits of alcohol such as the pleasure and social lubrication from modest amounts are also part of the benefit/harm tradeoff—as a society we seem, as the Law Commission identified, to be paying an excessively large price in the form of alcohol-related harm.

To put the results of Connor et al into context it is possible to consider related bodies of work. That is the NZBDS ranked alcohol as the sixth most important risk factor for health loss (after tobacco use, high BMI, high blood pressure, high blood glucose, and physical inactivity). For Australasia (Australia and NZ combined) the GBD2010 ranked alcohol use as the ninth most important risk factor.

Fortunately, there are many effective and evidence-based solutions to the alcohol problem and some of these, such as higher alcohol taxes, are likely to be either cost-saving or very cost-effective. Government could then use additional alcohol tax revenue to lower income taxes, or alternatively for projects to improve societal wellbeing in other ways (e.g., funding healthy school meals).

But organisational and research funding issues are also important for addressing the alcohol problem—and that is where the other article in this issue of the Journal by Kypri et al comes in. The three authors are all professors and long-term experts in researching alcohol issues. They thoughtfully...
describe the demise of the Alcohol Advisory Council (ALAC) and how its functions have been “ostensibly taken over by the Health Promotion Agency (HPA).” However, they argue that the HPA has been given less autonomy than ALAC and they raise concerns that its broad remit might reduce its capacity to deal with alcohol. They also note how the “HPA was compromised from the start by the appointment of a food, alcohol and tobacco industry representative to its Board.” The latter issue has drawn concern from others as detailed in an article in the BMJ and from public health experts as reported in the New Zealand media.

We find the analysis by Kypri et al to be a well-considered one and agree with the recommendations around the need for greater transparency and independent analysis and monitoring. Such measures may help ensure the best use of taxpayer funds—and minimise the risk of problematic industry influence (as per aspects of the “dirty politics” saga). This would all help with New Zealand moving forward to realise the health, social and economic gains of improved alcohol control.

Competing interests: Nil. More specifically the authors have no financial interests in the alcohol sector and have never received research funding from ALAC or the HPA.

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Euthanasia and physician-assisted dying: editorial comment and reply to the Waikato GP survey findings by Dr Havill

A D (Sandy) Macleod

The Roman Emperor, Augustus, prayed for euthanasia, a good, easy and painless death. He did achieve this in his old age. His fear was an early death by violent assassination. In contrast, General Franco’s death in 1975 stands for the most horrible medical death, a death that only doctors could devise.

Heroic interventions and life support systems can delay death. The Greek myth of Tithonus tells of the pain of prolonged life in loathsome old age. Begging for death to overcome him, his pleas were ignored. Modern euthanasia advocates rile against the violent death made possible by modern medicine’s ability to sustain life.

A fear of dying badly (dysthanasia), rather than the fear of death itself, whilst not new, is more prevalent and probably more likely an eventuality in modern Western societies. Multiorgan failure, including brain failure, and the elongation of dying, can be the cost of a few weeks or months granted by chemotherapy.

Science has driven medical progress but the art of sensible caring is at risk of being lost. How and when we die has always been a concern of mankind. It is encouraging that our profession is beginning to more openly discuss the issues of euthanasia, actively killing patients, and physician-assisted suicide (PAS).

The letter of Dr Havill (in this issue of the Journal) does relatively little to further our knowledge. The response rate was poor and it is likely that it was only those GPs with firm views, supportive or otherwise, who bothered to reply. But nonetheless, that about 40 Waikato GPs would be willing to hasten the deaths of their patients, even if they are mentally incompetent, is noteworthy.

Certainly there has been a thawing of the trenchant opposition by Medical Governing bodies over recent decades, implying an invitation to consider the issues in a reasoned and reasonable fashion. However there are many ethical, philosophical, legal, fiscal, religious and political influences to ponder, let alone the medical ones.

One of the surprises emerging from the Netherlands, Belgium and Oregon, legislations in which medicinal killings are allowed, is that it is not unrelieved physical symptoms or the inadequacy of palliative care services that stimulate euthanasia requests, but fears of loss of control and being a burden on others. It is for psychosocial and existential reasons that most request hastened death.

The stability of request is a critical factor. Thirteen percent of Dutch requesters changed their minds and up to a third of Oregonians provided a lethal prescription opted to die naturally. Medically estimating prognosis is notoriously imprecise. A definition of “unbearable suffering” has yet to be determined. The accurate assessment of competency and mood in those seriously ill is by no means well established. These medical tasks are difficult and as yet, uncertain. I doubt our bedside skills are as yet sufficient to competently assess medical fitness for physician-assisted dying.

In those with neurodegenerative disorders, such as the dementias, it is even more challenging. Neurology and psychiatry are the specialties likely to be most affected by liberalised euthanasia legislation and the early reports of euthanasia requests by Belgium psychiatric patients are likely to catapult discussion into further controversy (unpublished correspondence). A difficulty of caring for dementia, which is a terminal disease, is often the mismatch between the patient’s perception of quality of life and that of the relatives (and maybe the attending staff).
Paradoxically with fading cognitions the determination to live is usually enhanced. This, a variant of the disability paradox, is a challenge to the validity of Advance Directives and it also complicates the palliative care of this increasingly common disorder. Thus the answer to a question posed by Havill may depend on when and to whom it is addressed.

The surveys of the general public indicate that the closer one may be to death, the less certain is the ideology to foreclose life. The enthusiastic support for euthanasia fades in oncology outpatient clinics and is virtually dead in hospices. Surveys of medical specialists parallel this trend.8

GP's may well differ from specialists and indeed it is GPs who field the majority of requests. The incidence of medicinally hastened death is poorly researched. Not uncommonly an alteration of opioid dose is attributed for a community death that was imminent anyway, so medicinal killing is assumed, but probably did not occur. Prompt relief of “unbearable” suffering is the persuasive argument in favour of euthanasia (though the medicolegal processes may take considerable time).

Relatives may be the beneficiaries of the hastened death of their love one. The grief of surviving relatives may be eased by a prescribed death.9 Appropriate analgesia and palliative sedation has been shown to prolong remaining life in those terminally ill rather than abbreviating it, thus potentially adding psychological burden to relatives (and perhaps staff). However there are medical problems to overcome in legalised euthanasia jurisdictions.

Allowing requests to be sanctioned is fraught with as yet uncertain method and practitioner idiosyncrasies. US states, in which capital punishment is practised, struggle to medicinally execute humanely, with speed and precision. And Dutch doctors are increasing hesitant to practise euthanasia and are deferring to nurses to administer the fatal drug.10

A solution to ‘unbearable’ suffering toward the end of life is, as proposed by Havill, physician-assisted dying (PAD). By removing “suicide” from the term, presumably to counter the concerns of suicide prevention educators, moves the proposed practice toward euthanasia, the deliberate ending of another person’s life at his or her request. Alternatives to PAD include regaining the art of good clinical judgement and decision-making, diagnosing dying and allowing natural death, and skilful palliative sedation if intractable symptoms (most frequently delirium) can’t be managed in other ways. Yet every clinician knows of cases in which mercy killing might appeal.

But the broader risks of prescribing death remain uncertain. The Dutch, Belgians and Oregonians are accumulating experience and opinion and they are deserving of admiration for these efforts. But the slippery slope has happened before in history, and legal protections are not always full-proof (and invariably very expensive).

A ‘peaceful’ death is desirable and Augustus was lucky in this regard. King George V was *euthanised* by his doctor in 1936. Maybe the King was lucky for he was enduring an undoubtedly terminal delirium caused by respiratory failure. Dr Dawson acted, he stated, to preserve the King’s dignity, to prevent further strain on his family, and to ensure that the announcement could be made in the following morning’s newspaper. I am not sure Dawson’s rationale was proper.

We certainly need to manage the dying better. We need good research, wise expert opinion and fair legislation. We lack these. Dying is not invariably easy, and clumsy medicine can aggravate it. But is it best to give up and terminate life by the violence of non-physiological pharmacology?

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Motor Neurone Disease: bringing New Zealand patients onto the world stage

Emma L Scotter

Motor neurone disease (MND) is the umbrella term for a group of degenerative diseases of the motor neurons, of which amyotrophic lateral sclerosis is the most common form. The incidence of MND is around 2 per 100,000 globally and median survival time is approximately 3 years from symptom onset.1

MND may be either familial or sporadic, with the vast majority of cases (95%) being sporadic.2 Genetic characterisation of international cohorts shows this to be a somewhat artificial distinction—at least 1 in 10 sporadic cases carry a heritable genetic mutation.3 However New Zealand MND patients are yet to be represented in those cohorts. Indeed, there is a woeful lack of publicly available data on the demographics, aetiology (including genetics) and provision of care of MND patients in New Zealand thus far.

Outside of New Zealand, there have been significant recent advances in the genetic characterisation of both sporadic and familial MND. This has been made possible by massively parallel DNA sequencing technology which can process more samples for a fraction of the time and cost of previous methods. To date, 33 disease-causing genes have been identified,4 accounting for around 15% of all ALS cases.3 Notably, the same genetic mutations are present in both sporadic and inherited forms of MND, dispelling previous notions that these were distinct diseases. Furthermore, many of these genes can also cause frontotemporal dementia, consolidating the clinical and neuropathological picture of MND and frontotemporal dementia as a spectrum of overlapping diseases.

Disease-causing genes implicate dysfunction of gene expression (RNA processing), protein degradation, and cargo trafficking in the degeneration of motor neurons which causes MND. It is likely that the genetic profile of MND in New Zealand will largely resemble that of the UK and Europe, given the ancestry of New Zealand Europeans, however the genetics of Māori and Pacifica MND is currently a black box.

Although a rare disease, MND is certainly under-studied in New Zealand when compared to the likes of Huntington’s disease, which has similar prevalence. This is surprising given that the MND studies conducted here have yielded findings worthy of follow-up.

A “cluster” of six port workers in the Nelson area developed MND within a 10-year reporting period (1995–2005), five of them within a 4-year period (2002–2005).5 The estimated population of port workers in the Nelson area at the time was 150. This led to postulation that these MND cases were linked to the use of methyl bromide gas as a fumigant for cargo.5 The small numbers involved mean that the cluster could simply have been due to chance5 and that continued reporting is needed.

In 2006 a larger, prospective longitudinal study in Christchurch reported the highest incidence of MND in the world thus far, finding a steady increase in incidence over a 22-year period from 1985, the cause of which is yet to be determined.6 When included in large international meta-analyses (and considered to represent the national average), these two studies paint New Zealand as having extraordinary disease rates1 not attributable to higher numbers of familial cases.2

There is a clear need for both regional and nationwide studies of MND incidence, prevalence and mortality rates in New Zealand, whether remarkable or unremarkable compared to international averages.
The article by Dayal et al in this edition of the Journal provides important and timely new information on the demographics of MND patients in the greater Wellington area, and on the provision of healthcare to those patients. The 40 MND patients identified would have represented around 15% of all MND cases in New Zealand at that time, based on a predicted prevalence of 300 MND patients in New Zealand at any point in time. It is of interest therefore that this study reports rates of incidence and other disease demographics (male to female ratio, median survival time) that align closely to international averages. Dayal et al. also provide some of the only available information on the ethnic makeup of MND patients in New Zealand.

The authors detail useful statistics on the provision of care of MND patients in New Zealand. Non-invasive ventilation, speech language therapy, and provision for percutaneous feeding can significantly improve quality of life for MND patients. Their complex and rapidly changing needs can be challenging for non-specialist MND care providers, such as hospice, so this report provides valuable information on the whether patients are receiving the needed services within the hospital setting. Before this can occur however, patients must be diagnosed with definitive or probable MND by a specialist neurologist. The median delay to diagnosis of MND according to international studies is about 14 months due to its heterogeneity and similarities to other diseases, and these delays are a major concern in a disease with relentless and rapid progression.

The aetiology of MND has been scrutinised for nearly 150 years since it was first described by Charcot in 1869, and yet only in the last decade has real progress been made in identifying causal factors. Although analyses of disease clusters (temporal, geographical or both) have failed to confirm or refute the role of any one environmental factor, genetic studies have been illuminating. The ultimate goal will be to examine the interplay between all gene variants and environmental factors which increase risk, in order to best design therapeutic and prevention strategies. New Zealand must keep up with (and be included in) these analyses so that our unique profile of genetic and environmental factors can be understood.

Competing interests: Nil.


The burden of disease and injury attributable to alcohol in New Zealanders under 80 years of age: marked disparities by ethnicity and sex

Jennie Connor, Robyn Kydd, Kevin Shield, Jürgen Rehm

Abstract

Aim To update and improve estimates of morbidity and mortality due to alcohol consumption in New Zealand.

Method We applied the comparative risk assessment methods of the Global Burden of Disease Study at country level, and separately for Māori and non-Māori where possible. Analysis was restricted to 0–79 year olds.

Results We estimated 5.4% of all deaths under 80 years old were attributable to alcohol in 2007 (802 deaths) and these represented 13,769 years of life lost (YLLs). Injuries accounted for 43%, cancer for 30% and other diseases for 27% of deaths. We also calculated 351 deaths were averted by alcohol use, but only 3095 YLLs, resulting in a net annual loss of more than 10,000 years of life.

Sex and ethnic disparities were marked, with twice as many deaths in men as women for both Māori and non-Māori, and the age-standardised death rate for Māori two and a half times the rate for non-Māori. Injury was the biggest cause of alcohol-related deaths and YLLs in the young and overall, but the leading cause of alcohol-related death in both Māori and non-Māori women was breast cancer.

We estimated 6.5% of all healthy life lost among 0–79 year olds in 2004 was attributable to alcohol (28,403 DALYs lost), and 6538 DALYs were prevented. The sex disparity in DALYs lost mirrored the mortality analysis, but no disaggregation by ethnicity was possible.

Conclusion Alcohol consumption results in substantial loss of good health across the life course in New Zealand. It makes an important contribution to Māori/non-Māori and male/female health disparities. High average consumption and heavy drinking occasions confer the greatest risk of harm to the drinker and others. At a population level there are no documented health benefits of drinking before middle-age and benefits in later life are increasingly uncertain.
Impacts of alcohol consumption extend beyond the drinker and effects on others are common. In a recent survey, 1 in 4 New Zealanders reported a heavy drinker in their life in the previous 12 months, and this was associated with reduced personal wellbeing and poorer health status, as well as a range of direct harms attributed to their drinking. In other recent New Zealand studies, a higher proportion of the general population reported harm from the drinking of others (18%) than from their own drinking (12%) in the past year, and it was estimated that 43% of car occupants injured in alcohol-related crashes were not the drinking driver.

More generally, alcohol use has a cost to societies in loss of amenity and in the very substantial opportunity costs of public spending on the additional health services, policing, courts, prisons, social welfare support, special educational services and other facilities that are required.

There are numerous challenges to measuring the complete health burden of alcohol in populations, starting with the breadth of definition of health to be adopted, and how far from the drinker the effects can be tracked. Even within boundaries of countable physical and mental health outcomes, gaps in mortality and morbidity data are evident in New Zealand as elsewhere, particularly for harms to others. Together with insufficient certainty about some epidemiological relationships, and the evolution of the knowledge about others, it is a complex task.

This study aimed to describe a well-demarcated but limited component of the burden of harm from alcohol in New Zealand, updating and improving on a previous study.

The first estimates of the “Burden of death, disease and disability due to alcohol in New Zealand” were published in 2004 by the Alcohol Advisory Council of New Zealand, and in this journal. That work estimated mortality in 2000 and disability-adjusted life years (DALYs) in 2002 attributable to alcohol consumption. It has been widely used, for example by the New Zealand Law Commission in reviewing the impacts of alcohol in New Zealand, and so this new study has updated the estimates with more recent alcohol consumption measures and improved methods.

Since the publication of the previous report, some of the alcohol burden calculation methods that were used have been revised by the Global Burden of Disease 2010 Risk Factors Collaborating Group, and together with the other changes described below, this means that it is not valid to draw conclusions about change over time by comparing the two studies.

The aim of this study was to update New Zealand estimates of: (i) alcohol-attributable deaths and years of life lost (YLLs) by age and sex for Māori and non-Māori (2007), and (ii) alcohol-attributable disability-adjusted life years (DALYs) lost by age and sex (2004).

**Methods**

The methods used in this study are described in detail elsewhere and are only outlined here.

**Comparative risk assessment (CRA)**

The CRA methodology, developed by the World Health Organization (WHO), is a systematic approach to measuring and ranking the burden of disease attributable to a range of important global risk factors. It was designed for application at a global and regional level, and first used in the World Health Report 2002. CRA aims to combine best estimates of the risk factor distribution in the population, with best estimates of risk factor-disease relationships from the international epidemiological literature to measure the impact of each major risk factor using common metrics of mortality, years of life lost (YLLs), and disability-adjusted life years lost (DALYs).

In this analysis we applied the CRA approach to alcohol at a country level, and for Māori and non-Māori separately where possible, using the updated methods of the Global Burden of Disease Study 2010. The contribution of alcohol consumption to the health burden was quantified with an alcohol-attributable fraction (AAF) for each alcohol-related condition or injury in each age/sex/ethnicity subgroup of the population.
The mortality and morbidity AAFs used the theoretical-minimum risk exposure of lifetime abstention and thus estimated the proportions of death and disability, respectively, that would have been prevented if alcohol was never consumed by anyone in a given subgroup. These were calculated by combining New Zealand alcohol consumption data with the best available estimates of risk at different levels of consumption (see for the exact formulas used to model the AAFs).

Death and disability due to alcohol consumption were then estimated for New Zealand subgroups by applying the mortality AAFs to New Zealand mortality data for 2004 and 2007, and morbidity AAFs to WHO’s 2004 disability estimates for New Zealand. The DALY is a summary health gap measure that integrates fatal and nonfatal outcomes (measured by years of life lost and years of life lived with disability).

Scope of the study

The selection of the conditions attributable to alcohol was based on evidence of established epidemiological relationships, assessed by the GBD2010 alcohol group, using meta-analyses, new research, and biological evidence. Three groups of conditions were considered: wholly alcohol-attributable conditions, with AAF of 100%; chronic conditions where alcohol is a contributing cause (detrimental or beneficial); and acute conditions where alcohol is a contributing cause. The conditions included in this study are listed in Table 1.

Outcomes of alcohol consumption are not included unless they are coded in ICD-10 (International Classification of Diseases Version 10) and so broader impacts such as crime, public disorder, disruption of families, or loss of employment are outside the study’s scope although it is recognised that they also contribute to population health. Mortality data were available for Māori and non-Māori separately but DALY data were not. The analysis was restricted to people over 15 years of age, apart from the secondary effects of drinking by an adult on a child where data were available. These effects included traffic injuries, fire injuries, other unintentional injuries, assault, low birthweight, and fetal alcohol syndrome.

Changes since the previous New Zealand report

The basic approach was similar to that used in the previous study. The main differences in methods are outlined here, with the rationale and further details available in the full report. Age range—We restricted this study to people less than 80 years of age, due to inadequate data quality for both alcohol consumption and relative risks over this age. Thus, health outcomes that are common in older people, such as CVD and falls, will be least comparable with previous estimates.

Conditions included—Four additional alcohol-attributable conditions have been included on the basis of epidemiological evidence supporting a causal association, and sufficiently detailed dose-response information being available: colon cancer, rectum cancer, tuberculosis, and pneumonia. Unipolar depression has been excluded since the last study, consistent with the GBD2010 assessment that characterisation of the causal association was insufficiently reliable. However, we conducted a sensitivity analysis with depression included using the methods of the previous study.

Alcohol consumption data—The ‘2003/04 Health Behaviours Survey – Alcohol Use’ conducted by the Centre for Social Health Outcomes Research and Evaluation (SHORE) was used to estimate alcohol consumption for 2004, and the 2007/08 New Zealand Alcohol and Drug Use Survey conducted by the Ministry of Health was used to estimate alcohol consumption in 2007. Extrapolations of consumption in older age groups also used data from the Harm to Others survey (2008/9) conducted by SHORE.

Relative risk estimates—The ‘abstainer’ reference category used in this study included only lifetime abstainers, with ex-drinkers included as a separate alcohol consumption category to reflect the difference in health risks of ex-drinkers compared to lifetime abstainers.

New systematic reviews and meta-analyses performed as part of the Global Burden of Disease Risk Factors Assessment for alcohol resulted in a number of substantial differences in the relative risk (RR) values used to calculate alcohol-attributable fractions in subgroups. These included some decrease in RRs for cancers, cardiac arrhythmias, pancreatitis, epilepsy, liver cirrhosis, and hypertensive heart disease; increased RRs for haemorrhagic stroke; and more protective RRs for diabetes at the lowest level of alcohol consumption with increased risk for the heaviest drinkers.
We used specific ischaemic and haemorrhagic stroke relative risk estimates, where the previous study applied ‘total stroke’ relative risk estimates to combined mortality data for all stroke subtypes.

Morbidity RRs were available for the calculation of specific morbidity AAFs for ischaemic stroke and haemorrhagic stroke, cirrhosis of the liver, and ischaemic heart disease. For all other non-injury conditions, mortality RRs were used as the best available estimates of morbidity RRs, as before.

Incorporating drinking patterns into AAF calculations—Methods for incorporating drinking pattern information have been further developed by the GBD2010 and are markedly different from those used for the first report. In the previous study a drinking pattern score was applied to a whole population (i.e. Māori and non-Māori each had a single score), but in this study we have estimated drinking pattern parameters for each age/sex/ethnicity subgroup from detailed consumption data.

Ischaemic heart disease (IHD)—AAF calculations for IHD used new methods based on recent meta-analyses that examined the relationship between IHD, average alcohol consumption, and irregular heavy drinking occasions (HDOs), and also showed the importance of separating ex-drinkers from abstainer reference groups. The authors (Roerecke and Rehm) calculated sex-specific relative risk values for use in our study.

In essence, the evidence shows no cardioprotective effect of consumption under 60g per day when this is accompanied by irregular heavy drinking occasions. The method used is conservative—i.e. probably underestimates the negative impact of irregular HDOs on ischaemic heart disease (Michael Roerecke, personal communication, 2013).

Table 1. Alcohol-related conditions included in the study, and whether the effect is detrimental (-), is protective (+), or varies by subgroup (+/-)

<table>
<thead>
<tr>
<th>Conditions arising during pregnancy</th>
<th>Respiratory disorders</th>
<th>Intentional Injuries</th>
<th>Other unintentional injuries</th>
<th>Other intentional injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low birth weight</td>
<td>Tuberculosis*</td>
<td>Unintentional injuries</td>
<td>Fails*</td>
<td>Self-inflicted injuries</td>
</tr>
<tr>
<td>Fetal alcohol syndrome</td>
<td>Lower respiratory infections: pneumonia*</td>
<td>Road traffic injuries</td>
<td>Fires*</td>
<td>Assault</td>
</tr>
<tr>
<td>Respiratory disorders</td>
<td>Diabetes mellitus*</td>
<td>Alcohol poisonings</td>
<td>Drownings</td>
<td>Other unintentional injuries</td>
</tr>
</tbody>
</table>

Table 1. Alcohol-related conditions included in the study, and whether the effect is detrimental (-), is protective (+), or varies by subgroup (+/-)

<table>
<thead>
<tr>
<th>Cancers</th>
<th>Conditions arising during pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouth and oropharyngeal cancers</td>
<td></td>
</tr>
<tr>
<td>Oesophagus cancer</td>
<td></td>
</tr>
<tr>
<td>Colon cancer</td>
<td></td>
</tr>
<tr>
<td>Rectum cancer*</td>
<td></td>
</tr>
<tr>
<td>Liver cancer</td>
<td></td>
</tr>
<tr>
<td>Laryngeal cancer</td>
<td></td>
</tr>
<tr>
<td>Female breast cancer</td>
<td></td>
</tr>
</tbody>
</table>

| Neuro-psychiatric disorders      |                                     |
| Alcohol use disorders            |                                     |
| Unipolar depressive disorders    |                                     |
| Epilepsy                         |                                     |

| Cardiovascular disorders         |                                     |
| Hypertensive heart disease       |                                     |
| Ischaemic heart disease          |                                     |
| Cardiac arrhythmias              |                                     |
| Ischaemic stroke                 |                                     |
| Haemorrhagic stroke              |                                     |

| Digestive disorders              |                                     |
| Oesophageal varices              |                                     |
| Alcoholic liver cirrhosis        |                                     |
| Cholelithiasis                   |                                     |
| Pancreatitis                     |                                     |

*New category added since the NZBoA2000/02 report.
**Results**

**Mortality**

We estimated that 5.4% of all deaths under 80 years of age were attributable to alcohol in 2007 (802 deaths). Figure 1 shows the broad causes of these deaths, and the comparison between men and women.

Overall, 43% of deaths were due to injuries, including unintentional injury deaths as well as intentional injury (predominantly suicide but also homicide). Most of the difference between male and female mortality was made up of injury deaths. Thirty percent of alcohol-attributable deaths were from cancers, and 27% were from a variety of other chronic diseases and pneumonia.

**Figure 1. Alcohol-attributable deaths under 80 years of age, by sex and cause, 2007**

There were marked disparities in mortality by sex and ethnicity. Twice as many deaths were seen in men as women, for both Māori and non-Māori, and the age-standardised death rate for Māori was approximately two and half times the rate for non-Māori (Table 2). In addition to this, the number of deaths estimated to have been prevented through reduction in chronic disease mortality among older people did not have as much benefit for Māori as non-Māori, widening the gap further.
Table 2. Alcohol-attributable deaths caused and prevented, by sex and ethnicity (0–79 years; 2007)

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Deaths caused (count)</th>
<th>% of all deaths</th>
<th>Deaths caused (rate*)</th>
<th>Deaths prevented</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>124</td>
<td>8.3%</td>
<td>46.5</td>
<td>28</td>
</tr>
<tr>
<td>Non-Māori</td>
<td>414</td>
<td>5.7%</td>
<td>19.0</td>
<td>198</td>
</tr>
<tr>
<td>Total</td>
<td>537</td>
<td>6.1%</td>
<td>22.7</td>
<td>226</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>62</td>
<td>5.7%</td>
<td>22.1</td>
<td>14</td>
</tr>
<tr>
<td>Non-Māori</td>
<td>203</td>
<td>4.0%</td>
<td>8.1</td>
<td>111</td>
</tr>
<tr>
<td>Total</td>
<td>265</td>
<td>4.3%</td>
<td>9.7</td>
<td>125</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>185</td>
<td>7.2%</td>
<td>33.8</td>
<td>42</td>
</tr>
<tr>
<td>Non-Māori</td>
<td>617</td>
<td>5.0%</td>
<td>13.5</td>
<td>309</td>
</tr>
<tr>
<td>Total</td>
<td>802</td>
<td>5.4%</td>
<td>16.1</td>
<td>351</td>
</tr>
</tbody>
</table>

* Rate per 100,000 age-standardised to WHO world population

Causes of alcohol-related deaths varied by age. Figure 2 shows the predominance of injury as a cause of death amongst the young and the transition to chronic disease causes of death with age. This can be seen in more detail in Table 3 below.

Figure 2. Causes of alcohol-attributable deaths by age, 2007
Injuries were the dominant cause of alcohol-attributable deaths in young adults, with only injury categories included in the five leading causes of alcohol-attributable deaths in 15–29 year old males, 30–44 year old males, and 15–29 year old females (Table 3).

For these three young adult subgroups, road traffic injuries and self-inflicted injuries were the first and second specific leading causes of alcohol-attributable deaths, with road traffic injuries responsible for more than half of the alcohol-attributable deaths in 15–29 year old males (n=58) and females (n=14) and almost one-third of alcohol-attributable deaths in 30–44 year old males (n=33).

Breast cancer was a leading cause of alcohol-attributable deaths in women over 30 years old, while alcoholic liver cirrhosis was a common cause of alcohol-attributable deaths in women older than 30 years and men older than 45 years.

On a population basis, injury was the leading cause of alcohol-related death in New Zealand. However, the leading cause of alcohol-related death in both Māori and non-Māori women was breast cancer. The top five causes of premature deaths from alcohol consumption for Māori and non-Māori men and women are shown in Table 4.

The pattern varies between the four sex-ethnicity subgroups, with injuries being dominant among Māori men, and slightly less so in non-Māori men; breast cancer, road traffic injuries and heart disease among Māori women, and breast cancer and other chronic diseases in non-Māori women.
Table 3. Top five causes of alcohol-attributable (AA) deaths, by age and sex (2007)

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Males</th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% of all AA deaths</td>
<td>% of all deaths</td>
<td></td>
<td>% of all AA deaths</td>
<td>% of all deaths</td>
<td></td>
</tr>
<tr>
<td>15-29 years</td>
<td>n = 104</td>
<td>26.6%</td>
<td></td>
<td>15-29 years</td>
<td>n = 24</td>
<td>14.8%</td>
</tr>
<tr>
<td>Road traffic injuries</td>
<td>55.9%</td>
<td></td>
<td></td>
<td>Road traffic injuries</td>
<td>58.0%</td>
<td></td>
</tr>
<tr>
<td>Self-inflicted injuries</td>
<td>17.8%</td>
<td></td>
<td></td>
<td>Self-inflicted injuries</td>
<td>13.8%</td>
<td></td>
</tr>
<tr>
<td>Non-alcohol poisoning</td>
<td>6.0%</td>
<td></td>
<td></td>
<td>Violence</td>
<td>9.6%</td>
<td></td>
</tr>
<tr>
<td>Other unintentional injuries</td>
<td>5.6%</td>
<td></td>
<td></td>
<td>Other unintentional injuries</td>
<td>6.0%</td>
<td></td>
</tr>
<tr>
<td>Violence</td>
<td>4.6%</td>
<td></td>
<td></td>
<td>Non-alcohol poisoning</td>
<td>4.9%</td>
<td></td>
</tr>
<tr>
<td>30-44 years</td>
<td>n = 98</td>
<td>16.2%</td>
<td></td>
<td>30-44 years</td>
<td>n = 38</td>
<td>8.9%</td>
</tr>
<tr>
<td>Road traffic injuries</td>
<td>33.9%</td>
<td></td>
<td></td>
<td>Female breast cancer</td>
<td>22.8%</td>
<td></td>
</tr>
<tr>
<td>Self-inflicted injuries</td>
<td>22.8%</td>
<td></td>
<td></td>
<td>Road traffic injuries</td>
<td>15.4%</td>
<td></td>
</tr>
<tr>
<td>Other unintentional injuries</td>
<td>15.7%</td>
<td></td>
<td></td>
<td>Alcoholic liver cirrhosis</td>
<td>13.1%</td>
<td></td>
</tr>
<tr>
<td>Non-alcohol poisoning</td>
<td>5.2%</td>
<td></td>
<td></td>
<td>Self-inflicted injuries</td>
<td>12.1%</td>
<td></td>
</tr>
<tr>
<td>Drowning</td>
<td>4.1%</td>
<td></td>
<td></td>
<td>Haemorrhagic stroke</td>
<td>6.2%</td>
<td></td>
</tr>
<tr>
<td>45-59 years</td>
<td>n = 122</td>
<td>7.4%</td>
<td></td>
<td>45-59 years</td>
<td>n = 70</td>
<td>6.3%</td>
</tr>
<tr>
<td>Alcoholic liver cirrhosis</td>
<td>24.6%</td>
<td></td>
<td></td>
<td>Female breast cancer</td>
<td>38.8%</td>
<td></td>
</tr>
<tr>
<td>Other unintentional injuries</td>
<td>9.9%</td>
<td></td>
<td></td>
<td>Alcoholic liver cirrhosis</td>
<td>12.8%</td>
<td></td>
</tr>
<tr>
<td>Self-inflicted injuries</td>
<td>9.3%</td>
<td></td>
<td></td>
<td>Haemorrhagic stroke</td>
<td>8.2%</td>
<td></td>
</tr>
<tr>
<td>Mouth/oropharynx cancers</td>
<td>6.1%</td>
<td></td>
<td></td>
<td>Alcohol use disorders</td>
<td>4.3%</td>
<td></td>
</tr>
<tr>
<td>Liver cancer</td>
<td>6.0%</td>
<td></td>
<td></td>
<td>Self-inflicted injuries</td>
<td>4.2%</td>
<td></td>
</tr>
<tr>
<td>60-69 years</td>
<td>n = 98</td>
<td>4.6%</td>
<td></td>
<td>60-69 years</td>
<td>n = 57</td>
<td>3.8%</td>
</tr>
<tr>
<td>Alcoholic liver cirrhosis</td>
<td>22.5%</td>
<td></td>
<td></td>
<td>Female breast cancer</td>
<td>32.8%</td>
<td></td>
</tr>
<tr>
<td>Mouth/oropharynx cancers</td>
<td>12.6%</td>
<td></td>
<td></td>
<td>Alcoholic liver cirrhosis</td>
<td>19.4%</td>
<td></td>
</tr>
<tr>
<td>Oesophagus cancer</td>
<td>11.8%</td>
<td></td>
<td></td>
<td>Colon cancer</td>
<td>10.9%</td>
<td></td>
</tr>
<tr>
<td>Rectum cancer</td>
<td>7.1%</td>
<td></td>
<td></td>
<td>Haemorrhagic stroke</td>
<td>8.8%</td>
<td></td>
</tr>
<tr>
<td>Haemorrhagic stroke</td>
<td>6.7%</td>
<td></td>
<td></td>
<td>Rectum cancer</td>
<td>4.5%</td>
<td></td>
</tr>
<tr>
<td>70-79 years</td>
<td>n = 102</td>
<td>2.7%</td>
<td></td>
<td>70-79 years</td>
<td>n = 68</td>
<td>2.5%</td>
</tr>
<tr>
<td>Haemorrhagic stroke</td>
<td>15.4%</td>
<td></td>
<td></td>
<td>Female breast cancer</td>
<td>23.8%</td>
<td></td>
</tr>
<tr>
<td>Oesophagus cancer</td>
<td>14.4%</td>
<td></td>
<td></td>
<td>Haemorrhagic stroke</td>
<td>22.7%</td>
<td></td>
</tr>
<tr>
<td>Alcoholic liver cirrhosis</td>
<td>6.9%</td>
<td></td>
<td></td>
<td>Colon cancer</td>
<td>13.9%</td>
<td></td>
</tr>
<tr>
<td>Colon cancer</td>
<td>7.6%</td>
<td></td>
<td></td>
<td>Ischaemic heart disease</td>
<td>9.7%</td>
<td></td>
</tr>
<tr>
<td>Rectum cancer</td>
<td>7.2%</td>
<td></td>
<td></td>
<td>Rectum cancer</td>
<td>4.8%</td>
<td></td>
</tr>
</tbody>
</table>

Years of life lost (YLL)

Years of life lost incorporate the impact of age of death when summarising mortality. The 802 deaths described above resulted in 13,769 years of life lost (YLLs) due to alcohol in 2007. Due to the predominance of injury deaths among the young, injuries were the dominant cause of alcohol-attributable YLLs overall, making up 73% in men (n=7,066) and 42% in women (n=1,708).

Major sex and ethnic disparities in YLLs were seen (Figure 3), reflecting not only the differences in mortality between men and women, and Māori and non-Māori, but also the distribution of age at death in these groups.
Table 4. Top five causes of alcohol attributable (AA) deaths, by ethnicity and sex, 2007.

<table>
<thead>
<tr>
<th>Causes</th>
<th>% of AA deaths Māori (%)</th>
<th>% of AA deaths Non-Māori (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Road traffic injuries</td>
<td>32.1</td>
<td>15.8</td>
</tr>
<tr>
<td>Other unintentional injuries</td>
<td>13.1</td>
<td>13.3</td>
</tr>
<tr>
<td>Self-inflicted injuries</td>
<td>10.2</td>
<td>10.6</td>
</tr>
<tr>
<td>Alcoholic liver cirrhosis</td>
<td>5.7</td>
<td>13.3</td>
</tr>
<tr>
<td>Drownings</td>
<td>5.6</td>
<td>7.6</td>
</tr>
</tbody>
</table>

Disability-adjusted life years (DALYs) lost

We calculated alcohol-attributable DALYs lost for the year 2004, the most recent year for which the required data were available from the WHO Global Burden of Disease study. DALYs incorporate time affected by non-fatal conditions (as a proportion of a healthy year of life lost), as well as premature deaths, to give a measure of overall health burden. Disaggregation by ethnicity was not possible with the WHO data.
Overall, 28,403 years of healthy life (DALYs) lost by New Zealanders under 80 years old in 2004 were attributed to alcohol, representing 6.5% of all DALYs lost. Alcohol consumption was estimated to have prevented the loss of 6,538 DALYs.

The DALY burden in men (n=18,803; 8.5% of all DALYs) was almost twice that in women (n=9,601; 4.3% of all DALYs). Alcohol use disorders were the biggest cause group for both men and women. Approximately 12% of DALYs lost in women were due to breast cancer.

**Table 5. Top five causes of alcohol attributable disability-adjusted life years (AA DALYs) lost under 80 years of age, 2004**

<table>
<thead>
<tr>
<th></th>
<th>% of AA DALYs</th>
<th></th>
<th>% of AA DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td><strong>Females</strong></td>
<td></td>
</tr>
<tr>
<td>Alcohol use disorders</td>
<td>42.5%</td>
<td>Alcohol use disorders</td>
<td>50.0%</td>
</tr>
<tr>
<td>Road traffic injuries</td>
<td>18.8%</td>
<td>Female breast cancer</td>
<td>12.2%</td>
</tr>
<tr>
<td>Self-inflicted injuries</td>
<td>7.8%</td>
<td>Road traffic injuries</td>
<td>10.3%</td>
</tr>
<tr>
<td>Other unintentional injuries</td>
<td>6.5%</td>
<td>Cirrhosis of the liver</td>
<td>4.3%</td>
</tr>
<tr>
<td>Cirrhosis of the liver</td>
<td>3.8%</td>
<td>Other unintentional injuries</td>
<td>3.5%</td>
</tr>
</tbody>
</table>
framework uses AAFs calculated as if the health consequences were immediate, that is, current consumption is used as an indicator of consumption at the appropriate time in the causal pathway. While this is reasonable for injury and for much chronic disease, it is not so for cancer consumption where the previous two decades may be implicated.

We endeavoured to take an approach consistent with the Treaty of Waitangi by conducting separate analyses for Māori and non-Māori where possible. However, the data required to conduct an analysis of DALYs separately for Māori and non-Māori were not available. In common with the previous study, detailed NZ data on risk relationships are not available except in road traffic injury, and the extrapolation from other populations to Māori may be less appropriate than for non-Māori.

We have restricted this analysis to New Zealanders under 80 years of age, due to concerns about the validity of relative risk estimates and consumption survey data for older people, particularly Māori. Therefore the mortality findings may be best interpreted as premature deaths due to alcohol. The changes made to methods and to the range of conditions included mean this study should be seen as a stand-alone assessment, and should not be compared with the previous study as an indication of change over time.

The recently completed global assessment of the burden of alcohol (GBD2010) found that alcohol is the fifth most important risk factor for death and disability, accounting for 3.9% of global DALYs. It was ranked third for men after high blood pressure and smoking, and twelfth for women, and accounted for 2.7 million deaths globally in 2010.

The first Australian study using similar methods was based on the year 2010, and found that 5.5% of DALYS lost in men and 2.4% of DALYs lost in women were attributable to alcohol, compared with net DALYs lost of 7.2% for men and 2.8% for women in New Zealand in 2004.

This paper has placed less emphasis on health benefits than harms from alcohol. There are two main reasons for this. The first is that the epidemiological evidence for benefits is less compelling. Over time, more sophisticated estimation of risk relations between alcohol and cardiovascular disease have demonstrated that benefit is confined to a relatively small number of subgroups of the population. The contention that observed benefits could be an artefact of the epidemiological methods used has also received support recently with the publication of a Mendelian randomisation study of alcohol and cardiovascular disease which suggests no benefit from drinking even at low levels. However, the study leaves unanswered questions, and Rehm and Roerecke have suggested that given the contradictions in the evidence “we should be very cautious in placing too much weight on the IHD effects in policy formulation.”

The second reason, which is more relevant to the public health response, is that evidence of benefit doesn’t change advice to individuals or healthy policy on alcohol. Alcohol could never be (ethically) promoted for its health benefits now that the harms are so well documented, e.g. increasing cancer risk even among non-heavy drinkers, the risk of addiction leading to persistence of heavy use, toxicity to the unborn, and the considerable social burden that heavy drinking has become. Reduction in the risk of cardiovascular disease can be achieved more effectively with the use of medications with better safety profiles than alcohol, if lifestyle change is insufficient.

The burden of harm described here is due both to the properties of alcohol and to the pattern of alcohol drinking in New Zealand. An appropriate public health response requires evidence-based interventions that reduce consumption across the whole population, and that do not further increase health disparities.

Key strategies have been laid out in the World Health Organization’s Global strategy to reduce harmful use of alcohol ratified in 2010, and a detailed set of recommendations tailored for the New Zealand context.
Zealand context were put forward by the New Zealand Law Commission at the conclusion of their extensive review in 2010.11

Interventions that have been shown to be effective and cost-effective in reducing harm are population-wide policies that increase alcohol prices, reduce availability and control marketing, along with specific traffic injury countermeasures such as lowering the drink-driving limit.29,30

Competing interests: Nil.

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Datasets and estimates were provided by: Centre for Social and Health Outcomes Research and Evaluation (2003/04 Health Behaviours Survey – Alcohol Use; 2008/09 Alcohol’s Harm to Others Survey); Ministry of Health (2007/08 New Zealand Alcohol and Drug Use Survey; 2006/07 New Zealand Health Survey; 2007 and 2004 New Zealand mortality data; Te Rau Hinengaro: 2003/04 New Zealand Mental Health Survey); and the Global Burden of Disease 2010 Risk Factors Collaborating Group (relative risks for alcohol-attributable conditions; modelled injury alcohol-attributable fractions; YLD estimates).

This study was commissioned with a project grant from the Alcohol Advisory Council of New Zealand.

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References


Motor neurone disease (MND) is a neurodegenerative condition that leads to progressive muscle weakness, cumulative disability and eventual death. The incidence of MND in Europe and North America is approximately 1.89/100,000.\(^1\) It has a relentlessly progressive course, and has a 5-year survival rate of 23%.\(^4\) There are multiple subtypes of MND, with amyotrophic lateral sclerosis (ALS) being the most common form.

The clinical features include manifestations of bulbar muscle weakness with dysarthria and dysphagia, limb weakness and respiratory muscle weakness as well as cognitive impairment.\(^5\) The hallmark of classic ALS is a combination of upper and lower motor neuron signs on examination, although other less common subtypes of MND present with signs of predominantly either upper or lower motor neurone disease.

The disease has been reported to be familial in approximately 5% of cases,\(^5\) and sporadic in the remainder. Cognitive impairment is found in many patients with MND, with executive dysfunction and frontotemporal dementia which may be present in up to 50% and 25% of all cases respectively.\(^7,8\)

The pseudobulbar affect is also a recognised manifestation in affected individuals, although it is not specific to the condition and is also found in patients with multiple sclerosis, stroke and traumatic brain injury. It consists of uncontrollable display of emotions such as laughter or crying and affects up to half of MND patients.\(^8\)

Our aim was to review the disease characteristics and multidisciplinary care of MND patients in the greater Wellington region over a 12-month period. This data was then compared to established guidelines\(^16\) to determine if present practice matched expected levels of care.
Method

This was an observational and descriptive study using data from clinical records. The district health board protocol for ethics approval was followed, and no ethics committee approval was required for this study given it consisted of collecting observational information in the form of an audit, with no patient contact or interventions carried out.

Patients with a diagnosis MND were identified using hospital clinical coding data at Hutt Valley Health and Capital and Coast District Health Boards, which included presentations to Wellington Regional, Hutt and Kenepuru Hospitals. We reviewed all outpatient visits as well as inpatient encounters between 1 June 2011 and 31 May 2012.

In addition to this, the MND field workers for the Wellington and Hutt Valley regions were contacted to provide registries of all known patients with MND during this 12-month period, and any additional patients not identified via clinical coding information were added to the study group. No patients or families were contacted as part of the study.

The following data was collected: Age at diagnosis, sex, ethnicity, source of referral, clinical manifestation of MND at presentation (i.e. predominantly bulbar, limb or respiratory involvement), MND subtype, family history of MND, presence of cognitive impairment, and use of the disease-modifying drug riluzole (Rilutek).

Survival data was collected at 24 months following the end of the study period, in June 2014. This was done by calculating the time from onset of symptoms to death for each deceased patient. Aspects of community health care provision for MND patients that were evaluated included the number of patients who received respiratory assessment and non-invasive ventilation, percutaneous endoscopic gastrostomy tube placement, speech language therapist and dietitian input, and palliative care input.

Results

A total of 40 patients were identified as having a diagnosis of MND during the study period in the greater Wellington region. Twenty-eight of these were from the Capital and Coast District Health Board catchment zone and 12 from Hutt Valley Health.

The mean age at diagnosis was 66.2 years (SD of 13.2) and the median was 67 years, with a range of 27–94 years. There were 22 males and 18 females. The 12-month prevalence of MND in the Wellington region based on this data and the most recent census is 8.5 cases per 100,000.

The breakdown on subtype of MND was as follows: 35 patients had amyotrophic lateral sclerosis (ALS), three patients had primary lateral sclerosis (PLS), one had progressive muscular atrophy and one had progressive bulbar palsy.
Table 1. Demographic and clinical characteristics of MND patients in the Wellington region

<table>
<thead>
<tr>
<th>Age interval (years)</th>
<th>N (%)</th>
<th>Ethnicity</th>
<th>N (%)</th>
<th>Clinical manifestation</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21–30</td>
<td>1 (2.5)</td>
<td>NZ European</td>
<td>30 (75)</td>
<td>Limb involvement</td>
<td>12 (30)</td>
</tr>
<tr>
<td>31–40</td>
<td>2 (5)</td>
<td>Maori</td>
<td>2 (5)</td>
<td>Bulbar involvement</td>
<td>11 (27.5)</td>
</tr>
<tr>
<td>41–50</td>
<td>1 (2.5)</td>
<td>Asian</td>
<td>2 (5)</td>
<td>Mixed limb and bulbar</td>
<td>14 (35)</td>
</tr>
<tr>
<td>51–60</td>
<td>5 (12.5)</td>
<td>Pacific Islander</td>
<td>1 (2.5)</td>
<td>Limb with respiratory involvement</td>
<td>1 (2.5)</td>
</tr>
<tr>
<td>61–70</td>
<td>20 (50)</td>
<td>Other European</td>
<td>5 (12.5)</td>
<td>Bulbar with respiratory involvement</td>
<td>1 (2.5)</td>
</tr>
<tr>
<td>71–80</td>
<td>6 (15)</td>
<td></td>
<td></td>
<td>Isolated respiratory involvement</td>
<td>1 (2.5)</td>
</tr>
<tr>
<td>81–90</td>
<td>4 (10)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>91–100</td>
<td>1 (2.5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The referral sources to neurology in this group of patients were from the patient’s general practitioner 40% (16/40), hospital physician 27% (11/40), other hospital team 8% (3/40) and unknown 25% (10/40).

Hospital physician referrals consisted of six from general Medicine, four from geriatric services, and one from rheumatology. Other hospital services referring were orthopaedics (two) and ENT (one). Of the unknown group, some patients were transferred to the public hospital service from private Neurology consultations and did not have any referral on record.

Fronto-temporal dementia was diagnosed in one of the 40 patients. Mild cognitive impairment was documented in a further three. Emotional lability with a pseudobulbar affect was noted to be present in 7 patients, with 5 of these having bulbar predominant disease.

A positive family history of MND was noted in 22% (4 of 18 patients) in whom a documented enquiry was found. This is higher than expected but is likely to represent a reporting bias in the subgroup.

Only 2 of the 40 patients in the cohort were on the medication riluzole. With review of the survival data at 24 months, we found that 32 patients in the cohort were deceased and the median survival from the time of symptom onset was 29 months (range 6-126 months). The mean survival time was 37.5 months (SD of 29.1).

Twenty-one patients received a Respiratory physician assessment with spirometry. A further six had spirometry only. Ten patients (25%) in total received non-invasive ventilation with Bi-level positive airway pressure (Bipap).

Dietitian input was recorded in 21 patients and speech language therapist (SLT) input was recorded in 27 of the 40 patients. All except three patients with bulbar disease were assessed by an SLT. A PEG tube was placed in 12 patients out of 26 with bulbar disease. A discussion regarding consideration of a PEG was documented in three further patients, two of whom declined the procedure and one died prior to tube placement. One patient with bulbar predominant disease received salivary gland Botulinum toxin injections for excessive salivation.

Palliative care services are provided by Mary Potter Hospice in the Capital and Coast region and Te Omanga Hospice in the Hutt Valley region. Twenty-four patients in total were on the respective hospice programmes, and documentation of a discussion regarding future referral was made in a further three.

Discussion

Our findings show that the peak prevalence of MND in this population is in the seventh decade with a male to female ratio of 1:1.2. This is consistent with data from other epidemiological studies overseas.\(^1\)-\(^3\),\(^9\),\(^13\)

We found a 12-month prevalence of 8.5 per 100,000 in the Wellington region, and although this is slightly higher than reported figures of 5-8 per 100,000 elsewhere,\(^2\),\(^3\) the point prevalences in these studies may not be directly comparable. Despite this, there is a suggestion from another study\(^20\) that the incidence rates of MND in New Zealand is high relative to overseas populations, and has been increasing over the last few decades.

The majority of patients had the ALS form of MND in this group and 35% presented with mixed limb and bulbar symptoms. Overall, 65% of patients had some bulbar symptoms at presentation. Bulbar presentation is associated with a worse prognosis than spinal onset disease\(^19\),\(^20\) and early identification of this patient subgroup is important in planning subsequent management.
Respiratory involvement in isolation was uncommon at 2.5%. Other studies looking at the first clinical manifestation of MND report bulbar onset in 15-25%, respiratory muscle weakness as the first manifestation in less than 3% and spinal or limb onset in the remainder, although one large study of MND phenotypes reported approximately equal incidences of classic and bulbar types, as we found in this cohort.

Our data also confirms that general practitioners were the single largest group of doctors who had first contact with MND patients and made the most referrals to the neurology service for a specialist opinion.

The median survival time of 29 months (2.4 years) for the 32 of the deceased patients on collection of data at 24 months may be influenced by a selection bias of excluding patients with relatively slowly progressive disease who tend to survive longer and did not have survival data available as they were still living at this follow up period. Nevertheless, this figure is similar to reported longevity in MND patients in larger studies, including one that reported data from a local Canterbury cohort.

Treatment options for MND are limited and riluzole is the only drug available that has any impact on survival, albeit with a modest effect of prolonging survival by 2–3 months. Riluzole has recently been approved for funding for MND patients by PHARMAC upon application via special authority, but was not funded during the period of the study and had a cost to the patient of $700 per month. This may explain the low utilisation rate in this cohort. It is possible that more MND patients would now opt to take the drug given it is subsidised.

The American Academy of Neurology (AAN) guidelines recommends respiratory function tests at diagnosis for all MND patients. Almost seventy percent of patients in this cohort had spirometry assessments early after their initial diagnosis. This could be improved with the goal of aiming to objectively assess respiratory function in the remaining patients. However, some of these patients had significant bulbar dysfunction which would preclude them from the use of non-invasive ventilation and may explain why respiratory function tests were not performed.

A PEG tube placement or a documented discussion about future insertion was carried out in over half of patients in the cohort with bulbar dysfunction. The AAN guidelines recommend that a PEG be offered to all patients with symptomatic dysphagia, and that the optimal efficacy and safety of the procedure is when the vital capacity on spirometry is greater than 50% predicted.

Overall, while there remains some room for improvement, it appears that the hospital and community MND services within the Wellington and Hutt Valley regions are managing to provide a comprehensive service when compared to international guidelines, for these patients who have a very challenging set of physical and emotional problems.

Competing interests: Nil.

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References


Does Pūkawakawa (the regional-rural programme at the University of Auckland) influence workforce choice?

Christina Matthews, Warwick Bagg, Jill Yielder, Vernon Mogol, Phillippa Poole

Abstract

Aims: Relative shortages of rural doctors persist. In 2008 the University of Auckland medical programme introduced a Year 5 regional and rural immersion programme, Pūkawakawa, based in Northland, New Zealand (NZ). This study evaluates the early workforce outcomes of graduates of this programme.

Methods: During 2013 we surveyed Auckland medical graduates who were in the 2008–2011 Pūkawakawa cohorts. Questions were asked regarding recent and current place of work, future intentions for place of work, and career preference with reasons why. Qualitative analysis was undertaken to analyse free text responses about experiences of Pūkawakawa on this choice.

Results: Of the 72 Pūkawakawa participants, 45 completed the survey, for a response rate of 63%. In 2013, 62% were working in rural or regional areas, with 31% in the Northland DHB. The great majority intend to work rurally or regionally, with 35.6% intending to return to Northland DHB. Of the respondents, 68% listed general practice in their top three future career intentions.

Conclusions: In the early postgraduate years, medical graduates who participated in Pūkawakawa are very likely to be working in rural and regional areas. These graduates also show an intention to work in general practice and rural medicine.

There is a mal-distribution of doctors in New Zealand (NZ) with too few doctors working in regional and rural areas. The shortage of rural health professionals in NZ contributes to disadvantage and disparity in health status, health infrastructure and economic vitality.1,2

The 2012 New Zealand Medical Council workforce survey identified that rural areas, as defined by less than 20 people per square kilometre, have less doctors and general practitioners per population and the doctors’ average age is higher at 48.3 years compared to 44.8 years in urban areas.7 To meet their social contract, medical programmes are introducing measures designed specifically to enhance student interest in practising in regional or rural areas.

Several factors are known to predict which students will eventually practice rurally. The strongest of these is the impact of place of birth on future practice.4–7 Students who have lived and worked in regional / rural areas are more likely to practice in rural areas.4,8–16 This finding underpins the presence of dedicated entry pathways for rural students into medicine in NZ for over a decade. In terms of curriculum, there is an effect of rural exposure, with this shown to be stronger with prolonged attachments and within clinical years of training.13

A systematic review by Laven and Wilkinson found four out of five studies showed rural undergraduate training to be associated with rural practice, with a typical odds ratio of approximately 2.0.17 However, the relative contributors to long-term rural work place choice remain unclear, largely due to the failure to adjust for critical independent predictors of rural practice.16

Northland forms the most northern part of NZ. It has a population of 151,68918 and, based on the deprivation index, 35% of the population are in the lowest quintile compared with 20% of the total population of NZ.19 The Pūkawakawa programme is a partnership between the University of...
Auckland, Northland District Health Board and Hokianga Health. Up to 24 Year 5 medical students live and learn in Northland for most of their penultimate year of study. The majority of clinical placements are undertaken at Whangarei Hospital, with one 7-week general practice and integrated care placement in Kawakawa, Kaitaia, Rawene or Dargaville.

To be selected for the Pūkawakawa programme, students must have sound academic standing, submit a written application and go through an interview process. There are four entry pathways into the medical programme: general admission; international admission; the Māori and Pacific Admission Scheme (MAPAS) and the Rural Origin Medical Preferential Entry (ROMPE). Students in the two latter categories receive preference for Pūkawakawa.

The aim of the study was to evaluate the early outcomes of the University of Auckland rural-regional placement, Pūkawakawa, on location of practice and future career intentions. Additionally, we explored the reasons for career choices and whether or not Pūkawakawa has had an effect on students’ choices and consequently New Zealand’s workforce.

Methods

Participants—Between 2008 and 2011, a total of 78 students participated in Pūkawakawa. Of these 27 were ROMPE, 27 MAPAS, 23 general entry students and one international. Six students had not yet graduated, or started work after graduation. One student’s contact information was marked confidential on the Medical Council’s database. A survey link was sent by mail using the address on the Medical Council of New Zealand database and followed up through the University of Auckland database, by email and letter. 72 graduates were sent questionnaires.

The Survey—Survey questions (n = 36) examined a range of factors that are known predictors of future entry into the rural medical workforce, as well as questions regarding graduates’ workforce journey, the reasons for workforce choices and future intentions. The anonymous survey was conducted using Survey Gizmo. Free text responses to three questions were gathered to explore the reasons for choices made and to explore future intentions. Ethics approval was granted by the University of Auckland Human Participants Ethics Committee.

Definitions—Participants self-identified whether they had a rural or regional origin, or an urban origin. The place of work was defined by hospital or practice location within the DHB regions of New Zealand. Regional or rural areas are defined in this paper according to the parameters of the recent Regional Rural Admission Scheme at the University of Auckland. Thus, any DHBs largely outside of Auckland, Hamilton, Tauranga, Wellington, Porirua, Hutt, Upper Hutt, Christchurch or Dunedin City Councils are considered rural or regional.

The first year since graduation from the medical programme is denoted PGY1, the second, PGY2 and so on.

Analysis—Quantitative data were collected and analysed for summary statistics using Excel. A chi-square test was used for datasets with over 80% of the variables being over 5. The level of significance was set at a p < 0.05. We analysed whether there was an effect by entry pathway into the medical programme.

A dichotomous variable was created to classify the respondents’ preference for working at either an urban (0) or rural/regional hospital (DHB) (1). Phi correlation was computed to examine which of the possible 22 factors influencing intended future place of work have a relationship with preference for working in a rural/regional hospital versus an urban hospital. Similarly, respondents who selected regional and rural medicine or GP as one of their first three choices were assigned (1), whereas those who did not were assigned (0). Phi correlation was computed to examine the 25 possible factors affecting choice for future specialty training in regional and rural medicine or general practice versus all other specialties.

The qualitative data were analysed by two of the researchers independently, using a process of cross-sectional thematic analysis. Themes were established for each of the three open-ended questions:

1. How did Pūkawakawa affect views about regional/rural practice?
2. How do you think the participation in the Pūkawakawa scheme affected your career choices?
3. What were your best experiences of Pūkawakawa?
The themes have been included in the results and comments integrated into the discussion section of this article.

**Results**

The response rate was 62.5% (45/72). The respondents were representative of the whole cohort and similar across cohorts and entry pathways (Table 1). Over two-thirds of Pūkawakawa participants had entered the medical programme by the Rural Origin Medical Preferential Entry (ROMPE) or Māori and Pacific Admission Scheme (MAPAS) pathways.

**Table 1. Participants in Pūkawakawa by year and entry route into the medical programme. The numbers of responders are brackets**

<table>
<thead>
<tr>
<th>Variables</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>6 (1)</td>
<td>6 (5)</td>
<td>4 (3)</td>
<td>7 (5)</td>
<td>23 (14)</td>
</tr>
<tr>
<td>MAPAS</td>
<td>5 (3)</td>
<td>7 (5)</td>
<td>8 (4)</td>
<td>7 (3)</td>
<td>27 (15)</td>
</tr>
<tr>
<td>ROMPE</td>
<td>8 (3)</td>
<td>7 (4)</td>
<td>7 (4)</td>
<td>5 (5)</td>
<td>27 (16)</td>
</tr>
<tr>
<td>International</td>
<td>1 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Total</td>
<td>20 (7)</td>
<td>20 (14)</td>
<td>19 (11)</td>
<td>19 (13)</td>
<td>78 (45)</td>
</tr>
</tbody>
</table>

In 2013, 62% of the Pūkawakawa graduates were working in regional or rural hospitals compared to urban DHBs (Table 2). Of the respondents, 31% were working in Northland DHB, 16% Counties Manukau DHB and 18% in Lakes DHB at the time of the study.

Since graduation, the highest proportion of Pūkawakawa graduates have been working in the Northland DHB; PGY1 (45 responses), PGY2 (33 responses) or PGY3 (22 responses). These figures are followed closely by Counties Manukau DHB and Lakes DHB (Table 3).

Of those working in Northland DHB, 93% reported their experience there as a Pūkawakawa medical student affected their choice of current place of work; 79% cited the opportunity to do more hands on work at that site; and 71% identified that hobbies in the area and the atmosphere/work culture affected their current place of work.

**Table 2. Place of work in 2013 and intention to work for the 45 study participants, by entry pathway**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Current place of work</th>
<th>Intention to work</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Urban</td>
<td>Regional/Rural</td>
</tr>
<tr>
<td>Entry pathway</td>
<td></td>
<td></td>
</tr>
<tr>
<td>General (n=14)</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>MAPAS (n=15)</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>ROMPE (n=16)</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>17 (38%)</td>
<td>28 (62%)</td>
</tr>
</tbody>
</table>
### Table 3. Employment history by DHB in the early postgraduate (PG) years

<table>
<thead>
<tr>
<th>DHB</th>
<th>PGY1 n (%)</th>
<th>PGY2 n (%)</th>
<th>PGY3 n (%)</th>
<th>PGY4 n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>13 (28.9%)</td>
<td>9 (27.3%)</td>
<td>5 (22.7%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Waitemata</td>
<td>3 (6.7%)</td>
<td>1 (3.0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Auckland</td>
<td>3 (6.7%)</td>
<td>4 (12.1%)</td>
<td>1 (4.6%)</td>
<td>1 (10%)</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>7 (15.6%)</td>
<td>5 (15.2%)</td>
<td>5 (22.7%)</td>
<td>3 (30%)</td>
</tr>
<tr>
<td>Waikato</td>
<td>5 (11.1%)</td>
<td>4 (12.1%)</td>
<td>3 (13.6%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Lakes</td>
<td>6 (13.3%)</td>
<td>5 (15.2%)</td>
<td>4 (18.2%)</td>
<td>2 (20%)</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>2 (4.4%)</td>
<td>1 (3.0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Taranaki</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (4.6%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Whanganui</td>
<td>0 (0%)</td>
<td>1 (3.0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Capital and Coast</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (10%)</td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>1 (2.2%)</td>
<td>1 (3.0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Canterbury</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>2 (20%)</td>
</tr>
<tr>
<td>Overseas</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (4.6%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

The Pūkawakawa graduates were asked to identify their intentions for future work and career choice. When they identified in which DHB they would most like to work, 35.6% identified Northland DHB, with Lakes DHB identified by a further 20% of participants (Table 4). Within DHB regions, graduates were asked to specify which hospital they intended to work in; overall 80% of the graduates intend to work in a rural or regional hospital (Table 4).

### Table 4. Intended future DHB region

<table>
<thead>
<tr>
<th>DHB region</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>16 (35.6)</td>
</tr>
<tr>
<td>Auckland</td>
<td>2 (4.4)</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>3 (6.7)</td>
</tr>
<tr>
<td>Waikato</td>
<td>3 (6.7)*</td>
</tr>
<tr>
<td>Lakes</td>
<td>9 (20)</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>3 (6.7)</td>
</tr>
<tr>
<td>Taranaki</td>
<td>2 (4.4)</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>2 (4.4)</td>
</tr>
<tr>
<td>Hawke’s Bay</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>2 (4.4)</td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Southern</td>
<td>1 (2.2)</td>
</tr>
</tbody>
</table>

*Waikato DHB included Waikato Hospital (1), considered urban; and Thames hospital (1) and rural general practices (1), considered rural/regional.

In terms of place of work, there were significant subgroup differences in the rating of 22 influencing factors. Those who intend to work at a rural/regional hospital cited the hours of work (phi = 0.34, p<0.05) and the types of patients (phi = 0.36, p<0.05) as important. On the other hand, those who intend to work at an urban hospital indicated that prestige (phi = -0.47, p<0.1), teaching at the hospital (phi = -0.4, p<0.1), and the ability to do research (phi = -0.32, p<0.05) influenced their choice.
Students ranked their top three areas of future specialisation (Table 5). Surgery was the most popular first choice, with General Practice the most popular overall, with 68% of respondents listing it as one of their top three choices.

Table 5. Intended future medical discipline of Pūkawakawa graduates

<table>
<thead>
<tr>
<th>First choice</th>
<th>Second choice</th>
<th>Third choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery</td>
<td>General practice</td>
<td>General practice</td>
</tr>
<tr>
<td>General practice</td>
<td>Emergency medicine</td>
<td>Pediatrics</td>
</tr>
<tr>
<td>Paediatrics</td>
<td>Internal medicine</td>
<td>Rural and remote medicine</td>
</tr>
<tr>
<td>Internal medicine</td>
<td>Rural and remote medicine</td>
<td>Internal medicine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Emergency medicine</td>
</tr>
<tr>
<td></td>
<td>18%</td>
<td>33%</td>
</tr>
<tr>
<td></td>
<td>15%</td>
<td>9%</td>
</tr>
<tr>
<td></td>
<td>13%</td>
<td>9%</td>
</tr>
<tr>
<td></td>
<td>13%</td>
<td>7%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There were significant differences in responses to the 26 potential reasons for intended specialty between those planning to work in rural and remote medicine or general practice and those who are not. We combined these two pathways, as they are both areas of workforce need in New Zealand.

Hours of work (\( \phi = 0.33, p<0.05 \)), ability for flexible hours (\( \phi = 0.3, p<0.5 \)), location (\( \phi = 0.5, p<0.001 \)) and hobbies in the area (\( \phi = 0.34, p<0.05 \)) were important to the former, and experience in that specialty (\( \phi = -0.33, p<0.05 \)), and ability to do future research (\( \phi = -42, p<0.01 \)) important to the latter.

Qualitative results—Respondent comments about how their Pūkawakawa experience affected their views about regional/rural practice focused on five primary themes, shown below in order of decreasing frequency:

- Confirmed and consolidated their views on desire to work in a regional/rural setting (20).
- Demonstrated the positive aspects of work-life balance and lifestyle (17).
- Changed their views to a positive consideration of future regional/rural work (15).
- Demonstrated the positive aspects of teamwork in a collegial and supportive work environment (9).
- Helped them to appreciate sociological determinants of health (3).

Comments about whether participation in the Pūkawakawa programme affected career choices focused on four themes:

- Working in a rural hospital (14).
- Working as a rural GP (8).
- Didn’t have an influence (5).
- Two individual comments indicated influencing a decision to work in ED, and to general rather than sub-specialty work.
Finally, five themes emerged regarding the best experiences in the Pūkawakawa programme:

- Being part of the hospital team with collegial relationships (21).
- The social aspects experienced with peers, the hospital and community (18).
- Lifestyle factors (15).
- The learning experience (14).
- Involvement in community and sense of belonging (9).

Discussion

The results of this study have indicated that there is a benefit to the health system with possible changes to workforce distribution of graduating medical students. There is a change in the Pūkawakawa students’ views of regional/rural practice and future career choice.

A large proportion of the medical graduates who participated in Pūkawakawa in the years 2008–2011 are currently working or intend to work in rural or regional areas in New Zealand. The response rate at 62.5% is adequate to be confident that the data collected is valid and representative of the student population who were part of Pūkawakawa.

In the literature, the link of future rural practice to rural origin is clear and further is highlighted in this study, with ROMPE students most strongly associated with current and intended work in rural/regional areas. However, we noted a high retention in rural and regional DHBs throughout all entry pathways.

It is encouraging that the general entry students have also chosen to work in rural/regional areas; with 64% currently employed and 64.3% intending to in the future, compared to the 80% intention of all of the total Pūkawakawa graduates. This suggests a different pattern to where clinicians are working in New Zealand.

Students confirm the beneficial aspects of their experience in Pūkawakawa. The most obvious theme to come out of our analysis was the consolidation of views of rural and regional practice, but some also commented that it changed their view positively to working outside urban areas.

The positive experiences relate to the work-life balance, collegial relationships and communal living that encompass the Pūkawakawa experience. Students see Pūkawakawa as a unique experience and “enjoyed the atmosphere and support of a smaller hospital.” These points need to guide the development of future medical student placements in rural/regional areas to maximise the positive experience. Interestingly none of the open answers resulted in negative viewpoints on the programme.

There is clear evidence from the analysis of the data that the Northland DHB is a popular workforce choice for graduates of Pūkawakawa. Of these currently working in Northland DHB, the most cited reason was experience there as a medical student. Majority of the respondents intend to work in a rural or regional area. These findings add to the literature that living and working in a rural area increases future intention to work in a rural area.

An interesting theme that came out of the analysis was the appreciation of sociological determinants of health which links to the types of patients with whom graduates will work. Some comments from students about Pūkawakawa included: “It showed me the personal rewards of serving a population with poorer access to health services than other New Zealanders” and it “made me more committed to the health of rural and indigenous populations.”
Overall the receptivity of the community and health workforce allowed a positive experience of Northland and encouraged them to return. They were also able to gain a deeper understanding of cultural aspects that play a part in a patient’s health and wellbeing.

The communities they are working in is important to the graduates and developing a relationship and understanding of what faces Northland may encourage them to return: “Encounters with patients from the Hokianga in Whangarei Hospital were a highlight—as these were profoundly more rewarding because I could identify with places the patients were connected to.”

A connection with a place and community developed in medical school may be a factor for DHBs and universities to consider for the future.

**Limitations**—Students self-select to participate in the programme. In addition the University positively selects for ROMPE and MAPAS students to participate in the Pūkawakawa programme, thus there is a risk of selection bias. The selection process also includes an evaluation of a student’s likelihood of continuing in rural/regional medicine.

Moreover, some of the MAPAS students could be classed under the ROMPE criteria as well. We did not compare our findings to a control group of medical students—i.e. students of all entry pathways who did not complete rural undergraduate training. This may have helped to establish if there is a link of entry pathway to career and workplace choices.

Our study did not intend to split intentions for graduates to work in rural general practice versus urban general practice given the limited numbers that have come through the programme. We have no prior data on the cohort groups before exposure to the Pūkawakawa, therefore cannot accurately comment on change of intentions. At the very least, Pūkawakawa seem to be consolidating and maintaining rural and regional career intentions in these students, which might not be the case if they remained in urban settings.

Further areas of work are to evaluate the effect of rural placements on medical students with no intent to work rurally/regionally in the future, as well as to compare these results from those of students who did not have the opportunity to take Pūkawakawa. Longer-term follow-up, plus multivariate analysis may improve precision estimates of the important student characteristics that predict eventual practice in rural and regional areas.

Further research might include a comparison of academic performance over time between Pūkawakawa and other students to quantify whether there may be discernible differences in learning in this environment. This information will help to refine iteratively selection and educational policy for this programme.

At this stage no major changes are planned for the Pūkawakawa experience and we are seeking ways to replicate aspects in other regions.

**Conclusion**

In conclusion, this study demonstrates that in the short-mid term, Pūkawakawa’s workforce aims are being achieved. A large proportion of graduates are choosing to work in rural/regional areas. Encouragingly, the future career intention in general practice, and rural and remote medicine bode well for meeting the workforce need in New Zealand.

**Competing interests:** Nil.
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References


Prevalence predictions for age-related macular degeneration in New Zealand have implications for provision of healthcare services

David Worsley, Andrew Worsley

Abstract

Aim: To predict the prevalence of age-related macular degeneration (AMD) in New Zealand from 2014 through to 2026.

Method: Prevalence estimates for AMD in New Zealand for 2014 through to 2026 were generated by applying ethnic prevalence rate estimates for any, early and late AMD to New Zealand population projections for European, Māori, Pacific and Asian peoples.

Results: The prevalence of any AMD in New Zealand for the 45–85 year age group is estimated to be 184,400 in 2014 (10.3% of this age group) and increase 12.9% to 208,200 (9.9% of this age group) in 2026. For 2014 and 2026 respectively, early disease is estimated to be 167,500 and increase to 189,200 and late disease is estimated to be 7,600 and increase to 8,600.

Conclusion: The prevalence of AMD is expected to markedly increase from 2014 through 2026. New Zealand has the lowest funding of treatment for AMD in the OECD and a relatively low ophthalmic workforce. As such, there is a need to plan for an increasing demand for intervention strategies and associated ophthalmic services.

Age-related macular degeneration (AMD) is the leading cause of visual loss in individuals older than 50 years in New Zealand, as it is for the developed world as a whole.¹² Forty-nine percent of blind registrations in New Zealand are for AMD (Blind Foundation figures).

Early AMD is the presence of soft drusen in the macula.³ Soft drusen are accumulated extracellular material beneath the retina, seen clinically as small yellow spots. Late AMD takes two forms; geographic atrophy (GA) is loss of demarcated patches of retina, and neovascular AMD (nAMD) is growth of neovascular tissue beneath the retina, with secondary fluid leakage, bleeding and scar formation.

AMD is a disease of over 45 years of age and prevalence increases with age.² Late AMD is a feature of older age, with 10% of people over 80 years of age having late AMD.⁴ Significant vision loss occurs with late AMD.¹ This has widespread implications: reduced quality of life (QOL), other health issues such as hip fracture, depression and increased mortality and economic costs such as reduced income, treatment costs and increased need for care services.³

New Zealand is entering a period of demographic shift to an ageing population.⁶ This is due to two main factors.

Firstly, the European population is in transition from higher birthrate to lower fertility and mortality. Ageing of the European population is now moving from the under 65 year age group to the over 65 year age group. The largest increases in the over 65 age group will occur between 2020 and 2040 with ageing of the large birth cohorts of the 1950s and 1960s.⁵

Secondly, the Asian population, which was 9.7% of the total population in 2006, is estimated to be 15.8% by 2026, largely due to migration.⁷ The Asian over 65 year age group is projected to increase fivefold from 2006 to 2026 to be 11.2% of the Asian population.
Until a decade ago, AMD was largely untreatable. However, the finding that antioxidant therapy reduces progression to late AMD and anti-vascular endothelial growth factor (VEGF) agents are highly effective in nAMD has radically improved visual outcomes.\(^8\),\(^9\)

Progression to late AMD can be slowed with antioxidant therapy and vision loss from nAMD dramatically reduced with anti-VEGF therapy. Antioxidant and anti-VEGF therapy are cost-effective with robust health economic benefits.\(^3\),\(^5\) However, optimal anti-VEGF therapy requires 4–6 weekly ongoing intravitreal injections, resulting in a significant financial and logistical burden on healthcare systems.\(^10\)

The ageing of the population implies a rising prevalence of AMD with an associated treatment burden. Thus, best available estimates of prevalence are essential for healthcare planners to design and implement strategies to manage increasing need and to prevent avoidable vision loss.

This study aims to provide prevalence predictions for AMD in New Zealand for 2014 through to 2026.

**Methods**

**New Zealand ethnic population projections**—New Zealand ethnic population projections for each year from 2006 to 2026 were provided by Statistics New Zealand.\(^7\) These comprise 11 separate data series of population projections reflecting assumptions of mortality, fertility, ethnic mobility and migration (Table 1).

Series 6 is considered to be the most likely outcome. For this study we used series 1, 6 and 11 (respectively the lowest, mid and highest population projections) for each year 2014 through 2026.

**Table 1. Ethnic population projection series reflecting different assumptions sourced from Statistics New Zealand.**

<table>
<thead>
<tr>
<th>Assumptions</th>
<th>Series</th>
<th>Fertility</th>
<th>Mortality</th>
<th>Migration</th>
<th>Inter-ethnic mobility</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>low</td>
<td>high</td>
<td>low</td>
<td>high</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>medium</td>
<td>medium</td>
<td>medium</td>
<td>medium</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>high</td>
<td>low</td>
<td>high</td>
<td>low</td>
<td></td>
</tr>
</tbody>
</table>

Population projections made in 2010 are for ‘European or other (including New Zealander)’, ‘Māori’, ‘Asian’ and ‘Pacific’ peoples. ‘Other’ (Middle Eastern, Latin American and African), which make up about 1% of the population, are not large enough to alter a prevalence estimate. Therefore, we considered ‘European or other (including New Zealander)’ to be equivalent to ‘European’, and is referred to as ‘European’ throughout.

**Definitions of age-related macular degeneration**—Definitions are those used in the source publication of ethnic prevalences.\(^2\) In essence these are: ‘early AMD’, which is a minimum of either any soft drusen with pigment abnormalities or large soft drusen (125 micrometres or more in diameter), and ‘late AMD’, which is either GA or any features consistent with past or present nAMD. ‘Any AMD’ is the presence of either ‘early’ or ‘late’ AMD.

**Prevalence rates by ethnicity**—We sourced European and Asian prevalence rate estimates from the meta-analysis by Wong et al\(^2\) (Table 2). They estimated ethnic prevalence rates by applying hierarchical Bayesian approaches to pooled data from population-based studies of AMD that met strict quality criteria. Early, late, and any AMD data were pooled separately. Therefore, prevalence rate estimate for any AMD will not be equal to the sum of the prevalence rate estimates of early and
late AMD. Derived prevalence estimates were for an age range of 45–85 years, male and female combined.

We have used a zero prevalence of AMD for Māori and Pacific peoples as there is no published or anecdotal case of AMD in Māori or Pacific people. We conjecture that if the prevalence is higher than zero, it is likely to be very low and therefore inconsequential for this study.

Table 2. Estimated prevalence rates and credible intervals for early, late and any AMD by ethnicity. Sourced from Wong et al. †Bayesian credible interval.

<table>
<thead>
<tr>
<th>AMD Stage</th>
<th>Prevalence percentage estimate by ethnicity</th>
<th>Credible interval (95%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early AMD</td>
<td>Asian 6.81, 95% Crl†(3.14–13.94)</td>
<td>European 11.19, 95% Crl†(5.63–20.39)</td>
</tr>
<tr>
<td></td>
<td>Late AMD 0.37, 95% Crl†(0.17–0.85)</td>
<td>European 0.50, 95% Crl†(0.26–1.08)</td>
</tr>
<tr>
<td></td>
<td>Any AMD 7.38, 95% Crl†(3.40–14.46)</td>
<td>European 12.33, 95% Crl†(6.46–22.75)</td>
</tr>
</tbody>
</table>

New Zealand prevalence calculations—New Zealand prevalence projections for any, early and late AMD were calculated for each year from 2014 through to 2026 by applying the European and Asian ethnic prevalence rates for 45–85 year olds to the New Zealand European and Asian, aged 45–85 years, male and female combined population projections.

Of note, the 86 years and older age group is not included in the study as the source meta-analysis does not provide a prevalence rate for this age group. This is because of insufficient numbers in many epidemiology studies.”

Results

All estimates are using series 6, unless stated otherwise and are rounded to the nearest 100. This is a descriptive study and therefore no statistical tests have been applied.

New Zealand population estimates are in table 3. The 45–85 year age group is estimated to be 1,789,700 persons (39.1% of the population) in 2014 and 2,095,300 (41.1%) in 2026. Of note, the 86 years and older age group (estimated to be 68,400 persons in 2014 and 97,900 in 2026) is not included as AMD prevalence rates are not available.

Table 3. New Zealand population and ethnic subpopulations 2014 and 2026 in the 45–85 year age group (series 6).

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Number of persons (2014)</th>
<th>Number of persons (2026)</th>
<th>Percentage change 2014–2026</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>1,405,300 (30.7)</td>
<td>1,542,400 (30.3)</td>
<td>9.8</td>
</tr>
<tr>
<td>Asian</td>
<td>150,300 (3.3)</td>
<td>243,800 (4.8)</td>
<td>62.2</td>
</tr>
<tr>
<td>Māori</td>
<td>161,200 (3.5)</td>
<td>206,300 (4.0)</td>
<td>28</td>
</tr>
<tr>
<td>Pacific</td>
<td>73,000 (1.6)</td>
<td>103,000 (2.0)</td>
<td>41.1</td>
</tr>
<tr>
<td>Total</td>
<td>1,789,800 (39.1)</td>
<td>2,095,500 (41.1)</td>
<td>17.1</td>
</tr>
</tbody>
</table>

New Zealand AMD prevalence estimates for the 45–85 year age group for 2014 and 2026 are in Table 4. Prevalence of any AMD in this age group in 2014 is estimated to be 184,300 persons. Early AMD
is estimated to be 167,500 and late AMD 7,600 persons. In 2026 we estimate any, early and late AMD to be 208,200, 189,200 and 8,600 persons respectively.

Table 4. New Zealand prevalence estimates for any, early and late AMD in 45–85 year age group, for ethnic populations combined (European, Asian), European and Asian, and for series 6. Maori and Pacific are not entered as prevalence is zero. Numbers are rounded to the nearest 100

<table>
<thead>
<tr>
<th>AMD by ethnicity (series 6)</th>
<th>Number of persons</th>
<th>Percentage change: 2014–2026</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2014</td>
<td>2026</td>
</tr>
<tr>
<td>Combined any</td>
<td>184,400</td>
<td>208,200</td>
</tr>
<tr>
<td>European any</td>
<td>173,300</td>
<td>190,200</td>
</tr>
<tr>
<td>Asian any</td>
<td>11,100</td>
<td>18,000</td>
</tr>
<tr>
<td>Combined early</td>
<td>167,400</td>
<td>189,200</td>
</tr>
<tr>
<td>European early</td>
<td>157,200</td>
<td>172,600</td>
</tr>
<tr>
<td>Asian early</td>
<td>10,200</td>
<td>16,600</td>
</tr>
<tr>
<td>Combined late</td>
<td>7,600</td>
<td>8,600</td>
</tr>
<tr>
<td>European late</td>
<td>7,000</td>
<td>7,700</td>
</tr>
<tr>
<td>Asian late</td>
<td>600</td>
<td>900</td>
</tr>
</tbody>
</table>

Plots of any, early and late AMD prevalence for each year 2014 through to 2026, for combined European and Asian and European and Asian separately, are shown in Figures 1 to 5.

Legend for all figures (graphs)

Figure 1. New Zealand prevalence estimates: combined European and Asian any AMD, 45–85 years age, 2014 through to 2026.
Figure 2. New Zealand prevalence estimates: combined European and Asian early AMD, 45–85 years age, 2014 through to 2026.

Figure 3. New Zealand prevalence estimates: combined European and Asian late AMD, 45–85 years age, 2014 through to 2026.
Figure 4. New Zealand prevalence estimates: European any, early and late AMD, 45–85 years age, 2014 through to 2026.

Figure 5. New Zealand prevalence estimates: Asian any, early and late AMD, 45–85 years age, 2014 through to 2026.

Discussion

This study predicts a 12.9% increase in the prevalence of any AMD in New Zealand from 2014 through to 2026 (using series 6 which is the most likely). For series 1 and 11, the prevalence increases from 2014 through to 2026 are estimated to be 17.4% and 8.4% respectively.

For European, any AMD is estimated to increase by 9.8% from 2014 through to 2026. For Asian people, the increase is estimated to be 62.2%.

Significant factors in the rising ethnic prevalences are a shift of the European demographic to an aged population, an increasing Asian demographic due to the combined effect of migration and a shift to an aged population.
Early age-related macular degeneration—This study estimates a prevalence of early AMD in the whole 45–85 year group of 167,482 persons in 2014 and to increase by 13% to 189,197 in 2026. The meta-analysis of pooled population studies used for this study showed early AMD to have a much higher prevalence in European populations (11.2%) than in Asian populations (6.8%).

The risk of progression to late AMD increases with age and early AMD severity. Altering modifiable risk factors such as smoking, heavy alcohol consumption, systemic hypertension, exercise, obesity and high dietary fat intake may substantially lower the risk of developing early AMD or of early AMD progressing to late AMD.

Strategies to reduce the rate of progression of early AMD can have a significant impact on the prevalence of late AMD. A diet rich in carotenoids, omega-3 fatty acids and fish products is associated with a decreased risk of progression of early to late AMD. The Age-Related Eye Disease Study (AREDS) 1 and 2 showed antioxidant therapy to reduce progression to late AMD by 25%. AREDS antioxidant medications are readily available over-the-counter and, at approximately $NZ400 per patient per annum, are cost-effective. Efforts to promote antioxidant therapy are important as otherwise uptake and compliance has been shown to be poor. AREDS antioxidant medications are readily available over-the-counter and, at approximately $NZ400 per patient per annum, are cost-effective. Efforts to promote antioxidant therapy are important as otherwise uptake and compliance has been shown to be poor.

Late age-related macular degeneration—The prevalence of late AMD in the whole 45–85 year group is estimated to be 7583 persons in 2014 and increase 13.6% to 8614 in 2026. Although nAMD represents only 10–20% of late AMD, untreated it has a devastating visual prognosis with loss of 1–3 lines LogMAR visual acuity at 3 months and 3–4 lines by 1 year.

Anti-VEGF treatment is highly effective and the standard of care for nAMD. Vision loss is prevented in over 95% of study eyes and vision significantly improved in 40%. Even very short delays in starting treatment of nAMD can result in significant preventable vision loss. Regular treatment is required for years and possibly for life to maintain vision gain. U.S. Medicare data on anti-VEGF therapy demonstrates a 40% reduction of vision loss, 46% reduction of blindness and 19% reduction of admission to long-term care.

Education on nAMD symptom awareness and vision self-monitoring may lead to earlier diagnosis of nAMD, timely access to treatment and thereby better vision outcomes.

Screening—Early AMD has little impact on vision and patients are usually asymptomatic. As such, there is a need for measures to identify patients with early AMD for timely dietary modification and antioxidant therapy.

Screening of over 45 year olds for early AMD, such as by optometry eye examinations or retinal photography, is debated and warrants ongoing consideration. Screening arguably meets WHO screening guidelines but suffers from the issue of a potentially very large screening population.

To reduce the screening population, screening might be confined to only target European, Asian and other at-risk ethnicities and may be further refined by selecting an older age threshold than 45 years.

Health economics of interventions for age-related macular degeneration—AMD has a major impact on quality of life (QOL). Mild AMD has a self-reported 17% QOL decrease (similar to moderate cardiac angina); moderate AMD a 40% QOL decrease (similar to severe cardiac angina); and very severe AMD a 63% QOL decrease (similar to severe stroke with incontinence requiring constant nursing care).

Bilateral nAMD patients with a major decrease in QOL and increased need of daily living report 45% worse vision-related functioning, 13% worse overall well-being, 30% more anxiety, and 42% more depression than controls. Consequently, health care utilisation costs are more than seven times
higher for nAMD patients than healthy individuals.\textsuperscript{36} The burden of illness related to nAMD has a significant adverse impact on the economy.

Robust health economic benefits are gained from measures to reduce vision loss from late AMD such as lifestyle and diet modification, antioxidant therapy and anti-VEGF therapy.\textsuperscript{5} Severe vision loss from late AMD has declined by around 50\% since anti-VEGF treatment was introduced in 2005.\textsuperscript{37} In New Zealand, blind registration for AMD has declined by nearly one-third from 2005 to 2010 (Blind Foundation figures). Health economic analysis confers anti-VEGF therapy a 16–28\% value gain.\textsuperscript{5} Even treatment of a worse-seeing eye gives significant QOL improvements.\textsuperscript{38} Direct non-ophthalmic, non-medical and indirect costs of nAMD exceed the direct ophthalmic treatment costs by several hundred percent.\textsuperscript{5}

Long-term anti-VEGF therapy places substantial financial burden on a healthcare system.\textsuperscript{10,39} In the United States ranibizumab accounted for nearly 10\% of the drug budget in 2010.\textsuperscript{9} Bevacizumab, with a similar efficacy to ranibizumab but a large price differential (approximately $NZ100 and $NZ2250 per treatment respectively), has superior cost-effectiveness.\textsuperscript{39} Economics is the major driver of a widening call to use bevacizumab as the primary anti-VEGF agent.\textsuperscript{10}

**Provision of age-related macular degeneration healthcare in New Zealand**—An ageing population with a consequent rising AMD prevalence will progressively increase demand for ophthalmic healthcare services. Increasing numbers of anti-VEGF treatments and related clinic visits risk over-burden of ophthalmic services.\textsuperscript{40}

Compared to other developed countries, New Zealand has a low per capita number of ophthalmologists, currently approximately 1 per 38,000 people.\textsuperscript{41} A recent survey of the just over 120 ophthalmologists on the New Zealand medical register found 60\% currently engage in treating nAMD patients with anti-VEGF therapy (Goh Y.W., Worsley D.R. Survey of AMD treatment practices by NZ ophthalmologists. 2014). Eighty-eight percent perceive provision of treatment for nAMD to be a significant current burden in the public system. This echoes the experience in other countries.\textsuperscript{42}

New Zealand has the lowest public funding of anti-VEGF drugs of all Organization for Economic Co-operation and Development (OECD) countries.\textsuperscript{41} Public funded access to anti-VEGF treatment varies nationwide, being determined autonomously by each district health board. Each sets its own funding level and treatment criteria. Thereby, in all but one of the 20 district health boards, access to anti-VEGF therapy is restricted. Bevacizumab is the only routinely available agent. Furthermore, there is no funding of antioxidant therapy or education on lifestyle modification, nAMD symptom awareness and vision self-monitoring.

Only 53\% of New Zealand ophthalmologists perceive access to treatment of nAMD in the public sector to be adequate (Goh Y.W., Worsley D.R. Survey of AMD treatment practices by NZ ophthalmologists. 2014).

The only other major ophthalmic condition with widely restricted treatment access is cataract surgery with national criteria set by an ophthalmologist advisory board and based on strong health economics. Many patients have no option but to pay for anti-VEGF therapy in the private sector. Within a two year period, 46\% of ophthalmologists treating AMD report that patients without access to public care have declined starting, or have discontinued, anti-VEGF therapy because of cost. Furthermore, 61\% report patients not responding to bevacizumab declining to change to other, more expensive, anti-VEGFs because of cost (Goh Y.W., Worsley D.R. Survey of AMD treatment practices by NZ ophthalmologists. 2014).
Limitations of the study—Our projections are subject to the assumptions outlined in Table 1. These give a degree of uncertainty which may increase as projections are made into the future. These assumptions may result in future prevalence being under- or over-estimated.

The divergence over time of series 1 and 11 gives some idea of the range of prevalence that may occur. The ethnic prevalence rates we used are taken from a meta-analysis of a number of large population studies and therefore are not specific to New Zealand.

Several factors may lead to an underestimation of prevalence.

- The source publication for ethnic prevalences didn’t include those over 85 years age in prevalence estimates, as the numbers in this age group were insufficient in most of the pooled studies, yet this age group is likely to have the highest prevalence. In population studies, those with late AMD are more likely to either not participate or dropout for a number of reasons including older age, transportation difficulties and comorbidities. Furthermore, the dropout rate in the pooled population studies is high at approximately 20%, which gives further potential for inaccuracy. The New Zealand over 85 year age group comprises an estimated 68,400 persons in 2014 and 97,900 in 2026, of which the combined European and Asian comprise 66,100 and 84,200 respectively.

- The Blue Mountains Eye Study (BMES) is one of very few studies to calculate a prevalence rate for the over 85 year age group, although based on examining only 135 subjects. The BMES found a prevalence rate in the over 85 year age group of 38.9% for any AMD, 28% for early AMD and 18.5% for late AMD. These rates indicate New Zealand over 85–year age group prevalences in 2014 of 25,700 any AMD, 18,500 early AMD and 12,200 late AMD and in 2026 of 32,800 any AMD, 23,600 early AMD and 15,600 late AMD.

- The Māori and Pacific 45–85 year age group makes up 13.1% of the whole 45–85 year age group in 2014 and 14.8% in 2026. In this study Māori and Pacific peoples are given a zero prevalence of AMD as, to our knowledge, there is no published or anecdotal case. Our approach differs from that of a previous report on vision loss from AMD in New Zealand which arbitrarily assigned a prevalence for Māori. They assumed the Māori prevalence of AMD will be low but under-reported due to a low utilisation of health services.

- There are some potential errors in the population statistics compiled by Statistics New Zealand. These include error introduced due to double counting (because an individual may identify with more than ethnic group) and rounding in ethnic population projections.

Conclusion

AMD is prevalent in the elderly. Due to an ageing population demographic, AMD prevalence in New Zealand is expected to substantially increase from 2014 through to 2026. AMD is currently the leading cause of significant permanent vision loss in New Zealand and the impact of late AMD on QOL, burden of disease and the economic impact is marked. Treatment of AMD has a high health economic benefit that easily justifies public funding of interventions proven to reduce AMD disease progression and prevent vision loss.

This study highlights a need for New Zealand healthcare planners to review current strategies and funding of interventions that minimise vision loss from AMD. Additionally, there is a need to plan for a rising demand for AMD interventions and related ophthalmic services.

Competing interests: Nil.
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The dissolution of the Alcohol Advisory Council: a blow for public health
Kypros Kypri, Jennie Connor, Doug Sellman

Abstract
In June 2012 the Alcohol Advisory Council (ALAC) ceased to be after more than three decades of providing advice on alcohol policy, undertaking health promotion activities, and funding research on the prevalence and causes of unhealthy alcohol use and strategies to address alcohol-related harm. Perversely, its dissolution followed soon after the Law Commission’s “once in a generation” review recommending law reform to address New Zealand’s substantial alcohol-related health burden.

ALAC’s functions were ostensibly taken over by the Health Promotion Agency (HPA) but this new entity was given less autonomy than ALAC and a remit including areas as disparate as rheumatic fever and sun safety. In addition, HPA was compromised from the start by the appointment of a food, alcohol and tobacco industry representative to its Board. ALAC sometimes fell short of community and scientists’ expectations that it provide independent and fearless advice on politically contested matters, such as controls on alcohol marketing. However, it seems that the way the HPA has been set up makes effective action to address health and social problems caused by alcohol consumption in New Zealand unlikely.

The latest burden of disease estimates show alcohol consumption is responsible for 5.4% of deaths and 6.5% of disability-adjusted life years lost in New Zealanders <80 years of age. Of the 802 premature deaths in 2007, 43% were due to injuries, 30% to cancer and 27% to other chronic conditions combined. These direct harms are suffered disproportionately by men and Māori, largely determined by underlying alcohol consumption patterns and contributing to health disparities. There are also harms arising from others’ drinking (e.g., domestic violence) that are less well documented and are more often suffered by women and children.

This article examines the dissolution of the lead government agency on alcohol-related harm and the implications of this decision for New Zealand’s alcohol policy.

As a consequence of a Royal Commission of Inquiry into the sale of alcohol, the Alcohol Liquor Advisory Council was established by Act of Parliament in 1976. “Liquor” was dropped from the name in 2000 but the acronym ALAC remained part of the New Zealand vernacular. ALAC was an Autonomous Crown Entity funded by a levy on alcoholic beverages, with its primary role being: “the encouragement and promotion of moderation in the use of liquor, the reduction and discouragement of the misuse of liquor, and the minimisation of the personal, social, and economic harm resulting from the misuse of liquor.”

The legislation specified 12 functions, including: encouraging and funding policy-relevant research, health promotion, funding treatment and rehabilitation, making recommendations to government about the advertising and sale of alcohol, and the dissemination of relevant research findings from New Zealand and abroad. The development of ALAC is put in historical context in Table 1 which presents a history of New Zealand alcohol legislation over the last 40 years.

On 30 June 2012, ALAC was disestablished and its functions were ostensibly transferred to a new body, the Health Promotion Agency (HPA), which came into being on 1 July 2012 with a broad health promotion remit. The Government gave assurances that ALAC’s functions would be preserved in the new body, however, the HPA is a Crown Agent “which must give effect to government policy when directed by the responsible Minister” (the Crown Entities Act 2004). This arrangement provides for an organisation oriented toward assisting in the implementation of Government policy, in contrast with the more independent role of an Autonomous Crown Entity.
### Table 1. A brief history of New Zealand alcohol legislation 1974-2014

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1974</td>
<td>Royal Commission on the Sale of Liquor Recommended changes to unfreeze and change distribution of licencing, which influenced the 1976 Sale of Liquor Amendment Act and resulted in major increases in outlet numbers, licenced sports clubs, and BYO licences.</td>
</tr>
<tr>
<td>1976</td>
<td>Alcohol Liquor Advisory Council Act Establishes the Alcohol Liquor Advisory Council which began operation in 1978</td>
</tr>
<tr>
<td>1976</td>
<td>Sale of Liquor Amendment Act Established caterer’s licences and ancillary licences, greatly expanding number of licenced venues, and BYO restaurants. Hotels and taverns permitted to close at 11pm on Friday and Saturday nights where previously limited to 10pm.</td>
</tr>
<tr>
<td>1978</td>
<td>Transport Amendment Act (No 3) Introduction of evidential breath testing; lowering of permitted blood alcohol from 0.10 g/dL to 0.08 g/dL [20]</td>
</tr>
<tr>
<td>1978</td>
<td>Sale of Liquor Amendment Act (No 3) Laking Review explicitly rejects the notion that greater availability of alcohol contributes to increased consumption. The new act removed the need to show the ‘need’ for an outlet, substantially reduced the cost of obtaining a liquor licence, and permitted supermarkets to sell wine [12].</td>
</tr>
<tr>
<td>1978</td>
<td>Transport Amendment Act (No. 3) Blood alcohol limit for drivers under 20 years of age reduced from 0.08 g/dL to 0.03 g/dL [20] (commenced Apr 1993)</td>
</tr>
<tr>
<td>1989</td>
<td>Transport Amendment Act (No. 3) Compulsory breath testing introduced [20] (commenced Apr 1993)</td>
</tr>
<tr>
<td>1989</td>
<td>Sale of Liquor Act Parliament passed legislation lowering the alcohol minimum purchasing age from 20 to 18 years. Beer sales were permitted in supermarkets and alcohol was allowed to be sold on Sundays [12].</td>
</tr>
<tr>
<td>1992</td>
<td>Transport Amendment Act (No. 3) Blood alcohol limit for drivers under 20 years of age and repeat drink drivers reduced to zero</td>
</tr>
<tr>
<td>1999</td>
<td>Sale of Liquor Amendment Act</td>
</tr>
<tr>
<td>2008</td>
<td>Law Commission asked by government to conduct a ‘root and branch’ review of laws concerning the sale and supply of alcohol.</td>
</tr>
<tr>
<td>2009</td>
<td>Law Commission Issues paper published [12].</td>
</tr>
<tr>
<td>2010</td>
<td>Law Commission Advice to Government published [21].</td>
</tr>
<tr>
<td>2011</td>
<td>Land Transport (Road Safety and Other Matters) Amendment Act Blood alcohol limit for drivers under 20 years of age and repeat drink drivers reduced to zero</td>
</tr>
<tr>
<td>2012</td>
<td>ALAC disbanded and Health Promotion Agency created.</td>
</tr>
<tr>
<td>2012</td>
<td>Sale and Supply of Alcohol Act (coming into effect in 2012-2013) Territorial Authorities (local governments) are empowered (but not required) to develop Local Alcohol Policies with potential to affect where and how alcohol is sold locally (for discussion see [22]). Introduction of maximum default trading hours of 4am for on-licence outlets and 11pm for off-licences (for discussion see [22]). It became illegal to supply alcohol to anyone under 18 years of age without the express consent of the child’s parent(s) from 18 December 2013.</td>
</tr>
<tr>
<td>2014</td>
<td>Land Transport Amendment Act (No 2) Drink-driving limits for drivers aged 20 years and over reduced from 0.08 to 0.05g/dL, from 1 December 2014</td>
</tr>
</tbody>
</table>
We are reminded of the dissolution of the Public Health Commission in 1995. The Commission was established as part of the health service reforms of 1992 to conduct health monitoring, purchase health services and provide arm’s length policy advice. In its short life the Commission produced comprehensive advice on a range of issues, including alcohol policy, with recommendations for increased alcohol taxes, restricting the physical availability of alcohol, and substantial limitations on broadcast advertising of alcohol.  

The reports were explicitly informed by public health science [5] and by systematic reviews of the empirical literature. It has been suggested that pressure brought to bear on the Shipley government by the tobacco, alcohol, dairy and processed food industries was instrumental in its demise in 1995 when the Commissioner, Professor Sir David Skegg, and all of the Commission’s members, resigned *en masse* in protest against government interference in its activities [6].

Because of the change in statutory designation only some of ALAC’s functions persist in the new HPA. The critical permission to publicly express views that might offend government and to undertake or fund research examining the direction and effects of alcohol policy appears diminished. As health researchers and advocates we were not always happy with ALAC’s approach, finding it too closely aligned with industry at times, muddled on some issues, and apparently unwilling to offer frank and fearless criticism on occasion.

It did, however, highlight alcohol harm and made a substantial contribution to the development of community alcohol and other drug services and brief intervention in primary healthcare. Its single issue focus, policy expertise, and ring-fenced financial resources made it a welcome ingredient in the public health response to alcohol-related harm in a small country where commercial interests can dominate in public affairs.

The move away from an alcohol-focused agency to a multifunction one with responsibilities including immunisation, mental health, gambling, heart and diabetes checks, rheumatic fever, nutrition, physical activity, tobacco control and sun safety is a concern given the potential for dilution of the expertise necessary to provide advice on often technical aspects of alcohol policy, fund high quality research, and implement effective interventions.

Of additional concern is the appointment of a leading alcohol industry figure, Katherine Rich, to the Board of the HPA. A former National Party MP, Rich is Chief Executive of the New Zealand Food and Grocery Council, a lobby group representing the food, tobacco and alcohol industries. Prime Minister Key’s assurance that Rich would be able to manage the conflict of interest in the performance of her role guiding the HPA was unconvincing given the well-documented tactics of the tobacco and alcohol industries to influence government policy, which include industry membership on the boards of public agencies. Key’s assurances have now been undermined by allegations that Rich paid for a smear campaign against health experts; allegations that have not been denied by Rich.

New Zealand alcohol policy is in crisis. The alcohol burden is reflected in unprecedented public and official concern but little action from government. In the latest major review of New Zealand’s liquor laws, the Law Commission Issues Paper attracted 3000 public submissions, and the review finally yielded a comprehensive set of recommendations, many of them the same as proposed by the Public Health Commission 20 years ago. 

The most crucial recommendations, including increasing the price of alcohol, were excluded from the Government’s Alcohol Reform Bill. In the passage to legislation, the Bill was watered down further such that Local Alcohol Policies, which will supposedly underpin community approaches to preventing and ameliorating alcohol problems, offer the only hope of change, yet there is substantial uncertainty about whether they will empower communities or be subverted by commercial interests.

Early signs are that policies seeking to restrict the density or opening hours of alcohol outlets are being fiercely contested by the alcohol industry. The alcohol industry has paid a University economist to provide expert testimony seeking to undermine the research evidence tendered in
opposition to industry demands for longer trading hours than were permitted in new Local Alcohol Plans (e.g., 16). Such legal proceedings are costly for local councils and will deter some from defending policies developed through public consultation.

Funding for independent evaluation is critical to ensure that something is learned about whether the new legislation achieves its stated objectives which include facilitating greater public participation in decision making about alcohol. The hypothecated tax levied on alcohol products that financed ALAC ($12M in 2012 17) has been retained and now pays for the alcohol work of the HPA. The alcohol industry sometimes portrays this as a tax on its activities but it is of course a tax on consumers and therefore public money for which the HPA should be accountable.

We are concerned that the dissolution of ALAC reflects a move by the Government away from funding independent public good research on alcohol-related harm and strategies to address it. We call on the HPA to adopt a transparent strategy for funding policy-relevant research including independent assessment of proposals. This could be undertaken via a subcontract with the Health Research Council (HRC), or the proceeds of the hypothecated tax could go directly to the HRC to be distributed through its competitive grant review processes.

We have previously expressed concern at ALAC’s involvement in social marketing campaigns which are continuing as a major focus of the HPA. These are of dubious effectiveness, may increase health disparities, 18 and therefore represent poor use of public money. The activities of the HPA must build on existing research that has been systematically appraised, and should be guided by an evaluation plan. Anything else risks wasting resources and opportunities, or causing inadvertent harm. When there is no evidence to guide intervention programmes, innovation should be guided by public health theory and research should be undertaken to directly inform policy and practice so that learning occurs and mistakes are not repeated. 19

Competing interests: All of the authors have received research funding from ALAC. KK and JC have received research funding from the HPA. The authors, along with the rest of the research community, may be more likely to have their competitive research applications funded if money generated from the hypothecated tax were to be allocated via an independent, transparent, peer-reviewed process.

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References


CASE REPORT

Rare presentation of a treatable disorder: glutaric aciduria type 1

Monica S Badve, Sandeep Bhuta, Jim Mcgil

Abstract

A 32-year-old female patient presented with migraine and a bipolar disorder with frontal lobe dysfunction and bilateral pyramidal tract signs on examination. MRI brain revealed confluent bilateral symmetric white matter signal abnormality on T2 and FLAIR images with mild cerebral atrophy. Classic widening of Sylvian fissures and CSF space anterior to temporal lobes was seen. In view of the clinical and radiologic findings suggestive of a leukodystrophy, she was investigated for the same. Her investigations revealed an high level of urinary glutaric acid 857 mmol/mol creatinine (normal <4mmol/mol creatinine) and 3-hydroxyglutaric acid 44 mmol/mol creatinine (normal <1 mmol/mol creatinine) and plasma glutaryl carnitine 1.2 micromol/L; (normal <0.34 micromol/L). This was diagnostic of glutaric aciduria type 1. She was started on L-carnitine with which she showed clinical improvement.

Testing for urinary organic acids is important when looking for treatable metabolic disorders (such as glutaric aciduria type I) in patients with leukodystrophy.

Glutaric aciduria type I (GAI) is a rare autosomal recessive disorder caused by the deficiency of mitochondrial enzyme glutaryl-CoA dehydrogenase (GCDH). This results in the accumulation of glutaric acid, 3-hydroxyglutaric acid and glutarylcarnitine. Usually this disorder presents in infancy with striatal necrosis resulting in dystonia and spasticity.

We report a rare late-onset presentation of glutaric aciduria type 1 in adulthood.

Case report

A 32-year-old, right-handed married female patient presented with worsening migraine headaches that had developed at the age of 17 years. She also suffered from bipolar disorder. Neurologic examination revealed broken pursuit movements with normal saccades and fusion. Tone, power and deep tendon reflexes were normal with bilateral extensor plantars. There was no family history of neurologic illness.

The MRI brain showed a confluent bilateral symmetric white matter signal abnormality on T2 and FLAIR images with mild cerebral atrophy. These lesions demonstrated no restricted diffusion or enhancement on post contrast images. MRI brain also revealed wide opercula giving rise to dilatation of Sylvian fissures secondary to fronto-temporal atrophy (Figure 1).

Neuropsychologic testing revealed reduced verbal fluency and motor sequencing suggestive of frontal lobe dysfunction. In view of the clinical history, frontal lobe dysfunction, bilateral pyramidal tract signs and MRI appearance of white matter disease, she was investigated for a leukodystrophy. Her plasma lysosomal enzyme studies, plasma amino acids, blood lactate and blood very long chain fatty acids were normal. However, on urinary organic acid testing, there was an abnormally high level of urinary glutaric acid 857 mmol/mol creatinine (normal <4 mmol/mol) and 3-hydroxyglutaric acid 44 mmol/mol creatinine (normal <1 mmol/mol). Plasma glutaryl carnitine was 1.2 micromol/L (normal <0.34 micromol/L).
Genetic testing revealed that she was compound heterozygous for p.Asn215fs, c.636-10_642dup mutation in exon 8* and the p.Glu365Lys, c.1093G>A mutation in exon 11* of the GCDH gene. The p.Asn215fs, c.636-10_642dup mutation in exon 8* has not been previously described. She was started on L-carnitine at 100 mg 4 times a day with monitoring of plasma carnitine levels.

On follow-up, 3 months later, she was found to have improved mood with reduced mood swings and reduced migraine episodes. Her migraine also improved with propranolol 10 mg BD.

**Figure 1. MRI brain. FLAIR axial images (A&B) demonstrate bilateral widened opercula (arrows) with prominent Sylvian cisterns. Confluent diffuse subcortical white matter signal abnormality in frontoparietal and occipital lobes. Note normal appearance of basal ganglia. No restricted diffusion seen on ADC maps (C). No enhancement seen post contrast (D)**
Discussion

GAI usually presents in infancy with acute encephalitis-like metabolic crisis leading to a severe dystonic movement disorder. With age, muscle tone tends to increase, and dystonia may be accompanied by akinetic-rigid parkinsonism. The usual neuroradiologic findings in children are macrocephaly, frontotemporal atrophy, and after the encephalopathic crisis, atrophy of the caudate nucleus and putamen. Rarely have white matter abnormalities been observed, without the involvement of the basal ganglia, as in our patient. These white matter abnormalities suggestive of leucoencephalopathy are usually seen in adult-onset presentations.

There is tremendous clinical and neuroradiologic variability in this disease. The white matter changes on MRI brain possibly represent underlying mitochondrial dysfunction. Our patient reported a migraine which is similar to headaches described in adult patients previously reported. This is possibly related to the underlying GAI.

Patients with an adult or late-onset presentation usually do not develop dystonia or parkinsonism unlike the early-onset presentation. Our patient showed clinical improvement with L-carnitine supplementation. Patients with GAI can develop secondary carnitine depletion. Carnitine supplementation can result in conjugation of glutaryl-CoA resulting in physiological detoxification and replenishes the intracellular coenzyme A pool.

GAI should be included in the differential diagnosis of diffuse white matter disease in adults with appropriate clinical history and investigated accordingly. Testing for urinary organic acids should be done in adult patients with leukodystrophy since GAI is potentially treatable.

L-carnitine supplementation may halt disease progression and reduce disability in affected patients.

Competing interests: Nil.

References


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An unusual cause of right lower quadrant pain
Michael O’Grady, Marianne Lill

An 80-year-old male presented with right lower quadrant abdominal pain, and subjective fevers. There was guarding and percussion tenderness in the right lower quadrant. He had multiple comorbidities [American Society of Anaesthesiologist (ASA) classification 3], and was taking dabigatran.

Figure 1. An axial view CT image of the patient’s lower abdomen

What is the diagnosis, and best option for management?
**Answer—Perforating foreign body**

The CT scan shows a 35mm linear foreign body penetrating through the medial wall of the caecum into the pericolic fat, with associated inflammatory changes.

Laparotomy under general anaesthesia was considered high risk, given his comorbidities and use of dabigatran, therefore retrieval of the foreign body via colonoscopy was attempted, and achieved. The patient received intravenous antibiotics and bowel preparation overnight.

The foreign body (a toothpick) was located protruding into the lumen of the caecum (Figure 2) and was removed using a colonoscopic snare.

The patient was well the following day and was discharged home on a short course of oral antibiotics.

**Figure 2. Endoscopic view of the foreign body protruding from the wall of the caecum**

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**Discussion**—The majority of foreign body ingestions occur accidentally and are usually limited to children and the elderly. A history of foreign body ingestion is often absent. In the elderly, diagnosis is usually made only after a CT scan is obtained.

Foreign bodies that reach the stomach will pass through the gastrointestinal tract without complication in 80–90% of cases.\(^1\)-\(^3\) Complications include obstruction, bleeding and perforation, with possible subsequent abscess, fistula, damage to adjacent structures, and peritonitis. Perforation occurs in less than 1% of cases and most often occurs at the ileocaecal region.

Chicken or fish bones and toothpicks pose the greatest risk of causing a perforation.\(^1\),\(^2\) Colonoscopic retrieval of a colonic-penetrating foreign body is a feasible option, and removes the necessity for general anaesthesia and laparotomy. The decision should be weighed against the likelihood of ongoing peritonism, and subsequent need for surgery.
Learning points

- A history of foreign body ingestion may not be reported in elderly or paediatric populations.
- The majority of foreign bodies will pass through the gastrointestinal tract without complication.
- In cases of perforation, endoscopic retrieval of foreign bodies is a good option, removing the need for invasive procedures.

Competing interests: Nil.

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**References**

Neuromyelitis optica masquerading as sepsis

M Atif Mohd Slim, Jan Schepel

Clinical—A 24-year-old female was diagnosed with neuromyelitis optica in March 2014 following an admission under our care for right hemiparesis secondary to relapsing medullary myelitis. She was at the time successfully treated with intravenous methylprednisolone. She presented again in April 2014 with one day of malaise and fevers, a week following commencement of prophylactic azathioprine in the community.

On examination, she was tachycardic, tachypnoeic, febrile, and lethargic, with pronounced right hemiparesis and a right tongue deviation. No source of infection could be ascertained clinically. Given recent immunosuppression, sepsis of indeterminate origin was suspected.

Azathioprine was withheld, and empiric intravenous ceftriaxone, amoxicillin, and acyclovir were started following a full septic screen, including lumbar puncture, blood cultures, and chest radiograph. Additionally, urgent MRI brain with contrast was ordered to investigate the hemiparesis and rule out progressive multifocal leukoencephalopathy, a rare consequence of immunosuppression in inflammatory neurological disorders.

T2 imaging with a fluid attenuated inversion recovery (FLAIR) sequence revealed extension of the previous medullary lesion, with a new left perithalamic focus (Figure 1).

Antimicrobial therapy was immediately stopped in place of high-dose intravenous methylprednisolone. Her symptoms resolved within 24 hours, and she was discharged a week later on a tapering course of oral prednisone. The full septic screen done prior to antimicrobial therapy was negative, including all cultures following full period of incubation. There were no complications at follow-up in May 2014.

Figure 1. MRI brain showing an (arrowed) inflammatory left parathalamic lesion enhanced on an axial T2 FLAIR slice (left), with a T1 slice (right) at the same level for comparison.
Discussion—Patients on immunosuppressive therapy are at increased risk of severe infections. The pre-optic and posterior nuclei of the hypothalamus have an important role in thermoregulation. Our case illustrates how a primary inflammatory lesion in or adjacent to this area can masquerade as sepsis. It is important that perithalamic lesions remain in the differential diagnosis when treating the newly febrile immunosuppressed patient with a background of inflammatory neurological disorder.

Definitive management may appear counter-intuitive to the infection-like presentation; as with any relapse of autoimmune myelitis, further immunosuppression is indicated.

Competing interests: Nil.

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Physician-assisted dying—a survey of Waikato general practitioners

Jack H Havill

General practitioners (GPs) are central to the safe and successful introduction of physician-assisted dying (PAD) into a jurisdiction. Although medical participation is integral in the intended End-of-Life Choice Bill (Maryan Street), which was a Member’s Bill in the Parliamentary Ballot Box during part of the years 2013–2014, there has been little information published about views of New Zealand medical doctors as a group. However, a 2004 study in the New Zealand Medical Journal reported that GPs caring for patients in the last year of their life, deliberately hastened death in 39 (5.6%) cases.

This letter reports on a survey of opinions of GPs in the Waikato District Health Board (DHB) area on some of the main issues in the End-of-Life Choice debate in New Zealand. The survey was conducted at the end of 2014.

200 letters were sent and 78 replies were received (39% response).

There were 3 questions. Question 1 covered the basic issues as they applied to a competent patient who was still able to make a request while knowing what they were asking. Question 2 was about the End of Life Directive, which is a special type of advance directive and is intended to allow a person to have assistance to die after they have become incompetent. Question 3 addressed the issue of severe dementia as it could possibly be part of an End-of-Life Directive.

Each question had 5 choices (totally support, probably support, unsure, probably oppose and totally oppose), with one possible answer. The questions and responses are outlined in this table (n=78):

<table>
<thead>
<tr>
<th>Question</th>
<th>Fully support/ probably support</th>
<th>Unsure</th>
<th>Totally oppose/ probably oppose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Question 1:</strong> Given adequate safeguards against abuse, do you support the passing of a law to allow a medical practitioner to give assistance to die, on request from a competent patient, 18 years and older, where the patient has end-stage terminal disease (e.g. cancer), or is suffering from irreversible unbearable suffering (e.g. motor neurone disease, end-stage respiratory failure)?</td>
<td>47.3%</td>
<td>5%</td>
<td>47.3%</td>
</tr>
<tr>
<td><strong>Question 2:</strong> The End-of-Life Directive, as intended in the EOLC Bill, allows a person while still competent to write the Directive, seeking medical assistance to die, should the above conditions in Question 1 occur after they become incompetent. Would you support passing a law which would legalise such a request in an End-of-Life Advance Directive?</td>
<td>47.3%</td>
<td>9%</td>
<td>43.6%</td>
</tr>
<tr>
<td><strong>Question 3:</strong> It is possible that the patient may include the following statement about dementia in their End-of-Life Directive (as a condition that they would find unbearable): ‘If I develop severe dementia resulting from Alzheimer’s disease, or degenerative brain disease due to arterial disease or other agency, where my mental competence has deteriorated to the extent that I am no longer able to recognise close relatives or friends, and am totally dependent on others for basic physical needs e.g. eating food and drinking fluids, spoon feeding, toileting for incontinence, dressing, I would request that I be given medical assistance to die.’ Would you support passing a law which would legalise such a request with regard to dementia, as above in this question?</td>
<td>39.5%</td>
<td>10.5%</td>
<td>50%</td>
</tr>
</tbody>
</table>
In 2012 the Horizon Poll showed 63–65% support for all sections of the proposed End-of-Life Choice Bill.\(^3\) A 2014 study shows that public support for legalisation of PAD is now 82%.\(^4\) However, medical practitioner support has been moving more slowly and information is more sparse. For instance a recent survey of 17,000 USA physicians and 4000 European physicians,\(^5\) as part of the Medscape Ethics Report 2014, showed support for PAD as 54% (31% said no and 15% said ‘it depends’). However, support from physicians in the Netherlands and Belgium is strong and is said to be continually growing.\(^6\)

Naturally GPs will be anxious about taking part in PAD due to inexperience. When legalisation occurs in New Zealand, it would be desirable to have proper support systems in place for GPs—e.g. the Royal Dutch Medical Association set up Support and Consultation on Euthanasia in the Netherlands (SCEN): a programme aimed at structuring the consultation and decision-making process before accepting a request for PAD.\(^6\)

In conclusion, it is apparent that 45–50% of GPs ‘support or would probably support’ PAD in New Zealand. It is also reasonably certain that the New Zealand law will allow aspects of this within the next few years, following on from other jurisdictions where assistance has been legalised. Matters are also rapidly progressing in Britain and Australia, following on from Quebec,\(^7\) where the substance of the new law is similar to the ‘Maryan Street’ End-of-Life Choice Bill. Hopefully our professional medical and nursing bodies can take part in the framing of the law and regulations as this happens.

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References

1. Online: http://www.ves.org.nz/theBill
Pacific women’s experiences and views of participating in a novel dietary intervention for weight loss
Asmita Patel, Micalla Williden, Caryn Zinn, Nigel Harris, Dee Holdsworth-Perks, Grant Schofield

In comparison to other ethnic groups in New Zealand, Pacific People have higher prevalence rates for chronic conditions, such as type 2 diabetes and for obesity. This is predominantly associated with a poor diet.1

A low carbohydrate healthy fat (LCHF) dietary approach has been successfully used across a range of populations to help manage conditions such as epilepsy, polycystic ovary syndrome, type 2 diabetes and in reducing body fat (i.e., weight loss).2–7

A LCHF diet has an emphasis on whole foods. It generally consists of consuming around 100 grams of carbohydrates per day.3 The remainder of the macronutrients are comprised of a moderate amount of protein, and a higher fat intake than the current dietary guidelines recommend.8

The emphasis is on non-starchy vegetables, small amounts of starchy vegetables and fruit, fish, poultry, unprocessed meat, eggs and nuts. Healthy fats refer to a mix of monounsaturated fat, omega 3 polyunsaturated fat and some natural saturated fat from whole food sources.

The aim of this study was to examine the experiences and views of a sub-group of obese, Auckland-based Pacific women who took part in a 12-week dietary intervention designed to assess the feasibility and effectiveness of an LCHF dietary approach to weight loss.

The LCHF diet emphasised the use of culturally appropriate foods (i.e., fresh fish and coconut milk). The use of culturally appropriate foods may facilitate long-term adherence to an LCHF dietary approach, which may translate to health-related gain.

Five participants took part in an audiotaped focus group at the conclusion of the intervention. Participants were aged 41 years and older. Transcribed data were analysed using an inductive thematic approach.9

Four main themes emerged:

**Theme 1: Family health history**—This theme highlighted how participants’ own health status and/or the health status of family members influenced their decision to take part in the intervention. The following quotes demonstrate this:

“*I was pre diabetic. All your (sic) relatives are diabetic. So it’s about looking after ourselves and being around for our children.*”

“*We have grandchildren. We need to be mindful that there are other lives involved and effect not just our own.*”

**Theme 2: Perceived benefits of LCHF**

**Subtheme: Increased energy**—Some participants discussed how they gained more energy as a result of the LCHF approach. The following quotes convey this:

“*I go for a run. I’ve noticed I have all that energy again and I’m back into my netball again.*”

“*The energy. I felt the weight I lost has been so worth it.*”
Subtheme: Satiety and versatility of LCHF food—The following quotes illustrate participants’ experiences of LCHF food in terms of preparation and consumption:

“Eggs for breakfast and onions and mushrooms. It just fills you up. Lunch time is when you are starting to get hungry again. You don’t actually feel like snacking.”

“I’m becoming quite creative with my salads. It’s easy you can just throw some ham on top, cheese and apple.”

Subtheme: Cost—In the following quotes, participants discussed how LCHF food can be less expensive in terms of food purchasing costs:

“I find the food costs less.”

“It depends on what vegetables are in season. Sometimes lettuce is $2.60 and then you go to the food markets and the quality of the veggie is not as good but there is the price difference.”

Theme 3: Family and LCHF—For health reasons some family members also undertook a LCHF approach. The following quotes illustrate this:

“My middle child unfortunately inherited his mother’s genes. So he decided he would do it with me. That was quite helpful because it felt like I had a partner.”

“One of my boys was overweight. We have made a family change with eating. So we cook healthy.”

Theme 4: It’s a lifestyle, not a diet—The following quotes convey how a LCHF approach was perceived to be a lifestyle change and not a diet:

“It’s a lifestyle not a diet. Making it a part of your life to try (and) eat healthy.”

“It wasn’t a diet, it was more of a lifestyle change.”

The LCHF approach appeared to be acceptable and feasible for this group of women. Participants identified a number of perceived benefits to undertaking LCHF. Namely, a perceived increase in energy levels and feelings of satiety, as well as a reduction in food costs.

Satiety can be an important factor in terms of adherence to a weight loss programme. Individuals who constantly feel hungry are less likely to comply with certain diets. Cost was not a barrier to adopting LCHF, as there was a focus on purchasing seasonal vegetables and moderate consumption of protein-based foods.

Future research with Pacific women will examine the physical health-related benefits that may result from following a LCHF approach.

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LETTER

Hypertension in young adults
Walter van der Merwe, Veronica van der Merwe

Hypertension is common, even in young adults. The NHANES survey 2011–2012 put the incidence of hypertension in the 18–39 year age group at 7.3%. There are no outcome studies of hypertension treatment in young adults, but short to medium-term risks (5–10 years) of untreated mild and moderate hypertension are likely to be low. However, long-term outcomes (30–40 years) are much more important to people in their 20s than to those in their 60s and 70s and it seems likely that hypertension from a young age, particularly if undiagnosed or untreated for a long period of time would carry a very substantial long-term cardiovascular risk.

General practitioners may be reluctant to make a diagnosis of hypertension in a young person, and may also lack confidence about how to investigate and treat it. There is evidence that hypertension in this age group is less likely to be diagnosed or treated even when young individuals have good access to primary care.

Between 22 February 2009 and 10 June 2013, 1000 consecutive new patients were seen through the North Shore Hospital Hypertension Clinic; mean age 55 years. From this group we extracted and reviewed the data on those aged 30 years or less at their first visit.

Ninety-two (9.2% of the total) were aged ≤30 years on the date of their first clinic visit (range 15–30 years, mean age 24); 70 were European, 9 Asian, 8 Māori and 9 Pacific people. 24-hour ambulatory blood pressure monitoring was used in 51 patients (55%).

Average number of clinic visits was 2.6 (1–8). Secondary causes were identified in 12 (13%) patients: primary renal disease (6), obstructive sleep apnoea (5), primary aldosteronism (1). Average BMI was 31.8.

Forty-seven (51%) were on antihypertensive medication at the first clinic visit, and 53 (58%) at discharge. Mean blood pressure at the first visit for all patients was 145/86 mmHg. Mean discharge blood pressure was 129/75 mmHg and mean blood pressure drop was 16/11 mmHg.

Of those on antihypertensive medication, both at admission and discharge average number of drugs used was 2, although there was increased use of ACE-inhibitors and DHB calcium channel blockers, and reduced use of thiazide diuretics and beta blockers on discharge.

At discharge, the 92 patients could be categorised as follows:

- 41 – Essential hypertension on antihypertensive medication.
- 12 – Secondary hypertension on antihypertensive medication.
- 27 – Prehypertension (BP 120–139/ 80–89) not currently on antihypertensive medication.
- 12 – White coat hypertension not currently on antihypertensive medication.

In other words none of the patients was completely “normal”. Clearly those with treated essential hypertension and secondary hypertension require long-term monitoring and follow-up.

Prehypertension is not a completely benign condition, despite not mandating blood pressure medication immediately; it has been shown that the majority of individuals with prehypertension will progress to hypertension.

White coat hypertension, similarly, despite not mandating immediate use of antihypertensive medication does not have the same prognostic implications as true normotension; it confers a
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significantly higher risk of progression to chronic hypertension, and separate from that, also a higher stroke risk.\footnote{6}

In short, all 92 patients aged 30 years and less referred to the hypertension clinic, including the 39 (42\%) discharged on no antihypertensive medication were at significantly higher risk for long-term cardiovascular complications than age-matched individuals from the general population.

An additional important consideration in young individuals with treated hypertension and those with prehypertension is that they are at an age where lifestyle intervention may provide an important component both in the treatment of hypertension, and in the prevention of, or delayed progression to established hypertension.\footnote{6,9} This is particularly relevant to our group of young patients whose mean BMI (30.8) was significantly above the healthy range.

Hypertension is by no means uncommon in very young adults, whose lifetime risk of cardiovascular disease and premature death may be substantially higher than those who develop hypertension in middle-age or later life. Although secondary causes of hypertension do occur, the majority still have essential hypertension.

All young adults, including adolescents should have an annual blood pressure check, and whilst a one-off elevated reading does not make a diagnosis of hypertension, it should not be ignored, and the individual should be recalled for further evaluation.

24-hour ambulatory blood pressure monitoring should probably be mandatory for diagnosis of hypertension in young people, and is particularly useful prior to commencing medication.\footnote{10}

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LETTER

Total ambulatory management of patients undergoing coronary angiography and intervention: a pilot study

Theo Gudex, Jonathon White, Steph Madenholt-Titley, Michael McAleer, Sue Savage, Peter Ruygrok

The use of transradial access (TRA) for coronary angiography and percutaneous coronary intervention (PCI) has pervaded cardiac catheterisation centres internationally\(^1\)\(^-\)\(^2\) with the benefits of reduction in access site bleeding, rapid post-procedural ambulation, shorter hospital stay and improved patient satisfaction.\(^3\)\(^-\)\(^7\)

Recent studies have suggested that excellent outcomes are achievable in patients undergoing day case angiography and PCI employing a total ambulatory approach.\(^8\) This approach, using medically approved reclining chairs rather than beds, with a more relaxed, ‘de-hospitalised’ environment and allowing patients to ‘walk-in’ and ‘walk-out’, aims to reduce anxiety and need for intravenous sedation.

We aimed to assess the feasibility of implementing a ‘radial lounge’ for day case coronary angiography/PCI at Auckland City Hospital by screening consecutive outpatients undergoing elective day-stay angiography. The study protocol was approved by the institutional ethics committee and written informed consent was obtained.

Participating patients were admitted to the angiography day ward on the morning of their procedure and underwent usual assessments and insertion of upper limb intravenous access, while in medically-approved reclining chairs. They remained in their own clothing, drank clear fluids up until one hour before and ate until 4 hours prior to the procedure.

Patients walked into the catheterisation laboratory where intravenous sedation was administered at the discretion of the operator. At the conclusion of the procedure, under nursing supervision, patients walked from the catheterisation laboratory to the ‘radial lounge’ where they recovered in medically-approved reclining chairs. Those administered intravenous sedation returned in a wheelchair. All patients were encouraged to drink at least 1 litre of water. Satisfaction survey forms were posted following discharge.

Over a 2-month period (December 2013–January 2014), 199 patients were screened for inclusion. Fifty (25%) were enrolled and underwent angiography on the total ambulatory care pathway, 36 (72%) were male and 14 (28%) female, with a median age of 65 (range of 41–83 years). New Zealand European and Maori comprised 68% and 18% of the population respectively. Clinical indications for angiography were suspected coronary artery disease in 38/50 (76%), valvular heart disease in 9/50 (18%) and other indications (abnormal echocardiogram, arrhythmia, syncope) in 3 (6%).

Transradial angiography was successfully completed in 47/50 patients (94%) with 3 failing due to: radial arterial spasm, radial loop and guidewire perforation, respectively who underwent transfemoral angiography without complication. Intravenous sedation was administered to 9/50 (18%) patients, due to anxiety, puncture site pain, radial spasm or operator preference (Figure 1). Intravenous fluids were administered to 6/50 (12%).
All patients walked into the catheterisation laboratory prior to the procedure while 40/50 (80%) walked out following, without assistance. Five (10%) patients left in a wheelchair due to the administration of intravenous sedation while 5 required bed transfer (vasovagal reaction, critical coronary anatomy, 3 due to femoral access). Admission to hospital was required in one case for observation of a forearm hematoma while two patients were admitted because of critical coronary disease. All other patients, 45/50 (90%), were discharged on the same day at a median time of 185 minutes (range 130–422 minutes) following return to the ‘radial lounge’. No major complications were observed with 2 minor complications (vasovagal reaction and haematoma).

Ad hoc percutaneous coronary intervention was performed in 10/50 (20%) patients of whom 9 (90%) were discharged on the same day as the procedure after a median time of 237 minutes after return to the radial lounge (range 130–420 minutes). Sedation was administered to 4 of this group (40%) and IV fluids to 3 (30%) (Figure 1).

All 50 patients were posted satisfaction surveys following their discharge. The 30 responders (60%) were satisfied with the ambulatory care pathway and environment. Most were highly satisfied with the
comfort of the reclining chair, information delivery, treatment with respect and dignity and involvement in decisions regarding their care.

Although radial access for angiography and PCI has become increasingly common, radial spasm can limit to procedural success in a minority of cases and is frequently managed pre-emptively with conscious sedation. Although one randomised trial has shown that moderate doses of an opioid/benzodiazepine combination resulted in a reduction in spasm, we felt a selective approach could be considered as reduced sedation may speed ambulation and recovery, in an era when avenues to shorten hospital stay are being explored, yet retaining high levels of safety. This pilot study suggests that total ambulatory care of selected transradial coronary angiography and interventional patients can be successfully introduced with a decreased sedation, low complication rate and high patient satisfaction. Further studies to evaluate this management strategy and compare it more rigorously with bed-based care appear warranted. Greater degrees of ambulation could also be considered in other areas of hospital based medical care.

Competing interests: Nil.

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References


Medical Service in Warfare

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“War is Hell,” and never more hellish than in this year of grace. Highly-civilised men are striving to take one another’s lives on a scale that the mind of no savage ever conceived.

We read of straw being spread over heaps of corpses and set fire to, and this cremation hardly more speedy than the making of a large cemetery by a battery of artillery. No doubt war develops courage and self-sacrifice, and leads often to a regeneration of rational virtues, but in the main its attendant barbarity and cruelty seem sadly out of keeping with the enlightenment of the world.

The only difference between the attack of Germany on Belgium and the assault of a burglar on a peaceful household is that the former is immeasurably more heinous. Where are we to find in warfare something morally in advance of previous generations, some augury that mankind progresses? Are there no Good Samaritans now? It is the medical service of the contending armies that shows that in warfare civilisation has not altogether given way. The ambulances work not only to save the lives of comrades but also fallen foes and altruism has never reached a higher pitch of perfection.

There is more glory in saving life than in taking it. The prompt relief of the sick and wounded is part of the art of war, for wounded men are valuable if promptly cured and returned to their regiments, but worse than useless if they cannot rejoin the fighting line. Thus the medical officer has a double share of honour as a practitioner of the art of war in strengthening the ranks of his country, and as one who plays the part of the Good Samaritan to friend or foe, dealing mercy and not vengeance.

Napoleon was probably the first great military leader to provide a system for prompt attention to the wounded in war, but the modern ambulance dates from the Civil War in America, and was largely developed in the Franco-Prussian War. The importance of the medical services of armies has become increasingly great by the establishment of preventive medicine and sanitation, and the mobility of an army depends largely upon the proper evacuation of the sick and wounded without undue interference with the supplies of reinforcements and munitions along the lines of communication. To successfully attain this end much skill is required and co-operation among many units, from the, medical officers with the firing line to the bearer subdivisions and dressing stations, the convoys, the clearing hospitals, the ambulance trains and ships, and the base hospitals.

Various nations have modified ambulance work in accordance with their own particular views. Thus Japan discards elaborate equipment and relies greatly on improvisation; Germany has in peace time civil surgeons properly organised to take their places with their units in the field, and has provided, as far as possible, against emergencies; France depends to a very large extent upon voluntary aid; and Great Britain has learned much from lessons in the Boer War, when the medical service was overworked to breaking strain.

In the Franco-Prussian War the British people subscribed no less than £300,000 for Red Cross work in aid of both the French and Germans, and in the present crisis there has been no want of money for ambulance work. It is a source of great encouragement to the medical profession that the work of the doctor in the field attracts the sympathy and admiration of the public mind, and an honour that to him falls duty that is congenial and humane, and both arduous and dangerous.
METHUSELAH

Efficacy of beta-blockers in patients with heart failure plus atrial fibrillation

Beta-blockers are indicated in patients with symptomatic heart failure with reduced ejection fraction; however, the efficacy of these drugs in patients with concomitant atrial fibrillation is uncertain. In order to elucidate this issue, these researchers have performed a meta-analysis of data from ten randomised controlled trials of the comparison of beta-blockers versus placebo in heart failure. The presence of sinus rhythm or atrial fibrillation was ascertained from the baseline electrocardiograph.

Over 18,000 patients were assessed. 76% were in sinus rhythm and 17% had atrial fibrillation. Beta-blocker therapy led to a significant reduction in all-cause mortality in patients with sinus rhythm but not in patients with atrial fibrillation.

The conclusion reached was that beta-blockers should not be used preferentially over other rate-control medications and not regarded as a standard therapy to improve prognosis in patients with concomitant heart failure and atrial fibrillation.


Cytisine versus nicotine for smoking cessation

Placebo-controlled trials indicate that cytisine, a partial agonist that binds the nicotinic acetylcholine receptor and is used for smoking cessation, almost doubles the chances of quitting at 6 months.

This report concerns a non-inferiority trial comparing cytisine with nicotine. Participants were recruited from smokers motivated to quit who had sought advice from the New Zealand national quitline. 1310 adult daily smokers were randomly assigned to receive cytisine for 25 days or nicotine replacement therapy for 8 weeks.

The effectiveness of cytisine for continuous abstinence was superior to that of nicotine-replacement therapy at 1 week, 2 months, and 6 months. A downside noted was that there were more self-reported adverse events in the cytisine cohort, primarily nausea and vomiting, and sleep disorders.


Cobalt-chromium everolimus eluting stents or bare metal stent in the management of coronary artery disease

Network meta-analyses have suggested that cobalt-chromium everolimus eluting stents are associated with a reduction in stent thrombosis compared with bare metal stents but the clinical implications of this finding remains unclear. This report concerns a meta-analysis which seeks to clarify the issue.

Screening the extensive literature produced 5 randomised controlled trials. They involved 4896 patients. The results obtained demonstrated that cobalt-chromium everolimus eluting stents are associated with improved cardiovascular outcomes including cardiac survival, myocardial infarction, and overall stent thrombosis compared with bare metal stents.

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NOTICE

Medical Benevolent Fund

NZMA Members, and families of deceased Members, may apply for aid when in situations of financial hardship or distress.

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