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SUMMARIES

Comparison of cancer survival in New Zealand and Australia, 2006–2010

Phyu S Aye, J Mark Elwood, Vladimir Stevanovic

This study compares the survival of cancer patients diagnosed in 2006 to 2010 in the whole populations of New Zealand and Australia. Survival rates were lower in New Zealand, with 5-year relative survival being 4.2% lower in women, and 3.8% lower in men, for all cancers combined. Of 18 cancers assessed, 14 showed lower survival in New Zealand. For most cancers, the differences in survival were maximum at 1 year after diagnosis, becoming smaller later. We conclude that further improvements in the recognition, diagnosis, and treatment of cancer in New Zealand should be possible. As the survival differences are seen soon after diagnosis, the appropriate investigation of patients in primary care, which is a very complex process, and so reducing time intervals to diagnosis and treatment, may be particularly important.

Additional points: 'Survival' means the proportion of people who are still alive at various times from 1 to 10 years after their diagnosis, and is adjusted to allow for general non-cancer causes of death. Thus a lower 'survival ratio' means that a higher proportion of patients have died from their cancer. Overall, in New Zealand, for all people diagnosed with cancer in 2006-2010, for women, 77% were alive 1 year later, 63% at 5 years, and 59% at 10 years. For men, the figures are 75% at 1 year, 61% at 5 years, and 57% at 10 year. The 4 cancers with similar survival in each country, were melanoma, myeloma, mesothelioma, and cervical cancer. In this study, the whole populations of each country were compared. Several other studies by other investigators have shown differences in cancer survival within New Zealand by ethnic group and by socioeconomic factors.

Health and wellbeing of older Pacific Peoples in New Zealand

Fialupe Lotoala, Mary Breheny, Fiona Alpass, Annette Henricksen

Older Pacific people have poorer physical and mental health than older people from other ethnic groups in New Zealand. They also have higher rates of health condition such as diabetes, respiratory conditions, stroke, and chronic kidney conditions. Older Pacific people have lower income, wealth and assets. Poorer socioeconomic status contributes to the poor health of older Pacific people. Improving the health of older Pacific people relies on improving their economic standard of living.

Late-onset rheumatoid arthritis in the Counties Manukau District Health Board region of New Zealand: an observational study of treatment

Laurence Teoh, Ravi Suppiah, Peter Gow

Our study investigated the early treatment of rheumatoid arthritis in patients with late onset (starting at or after 60 years of age) and those with young onset rheumatoid arthritis (starting before 60 years of age). Both groups benefit from treatment with disease modifying medications (a groups of medications that can modify the course of rheumatoid arthritis progression). At Counties Manukau District Health Board rheumatology outpatient clinic, patients with late onset rheumatoid arthritis had similar treatment with disease modifying medication to those with young onset rheumatoid arthritis, which is appropriate.

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Diagnostic category agreement and malignancy rates in clinician-categorised, non-standardised thyroid cytology reports
Mark J Bolland, Carl Eagleton, Brandon Orr-Walker

Thyroid cytology is a standard investigation in suspected thyroid cancer. It is recommended that thyroid cytology samples are reported using a standardised system, but this has not been universally adopted yet in New Zealand. We asked two clinicians to classify thyroid cytology reports that had not been reported using the standard system, and found moderate levels of disagreement between clinician classifications. In addition, the rates of thyroid cancer for each category of thyroid cytology were different from published rates. These results suggest that standardised reporting of thyroid cytology should be universally adopted, and that local malignancy rates should be determined and reported regularly to guide management of patients locally.

Herpes zoster (shingles) at a large New Zealand general practice: incidence over 5 years
J Stewart Reid, Brendon Ah Wong

This is the first paper to describe the occurrence of shingles in New Zealand and suggests that the rate of shingles in NZ is similar to that reported internationally. The data were derived from a study of the recorded cases over a five year period in a large group practice in Lower Hutt with 19,000 registered patients. The incidence rose with age and for those aged >50 years, females have about a 10% higher occurrence rate than males. These data suggest that there is a 1 in 5 chance of suffering shingles from age 51 to 80 years.

Impact of improved treatment on disease burden of chronic hepatitis C in New Zealand
Edward Gane, Catherine Stedman, Cheryl Brunton, Sarah Radke, Charles Henderson, Chris Estes, Homie Razavi

In this study, local experts have reviewed how many New Zealanders have hepatitis C virus (HCV) infection, how many will develop liver cancer or liver failure, and how many will need liver transplantation or will die from these complications. This year, approximately 140 New Zealanders died from HCV this year and by 2030, this number will climb to 350. The only way to prevent these deaths is by curing patients with successful antiviral treatment. Despite this, very few New Zealanders are being treated because current treatment has a low chance of success, bad side-effects and requires weekly injections of interferon for up to 1 year. Newer antiviral treatments have been developed which are all oral, have few side-effects and can cure 95% of patients after only 12 weeks. Timely funding of these new treatments could prevent most complications and deaths in patients with HCV. Widespread access should also reduce new cases of infection and if combined with other efforts to prevent disease transmission like needle exchange, should eliminate HCV from NZ within our lifetime.

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EDITORIAL

Food in schools: targeting versus the right to food

Donna Wynd, Mike O'Brien

Food in schools has been the topic of a great deal of public discussion in recent years. While schools have been providing breakfasts, and in some cases lunches, to children for many years, the bigger question of whether the need warrants government intervention remains largely unresolved.

While the New Zealand public are generally in favour of providing food to children,[1] others are less certain of the need for, or wisdom of, such provision. The chief argument against state provision—and this is the view of the current National Party-led government—is that feeding children is the parents' responsibility. Other arguments are that (1) providing food in schools makes parents 'create an expectation' of provision, and (2) there is always money in the household budget for breakfast, if only parents chose to prioritise it. Ranged against these views are those in favour for a range of reasons including that if children are hungry, then they should be fed.

This editorial considers the government response to food in schools, including recent advice from Treasury; it then outlines some reasons to support food in schools, and finishes by reflecting on broader issues of children's right to food.

Government response

In 2012 the Children's Commissioner's Expert Advisory Group on child poverty recommended the New Zealand Government give immediate attention to designing and implementing "a collaborative food-in-schools programme, commencing with lower socioeconomic (decile 1 to 4) primary and intermediate schools."[2] The government's response to the EAG's recommendation was to provide $9.5 million over 5 years to support the provision of food in schools by food manufacturing corporates Fonterra and Sanitarium, and private charity KidsCan. Although the Fonterra and Sanitarium's food in schools programme is expanding, an estimated 80,000 children miss out on breakfast every day due to the lack of nationwide coverage.

The government's tepid response to the EAG's recommendation was largely informed by a Treasury briefing paper which outlined the "two immediate problems" food in schools might be expected to address as "improving educational outcomes", and "alleviating hardship and suffering".[3] While the paper acknowledged "low parental income" as a factor in children's hunger, it suggested other measures such as budgeting classes and parenting programmes could deal with less well defined issues including "different cultural norms" and children who refuse to eat breakfast.

Treasury based much of its analysis on the 2007 *Children's Food and Drink Survey*.[4] This shows that the likelihood of a child not eating breakfast increases sharply as they get older. For younger children (5–7 years), 91% had breakfast every school day, falling to 71% for 13–16 year olds. This suggests that food in schools aimed at primary schools is more likely to improve children's nutritional status in those cases where food is not served at home. It also suggests that Treasury's claims that children 'choose not' to eat breakfast is more applicable to adolescents and teens. This is one reason the EAG and others such as Child Poverty Action Group have focused on younger children.

Treasury's suggested role for the government was to contribute to KidsCan or create a contestable fund. In the event, KidsCan and Fonterra received funding. More problematically—and this is emerging as a general trend in the government's efforts to deal with child poverty—was Treasury's 'principle' of targeting those most in need, along with the possibility of 'reprioritising' existing scarce funds to provide food in schools.

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Reasons to support school food programmes

Treasury's reasoning largely rested on a New Zealand study that provided food in schools over a year and found no statistically significant effect on children's school attendance or academic achievement.[5] The study did, however, find "a significant decrease in children's self-reported short-term hunger." Yet a child's rights perspective dictates that the alleviation of hunger must be the primary focus of food in schools: no child is responsible for his or her own hunger.

While the New Zealand study did not find any improvement in children's school attendance or academic performance, other studies have found such improvements,[6] although using different methodologies and reporting methods. Teachers and principals interviewed by CPAG during its research on the impact of food in schools[7] argued that breakfast improved both children's school attendance and classroom performance.

A key reason to support food in schools for all children that need it (not just those "most in need') is improved nutrient uptake, and the longer term benefits associated with this. In the first instance, there is the immediate benefit of a nutritious meal, but there are longer-term benefits associated with improved nutrient uptake over time (including improved academic performance), as well as evidence that school breakfasts can reduce obesity.[8]

A surprise finding of CPAG's research, and perhaps one of the more compelling arguments for breakfast in schools, was the positive social impact of school breakfasts. Most schools strive to provide a safe environment for children, and often breakfast clubs doubled as spaces for children to socialise or do homework if they were unable to do so at home. Indeed, school staff felt the social and community benefits were as important as the food itself.

Clearly, not all the benefits of food in schools can be captured in a time-limited experiment or in purely dollar terms, especially those around positive social outcomes. Nonetheless, we consider they are sufficiently well-established to justify the investment in a food in schools programme along the lines outlined by the EAG.

Broader issues of support for children

There is little doubt the underlying reason so many New Zealand children go without breakfast is that households simply do not have sufficient money. Data from the Ministry of Social Development shows that from 2010–2013, the number of children living in households with incomes less than 40% of the median wage increased by 20,000 to 135,000 (13% of all New Zealand children).[9] New Zealand research has also identified children of working parents as regular users of school breakfast clubs. Many low-income parents work long hours and it is not uncommon for children to arrive at school very early not having eaten. Poverty is also a feature of many of these working households, with 40% of children in poverty living in households with at least one adult in work.

As a signatory to the UN Convention on the Rights of the Child, the New Zealand Government has an obligation to ensure all children are provided adequate protection and care to ensure their wellbeing. Article 27(1) recognises the right of every child to a standard of living adequate for the child's physical, mental, spiritual, moral and social development, and Article 27(2) says that states shall take appropriate measures to assist parents and others responsible for the child to provide material assistance and support programmes. Similarly, Article 11 of the International Covenant on Economic, Social and Cultural Rights states: "…Parties to the present Covenant, recognising the fundamental right of everyone to be free from hunger". Furthermore, child poverty in New Zealand has a steep ethnic gradient, and the Crown has an obligation under the Treaty of Waitangi to protect and nurture Māori children.

The large number of New Zealand children who do not receive adequate nutrition is not only a violation of New Zealand's international and Treaty obligations, it violates their rights as children.

Children must be protected not because they are future productive workers, but because they are today's citizens.

This 'right to food', of which universally available food in low-decile schools is but a small facet, cuts across the neoliberal foundations of New Zealand's social assistance architecture. Far removed from the notion of citizenship and participation, New Zealand's current social assistance is framed in the language of independence, individual responsibility, and targeting towards those "who need it most". The support of central government for privatised provision of food in schools not only fails to address the rights of children but has opened the way for schools to become sites for corporate marketing and entrepreneurial private charities.[10] Troublingly, despite the laudable intentions of community organisations, there is evidence that this charitable provision of food in schools acts against the best interests of children it purports to help.[11]

As O'Brien and Wynd both observe, while there is a nascent 'politics of hope' in the food in schools debate, the dominant discourse of personal responsibility, along with the new interest in schools as marketing venues undermines the right to food that should be part of every child's bundle of rights and expectations. After more than 20 years of foodbanks and breakfasts in schools, charity has failed to plug the gaping hole left by the inadequate incomes of many thousands of New Zealand families. Indeed, charities cannot be expected to ensure adequate incomes. In the end, it is improved families' incomes that will ensure that children's right to food is secured and government has the key role here.

Conclusion

An estimated 80,000 New Zealand children go without breakfast every day. While the government has put in some money to help Fonterra, Sanitarium and KidsCan extend their existing programmes, it falls short of the need, particularly as the number of children in very low-income households continues to rise. Implicitly blaming parents (and suggesting this gap can be filled by parenting and budgeting programmes) demeans and insults many thousands of parents who are highly motivated to do the best by their children, and often work long hours to accomplish this.

In light of New Zealand's persistently high rates of child poverty, food in schools programmes (and other assistance for low-income households) need to be expanded. Tighter targeting moves away from provision based on the rights of children, and may result in children from disengaged or hard-to-reach families falling through the gaps. Likewise, shifting funding from one needy group to another group (arbitrarily designated as a more needy group) is a zero-sum game.

As a matter of equity, greater resources must be provided to children in low-decile schools. The alleviation of hunger and recognition of children's right to adequate food is sufficient justification.

Note: The views and statements of the authors do not necessarily reflect NZMA policies, unless stated as such.

Competing interests: Nil.

Author information: Donna Wynd, Researcher, Child Poverty Action Group, Auckland; Mike O'Brien, Associate Professor Counselling, Human Services and Social Work, University of Auckland, Auckland

Correspondence: Donna Wynd, Child Poverty Action Group, PO Box 56 11, Wellesley St, Auckland 1141, New Zealand. donna.wynd@gmail.com

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EDITORIAL

Are New Zealand's cancer services second best?

Brian Cox

Comparisons of population-based relative survival for cancer between Australia and New Zealand using cancer registry data are provided in this issue of the *NZMJ*.[1] For most cancer sites relative survival was higher in Australia than in New Zealand. Intriguingly, the difference between the countries in 1-year relative survival was usually greater than for 5-year or 10-year relative survival.

New Zealand and Australian cancer survival was compared using a technique based on a period of time, rather than the usual approach of following a cohort of patients from diagnosis.[1,2] Numerous uncontrolled factors can influence population-based relative survival studies. Therefore, it is necessary to understand the method used, and its strengths and weaknesses,[2] when interpreting the results. The Australian period-based relative survival ratios were obtained from a publication of the Australian Institute of Health and Welfare. The same Ederer II method[3] was used for the New Zealand estimates but the preparation of the data is not described and the lack of age-standardisation for the comparison a weakness.[4]

The focus on population-based cancer survival analysis in New Zealand was triggered by the observed difference in mortality to incidence ratios between Australia and New Zealand.[5] Although cancer survival estimates are frequently used as an assessment of diagnostic and treatment services, population-based survival analysis using cancer registry data actually assesses the combined effects of the time to the presentation of symptoms, changes in disease classification, the adequacy of diagnostic services, the timeliness of diagnosis and treatment, the effectiveness of primary therapy, and the treatment of recurrent disease and metastases. Population-based survival analyses are descriptive only and can be useful for monitoring cancer services, but are seldom able to differentiate among the sources of discrepancies in survival observed.

The relative survival ratio is the ratio of the probability of the survival of people who develop the disease of interest relative to that expected in the relevant population in which they were living at the time of their diagnosis. Overall mortality is usually used to approximate the mortality rate of all other causes of disease when the mortality from the disease in the general population is very low. Shorter overall life-expectancy of the reference population produces a lower relative survival for cancer. Thus, relative survival for cancer is usually significantly reduced with increasing age making adjustment for age desirable in population group comparisons.[4] In addition, the estimation of relative survival assumes that survival from cancer is unrelated to survival from other diseases, which in the case of smoking-related cancers, for example, may not be valid.

Among the Australia states there are differences in registry methodology and data definitions. In addition, a wider number of sources provide data to many of the State registries than occurs in New Zealand. For example, the NSW Cancer Registry receives information from; pathology laboratories, radiotherapy and medical oncology departments, hospitals, multi-purpose services, forensic medicine, residential aged-care facilities, and day procedure centres. In addition, specialist registers also provide registration information in many states of Australia.

A key variable for relative survival analysis is the date of diagnosis. Multiple sources of information are likely to result in the recording of an earlier date of diagnosis than pathology reports alone, particularly for cancers diagnosed clinically. In addition, some of the Australian state cancer registries allow an estimated date of diagnosis to be entered by the clinician with a flag indicating when this occurs, whereas this is not a feature of New Zealand cancer registration.

The examination of the differences in incidence and mortality between population groups should, ideally, assess cancer in patients from its origin to death or end of follow-up. Unfortunately, the

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assessment of survival from a relatively random point in time, the date of diagnosis, limits its ability to explain differences in the relationship between incidence and mortality.

For example, if it takes longer for a patient referred with suspected cancer to be seen by a specialist in New Zealand compared to Australia, then 1-year survival after diagnosis will be better in Australia solely because the diagnosis was made sooner. However, the earlier diagnosis would result in less of a direct effect on 5-year and 10-year survival, as has been observed by Aye and colleagues.[1] However, as delay is biologically linked to increased severity, longer-term survival may be compromised. The relative survival advantage at 1-year in Australia across most cancer sites suggests that greater diagnostic delay occurs in New Zealand.

For some forms of cancer, melanoma, myeloma and haematological malignancies, for example, the pathological confirmation of cancer may be less likely to require referral to a specialist. Therefore, it is noteworthy that for melanoma and myeloma, little difference in 1-year relative survival was observed.

If the average delay from presentation to diagnosis was 4 weeks longer in New Zealand due to delay in presentation by the patient, experimentation with alternative therapy, or difficulty in diagnosis by the doctor, the 1-year relative survival would be about 7% poorer compared to Australia. The range of delay among patients is even more important and if even relatively few patients have considerable delay this can greatly influence overall relative survival due to a lower chance of cure. Conversely, where treatment is seldom effective, 1-year survival may be affected by delay but it may have little influence on long-term survival differences. This was apparent for trans-Tasman differences in relative survival for cancers of the pancreas, brain and stomach.[1] However, relative survival for non-Hodgkin lymphoma was uniformly poorer in New Zealand suggesting features other than delay in diagnosis are important.

In period-based relative survival the 10-year relative survival does not apply to the same population as the 5-year relative survival or the 1-year estimate.[6] For example, the increased difference in 1-year, 5-year and 10-year relative survival for breast cancer appears to represent the slightly later introduction of organised breast screening in New Zealand (December 1998) compared to Australia (1991). The three main influences of screening, lead time, length bias, and overdiagnosis,[7] are likely to explain the higher 10-year and 5-year, 2006-10 period-based, breast cancer relative survival across the Tasman, as many of the diagnoses in these patients would have occurred between 1991 and 1998, whereas the 1-year estimates are influenced more by recent diagnoses.

If New Zealand has not achieved the improvements in cancer survival seen in other countries then audits of disease presentation, diagnostic delay (both patient-related and doctor-related delay), primary treatment, and the follow-up management of cancer patients are warranted. The Faster Cancer Treatment Indicators initiative of government suggests that delay in the time from referral to treatment was thought to contribute to our relatively poor cancer mortality rates compared to Australia. However, independent review by the Auditor-General found the definitions of the indicators to be ambiguous, interpreted differently, and often unable to be calculated accurately by District Health Boards.[8] Despite a shift of resources to meet these targets, no assessment of their effectiveness appears to have been included as part of their implementation.

The results of the comparison of relative survival between Australia and New Zealand in this issue of the *Journal* suggest that all delay in obtaining a diagnosis and not just the time from receiving the referral letter of the general practitioner to treatment needs to be monitored.

It is very important for cancer control that there is adequate support for general practitioners to investigate potential cancer symptoms. Nationwide ease of access to imaging techniques and biopsy services, as well as timely assessment by specialists of referred patients, is essential for patients with cancer. This may have been severely compromised by the historical reductions in outpatient services as hospitals have attempted to reduce costs. A lack of timely specialist assessment can lead to

increased presentation to emergency departments of cancer patients in medical crisis with consequent limited options for curative treatment.

Failure to treat with curative intent in those for whom it appears possible[9] can result in long protracted periods of care before death from the disease. For those who die of cancer, the cost of ongoing care until death often greatly exceeds the cost of primary treatment.

In a hospital cost-cutting climate, appropriate medical care can be difficult for doctors to provide, and the needs of medical staff difficult for management to acknowledge, particularly for expensive cancer treatment. It is important that the structures and decisions of management do not unduly affect the ability of the specialist and the general practitioner, along with the staff of the available ancillary secondary care services, from acting as a team in the care of the cancer patient so that delays in diagnosis and treatment are minimised.

The greatest improvements recently in cancer survival have occurred among older cancer patients. An 80 year-old man or woman in New Zealand will, on average, live until aged 88.5 or 89.8 years, respectively, but their life-expectancy is 1.6% and 4% less than their trans-Tasman counterparts. If age, or a false perception of life expectancy, unduly influences whether curative therapy is offered, improvements in cancer survival may not keep up with other countries and could even decline.

Australia has been reported to have some of the highest cancer survival rates in the world[10] and if New Zealand is second best to Australia that may be acceptable. However, considerably lower relative survival, consistent across most cancer types does suggest that our cancer services may not even be second best.

Competing interests: Nil.

Author information: Brian Cox, Director and Research Associate Professor, Hugh Adam Cancer Epidemiology Unit, Department of Preventive and Social Medicine, Dunedin School of Medicine, University of Otago, Dunedin

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Correspondence: Associate Professor Brian Cox, Hugh Adam Cancer Epidemiology Unit, Department of Preventive and Social Medicine, PO Box 913, Dunedin, New Zealand. brian.cox@otago.ac.nz

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ORIGINAL ARTICLE

Comparison of cancer survival in New Zealand and Australia, 2006–2010

Phyu S Aye, Mark Elwood, Vladimir Stevanovic

Abstract

Background and aims Previous studies have shown substantially higher mortality rates from cancer in New Zealand compared to Australia, but these studies have not included data on patient survival. This study compares the survival of cancer patients diagnosed in 2006–10 in the whole populations of New Zealand and Australia.

Method Identical period survival methods were used to calculate relative survival ratios for all cancers combined, and for 18 cancers each accounting for more than 50 deaths per year in New Zealand, from 1 to 10 years from diagnosis.

Results Cancer survival was lower in New Zealand, with 5-year relative survival being 4.2% lower in women, and 3.8% lower in men for all cancers combined. Of 18 cancers, 14 showed lower survival in New Zealand; the exceptions, with similar survival in each country, being melanoma, myeloma, mesothelioma, and cervical cancer. For most cancers, the differences in survival were maximum at 1 year after diagnosis, becoming smaller later; however, for breast cancer, the survival difference increased with time after diagnosis.

Conclusion The lower survival in New Zealand, and the higher mortality rates shown earlier, suggest that further improvements in recognition, diagnosis, and treatment of cancer in New Zealand should be possible. As the survival differences are seen soon after diagnosis, issues of early management in primary care and time intervals to diagnosis and treatment may be particularly important.

Cancer survival, the survival of cancer patients from the time of diagnosis, is the key indicator used to assess the effectiveness of cancer care in diagnosis and treatment. In principle, if equivalent cancer care at an equivalent time is provided for patients with the same cancer and the same background health status, the outcomes for cancer patients should be similar regardless of variations in geography, ethnicity, or socioeconomic position. Thus differences in cancer survival may reflect possible deficiencies in cancer care and indicate the potential for improvement in cancer care services.[1–3]

Differences in cancer survival are substantial, between and within countries. It has been estimated that 6.5% of cancer deaths in Britain could have been avoided annually if Britain's cancer survival had been equal to the mean European level during 1995–99.[4] A study of Nordic countries estimated that 2.5% of deaths from 12 cancer sites between 2008 and 2012 could be saved by eliminating the regional variations.[5] Variation in 3-year survival between deprived and affluent groups accounted for 11% of cancer deaths in England during 2004–06.[6] These findings signal that discrepancies in cancer survival should be investigated.

Comparisons of cancer survival to investigate international differences have been conducted in Europe, e.g. by the EUROCARE studies [1], and extended globally, e.g. the CONCORD studies [2]. The published CONCORD studies have involved Australia, but not New Zealand, although New Zealand is involved in the current studies. Internal comparisons of cancer survival have been reported addressing variations with regard to ethnicity, geography, and socioeconomic position in both New Zealand[7–11] and Australia.[12–14] New Zealand and Australia have taken part in recent OECD health policy studies based on exploratory survival analysis of four selected cancers (breast, cervix, colorectum, and lung) among OECD countries [15].

New Zealand and Australia have similar data systems and health care systems, and their training programmes for health professionals are similar and often integrated. Despite that, New Zealand had a higher cancer mortality rate than Australia in 1996-97, and the mortality to incidence ratios for New Zealand were higher for many cancer sites.[16] A more recent study of 2000–07 data found a persisting discrepancy, despite reductions in overall cancer death rates in a both countries; overall cancer mortality was 15.1% higher in women, and 4.7% higher in men, in New Zealand compared with Australia [17]. These studies had no survival data. For this analysis, we explored the differences in cancer survival between the two countries using whole-population survival data for 2006–10.

Methods

Survival data for Australia were from the Australian Institute for Health and Welfare (AIHW).[14] These data relate to all new primary cancers, excluding basal cell and squamous cell carcinoma of the skin, diagnosed in Australia and recorded by state and territory cancer registries, with standardised coding practices to minimise errors and duplications [14]. New Zealand data were from the New Zealand Cancer Registry (NZCR), with similar coverage and quality controls [18], and required specific analyses to produce data for the same time periods given in the Australian data.

For both countries, relative survival ratios (RSRs) yearly from 1 to 10-year time points with corresponding 95% confidence intervals (95%CIs), for the whole population in each country, were extracted by type of cancer and sex for patients registered (diagnosed) in 2006-10, using a period approach.[19;20] The expected survivals by year, age, and sex for the whole population of each country were derived by the Ederer II method, used in recent US studies [21]. Cancer sites were coded using ICD-10 for site[22] and ICD-O for morphology [23] in both countries; comparisons were made for 24 sites in men and 26 cancer sites in women, although only sites accounting for more than 50 deaths per year in New Zealand are presented here.

The main comparisons shown are for three time points; 1, 5, and 10 years. Conditional survival ratios from 1 to 5 years, and from 5 to 10 years, were also calculated, but not shown. The differences of RSRs between the two countries were calculated for each site of cancer, for men and women separately, and the statistical significance of the difference determined by a z test. The age distributions were compared between the two countries, by 5 year age group in each sex, for all cancers and for the most common cancers, and found to be virtually identical; thus, age adjustment was not used in the survival analyses.

The numbers of 'potentially avoidable' deaths were estimated, using the terminology and method defined in other studies [4], for total cancer at all ages. Here it is the difference in numbers of deaths from cancer that occur within 5 years of diagnosis in New Zealand, and the (lower) numbers that would occur if the survival rates had been equal to the Australian rates. To make this estimate, the expected non-cancer mortality was assessed from New Zealand life tables applied to the 5-year age distributions of cancer patients by sex, and the observed relative survival ratios in each country used.

Results

Survival for all cancers combined shows that in both men and women, survival ratios up to 10 years from diagnosis were higher in Australia (Figure 1), with 5 year relative survival ratios in males of 65% in Australia and 61% in New Zealand; and in females 67% in Australia and 63% in New Zealand (both comparisons $p<.01$). [Relative survival represents cancer survival in the absence of other causes of death; the relative survival ratio is the observed survival of a group of cancer patients at a given time from diagnosis, divided by the expected survival of a sex- age- and time- matched group from the general population; thus adjusting for the expected mortality without cancer].

Figure 1 shows that the differences in survival occur early; at 1 year from diagnosis there is a significantly higher survival in Australia, and that difference is little changed in later years, showing that survival ratios after the first year are similar in the two countries.

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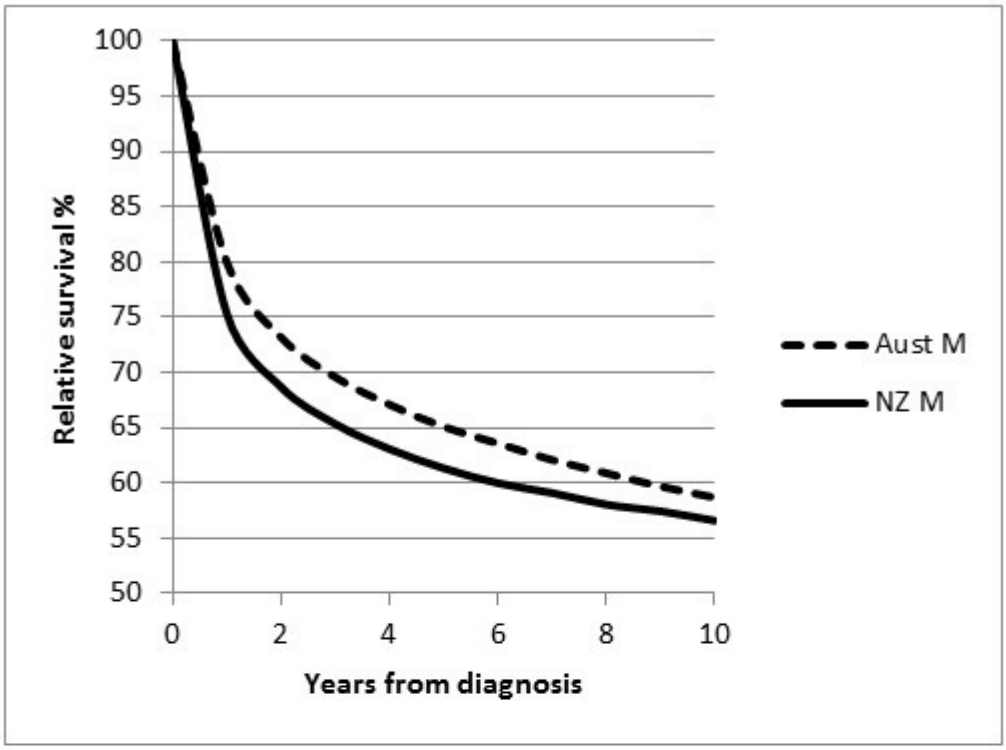
Figure 1: Survival (relative survival ratios) for all cancer combined, in males and females, in New Zealand and in Australia, patients diagnosed in 2006-2010



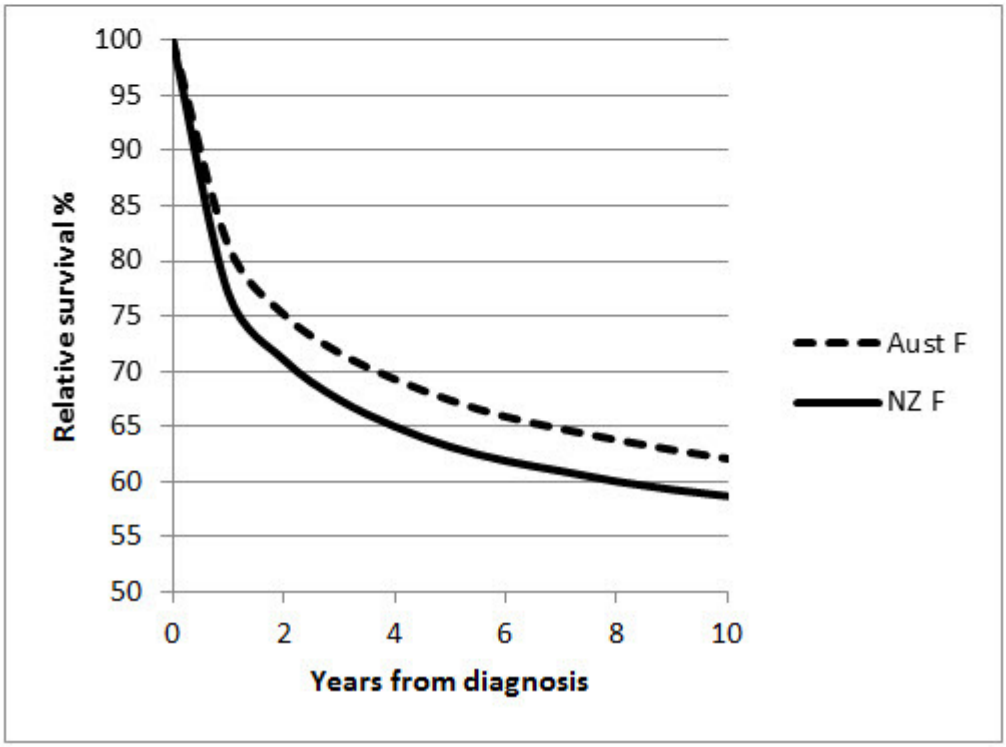


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Table 1: Cancer relative survival ratios in New Zealand, by time from diagnosis, and differences from Australia

Cancers in order by related annual numbers of deaths

Sex	cancer site	NZ deaths annual (2008)	New Zealand relative survival rates						Difference from Australia rates (bold/ul if significant)			Pattern
			1 yr	5 yr		*5 yr 95% limits*		10 yr	1 yr	5 yr	10 yr	
female	all cancers	4005	77.2	63.2	(	*62.7*	*63.7*	) 58.7	-4.4	-4.2	-3.4	Max diff yr 1, reduces but still sig
male	all cancers	4561	75.4	61.3	(	*60.8*	*61.9*	) 57.0	-4.6	-3.8	-2.0	Max diff yr 1, reduces but still sig
female	lung	745	32.1	10.6	(	*9.6*	*11.6*	) 8.3	-10.4	-6.0	-4.0	Max diff yr 1, reduces but still sig
male	lung	889	25.8	8.5	(	*7.7*	*9.4*	) 7.0	-10.2	-4.1	-2.0	Max diff yr 1, reduces but still sig
female	colorectal	580	78.2	62.2	(	*60.7*	*63.7*	) 58.6	-6.1	-4.9	-3.7	Max diff yr 1, reduces but still sig
male	colorectal	684	79.7	60.4	(	*58.9*	*61.8*	) 55.0	-5.8	-5.0	-4.0	Max diff yr 1, reduces but still sig
female	pancreas	197	12.6	4.3	(	*3.2*	*5.6*	) 4.2	-7.9	-1.3	0.0	Max diff yr 1, later ns
male	pancreas	176	14.1	4.7	(	*3.5*	*6.1*	) 5.0	-8.7	-0.2	0.9	Max diff yr 1, later ns
female	melanoma	115	98.4	93.8	(	*92.6*	*94.9*	) 92.9	-0.1	-0.2	-1.5	No sign diffs
male	melanoma	202	96.3	88.2	(	*86.8*	*89.5*	) 85.0	-0.1	-0.4	-0.1	No sign diffs
female	stomach	110	41.0	22.8	(	*19.4*	*26.4*	) 21.3	-9.1	-3.6	-2.5	Max diff yr 1, later ns
male	stomach	173	43.7	24.2	(	*21.5*	*27.0*	) 23.0	-7.5	-2.7	0.0	Max diff yr 1, later ns
female	NHL	134	75.7	64.0	(	*61.2*	*66.8*	) 54.8	-7.1	-7.3	-8.4	Diff constant or increase
male	NHL	160	79.6	65.2	(	*62.6*	*67.8*	) 57.0	-4.1	-4.8	-4.0	Diff constant or increase
female	brain	98	41.8	23.1	(	*19.6*	*26.7*	) 18.2	-5.0	-0.8	-1.7	Max diff yr 1, later ns

male	brain	109	41.6	18.5	(	*15.7*	-	*21.5*	)	15.0	-5.4	-1.9	-1.0	Max diff yr 1, later ns
female	oesophagus	75	36.9	11.0	(	*8.0*	-	*14.5*	)	7.4	-6.9	-6.0	-5.7	Max diff yr 1, reduces but still sig
male	oesophagus	154	35.5	10.3	(	*8.2*	-	*12.7*	)	9.0	-8.1	-5.2	-3.0	Max diff yr 1, reduces but still sig
female	bladder	66	61.0	45.7	(	*40.7*	-	*50.8*	)	44.5	-8.9	-3.9	0.7	Max diff yr 1, later ns
male	bladder	134	72.5	53.1	(	*49.9*	-	*56.3*	)	49.0	-9.2	-6.9	-3.0	Max diff yr 1, later ns
female	liver	66	22.4	13.1	(	*9.9*	-	*16.8*	)	8.7	-14.7	-2.3	-3.6	Max diff yr 1, later ns
male	liver	124	31.0	12.9	(	*10.5*	-	*15.6*	)	12.0	-7.1	-2.6	1.0	Max diff yr 1, later ns
female	kidney	67	78.6	67.4	(	*63.3*	-	*71.3*	)	64.2	-5.3	-5.1	-2.6	Max diff yr 1, later ns
male	kidney	98	78.7	62.2	(	*59.0*	-	*65.3*	)	55.0	-5.9	-9.4	-9.2	Diff constant or increase
female	myeloma	68	75.9	39.0	(	*34.4*	-	*43.7*	)	24.6	-1.7	-3.8	0.2	No sign diffs
male	myeloma	96	76.1	43.5	(	*39.2*	-	*47.8*	)	26.0	-2.8	-0.4	2.0	No sign diffs
male	prostate	670	96.5	90.3	(	*89.5*	-	*91.2*	)	85.0	-1.3	-1.7	0.0	Max diff yr 1, later ns
male	mesothelioma	65	40.1	3.4	(	*1.7*	-	*5.9*	)	3.0	-3.5	-2.0	1.0	No sign diffs
female	breast	618	97.2	86.6	(	*85.9*	-	*87.4*	)	79.7	-0.7	-2.8	-3.5	Diff constant or increase
female	ovary	184	64.9	35.9	(	*33.2*	-	*38.5*	)	31.0	-11.6	-7.5	-3.0	Max diff yr 1, reduces but still sig
female	uterus	82	90.2	77.5	(	*75.2*	-	*79.7*	)	75.5	-2.6	-4.5	-3.0	Max diff yr 1, later ns
female	cervix	59	86.3	71.0	(	*67.5*	-	*74.3*	)	67.9	-0.3	-1.1	-0.7	No sign diffs

Explanations of descriptions:

Max diff yr 1, reduces but still sig	Maximum NZ-Australia difference at year 1, reduces at year 5 or 10, but remains significant
Max diff yr 1, later ns	Maximum NZ-Australia difference at year 1, reduces at year 5 or 10, becomes non-significant
Diff constant or	Difference ay year 1 remains similar or increased at year 5 or 10

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increase	
No sign diffs	No differences at years 1, 5, or 10 are significant
NHL = non-Hodgkin lymphoma	

Compared to Australia, cancer survival at 1 year is lower in New Zealand by 4.4 % in females and by 4.6% in males; these differences persist, with some reduction, at 5 and 10 years from diagnosis (Table 1).

The conditional survivals after 1 year (that is, the survival over further time of those who have survived 1 year) were very similar: of those surviving 1 year, 76% of females survived to 10 years in both countries; in males, 74% in Australia, 76% in New Zealand.

Results for major cancers, those accounting for 50 or more deaths per year in New Zealand, are shown in Table 1. The 1, 5, and 10 years' survival in New Zealand are shown, with the confidence limits at 5 years (the width of the limits is similar at other time points), and the differences from Australia, in absolute terms.

Most cancers show the pattern seen with all cancers combined: that is, survival in New Zealand is significantly ($P<0.01$) lower than in Australia at 1 year, and the difference persists at 5 and 10 years, with a similar or smaller difference in absolute terms. For lung, colorectal, and oesophageal cancer, in both sexes, and for ovary, the differences remain significant. For many other sites, the same pattern is seen, but the differences become non-significant at 5 or 10 years. This of course is partially due to the numbers of observations reducing. This pattern is shown by stomach, brain, pancreas, liver, and bladder cancer, in both females and males; and in prostate, and uterus (endometrial) cancer. The differences in survival at 5 years are up to 7 percent.

A few cancers show no inter-country differences, with similar survival ratios at all time-points: melanoma and myeloma in both sexes, cervical cancer, and mesothelioma in men (where there were too few cases to assess in women).

Breast cancer is unusual, as the lower survival in New Zealand increases over time; 1 yr. survival is 97.2% in NZ and 97.9% in Australia, but 10 yr. survival is 79.7 and 83.2% respectively. For non-Hodgkin lymphoma in both sexes, the survival difference remains similar from 1 to 10 years. Kidney cancer is the only site showing different patterns in the two sexes: in males the survival difference increases by time of follow up, while in females the difference decreases.

So while survival from melanoma, myeloma, mesothelioma in men, and from cervical cancer is similar in the two countries, for every other major cancer survival in New Zealand is lower, especially in the first year.

Discussion

In this study, we found that the survival from all cancers combined in New Zealand (NZ) was significantly lower than in Australia, for both men and women, at 1 year and up to 10 years after diagnosis, for patients diagnosed in 2006-2010. The 5-year relative survival ratios in NZ were 3.8% lower (61.3% vs 65.1%) for men, and 4.2% lower (63.2% vs 67.4%) for women, than in Australia. These numbers may appear small; however, this difference in survival represents a substantial number of deaths.

In New Zealand, the difference for all cancer combined equates approximately to 341 deaths annually in men in New Zealand, and 364 deaths in women, calculated as deaths from cancer in 5 years from diagnosis, taking into account background mortality from other causes [4]. This represents 11.7% of cancer deaths within 5 years of diagnosis in men, and 12.1 % in women. These estimates are approximate and may be conservative; a fuller assessment would use ethnic- and age-specific comparisons. However, these estimated proportions are considerably greater than those estimated for Britain in 1985-89, using the average European relative survival ratio as the comparator, 6–7% [4]: a result which was a trigger for major cancer health systems reform in England.[24;25]

The generally lower survival shown in New Zealand raises the question of differences in how the data has been collected, coded, or analysed. While a full audit would be needed to rule this out, the systems of cancer registration, death coding, and linkage of cancer registry and mortality data are the

same in the two countries, as far as we can determine; and the methods of survival analysis used are the same. Relative survival ratios are based on deaths from all causes in cancer patients, and deaths expected in that year-age-sex group in the whole population, so do not depend on the cause of death recorded in the death certificates.

The same issues of data quality have been extensively studied in European and world-wide comparisons of similar data systems [1;2;26;27]. In our study, comparing the whole populations of each country, the age distributions of patients for all cancer combined and for major cancer sites (lung, colorectal, prostate, breast, melanoma) between NZ and Australia were found to be virtually identical; thus, the survival differences were not due to different age distributions.

In international comparisons, Australia shows very good overall cancer survival outcomes, similar to those from Canada and Sweden, and better than those in the UK or Denmark.[2;28] Cancer survival has improved substantially in both Australia and New Zealand over recent years.[14;29]

The lower survival in New Zealand than in Australia is seen for most cancers, including the leading causes of death of lung and colorectal cancer, and for 14 of the other 18 cancers accounting for over 50 deaths per year in New Zealand (Table 1). This suggests a health system issue, rather than a biological or treatment issue specific to certain types of cancer.

It is easy to say that the NZ deficit in survival means that NZ cancer care could be improved to match the Australian processes and outcomes, and we could regard the differences in cancer survival as representing 'avoidable deaths' in New Zealand. This terminology has been used in arguing for improvements in the cancer care in the UK [4;30]. However, specifying the changes needed in New Zealand, and prioritising these with regard to costs and effectiveness, is more challenging. However, the demonstration of these substantial and general survival deficits compared to a neighbouring country should stimulate both local and national, clinical and health management, attention and actions.

This study on cancer survival comparisons complements the previous studies on cancer mortality comparing the two countries. In a similar period 2000–2007, NZ had substantially higher overall cancer mortality than Australia, an average of 5% more deaths in NZ men and 15% in NZ women each year; while overall cancer incidence for NZ men was 5% less than that for Australia, and incidence for NZ women was only slightly higher (3%) for Australian women.[17] Thus, the differences were mainly found to be in mortality, implying differences in survival. These differences were only slightly reduced compared to an earlier period, 1996-97.[16]

The modest differences in incidence imply that the two countries were not greatly different in cancer primary prevention. However, the lower survival in NZ found in the current study, supported by the differences in cancer mortality, implies that NZ is lagging behind in diagnosis and treatment. While the current survival study and the previous mortality studies all show less good outcomes in New Zealand, a difference is that the current study gives similar results for males and females, whereas the mortality studies showed greater differences in females. The recent mortality study related to cancer deaths in 2000–-2007, and therefore to cancers diagnosed in earlier years. The current study relates to cancers diagnosed in 2006-2010, so is more recent; the similarity between male and female results may better reflect the present situation.

The pattern of the survival differences seen for most cancers, being apparent in the first year from diagnosis and continuing at 5 and 10 years without much change, strongly suggests the reasons relate to diagnosis and initial presentation, relating in turn to awareness of symptoms, time intervals to referral, investigation, and diagnosis. These aspects will affect when the cancer is diagnosed in its biological progression, determining its stage distribution.

This pattern of survival differences has also been seen in European and world-wide comparisons.[1,2] As a response, efforts to improve the early management of patients in primary care and through referral processes have been made. These include studies of cancer presentation in primary care ,[31-33]

studies of the diagnostic time intervals [34] ('delays', although that word can be pejorative and maybe should be avoided), and studies of health system issues related to these. [35] A major international focus is through the International Cancer Benchmarking Project (ICBP), which is conducting studies in primary and secondary care to clarify reasons for international variations in cancer survival. [28;36;37].New Zealand is involved in one part of that work; we are conducting a study of primary care in relation to cancer diagnosis.[37]

Assessments in the UK have concluded that more premature cancer deaths can be avoided from small gains in survival for common cancers rather than large gains for uncommon cancers [30]. Other factors such as comorbidity are also relevant; comorbidity in common in cancer patients, and in New Zealand it contributes to ethnic differences in outcomes and to variations in treatment choices [38-40].

With regard to specific cancers, colorectal cancer showed the highest excess deaths in NZ compared to Australia in mortality studies,[17] and in this study, 5-year relative survival was 5% lower in NZ for both males and females. A Bowel Cancer Programme was set up by the NZ Ministry of Health in 2009 aiming at health service improvement in diagnosis, surveillance and treatment, and a pilot bowel cancer screening programme started in 2011.[41]

In Australia, bowel cancer screening has been available since 2006 [42] and it was stated in a 2010 report that 1,056 people had been detected with bowel cancer or suspected cancer through screening.[43] In New Zealand, the PIPER project (Presentation, Investigation, Pathways, Evaluation, Rx (treatment)) is a comprehensive management and outcome study of some 6000 colorectal cancer patients, which will indicate priorities for improvement .[44]

Lung cancer has low survival in both countries, but NZ is still at a disadvantage, lagging in 5-year survival ratios by 4% in males, and 6% in females. Prevention has been successful, with a reduction in NZ smoking rates by half;[45] however, the higher mortality and lower survival in NZ show potential for improvements in diagnosis and effective treatment. Lung cancer shows large differences between Maori and non-Maori in New Zealand[46;47] and was the subject of the first New Zealand service standards report.[45]

Prostate cancer had high survival ratios in both countries, but 1- and 5-year survival ratios were slightly lower in New Zealand. Prostate cancer survival can be considerably affected by overdiagnosis, related mainly to PSA screening, and incidence trends have shown divergence, with increases in Australia but decreases in New Zealand over the 2000-07 period;[17] so these differences are difficult to interpret.

For breast cancer, unlike bowel and lung cancers, survival difference increased with time, being 1%, 3%, and 4% lower in NZ at 1,5, and 10 years, respectively. This suggests that early diagnosis, including successful mammographic screening, may be comparable in the two countries but there may be differences in further treatment. Reductions in breast cancer mortality in Australia have been shown to be linked with the increased use of adjuvant hormonal and chemo-therapy [48].

Studies in NZ show that there are internal ethnic disparities, particularly for Maori, who are significantly more likely to have adverse survival in major cancer sites than other New Zealanders due to residing in deprived areas, late stage at presentation, longer waiting time from diagnosis to initial treatment, and lower curative treatment rates.[7;8;10;47;49]

A major analysis of survival trends from 1991-2004 in New Zealand showed lower survival in Maori and in low income groups, with little change in the ethnic differences over time, and some evidence of widening of the income-based differences [7]. Pacific peoples also show more disadvantage in many cancers.[50]

Clearly, improving NZ cancer care for early diagnosis and treatment of disadvantaged groups is a high priority. In this study we have compared the whole of each country. Australia also has the challenges of providing good care to disadvantaged groups, including its indigenous people and ethnic minorities, and has socioeconomic inequities and even greater geographic disparities than New

Zealand; cancer survival is similarly lower in indigenous peoples, and varies by geographic and socioeconomic factors;[12;51–53] although the indigenous population forms a much smaller proportion of the total than in New Zealand.

Australia has conducted many state based and several national audit studies of cancer management on population-based groups of patients, which have allowed comparisons of actual management with evidence-based guidelines, and may have stimulated improvements.[54] There have been few similar comprehensive studies of cancer management in NZ until recently; but the PIPER study noted above is more comprehensive than most Australian management studies.

Recent studies have found that economic factors such as total national expenditure on health, and number of CT scanners per million population, are significant predictors of survival outcomes when comparing countries. Setting targets and timeframes, monitoring, case management, and establishing cancer networks may all improve cancer outcomes.[15;55] Insightful studies on health system issues could also be beneficial for the development of further policies to strengthen NZ cancer care.

In conclusion, the survival differences found in this study are likely due to differences in diagnosis and treatment services. As significant differences were mainly found in initial years after diagnosis for most cancers, attention needs to be given particularly to aspects of early diagnosis.

Competing interests: Nil.

Author information: Phyu S Aye, MPH Student[1]; J Mark Elwood, Professor[1]; Vladimir Stevanovic, Principal Technical Specialist[2]

[1] Department of Epidemiology and Biostatistics, School of Population Health, University of Auckland

[2] Health and Disability Intelligence Unit, Ministry of Health, Wellington

Correspondence: Professor Mark Elwood. Email: mark.elwood@auckland.ac.nz

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ORIGINAL ARTICLE

Health and wellbeing of older Pacific Peoples in New Zealand

Fialupe Lotoala, Mary Breheny, Fiona Alpass, Annette Henricksen

Abstract

Aim Ethnic group membership has a robust association with health in New Zealand. To understand this relationship with particular attention to the health of older Pacific peoples, an analysis was undertaken using the Health, Work and Retirement Study.

Method The study was initiated in 2006 with 2-year re-assessment intervals. The sample consisted of 6653 individuals aged 55–70 randomly sampled from the electoral roll. Of these, 108 identified as belonging to a Pacific ethnic group. The rest of the sample comprised New Zealand European, Māori, Asian and other ethnic groups.

Results Older Pacific people scored lower on measures of physical and mental health, and reported higher rates of health conditions. The relationship between ethnicity and health was partially explained by lower socioeconomic status, less physical activity, and greater alcohol consumption. After controlling for multiple health risks, socioeconomic and demographic variables, ethnicity continued to predict lower levels of physical health, suggesting that there are other factors which contribute to higher rates of poor health for people of Pacific ethnicity. NZ-born Pacific people had better health than those born outside NZ.

Conclusion Taken together, this indicates that ethnicity intersects with structural variables which restrict access to resources related to good health and expose older Pacific people to risk factors for poor health. Combined with the effects of migration this contributes to health disparities found between Pacific and other ethnic groups in in New Zealand.

Ethnic group membership is associated with health outcomes in New Zealand.[1] In particular, Pacific people have lower life-expectancy than non-Māori, non-Pacific peoples (nMnP).[2] Disparities in health between different ethnic groups have been closely linked to differences in socioeconomic status (SES) and SES explains a large proportion of the differences in health outcomes between ethnic groups in New Zealand.[1,3] People with low SES have more fragile health, higher mortality, morbidity and disabilities rates, and are prone to certain diseases, cognitive impairment and depression.[4–6]

There are a variety of explanations of the relationship between SES and health.[7,8] Materialist explanations focus on the ways that increased resources enable greater access to goods and services as well as providing the capacity to limit exposure to risk factors. Psychosocial explanations focus on how social and economic risk factors are unequally distributed among the population, and these risk factors have biological and health consequences. Cultural and behavioural explanations focus on the differences in health-related behaviour among different cultural groups. These differences in health behaviour are viewed as a consequence of disadvantage, and unhealthy behaviours may be more culturally accepted among certain groups. Life course approaches view health inequalities as the result of the cumulative impact of social, psychological and biological advantages and disadvantages over time.

These explanations for the relationships between SES and health may work differently in different population groups and for different health outcomes.[9] To advance understandings of inequalities in the health of Pacific peoples we need to understand the mechanisms through which SES produces health inequalities.

The gradient in health according to SES holds with a variety of measures of SES, and examining the relationships between these measures, ethnicity, and health tells us about the social structure which underlies these health inequalities. To begin to evaluate the relative importance of these aspects of the relationship between SES and health in New Zealand, this study included a range of measures.

Measures of economic living standards, income, wealth, and assets gauge access to material resources and a strong relationship between these measures and health support materialist explanations for health inequalities. Differences in health outcome due to education and ethnicity reflect psychosocial explanations of unequal exposure to risk factors that influence health. By including health behaviours we can account for the contribution of cultural prevalence of negative health behaviours on later life health. Including a range of measures of SES and health behaviours in a study of the relationship between Pacific ethnicity and health enables a more nuanced understanding of inequalities in this population.

There has been much discussion on which measures best assess SES of an individual. Income has been widely established as the main positive determinant of health outcomes.[10–12] Measures of economic living standards also strongly predict health.[13] Likewise, educational qualifications, occupational status and wealth are also known to be key factors in determining health outcomes.[14] Education has been widely used in health-related research as a measure of SES and education is associated with health, independent of income and wealth.[14] It has been suggested that educated people have better knowledge of risky health behaviours, of preventive care and medical treatments, and are better able to utilize the health care system, and thus are less prone to ill health.[15]

Although occupational status is a key aspect of SES, it is a poor indicator of economic situation among older people as many in this age group have retired and many women in this cohort have not been regularly engaged in paid employment. Although there is evidence that individual health behaviours, such as smoking, alcohol consumption, and physical activity, affect health outcomes these behaviours are also socially structured and interact with ethnicity, SES, age and gender.[16]

New Zealand's older adult population is gradually increasing, in line with a global increase in the number of older people. Inequalities in health have been shown to persist and increase in older people, even after retirement from working life.[17] There is limited research on the health and well-being of non-clinical, community-based older Pacific peoples in New Zealand. In the next 15 years there will be a projected increase of more than 125% in the proportion aged 65 and over, relative to the overall size of their ethnic population,[18] thus older Pacific people represent an important focus for research on ageing and health.

The present study aimed to: a) describe the health and wellbeing of a community sample of older Pacific people in New Zealand, including examining any differences between NZ-born and Pacific Island-born participants; b) compare the health of this group to similar aged Māori and nMnP samples; and c) investigate the socioeconomic factors (e.g., economic living standards, income, education) and behavioural factors (smoking, alcohol consumption, physical activity) which may account for any disparities in physical and mental health among these groups.

The study involved a secondary analysis of wave one data from the HRC-funded Health, Work and Retirement (HWR) study (http://hart.massey.ac.nz/). This is a large-scale longitudinal study of New Zealanders aged 55-70 years, which began in 2006 and collects data biannually on health, physical activity, social support, work status and attitudes, retirement status and attitudes, socio-demographic information and whakapapa/whanaungatanga.[13,19,20]

Method

Participants

The sample consisted of 6653 individuals aged 55–70 randomly sampled from the electoral roll. Of these participants, 108 identified as belonging to either Samoan, Cook Island Māori, Tongan, Niuean or other Pacific ethnic groups. The rest of the sample comprised of 3120 New Zealand European, 3117 Māori, 118 Asian and

122 belonging to other ethnic groups. A full description of methodology and sampling for the wave one HWR study is described elsewhere.[21,22]

Measures

Health—Health was assessed using the SF36 health measure.[23] The SF36 consists of 36 questions and has eight subscales (physical function, role limitations for physical and emotional problems, pain, general health perception, general mental health, energy/vitality and social functioning). These were combined via principal components analysis with orthogonal rotation to derive coefficients forming two components (physical and mental health), with scores ranging from 0 to 100. Norm-based methods were employed to standardise summary scores using means, standard deviations, and factor score coefficients for the scales, resulting in physical health (*M*=50.15, *SD*=9.89) and mental health scores (*M*=50.15, *SD*=9.22), with higher scores reflecting better self-reported health. In addition, participants completed a chronic illness and disability checklist to assess the prevalence of specific health conditions. They were asked whether a nurse or doctor had ever told them they had any of a list of health problems including diabetes, heart trouble and asthma (see Table 3 for the conditions listed).

Health-related behaviours—Behavioural factors likely to influence health included alcohol consumption, smoking, and physical activity. Alcohol intake was measured by a summary of three alcohol questions from the AUDIT questionnaires[24] which assess the frequency of drinking for each participant. Physical activity was the total amount of time (in minutes) the respondent engaged in physical exercise within the previous seven days. Participants also indicated whether they smoked regularly.

Socioeconomic status (SES)—SES measures included the Economic Living Standards Index (ELSI[25]), along with education, income, assets, and wealth measures. The ELSI-short form[25] assesses restrictions in the ownership of household items (8 items), restrictions in social participation (6 items), the extent to which respondents economised to keep living costs down (8 items), and 3 self-rated indicators of living standards. Scores were combined and transformed[25] to form a composite score (0-31), with higher scores reflecting greater economic living standards (*M*=23.51, *SD*=5.99).

In the present study education was categorised into two levels: no post-secondary school qualification and at least one post-secondary qualification. Income was the summation of all personal income, before tax, for each respondent (for the 12 months prior to survey completion). It consisted of wages and salary, interest earned, NZ superannuation, other superannuation, insurance, and welfare benefits.

Assets were the sum of all assets self-reported by each respondent. It consisted of both financial assets (such deposits, financial investments) and non-financial assets (such home ownership, property, car, farm etc). A New Zealand treasury calculated wealth score was also included as a measure of economic position.[26]

Data analysis—Data were analysed using SPSS Statistics 21.0 software.[27] The dataset was weighted in order to match the New Zealand population. Descriptive analyses were conducted to examine and compare the health and wellbeing of the Pacific and other main ethnic groups (Māori, NZ European and Asian) within the sample, and to examine any differences between Pacific people born in New Zealand (NZ) and not born in NZ. Chi-square tests (for categorical variables) and independent samples *t*-tests (for continuous variables) with Bonferroni correction were conducted to evaluate differences across groups (e.g., ethnicity, location of birth). Regression analysis was employed to determine if the addition of ethnicity contributed to the prediction of physical health and mental health beyond that of socioeconomic and behavioural factors.

Results

Table 1 compares a range of health and SES factors across different ethnic groups. As can be seen, those indicating Pacific ethnicity had, on average, the lowest reported physical health and mental health scores of the four ethnic groups. They also had the lowest ELSI scores and were the least likely to have a post-secondary qualification. In regard to health-related behaviors, they reported the highest proportion of non-drinkers; however, they also reported the least amount of exercise per week and had the second highest proportion of smokers, following Māori.

New Zealand Europeans had the highest mean scores across physical health, mental health, income, wealth, assets, and ELSI, indicating better health and SES. However, they also had the highest proportion of drinkers than the other ethnic groups. Chi square (χ^2) tests performed on the categorical

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data were all significant at the 1% level ($p<0.01$), indicating that the differences between the groups across these variables are not likely to have occurred by chance.

Table 1. Comparison of health and socioeconomic status factors across different ethnic groups

	Pacific (n=147)			Maori (n=459)			NZ European (n=4952)			Asian (n=289)		
	Mean	SD	Valid %	Mean	SD	Valid %	Mean	SD	Valid %	Mean	SD	Valid %
Age	$60.1_{a,b}$	4.2		$60.4_{a,b}$	4.8		60.8_{a}	4.7		59.7_{b}	4.6	
Physical health	40.2_{c}	10.5		47.8_{b}	10.9		50.7_{a}	9.7		48.1_{b}	10.1	
Mental health	46.2_{b}	9.3		$47.9_{b,c}$	10.7		50.4_{a}	9.0		$49.9_{a,c}$	9.1	
ELSI	16.6_{c}	6.6		20.5_{b}	7.5		24.0_{a}	5.7		$23.2_{a,d}$	5.3	
Physical activity	320_{b}	422		347_{b}	391		$419_{a,c}$	371		442_{a}	395	
Gender												
Male			46% $_{a}$			48% $_{a}$			49% $_{a}$			53% $_{a}$
Female			54% $_{a}$			52% $_{a}$			51% $_{a}$			47% $_{a}$
Postsecondary qualification												
Yes			34% $_{b}$			39% $_{b}$			51% $_{a}$			60% $_{a}$
No			66% $_{b}$			61% $_{b}$			49% $_{a}$			40% $_{a}$
Alcohol use												
Non-drinker			59% $_{c}$			27% $_{b}$			13% $_{a}$			41% $_{d}$
Light drinker			25% $_{b}$			31% $_{b}$			41% $_{a}$			48% $_{a}$
Moderate-heavy drinker			16% $_{b}$			43% $_{a}$			46% $_{a}$			11% $_{b}$
Smoker identity												
Smoker			16% $_{a,b}$			22% $_{b}$			12% $_{a}$			3% $_{c}$
Non-smoker			84% $_{a,b}$			78% $_{b}$			88% $_{a}$			97% $_{c}$

Note: Values in the same row and subtable not sharing the same subscript are significantly different at $p<0.05$;

ELSI=Economic Living Standards Index.

Table 2 compares identified health problems across the ethnic groups. Pacific people had the highest reported rates of diabetes, respiratory conditions, stroke, ulcers, and chronic kidney or urinary tract conditions. High blood pressure was the most common health problem across all groups, with higher prevalence in Māori (50%) and Pacific (37%) participants. Māori were also found to have the highest proportion of heart trouble, hearing and vision impairments, and arthritis or rheumatism. NZ European participants had the highest proportions for cancer. χ^2 values for all the health problems were significant at the 1% level ($p<0.01$).

Table 3 shows a comparison of personal income, assets, and a treasury calculated wealth score across the ethnic groups. NZ Europeans had the highest mean and median values across all three economic measures (personal income, assets and wealth), indicating better economic position. Pacific participants were found, on average, to have the lowest assets and wealth. Although Asian participants reported the lowest mean personal income, Pacific had the lowest median income (along with assets and wealth). Median values were substantially lower than the averages, which is not surprising given the skewed nature of the economic data. It is interesting to note that median values indicated that at least half the Māori, Pacific and Asian participants reported no assets.

Mean differences between ethnic groups were tested using one-way ANOVA. Owing to the different group sizes and associated concerns regarding violation of the homogeneity of variance assumption, the Welch test was used to test equality of means. The test revealed significant differences in the mean income, wealth and assets across all ethnic groups at the $p<0.001$ level.

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Table 2. Comparison of health problems across the different ethnic groups

	Pacific		Māori		NZ European		Asian	
	Count	Valid %	Count	Valid %	Count	Valid %	Count	Valid %
Diabetes	52_{c}	39.3%	86_{b}	20.1%	315_{a}	6.6%	45_{b}	15.6%
Asthma	$39_{b,c}$	29.2%	87_{b}	20.5%	533_{a}	11.2%	23_{a}	7.9%
Respiratory conditions	28_{b}	22.1%	84_{b}	19.8%	450_{a}	9.5%	12_{c}	4.3%
Chronic kidney or urinary tract condition	20_{b}	14.8%	$44_{b,c}$	10.3%	182_{a}	3.8%	$17_{a,c}$	6.0%
Ulcers	18_{b}	13.3%	$21_{a,c}$	4.9%	197_{a}	4.2%	$30_{b,c}$	10.4%
Stroke	9_{a}	6.7%	20_{a}	4.8%	127_{a}	2.7%	6_{a}	2.2%
Blood pressure	$49_{a,b}$	37.1%	218_{b}	50.4%	1571_{a}	32.8%	92_{a}	31.8%
Arthritis or rheumatism	36_{a}	26.0%	167_{a}	39.0%	1602_{a}	33.5%	40_{b}	13.9%
Hearing impairment	7_{c}	5.0%	119_{b}	27.7%	984_{a}	20.7%	14_{c}	4.8%
Heart trouble	$21_{a,b}$	16.0%	97_{b}	22.7%	613_{a}	12.9%	$22_{a,c}$	8.0%
Sight impairment	$10_{a,b,c}$	7.4%	55_{b}	13.0%	366_{a}	7.7%	8_{c}	2.8%
Cancer	$5_{b,c}$	3.9%	$38_{b,d}$	9.0%	902_{a}	19.0%	6_{c}	3.9%

Note: Values in the same row not sharing the same subscript are significantly different at $p<0.05$.

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Table 3. Comparison of economic factors across the ethnic groups

	Pacific (n=147)	Māori (n=459)	NZ European (n=4952)	Asian (n=289)	*F*
Annual Income					
Mean	$26,905	$35,747	$53,868	$23,997	19.66
Median	$15,000	$30,000	$35,000	$17,400	
Wealth					
Mean	$184,455	$368,075	$705,977	$295,103	34.36
Median	$0	$135,000	$425,000	$220,888	
Total value of owned assets					
Mean	$102,499	$378,255	$514,362	$218,691	23.26
Median	$0	$0	$160,000	$0	

To further investigate the relationship between ethnicity and health controlling for socioeconomic factors, behavioural factors, and demographic variables a regression was conducted.The regression results are displayed in Table 4.

The impact of the demographic, socioeconomic and behavioural covariates was controlled for by entering them at the first step of each analysis. These control variables together accounted for 13.7% of the variance in physical health ($p<0.001$) and 13.1% of the variance in mental health ($p<0.001$); age, economic living standards and physical activity were the strongest unique predictors of health. Income, wealth and assets did not predict health over and above the strong effect of economic living standards.

When taking into account the demographic, socioeconomic and behavioural factors, ethnicity added to the explanation of health outcome, suggesting that there is a distinctive factor associated with Pacific ethnicity that is also likely to affect health outcomes. The addition of ethnicity to the equation resulted in a statistically significant (though small) increment in R^2 in both physical health and mental health models. Being of Pacific ethnicity was found to contribute negatively to physical health, but made no unique contribution to mental health.

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Table 4. Regression analyses predicting physical health and mental health

	Physical health				Mental health			
	Model 1	Model 2			Model 1	Model 2		
			95% CI				95% CI	
	Beta	Beta	LL	UL	Beta	Beta	LL	UL
Age	-.17 ***	-.17 ***	-.39	-.27	.08 ***	.08 ***	.10	.21
Gender (male/female)	.03	.03	-.11	1.06	.05 ***	.05 ***	.40	1.51
ELSI	.24 ***	.23 ***	.31	.42	.33 ***	.33 ***	.45	.55
Annual personal income	-.01	-.01	.00	.00	.02	.02	.00	.00
Wealth	.02	.02	.00	.00	.01	.01	.00	.00
Total value of owned assets	.00	.00	.00	.00	.00	.00	.00	.00
Physical activity (min/week)	.14 ***	.14 ***	.00	.00	.07 ***	.07 ***	.00	.00
Postsecondary qualification (no/yes)	.05 **	.05 **	.28	1.43	.02	.02	-.25	.84
Alcohol use	.09 ***	.08 ***	.66	1.52	.03	.04 *	.06	.87
Smoker (yes/no)	.00	.00	-.92	.87	.01	.01	-.67	1.03
EthnicityD1 (Non-European)		-.02	-1.92	.89		.09 ***	1.17	3.83
EthnicityD2 (Maori & Pasifika)		.01	-1.61	1.99		-.07 **	-4.08	-.67
EthnicityD3 (Pasifika)		-.04 *	-5.52	-.69		.01	-1.94	2.64
R^2	0.137	0.139			0.131	0.131		
F	58.23 ***	45.66 ***			55.62 ***	43.96 ***		
ΔR^2		0.002				0.003		
ΔF		3.37 *				4.53 **		

Note: *p<0.05; **p<0.01; ***p<0.001

Table 5 presents a comparison of Pacific participants born in NZ with those not born in NZ across different health and SES variables. As the table indicates, NZ-born Pacific participants have, on average, higher scores across most health and SES measures than those not born in NZ.

In regard to health, NZ-born Pacific people have significantly better physical health than those not born in New Zealand; however, there was no significant difference found in mental health scores, physical activity, or smoking or drinking behaviour. In terms of SES factors, NZ-born Pacific people reported significantly better economic living standards than those not born in New Zealand; although no significant differences were found in regard to personal income, wealth or assets.

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Table 5. Comparison of Pacific People born in NZ versus not born in NZ

	NZ born				Not NZ born				
	Mean	SD	n	Valid %	Mean	SD	n	Valid %	Sig.
Age	60.6$_a$	4.8	30		60.8$_a$	4.3	75		
Physical health	48.6$_a$	10.0	30		41.4$_b$	9.9	75		**
Mental health	48.2$_a$	7.8	30		44.9$_a$	10.7	75		
ELSI	21.4$_a$	6.7	30		15.3$_b$	7.6	75		***
Annual personal income	46,973$_a$	65,273	30		32,811$_a$	44,032	75		
Wealth	449,905$_a$	558,982	30		209,688$_a$	1,095,984	75		
Total value of owned assets	282,333$_a$	505,569	30		157,046$_a$	924,109	75		
Physical activity (min/week)	291.3$_a$	377.9	30		290.7$_a$	331.3	75		
Gender									*
Male			10$_a$	33.3%			44$_b$	58.7%	
Female			20$_a$	66.7%			31$_b$	41.3%	
Postsecondary qualification									*
Yes			13$_a$	48.1%			12$_b$	19.7%	
No			14$_a$	51.9%			49$_b$	80.3%	
Alcohol use									
Non-drinker			10$_a$	35.7%			37$_a$	52.9%	
Light drinker			10$_a$	35.7%			14$_a$	20.0%	
Moderate-heavy drinker			8$_a$	28.6%			19$_a$	27.1%	
Smoker identity									
Smoker			4$_a$	13.8%			17$_a$	23.0%	
Non-smoker			25$_a$	86.2%			57$_a$	77.0%	

Note: Values in the same row and subtable not sharing the same subscript are significantly different at $p<0.05$

Discussion

The findings of this paper support the existence of health disparity among different ethnic groups in older age. Comparing the four ethnic groups revealed significant differences in both physical and mental health. Overall, Pacific people had poorer physical and mental health, while New Zealand Europeans reported better physical and mental health than the other ethnic groups. In this study, the greatest contributor to this disparity in health is SES.

The results support previous findings on the poor health of Pacific people. A report from the Ministry of Health stated that between 2002 and 2004, the rate for new cases of stroke in Pacific adults was 318 per 100, 000 compared with 179 per 100, 000 for the total population. Pacific people have been shown to have poorer health outcomes compared to all ethnic groups.[18] These results are consistent with previous studies of health using the SF-36.[28–30] These differences have also been evident in other studies in New Zealand using different health outcomes,[3,31] including mortality.[32] Dulin[20] found even when controlling for multiple socio-contextual and physical variables, ethnicity continued to predict health status.

The lower SES of Pacific people explains much of their comparatively poor health status. Economic living standards was the strongest predictor of health, and less education also explained some of the difference in health outcomes. Consequently, this study supports the well-established finding in the literature of the existence of the strong inverse relationship between SES and health.[4,6,33–35] Consequently, older Pacific people are not uniformly disadvantaged in terms of their health; those

with higher economic living standards have better health. This supports Nazroo's[36] assertion that health disadvantage associated with minority ethnicity is not inevitable, but reflects differences in access to resources necessary for good health.

Similarly, Harris et al[37] found that differences in deprivation and experience of discrimination accounted for most of the difference in health between Māori and non-Māori. As the present study did not control for experiences of discrimination, it is possible that the additional variance in health associated with Pacific ethnicity may reflect experiences of discrimination. Experience of discrimination has been associated with poor health and negative experiences of healthcare utilisation among Māori.[31,38,39]

These findings provide a way to consider the materialist, cultural and behavioural, and psychosocial explanations for health inequalities in New Zealand. Improved economic living standards are associated with improved health, suggesting that access to material resources is a foundational element of health.

The relationship between Pacific ethnicity and some health behaviours suggests that there are perhaps both cultural differences in accepted patterns of health behaviour and also health conditions (such as stroke or gout causing reduced physical activity) which impact on later life health. Although the effect was small, ethnicity was related to health over and above SES and health behaviours. The additional unique contribution of ethnicity to health over and above these predictors suggests that there are additional psychosocial risk factors associated with Pacific ethnicity that influence health. It is important to identify and understand factors that protect and promote good health in this vulnerable group.

Although New Zealand has low rates of material hardship among older people in general, older Pacific people have the highest rates of hardship and the lowest economic living standards in New Zealand.[40] There is evidence that health and wellbeing is significantly poorer for older people who live in poverty, who are much more likely to be of Māori and Pacific ethnicity.[13,20] Lifetime experiences of poverty accumulate to produce inequalities in mortality in older age[41]. Moreover, Barrett et al.'s[33] re-analysis of the data from the living standards study by Fergusson, Hong, Horwood, Jensen, and Travers[42] revealed the consistent fragility of older people with lower SES.

Pacific elders had poorer health than all other ethnic groups. The indication that place of birth may have long term health impacts also suggests that life course understandings of health may be important for non-New Zealand born Pacific people. This study showed an interesting trend in that New Zealand born Pacific people had better health than those born in the Islands. We cannot make strong claims regarding whether Pacific ethnicity and place of birth explains an additional part of the variance in health status in older age due to the limited sample size. However, this provides some indication of what may be influencing the relationship between ethnicity and health.

Much of the work on ethnicity and health focuses on categorising *who is* a member of a particular ethnic group rather than understanding *what it means* to be a member of that group in a particular social context.[43] Among older Pacific people, those born in the Islands may subscribe to traditional views of health and health care, which may contribute to difficulties in accessing healthcare in New Zealand that recognises these understandings of health.[44] Findings such as this point to a more nuanced understanding of how membership of multiple intersecting groups contributes to health outcomes.

Due to the limited sample size, further research is needed with a much larger sample to compare health of New Zealand born Pacific and non-New Zealand born older Pacific people[45]. Examining this in detail will advance our understanding of the relationship between ethnicity, SES and health.[46] Evidence is increasingly pointing to the long term impact of early life events on later life health and wellbeing[8]. Place of birth and migration may have long term health impacts and tracking the early life events of the current cohort of older people will enable theoretical relationships between SES, ethnicity and health to be untangled with much greater sophistication.

As Lorant and Bhopal[7] suggest, thinking about the effect of social structural categories on health is best achieved by considering how category membership aligns with access to resources that influence health behaviour and health status. In terms of the present study, the health of Pacific peoples can be understood in terms of intersecting but non-overlapping categories of Pacific/non-Pacific and NZ born/non-NZ born, high English language proficiency/low English language proficiency. These categories are associated with different access to socially valued resources that influence health. Differences in health behaviours also reflect access to resources, with rates of cigarette smoking, alcohol consumption and physical exercise reflecting differences in education, employment, income, and deprivation[47]. Understanding health outcomes in this way could explain the variation in health-status risks between ethnic minority groups as due to exclusion from resources[7].

Although these results are consistent with previous studies of SF-36 as described above, there is a question of whether the SF-36 is an appropriate measure to assess health across different ethnic groups. Scott, Sarfati, Tobias, and Haslett[48] question the validity of the SF-36 summary scores (which attempt to separate mental and physical health as separate constructs) with regard to Māori and Pacific. They demonstrated that SF-36 summary scores did not fit Māori and Pacific ethnic groups' understandings of health and therefore comparative results using these measures should be treated with caution. Other ways of assessing health that acknowledge differences in conceptualising health and wellbeing across ethnic groups must be explored.

Although health was related to SES in this sample, some of the measures of SES used in this study have limitations when used with older people. Education has poor spread among people aged over 65. Income has been widely established as a main positive determinant of health outcomes.[10–12] However, Pollack et al[49] in their review of health studies suggested that, wealth relative to income is a more suitable measure due to its ability to absorb the effect of sudden loss or temporarily low income. In addition, Pollack et al.[49] and Huisman, Kurnst, and Mackenbach[50] mentioned that wealth is preferred to income when undertaking studies on older adults and retirees because of their relatively low or no income earned during this time. In this case, although income and wealth are positively correlated they are distinct as some older people may have relatively low income but substantial wealth[51]. Occupational status was excluded from the analysis, as its role is questionable given that those still in employment will be a minority.[14]

As SES has been shown to be the most important determinant of health it is vital to focus on improving the SES of Pacific elderly to improve their health. The study therefore highlights the need to identify policies that target the main determinants of health to reduce health disparity.[10] Future health and wellbeing of older Pacific peoples relies on improving employment opportunities, education and economic standard of living. Public education and awareness on healthy lifestyle and healthy living may also be contributing factors to improving health outcome for Pacific peoples, but differences in these are also patterned by SES.

This paper highlights the need to identify and understand factors that protect health and promote good health across all ethnic groups, in order to implement effective intervention for improving older people health outcomes.

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Author Information: Fialupe Lotoala, Research Assistant, School of Psychology, Massey University, Palmerston North; Mary Breheny, Senior Lecturer, School of Public Health, Massey University, Palmerston North; Fiona Alpass, Professor, School of Psychology, Massey University, Palmerston North; Annette Henricksen, Research Officer, School of Psychology, Massey University, Palmerston North

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Correspondence: Dr Mary Breheny, School of Public Health, Massey University, Private Bag 11 222, Palmerston North, New Zealand. M.R.Breheny@massey.ac.nz

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ORIGINAL ARTICLE

Late-onset rheumatoid arthritis in the Counties Manukau District Health Board region of New Zealand: an observational study of treatment

Laurence S G Teoh, Ravi Suppiah, Peter Gow

Abstract

Aim The aim of this retrospective study was to investigate whether there are differences in the early treatment of Rheumatoid Arthritis (RA) depending on the age of the patient at diagnosis.

Methods The electronic records of 127 newly diagnosed RA patients presenting to the Counties Manukau District Health Board Rheumatology outpatient clinic between January 2008 and December 2010 were reviewed. Demographics, disease severity, relevant investigations, and medication use were analysed using Pearson's Chi squared test, Fisher's exact test and *t*-test.

Results The cohort included 32 aged ≥60 years with Late Onset RA (LORA) and 95 aged <60 years with Young Onset RA (YORA). No significant differences in baseline disease severity, disease modifying anti-rheumatic drug or prednisone use rates were observed between the LORA and YORA groups, with methotrexate use rates of 26/32 (81.25%) and 74/95 (77.89%) respectively. Nonsteroidal anti-inflammatory (NSAID) rate was significantly lower (p=0.013) in the LORA group 14/32 (43.75%) compared to the YORA group 65/95 (68.42%), reflecting an awareness of the adverse effects of these drugs in an older population.

Conclusion Patients with new onset rheumatoid arthritis at our institution received similar disease modifying anti-rheumatic drug treatment irrespective of their age.

Rheumatoid arthritis (RA) is the most common inflammatory arthritis in older adults.[1] Ten to twenty percent of people with RA have late onset rheumatoid arthritis (LORA) and present with de novo disease after age 60.[1,2] RA can be treated with disease modifying anti-rheumatic drugs (DMARDs) such as methotrexate, either alone or in combination with other synthetic or biological DMARDs, depending on disease severity and individual patient factors.[3]

LORA patients have some different characteristics to young onset rheumatoid arthritis (YORA) patients, including a greater proportion of men affected, greater large joint involvement,[4] association with systemic symptoms,[5] longer duration of early morning stiffness and higher rate of myalgic onset.[6]

Patients with LORA and YORA derive similar reduction in disease activity, improvement of disability, and reduction in radiographic progression when treated with methotrexate, and also combination methotrexate and anti-tumour necrosis factor (anti-TNF) treatment.[7] Despite this, previous clinical studies comparing treatment of LORA and YORA patients demonstrate differences in RA treatment dependent upon age.[8,9]

The aim of this study was to investigate in a real world setting whether there are differences in the early treatment of RA depending on the age of the patient at diagnosis.

Methods

Study design—The study cohort included all patients with a new diagnosis of RA made at Counties Manukau District Health Board (CMDHB) Rheumatology outpatient clinic (OPC) between 1 January 2008 and 31 December 2010, who had at least two clinic visits. LORA was defined as a diagnosis of RA at or after 60 years of age. YORA was defined as a diagnosis of RA before 60 years of age.

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Patients were excluded from the cohort when their first Rheumatology appointment regarding RA occurred prior to 2008 or after 2010, or at an external (non-CMDHB) provider. Patients who were seen only once and patients who did not fulfil the American College of Rheumatology (ACR) European League Against Rheumatism (EULAR) 2010 RA classification criteria were also excluded.[10]

Data was obtained by the review of electronic medical records (clinic letters, electronic discharge summaries, relevant blood tests and radiology reports) from the first OPC until the 2-year follow-up OPC. The 2-year follow-up OPC was defined as the next follow-up OPC after a 2-year period from the first OPC and demarcated the end of the study period. If a patient did not remain in follow-up for at least 2 years then the last recorded OPC was accepted as the 2-year follow-up OPC for the study period.

Data recorded included age at first OPC, gender, ethnicity, and smoking status. Serologic status for rheumatoid factor (RF) and anti-cyclic citrullinated peptide (CCP), and fulfillment of 2010 ACR EULAR classification criteria were noted. Data recorded from first and 2-year follow-up OPC included date, tender joint count out of 28 joints (TJC28), swollen joint count out of 28 joints (SJC28), Erythrocyte Sedimentation Ratio (ESR), C reactive protein (CRP), presence or absence of early morning stiffness, and radiographic erosions. Composite 28 joint disease activity scores (DAS28 ESR 3 variable and DAS28 CRP 3 variable) were retrospectively calculated using this data where available.

Use of prednisone, nonsteroidal anti-inflammatory drugs (NSAIDs), and synthetic and biological DMARDs was recorded for two time periods, the first OPC time period (defined as time period from first OPC up to and including the next OPC), and the 2-year follow-up OPC time period (defined as time period from the first OPC up to and including the 2-year follow-up OPC, or last recorded OPC if the patient did not remain in follow-up for at least 2 years). A medication was recorded as used during a time period if it was used at any point during the defined period (even if it was subsequently stopped). Maximum tolerated dose of methotrexate during the study was recorded.

The binary presence of comorbidities in particular categories listed in the electronic clinical documents until the time of the first two rheumatology OPC clinic letters was recorded. The number of categories of comorbidities present for each patient was recorded. Categories assessed for binary presence were cardiac/cardiovascular, endocrinological, gastro/liver, gynaecological/obstetric, haematological, infection, malignancy, neurological, orthopaedic, otorhinolaryngological, psychiatric, pulmonary, renal, rheumatological (other than RA), or other category.

After analysis was completed for the LORA and YORA groups, a decision was made to conduct a post-hoc analysis of methotrexate and DMARD use by gender for those aged 15 to 45 years (roughly equating to child bearing age) to investigate if a lower use of methotrexate in females aged 15 to 45 years was a contributing factor to the first OPC methotrexate rates in the YORA group.

Ethics—Ethics approval was obtained from the Northern X Regional Ethics Committee (Ref No: NTX/12/EXP/068).

Statistical analysis—Data was collected and stored in an Access Database. Data validation and preparation was completed using Microsoft SQL Server 2012 Express. Statistical analysis was performed using SPSS and Excel.

Comparisons of proportions of binary outcomes between groups were made using Pearson's Chi-squared test and Fisher's exact test. Comparisons of continuous data sets between groups were made using the *t*-test for two independent samples.

Results

With the assistance of CMDHB decision support staff, 144 patients diagnosed with RA who attended an initial rheumatology OPC appointment between 1 January 2008 and 31 December 2010 were identified.

After review of the electronic medical records 17/144 were excluded (seven did not fulfil RA classification criteria, seven had only one appointment, one had no electronic medical record, one lacked a first appointment letter in the electronic medical record, and one was seen for single appointment then discharged, with subsequent re-referral for RA treatment after 31 December 2010).

Of the cohort of 127 that met the inclusion/exclusion criteria, 32 were aged ≥60 years of age, while 95 were <60 years of age. The age distribution of the cohort is illustrated in Figure 1.

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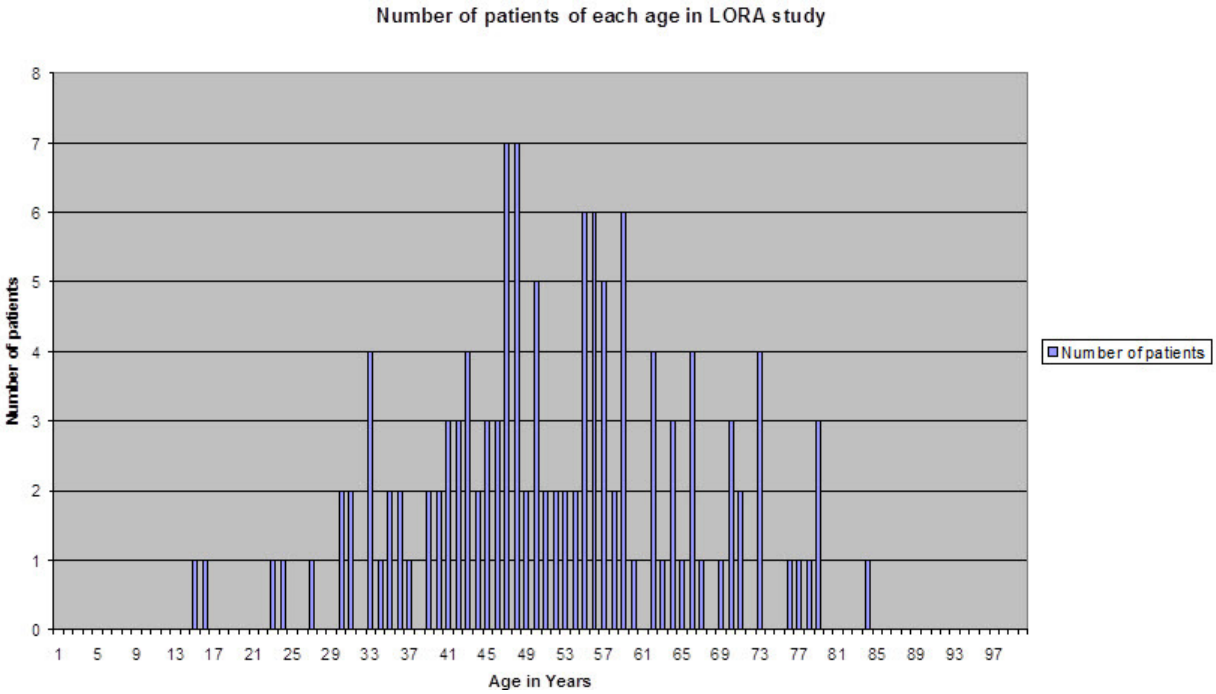
Figure 1: Number of patients of each Age in LORA study population



Patient demographics are recorded in Table 1 and reveal similar gender ratios and RF presence, although the LORA patients had a lower rate of presence of CCP ($p=0.045$), and higher number of comorbidities ($p<0.001$). Other first OPC parameters were similar between the LORA and YORA groups (including ESR, CRP, TJC28, SJC28 and presence of erosions). Mean duration of follow-up was not significantly different between the LORA and YORA groups as shown in Table 1.

A minority of the cohort (7/32 (21.88%) for the LORA group and 19/95 (20%) for the YORA group) did not remain in follow-up at 2 years, and their last recorded OPC was therefore used as their 2-year follow-up OPC.

Mean DAS28 ESR 3 variable and mean DAS28 CRP 3 variable were calculated for first and 2-year follow-up OPC and are similar in LORA and YORA patients as shown in Table 2. Mean DAS28 CRP 3 variable fell from 4.14 at first OPC to 2.6 by follow-up OPC in LORA patients, and fell from 3.93 at first OPC to 2.54 by follow-up OPC in YORA patients.

The medication rates refer to the use of these medications at any time between the first two clinic appointments (first OPC time period) and also between the first OPC visit and the 2-year follow-up clinic appointment (follow-up OPC time period). If a patient did not remain in follow-up for at least 2 years then the last recorded OPC was accepted as the 2-year follow-up clinic appointment for the study period. When comparing the LORA and YORA groups, no statistically significant differences in rates of methotrexate use at either first or follow-up OPC time periods were detected as shown in Table 3.

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Table 1: Baseline patient demographics

	Age ≥60 years		Age <60 years			
	Proportion	Percentage / Mean	Proportion	Percentage / Mean	P value	Test
Mean Age (yrs)		69.50		45.96	N/A	
Female	24/32	75.00%	71/95	74.74%	0.976	Pearson
RF positive	32/32	100.00%	88/94	93.62%	0.336	Fisher's
CCP Positive	24/31	77.42%	84/91	92.31%	0.045	Fisher's
ESR (mm/Hr) (1st OPC)		32.86		31.98	0.846	T Test Un
CRP (g/L) (1st OPC)		22.18		15.26	0.147	T Test Eq
TJC28 (1st OPC)		7.53		6.17	0.449	T Test Un
SJC28 (1st OPC)		7.35		6.45	0.550	T Test Eq
Erosions (1st OPC)	8/31	0.26	34/92	0.37	0.258	Pearson
Mean Number of Comorbidities		2.91		1.74	<0.001	T Test Eq
Duration of Follow-up (days)		848.13		764.36	0.091	T Test Eq

Legend:
Row Names: *RF* – rheumatoid factor, *CCP* – anti-cyclic citrullinated peptide, *ESR* – erythrocyte sedimentation ratio, *CRP* – C-reactive protein, *TJC28* – tender joint count out of 28, *SJC28* – swollen joint count out of 28, *1st OPC* – First Outpatient Clinic.
Statistical Tests: *Pearson* – Pearson's Chi-squared test, *Fisher's* – Fisher's exact test, *T Test Un* – T Test assuming unequal variance, *T Test Eq* – T Test assuming equal variance. All tests 2 sided.

Table 2: Comparison of DAS28 3 variable by LORA and YORA groups

	Age ≥60 years		Age <60 years			
	Proportion Missing Data	Mean	Proportion Missing Data	Mean	P value	Test Type
DAS28 CRP 3v (1st OPC)	2/32 (6.25%)	4.14	16/95 (16.84%)	3.93	0.536	T Test Eq
DAS28 ESR 3v (1st OPC)	6/32 (18.75%)	4.79	24/95 (25.26%)	4.33	0.177	T Test Eq
DAS28 CRP 3v (FU OPC)	14/32 (43.75%)	2.60	21/95 (22.11%)	2.54	0.864	T Test Eq
DAS28 ESR 3v (FU OPC)	17/32 (53.13%)	2.87	30/95 (31.58%)	2.93	0.873	T Test Eq

Legend:
Row Names: *DAS28 CRP 3v* – DAS28 CRP 3 variable, *DAS28 ESR 3v* – DAS28 ESR 3 variable, *1st OPC* – First Outpatient Clinic, *FUOPC* – 2-year Follow-up Outpatient Clinic (or last follow-up OPC for those patients that did not remain in follow-up at 2 years after their first OPC)
Statistical Tests: *T Test Eq* – T Test assuming equal variance. All tests 2 sided.
Proportion Missing Data: Proportion of patients with missing data preventing calculation of a particular composite disease activity score

Methotrexate use rates were 26/32 (81.25%) and 74/95 (77.89%), single DMARD rates were 30/32 (93.75%) and 85/95 (89.47%), and prednisone rates were 21/32 (65.63%) and 57/95 (60%) for the LORA and YORA groups respectively for the follow-up OPC time period. No statistically significant differences in rates of use of biological DMARDs, no DMARD, single DMARD, two or three DMARDs in combination or prednisone were detected between LORA and YORA groups.

LORA patients had a significantly lower NSAID use rate of 14/32 (43.75%) compared to YORA patients whose rate was 65/95 (68.42%) for the follow-up OPC time period (p=0.013). NSAID use rates were also significantly lower in LORA patients for the first OPC time period (p=0.046). There was no significant difference between LORA and YORA groups in disease severity or presence of erosions at 2-year follow-up OPC as shown in Table 3.

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Table 3: Comparison of DMARD use, follow-up disease severity and presence of erosions by LORA and YORA groups

	Age ≥60 years		Age <60 years			
	Proportion	Percentage	Proportion	Percentage	P value	Test Type
MTX (1st OPC TP)	22/32	68.75%	58/95	61.05%	0.435	Pearson
MTX (FUOPC TP)	26/32	81.25%	74/95	77.89%	0.688	Pearson
1 DMARD (FUOPC TP)	30/32	93.75%	85/95	89.47%	0.375	Fisher's
2 DMARD combined (FUOPC TP)	14/32	43.75%	44/95	46.32%	0.801	Pearson
3 DMARD combined (FUOPC TP)	6/32	18.75%	19/95	20.00%	0.878	Pearson
Biologics (FUOPC TP)	0/32	0.00%	2/95	2.11%	1.000	Fisher's
No DMARD (FUOPC TP)	1/32	3.13%	3/95	3.16%	1.000	Fisher's
Prednisone (1st OPC TP)	21/32	65.63%	51/95	53.68%	0.238	Pearson
Prednisone (FUOPC TP)	21/32	65.63%	57/95	60.00%	0.572	Pearson
NSAID (1st OPC TP)	12/32	37.50%	55/95	57.89%	0.046	Pearson
NSAID (FUOPC TP)	14/32	43.75%	65/95	68.42%	0.013	Pearson
DAS 28 ESR 3v ≤2.6 (FUOPC)	8/15	53.33%	30/65	46.15%	0.616	Pearson
DAS 28 CRP 3v ≤2.6 (FUOPC)	13/18	72.22%	51/74	68.92%	0.785	Pearson
Erosions (FUOPC)	12/32	37.50%	46/93	49.46%	0.242	Pearson

Legend:

Row Names: *MTX* – methotrexate, *1 DMARD* – single disease modifying anti-rheumatic drug during study period, *2 DMARD* – two disease modifying anti-rheumatic drugs in combination during study period, *3 DMARD* – three disease modifying anti-rheumatic drugs in combination during study period, *No DMARD* – no disease modifying anti-rheumatic drugs taken during study period, *NSAID* – Nonsteroidal anti-inflammatory drugs, *OPC* – Outpatient Clinic, *FUOPC* – 2-year follow-up Outpatient Clinic, *DAS28 ESR 3v ≤2.6* – DAS28 ESR 3 variable less than or equal to 2.6 (i.e. in remission), *DAS28 CRP 3v ≤2.6* – DAS28 CRP 3 variable less than or equal to 2.6. Note medication use rates were recorded for two time periods: *1st OPC TP* – first Outpatient Clinic time period was defined as the time period from first OPC up to and including the next OPC; and *FUOPC TP* – follow-up Outpatient Clinic time period was defined as the time period from the first OPC up to and including the 2-year follow-up OPC (or last follow-up OPC for those patients that did not remain in follow-up at 2 years after their first OPC). A medication was recorded as used during a time period if it was used at any point during the defined period.

Statistical Tests: *Pearson* – Pearson's Chi Squared test, *Fisher's* – Fisher's Exact Test. All tests 2 sided.

For the 2-year follow-up OPC time period the mean maximum tolerated dose of methotrexate was 16.3 mg for the LORA group and 16.86 mg for the YORA group. No significant difference between the groups was observed (p=0.660).

No statistically significant difference was seen in mean time from first OPC to methotrexate use or first DMARD use between groups; however the LORA patients had a significantly shorter mean time to prednisone commencement of 7.1 days compared with YORA patients whose mean time was 32.5 days (p=0.049) as shown in Table 4.

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Table 4: Comparison of Time (in days) to DMARD and prednisone use in LORA and YORA groups

	Age ≥60 years		Age <60 years			
	Mean	SD	Mean	SD	P value	Test Type
Time to MTX (days)	88.27	184.53	83.91	179.34	0.920	T Test Eq
Time to 1st DMARD (days)	35.16	53.09	63.61	147.45	0.118	T Test Un
Time to Prednisone (days)	7.14	21.32	32.54	89.14	0.049	T Test Un

Legend:
Row Names: *Time to MTX* – time from first outpatient clinic to commencement of methotrexate, *Time to 1st DMARD* – time from first outpatient clinic to commencement of first disease modifying anti-rheumatic drug. *Time to prednisone* – time from first outpatient clinic to commencement of prednisone
Statistical Tests: *T Test Eq* – T Test assuming equal variance. *T Test Un* – T Test assuming unequal variance. All tests 2 sided. SD – standard deviation.

Post-hoc analysis of the time to medication data arrayed into categories revealed that there was no statistically significant difference between the proportions of LORA and YORA patients who had a time of 0 days or a time >90 days for those with time to methotrexate, time to first DMARD and time to prednisone recorded. Regarding the LORA and YORA patients with time to first DMARD use recorded, 19/31 (61.29%) and 50/92 (54.35%) had a time to first DMARD of 0 days.

Post-hoc analysis revealed no statistically significant differences between males and females aged 15 to 45 years in ESR, CRP, TJC28, SJC28, or DAS28 ESR 3 variable, presence of RF, CCP, erosions or comorbidities at first OPC, or use rates of methotrexate, DMARDs or biologics. Methotrexate use rate for the first OPC time period in males aged 15 to 45 years was 3/7 (42.86%) compared with the rate in females of 16/31 (51.61%).

Discussion

Patients with new onset rheumatoid arthritis at our institution received similar disease modifying anti-rheumatic drug treatment irrespective of age. The absence of a statistically significant difference in disease modifying agent use between late and young onset rheumatoid arthritis patients is encouraging since they achieve similar patterns of benefit including reduction in disease activity and radiographic progression, and similar anti-tumour necrosis factor discontinuation rates when treated appropriately with disease modifying agents.[7,11–13]

Late and young onset rheumatoid arthritis patients have a similar hazard ratio for infection with anti-tumour necrosis factor use compared with synthetic anti-rheumatic disease modifying drug use, although a higher crude rate of infection occurs with increasing age in patients with rheumatoid arthritis.[14] Some previous studies have revealed differences in treatment between LORA and YORA patients.[8,9,15]

Tutuncu et al used data from the CORRONA database which included 2101 LORA patients from multiple centres in USA with a mean disease duration of 5.3 years to demonstrate statistically significant differences in treatment between LORA patients and matched YORA patients.[9] They noted LORA patients had a 25% rate of biological agent use compared with 33.1% in YORA patients, while 30.9% of LORA patients used >1 DMARD compared with 40.5% of YORA patients.[9]

Tutuncu et al observed that methotrexate use was higher in LORA patients (63.9% in LORA patients compared to 59.6% in YORA patients) although the LORA patients had a lower mean methotrexate dose (11.96 mg in LORA patients and 13.53 mg in YORA patients).[9] In contrast the current study revealed no significant difference in methotrexate use for the 2-year follow-up OPC time period with use rates of 81% and 78% in LORA and YORA patients respectively.

Huscher et al reported data for a large multicentre German cohort of 1551 LORA and YORA pairs matched for disease duration with a mean disease duration of 4.1 years.[15]

Huscher et al did not find statistically significant differences in methotrexate use in their cohort with 58.7% of YORA patients and 56.9% of LORA patients using methotrexate, in agreement with the current study.[15] However, in contrast with the current study, they demonstrated statistically significant differences in treatment between LORA and YORA patients, reporting synthetic or biologic DMARD use by 84.7% of YORA patients and 78.9% of LORA patients, and biologic DMARD use by 16% of YORA patients but only 5.7% of LORA patients.[15]

Arnold et al analysed data from 1889 patients from the Canadian Early Arthritis Cohort (CATCH), 442 aged <42 years, 899 aged from 42 to 63 years and 468 aged ≥64 years.[16] In contrast to the current study but in keeping with Tutuncu et al they demonstrated LORA group (aged ≥64 years) had a significantly different methotrexate rate of 75.8% compared to those aged 42 to 63 years and <42 years whose methotrexate rates were 77.4% and 64% respectively.[9, 16]

Radovits et al demonstrated in a Dutch cohort that RA patients >68 years of age were less likely to receive anti-TNF treatment within an equal period of time compared with younger patients, taking disease activity, disease duration and comorbidities into account.[8]

Huscher et al, Tutuncu et al and Arnold et al also reported a lower rate of biological DMARD/anti-TNF use in LORA patients.[9,15,16] Only two of the cohort of 127 in the current study utilised anti-TNFs during the study period prior to their 2-year follow-up OPC. The majority of patients commencing on anti-TNFs or biological DMARDs at our institution are doing so after a disease duration of greater than 2 years.

Possible reasons for the difference in LORA and YORA patient variance of synthetic and biological DMARD use between previous studies and the current study include the current study's smaller size and use of a recent onset cohort with a shorter patient disease duration.[9,15]

Previous studies revealed higher rates of glucocorticoid use in LORA patients compared with YORA patients. Tutuncu et al reported prednisone use was higher in LORA patients (41% in LORA patients compared with 37.64% in YORA patients),[9] and Huscher et al reported a higher rate of glucocorticoid use in LORA patients (60.5% in LORA patients compared with 55.6% YORA patients).[15] Arnold et al also observed a significantly higher rate of prednisone use amongst the LORA group compared with other age groups.[16]

The current study revealed no statistically significant difference in prednisone use rate between LORA and YORA patients although LORA patients had a significantly shorter mean time to prednisone use than YORA patients. It should be noted that the 2-year follow-up OPC time period prednisone use rate recorded prednisone use at any point during the study period (even if it was subsequently discontinued), and included patients who used short term prednisone for disease control at presentation or for a subsequent flare. The LORA patients had a significantly lower rate of NSAID use in the current study compared to YORA patients, which is appropriate clinical practice.[17]

In the current study the mean time to first DMARD use was 35 days and 64 days for the LORA and YORA patients respectively, and for methotrexate use was 88 days and 84 days. The majority of LORA and YORA patients commenced their first DMARD during the first OPC time period. Some patients chose to defer or decline DMARD commencement, despite recommendation by the rheumatologist or registrar at clinic. For many patients establishment of the diagnosis and discussion with the rheumatology nurse specialists occurred prior to commencing DMARD or Methotrexate use in this multicultural population.

The authors also conducted post-hoc analysis of recorded data to see if the low initial utilisation of methotrexate was influenced by gender difference in the childbearing age group. No significant reduction in initial or follow-up methotrexate use or DMARD use was found when comparing the 15 to 45 year age group by gender, although the number in the male 15 to 45 years of age subgroup was very small.

The limitations of this study include the relatively small size of the study and its retrospective nature. While some previous studies have also defined LORA as RA with an age of onset at or after 60 years of age,[9, 18-21] others have defined LORA as after 65 years of age.[4,6,15] Given the small size of this study's cohort, LORA was defined as newly diagnosed recent onset RA at or after the age of 60 years. Patients attending clinics with RA prior to 2008 were unable to be identified from the rheumatology database.

The retrospective nature of the study led to some data not being available for collection from the electronic medical record clinic letters and discharge summaries, including TJC and SJC which were interpreted from clinic letter descriptions of examinations. DAS28 (3 variable) scores were retrospectively calculated from ESR, CRP, TJC and SJC, though the number of patients where this data was not uniformly collected makes this information more difficult to interpret.

In summary the current study has shown late onset rheumatoid arthritis patients had a significantly lower rate of nonsteroidal anti-inflammatory medication use, which is appropriate, and no statistically significant differences in disease activity or rates of use of methotrexate and other disease modifying anti-rheumatic drugs compared with young onset rheumatoid arthritis patients.

Competing interests: Nil.

Author information: Laurence S G Teoh, Clinical Tutor Specialist, Rheumatology Department, Middlemore Hospital, Otahuhu, Auckland; Ravi Suppiah, Rheumatologist, Rheumatology Department, Middlemore Hospital &Rheumatology Department, Auckland City Hospital, Grafton, Auckland; Peter J Gow, Rheumatologist, Rheumatology Department, Middlemore Hospital, Otahuhu, Auckland

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Correspondence: Laurence S G Teoh, Rheumatology Department, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland 1640, New Zealand. laurence_teoh@xtra.co.nz

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ORIGINAL ARTICLE

Diagnostic category agreement and malignancy rates in clinician-categorised, non-standardised thyroid cytology reports

Mark J Bolland, Carl Eagleton, Brandon Orr-Walker

Abstract

Aim Standardised reporting of thyroid cytology is recommended but not universally practiced. We compared agreement between clinicians categorising non-standardised thyroid cytology reports, and determined malignancy rates in clinician-assigned cytology categories.

Methods We identified all thyroid cytology reports from 2008–9 and any reports prior to histology samples from 2008–9 at Middlemore Hospital. Two clinicians independently classified these cytology reports using the Bethesda System, and we assessed agreement between their classifications. We classified histology results following the cytology sample as benign or malignant according to the primary diagnosis in the histology report, and calculated malignancy rates for each cytology category.

Results Agreement between the classifications of 259 cytology results from 227 patients was moderate (κ=0.67, 95% confidence interval 0.61– 0.74), with good agreement (>80%) for only 3 of the 6 Bethesda categories. 122 patients had subsequent thyroid histology samples. 88% had benign primary diagnoses, with 15 (12%) primary thyroid cancers and an additional 11 (9%) incidental cancers. Malignancy rates for each Bethesda category varied from published rates for the majority of clinician-assigned categories.

Conclusions Low agreement between interpretation of non-standardised thyroid cytology reports suggests that standardised reporting should be universally adopted. Malignancy rates for thyroid cytology categories should be reported to inform local clinical practice.

Palpable thyroid nodules occur in about 5% of middle aged and older adults,[1,2] and impalpable nodules are even more common.[3,4] Once a nodule is recognised, concern is often raised about the risk of malignancy.

Thyroid ultrasonography can be used to stratify the risk of malignancy in a nodule, but specific ultrasound findings associated with a very low risk of malignancy risk are only found in a small proportion of thyroid nodules.[5] Therefore, cytological assessment of fine needle aspiration (FNA) samples from thyroid nodules remains the most useful test to rule out malignancy without surgical excision of the lesion.

Different systems for standardised reporting and classification of thyroid cytology have been proposed.[6] In 2008, a consensus conference recommended standardised reporting of thyroid nodules with classification into six groups, termed the Bethesda System (Table 1).[6] The risk of malignancy increases in the higher Bethesda categories (Table 1).

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Table 1: The Bethesda System for reporting thyroid cytology with risk of malignancy and recommended clinical management

Diagnostic category	Risk of malignancy	Suggested management
1. Nondiagnostic	1–4%	Repeat FNA with ultrasound guidance
2. Benign	0–3%	Clinical follow-up
3. Atypia of Undetermined Significance	5–15%	Repeat FNA
4. Follicular Neoplasm	15–30%	Lobectomy
5. Suspicious for Malignancy	60–75%	Total thyroidectomy or lobectomy
6. Malignant	97–99%	Total thyroidectomy

Adapted from Baloch and colleagues[6] and Cibas and Ali[7]

Reporting of cytology with the Bethesda System is becoming more widespread in New Zealand, although it is not universally used. When cytology results are not reported using standardised categories, many clinicians will nevertheless attempt to categorise the lesion based upon the report, and use the reported malignancy risks for that category to guide further management.

We investigated the reliability of this approach, by firstly comparing the agreement between two clinicians categorising non-standardised cytology reports into Bethesda categories. Secondly, we determined the rates of malignancy in clinician-assigned Bethesda categories from thyroid nodules with both cytology and histology samples, and compared these rates with those published for the Bethesda System.[6,7]

Methods

We identified all thyroid cytology and histology samples reported at Middlemore Hospital in 2008 and 2009. We extracted the text from all these cytology reports, and also any earlier cytology report taken before a histology sample.

Two clinicians (MB, CE) independently classified the cytology report text using the Bethesda System without knowledge of any patient, clinical or laboratory data related to the cytology report. Agreement between cytology classification was assessed using the kappa coefficient. Any histology samples taken after the cytology report were retrieved and classified as benign or malignant according to the primary diagnosis in the histology report.

Where a cancer was identified on histological examination, we classified it as a primary malignancy when it was present in the nodule that led to clinical presentation or was the subject of the preceding FNA. Otherwise, any cancer identified was considered incidental. Relevant demographic and clinical details were obtained from electronic medical records. This is an audit as defined by the New Zealand National Ethics Advisory Committee guidelines and therefore it did not require ethical approval.[8]

Results

Of 298 patients with samples coded as thyroid cytology or histology in 2008–9, we excluded 25 patients: 14 had thyroid tissue taken during parathyroid surgery, 5 had investigations of non-thyroidal neck masses, 2 had no clinical details because all management occurred in the private medical system, 2 had a known primary malignancy and investigation of incidental thyroid nodules as possible metastases, and for 2 patients with histological samples we were unable to obtain prior cytology results. Thus, of the remaining 273 patients, 227 had cytology samples, 168 had histology samples and 122 had both. Table 2 shows relevant demographic and clinical characteristics of the patients who underwent thyroid FNA, and those who underwent thyroid FNA followed by thyroid surgery.

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Table 2: Baseline characteristics of cohort

Variables	All patients	Patients with cytology and histology samples
	N=273	N=122
Gender		
Female	234 (86%)	104 (85%)
Male	39 (14%)	18 (15%)
Age (y)	48.4 (15.4)	46.5 (15.4)
Referral source		
General practitioner	217 (79%)	101 (83%)
Palpable nodule/goitre	221 (81%)	107 (88%)
Ultrasound performed	241 (88%)	111 (91%)
Proportion with		
Cytology sample	227 (83%)	122 (100%)
Histology sample	168 (62%)	122 (100%)
Cytology and histology sample	122 (54%)	122 (100%)
Fine needle aspirate technique		
Freehand	83 (37%)	55 (45%)
Ultrasound-guided	144 (63%)	67 (55%)

Data are n (%) or mean (SD).

259 FNA results were available from 227 patients. Table 3 shows the classification of these results into Bethesda categories by two clinicians. The agreement between clinicians was moderate (κ=0.67, 95% confidence interval 0.61–0.74).

Table 4 shows that the agreement was >80% for 3 categories (Bethesda 1, Bethesda 2, and Bethesda 6), but was only moderate for the Bethesda 5 category (67%) and was poor for both the Bethesda 3 (6%) and Bethesda 4 (26%) categories. However, when the Bethesda 3 and 4 categories were pooled, agreement between clinicians for this pooled category was high (89%, Table 4).

When this pooled category was used with the other 4 Bethesda categories to create 5 categories overall, there was a high level of agreement overall between clinicians (κ=0.91, 95% confidence interval 0.86–0.95).

Of the 227 patients with FNA results, 105 did not have thyroid surgery. For these 105 people, the proportions of final FNA in each Bethesda category were: 1 – 14% (clinician 1), 11% (clinician 2); 2 – 70%, 73%; 3 – 1%, 11%, 4 – 12%, 3%; 5 – 2%, 1%; 6 – 0%, 0%.

Of individuals without benign cytology (Bethesda categories 1, 3, 4, or 5) who did not proceed to surgery, the majority of patients either declined further intervention or were reassured by their clinician that this was not necessary.

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Table 3: Comparison of Bethesda classifications of all 259 cytology reports by 2 clinicians

		Clinician 1						
	Category	**Bethesda 1**	**Bethesda 2**	**Bethesda 3**	**Bethesda 4**	**Bethesda 5**	**Bethesda 6**	**Total**
Clinician 2	**Bethesda 1**	44	4	0	0	1	0	49
	Bethesda 2	4	123	0	2	0	0	129
	Bethesda 3	0	0	3	43	5	0	51
	Bethesda 4	0	0	0	16	0	0	16
	Bethesda 5	0	0	0	0	12	0	12
	Bethesda 6	0	0	0	0	0	2	2
	Total	**48**	**127**	**3**	**61**	**18**	**2**	**259**

Bethesda categories: 1 Inadequate; 2 Benign; 3 Atypia of uncertain significance; 4 Follicular neoplasm; 5 Suspicious for malignancy; 6 Malignant.

Table 4: Agreement between Bethesda classifications by 2 clinicians

Bethesda category	Samples classified by either clinician (n)	Samples classified by both clinicians (n)	Agreement (%)
1. Inadequate	53	44	83%
2. Benign	133	123	93%
3. Atypia of uncertain significance	51	3	6%
4. Follicular neoplasm	61	16	26%
3 or 4. Atypia of uncertain significance or follicular neoplasm	69	62	89%
5. Suspicious for malignancy	18	12	67%
6. Malignant	2	2	100%

Table 5 shows the comparison between the cytology results from the final FNA and the histological samples by clinician for the 122 patients with cytology and histology results. Overall, 88% of patients had benign primary diagnoses, with 15 primary thyroid cancers (12%), and an additional 11 incidental cancers (9%).

Table 1 shows the reported risk of malignancy for each Bethesda category.[6,7] Thus, malignancy rate was slightly higher than reported for the Bethesda 2 category (4% vs reported rate 0–3%). Clinician 1 used the Bethesda 3 category infrequently, and malignancy rate was lower than reported for the Clinician 1 Bethesda 4 category (9% vs reported rate 15–30%). Clinician 2 used the Bethesda 3 category more frequently, and malignancy rates were similar to reported rates for both Clinician 2 Bethesda 3 and 4 categories.

Malignancy rate was lower than reported for the Bethesda 5 category (30–36% vs reported rate 60–75%). Both FNA results classified as malignant were confirmed as cancer on histological samples.

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Table 5: Comparison between final thyroid cytology and histology results (n=122)

	Clinician 1						Clinician 2					
Bethesda cytology category	**1**	**2**	**3**	**4**	**5**	**6**	**1**	**2**	**3**	**4**	**5**	**6**
N	**11**	**47**	**1**	**47**	**14**	**2**	**14**	**47**	**37**	**12**	**10**	**2**
Benign primary pathology (%)	82	96	100	91	64	0	79	96	92	83	70	0
Malignant primary pathology (n)												
Papillary thyroid cancer	1	1	0	1	2	2	1	1	1	0	2	2
Follicular variant PTC	1	1	0	1	2	0	2	1	2	0	0	0
Follicular carcinoma	0	0	0	2	1	0	0	0	0	2	1	0
Percent of total (%)	18	4	0	9	36	100	21	4	8	17	30	100
Benign primary pathology with incidental cancer (n)												
Papillary thyroid cancer	0	4	0	4	1	0	0	4	3	1	1	0
Follicular variant PTC	0	1	0	1	0	0	0	1	0	1	0	0
Percent of total (%)	0	11	0	11	7	0	0	11	8	17	10	0

Bethesda categories: 1 Inadequate; 2 Benign; 3 Atypia of uncertain significance; 4 Follicular neoplasm; 5 Suspicious for malignancy; 6 Malignant. **Abbreviation:** PTC – papillary thyroid carcinoma.

Discussion

We found only moderate agreement between two clinicians classifying non-standardised cytology reports into Bethesda categories. This was largely due to very poor agreement between the use of the Bethesda 3 and 4 categories and only moderate agreement for the Bethesda 5 category. The lack of agreement between interpretation of non-standardised cytology reports by clinicians suggests that standardised cytology reporting should be universally adopted.

The proportions of primary malignancy differed from those reported for the Bethesda system. This has been reported previously[9–12] and reinforces the importance of regular auditing and publication of local results. Higher-than-expected malignancy rates following benign cytology results could lead clinicians to falsely reassure patients about their malignancy risk and need for follow-up.

Conversely, lower-than-expected malignancy rates following Bethesda 3, 4, and 5 category cytology results could lead to overdiagnosis where the risk of malignancy is inflated, and therefore the need for surgical procedures is exaggerated.

In our results, there were 2 primary malignancies in 47 patients with benign cytology (Bethesda 2). This proportion (4%) is slightly higher than the expected rate (0–3%), but because of the small number of patients and cancers, there remains considerable uncertainty in the result. It is also difficult to know whether malignancy rates in individuals proceeding to surgery can be applied to those who do not.

The low level of agreement between clinicians for the Bethesda 3 and 4 categories is likely due to a number of factors. In the original report of the Bethesda System, the Bethesda 3 category was described as optional, and its use was discouraged.[6] Others suggest it accounts for 3–6% of cytology reports, that higher rates suggest overuse of the category, and that it should only be used as a last resort.[7] Furthermore the Bethesda 3 category is labelled Atypia of Undetermined Significance or Follicular Lesion of Undetermined Significance, but “Follicular lesion” is a term commonly used previously for Bethesda 4 category lesions.[13]

In our results, there was high agreement (89%) between clinicians that lesions should be categorised as either Bethesda 3 or 4, but one clinician used the Bethesda 3 category very sparingly (3 of 64 lesions categorised as either Bethesda 3 or 4), whereas the other clinician used the Bethesda 3 category commonly (51 of 67 of lesions categorised as either Bethesda 3 or Bethesda 4).

The original purpose of the Bethesda 3 category was to identify individuals whose malignancy risk was greater than for a benign lesion but not high enough to warrant a surgical procedure. Thus, management recommendations for this category include repeat FNA, or further radiological imaging.[7,13] Other authors have suggested that the Bethesda 3 and 4 categories could be combined,[12] which would be more consistent with clinical practice at our hospital, in which the majority (~80%) of patients with either Bethesda 3 or 4 cytology undergo a surgical procedure.

There was only moderate agreement between clinicians for the Bethesda 5 category, but all patients in this category were recommended to undergo a surgical procedure. From a clinical management perspective, the major difference between a Bethesda 5 and 6 category result is the operative procedure that might be undertaken. For individuals with definite malignancy, a total thyroidectomy is recommended, whereas for a suspicious lesion, consideration could be given to a lobectomy as the initial procedure.

Thus, some authors have suggested combining the two categories.[12] However, in our results, the rate of primary malignancy in the Bethesda 5 category was 30–36%, which is lower than published results for this category, and closer to rates for the Bethesda 4 category,[6,7,9–12] for which lobectomy is the recommended management.[6,7,13]

The decision about which surgical procedure to undertake is likely to be influenced not only by malignancy rates, but also the size of the nodule (and hence the size and stage of the potential tumour), the presence and size of nodules in the contralateral lobe and the patient preference. In some situations, the advantages of a lobectomy in preserving thyroid function may outweigh the disadvantages of performing a two-stage procedure if malignancy is confirmed, whereas in other cases an initial total thyroidectomy may be preferred.

A further complicating issue is that there is often substantial disagreement amongst cytologists, with consensus review of cytology often leading to changes in the categorisation of the lesion.[14] Thus, clinicians need to factor in the inherent limitations of cytology when formulating treatment plans, especially when considering surgical procedures.

Given the local variations in thyroid cytology reporting, malignancy rates and clinical practice, we think the best approach to optimise care of patients with thyroid nodules is that all thyroid cytology is reported using a standardised reporting system, that regular audits of the frequency of cytology categories used and the malignancy rates for those categories are undertaken and published locally, and for clinicians and cytologists to meet regularly to discuss these results to inform local clinical practice.

Competing interests: Nil.

Author information: Mark J Bolland, Associate Professor of Medicine[1,2]; Carl Eagleton, Endocrinologist[1]; Brandon Orr-Walker, Endocrinologist[1]

[1]Department of Endocrinology, Middlemore Hospital, Otahuhu, Auckland

[2]Department of Medicine, University of Auckland

Correspondence: Mark Bolland, Department of Endocrinology, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland 1640, New Zealand. m.bolland@auckland.ac.nz

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ORIGINAL ARTICLE

Herpes zoster (shingles) at a large New Zealand general practice: incidence over 5 years

J Stewart Reid, Brendon Ah Wong

Abstract

Aim The objective of this study, in a large group practice in Lower Hutt with a stable population of around 19,000 patients, was to describe the retrospective incidence of shingles over a 5-year period so that it could be compared to international data.

Method The practice database was interrogated for patients whose disease code indicated they suffered shingles, herpes zoster, or post herpetic neuralgia between January 1 2009 and December 31 2013. The charts of the identified patients were reviewed to assess whether the diagnosis was confirmed, to ascertain that onset occurred within the specified time period, to describe the site of the rash and the age, sex and ethnicity of the patients. Rates of disease were calculated using a denominator derived from a comparison of patients registered during the 5 years studied.

Results The results indicate that incidence of shingles rose with age, females were more frequently affected than males and that the thorax was the commonest site.

Conclusion The incidence at this New Zealand medical centre was similar to that reported internationally.

A vaccine against herpes zoster (shingles) has recently become available in New Zealand.[1,2] "How likely am I to get shingles?" is an important frequently asked question by potential vaccine recipients. Few data exist concerning the incidence of shingles in New Zealand. In the 2014 New Zealand Immunisation Handbook,[3] only hospitalisation data from New Zealand are presented and it is assumed that the incidence of shingles in New Zealand is similar to that reported in other countries. It is widely reported that the incidence of shingles rises with age, from around 1–2/1000 per annum under age 45 to 12–15/1000 per annum over age 80.[1,4–7]

The lifetime risk of shingles is reported as being 1 in 3 and there is a 50% chance of suffering shingles for those who live to age 85.[5] Women are affected more often than men.[8]

The objective of this study, in a large group practice in Lower Hutt with a stable population of around 19,000 patients, was to describe the retrospective incidence of shingles over a 5-year period so that it could be compared to international data.

Methods

The practice is fully computerised using Medtech 32 (version 20.11) software. The database was retrospectively interrogated for those whose disease coding indicated they had suffered shingles, herpes zoster or post herpetic neuralgia between 1 January 2009 and 31 December 2013. In addition those who were prescribed acyclovir 800 mg 5 times daily but who did not have any of the above disease codes were identified.

A chart review of all those identified, by either method, was conducted to establish whether the diagnosis was confirmed. The confirmation of diagnosis following chart review was one of interpretation given that this was a retrospective study and the diagnoses made by up to 15 doctors of varying experience were being reviewed.

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A case of shingles was confirmed if its onset was within the specified time period and either:

- A swab from the rash was PCR positive for varicella zoster virus
- The rash was described as "classical shingles" or similar description
- The rash was described as unilateral, dermatomal and vesicular
- A subsequent consultation suggested that the diagnosis was confirmed – further description of rash, ongoing allodynia or pain

A case was excluded if:

- A swab from the rash was PCR positive for herpes simplex virus (HSV)
- The diagnosis was "recurrent shingles", which was assumed to be HSV
- Diagnostic uncertainty was expressed and not clarified by further consultations.

For each confirmed case, age, sex, ethnicity, site of rash, referral and antiviral prescription were recorded. It was initially thought that it would be possible to ascertain the duration of symptoms but the data recorded were too variable to make any assessment of this feasible.

The denominator was chosen following comparison of the number of patients registered each year from 2009 until 2013 and the age breakdown of registered patients for the years for which such data were available.

Rates of disease per thousand patients per annum were calculated by multiplying the number of confirmed cases in each population by 1000 and dividing that figure by 5 times the denominator population.

Results

A total of 339 cases had a diagnosis of shingles, herpes zoster or post-herpetic neuralgia (diagnosis group) recorded from 1 January 2009 until 31 December 2013. An additional 44 cases were prescribed acyclovir 800mg five times daily and did not have a diagnosis recorded (prescription group). Following the chart review 287 cases confirmed cases remained of which 273 were from the diagnosis group and 14 were from the prescription group.

203 cases (70.7%) occurred in those aged 51 and older. 149 cases (51.9%) were thoracic, 44 lumbar (15.3%), 76 cervical (26.5%) and 18 ophthalmic (6.3%). Only ophthalmic cases were the subject of referral and, with one exception, an individual leaving for overseas the following day, all were referred.

Almost all of the 70.7% of cases in those aged 51 and older were of New Zealand European or other European ethnicity. Six cases occurred in those of Maori, two of Pacific Island and seven of Asian ethnicity.

Thus 92.6% of cases in those aged 51 and greater were of European ethnicity. The ethnic distribution of the practice differs from New Zealand as a whole and is 6% Maori, 3% Pacific, 10% Asian and 81% European. The 2013 New Zealand Census indicated that 14.9% of the population were Maori, 7.4 % Pacific and 11.8% Asian[9].

Acyclovir 800mg five times daily, usually for seven days (range five to ten), was the only antiviral prescribed. 169 (83%) of those aged greater than 50 years were prescribed acyclovir. Forty-nine (58%) of those aged 50 years or less were prescribed acyclovir. For both age groups the usual reason for not prescribing was late presentation though, in the younger age group, less severe disease with minimal pain was also a reason for not prescribing.

The denominator was taken as the March 2014 registered population; see table 1. Data were available on the total number of patients registered for each year from 2009 until 2013, but the age breakdown of patients registered was only available from 2011. The highest number of registered patients occurred in 2011 but the excess over March 2014 was because of a much larger number of patients

aged 50 years and under. Therefore we considered that the denominator chosen was conservative and would not inflate the rate of shingles in the older age groups.

Table 1: Registered population 2009–2014

Year	2009	2010	2011	2012	2013	March 2014
Registered patients	19,386	19,371	19,413	18,722	18,893	19,328

Rates of disease are described in Table 2. The overall rate of disease was 2.97 per thousand patients per annum. The rate of disease rises with age and for those age greater than 80 years is 13.91/1000/annum. For those aged greater than 50, the rate of disease in females was 6.38/1000/annum compared to the rate in males which was 5.75/1000/annum. Thus women over 50 years had an approximately 10% higher rate than men of the same age.

According to these data during the ten year period from age 51 to 60 years approximately 3.5% of individuals will suffer shingles: from age 61-70 years 6.3% will suffer shingles and from 71 to 80 years 8.3% will be affected. The cumulative risk of suffering shingles for the 30 years period from age 51 to 80 is therefore approximately 18%.

Table 2: Rates of disease by age group

Group (gender & years of age)	Number identified	Number confirmed	Denominator	Rate (1000/annum)
All ≤50	103	84	12,647	1.33
All ≥51	245	203	6,681	6.1
All >60	187	154	3,826	8.1
F 51–60	33	26	1,534	3.39
F 61–70	49	39	1,079	7.23
F 71–80	32	27	649	8.32
F > 80	27	23	345	12.96
M 51–60	25	23	1,310	3.51
M 61–70	34	26	990	5.25
M 71–80	25	22	533	8.25
M >80	20	17	230	14.78

Discussion

Hope-Simpson[10] in his classic epidemiology study over 25 years in his practice of approximately 3800 patients found that the incidence rises with age and is around 11/1000 per annum by age 80, similar to the rate seen in this paper. These data are replicated in other studies with, in general, an incidence of around 12–15/1000/annum for those aged 80 years and older.

In Australia, for those aged >60 years, the rate of shingles is now 15/1000 per annum.[7] Yawn et al[11] indicated that the population rate in the US is 3.6/1000 person years and in our study the rate was comparable at 2.97/1000/annum. Our data indicate that the incidence rose with age, was higher in females than males and our rates corresponded to those reported in the international literature.

Dworkin[12] reported that the site of disease was 50–70% on the trunk, 10 - 20% cervical and 10 - 20% ophthalmic. Our data, though similar at least for the trunk, had a rather higher rate for cervical disease and a lower rate for ophthalmic. We have no explanation for this variation other than the relatively small numerator in our study.

There are limitations to these data. Retrospective case reviews have inherent inaccuracy in that data are not recorded systematically. The quality of note taking varied and the clinical experience of the doctors in the practice ranged from 30 plus years of general practice to general practice registrars in their first year of practice.

In the course of the chart review we attempted to be quite strict about which cases were confirmed and we have confidence that those counted are highly likely to be cases of shingles. It is possible, however, that we have excluded too many cases given the strict approach we took, and there may have been some cases not disease coded who were not prescribed acyclovir 800 mg five times daily.

The population denominator is also subject to some imprecision but we consider that the denominator chosen did not result in any inflation in the observed rates.

This is the first report of shingles incidence in New Zealand. Given the predominately European ethnicity of our practice we consider that these data are likely to reflect the incidence of shingles in the European New Zealand population as a whole. The data are too few to make any comment about other ethnic groups.

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Author information: J Stewart Reid, Partner; Brendon Ah Wong, FMTP Registrar. Ropata Medical Centre, Lower Hutt

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Correspondence: Dr J Stewart Reid, Ropata Medical Centre, 577 High St, Lower Hutt 5010, New Zealand. stewart_christine@mac.com

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ORIGINAL ARTICLE

Impact of improved treatment on disease burden of chronic hepatitis C in New Zealand

Edward Gane, Catherine Stedman, Cheryl Brunton, Sarah Radke, Charles Henderson, Chris Estes, Homie Razavi

Abstract

Background Chronic hepatitis C is an important cause of liver failure, liver cancer and liver-related deaths in New Zealand. Although these complications can be prevented by HCV eradication, current treatment uptake is <1% per annum. We describe the burden of HCV infection and estimate the effect of four different treatment strategies to reduce HCV-related morbidity and mortality.

Methods Baseline model parameters were based upon literature review and expert consensus, focusing on New Zealand data. Four scenarios were modelled: Scenario 1 estimated the impact of increased treatment efficacy, while Scenario 2 estimated the effect of increased treatment efficacy and gradual increases in numbers treated. Scenarios 3 and 4 estimated the impact of deferred introduction of new DAAs for either 1 or 2 years.

Results Prevalence of HCV infection peaked in 2010 (50,480 cases). Peak prevalence of cirrhosis and HCC will occur after 2030. Scenario 2 resulted in sizeable decreases in HCV-related morbidity and mortality. The impact of Scenario 1 was smaller. Deferring funding for new DAA treatments for a further 1 or 2 years resulted in an 18-36% increase in liver-related deaths in 2030.

Conclusions While prevalence of chronic HCV infection may have peaked, disease burden continues to grow. Increased treatment uptake and efficacy combined with efforts to reduce disease transmission, will help prevent advanced liver disease and deaths.

About 2% of the world's population (almost 80 million people) has been infected with the hepatitis C virus (HCV).[1] Almost 30 years after the first notified case of post-transfusion non-A, non-B hepatitis, the exact prevalence of chronic HCV infection in New Zealand remains unknown because most cases remain undiagnosed and only acute infection is notifiable. However, the epidemiology of HCV is assumed to be similar to that in Australia, which has obtained accurate HCV prevalence data through high diagnosis rates and compulsory notification of prevalent, as well as incident cases (270,000 identified cases, prevalence 1.28%). The same rate in New Zealand would equate to approximately 54,000 people living with past or present HCV infection. From laboratory data, it is estimated less than one half of HCV-infected New Zealanders have been diagnosed and less than 10 per cent have accessed treatment of whom only half have been cured (Gane E, personal communication, 2014).

In many developed countries, the incidence of HCV infection was high during the 1960s, 70s and 80s, secondary to the rise in injecting drug use (IDU) over those decades. Since 2000, the incidence of HCV infection in Australia has dropped by more than 50% reflecting both a reduction in IDU and safer injecting practices.[2,3] Epidemiologic studies have identified that most chronic infections are now in the 40-to-60-year age group.

In the USA, the total size of the HCV-infected population has been stable since 2000[4] and this may also be the case in New Zealand. However, the very low rates of treatment uptake and an aging cohort effect will result in a steady increase in the proportion with established cirrhosis and numbers with the complications of HCV-related liver failure and hepatocellular carcinoma.[5–7] This will be associated with an increase in both liver-related mortality and demand for liver transplantation. A recent pharmacoeconomic analysis has costed the future

health burden of the current HCV epidemic in New Zealand at more than 0.5 billion dollars over the next 20 years.[8]

Eradication of HCV infection will halt liver disease progression and prevent liver-related complications. However, the poor efficacy and tolerability of current interferon-based (IFN) therapies have limited treatment uptake. Real world studies report that more than half of all patients with chronic HCV infection are either IFN-ineligible or intolerant.[9] Reduction of the projected future health burden associated with chronic hepatitis C will require widespread access to new regimens with better efficacy and less side effects.

Direct acting antiviral agents (DAAs) provide new opportunities for treatment of HCV whilst reducing the need for both interferon and ribavirin. Two combination DAA regimens are approaching approval for treatment of patients infected with HCV genotype 1 (G1): ledipasvir and sofosbuvir, and ABT-450, ombitasvir, and dasabuvir, with ribavirin.[10–13]

These new regimens have certainly raised the bar for future DAA development to at least 95% sustained virologic response (SVR) with only 12 weeks of treatment. Current Phase II studies combining three or more next generation DAAs are looking at reducing duration to as short as only 4 weeks (see clinicaltrials.gov NCT02133131 and NCT02175966), and also providing a one-size-fits-all regimen for all HCV genotypes.[14] It is expected that within the next 5 years, highly effective and well tolerated all-oral DAA regimens will be available and affordable for most patients living with HCV infection

This current study estimates the impact of increased treatment uptake and efficacy on the projected health burden from liver-related complications in New Zealanders with chronic HCV infection and also estimates the impact of delaying any access to new therapies for 1 or 2 years.

Methods

As previously described,[7,15,16] country-specific inputs were used to construct a disease progression model in Microsoft Excel software (Redmond, WA, USA) to quantify the HCV-infected population from 2013-2030. Uncertainty and sensitivity analyses were completed using Crystal Ball, an Excel add-in by Oracle. Beta-PERT distributions were used to model uncertainty associated with all inputs. Sensitivity analysis was used to identify the uncertainties that had the largest impact on peak prevalence in 2030. Monte-Carlo simulation was used to determine the 95% uncertainty intervals for prevalence.

The following model parameters were used:

Total population

Population data were organised by sex, 5-year age groups, and year (1950–2030) and obtained from the United Nations population database.[17]

Total infected

In New Zealand, the viraemic prevalent HCV infected population was estimated at 50,000 individuals in 2013 (Table 1).[18] A viraemic rate of 74.6% was assumed (Table 1).[19] The age and sex distribution of the infected population was based on demographic data collected through March 2014 from over 1000 clients with HCV attending a HCV clinic (Figure 1) (Personal communication, Brunton C, 2014). Christchurch Hepatitis C Community Clinic data, 2014. Razavi H, editor. 2014. 5-6-2014). The genotype distribution of the prevalent population was based upon distribution in more than 2000 individual results from a reference laboratory G1=56%, G2=8%, G3=35%, G4=0.5%, G6=1%.

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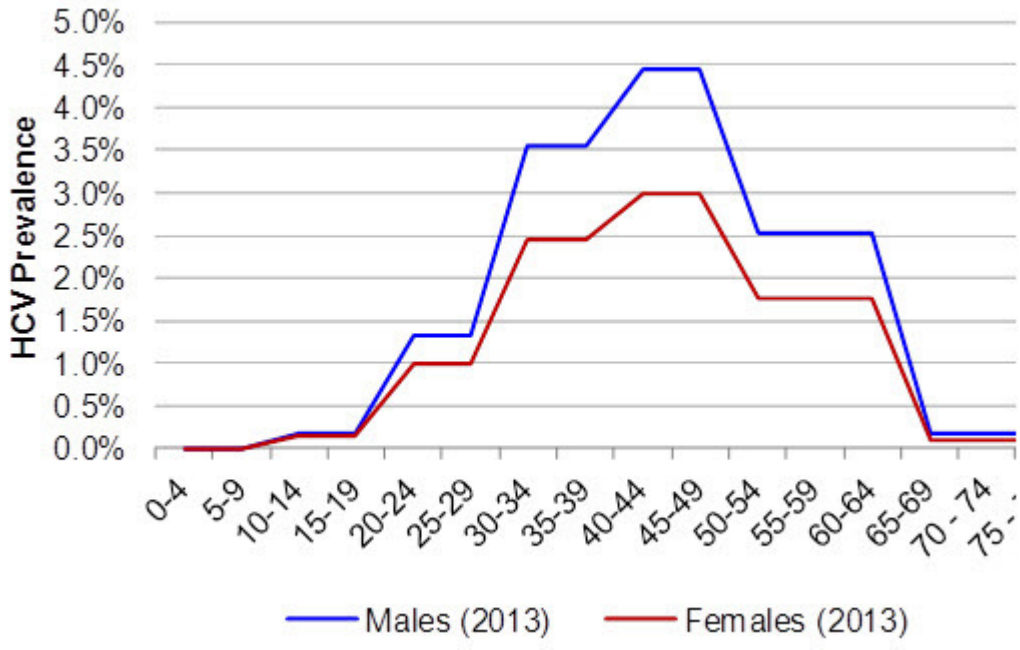
Table 1. Model inputs and 2013 estimates

	Historical	Year	2013 (Est.)
HCV Infected Cases	66,980 (36,240 - 96,630)	2013	66,980
Anti-HCV Prevalence	1.5% (0.8% - 2.1%)		1.5%
Total Viremic Cases	50,000 (27,050 - 72,130)	2013	50,000
Viremic Prevalence	1.1% (0.6% - 1.6%)		1.1%
Viremic Rate	74.6%		
HCV Diagnosed (Viremic)	20,000	2013	20,000
Viremic Diagnosis Rate	40%		40.0%
Annual Newly Diagnosed	910	2013	910
New Infections			1,020
New Infection Rate (per 100K)			22.6
Treated			
Number Treated	900	2011	900
Annual Treatment Rate	1.8%		1.8%
Risk Factors			
Number of Active IDU with HCV			10,100
Percent Active IDU			20.2%
Previous Blood Transfusion			1,790
Percent Previous Blood Transfusio			3.6%

Figure 1. Age and gender distribution of anti-HCV prevalence, New Zealand, 2013



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Transition probabilities

Age and sex specific transition probabilities were used to progress patients annually through each disease state, as described in earlier work.[7]

New cases

Historical changes in incidence were estimated based on expert consensus, previous New Zealand HCV modelling[20] and notification data.

It was estimated that the incidence of HCV infection peaked in 1980, and has since decreased. In 2014, it is estimated that 1,020 new infections occurred in New Zealand.

Treated patients

In 2013, it is estimated that 900 patients were treated in New Zealand, based on expert consensus and IMS Health (NZ) Ltd data for standard units of Peg-IFN sold in New Zealand, with a multiplier to account for under-reporting. The New Zealand genotype distribution was used to estimate the average number of weeks of treatment per patient with 85% compliance/persistence.

Liver transplants

In 2013, there were 36 liver transplants performed in New Zealand of which 24 were in adults. Thirteen transplants were attributable to HCV (54% of all adult transplants). The total number of annual liver transplants was available from transplant registry reports for the years 1997 to 2013.[21] The proportion of all liver transplants attributable to HCV was reported as 37% of all transplants.

Hepatocellular carcinoma

The best indicator of the increasing health burden related to chronic HCV infection is the incidence of HCV-related hepatocellular carcinoma (HCC). The best estimate for HCC incidence in New Zealand is the number referred to the National Hepatoma Service at Auckland City Hospital. This number has increased by more than 10% each year –from 55 cases in 2003, to 110 cases in 2008 and 205 cases in 2013. This observed increase matches the base case model outputs (Figure 2).

The major contributor to this rising HCC incidence in New Zealand is now chronic HCV infection. In 2003, there were nine new cases of HCV-related HCC, increasing to 21 in 2008 and 75 in 2013. Of these, the proportion in whom the diagnosis of HCV was known prior to presentation with HCC has increased steadily from only 33% in 2003 to 38% in 2008 and 53% in 2013. These figures would suggest that overall, less than 50% of the HCV-infected population in New Zealand is aware of their HCV status.

Diagnosed patients

Based on expert consensus, it was assumed that 40% of the HCV-viraemic population in New Zealand in 2013 was previously diagnosed. Based on the ratio of newly to previously diagnosed HCV infection in Australia, it was estimated that 910 cases were newly diagnosed in New Zealand in 2013.[19,22]

Mortality & risk factors

Background mortality rate by year, age group and sex was calculated using the Berkeley Human Mortality database.[23] Based on expert consensus, it was estimated that approximately 1% of the New Zealand population were active IDU and 30% were infected with HCV. Based on these data, it was estimated that 20.2% of the total infected population are active IDU while notification data show that 82.8% of those with incident HCV infection report a history of IDU.[24] Increased mortality among active IDU was estimated using a standard mortality ratio (SMR) of 10.0 for individuals between the 15 and 44 years of age.[25-30] A national study reported that 3.8% of the viraemic population was infected through transfusion.[20] An SMR of 1.5 was applied for all age groups in this population.[31]

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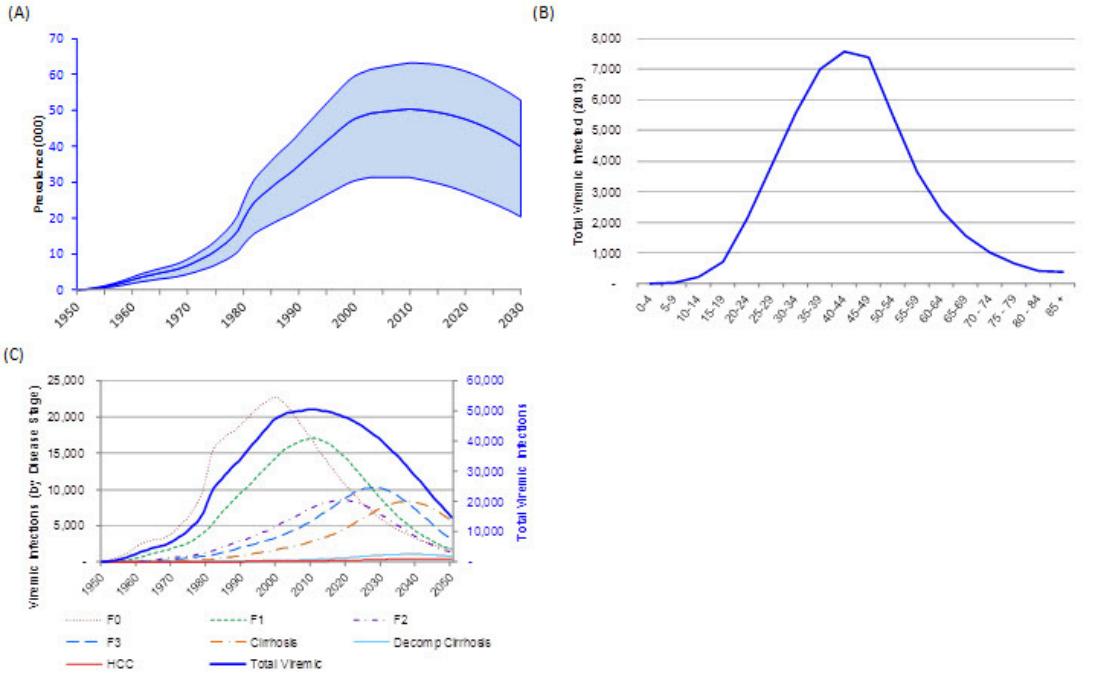
Figure 2. Base case model outputs



(A) Total viraemic cases, by year, 2013-2030;shaded areas represent interquartile range (B) Total viraemic cases, by age, 2013; (C) Number of viraemic cases, in total, and by disease stage, 1950-2030

Four model scenarios were explored

For the base case, it was assumed that all patients aged 15–59 years are considered for treatment with no restrictions based on fibrosis stage, and that 60% of potential patients in New Zealand were eligible and willing to complete treatment (Figure 3). It was also assumed that average SVR rates were 60% (G1), 80% (G2), 65% (G3) and 50% (G4). A treated population of 900 patients annually was modelled. It was further assumed that patients with decompensated cirrhosis, HCC, or transplant eligibility are not eligible for treatment until 2016.

- **Scenario 1: Increased SVR only**

  SVR and treatment eligibility rates gradually increased to 90% (all genotypes) by 2016. 2013 values for annual treated and newly diagnosed populations were held constant. Treatment restriction based on fibrosis stage (≥F3) was implemented during 2015-2017 in order to focus treatment on individuals with the most advanced liver disease. Beginning in 2018, fibrosis restrictions were removed (Figure 3).

- **Scenario 2: Increased SVR and increased annual treated population (Elimination Strategy)**

  This scenario included the same SVR and fibrosis restriction changes as Scenario 1. In addition, the annual number of treated patients was gradually increased to 4,040 by 2020. Beginning in 2016, treatment was expanded to include individuals up to 74 years old (Figure 3).

- **Scenario 3: Access to DAA therapy deferred for 1 year (Elimination [1-year delay] Strategy)**

  Scenario 2 was altered to estimate the impact of delaying access to new IFN-free DAA therapies to all patients, irrespective of stage of liver disease, for 1 year.

- **Scenario 4: Access to DAA therapy deferred for 2 years (Elimination [2-year delay] Strategy)**

  Scenario 2 was altered to estimate the impact of delaying access to new IFN-free DAA therapies to all patients, irrespective of stage of liver disease, for 1 year.

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Figure 3. Model inputs for Base Case, Scenario 1 and Scenario 2

Fig 3, continued

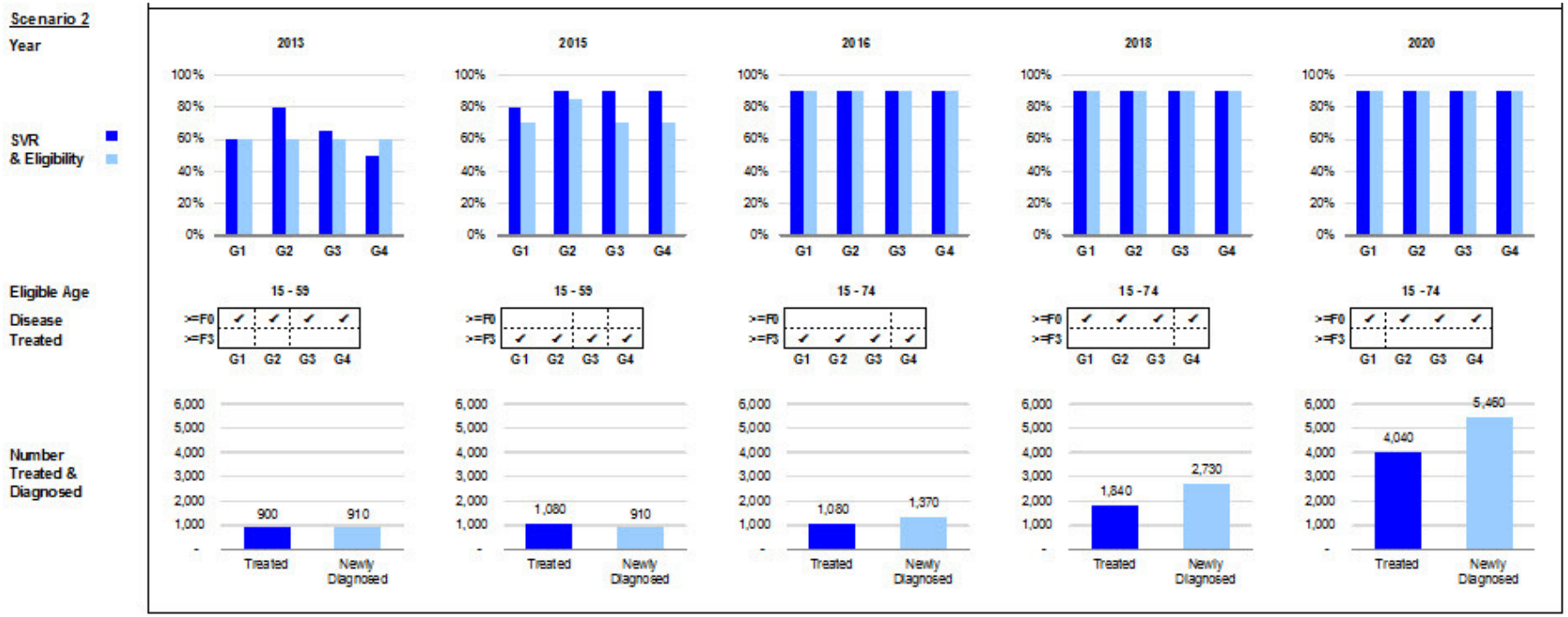

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Figure 4. Morbidity and mortality by scenario—New Zealand, 2013–2030

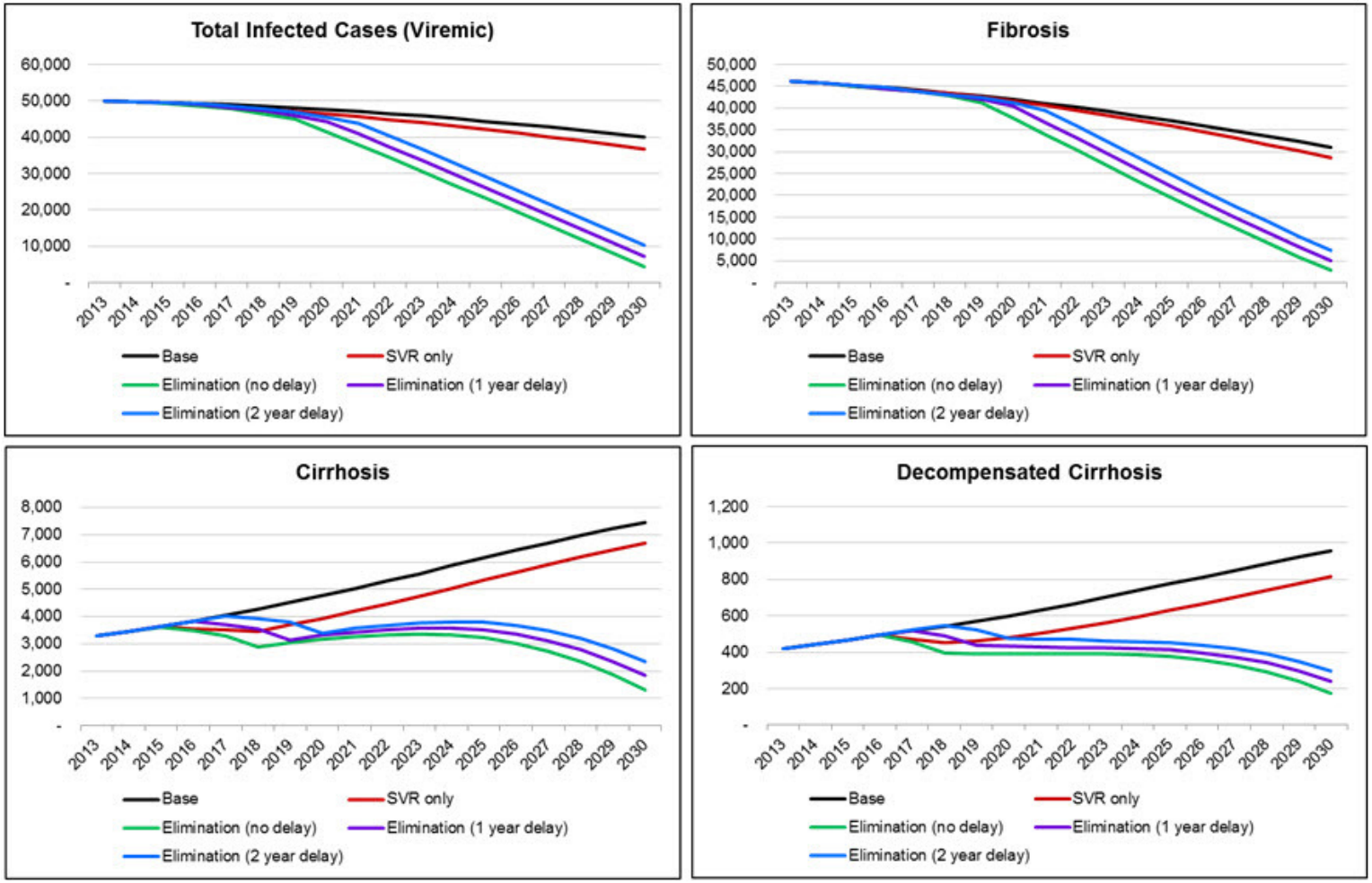

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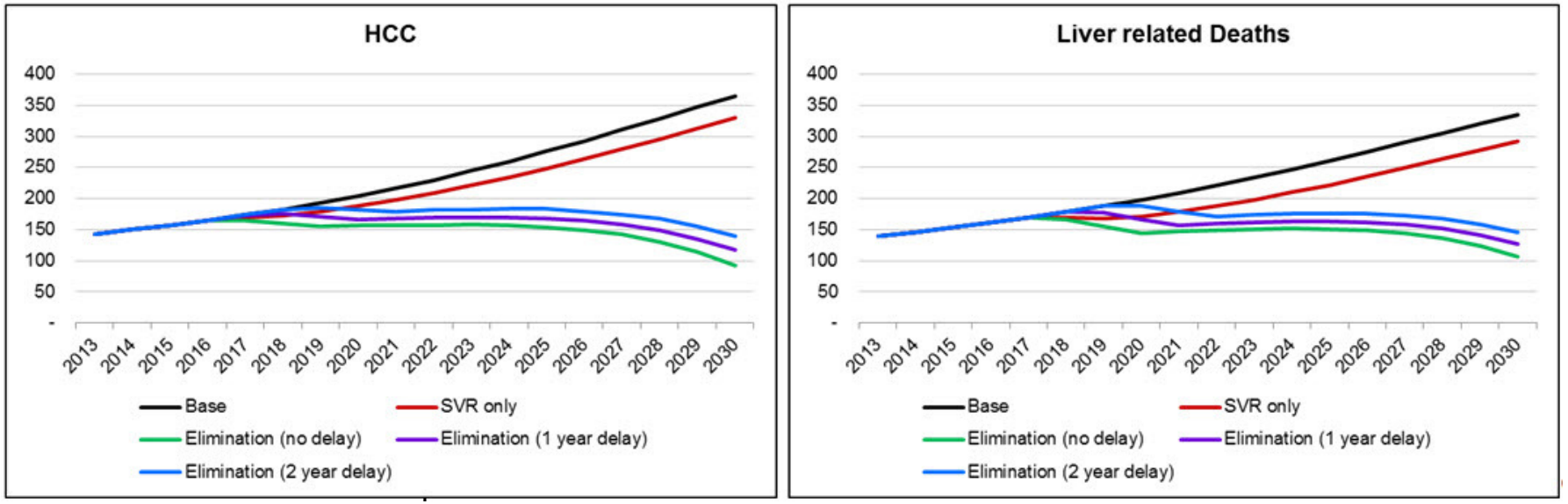
Fig 4, continued



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Results

Base case

The base case estimated 50,000 (30,440–63,130) infected individuals in 2013 (Figure 2A); their median age was 42 years (Figure 2B). Prevalence peaked at 50,480 patients in 2010, and declined to 39,950 by 2030. This model predicted an estimated 340 HCV-related deaths in 2030 compared to 140 deaths in 2013 (Figure 4). In 2013, 8% of viraemic cases are estimated to have compensated cirrhosis or more advanced liver disease (decompensated cirrhosis, HCC, or transplant), while this proportion will increase to 21% in 2030.

Scenario 1: Increased SVR only (SVR Strategy)

In this scenario, cumulative HCV-related mortality during the years 2013-2030 decreased by 11% (440 deaths averted) compared to the base case (Figure 4). HCC cases in 2030 decreased by 10% (35 cases), and the number of patients progressing to decompensation decreased by 15% (144 cases). The total infected population declined by 3,262 (8%) by 2030, compared to the base case

Scenario 2: Increased SVR and increased annual treated population (Elimination Strategy)

In this scenario, cumulative HCV-related mortality during 2013-2030 decreased by 35% (1,387 deaths averted) compared with the base case (Figure 4). The number of HCC cases in 2030 decreased by 75% (272 cases) and the number of patients progressing to decompensation decreased by 82% (784 cases). The total infected population decreased by 89% (35,526 cases) by 2030 compared to the base case.

Scenario 3: Access to DAA therapy deferred for 1 year: Elimination (1-year delay) strategy

In this scenario, cumulative HCV-related mortality was increased by 7% (195 extra deaths, the total number of HCC cases in 2030 increased by 26% (24 additional cases) and the total number of patients progressing to decompensation increased by 37% (65 additional cases) compared to the number predicted if the Elimination strategy was introduced in 2014 (Figure 4).

With this 1-year delay scenario, the size of infected population was 7287 in 2030, an increase of 2889 (62% increase) compared to the number predicted if the Elimination strategy was introduced in 2014.

Scenario 4: Access to DAA therapy deferred for 2 years: Elimination (2-year delay) strategy

In this scenario, cumulative HCV-related mortality was increased by 14% (377 extra deaths), the total number of HCC cases in 2030 increased by 50% (47 additional cases), and the total number of patients progressing to decompensation increased by 72% (124 additional cases), compared with outcomes if the Elimination strategy had been introduced in 2014 (Figure 4).

In this 2-year delay scenario, the size of the infected population in 2030 was 10,182, which was an increase of 5784 (132%) compared to the infected population size if DAA therapy had been introduced in 2014.

Discussion

Although the HCV-infected population in New Zealand has probably been relatively stable since 2000, the numbers with cirrhosis have doubled over the last decade, because of an aging cohort effect and very low rate of treatment uptake. The numbers presenting with life-threatening complications of decompensation and liver cancer are projected to treble over the next two decades. However, end-stage liver disease in a patient with chronic HCV infection can be prevented or even reversed by eradicating HCV with antiviral therapy. On a population scale, improvement in both treatment uptake and treatment effectiveness, along with ongoing measures to prevent new infections (such as needle exchange services), should reduce the future health burden from the epidemic of HCV infection.

This study modelled the impact of four different scenarios most likely to reflect PHARMAC responses to the introduction of the new IFN-free, DAA regimens. In the first scenario, the

new DAA regimens with SVR rates >90% are made available, but due to high cost, their funding is limited to patients with more advanced liver disease. Consequently, in this scenario the total numbers of patients treated will be similar to current levels but cure rates will be almost double, resulting in a small but significant reduction in the projected increase in liver-related complications. However, with this approach, the HCV-related health burden from HCV and related costs will continue to increase for the next two decades.

In the second scenario, access to the new DAAs is widened to include all stages of liver disease. The availability of more effective, better tolerated therapy will drive a rapid increase in treatment uptake, because there will be no more IFN-ineligible patients (those with contraindications to, or intolerance of IFN). Given that only 1.8% of diagnosed patients are currently treated per annum, a four-fold increase in treatment numbers adopted for this scenario would seem attainable.

The capacity of current treatment services would be boosted by replacing the complex 48 week boceprevir-triple therapy with IFN and RBV-free DAA regimens of very short duration (maximum durations 8–12 weeks), without any need for on-treatment viral load or safety monitoring . However, such increases in treatment numbers will rapidly exhaust the relatively small numbers already diagnosed with HCV, so broadening of access criteria to include all stages of liver disease, and increased HCV diagnosis would also be necessary. The latter would be achievable through improved community awareness and targeted testing programmes, which the current Government-funded National Hepatitis C Action Plan could address.

Finally, we included two additional scenarios in the model to estimate the impact of delaying funding the new DAA therapies for all patients, including cirrhotics, for either 1 or 2 years. These scenarios would reflect a potential PHARMAC decision to wait until there is increased competition from multiple manufacturers of new DAA therapies, which could drive down costs of treatment. However, such a strategy would come at a real human cost, resulting in an additional 377 preventable deaths.

This study estimated that immediate funding of IFN-free, DAA regimens for patients with established HCV cirrhosis would significantly reduce the life-threatening complications of hepatic decompensation and hepatocellular carcinoma, thereby dramatically reducing both liver-related mortality and demand for liver transplantation in New Zealand within the next decade. Widening of access to include all patients with all fibrosis stages could eliminate chronic HCV infection in this country within the next 20 years.

There are a number of limitations with this study. The lack of recent studies quantifying the total HCV-infected population in New Zealand means that our estimates are based on largely historical prevalence studies and expert opinion rather than large community-based seroprevalence studies. In addition, rates of SVR for current and future treatment protocols were based on clinical data from registration studies from centres experienced in treating patients and managing adverse events. SVR rates with DAAs in the real world could be lower than those published in journals if non-adherence is a significant occurrence outside the strict supervision provided during clinical trials.[32] There is variance in HCV prevalence estimates and the relative impact of each scenario may be more or less pronounced if true prevalence is higher or lower than the estimated values.[15]

Another limitation of this study is that modelled increases in treatment rate, diagnosis rate, eligibility and SVR were all assumed to take effect immediately. Adoption of new therapies

and strategies at the national level is more likely to take several years as new treatment guidelines are developed and as PHARMAC funding decisions are made. However, analyses examining the impact of accelerating or delaying increases in SVR or treatment consistently demonstrated that the desired outcomes were more likely to be achieved when the strategies were implemented earlier.

A final limitation is that disease progression was no longer followed when patients were cured. Among cured patients, risks for advanced liver disease and related mortality can remain, but at markedly lower rates.[33] Therefore, the model could overestimate the impact of curing patients on total HCV liver-related morbidity and mortality. Any underestimation is likely to be minimal as most reduction in HCV morbidity and mortality came from prevention of HCV progression in earlier disease stages where progression to more advanced liver disease is unlikely.

In conclusion, although the number of HCV infections in New Zealand is expected to decline over the next 15 years, HCV-related morbidity and mortality from cirrhosis and its complications will increase steadily. Reducing future HCV disease burden is possible with a two-pronged effort, through increased detection of undiagnosed New Zealanders living with HCV and increased treatment uptake, with new DAA regimens. Immediate funding of IFN-free, DAA regimens for patients with established HCV cirrhosis would dramatically reduce both liver-related mortality and the demand for liver transplantation in this country within a decade.

When combined with widening of access to treatment to include all patients, regardless of fibrosis stage, ongoing prevention efforts, targeted testing and treatment programmes in populations at high risk of infection, such as people who inject drugs and prisoners (so-called "treatment as prevention" strategy), these DAA regimens could eventually eliminate HCV infection in New Zealand within our lifetime.[34]

Competing interests: Nil.

Author information: Edward Gane, Professor of Medicine, University of Auckland, Auckland, NZ; Catherine Stedman Associate Professor of Medicine, University of Otago, Christchurch, NZ; Cheryl Brunton, Public Health Physician, University of Otago, Christchurch, NZ; Sarah Radke, Epidemiologist, Institute of Environmental Science and Research, Wallaceville, NZ; Charles Henderson, National Manager, New Zealand Needle Exchange Programme, Christchurch, NZ; Chris Estes, Center for Disease Analysis, Louisville, Colorado, USA; Homie Razavi, Center for Disease Analysis, Louisville, Colorado, USA

Correspondence: Professor Edward Gane, New Zealand Liver Transplant Unit, 15th Floor Support Building, Auckland City Hospital, Grafton, Auckland, New Zealand. edgane@adhb.govt.nz

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CASE REPORT

An uncommon side effect in a common procedure: a case report of an adverse reaction to prilocaine during a Bier's block

Gareth Rooke, Charlotte Blau, Ryan Johnstone

Abstract

This case report describes a rare side effect during a Bier's block. During local anaesthetic injection, the patient suffered a sudden onset painful petechial rash localised to the upper limb, distal to the tourniquet, without systemic effect. After deflation of the tourniquet, the pain resolved and no systemic effects were seen. The skin changes settled without treatment over one week. The discussion summarises standard technique and precautions required for a Bier's block. It also evaluates risks and complications.

A Bier's block is a commonly used technique for regional anaesthesia. It is safe and effective; however, it is not risk-free. This case demonstrates a rare side effect, exacerbated by standard precautions required during this procedure. Patient consent was granted for publication.

Case report

An 80-year-old lady presented with a 3-day-old angulated distal radius fracture. Planned treatment was manipulation under Bier's block. Medical history included stable angina, previous local anaesthetic use during dental procedures, with no known drug allergies. No absolute contraindications to Bier's block were present.[1] Informed consent was gained.

The block was performed in the standard fashion. After 8 ml of 0.5% prilocaine, the patient felt proximal forearm discomfort. This was initially interpreted as pain secondary to tourniquet pressure. The injection ran freely. After 12 ml the patient felt pain in her hand and wrist. Pain increased despite cessation of prilocaine infiltration.

Skin changes with swelling and petechiae (Figure 1) became obvious. There was no evidence of extravasation of local anaesthetic.

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Figure 1. Note the line of demarcation where the tourniquet was present

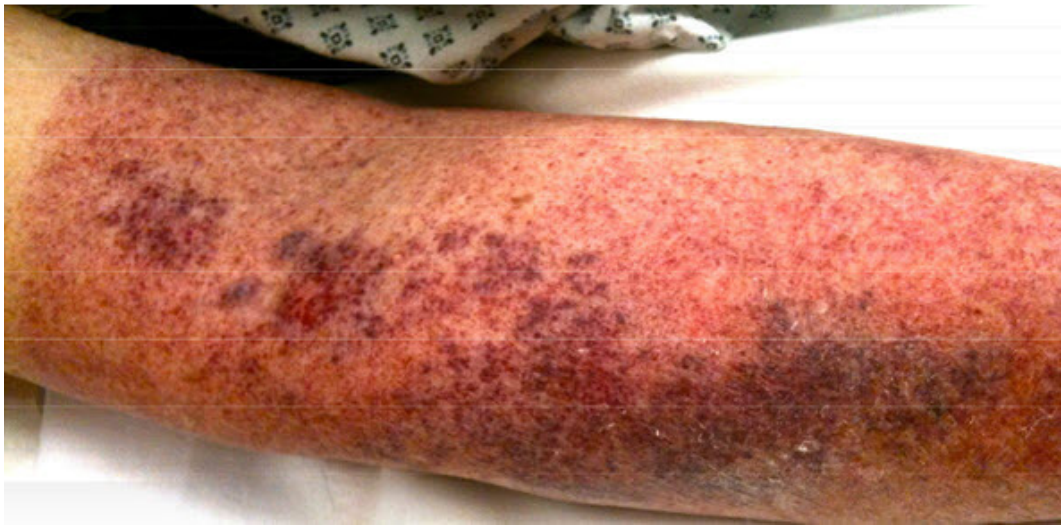

Figure 2. 24 hours after infiltration of prilocaine. Note that the purpura is not specifically concentrated around the cannula site

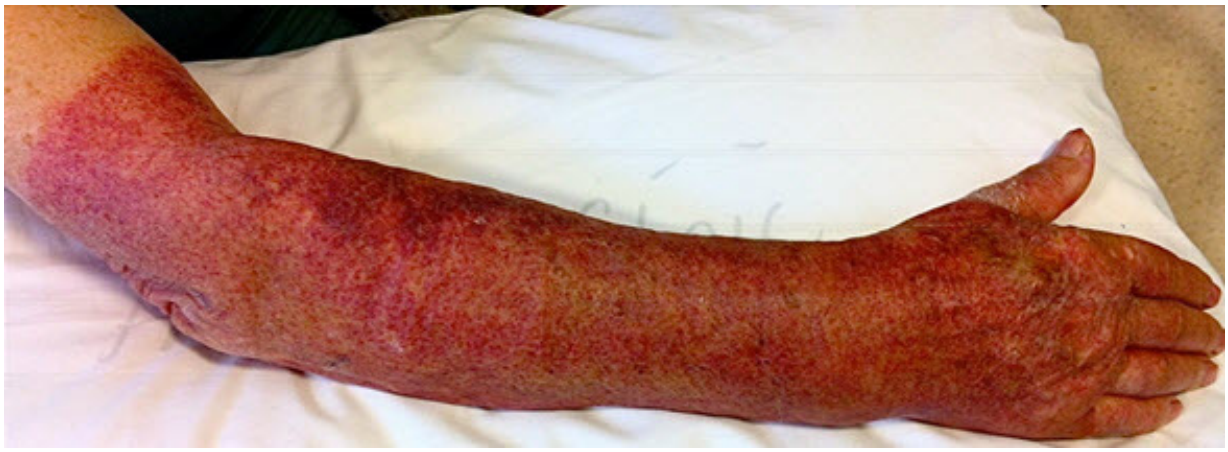

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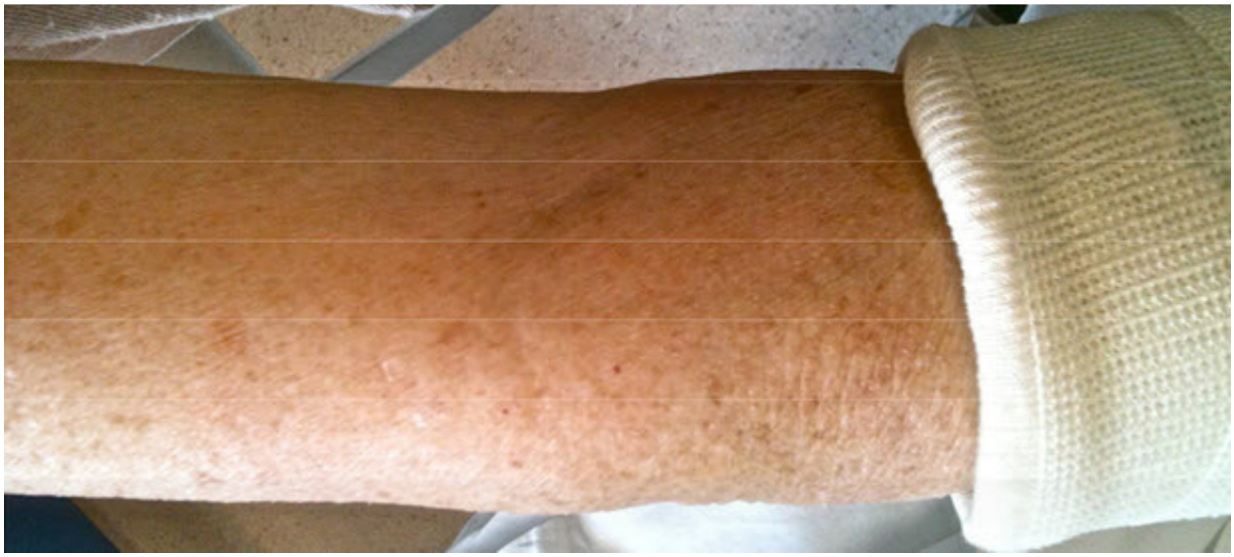
Figure 3. Patient in below elbow cast 13 days post Bier's block with prilocaine. No persistent skin changes observed.



Routine bloods were taken, IV fluids plus antihistamine (promethazine 25 mg) were commenced through the contralateral cannula, oxygen applied and a systemic review conducted. Heart rate was 92 beats per minute, blood pressure 142/86 mmHg and pulse oximetry showed an oxygen saturation of 99% on 6 litres of oxygen via a Hudson Mask. There were no immediate systemic effects of this local reaction. No further treatment was required at this time.

After 20 minutes the tourniquet was removed and the arm pain subsided. The patient suffered chest pain similar to her usual angina. This lasted three minutes and resolved without treatment. Systemic observations remained within normal limits. No electrocardiogram (ECG) changes were evident. Baseline and follow-up cardiac markers were not elevated. International Normalised Ratio (INR) was 1.1 with an activated partial thromboplastin time (APTT) of 32. The patient was monitored for 24 hours. Skin changes remained stable without blistering or necrosis (Figure 2).

No further cardiac symptoms reported. Subsequent treatment of the fracture was a below elbow non-moulded plaster of Paris cast and regular review over the following week. No further adverse events were noted. Two weeks later the skin changes (Figure 3) had completely resolved, without evidence of further complications.

Given the speed of onset, our clinical findings suggested an anaphylactoid reaction to prilocaine which is a rare but significant complication. Few cases have been reported in the literature.[2,3] A serum tryptase, a marker of mast cell activation,[4] may have been useful to confirm or refute this theory.

Discussion

A Bier's block should be performed with blood pressure and cardiac monitoring. Local anaesthesia is inserted into a cannula placed as distally as possible in the affected extremity, with a larger 'safety' cannula in the contralateral antecubital fossa. An appropriately padded tourniquet is placed proximally on the injured arm and the limb exsanguinated to increase concentration of the anaesthesia. The tourniquet should be inflated to 100 mmHg greater than systolic blood pressure.

Prilocaine is most commonly used (0.5 mg/kg) via slow injection, utilising a large syringe. Patients commonly feel discomfort around the tourniquet site, note mottling or a hot flush in the lower

arm. The planned procedure may commence after five minutes. The tourniquet must remain inflated for a minimum of 20 minutes for adequate metabolism of local anaesthetic and to minimise systemic absorption. Then, the tourniquet can be slowly deflated, with monitoring continued for 10 minutes.

A wide variety of complications from Bier's blocks have been reported.[5] Anaphylaxis is possible with any medication. Prilocaine has a low risk of anaphylaxis. Bupivacaine is contra-indicated,[6] as it has a higher risk of cardiotoxicity.

Localised nerve damage, extravasation of local anaesthetic and compartment syndrome are all possible early sequalae.[7] Thrombophlebitis is possible and usually presents late. Methemoglobinaemia is a condition characterised by an altered haemoglobin molecule being present in the blood at a concentration greater than one percent. It has been associated with local anaesthetic use.[8] Increasing concentrations may lead to shortness of breath, cyanosis and mental status changes. Greater concentrations can cause cardiac dysrthymias and death. Severe cases require treatment with methylene blue or hyperbaric oxygen.

Following premature deflation of the tourniquet, or with an inadequately inflated device, systemic absorption of local anaesthesia can occur. Patients may complain of nausea, tinnitus or perioral tingling, and progress to vomiting, muscle twitching, loss of consciousness, or convulsions. In severe cases, ECG changes of prolonged PR, QRS, and QT intervals may occur. This may progress to cardiac arrest.

Toxicity is typically short-lived and treated with airway protection and resuscitation if required, however, inotropes increase survival rates.[9] Local anaesthetics are lipid soluble, therefore in prolonged cardiovascular collapse, treatment with a lipid emulsion can relieve the effects of local anaesthetics on myocytes and increase survival.[10]

It is our conclusion that a prilocaine Bier's block remains a relatively safe procedure, but (as during any procedure) vigilance is mandatory.

Author information: Gareth Rooke, Department of Orthopaedic Surgery, Christchurch Public Hospital, Christchurch; Charlotte Blau, Department of Plastic Surgery, Hutt Valley District Hospital, Lower Hutt; Ryan Johnstone, Department of Orthopaedic Surgery, Hutt Valley District Hospital, Lower Hutt

Correspondence: Dr Gareth Rooke, Department of Orthopaedic Surgery, Christchurch Public Hospital, Riccarton Avenue, Christchurch 8011, New Zealand. gmjrooke@hotmail.com

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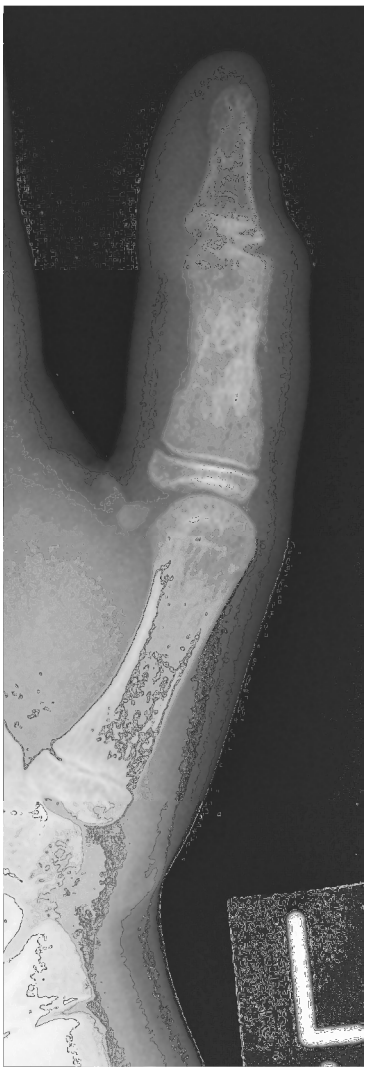
MEDICAL IMAGE

Pain and swelling in a child's thumb

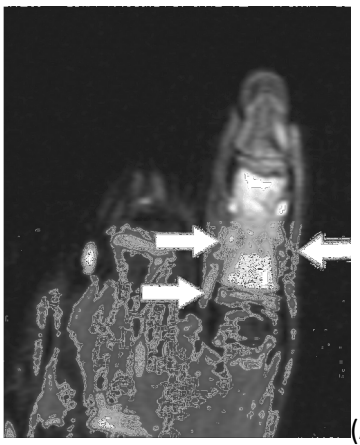
S Claire Gowdy, Anne Paterson, Anthony McCarthy

Clinical—A 10-year-old girl presented to the Emergency Department with a 2-week history of pain and swelling of the thumb. There was no history of trauma nor signs of sepsis. Plain radiographs revealed an aggressive lesion in the proximal phalanx of the thumb (Figure 1). MRI showed the lesion to have breached the cortex, but confirmed sparing of the adjacent epiphysis (Figure 2 A and B). *What is the diagnosis?*

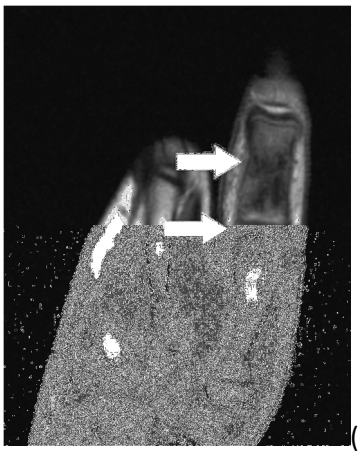
Figure 1. Oblique view of the left thumb showing a mixed sclerotic and lytic lesion in the proximal phalanx, with associated periosteal new bone formation



Figures 2A & 2B: Coronal MR images of the left thumb (A) STIR, and (B) T1-weighted



(A)



(B)

Note: There is a destructive lesion in the proximal phalanx, which has breached the cortex and elicited periosteal new bone formation (respectively indicated by the opposing arrows on the STIR image. Cortical destruction is signalled by the uppermost arrow on the T1-weighted image). The lesion is heterogeneous and there is associated marrow oedema. The adjacent soft tissue involvement is minimal. The proximal phalangeal epiphysis remains normal (more inferiorly placed arrows on both STIR and T1-weighted images).

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Answer—*Ewing sarcoma of the proximal phalanx of the thumb*

Biopsy confirmed a diagnosis of *Ewing sarcoma*. The thumb was surgically removed and the patient received adjuvant chemotherapy; she continues to do well more than 15 months later.

Discussion—Ewing sarcoma is the second most common malignant tumour of bone but it occurs only rarely in the extremities. Within the exception of lesions in the calcaneus, the prognosis for extremity tumours is excellent when compared with axial tumours. The Intergroup Ewing Sarcoma Study document the radiological findings of a combination of permeative bone destruction, irregular areas of sclerosis and a soft tissue mass as the most oft reported findings in primary extremity tumours. Bone expansion, a cystic or honeycomb pattern, and a lack of a laminated periosteal reaction are also described. The radiological differential diagnosis includes osteomyelitis and biopsy is required to confirm the diagnosis.[1,2]

Learning point:

- In the absence of clinical signs of sepsis, malignancy must be considered when an aggressive bone lesion is encountered, even in an unusual anatomic location.

Author information: S Claire Gowdy, Specialist Registrar; Anne Paterson, Consultant Paediatric Radiologist, Radiology Department; Anthony McCarthy, Consultant Paediatric Oncologist, Haematology/Oncology Department. Royal Belfast Hospital for Sick Children, Belfast, Northern Ireland, UK

Correspondence: Dr Anne Paterson, Royal Belfast Hospital for Sick Children, 180 Falls Road, Belfast BT12 6BE, UK. annie.paterson@belfasttrust.hscni.net

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LETTER

Options for expanding community water fluoridation in New Zealand

Nick Wilson, Rob Beaglehole

The expansion of community water fluoridation (CWF) in New Zealand has been recommended in a recent report from the **Royal Society of NZ and the Office of the Prime Minister's Chief Science Advisor.**[1]

CWF is safe and cost-effective and, since only around half the New Zealand population live in areas with fluoridated water supplies, it has the potential to further reduce the burden of oral disease and reduce health inequalities in New Zealand.

Here we outline three key steps for expanding CWF in New Zealand:

- **Key role for Central Government—**Given the health benefits to the community (and financial benefits via the savings to taxpayers who pay for child dental care costs), there is a major role for central government action. This role could logically cover fully paying local government councils for providing CWF and potentially more of other water supply costs. Central Government could also legislate to require CWF in all district health board (DHB) areas except where a well-designed Statistics New Zealand (SNZ) run survey showed less than majority public support. Any such survey could follow a period of community consultation run by the relevant DHB (with appropriate information campaigns). This process would be superior to having referenda which sometimes engage only a minority of voters.

  If instead of the above, the decision on CWF was delegated by Central Government to the 20 DHBs (and no longer the 67 territorial authorities), this could still involve DHB-led community consultation processes and SNZ-run surveys.

  A strong central government role has been advocated for by the NZ Dental Association[2] and the NZ Medical Association.[3] A Health Select Committee report on child health[4] also recommended a shift in responsibility for CWF to the Ministry of Health and District Health Boards. Furthermore, after the issues over stopping/re-starting CWF by the Hamilton City Council and litigation involving other local territorial authorities, Local Government New Zealand requested that the central government take the lead and give responsibility for CWF to the Director General of Health.[5]

- **Research on public knowledge levels and citizen panels—**So that the public can make informed decisions in any community consultation processes, informed discussion is essential around issues of health benefits and risks, health inequalities reduction, and cost saving issues. Research around the knowledge gaps in regions without CWF may help inform how best to design future informational campaigns. There might also be a place for a citizen jury to consider the issues (as per a recent systematic review of such juries[6] and a citizen jury in Australia that considered the issue of taxing soft drinks – which would benefit oral health as well as obesity control[7]).

- **Funding targeted mass media campaigns—**After research on the public knowledge issues (as per the above), central government could also run mass media campaigns to provide information for informed public decision-making in any community consultation processes at the DHB level. Such campaigns would ideally need to be evaluated carefully for effectiveness and cost-effectiveness in improving public knowledge of the issues – and only continued if they meet these criteria.

While expanding CWF is a key strategy to improve oral health in New Zealand, other plausible options exist. These include taxing sugary drinks, taxing snack foods, restricting the advertising of such products to children, and achieving the Government's Smokefree 2025 goal (since smoking contributes to gum disease and oral cancer).

Competing interests: Nick Wilson was an unpaid panel member involved in the new OPMCSA/Royal Society Report on fluoridation.[1] He previously authored a report on CWF for the Public Health Commission.[8] Rob Beaglehole is the NZ Dental Association's Spokesperson on Community Water Fluoridation.

Nick Wilson
University of Otago, Wellington, New Zealand
nick.wilson@otago.ac.nz

Rob Beaglehole
New Zealand Dental Association, Auckland, New Zealand

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LETTER

Health effects of water fluoridation—how "effectively settled" is the science?

David B Menkes, Kathleen Thiessen, Jonathan Williams

The recent publication of a high-profile review[1] marks a milestone in the New Zealand (NZ) discourse regarding this controversial public health practice. Jointly sponsored by the Royal Society (RSNZ) and the Prime Minister's Chief Science Advisor, the review strongly supports the Ministry of Health's promotion of community water fluoridation (CWF) and concludes "there are no adverse effects of fluoride of any significance arising from fluoridation at the levels used in New Zealand."

The developmental neurotoxicity of fluoride is well established, with plausible mechanisms, based on observational studies and preclinical toxicology,[2–4] but its relevance to CWF remains controversial due to uncertainties about exposure (dose × duration, across all sources), effect thresholds, and uncontrolled confounders. Significant IQ deficits in children exposed to high fluoride were reported by two meta-analyses;[3,4] the RSNZ review[1] considered only one of these and dismissed its relevance, apparently due in part to mistaking the average deficit to be around half an IQ point rather than half a standard deviation (seven IQ points).[5]

We consider below a recent NZ study that reported no IQ differences attributable to childhood exposure to CWF.[6] Although the RSNZ review cites this as indicating neurodevelopmental safety, several considerations suggest that the study failed to adequately assess possible IQ effects of fluoride exposure.

The IQ of the Dunedin birth cohort was analysed in relation to childhood residence in suburbs with or without CWF.[6] The authors control for socioeconomic status, but unmeasured confounders operating at suburb level are likely to influence IQ and were not considered in the analysis.[7] This is important, because residence determines CWF exposure and thus measurements are clustered within the same geographic groupings that determine the study's 'design'.[8]

Suburbs with CWF were mostly in central Dunedin while those without were peripheral; it is thus very likely that unmeasured factors co-vary with suburb. For example, most children without CWF exposure lived in Mosgiel, which borders a farming district and has rural characteristics. Rural children, as the authors note, typically have lower IQs than those from urban areas.

Controlling such confounders may be possible with hierarchical mixed-effects modelling, using random intercepts for the clustering factor—in this case, suburb.[7] Analyses could also include structured random effects to model spatial relationships within the data—for example, distance from the city centre. Hierarchical modelling may strengthen the study's results, provided the geographical clusters are not inextricably confounded with fluoride exposure.[8]

Breastfeeding and fluoride supplementation also deserve further analysis. Given data suggesting the beneficial IQ effect of breastfeeding is more pronounced in CWF areas (Table 3),[6] a CWF × breastfeeding interaction, including all available duration data, should be reported. This is important because the breast actively excludes fluoride from milk,[9] particularly reducing exposure in CWF areas since infant formula is typically prepared with tap water.

Likewise, the authors do not report the distribution of fluoride tablet or toothpaste use according to CWF status; these additional fluoride sources, and available duration data, warrant inclusion in the multivariate model to better estimate exposure and enable more sensitive hypothesis testing.

More children were exposed to tablets (n=139) or toothpaste (n=874) than resided in non-CWF areas (n=99); comparing groups with overlapping total fluoride exposure thus compromises the study's

statistical power to determine the single effect of CWF. Individualising exposure estimates is thus essential to test the possible impact of fluoride exposure on IQ. For example, a subset of children was exposed to CWF, fluoride tablets and toothpaste; their IQs would be of particular interest given intensified exposure.

Finally, the Dunedin study's analyses assumed that IQ is normally distributed. In fact this distribution has a negative skew, plausibly because infrequent combinations of environmental and genetic factors may disrupt brain development.[10] Neurotoxin exposure may thus have little effect on mean IQ, while markedly increasing the number of children with impairment (IQ<70, say).[4] The Dunedin dataset offers a unique opportunity to explore this possibility, taking into account all factors affecting fluoride exposure (CWF, tablets, toothpaste, breastfeeding) including duration of each. Individualised exposure and effect assessment, as suggested here, were recommended by the National Research Council (USA) in 2006.[2]

Available evidence regarding both benefits and harms of CWF is generally of low to moderate quality, making firm conclusions doubtful.[11] There are, for example, no prospective, randomised trials, and a Cochrane Review is not yet available. Public debate is typically polarised, reflecting disparate ethical and professional views of CWF; both sides are known to 'cherry pick' and distort evidence to suit their respective arguments.[11]

In describing the Dunedin study[6] as "extensive" and the risk of IQ effects from CWF as "imperceptibly small" the RSNZ review[1] appears to have overstated available evidence. Given the importance of the issue, and the numbers of children exposed, exhaustive and impartial evaluation of scientific evidence should be required to inform public policy.

David B Menkes
Associate Professor
Psychiatry, Waikato Clinical Campus, University of Auckland
Hamilton, New Zealand
David.Menkes@auckland.ac.nz

Kathleen Thiessen
Senior Scientist
Oak Ridge Center for Risk Analysis
Oak Ridge, TN, United States

Jonathan Williams
Psychiatry
Far North Mental Health & Addictions Services
Northland DHB, Kaitaia, New Zealand

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LETTER

Crisis checklists at every hospital bedside?

Hamish M Lala, Robert A Martynoga

Cognitive aids designed to supplement and support clinical management during crises are becoming commonplace. Most development has happened within the operating theatre environment led by anaesthesia. Observational evidence supports the utility of crisis checklists.[1]

Controlled trials outside of simulation are difficult to perform. It is conceivable that critical incident checklists should have clinical, educational, and organisational benefits. Their development within each individual hospital's particular working structure encourages teamwork between medical disciplines, nursing and allied health professionals**.**

The goal is not to provide a prescription of management for any given situation, rather a 'first 5 minutes' path of well-established critical care for uncommon but important clinical events, followed by suggestions relating to differential diagnosis, investigation and immediate treatment.

The next most logical place to consider their implementation is in the intensive care unit (ICU). We recently conducted a survey of 58 Australasian ICUs which showed that checklists in the ICU that provide such guidance are rare. A medical representative from 37/58 (64%) ICUs responded to the survey request. 10/37 (27%) units had some form of bedside clinical checklist. Of those ICUs that had a clinical checklist, 8/10 (80%) found it to be a useful resource. The checklists in use were almost exclusively limited to airway algorithms or cardiac arrest scenarios. The development of a checklist that explored a variety of important events was thought to be a potentially worthwhile undertaking by the remaining 21/27 (78%) ICUs.

Could a simple crisis checklist relevant to the patient's environment (emergency department, operating room, post-anaesthetic recovery unit, critical care unit, ward) improve patient safety at every hospital bedside? Clearly there is a risk of checklist fatigue. Evaluation of effectiveness will be required, as will continuous critical appraisal to ensure their relevance – but it will be important to consider changes in staff psychology and attitudes to crisis management as part of that process.

Hamish M Lala
Registrar
Intensive Care Unit, Department of Critical Care
Waikato Hospital, Hamilton, New Zealand
hamish.lala@waikatodhb.health.nz

Robert A Martynoga
Anaesthetist & Intensivist
Intensive Care Unit, Department of Critical Care
Waikato Hospital, Hamilton, New Zealand

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LETTER

New Zealand Emergency Medicine Network (NZEMN): collaboration for acute care research in New Zealand

Martin Than, Peter Jones, Stuart Dalziel, et al; The NZEM Network

Emergency Medicine (EM) is a relatively new specialty in Aotearoa New Zealand that has come a long way in a short time. The Australasian College for Emergency Medicine (ACEM) was founded in 1984 and the first New Zealand Fellowship of the College was awarded in 1989. EM was formally recognised as a specialty by the New Zealand Medical Council in 1995 and now there are almost 200 specialists providing and supervising care to over one million patients per year. In parallel to the development of the medical workforce the nursing workforce has also embraced the specialty; the Emergency Nurses Section of the New Zealand Nursing Organisation started in 1993 and became the College of Emergency Nurses (New Zealand) in 2001.

The initial focus of any new specialty is the establishment of adequate clinical capacity and associated training. However knowledge generation, academic development, and scholarship are the true hallmarks of specialty maturation. The first Chairs of EM were conferred in 2000 at the University of Otago, Christchurch Clinical School and in 2011 at the University of Auckland, School of Medicine.

Alongside formal teaching the development of research in EM has been a relatively recent phenomenon. The first article relevant to EM was published in the NZMJ in 1968.[1] Since then there has been a steady rise in the number of articles to around 10 per year. Most of these articles have been opinion pieces, descriptive studies or non-randomised comparative studies. To date, few have been randomised controlled trials (RCT) and most of these have been small, single-centre studies.[2-5] More recently, EM specialists have lead multicentre RCTs[6] and obtained major research grants from the Health Research Council and from the National Health and Medical Research Council (Australia) to conduct research either within New Zealand, or across EDs in Australia and New Zealand.[7]

It is now time for New Zealand's EM community to develop a high quality evidence base for acute care through collaborative multicentre research within an established research network. This will help overcome the barriers to research and knowledge translation in emergency settings such as the balance between service delivery and research, consent, data quality, and delays in translating findings.

With this in mind a group of EM specialists with a shared vision convened the inaugural meeting of NZEMN on 6/11/2013. At this meeting the core principles of the network which form our Vision and Values were adopted (Box). The NZEMN is based on a voluntary collaboration of committed people and local champions with national representation, working together in good faith to answer questions that would be possible otherwise. While such an egalitarian structure is perhaps less stable than a central coordinating centre run by permanent staff, it is more flexible financially and more nimble from a research content perspective.[8]

First projects

At the inaugural meeting steering committee members were asked to table ideas for projects that may be considered by the group. Twenty-one ideas were tabled, ranging from descriptive studies of the demographics and type of care provided in New Zealand emergency departments to quality and standardisation of care and studies.

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New Zealand Emergency Medicine Network Vision and Values

Our Vision

"One emergency department for New Zealand/Aotearoa"

New Zealand/Aotearoa will have a world-leading, patient-centred emergency care research network, which will improve emergency care for all, so that people coming to any emergency department in the country will have access to the same world-class emergency care.

Our Mission

To work with stakeholders at all levels to foster discovery and advance the art and science of emergency medicine. To achieve this mission we will pursue the aims of:

- Knowledge creation through high quality research and innovation
- Knowledge translation through stakeholder involvement and networks, both local and international
- Stewardship and value for both patients and the general public

Our Values

Person-centred
By having the patient/consumer at the heart of everything we do, we support individual and family/whanau participation and decision-making about emergency care research at every level.

Practice-informing
By conducting research of a high standard and evaluating the effectiveness of interventions while maintaining a practical approach, we create strong evidence to inform best practice.

Partnership & Openness
By working alongside local and international stakeholders we improve the quality of emergency care knowledge translation in New Zealand/Aotearoa and globally. We value the views of other professionals and diversity of culture and opinion. We openly share ideas and knowledge amongst research network members while respecting individual contributions.

Transparency & Integrity
We share information with integrity and advocate for each other's projects in a culture of support and collaboration. We are trustworthy and communicate in clear language for all to understand.

Leadership
By showing strong leadership we set the direction for emergency care research in New Zealand/Aotearoa and encourage innovation and change to achieve our shared vision.

After a formal selection process, two studies were chosen to be the first project for the NZEMN:

- **Project A. The New Zealand Emergency Department Airway Registry—**This project will establish the current state of airway management in New Zealand's EDs, reporting the first pass success and adverse event rates and evaluating and promoting adherence to best practice for emergency intubations, with the aim of standardizing care across the country.
- **Project B. The Pain Relief in New Zealand Emergency Departments Study (PRiZED 1)—**The timely and adequate relief of pain has been identified as one of the most important facets quality of care in the ED.[9] The PRiZED 1 study will explore the timeliness and adequacy of analgesia in NZ EDs from 2006-2012 and explore the influence on the Shorter Stays in Emergency Departments target on this aspect of quality of care.[10]

Initial funding for NZEMN has come from established departments of EM research at Christchurch, Starship and Auckland hospitals, and from the University of Auckland Lion Foundation Chair in EM. The PRiZED 1 study is funded by the Health Research Council as part of the Shorter Stays in Emergency Departments National Research Project (10-588). In the future, specific funding for projects will come primarily through competitive research grant applications and charitable trusts.

The Network has a focus on public good research and knowledge translation and will not serve as a vehicle for pharmaceutical or diagnostic company research. In order to sustain research capacity and continued growth, NZEMN will seek out broad collaborative relationships with government bodies, health boards, registered charities, and academic entities both domestic and abroad.

The NZEM Network members: Martin Than, Peter Jones, Stuart Dalziel, Luke Larkin Andrew Munro, Craig Ellis, Kim Yates, Paul Quigley, Mike Shepherd, Mike Ardagh, Grant Cave, Adam McLeay, Martyn Harvey, Marc Gutenstein, Thomas Cheri, Dominic Fleischer, Mark Hussey, Alastair MacLean, Brad Peckles, Andrew Swain, Derek Sage, Martin Watts, Andrew Brainard, Marama Tauranga, TeRina Joseph.

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100 YEARS AGO

Notes on the treatment of pulmonary tuberculosis

Extract of an article written by GJ Blackmore, MD, DPH, Medical Superintendent, Cashmere Hills Sanatorium, Christchurch. Read at the Annual Meeting of the NZ Branch, BMA, 1913. Published in NZMJ June 1913;12(46):398–407.

These methods, as you are well aware, comprise the giving of an abundance of fresh air and nourishing food, the employment of rest, the regulation of work and exercise, and the use of certain drugs. To these may be added the giving of tuberculin, as being a form of treatment now in use in most sanatoria. It is of these measures and their method of employment that I propose speaking.

As regards the treatment by means of fresh air, it is first necessary to decide on the kind of climate best suited for the purpose. Undoubtedly good results are to be obtained by living out of doors in any climate, but some climates are much better than others. Fortunately in New Zealand we possess many ideal places. Speaking generally, seaside places do not give the best results.

The kind of place best suited to the majority of phthisical patients is one situated inland, some distance above sea level, where the air is dry, invigorating and free from dust. A bracing cold is much better than warmth. Warm, moist air is the least favourable for consumptives. It is generally considered that the place selected should be sheltered from winds, and on the Continent much stress is laid on this fact. I am convinced from experience that this not necesaary, and that patients do as well in a moderately windy place as they do in still air.

The open-air life should be as complete as possible, that is, the patient should live outside both night and day. A word may be said about the accommodation necessary for sleeping in the open air. The ideal would be to sleep under the open sky, but that is scarcely practicable in most countries. The next best arrangement is a shelter which approximates as nearly as possible to a roof supported on posts. Other sleeping-out places are verandahs, balconies and tents.

In using tents it is necessary to impress the patient with the fact that the tent should have high sides and that these sides should be kept up night and day, only being let down when it is necessary to protect against rain or boisterous winds, and then not any more than is absolutely necessary. Patients have the most extraordinary ideas as to what constitutes the open-air life, so that instructions on the subject must be very precise and definite. I once had a man come to me who could not understand how it was that he was making no progress towards recovery, seeing that he had been living in a tent for months. I found on inquiry that the so-called tent had built-up sides and ends, with a door at one end. He didn't even keep the door open at night! He had been told to live in a tent, and this is how he did it. He did very well when he was set to live the true open-air life.

Whatever kind or sleeping place is adopted the object to be aimed at is to allow a free movement of air over the face. It doesn't matter how cold the air is. It has been shown that cool fresh air passing over the face not only provides a full supply of unpolluted air, but that the passage of such air over the face stimulates the respiratory function and causes deeper breathing. In cold weather the body can be kept warm by a sufficiency of clothing and, if necessary, the use of a hot water bottle. Consumptive persons should always have a long night’s rest in bed. They should go to bed early and get up late.

I do not propose to deal at length with the feeding of consumptives. It is still believed on the Continent that it is necessary to stuff patients. I think most English physicians have given up that notion, while still believing that a considerable amount of nourishing food is necessary. As a rough guide it may be said that adult patients should take about the same amount of food as would be taken by a labourer doing an ordinary day's work. Milk, cream, eggs, and meat should be looked upon as essential articles of diet. In most cases, not less than three pints of milk should be taken daily. Some of the eggs should be taken beaten up in milk.

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METHUSELAH

Efficacy of paracetamol for acute low-back pain

Regular paracetamol is the recommended first-line analgesic for acute low-back pain; however, no high-quality evidence supports this recommendation. This report concerns a randomised trial concerning this hypothesis.

1652 patients with acute low-back pain were randomly assigned to receive regular doses of paracetamol, as needed doses of paracetamol, or placebo. The median time to recovery was 17 days in the regular group, 17 days in the as-needed group, and 16 days in the placebo group. Adverse effects were reported in 18.5%, 18.7%, and 18.5% in the 3 groups. No differences were noted in secondary outcomes (short-term pain relief between 1 and 12 weeks, disability, function, global rating of symptom change, sleep, or quality of life) between the 3 groups.

All patients received advice to remain active, avoid bed rest, and were reassured of a favourable outcome. At 12 weeks about 85% of participants had recovered. The researchers concluded that regular or as-needed dosing with paracetamol does not affect recovery time compared with placebo in low-back pain, and question the universal endorsement of paracetamol in this patient group.

Lancet 2014;384:1586–96.

Postherpetic neuralgia

Approximately a fifth of patients with herpes zoster report some pain at 3 months after the onset of symptoms, and 15% report pain at 2 years. Approximately 6% have a score for pain intensity of at least 30 out of 100 at both time points. The incidence of both herpes zoster and postherpetic neuralgia are both related to age. In one study, the incidence of the neuralgia rose from 8% in the 50–54 year old cohort to 21% at 80–84 years of age.

This review looks at treatment of this painful condition. Lidocaine patches and capsaicin cream may be effective but can cause erythema, and the capsaicin may cause pain. Gabapentin, pregabalin and tricyclic antidepressants may help but their adverse effects may limit their use. Opiates may cause more problems than benefits. Unfortunately, clinical trials reveal that fewer than half these patients have a 50% reduction in pain.

A live attenuated varicella-zoster virus vaccine which became available (in the UK) in 2006 for use in immunoincompetent individuals can significantly reduce the incidence of both zoster and postherpetic neuralgia.

N Eng J Med 2014;371:1526–33.

Effects of hospital-acquired conditions on length of stay for patients with diabetes

This study evaluates the rates and types of hospital-acquired conditions among patients with and without diabetes and assesses any effects on their length of stay (LOS) in hospital. 47,615 admission episodes in a Victorian Hospital were reviewed. The incidence of Hospital-Acquired Diagnoses and the LOS were compared between diabetics with end organ sequelae (EOS), diabetics without EOS, and non-diabetics.

Almost 30% of patients with diabetes and EOS had at least one Hospital-Acquired Diagnosis compared with 13% for non-diabetics. The predominant complications were infections, cardiovascular complications, and metabolic complications. The mean LOS in hospital was found to

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1407/6398

be 8.26 days for diabetics with EOS, 4.01 days for diabetics without EOS, and 2.52 days for non-diabetic subjects.

Internal Medicine Journal 2014;44:1109–1116.

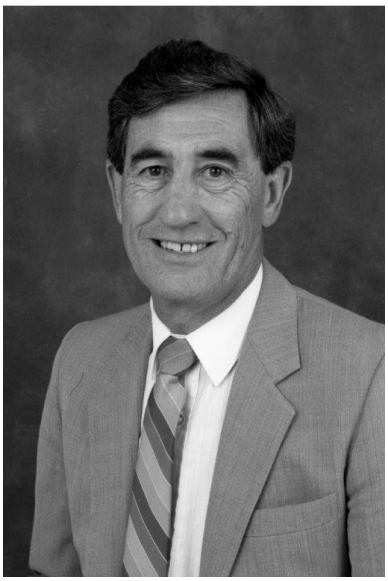
http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1407/6399

OBITUARY

Brian Ernest Tomlinson

MBChB DCH FRACP FRCP

Brian Ernest Tomlinson was born on 3 November 1928 in Gisborne. His father was a surveyor and Great War veteran who had migrated from England, and his mother was a fourth-generation New Zealander. The family moved around the East Coast area, briefly living in Wairoa before settling in Gisborne.



Brian was educated at Nelson College where he played in the top school cricket and rugby teams and was duly awarded the prize for best all-round student.

He then proceeded to study medicine at Otago University where he resided at Knox College.

He spent 5 very happy years there, sharing rooms with his school friend Peter Rothwell and together they explored much of the lower South Island by hitch hiking and, briefly, in a Model T Ford. He was on the Knox College student executive for 1951 and 1952.

His final year as a Medical Student was spent in Auckland where he met Dorothy, his first wife. He was appointed House Surgeon in New Plymouth for two years and then Medical Registrar at Waikato Hospital.

After he had a period of General Practice in Hamilton East in partnership with Russ Freeman, a 2 year locum undertaken by Peter Rothwell, recently returned from overseas study, enabled Brian to travel to Britain in 1961. There he undertook paediatric specialisation, his goal since school days, gaining a Diploma in Child Health from London. He also obtained memberships of the Royal Colleges of Physicians in both London and Edinburgh.

The young family spent time in William Goodenough House and then later in Aylesbury, where Brian was Registrar to Dermot McCarthy at Stoke Mandeville hospital. He was subsequently appointed as a visiting Paediatrician at Waikato Hospital, alongside Colin Watson and David Pullon.

Waikato Hospital was rapidly expanding and it became apparent that three paediatricians were not sufficient, particularly with the emergence of the new field of neonatal intensive care. At the other end of the paediatric age scale, Brian began to express an interest in Adolescent Medicine. He became Head of the Paediatric Department for a time and was later elected Treasurer of the Paediatric Society. He had a busy private practice and he particularly enjoyed conducting newborn baby checks.

Brian was always interested in continuing medical education and regularly travelled overseas, gathering new ideas for practice back home and identifying visiting speakers for future conferences. He actively supported Waikato Registrars who were sitting postgraduate examinations and saw some success, leading to the emergence of Waikato Hospital as a training centre of excellence. He had earlier been appointed to the post of Director of Postgraduate Medical Education at Waikato Hospital, which he took very seriously, promoting the concept of lifelong learning at every opportunity. He continued in this role for 22 years.

Brian retired from paediatrics at Waikato Hospital and subsequently undertook locums in Taranaki, Whakatane and Invercargill in New Zealand and in the UK.

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Brian was a fit and active man and keen sportsman. He was awarded University Blues in shooting and tennis and continued to play tennis at home until his retirement. He worked hard in his garden at home and built several paths beside the Waikato River. He was a keen skier from student days and continued into his seventies, delighting in earning his free ski pass. He had a lengthy association with Christiania Ski Club prior to obtaining his own property near Turangi.

Soon after finally retiring Brian had an abdominal aortic aneurysm repair and after this procedure was completed he became troubled by progressive dementia. He spent his last few years in institutions, latterly in the wonderful care of the staff at Eventhorpe Home in Hamilton. He died on 13 May 2014 at 85 years of age.

Brian is survived by Joanne (his wife of 34 years), 3 sons, 2 stepchildren and 10 grandchildren. Of his 3 sons, Paul is a paediatrician in Invercargill, Matthew is an orthopaedic surgeon at Middlemore Hospital, and Nicholas is a corporate lawyer in Singapore.

A son Paul Tomlinson (Invercargill) and friend Peter Rothwell (Hamilton) wrote this obituary.

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OBITUARY

Charles Marshall Luke

MB ChB (NZ) 1949, MRCP (Lond) 1954, MRACP 1957, FRACP 1965, FRCP (Lond) 1972

Dr Marshall Luke died peacefully on 3 March 2014 at the age of 87 years.

Born at home in Roseneath, Wellington to Ken and Alice Luke in 1926, Marshall was schooled at Scots College.

The only boy amongst four sisters, he studied medicine at Otago University, graduating in 1949. It was there he met his wife and lifelong partner Beverley Brown, a Doctor of Medicine herself.

Marshall spent his years as a house surgeon and registrar at Wellington Hospital.

In those years he assisted in a number of cardiac catheterisations under the guidance of Verney Cable. He well remembered the delight shown by Verney during such an investigation in 1952 when the catheter proceeded directly from the RV into the aorta in a patient with Tetralogy of Fallot.

Of course, these days, there are easier methods of making such a diagnosis and intervention, but with Verney, Marshall (as a graduate doctor) was in a pioneering field.

Taking "The Queen's shilling" he travelled by ship to England as the Ship's Doctor in 1953, where he then enrolled as a PG student at Hammersmith Hospital in 1953. Travelling separately on another ship, Beverley and their two young children joined him afterwards. In 1954 he was appointed SHO to Drs' J G Scadding, Charles Fletcher and Emeritus Professor McNee. In 1954 he became Medical Registrar at the Cardiothoracic Hospital in Sully, Wales.

Interspersed with some camping journeys in Europe with Beverley and their children, they all returned to Wellington in 1956 where he became a Medical Tutor to Wellington Hospital (1956–57) and Acting Staff Health Officer (1957–58). This, at the time, was a typical pathway for a young Doctor returning from his/her OE and post-graduate training.

In 1958, Marshall started a private practice in Khandallah. He also held the appointment of Visiting Physician in the Cardiology Department at Wellington Hospital, from then until 1991. He was also appointed Visiting Physician (general medicine) until 1989. He served on Ward 9 in Wellington Hospital through these years. Simultaneously, in 1959, he established his own practice in Kelvin Chambers on The Terrace as a Consultant Physician. During these years and into the 1960s he served as Commanding Officer, with the rank of Colonel, of the 2nd General Hospital at the Waiouru Army Camp. Other roles included Visiting Medical Officer at the VD Clinic from 1959–1960.

Retiring in 1995, Marshall continued a number of ongoing consultancies; to Wellington Hospital, to Civil Aviation (famously in this role, he grounded the pioneer aviator Fred Ladd), to Colonial Mutual Life and to Swiss Reinsurance.

In his personal life, Marshall, with Beverley, brought up one daughter and three sons. In later years, between them, he also enjoyed the company of 10 grandchildren. His passions were for gardening especially roses (like his own father), golf which he loved but played badly, bridge, holidays at Lake Taupo where he and Beverley perfected trout fishing by harling the reefs, and fast cars! Marshall's ideal day was a millpond-like lake, a beach in the Western Bays, and some trout to take home. These were his "Taupo days".

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Less is perhaps known about Marshall's spontaneous generosity. He gave money to those he thought to be in plight. After driving through a flood-stricken Manawatu he contacted an affected farmer and donated him money, no questions asked. This generosity extended to others in a wide array of circumstances.

As a physician, Dr Luke was noted for his astute and accurate readings of X-ray's and ECGs. He had the mind and eye to see through the fuzzy images of these technologies and really see what was going on. Diagnostically he was pitch perfect. Perhaps better though, was his ability to talk with his patients and see the signs and causes of their ailments where others had misunderstood them. One case was a man who had been bounced around the medical system presenting bruises and loose teeth. After some gentle discussions with the patient about his diet, Marshall diagnosed scurvy as the cause, an ailment that no-one else had even dreamt of.

In the medical profession, a doctor of Marshall's capabilities and intuition is a very valuable person. In his career he combined a remarkable knowledge with a very humane sensibility towards his patients. His career is exemplary for this combination and he detected those little tricks and ticks that many others missed. His humanity and care, combined with an indepth medical knowledge, is a career skill that is so important. He saved, through these skills, many lives that could have otherwise been lost.

In his retirement years, Marshall, with Beverley, lived in a new, architecturally-designed house. This and its garden gave him enormous pleasure, all the better for it being right next to a golf course. There he practiced his (never very good) golf skills, but he took much delight in walking the course, despite the many times he had to try to hit the ball. He became involved with learning to use a computer and through this went on to become a teacher for Senior Net, helping other elderly people access and use computers.

Marshall's failing health took him into respite care in late 2012 and latterly into hospital care in early 2014. He retained a sense of humour, a reasoned mind and a great kindness to all around him right up until his death.

Marshall is survived and missed by his wife Beverley; his daughter Adrienne and her husband Lawrence; his sons Richard and his wife Jill, Ken and his wife Gill, Alistair and his wife Sharon; and his grandchildren Nicki, Sophie, Jonathan, Michael, Simon, Sam, Katie, Ben, Lise, and Helena.

He was a very special man and a remarkable medical professional.

Alistair Luke wrote this obituary.

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OBITUARY

Murdoch Macrae Herbert

QSO (born 28/2/1924 – died 4/8/2014)

Murdoch was born and educated in Glasgow, Scotland.

After obtaining his MB ChB in 1946, he served with the Royal Army Medical Corps in Palestine in 1947–48.

Murdoch re-turned to Glasgow where he trained as a GP Obstetrician. After initially practising in Clydebank, Murdoch became dissatisfied with working in the UK's new National Health Service (NHS).

As a result, Murdoch brought his wife Jessie and three children to New Zealand. In 1963 he worked as a GP in Wainuiomata, Wellington. In 1965, Murdoch was invited to join Gibbie Abercrombie and Keith Watt in their practice in Milford, Auckland.

For the next 40 years Murdoch has been regarded as a most respected GP Obstetrician who attended in excess of 3000 deliveries.

Murdoch actively involved himself in medical politics and other local issues. Through his membership of the North Shore NZMA, Murdoch, with his colleagues, set up the North Shore After Hours GP Service, now called Shorecare.

He strongly advocated for improved access to services for North Shore patients, particularly urgent access to Caesarean sections for maternity patients. These campaigns resulted in the opening of a General Hospital on the North Shore in 1984. Murdoch's advocacy on obstetric and other issues resulted in his membership of national maternity service tribunals in the 1970s.

He was a foundation member of the Royal NZ College of General Practitioners and became the President in 1991–1992. In 2005 he held the office of President of NZMA. Furthermore, he served on many Association and College committees during the 1980s.

Murdoch was also an active and respected member of New Zealand's senior medical body, the Medical Council, for 13 years from 1987 to 2000.

In 1991, Murdoch was awarded the Queen's Service Order (QSO) for services to Medicine and the Community.

Dr Alan Sutherland (a colleague of Murdoch at Dodson Medical Centre Milford for many years) wrote this obituary, which originally appeared in the bulletin of the Probus Club of Milford.

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BOOK REVIEW

Are we all scientific experts now?

Harry Collins, Published by Polity Press, 2014, Contains 140 pages, ISBN 978-0-7456-8204-4.
Price AUD $20.95 (paperback)

Harry Collins is a Research Professor at Cardiff University who works alongside physicists, as Jane Goodall did with chimpanzees. He learned their language, studied expertise, and advanced the sociology of scientific knowledge to place it securely alongside the history and philosophy of science.

In the public mind, mid-twentieth science was an activity done by heroes in white coats selflessly pursuing knowledge for the common good, and Collins calls that attitude Wave 1. But then there was a succession of failures; promises of energy too cheap to measure, nylon shirts that became sweaty and turned yellow, Comet jets that crashed, new economics that failed to prevent stagnation, "mad cow disease", and the promise of fusion power that became a joke. Failures like these and the Cartwright Inquiry in New Zealand changed the public's attitude to science.

"If our science is being made by puppet-masters pulling the financial strings of scientists", the critics asked, "Then aren't the opinions of Jane Doe and the man on the Clapham omnibus as good as anyone's?"

When hackers motivated by climate change skepticism publicised e-mail correspondence from a research unit "the public saw how they talk to each other when they are off the record, and it was horrifying: it did not look like science was supposed to look. It looked like scientists were fiddling the books to keep their data hidden from skeptics. The tête-à-tête among professionals was not understood by amateurs but in the public mind scientists were no longer infallible heroes.

But hold on, we see scientific and technological success all around us. Neil Armstrong walked on the moon; smallpox has been eradicated and much else besides, so why in the public mind has the balance sheet of benefit and loss been added up negatively? It's the world view, the zeitgeist. The overarching attitude to science is why the public adds things up that way. "Anyone who lived through the latter half of the twentieth century has seen the zeitgeist change in science along with so much else." The long-established norms of every social hierarchy began to be challenged from 1960. Like Collins this reviewer lived through the 1960s and the 1970s and watched himself and his colleagues drinking deep and refreshing draughts from the new way of living.

In the 1950s with radar, penicillin, nylon, and all the rest it was impossible to doubt the pre-eminence of science as a way of making knowledge. The job for historians, philosophers, and sociologists of science was to explain how the scientific miracle worked. In that "Wave 1" they never challenged either the pre-eminence of science or that scientific ideas were good because they were contestable against reality.

But since the 1960s, academics (mostly from the social sciences and humanities) have been trying to turn us all into default experts by asserting that there is nothing special about science. The change came at about the time Kuhn published "The Structure of Scientific Revolutions" (1962). He noted that science textbooks often start with potted histories—little fairy stories, not serious history, but a good way to teach science. These are harmless unless policymakers and the public believe that science is that simple. Scientists who do serious stuff live in a very untidy world. The subtleties of Kuhn's ideas were lost on those academics who were attracted to the simple, punchy version of his new idea. Collins marks this time as the change from Wave 1 to Wave 2. "Academics often engage in a kind of journalism" he says: "they pick up the headline, not the detail when they make use of another's work, and distance lends enchantment."

In the middle of any scientific dispute are the specialists who do the experiments, build the theories, and meet together to argue. What happens inside this core is complicated, filled with others'

calculations, arguments, measurements, and judgments of other's capabilities. That is what a committed professional's life is like. To a non-specialist outside the core, things inevitably become simplified as distance lends enchantment.

Paradoxically what is nuanced and unclear inside the core becomes sharp and clear to those on the outside as all the uncertainties get lost, and journalists give us the latest breakthrough since lunch time. An untidy set of doubts in the centre becomes a compelling and polarising set of certainties as the distance increases. Look how both the climate-warming skeptics and the climate-warming enthusiasts are certain of their respective positions. Whilst the scientists may be pretty sure they are right they do not have the religious certainty of either the skeptics or the true believers.

Inside the core the aim is to get to the truth, and this requires an understanding of the opponent's position. "The distinction between a campaign and a debate may seem subtle but it is not: scientists know when their opponents have ceased to play by the rules and instead of taking their opponents' arguments seriously they are ignoring them or caricaturing them and playing to the public audience, as politicians do." The aim to grasp both sides of the argument is the quality that distinguishes scientific disputes from political arguments.

Collins divides expertise into four categories: default expertise, ubiquitous expertise, specialist expertise, and meta-expertise.

We unconscientiously develop ubiquitous expertise about how to live in our society as we grow up, and we overlook this until attempts to make computers that act like humans cause frustration, or we go to live in another country. We acquire the ability to speak a language without explicit lessons, and learn to read and write, but it is an illusion to think that deep knowledge has been acquired from reading the literature if we lack the skills that tell us what to take seriously and what to ignore. Thus ubiquitous expertise cannot give rise to sound decision making in scientific disputes.

Collins is an interactional specialist, skilled through immersion in physicist's discourse without the participation in their research activities that would gain him contributory expertise. He was shocked to find how few interactional experts there were among the journalists who write about science, especially medical science.

Many citizens have the specialist expertise got from many hours of training for the job, but this provides no warrant to claim "we are all scientific experts now."

Sometimes those who live with chronic diseases become interactional experts in their disease who can contribute to better ways of living with it, and less commonly contribute to training professionals how to manage it. In "Madness made me" Mary O'Hagan reveals how she first became an interactional and then a contributory expert.

The specialist expertise gained from training and experience is sometimes relevant to scientific debate outside the individuals' field, and Collins provides good examples.

Rarely dedicated activists can acquire interactional, and even a degree of contributory expertise, but those who get their information from the internet, and think that have true expertise are dangerous. They do not have the interactional or specialist meta-expertise that goes with the oral community of the contributory specialists. Thabo Mbeki's argument for the non-distribution of AZT was bad enough but the anti-vaccination campaigns—insofar as they are based on the campaigners' impression that they are in possession of technical knowledge are worse. A single child with polio or one who dies, or is maimed by measles, is a tragedy.

Meta-expertise is all about choosing between experts and is a skill that can aid the choice between salespersons and politicians by reference to their demeanour and their readiness with answers: recognising evasiveness is one element of the ubiquitous meta-expertise we gain from experience. Meta-expertise is the only possibility in democratic elections and it is the idea behind juries, but juries require instructions from the judge, and sometimes juries are not used in highly technical cases.

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"Yet do we really believe ubiquitous meta-expertise can be a good enough foundation from which to mount a campaign that can damage future generations by re-establishing eradicated diseases?"

Collins thinks that journalists and whistle-blowers can serve a vital function, not by trying to do half-baked technical science, but from serious political investigation of science. However such investigation must be properly done and show that. Personal conviction is not expertise, nor is a good television personality.

Kaufman's study of anti-vaccination campaigners and their reported impact on parents shows how the zeitgeist influences default expertise. "With the explosion of anti-vaccination information on line", Kaufman said, "Many parents see the most respected vaccine experts' perspective as just one more opinion". But scientists are not people who have just one more opinion in their field. In Wave 1 it was thought that scientists had a special kind of opinion because their work led to true and efficacious results; science stood above the plane of other ways of being in the world when it came to accessing the workings of nature. But in Wave 2 "Mount science" was eroded, and many people came to believe that scientists have no special qualities. Collin' sociological studies of science show that this is wrong, because they are special in the values that drive them. Supposedly scientific results driven by financial or political motivation, for example by tobacco companies, is wrong and should be unmasked by investigative journalists.

Collins concludes that although we may be experts in one way or another we are not all scientific experts now. If we believe that we are then those with the most media appeal will make our truths, according to the interests they are pursuing. We have to raise the value of ordinary science in our minds. This is his prescription for Wave 3. Set aside scientific fraudsters, scientists who are primarily driven by greed or by fame. Set aside those for whom science is primarily about the generation of wealth, and those who allow themselves limitless licence to speculate. We need theorists who are ready to identify measurements that will support or falsify theories, and those who are driven to find out why the world is the way it is.

Collins' work is important for medicine; we must recognise that our specialist medical knowledge does not necessarily make us good managers; and think about how specialisation tends to confine medical practitioners' expertise to narrow areas making us default experts outside our own field. John Kellett found evidence of this when he asked, "When did doctors become scientists and why did it all go wrong?" Wave 3 science is required to solve health care challenges, but as Osler observed long ago the pursuit of wealth and a "bauble" reputation can obscure that goal.

John B Morton
Professor
Christchurch, New Zealand

References

1. O'Hagan M. Madness made me. Publisher: Open Box, 2014.
2. Kellett J. When did doctors become scientists and why did it all go wrong? Interna Med. 2008;8(1):8-11.

2015 GRANT APPLICATIONS

PROJECT GRANTS
Project Grant applications will be considered at the July 2015 meeting of the Scientific Advisory Group. The closing date is: **1 March 2015**.

FELLOWSHIPS AND SCHOLARSHIPS
Applications for **Overseas Training and Research Fellowships, Research Fellowships** and **Postgraduate Scholarships** will also be considered in July 2015. The closing date is: **1 June 2015.**

LIMITED BUDGET GRANTS

Small Project Grants
Applications for small project grants will be considered at the July 2015 meeting. The closing date is: **1 June 2015.**

Grants-In-Aid
Applications for grants-in-aid will be considered at the July meeting. The closing date is: **1 June 2015.**

Travel Grants
Applications for travel grants will be considered three times in 2015, with the closing dates being: **1 February**, **1 June and 1 October 2015.**

The Foundation broadly supports travel to relevant international conferences and symposia and locally would welcome applications for presentations at CSANZ meetings in Australia and NZ.

The scoring criteria for Travel Grants are available on the Heart Foundation website under http://www.heartfoundation.org.nz/our-work/grants/grant-advertisement. ***NB: Retrospective funding of travel will not be considered. Note the granting dates.****

***SCIENTIFIC GRANTING DATES**
Wednesday 25 March 2015 ***(for Travel Grants only)***
Wednesday/Thursday 22/23 July 2015
Wednesday 4 November 2015 ***(for Travel Grants only)***

PRIORITIES FOR RESEARCH
Applications are particularly encouraged in areas that align with National Heart Foundation strategic priority objectives.

CRITERIA FOR ASSESSING RESEARCH PROPOSALS
Each research proposal will be assessed on:

- Importance to the Heart Foundation
- Scientific merit
- Design and methods
- Project achievability, expertise and track record of the research team.

CV TEMPLATE
Applicants are encouraged to use the CV template available on the Grant Advertisement on the Heart Foundation website.

FORMAT FOR APPLICATIONS
Applicants should follow the format outlined in the revised *"A Guide to Applicants for Research and Other Grants"* which is available on the Heart Foundation website http://www.heartfoundation.org.nz.

Applications should be sent to:
Associate Professor Gerry Devlin
Medical Director
The National Heart Foundation
PO Box 17160
Greenlane
AUCKLAND 1546

NB: When the closing date falls on a weekend or a public holiday, the closing date will be the first working day following this day.

December 2014

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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.

Applications should be directed through the NZMA:

Central Office

P O Box 156

Wellington

Tel: 0800 656161

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1407/6405

NOTICE

2015 NZMJ Publication Dates

2015 Publication Dates (NZ Medical Journal)	
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February	20
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May	1
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June	12
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August	7
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September	4
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