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

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THE NEW ZEALAND MEDICAL JOURNAL
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This Issue in the Journal

Cancer mortality and incidence trends comparing New Zealand and Australia for the period 2000–2007
J Mark Elwood, Sally Ioannides, Lamees Alafeishat

A previous study showed that cancer mortality in New Zealand in 1996ï 97 was substantially higher than that expected from Australian rates. This study compared cancer mortality and incidence in New Zealand for 2000ï 2007 with rates in Australia, to assess if any differences had persisted or changed. The numbers of cancer deaths in New Zealand, by type of cancer, year, sex, and 5 year age group, were compared to the numbers that would have occurred if NZ rates had been the same as those in Australia. From 2000ï 2007, there were each year an average of 586 (15.1% of the total) more deaths from cancer in New Zealand women than expected from Australian rates; and 197 (4.7%) more deaths in men. Higher cancer mortality was seen for most common sites; the greatest excesses were for colorectal cancer in both men and women. Cancer incidence in New Zealand women was 3.3% higher, and incidence in men was 4.7% lower, than in Australia over this period. Cancer mortality remains substantially higher in New Zealand than in Australia, especially for women, although mortality has reduced in both countries. While the differences in 2000ï 07 were slightly smaller than in 1996ï 97, there has been little change since 2000. The greater differences in deaths than in incidence suggest that patient survival is lower in New Zealand.

A comparison of cancer statistics in New Zealand and Australia: 1996–2007
John Waldon, David S Lamb, Brett Delahunt, John N Nacey, Peter J Dady, Carol A Johnson, Alan G Hall, Peter B Bethwaite, Philip Weinstein

For the years 1996ï 1997 and 2006ï 2007, the incidence and mortality of cancer in New Zealand and Australia was compared to determine if differences between the two countries had changed over the decade under study. For the 11 year timeframe of this study, total rates of cancer incidence reduced in New Zealand and increased in Australia. Over the same 11-year timeframe, cancer-specific mortality rates decreased in both countries, but there was no change in the difference between New Zealand and Australian rates, which remained 10% higher in New Zealand. We conclude that the persisting different cancer mortality rates between the two countries is likely to have been partly due to lifestyle and ethnic differences in the populations, and partly due to New Zealanders presenting with more advanced cancers and having less easy access to some treatments.

Screening for pulmonary arterial hypertension in patients with scleroderma—a New Zealand perspective
Sanjib K Ghosh, Michael M Corkill, Hamish H Hart, Kristine P Ng

Screening for pulmonary arterial hypertension in patients with scleroderma is recommended. There is a wide variation of how NZ rheumatologists screen for pulmonary hypertension in patients with scleroderma. A national pulmonary arterial hypertension screening guideline may help standardise treatment in NZ.

Effect of age, gender, ethnicity, socioeconomic status and region on dispensing of CVD secondary prevention medication in New Zealand: The Atlas of Health Care Variation CVD cohort (VIEW-1)
Andrew Kerr, Dan Exeter, Grant Hanham, Corina Grey, Jinfeng Zhao, Tania Riddell, Mildred Lee, Rod Jackson, Sue Wells

There is good evidence to support the use of blood thinning, blood pressure (BP)-lowering, and cholesterol lowering statin medications to improve outcomes in patients with established atherosclerotic cardiovascular disease (CVD). In this national NZ study which included 86,256 patients with CVD in 2011 we found that there was significant under-utilisation of safe and inexpensive secondary prevention medication, particularly in younger people and women. This provides an opportunity to improve CVD outcomes in this easily identifiable high-risk population. Making the gaps in evidence-based practice visible to patients, health providers and health administrators is a critical first step. The data has been made available to the public and medical community in an interactive mapping tool as part of the Health Quality and Safety Commissionôs New Zealand Atlas of Health Care Variation (http://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/cardiovascular-disease/).

Construction and use of mapping techniques to describe the geographical distribution of medication dispensing for the secondary prevention of atherosclerotic CVD in New Zealand: VIEW-2
Daniel J Exeter, Jinfeng Zhao, Grant Hanham, Corina Grey, Sue Wells, Andrew Kerr

This paper outlines the development of two alternate methods of presenting results related to the epidemiology of CVD medication dispensing among patients who have been hospitalised for CVD (see article in this issue by Kerr et al). We demonstrate that the standard template, as used by the Health Quality and Safety Commission is effective as showing variations for a particular group (such as MŪori, or patients aged 50-59, for example). By contrast, our Advanced template was designed to make comparisons between age or ethnic groups more effectively. These maps integrate data in tables, maps and graphs to efficiently display variations in secondary prevention medication to promote opportunities for front-line change.

Stakeholder engagement for the New Zealand Atlas of Healthcare Variation: cardiovascular disease secondary prevention: VIEW-3

Corina Grey, Sue Wells, Daniel J Exeter, Grant Hanham, Jinfeng Zhao, Andrew J Kerr

After collecting data and creating maps of the differences in long-term medication use among people with cardiovascular disease in New Zealand, our research group sought the opinions of doctors and healthcare managers to improve the presentation of our data and identify reasons why certain groups of people receive less medication than others. Among those with cardiovascular disease, younger people, women and those of MÜori or Pacific ethnicity receive preventative medications least frequently. These differences are likely due to a combination of factors and barriers encountered by prescribers and patients. One strategy to increase the use of medication among cardiovascular patients is to regularly report the proportion of patients receiving medications to general practices to motivate these practices to ensure that all patients understand the importance and benefits of long-term preventative therapy. This feedback is also important so that practices are able to monitor their progress and identify groups of patients that may potentially be missing out.

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Charting progress in the battle against cancer

David C G Skegg

Two papers in this issue of the *NZMJ* appear at first sight to convey a disappointing messageð that the previously reported higher mortality from cancer in New Zealanders compared with Australians has persisted.[1,2] Yet these studies also point to good newsð that the overall risk of dying from cancer has been declining steadily in both countries.

The two studies were independently designed to update an earlier comparison of cancer mortality and incidence in New Zealand and Australia, which was published in 2002.[3] Using data from 1996 and 1997, the authors of that report found that New Zealanders of both sexes and in every age group experienced more deaths from cancer than would have been expected. If the Australian rates had applied here, there would have been 616 fewer cancer deaths each year among New Zealand women, and 215 fewer in men. The majority of excess deaths were in people under 70 years of age.

Of the two new papers, that by Alafeishat and colleagues replicates more closely the previous research.[1] Using data from 2000ï 2007, they report that each year there were an average of 586 more deaths from cancer in New Zealand women, and 197 more deaths in New Zealand men, than would have been expected from Australian rates. There was no significant change over time in these differentials. The higher mortality from all cancers combined cannot be attributed to higher incidence rates, and this suggests that overall patient survival is lower in New Zealand.

As in the earlier research, both studies show that the largest differences are due to colorectal cancer in both sexes, and to breast cancer and lung cancer in women. Each year, about 350 New Zealanders die from bowel cancer who would not die if the Australian rates applied here.

A higher risk of dying from cancer, in the presence of similar incidence rates, can be due to delayed presentation with symptoms (or later detection at screening), delayed clinical diagnosis, or having poorer access to the most effective treatment. As both groups of authors point out, it is difficult to disentangle such factors without having systematic data about the clinical stages of the cancers diagnosed (as well as the timeliness of treatment).

For some particular cancers, the higher mortality in New Zealand does partly reflect a higher incidence than in Australia. An example is lung cancer in women, for which the higher prevalence of smoking among New Zealand women must be a key factor.[2] In contrast, the recorded incidence of colorectal cancer is similar or even lower in New Zealand, whereas the death rate is far higher.

So where is the good news in these articles? First, the authors of the paper in 2002 predicted that the gap between New Zealand and Australia was likely to widen during the next few years.[3] This was because Australia had already implemented a national cancer control initiative, whereas repeated calls for such action in New Zealand had

gone unheeded during the 1990s. In fact the gap has not widened, and a New Zealand Cancer Control Strategy was launched in 2003.[4]

Secondly, and more importantly, there has been a remarkable decline in cancer mortality in both countries. Twenty-five years ago, I wrote a leading article for the *NZMJ* entitled *Losing the battle against cancer*.[5] In the years leading up to that time, cancer was the only major cause of death that was continuing to increase. Age-adjusted death rates for four of the five leading sites of cancer had risen over the previous 30 years. It is encouraging to see that, in the 17 years following 1990, the overall cancer death rate declined by about 24% in men and 20% in women.[1]

This is a major improvement in what is still the leading cause of premature death in this country. Progress has been achieved through both preventive efforts (such as measures to control tobacco smoking) and provision of more effective treatment regimens (as in the case of breast cancer). Since the launch of the New Zealand Cancer Control Strategy, there have been important advances in several areas such as the delivery of radiation treatment. Everyone involved in oncology services in New Zealand should take pride in the better quality of care being provided.

A cancer control programme is concerned not only with reducing mortality and morbidity, but also with improving the quality of life for everyone with cancer and providing high quality palliative care for those who cannot be cured. Implementing an effective strategy and action plan needs to involve not only the Ministry of Health and District Health Boards, but also voluntary organisations and researchers.[6]

If New Zealand is to continue to make progress, and perhaps even to catch up with our neighbours across the Tasman, we need to take concerted action. Special attention must be devoted to ethnic and socioeconomic disparities.[7] After the passage of more than a decade, urgent steps should be taken to update the New Zealand Cancer Control Strategy. Alafeishat et al provide an excellent discussion of ways in which we could learn from the Australians about how to improve our performance.

Analyses such as those presented here are valuable not only for charting progress in the battle against cancer, but also for identifying priorities for further action. It is sad that so much acrimonious debate has been devoted to the case for and against prostate cancer screening, while the *elephant in the room*ð colorectal cancerð has been largely ignored.

Do people realise that, while Australians and New Zealanders both have an exceptionally high incidence of bowel cancer, the risk of dying from this disease is much greater in our country? According to the World Health Organization (WHO) Cancer Mortality Database,[8] New Zealand women have nearly the highest mortality from colorectal cancer in the world.

Australia implemented a national bowel screening programme some years ago,[9] and there is a strong emphasis there on early detection and adherence to national management and treatment guidelines. In some parts of New Zealand, even patients with worrying and suggestive symptoms have experienced difficulties and delays in accessing colonoscopy services. Undoubtedly some people who could have been cured will have died as a result.

Randomised controlled trials have shown that screening of asymptomatic people with either faecal occult blood testing or flexible sigmoidoscopy can reduce mortality from colorectal cancer.[10] A pilot programme using the former approach was finally started in the Waitemata area of Auckland in 2011.

The Ministry of Health proposes that any decisions about national screening will be made some time after this pilot is completed in 2015. Presumably much practical information must already have been gleaned from this exercise, and the existence of a pilot should not be an excuse for delaying national planning and the necessary training of personnel.

New Zealanders should demand a greater degree of urgency in addressing their exceptional risk of dying from bowel cancer.

Competing interests: Nil.

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Cancer mortality and incidence trends comparing New Zealand and Australia for the period 2000–2007

Lamees Alafeishat, Mark Elwood, Sally Ioannides

Abstract

Background and aims A previous study showed that cancer mortality in New Zealand in 1996ï 97 was substantially higher than that expected from Australian rates. This study compared cancer mortality and incidence in New Zealand for 2000ï 2007 with rates in Australia, to assess if any differences had persisted or changed.

Methods The numbers of cancer deaths in New Zealand, by type of cancer, year, sex, and 5 year age group, were compared to the numbers that would have occurred if NZ rates had been the same as those in Australia. Trends over time, and also cancer incidence, were assessed.

Results From 2000ï 2007, there were each year an average of 586 (15.1% of the total) more deaths from cancer in New Zealand women than expected from Australian rates; and 197 (4.7%) more deaths in men. There was no significant change over time in these differentials. Higher cancer mortality was seen for most common sites; the greatest excesses were for colorectal cancer in both men and women. Cancer incidence in New Zealand women was 3.3% higher, and incidence in men was 4.7% lower, than in Australia over this period; thus the higher cancer deaths in New Zealand are not due simply to higher incidence. Over this time period, cancer mortality has fallen substantially in both countries; in New Zealand, it fell from 1990 to 2007 by 20% in women and 24% in men.

Conclusion Cancer mortality remains substantially higher in New Zealand than in Australia, especially for women, although mortality has reduced in both countries. While the differences in 2000ï 07 were slightly smaller than in 1996ï 97, there has been little change since 2000. The greater differences in deaths than in incidence suggest that patient survival is lower in New Zealand.

In 2002, Skegg and McCredie published a comparison of cancer mortality and incidence for New Zealand and Australia, using 1996ï 97 data.[1] They found that at that time New Zealanders of both sexes experienced more deaths from cancer than were expected on the basis of Australian rates, for all cancers combined, and for most of the 10 leading sites for cancer deaths.

They concluded that ócconsiderable scope exists for reducing cancer mortality in New Zealandô, but also predicted that óthe gap between New Zealand and Australia in controlling cancer is likely to widen during the next few yearsô.

To see whether the ógapô has persisted, we used a similar method to compare the numbers of deaths (mortality) and of new cases (incidence) in New Zealand in each year from 2000 to 2007, for all cancers combined and for the most common cancers, to the numbers that would have occurred in the NZ population on the basis of

Australian rates. We also assessed whether any ógapô was changing over that time period.

Methods

Information on cancer mortality and incidence from 2000 to 2007, by year, sex, cancer site, and five year age group from 0ï 4 to 85+, was obtained from official publications of the appropriate national groups in New Zealand and in Australia. In New Zealand, cancer incidence and death data is collated by the Ministry of Health, from the New Zealand Cancer Registry and the mortality collection [2]. Australian data is published by the Australian Institute of Health and Welfare (AIHW), which draws on the Australian cancer database, combining data from each State cancer registry, and from the national mortality database [3].

Cancer deaths were classified using the International Classification of Diseases version 10 (ICD 10) in both countries. Cancer registrations are recorded by the oncology conversion of the ICD, version ICD-O-2 being used in New Zealand in 2000 to 2002, and version ICD-O-3 from 2003 onwards; ICD-O-3 was used for Australian cancer incidence the whole period. To facilitate comparisons of incidence and mortality data, both countries link the ICD-O codes to ICD 10 codes, following internationally accepted rules. The Australian data give rates for bowel cancer (colon and rectum) and anal cancers separately, which were summed to be equivalent to New Zealand data on colorectal cancer, which includes the anus.

For each sex, year and cancer, the Australian incidence rates for each 5-year age group were applied to New Zealand population data to give ócexpected, Eô numbers of cancers, to be compared to the actual óobserved, Oô numbers (indirect standardisation), and 95% confidence limits calculated. Results are expressed as the O-E difference as a percentage of the observed New Zealand numbers. To assess trends, the log transform of the O/E ratio was regressed against year.

Results

Mortality and incidence for all cancers combined—Over the 8-year period 2000 to 2007, there were on average each year 7998 deaths from cancer in New Zealand, 3771 in women and 4227 in men.

Applying the Australian death rates to the New Zealand population shows that on average each year there were 586 (15.1%, 95% confidence interval [CI] 11.9 to 18.3) ócextraô deaths in New Zealand women, and 197 (4.7%, 95% CI 1.7 to 7.7) ócextraô deaths in New Zealand men. The proportions of extra deaths per year were fairly constant over time (Figure 1); statistical analyses showed a small increase over time, but not statistically significant, in both women (P=0.1) and men (P=0.7).

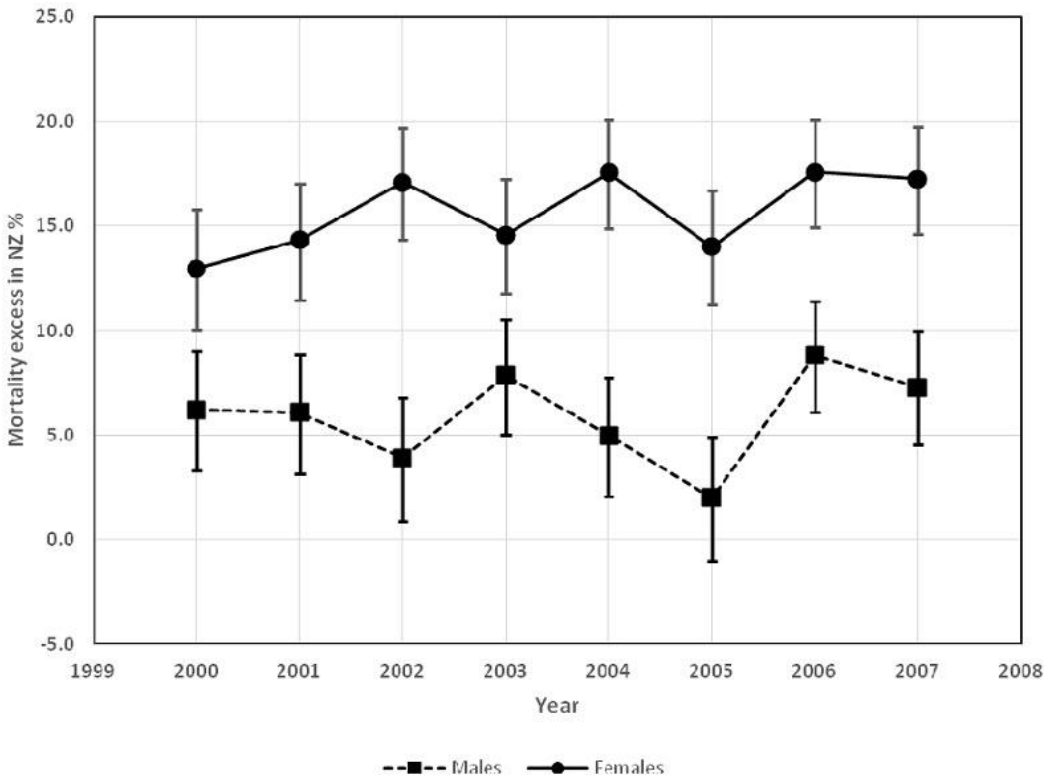
The equivalent analysis for cancer incidence shows an average annual excess in New Zealand women of 287 cases (3.3% of the annual total of 8782, 95% CI 1.2 to 5.4). This excess in New Zealand reduced over time, but the trend was not statistically significant (P=0.09).

In men, the numbers of incident cases were lower than those expected from Australian rates, the average annual difference being 465 (-4.7% of the annual incidence total of 9794, 95% CI -6.7 to -2.7). Comparisons of incidence numbers are greatly influenced by rapid variations in the recorded incidence of prostate cancer. Although over the whole period prostate cancer incidence was similar in the two countries, the incidence fell by 9% in New Zealand between 2000 and 2007, but increased by 10% in Australia.

Thus, for all cancer (including prostate), the incidence in New Zealand compared to Australia reduced over time, but this was only due to the different trends for prostate cancer incidence.

For all cancer sites except prostate, there were overall fewer incident cases in New Zealand compared to Australia, an average ódeficitô of 463 (-6.6% of the annual total of 7038, CI -8.9 to -4.2); and this proportion did not change significantly from 2000 to 2007 (P=0.7).

Figure 1. Excess proportion of cancer deaths in New Zealand, compared to Australian rates, by year, 2000 to 2007. Error bars show 95% confidence limits



Mortality and incidence for major cancer sites—Results for specific cancers are shown in Table 1; the cancers shown are those accounting for the most deaths, plus cervical cancer, included as it has a screening programme. The cancers shown account for 76% of cancer deaths in women, and 70% in men. Leukaemia had to be omitted as the coding systems differ between the two countries. The cancers are ordered in terms of the numbers of annual deaths in New Zealand. For women, for most cancer sites there were more deaths than expected when compared with Australian rates. Colorectal cancer had the highest number and proportion of óexcessô deaths, 204 per year, 35% higher than expected; in contrast, in New Zealand women the incidence of colorectal cancer was substantially (25%) lower) than in Australia.

Table 1. Annual average deaths and incidence cases of cancer in New Zealand, 2000-2007, by sex and site; and differences compared to Australian rates (LL, UL: 95% lower and upper confidence limits)

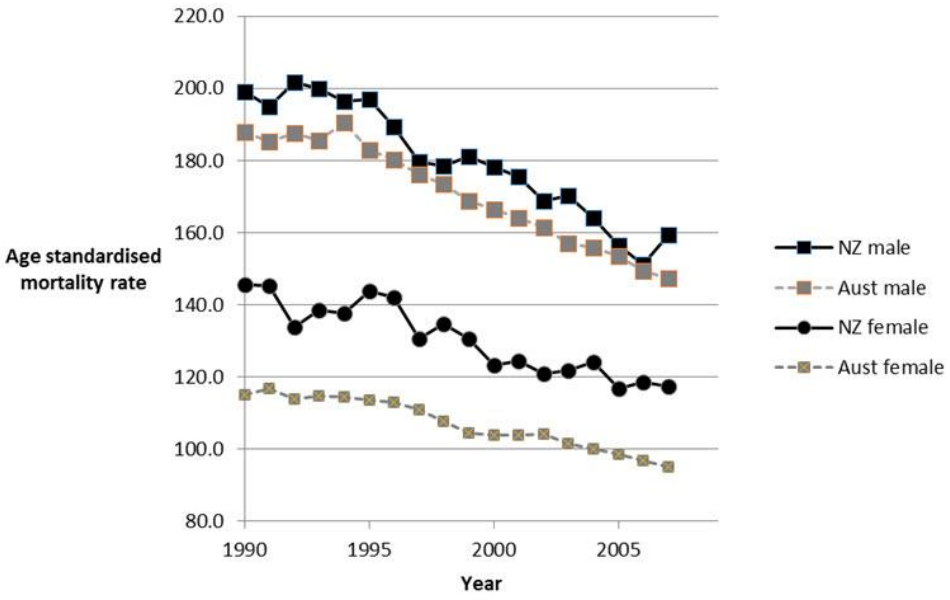
Sex	CANCER SITE	MORTALITY					INCIDENCE				
		NZ annual deaths	Excess / deficit	% excess / deficit	95% LL	95% UL	NZ annual cases	Excess / deficit	% excess / deficit	95% LL	95% UL
Female	**Total cancer**	**3771**	**568**	**15.1**	**11.9**	**18.3**	**8782**	**287**	**3.3**	**1.2**	**5.4**
	Breast	632	120	18.9	11.3	27.0	2403	25	1.0	-2.9	5.1
	Lung	612	120	19.7	11.9	27.9	722	93	12.9	5.8	20.5
	Colorectum & anus	586	204	34.9	27.0	43.4	1324	-338	-25.5	-30.9	-20.0
	Ovary	194	34	17.7	4.1	32.8	296	59	20.1	9.0	32.1
	Pancreas	171	-11	-6.4	-20.9	9.7	183	-15	-8.2	-22.2	7.4
	Non Hodgkin lymphoma	135	6	4.3	-11.8	22.7	301	-31	-10.3	-21.3	1.7
	Stomach	113	29	25.7	8.1	46.0	143	12	8.5	-7.2	26.4
	Melanoma	103	27	26.8	8.3	48.1	922	96	10.4	4.0	17.1
	Brain	90	1	1.5	-18.1	24.5	108	-8	-7.4	-25.5	13.4
	Uterus	88	26	30.0	10.2	53.4	341	3	0.8	-9.5	12.0
	Oesophagus	72	9	12.5	-9.2	38.5	83	11	13.2	-7.2	37.2
	Cervix	62	16	26.4	3.0	54.7	172	26	15.1	0.8	31.2
	All others	914	-15	-1.7	-8.0	5.0	1784	354	19.8	15.2	24.6
Male	**Total cancer**	**4227**	**197**	**4.7**	**1.7**	**7.7**	**9794**	**-465**	**-4.7**	**-6.7**	**-2.7**
	Lung	859	-17	-2.0	-8.6	4.9	972	-102	-10.5	-16.7	-4.0
	Colorectum & anus	589	143	24.3	16.4	32.7	1363	-7	-0.5	-5.8	4.9
	Prostate	577	54	9.4	1.4	17.9	2756	-2	-0.1	-3.8	3.7
	Stomach	181	46	25.2	11.2	40.9	230	-3	-1.3	-13.8	12.5
	Pancreas	166	-21	-12.7	-27.3	3.8	177	-27	-15.2	-29.4	0.6
	Melanoma	162	19	11.5	-3.3	28.2	978	-88	-9.0	-15.2	-2.5
	Non Hodgkin lymphoma	159	11	7.1	-7.9	23.9	347	-49	-14.1	-24.4	-3.0
	Brain	134	12	9.0	-7.2	27.5	152	-3	-2.0	-17.2	15.2
	Oesophagus	133	-8	-6.0	-22.3	12.5	161	-13	-8.1	-23.0	8.6
	All others	1268	-42	-3.3	-8.8	2.3	2659	-171	-6.4	-10.2	-2.6
	All except prostate	3650	143	3.9	0.7	7.2	7038	-463	-6.6	-8.9	-4.2

Substantial excess deaths were also seen in women for breast cancer (120 excess deaths per year, 19%), while incidence was about the same as Australia. For lung cancer, there were also more deaths (120 per year, 20%) compared to Australia, but also 13% higher incidence.

In women, deaths from cancers of the ovary, stomach, uterus, and cervix, and from melanoma, showed lower numbers of excess deaths as these cancers account for fewer deaths in total, but the proportions of excess deaths ranged from 12% to 30%. For cancers of the ovary, and cervix, and for melanoma, incidence was substantially higher in New Zealand. There were fewer deaths but also fewer incident cases of pancreatic cancer in New Zealand; and there was lower incidence of non-Hodgkin lymphoma and brain cancer, although these differences were not statistically significant.

For men, excess numbers of deaths were seen in New Zealand for several cancers. Colorectal cancer showed the greatest numbers of excess deaths, 143 per year, 24% of the total. Stomach cancer deaths were 25% higher in New Zealand, and prostate cancer deaths 9% higher.

Figure 2. Deaths rates for all cancer from 1990, New Zealand and Australia, both age-standardised using the WHO world standard population



For lung cancer, the number of deaths were similar to Australia (2% lower), but incidence was 10% lower. Melanoma, brain cancers, and non-Hodgkin lymphoma also showed higher mortality, although the differences were not statistically significant. For all of these sites, there were no excesses of cancer incidence in New Zealand; indeed, the incidence rates of lung, melanoma and non-Hodgkin lymphoma

were significantly lower. As in women, both deaths and incidence from pancreatic cancer were lower than expected from Australian rates.

These differences between the countries relate to a period when total cancer death rates adjusted for age decreased substantially in each country (Figure 2). The decrease in New Zealand over 17 years from 1990 to 2007 was about 24% in men, and 20% in women; but throughout this period, the death rates in New Zealand were higher than in Australia, especially in women.

Discussion

This study shows that the higher rates of cancer deaths in New Zealand compared with Australia, shown for 1996 to 1997 by Skegg and McCredie,[1] have continued through to 2007 with only a slight change. In 1996-97, there were 17.5% more deaths in New Zealand in women, and 5.6% more in men.

Overall for 2000 to 2007, there were 15.1% more deaths from cancer in women, and 4.7% more deaths in men in New Zealand than would have occurred if age and site-specific death rates had been equivalent to those in Australia. These differences have been reasonably constant over time from 2000 to 2007, not showing the increased mortality differential predicted in the earlier study, but also not showing any decrease. The analysis used adjusts for age differences between the two countries and over time, and also adjusts for the mix of cancer sites.

Over this time, and since around 1990, total cancer death rates adjusted for age have decreased substantially in each country, as shown in Figure 2, so there has been a substantial improvement. In men, cancer mortality rates in New Zealand are similar to those in Australia about 3 years earlier, but in women this time difference is more than 10 years.

The greater number of deaths in women in New Zealand in 2000-07 was produced by higher numbers of deaths from each of the three main causes of cancer death: breast, lung, and colorectal cancer. However, the excesses for ovarian, stomach, endometrial, and cervical cancer and from melanoma, while numerically smaller, ranged from 18 to 30%. For men, the greatest excess was for colorectal cancer, with substantial excesses for prostate and stomach cancer and for melanoma.

The methods of registration and coding of cancers by the cancer registries are essentially the same in the two countries. It would take a major study to assess whether there are systematic differences in clinical diagnostic procedures, the information that is sent to the registries, or in coding and death certification practices.

Any differences in procedures may be more likely to apply to incidence rather than to mortality data, as accurate and complete recording of incidence is more complex. Also, variations in screening and diagnostic procedures can have major effects. Here, the incidence trends were dominated by the rapid variations in prostate cancer; in New Zealand, the age-standardised incidence rate (WHO world standard) per 100,000 fell from133 in 2000 to 92 in 2007 (a 9% fall), while in Australia it increased from 95 to 135 (a 10% increase) in the same period. Such rapid variations are likely to be due to changes in the use of prostate specific antigen (PSA) testing.[4] No substantial changes in prostate cancer mortality were seen in either country over those years.

Thus, as in the earlier study, these differences were in mortality rather than in incidence. Total cancer incidence for women in these years was only slightly higher in New Zealand than in Australia, and for men, whether including or excluding prostate cancer, it was slightly lower. Thus the substantial excesses in cancer mortality in New Zealand are not explicable by differences in incidence, implying that for the factors which influence the case-fatality of cancer, New Zealand is at a disadvantage. These factors include the process of diagnosis of cancer and as a result the extent of cancer (the stage distribution) at the time of treatment, the treatment given and the efficiency of treatment services, including equity across the population, and the provision of appropriate treatment at the appropriate time. Information on the distribution by stage at diagnosis would be very valuable, but complete and comparable data is not available for the two countries, except on selected series of patients.

Australia has good cancer survival by world standards. In worldwide comparisons of cancer survival, Australia, along with the U.S., Canada and Sweden is usually ranked near the top, above for example the UK.[5,6] New Zealand has not participated in these major studies of international cancer outcomes, although there is good data on survival within New Zealand,[7] and it will participate in future studies. We are currently doing a comparison of cancer survival rates in Australia and New Zealand.

In 2000ï 07, the largest differences, both in absolute excess numbers of deaths and in the proportional excess, were in colorectal cancer, where New Zealand mortality was 35% higher than in Australia for women, and 24% higher for men.

Both New Zealand and Australia have high incidence rates by world standards, likely linked to dietary factors.[8] Australiaôs approach to this disease has emphasised adherence to national management and treatment guidelines, and early detection, including population based screening.[9,10]

The Australian national bowel cancer screening programme began in 2006, too recently to influence mortality; however, that was preceded by a pilot program started in 2001, and there was considerable awareness and interest in the earlier diagnosis of bowel cancer in advance of that. In New Zealand, a pilot program in Waitemata started in 2011, with results expected in 2015;[11] so this development is considerably later than that in many other countries.

Deaths from breast cancer in New Zealand were 19% higher than those in Australia. Both countries have population-based screening programs, starting in Australia in 1991 and in New Zealand in 1998, both offering two yearly mammographic screening. It has been estimated in the New Zealand programme, deaths were reduced by as much as 40% in women participating in the screening.[12]

The utilisation of clinical practice guidelines in Australia in 1985 led to increased use of adjuvant radiotherapy, chemotherapy and oral therapy, and the decrease in mortality in Australia has been linked to improvements in adjuvant therapy.[13] Treatment trends in New Zealand may have been similar, but less information is available.

Lung cancer deaths in women were 20% higher in New Zealand compared to Australia. Deaths in men were about the same as in Australia; however lung cancer incidence in men was 10% lower in New Zealand, so this suggests that clinical survival rates are lower in both sexes.

Successful inter-sectoral collaboration on smoking policy is a major reason for the success of preventive actions for lung cancer;[14] both countries have made legislative changes about packaging, advertising, and the sale of tobacco products. The lung cancer mortality rates fell since the 1990s for Australian men and women and New Zealand men, but did not decrease as much in New Zealand women. Smoking is particularly common amongst Maori women in New Zealand.[15]

The one cancer site showing lower mortality in New Zealand than in Australia in both men and women was pancreatic cancer, where both mortality and incidence were lower than in Australia. Early diagnosis of pancreatic cancer is not linked to better survival outcomes, and it has a poor prognosis and is difficult to treat.[16]

Cancer outcomes are influenced not only by specific treatments, but by the effectiveness and efficiency of cancer control programs. The data here suggest that for cancers which can be greatly benefited by early diagnosis and optimum treatment, such as breast and colorectal cancer, mortality outcomes in New Zealand lag behind those of Australia. For pancreatic cancer, for which neither early diagnosis nor recent advances in treatment have much effect, New Zealand does as well or better.

Progress in a systematic approach to cancer control in New Zealand began during the 1990s with an initiative focused on cervical, skin, breast and lung cancers.[17] The New Zealand Cancer Control Trust was set up in 2001, representing non-government agencies in partnership with the Ministry of Health, and produced a New Zealand Cancer Control Strategy in 2003, which set goals for improving access to high quality and timely cancer services.[17i 19]

The focus has been on identifying disparities in cancer outcomes between subgroups of the population. Relatively little effort has been taken to identify international trends or to compare outcomes between New Zealand and other countries. Recent developments have been based on the Regional Cancer Networks set up in 2006ï 08,[20] and include the development of service standards for 10 major cancers[21] ñto ensure patients receive timely, good quality care along the cancer management pathway.ò

The Australian approach has been to build a systematic framework to promote a comprehensive national approach to cancer control, involving all phases along the continuum of care. Cancer was recognised as a National Health Priority Area in 1986, and in 1987 the National Cancer Control Initiative was jointly established by the federal government and voluntary sectors, to identify areas of greatest potential benefit in cancer control and to support their implementation.[22]

A comprehensive nationwide consultation was held to assess priority control measures, and subsequently these identified measures were assessed by cost effectiveness analyses [23]. In addition, much emphasis was given for the development of evidence-based clinical practice guidelines, primarily supported by the Australian Cancer Network, a voluntary association primarily of cancer specialists. These guidelines cover all the most common cancers such as lung, colorectal and prostate cancer. New Zealand experts have frequently been involved in the development of these guidelines, and some are explicitly Australian and New Zealand joint productions, such as the melanoma guidelines.[24]

In Australia, there have been a large number of systematic surveys of the management of cancer, mostly carried out by the voluntary sector Cancer Councils. These have

included national surveys of population-based series of breast and colorectal cancers, and State based surveys of many other cancers.[25] These have identified variations in management, and gaps between optimal management as defined in evidence-based guidelines and actual practice. There has also been systematic assessment of the actual use of radiotherapy compared to the utilisation expected on the basis of guidelines.[26] In New Zealand, there have been some studies of cancer management, and some large databases, for example for breast cancer,[27,28] but these have been less extensive than the Australian approaches.

The demonstration of international cancer outcome differences has been particularly influential in the UK. The demonstration that survival rates for cancer patients in the UK were lower than those in many other European countries led to a major reorganisation of cancer services in the UK, with clear national leadership [29]. The emphases in these reforms have been on the efficient provision of evidence-based cancer management, with attention being given to reducing barriers and time delays in access to care, effective primary care services, and appropriate referral services including the concentration of services to provide appropriate levels of expertise and workloads.[30]

More general economic issues are also relevant. Comparisons of cancer survival within 19 countries in Europe show strong positive correlations with total per capita expenditure on health and with the number of computer tomography scanners per million population, interpreted as a measure of investment in advanced health care.[31] Figures from the OECD show a modest difference in total health spending per capita between Australia and New Zealand ($US 3800 and $3042 respectively, 2010), but, if the figures are accurate, a very large difference in scanners per million population (Australia 50.6, New Zealand 15.3, 2012).[32]

Internationally there has been much emphasis on making comparisons between countries in regard to cancer outcomes, particularly survival [33]. The demonstration of survival differences has been followed by óhigh-definition studiesô in which patient management surveys in areas of contrasting cancer outcomes are used to identify reasons for the outcome differences.[34,35]

The results in the current study suggest that a systematic comparison of the diagnosis and clinical management of common cancers such as colorectal cancer between New Zealand and Australia could show important areas for improvement.

Competing interests: Nil.

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Acknowledgement: This work is based on the Honours BHSc dissertation of Ms Alafeishat. No external funding was used.

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

A comparison of cancer statistics in New Zealand and Australia: 1996–2007

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Abstract

Aim To compare the burden and outcomes of cancer in New Zealand with those in Australia.

Methods For the years 1996ï 1997 and 2006ï 2007, the incidence and mortality of cancer in New Zealand and Australia was compared to determine if differences between the two countries had changed over the decade under study. Summarised cancer data from New Zealand and Australia, age standardised to the 2002 World Health Organisationôs standard population, were used to make the comparisons.

Results For the 11 year timeframe of this study, total rates of cancer incidence reduced in New Zealand and increased in Australia. The incidence of cancer in New Zealand, relative to Australia, changed from an excess of +10.3 to a deficit of -27.5 per 100,000 people. When considering the excess in terms of gender, the annual excess of cancer registrations for New Zealand females fell from +19.9 to +0.9 per 100,000, and male cancer registration fell from an excess of +3.7 to a deficit of -58.0 per 100,000, due almost entirely to a surge in prostate cancer registration in Australia.

Over the same 11-year timeframe, cancer-specific mortality rates decreased in both countries, but there was no change in the difference between New Zealand and Australian rates, which remained 10% higher in New Zealand. Similar to findings on 1996/7 data, the main cancer sites responsible for the overall excess mortality in 2006/7 were colorectal cancer in both sexes, and lung and breast cancer in females.

Conclusion The persisting different cancer mortality rates between the two countries is likely to have been partly due to lifestyle and ethnic differences in the populations, and partly due to New Zealanders presenting with more advanced cancers and having less easy access to some treatments. Until we know the relative contributions of these factors, it will be difficult for New Zealand to plan interventions in the future which have a good chance of lifting our cancer survival rates to those of our closest neighbour. The collection of clinical stage on all new cancer registrations would provide the base information required.

In 2002, Skegg and McCredie showed that cancer incidence and mortality differed between Australia and New Zealand.[1] They pooled 1996 and 1997 cancer statistics for each country, and age standardised them to Australian 1991 population statistics before making inter-country comparisons. Pooled 2-year data were used as the small population of New Zealand made annual estimates of some cancer types unstable.

New Zealand was shown to have an excess cancer incidence (+25.5) and mortality (+21.7) per 100,000 people when data were compared to Australia.

In 2011, the Organisation for Economic Co-operation and Development (OECD) placed New Zealand 16th in ranking for lowness of cancer mortality, one position behind the United Kingdom and two places ahead of the OECD average.[2]

Australia was ranked nine places ahead of New Zealand, suggesting that there were still mortality differences between New Zealand and Australia.

We performed this study to see whether or not the New Zealand cancer incidence and mortality excesses over Australia detected by Skegg and McCredie were still present a decade later.

Methods

Incident cases of cancer and information relating to cancer mortality were retrieved from the New Zealand Cancer Registry (NZCR)[3,5] and the Australian Institute of Health and Welfare (AIHW).[6] Data were obtained and pooled for the 2-year periods 1996+1997 and 2006+2007, and were then age standardised to the 2002 World Health Organization (WHO) standard population.[7] The use of an international age standard set approximately halfway through the study period was to allow meaningful comparisons of data within each country and between countries across the whole 11-year time period of the study.

Leukaemia and myeloid cancers were the only major cancers not included in the study as differences in coding in New Zealand and Australia precluded meaningful comparisons between the two countries. As these cancers were responsible for only 5.5% of cancer registrations and 4.9% of deaths in New Zealand in 2006, their exclusion will have minimal impact on comparisons made on data for all other cancers. Minor non-melanoma skin cancers (ICD 173) were not included in comparisons, as data on these cancers are not routinely collected by cancer registries in either country on account of their extreme frequency and very low lethality.

In addition to the main analysis involving all cancer incidence and mortality events in the two countries, sub-set analyses were performed to identify differences according to site and gender of cancer.

The 95% confidence interval (CI) is provided in the tables for differences in incidence and mortality between similar cancer groups in New Zealand and Australia.

Results

In New Zealand, during the period 1996 to 2007, 178,231 cancers were diagnosed and 78,005 deaths recorded on the New Zealand Cancer Registry.

In 2007, cancer was the leading contributor to mortality in New Zealand, accounting for 4144 (53%) and 3950 (47%) deaths in men and women respectively.[8]

Table 1 shows cancer events in Australia and New Zealand in 1996/7 and 2006/7, with figures for the total population and for each gender.

Table 2 shows the differences in cancer incidence between New Zealand and Australia according to gender and anatomical site of cancer.

Table 3 shows the differences in cancer mortality between New Zealand and Australia according to gender and anatomical site of cancer.

All figures in the tables are age standardised to the 2002 WHO population, and are per 100,000 people at risk.

Table 1. Cancer events in Australia and New Zealand 1996/7 and 2006/7

Cancer events (rate per 100,000, WHO age standardised)	1996/7 (95% CI)			2006/7 (95% CI)			Within country change 1996–2007			Between country change 1996-2007 (95% CI)		
Australia	**f**	**m**	**total**	**f**	**m**	**total**	**f**	**m**	**total**	**f**	**m**	**total**
Incidence	299.4	405.0	344.1	307.0	435.3	365.8	+7.7	+30.3	+21.7			
Mortality	112.6	179.1	140.8	97.7	151.4	121.3	-14.9	-27.8	-19.5			
New Zealand												
Incidence	319.2	408.7	354.3	307.9	377.3	338.2	-11.3	-31.4	-16.1			
Mortality	136.4	184.5	155.7	117.9	155.4	133.7	-18.5	-29.1	-21.9			
NZ excess/deficit compared to Australia												
Incidence	+19.9 (±12.3)	+3.7 (±10.3)	+10.3 (±8.7)	+0.9 (±1.5)	-58.0 (±33.7)	-27.5 (±16.0)				+19.3 (±5.7)	-61.6 (±16.3)	-37.8 (±8.5)
Mortality	+23.8 (±14.5)	+5.4 (±5.2)	+14.9 (±9.6)	+20.3 (±11.7)	+4.0 (±4.0)	+12.4 (±7.2)				-3.6 (±10.7)	-1.3 (±11.2)	-2.4 (±9.6)

Table 2. Differences in cancer incidence in New Zealand relative to Australia by gender and anatomical site of malignancy

		Difference 1996/97	Difference 2006/07	Decade change in difference (95% CI)	Within country incidence change over decade	
					New Zealand	Australia
Total cancer	All	+10.3 (±8.7)	-27.5 (±16.5)	-37.8 (±8.5)	-16.1	+21.7
	Female	+19.9 (±13.2)	+0.9 (±1.6)	-19.3 (±5.7)	-11.3	+7.7
	Male	+3.7 (±10.3)	-58.0 (±34)	-61.6 (±16.3)	-31.4	+30.2
	All	+0.4	-1.6	-2.1 (±1.2)	-3.6	-1.5
	Female	+4.8 (±2.8)	+8.5 (±6.8)	+3.7 (±3.9)	+6.7	+3.0
Lung	Male	-2.8	-6.1	-3.3 (±3.5)	-9.6	-6.4
Colorectal	All	+5.7	+1.3	-4.4 (±2.2)	-5.0	-0.6
	Female	+8.0	+2.6	-5.4 (±2.5)	-5.0	+0.5
	Male	+3.1	+0.5	-3.1 (±2.1)	-5.2	-2.2
	All	-1.6	+0.5	+2.1 (±1.2)	+2.1	-0.1
Melanoma	Female	+1.6	+3.5	+2.0 (±1.6)	+0.4	-1.6
	Male	-5.0	-2.9	+2.1 (±2.1)	+3.7	+1.6
Breast	Female	-0.2	+1.6	+1.8 (±1.4)	+3.7	+2.0
Cervix	-	+2.3	+0.6	-1.7 (±1.4)	-3.9	-2.3
Uterus	-	+0.7	+0.4	-0.3 (±0.7)	+1.2	+1.5
Prostate	-	+20.4 (±12.6)	-30.9 (±19.1)	-51.3 (±12.0)	-12.7	+38.6

Table 3. Differences in cancer mortality in New Zealand relative to Australia by gender and anatomical site of malignancy

		Difference 1996/97	Difference 2006/07	Decade change in difference (95% CI)	Within country mortality change over decade	
					New Zealand	Australia
Total cancer	**All**	+14.9 (±9.8)	+12.4 (±7.2)	-2.4 (±9.6)	-21.9	-19.5
	Female	+28.8 (±9.6)	+20.3 (±5.8)	-3.6 (±10.7)	-18.5	-14.9
	Male	+5.4 (±5.4)	+4.0 (±4.1)	-1.3 (±11.2)	-29.1	-27.8
Lung	**All**	+2.8 (±1.8)	+1.1 (±0.7)	-1.6 (±1.9)	-5.3	-3.7
	Female	+5.1 (±3.0)	+4.2 (±2.5)	-0.9 (±1.8)	+0.1	+1.0
	Male	+0.5	-2.3	-2.7 (±4.4)	-12.5	-9.8
Colorectal	**All**	+4.9 (±3.0)	+7.2 (±4.2)	+2.3 (±3.1)	-4.2	-6.5
	Female	+4.8	+7.6	+2.8	-2.6	-5.4
	Male	+5.2	+6.7	+1.6	-6.3	-7.9
		+0.5	+0.6	+0.1	+0.4	+0.3
Melanoma	**All**					
	Female	+0.9	+0.7	-0.2	-0.1	+0.2
	Male	+0.1	+0.6	+0.4	+0.9	+0.5
Breast	**Female**	+6.1 (±3.8)	+4.2 (±2.4)	-2.0 (±3.0)	-6.6	-4.7
Cervix	**-**	+1.1 (±0.7)	+0.5 (±0.4)	-0.5 (±0.6)	-1.5	-1.0
Uterus	-	+0.5 (±0.3)	+1.1 (±0.6)	+0.6 (±0.4)	+0.6	+0.1
Prostate	-	+1.5 (±1.4)	+0.7 (±0.6)	-0.8 (±1.9)	-5.0	-4.2

Cancer incidence—From 1996/7 to 2006/7, the cancer incidence rate in New Zealand fell both for females (-1.3) and males (-31.4), resulting in an overall reduction of -16.1 per 100,000 for the total population. In Australia over the same period the incidence of cancer increased in both females (+7.7) and males (+30.2), resulting in an overall increase of +21.7 per 100,000 for the total population.

For 1996/7, the excess cancer incidence rate in New Zealand over Australia was +10.3 per 100,000 people when data were age standardized to the 2002 WHO population (less than the +25.5 reported by Skegg and McCredie who age standardized their data to the 1991 Australian population). By 2006/7, this excess had converted into a deficit of -27.5, a shift of -37.8 over the decade.

When cancer site is considered (Table 2), the main reason for the higher incidence of cancer in Australia in 2006/7, when compared to 1996/7, relates to a relative increase in prostate cancer diagnosis in Australia. Over the study period, prostate cancer was diagnosed less in New Zealand (-12.7) and more in Australia (+38.6), leading to a relative change of -51.3 per 100,000 men for New Zealand compared to Australia. Over the study period, there was a reduction in the incidence of lung cancer in men in both countries, with a change of -9.6 in New Zealand males and -6.4 in Australian males. However, the incidence of lung cancer in women increased more in New Zealand (+6.7) than in Australia (+3.0), so the excess in the incidence of lung cancer in New Zealand women seen in 1996/7 (+4.8) was larger in 2006/7 (+8.5).

The reduction in colorectal cancer incidence was larger in New Zealand (-5.0) than in Australia (-0.6). For other sites of cancer, changes in incidence over the study period were similar within each country and were generally small. For example, in New Zealand and Australia the increased incidence of breast cancer was +3.7 and +2.0 respectively, and the reduced incidence of cervical cancer -3.9 and -2.3 respectively.

Cancer mortality—In New Zealand, from 1996/7 to 2006/7, cancer mortality rates fell for both females (-18.5) and males (-29.1) leading to a reduction of -21.9 for the whole population. In Australia over the same period cancer mortality rates also fell for both females (-14.9) and males (-27.8) leading to a reduction of -19.5 per 100,000 for the whole population, a fall comparable to that seen in New Zealand.

For 1996/7, the New Zealand excess cancer mortality over Australia was +14.9 per 100,000 when data were age standardised to the WHO population (less than the +21.7 reported by Skegg and McCredie who age standardized their data to the 1991 Australian population). By 2006/7, the excess had reduced slightly to +12.4 per 100,000 people.

When cancer site is considered (Table 3), there was no single site responsible for the excess cancer mortality seen in New Zealand. In 2006/7, sites where there were large New Zealand excesses in mortality were colorectal cancer (+7.2), female lung cancer (+4.2) and breast cancer (+4.2), sites that also had sizable excesses in 1996/7. The 2006/7 excess mortality for prostate cancer was small (+0.7), much as it was in 1996/7 (+1.5).

Because the cancer sites with large mortality excesses are ones that occur commonly, each individual site excess has an impact on the mortality excess for the whole population. In 2006/7, the New Zealand population had a hazard ratio (HR) for cancer

death of 1.10 (12.4 +121.3/ 121.3) compared to the Australian population. The HR in 1996/7 was also 1.10 (14.9 +140.8/ 140.8).

Discussion

There is no reason to believe that the differences we have demonstrated in cancer incidence and mortality between New Zealand and Australia were due to differences in registration completeness and death notification between the two countries. In both countries notification of new cancer cases was compulsory in both countries for the whole of the study period.

In New Zealand, the Cancer Registry Act 1993 came into effect in July 1994, and in 1994 the collection of cancer data by Australian state registries was standardized so data could be pooled to provide national statistics. Differences demonstrated in this study are therefore likely to be real ones when the confidence interval indicates they are statistically significant.

From a public health point of view, it is disappointing that over the study period there was no change in the difference between New Zealand and Australian cancer mortality rates, which overall remained 10% higher in New Zealand. In 2006/7, the main cancer sites responsible for the different mortality rates were colorectal cancer (both sexes), and lung and breast cancer in females, the same sites that were mainly responsible for the difference in 1996/7.

Over the study period, the downward trend in the excess diagnosis of colorectal cancer in New Zealand compared to Australia (+5.7 to +1.3 per 100,000 people) appears encouraging at first glance. However, the worsening New Zealand excess mortality rate for this type of malignancy (+4.9 to +7.2 per 100,000 people) suggests that the reduction in excess incidence was due to New Zealand diagnostic rates for the early-stage more curable colorectal cancers falling behind those of Australia. A possible explanation for this is that New Zealand had no active screening programme in place during the study period, whereas in Australia the benefits of screening for colorectal cancer had a high public profile throughout the study period and systematic screening was initiated in some states in 2006.[10]

Over the study period, there was no downward trend in the incidence of lung cancer in New Zealand women, and in 2006/07 the New Zealand excess over Australia for this group remained high at +8.5 per 100,000 women. The main reason for the excess will be that tobacco smoking was more prevalent in New Zealand women during the study period.

In 2008, the World Health Organization reported that the incidence of smoking amongst males was only slightly higher in New Zealand when compared to Australia (29.7% versus 27.7%), but that the difference was larger for female smokers (27.5% versus 21.8%).[9]

Over the study period, the improvement in excess lung cancer mortality for males (-2.7) was not matched by a similar improvement for females (-0.9), which suggests that smoking practices in New Zealand women changed little from 1996-2007. It is also possible that New Zealand females are worse than their male counterparts in presenting late with symptoms of their lung cancer.[11]

The excess breast cancer mortality in New Zealand compared to Australia declined by only a small amount over the study period (+6.1 to +4.2 per 100,000 women). As screening programmes were in place in both countries during the whole study period, the persisting mortality excess could be due to differences in the target population for screening and/or differences in available (funded) treatments for the disease.

As an example of treatment differences, the anti-cancer drug trastuzumab (Herceptin) was available in Australia for most of the study period but was only funded in New Zealand from 2008, which post-dates the period covered by this study. Also, substantial delays to the start of radiation treatment were common in New Zealand during the study period, and radiation treatment in Australia was arranged for significant numbers of New Zealand women with breast cancer whose wait time in New Zealand would otherwise have exceeded twelve weeks.

Over the study period, New Zealand developed a large deficit in prostate cancer diagnosis relative to Australia. This is likely to have been the result of PSA testing of asymptomatic men being practiced more widely in Australia than New Zealand. Recently, the New Zealand Prostate Cancer Taskforce recommended that all New Zealand men aged 50ï 70 years should be given written information on PSA testing by their general practitioners,[12] and this could lead to PSA testing practices in the two countries becoming more aligned in the future.

Differences in prostate cancer mortality between the two countries remained small over the study period, and the New Zealand excess at the beginning and end was of marginal statistical significance. Mortality rates at the end of the study period could not have been affected by the different diagnostic rates that developed during the study period. The ERSPC screening trial[13] showed that it took nine years for a survival advantage of PSA testing to become evident, and the G¸ teborg screening trial[14] showed that the survival advantage was still increasing at 14 years.

The generally long natural history of prostate cancer after PSA diagnosis is confirmed by a study of men diagnosed in the PSA era which showed that prostate cancer mortality increased three fold after 15 years of follow-up.[15]

Conclusion

The persisting different cancer mortality rates between the two countries are likely to have been partly due to lifestyle and ethnic differences in the populations, and partly due to New Zealanders presenting with more advanced cancers and having less easy access to some treatments. Until we know the relative contributions of these factors, it will be difficult for New Zealand to plan interventions in the future which have a good chance of lifting our survival rates to those of our closest neighbour. The collection of clinical stage on all new cancer registrations would provide the base information required.

Competing interests: Nil.

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Journal of the New Zealand Medical Association

Screening for pulmonary arterial hypertension in patients with scleroderma—a New Zealand perspective

Sanjib K Ghosh, Michael M Corkill, Hamish H Hart, Kristine P Ng

Abstract

Background Pulmonary arterial hypertension (PAH) in scleroderma (SSc) patients is a devastating complication with high mortality if untreated. Early recognition and specific treatment of PAH may improve outcome. Regular interval screening for PAH is generally recommended in scleroderma patients especially with the availability of emerging new therapies. The aim of this study is to determine the self-reported screening and treatment practices for SSc-PAH amongst rheumatologists in New Zealand (NZ).

Methods An anonymous online questionnaire survey was emailed to all rheumatologists in New Zealand.

Results Responses were received from 65% (39/60) of rheumatologists. The majority of patients had limited SSc (lcSSc) (57%) versus diffuse SSc (dcSSc) (34%). Twelve percent of patients had PAH. Eighty-two percent of rheumatologists screened for PAH in all SSc patients regardless of symptoms. The most commonly used screening modalities were pulmonary function tests (PFT) (97%) followed by clinical examination (95%) and echocardiogram (TTE) (92%). The majority of rheumatologists performed screening tests on a yearly basis (80% used PFT and 64% used TTE). A right heart catheter was used to confirm PAH in 70% of patients. Sixty-four percent of rheumatologists extend screening interval time if their patients were clinically stable. The most common PAH-specific therapy used was sildenafil (57%) followed by bosentan (19%). Sixty-four percent of rheumatologists supported a national PAH-SSc screening guideline.

Conclusion This study has shown a wide variability of how NZ rheumatologists screen for PAH in scleroderma patients. The development of a PAH-SSc guideline for screening and diagnosis may help standardise treatment practices in NZ.

Scleroderma is an autoimmune connective tissue disease of unknown aetiology with an estimated prevalence of 19ï 75 cases per 100,000.[1] It has the potential to involve multiple systems. Scleroderma is generally classified into two subgroups; diffuse (dcSSc) and limited scleroderma (lcSSc) depending on the extent of skin involvement and serological pattern. Pulmonary complications are relatively common with reports of up to 26% of patients developing pulmonary arterial hypertension (PAH) and up to 40% developing interstitial lung disease (ILD).[2ï 5]

Pulmonary arterial hypertension (PAH) is a devastating complication and leading cause of death in up to 30% of patients in 2 years if left untreated.[6ï 8] The prognosis is worse in patients with ILD associated PAH compared to PAH alone with a five-fold increase in risk of death.[8] PAH is often under-recognised because of non-specific symptoms.[9]

With recent advances in PAH-SSc therapy and evidence that early recognition and treatment reduces disease progression and improves long-term outcomes, it is important to diagnose PAH-SSc early in the course of the disease.[10ï 13] Routine screening for PAH, even in asymptomatic patients, has assumed greater importance. Yearly echocardiogram and pulmonary function tests (PFT) with diffusion capacity for carbon monoxide (DLCO) are generally recommended.[14]

The management of PAH in scleroderma patients often requires multidisciplinary coordination between rheumatologists, respiratory physicians and cardiologists.[15] Early intervention with PAH-specific treatment is essential to improve outcome.[15,16] The prevalence of screening and treatment practices amongst rheumatologists in New Zealand (NZ) for PAH in scleroderma patients is unknown.

This study aims to determine self-reported screening, diagnosis and treatment practices for SSc-PAH amongst rheumatologists in NZ.

Methods

We surveyed 60 rheumatologists in NZ through an online questionnaire using website http://www.surveymonkey.com

The email contacts were obtained from the New Zealand Rheumatology Association membership. We emailed a questionnaire comprising of 10 questions (Figure 1). The results were analysed.

Figure 1. Questionnaire

1. Please estimate the number of SSc patients in your rheumatology practice?
2. How many of your SSc patients have interstitial lung disease? Please state number.
3. How many of your SSc patients have Pulmonary Arterial Hypertension (PAH)? Please state number.
4. Of the patients diagnosed with PAH, how many have had a right heart catheter study? Please state number.
5. Do you routinely screen your SSc patients for PAH? If "No" please omit question 6 & 7
6. If you do screen, please outline which of the following methods do you use and how often? Please select as many as you like from the following: history & examination, pulmonary function tests (PFT) with diffusion capacity for carbonmonoxide (DLCO), transthoracic echocardiogram (TTE), electrocardiogram (ECG) and six minute walk test (6MWT).
7. Do you extend the screening time if your patients are stable clinically? If yes. Please indicate how your screening time is changed.
8. How many of your SSc-PAH patients are treated with a pulmonary vasodilator, anticoagulant or both? Please state number.
9. For those SSc-PAH patients who are on a pulmonary vasodilator please specify number of patients on endothelin receptor antagonists (e.g. Bosentan), sildenafil and prostacyclin analogues (Iloprost).
10. Do you feel that a national NZ PAH screening guideline will be helpful? Please specify reason.

Results

Thirty-nine rheumatologists (65%) responded to our survey. The majority of patients had lcSSc (n=301/525, 57%) followed by dcSSc (n=176/525, 34%) and 9% (n=48/525) had overlap of connective tissue diseases. The mean number of patients for an individual rheumatologist was 8 (range 0ï 20) for lcSSc and 5 (range 0ï 20) for

dcSSc. Interstitial lung disease was present in 20% (n=106/525) of patients and 12% (n=63/525) had PAH.

Table 1 displays the self-reported screening patterns for PAH in SSc patients depending on symptoms. Eighty-two percent of rheumatologists screened all SSc patients (limited and diffuse) regardless of symptoms. Five percent of rheumatologists screened only symptomatic patients in both groups. Another 5% of rheumatologists screened all dcSSc and symptomatic lcSSc patients only. Two rheumatologists do not screen lcSSc patients at all.

Table 2 demonstrates a wide variability of different investigations and frequency of tests used for PAH screening among rheumatologists. Ninety-seven percent (n=38/39) of rheumatologists preferred PFT with DLCO as a screening test. Of these, 82% of rheumatologists performed this test yearly.

Ninety-five percent (n=37/39) of rheumatologists conducted clinical examination for screening and of these, 59% performed this 6-monthly. Ninety-two percent (n=36/39) of rheumatologists used echocardiogram as a screening method and of these, 64% ordered this test yearly.

Twelve rheumatologists requested yearly electrocardiogram (ECG). Twenty-six percent (n=10/39) of rheumatologists performed the 6-minute walk test (6MWT) for screening, of which 18% ordered this yearly.

Table 1. Screening patterns of rheumatologists in diffuse and limited scleroderma patients depending on symptoms

Diffuse scleroderma	Limited scleroderma	Number of rheumatologists (%) n=39
All patients regardless of symptoms	All patients regardless of symptoms	32 (82)
Symptomatic patients only	Symptomatic patients only	2 (5)
Symptomatic patients only	All patients regardless of symptoms	1 (2)
All patients regardless of symptoms	Symptomatic patients only	2 (5)
All patients regardless of symptoms	No patients screened	1 (3)
Symptomatic patients only	No patients screened	1 (3)

Table 2. Methods and frequency of screening

Method of screening	Less than 6 monthly n (%)	6 monthly n (%)	Yearly n (%)	2 yearly n (%)	Range in months
PFT with DLCO*		2/38 (5)	31/38 (82)	5/38 (13)	6 to 60
History & Examination	7/37(19)	23/37(62)	7/37(19)		2 to 12
Echocardiogram			25/36 (69)	11/36 (31)	12 to 24
ECG**			11/12 (92)	1/12 (8)	12 to 24
6MWT***	1/10 (10)		7/10 (70)	2/10 (20)	4 to 24

*PFT with DLCO=Pulmonary function tests with diffusion capacity for carbon monoxide, **ECG=Electrocardiogram, ***6MWT=6-minute walk test

Table 3. Extension of screening interval time

Method of screening	Number of rheumatologists extending screening time n=39 (%)	6 to 12 months n=39 (%)	12 to 24 months n=39 (%)	Range (months)
Hx & Ex*	23 (59)	12 (30)		3 to 12
PFT with DLCO**	14 (36)		7 (18)	6 to 36
ECG§	5 (13)		2 (5)	12 to 72
6MWT§§	4 (10)		2 (5)	6 to 36
Echocardiogram	14 (36)		8 (21)	12 to 36

* Hx & Ex=History and examination, ** PFT with DLCO=Pulmonary function test with diffusion capacity for carbon monoxide, § ECG=Electrocardiogram, §§6MWT=6-minute walk test

Table 4. Medications used for pulmonary arterial hypertension in scleroderma patients

Medications	Number of patients (%)
Sildenafil	36 (57)
Bosentan	12 (19)
Prostacyclin analogue	8 (13)
Anticoagulant	24 (38)
Both (pulmonary vasodilators and anticoagulation)	24 (38)
None	3 (5)

Sixty-four percent of rheumatologists extended their screening interval time provided their patients were clinically stable. Fifty nine percent of rheumatologists extended their clinical examination interval time and 30% extended the interval period from 6 months to 1 year.

Thirty-six percent of rheumatologists extended screening interval time for PFT with DLCO and half of them changed this interval from yearly to 2-yearly. Thirty-six percent of rheumatologists extended screening interval time for echocardiogram and 21% changed the screening interval from yearly to 2-yearly.

Thirteen percent and 10% rheumatologists extended their screening interval time for ECG and 6MWT respectively and almost half of them changed the interval time from yearly to 2-yearly (Table 3). Seventy percent of SSc-PAH patients were diagnosed by a right heart catheter (RHC) study.

In terms of PAH-specific treatment, 57% patients received sildenafil. Twenty four patients received anticoagulation in combination with other PAH-specific treatment. Only 12 patients and 8 patients received treatment with bosentan and prostacyclin analogue respectively. Five percent of patients did not receive any form of treatment (Table 4).

Discussion

Our descriptive study has shown that there is a wide variation of screening and treatment practices of PAH in SSc patients among NZ rheumatologists. Scleroderma patients have an increased risk of cardiopulmonary and other systemic organ involvement.

The mortality from scleroderma renal crisis has improved significantly since the advent of angiotensin converting enzyme[17] but mortality from lung involvement remains very high if untreated.[18] A number of studies recommend regular interval screening for early detection and introduction of specific treatments to improve outcome.[10-13]

Our study revealed that the majority of rheumatologists (82%) screen all their SSc patients for PAH regardless of symptoms using different methods, with no consistent interval between screenings. The number of rheumatologists screening for PAH in SSc patients has increased significantly since a prior audit conducted from 1999 to 2004[19]

The previous audit aimed to look at screening practice of SSc related lung disease in a single rheumatology tertiary centre. In that audit, only 50% of patients had echocardiographs performed. None of the patients in that audit were on specific PAH medications as the drugs were not funded at that time.

Our study has shown that NZ rheumatologists screening practices for PAH have changed with 92% of rheumatologists using echocardiograph as a screening test for PAH.

The screening interval time varied from months to years. The most frequently used screening tests are PFT with DLCO (97%) followed by clinical examination (95%) and echocardiogram (92%). Most rheumatologists are screening six monthly to yearly depending on which investigation is used.

Our study found that lcSSc patients are not screened as routinely compared to dcSSc patients. In the previous audit, limited SSc patients were also not screened as rigorously as diffuse SSc patients for any scleroderma related lung disease (67% versus 90%)[19]. The lcSSc patients require regular screening as the prevalence of PAH increases with prolonged duration of disease in lcSSc patients.

This study has highlighted that a small minority of rheumatologists (5%) do not screen for PAH in lcSSc at all.

Current screening recommendations for PAH in SSc patients are largely based on consensus. A variety of screening recommendations have been published by organisations such as the American College of Cardiology Foundation/American Heart Association and the European Society of Cardiology/European Respiratory Society (ESC/ERS).[20,21]

Most international screening guidelines for PAH in SSc patients recommend yearly echocardiogram and PFT with DLCO [15]. A recent study observed a better prognosis in patients identified in an active screening programme compared to those in routine clinical practice.[22]

This study has shown that a significant number of rheumatologists are not following international guidelines in screening for PAH-SSc. There may be a variety of factors for this including lack of resource, in particular constraint to investigations such as echocardiogram, lack of specialised PAH cardiothoracic units in smaller NZ regions and no clear guidelines on screening in the NZ setting.

The first evidenced based PAH detection study was published recently using a two-step internally validated algorithm. The algorithm uses simple clinical data and non-invasive tests to determine the likelihood of PAH and cut-off points for decision to refer a patient to echocardiograph and subsequent RHC.[23] The DETECT algorithm may optimise the use of already limited resources by identifying the appropriate high risk patient for echocardiograph and RHC. The application of classic screening criteria such as the Wilson and Jungner criteria will be more applicable in the future as more evidence based data is available to guide the principles of screening[24].

The 6-minute walk test (6MWT) is an outcome measure that is used in most PAH studies. Some groups suggest that it is a highly reproducible test in SSc-ILD patients, used as a primary outcome measure in the management of PAH in scleroderma patients [25]. However, this test has never been validated in the SSc population and the interpretation of this test can be difficult in SSc patients. SSc patients have other factors such as arthritis and a low fitness level that will affect the reproducibility of this test.

Furthermore, it is difficult to obtain a good measure of oxygen saturations with a finger probe in SSc patients due to poor peripheral circulation. Wilsher and colleagues have suggested using forehead probe to measure oxygen saturations[26]. In our study only 26% of rheumatologists are performing the 6MWT and 18% of rheumatologists are requesting this test annually.

The 6MWT is often not available in peripheral regions of NZ where there is no specialised respiratory unit. We suspect this is one of the main reasons as to why this test is under-utilised.

The RHC study is the gold standard for diagnosing PAH. Only 70% patients are reported as being diagnosed with PAH by RHC study. This may be due to the difficult access of this specialised test. Another possible explanation is that patients with mild to moderate PAH with good exercise tolerance have yet to proceed to confirmatory RHC study.

Our study has found no consistency with regards to increasing screening interval time amongst rheumatologists if their patients are clinically stable. Two thirds of

rheumatologists extended the screening interval time for their patients if they are clinically well. However there was a huge variability of increasing screening interval time depending on which investigation is used ï range from few months to years.

A number of studies have clearly demonstrated that early intervention with specific treatment for SSc-PAH patients improves outcome.[15,16] The EULAR recommendations for treatment of systemic sclerosis emphasise the benefit of PAH-specific treatment [27]. PAH-specific treatment is currently funded by PHARMAC (via an expert panel) and available in NZ.

Sildenafil is a phosphodiestarase 5 inhibitor, is funded for PAH patients with New York Heart Association (NYHA) class III and IV symptoms. Bosentan, an endothelial receptor antagonist (ERA) is available for those who have not responded to sildenafil. In our study, the majority of patients (57%) received sildenafil and followed by bosentan (19% of patients) which reflects the local funding criteria guidelines.

Our questionnaire had a good response rate from NZ rheumatologists compared to a similar study in Australia[9]. We feel that this study has highlighted a wide variability of how SSc patients are screened for PAH amongst NZ rheumatologists and provides a snap shot of current PAH-SSc practices in NZ.

One of the limitations of our study is that this is a small study and it was not designed to identify barriers to performing screening and diagnostic tests. It is also likely (although not addressed in the study questionnaire) that stricter criteria for PAH-SSc patients to qualify for treatment (compared to many overseas countries) may influence NZ Rheumatologistsô screening practices.

Resource constraint is a major issue revealed from the comments made by rheumatologists. The majority (64%) of rheumatologists believe a national screening guideline for PAH-SSc would be valuable and in line with international recommendations. PAH-specific medications are currently only funded for symptomatic patients in NZ. Therefore, screening asymptomatic patients would not be cost effective.

The application of a validated breathless assessment questionnaire may be useful to identify the patients that will need further tests to screen for PAH.[28] One of the difficulties in setting up a national screening guideline is the variability of access for tests such as PFT and echocardiograph in NZ regions. Some regions of NZ have no or very limited access to these investigations (personal communication).

In summary our study recognises the wide variability of screening and treatment practices for PAH-SSc patients amongst NZ rheumatologists. There may be a role for national screening guideline to standardise our approach in the management of PAH-SSc patients.

Competing interests: Nil.

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Acknowledgement: The authors thank all rheumatologists who responded to our survey.

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Journal of the New Zealand Medical Association

Effect of age, gender, ethnicity, socioeconomic status and region on dispensing of CVD secondary prevention medication in New Zealand: The Atlas of Health Care Variation CVD cohort (VIEW-1)

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Abstract

Background Triple therapy with anti-platelet/anti-coagulant, blood pressure (BP)-lowering, and statin medications improves outcomes in atherosclerotic cardiovascular disease (CVD). However, in practice there is often a substantial evidenceï practice gap, with sub-optimal initiation and longer-term adherence. Our aim was to enumerate a contemporary national cohort of people with significant CVD and report the variation in CVD secondary prevention dispensing by demographic variables.

Methods Using anonymised linkage of national data sets, we identified 86,256 individuals, alive and residing in New Zealand at the end of 2010, aged 30ï 79 years who were hospitalised for an atherosclerotic CVD event or procedure in the previous10 years. This cohort was linked to the national pharmaceutical dispensing dataset to assess dispensing of CVD prevention medications during the 2011 calendar year. Adequate dispensing was defined as being dispensed a drug in at least 3 of the 4 quarters of the year. Multivariate regression was used to identify independent predictors of adequate dispensing.

Results 59% were maintained on triple therapy, 77% on BP-lowering medication, 75% on anti-platelet/anti-coagulants and 70% on statins. From multivariate analysis, patients less than 50 years were about 20% less likely than older patients and women were 10% less likely than men to be maintained on triple therapy. Indian patients were about 10% more likely to be maintained on triple therapy than NZ European/Others. Those living in the Southern Cardiac Network region of New Zealand had slightly higher rates of triple therapy than National Cardiac Regions further north.

Conclusions The significant under-utilisation of safe and inexpensive secondary prevention medication, particularly in younger people and women, provides an opportunity to improve CVD outcomes in this easily identifiable high-risk population.

There is robust evidence to support the use of anti-platelet/anti-coagulant,[1,2] blood pressure (BP)-lowering,[3ï5] and statin[6ï8] medications to improve outcomes in patients with established atherosclerotic cardiovascular disease (CVD). International practice guidelines[9] recommend that, unless contraindicated, agents from each of these three classes be prescribed.

Adherence to ñtriple therapyò has the potential to reduce future CVD event rates by up to 80%.[10] However, in practice there is often a substantial evidenceï practice gap,

with sub-optimal initiation and longer-term maintenance of evidence-based therapy across the spectrum of CVD.[11ï 18] Better understanding the extent of this evidence-practice gap and its determinants is essential to optimise the benefits of secondary prevention.

Most prior studies of medication use in CVD cohorts have utilised prescribing data from secondary care cohorts or cross-sectional primary care cohorts. Data collection in such studies is typically resource intensive, follow-up time is short, and the data is difficult to update regularly. In New Zealand there is an opportunity to take a more comprehensive and cost-effective approach.

Every patient encounter with the public health system, which provides or subsidises the vast majority of primary and secondary health services, is linked to a unique national health identifier (NHI) number, which enables anonymised linkage of multiple electronically stored national health databases. Because virtually all subsidised pharmaceutical dispensing, all public hospitalisations, and all deaths are recorded in these linked datasets, it is possible to identify a comprehensive national cohort of people hospitalised with atherosclerotic CVD and regularly report on dispensing of secondary prevention medication.

The aims of this study are to describe the identification of a national atherosclerotic CVD cohort and to report the variation in maintenance of CVD secondary prevention medication by age, gender, ethnicity, socioeconomic status and geographical region. This is a collaborative programme of work involving researchers at the University of Auckland, analysts at the Northern District Health Board Support Agency (NDSA) and sponsored by the Health Quality & Safety Commission (HQSC).

Accompanying papers in this issue of the *NZMJ* (VIEW-2 & VIEW-3) describe the methodology used to display this data within the HQSC New Zealand Atlas of Healthcare Variation and the results of a national consultation process, respectively.

Methods

Generation of cohort—An encrypted version of the NHI number (a unique patient identifier allocated to every New Zealander) was used to anonymously link individual patient data from national mortality, hospitalisation and drug dispensing datasets. We identified 86,256 individuals, aged 30ï 79 years, alive and residing in New Zealand at the end of 2010, who had been hospitalised for an atherosclerotic CVD event or procedure in the prior 10 years. This cohort was then linked to the national pharmaceutical dispensing dataset to report dispensing of CVD prevention medication during the 2011 calendar year.

Our aim was to identify patients who were highly likely to have atherosclerotic CVD, for whom there is strong evidence for the benefits of long-term treatment with statins, BP-lowering and antiplatelet/anticoagulant agents. Using the International Classification of Diseases, version 10 (ICD-10) codes, we included those patients admitted to a public hospital with myocardial infarction, ischaemic stroke or coronary and peripheral procedures between 01/01/2001 and 31/12/2010(see Appendix 1 for full list of ICD-10 codes). Transient ischaemic attacks (TIAs), unstable angina, angina and haemorrhagic strokes were not included.

A comprehensive list of peripheral vascular procedures was identified and reviewed by a cardiovascular surgeon (ARK). Only those procedures likely to be performed as a consequence of atherosclerosis were included. The cohort was refined to include only those alive and living in New Zealand at the end of 2010. The national mortality and hospitalisations datasets were used to exclude those who had died prior to 2011. Those without a documented contact with the public health sector in 2009ï 2010 were also excluded.

A health contact was defined as: a public hospital admission (recorded in the National Minimum Dataset (NMDS)), a public hospital outpatient visit (in the National Non-Admitted Patients Collection

[NNPAC]), Pharmaceutical dispensing, a Community Laboratory Claim, a General Medical Subsidy Claim (for primary care services), or current enrolment with a Primary Health Organisation (PHO).Patients aged 30ï 79 years at the end of 2010 were included. We excluded 264 people with ñunmappableò Census area units and a further 223 who had duplicate NHIs.

These datasets were also used to obtain socio-demographic data (age, gender, ethnicity, deprivation score and Census Area Unit (CAU) of most recent address). CAUs are broadly equivalent in size and geographical coverage to health domicile codes available in routine health databases. Ethnicity was prioritised (in the following order: MŪori, Pacific, Indian, European/Other).

Socioeconomic status was assessed using the NZDep2006 score, an area-based measure from 1 (least deprived) to 10 (most deprived) that combines nine variables reflecting eight dimensions of relative deprivation.[19] As NZDep2006 was calculated for Census Meshblocks, which are considerably smaller (average population 87 residents) than CAUs (average population = 2000 residents), in these analyses we used the average NZDep2006 score for the CAU and combined the deciles, into quintiles.

Patients were geographically located according to the most recent CAU of residence and were assigned to one of 20District Health Boards (DHBs) and one of four national cardiac network regionsð Northern, Midlands, Central and Southern.

CVD medication maintenance—For this analysis CVD secondary prevention medication was grouped in three classes - anti-platelets/anticoagulants, statins and BP-lowering drugs. The medications included under each class are those licensed for use in New Zealand and are shown in Appendix 2. The cohort was linked to the national pharmaceutical dispensing dataset derived from subsidy claims from community pharmacies.

Virtually all CVD medications prescribed in New Zealand receive a government subsidy, so these data are likely to be complete. To be considered adequately maintained on a class of medication a patient needed to be dispensed at least one agent from that class in at least three of the four quarters of 2011. Triple therapy was defined as being on an anti-platelet/anticoagulant, a statin and a BP-lowering drug. Adequate maintenance on triple therapy required the patient to meet the definition of adherence for each of the three classes of drug in 2011.

Statistics—Log-binomial regression was used to identify predictors of medication maintenance in 2011 and calculate relative órisksô of being dispensed medication, adjusting for the following factors: age, gender, ethnic group, NZDep06 quintile and geographical region.

Results

Study cohort by age, gender, ethnicity and region (Table 1, Figure 1)—There were 86,256 patients aged 30ï 79 years with significant atherosclerotic CVD living in New Zealand at the end of 2010, of whom 67% were male. Overall, 13% were MŪori, 6% Pacific, 2% Indian and 79% European/Other patients. There were 15%, 16%, 21%, 25%, and 24% living in NZDep06 quintiles 1 to 5, respectively.

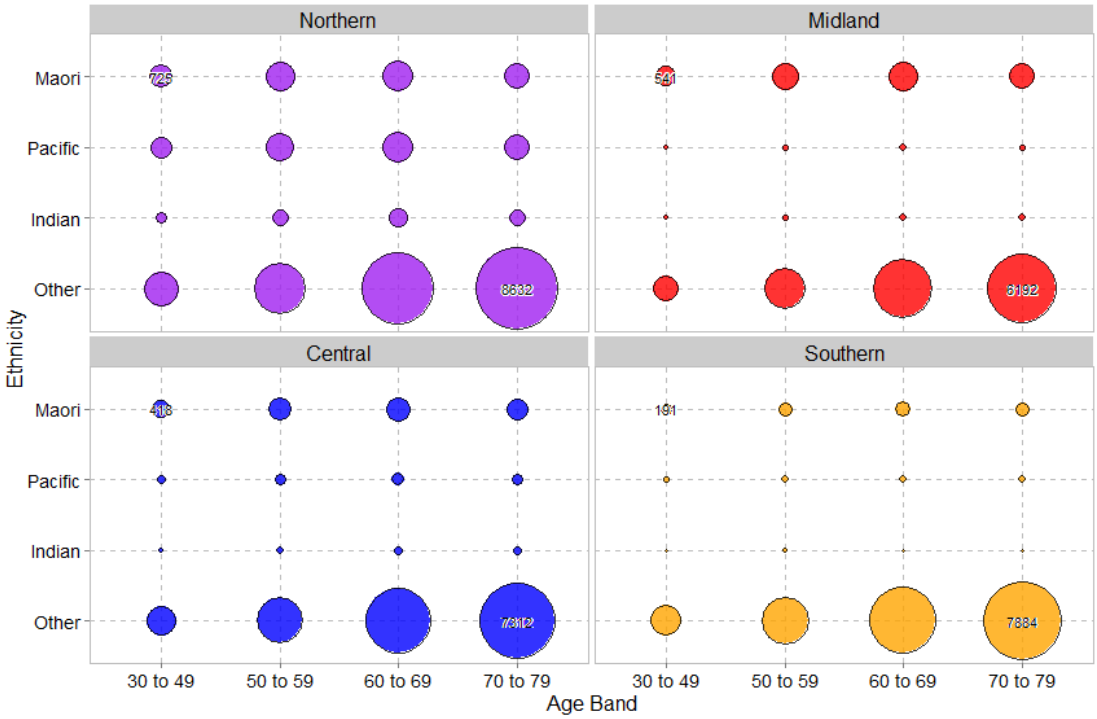
The age distribution was much flatter for MŪori, Pacific and Indian patients compared to Europeans (Figure 1) reflecting the underlying age distributions of these populations in New Zealand. There are also major regional ethnic differences with most Pacific and Indian patients living in the Northern region, and relatively few MŪori patients in the Southern region.

The proportions of patients classified as least deprived (NZDep Quintile 1) through to most deprived (NZDep Quintile 5) were 15%, 16%, 21%, 25%, and 24% respectively.

Table 1. number of CVD patients by age, gender, ethnicity and NZDep (1 to 5)

Ethnicity	Age band	1		2		3		4		5		#N/A	Grand total
		F	M	F	M	F	M	F	M	F	M	M	
Māori	30 to 49	27	63	52	75	97	175	193	284	418	490	1	1875
	50 to 59	56	102	78	157	168	253	301	455	641	853		3064
	60 to 69	74	110	115	171	193	289	385	447	736	910		3430
	70 to 79	62	93	81	125	154	195	321	327	597	634		2589
Māori total		**219**	**368**	**326**	**528**	**612**	**912**	**1200**	**1513**	**2392**	**2887**	**1**	**10958**
Pacific	30 to 49	8	26	13	26	32	70	49	119	165	290		798
	50 to 59	14	41	17	69	38	85	79	180	249	529		1301
	60 to 69	25	49	34	42	66	113	113	200	342	596		1580
	70 to 79	14	27	36	39	70	68	121	144	281	366		1166
Pacific total		**61**	**143**	**100**	**176**	**206**	**336**	**362**	**643**	**1037**	**1781**		**4845**
Indian	30 to 49	6	24	7	37	8	38	10	56	15	64		265
	50 to 59	20	70	11	67	21	96	19	115	24	109		552
	60 to 69	24	45	21	72	42	97	37	128	46	135		647
	70 to 79	21	44	35	66	36	59	52	80	50	86		529
Indian total		**71**	**183**	**74**	**242**	**107**	**290**	**118**	**379**	**135**	**394**		**1993**
European/Other	30 to 49	260	603	238	573	302	634	339	794	295	582		4620
	50 to 59	481	1591	513	1591	594	1771	718	1958	555	1440	1	11213
	60 to 69	972	3135	1088	3101	1396	3564	1564	3717	1253	2816	1	22607
	70 to 79	1528	3242	1873	3499	2458	4502	2967	4901	1934	3114	2	30020
Euro/Other total		**3241**	**8571**	**3712**	**8764**	**4750**	**10471**	**5588**	**11370**	**4037**	**7952**	**4**	**68460**
GRAND TOTAL		**3592**	**9265**	**4212**	**9710**	**5675**	**12009**	**7268**	**13905**	**7601**	**13014**	**5**	**86256**

Figure 1. Bubble plot illustrating the number of CVD patients by age, ethnicity and cardiac network region (size of bubbles proportional to number of patients)



Adequate CVD prevention medication maintenance by age and gender (Table 2, Figure 2)—Overall 77% of patients were adequately maintained on a BP-lowering medication, 70% on a statin, 75% on an antiplatelet/anticoagulant and 59% on triple therapy during 2011. For each medication class and for triple therapy, maintenance in younger age groups was lower.

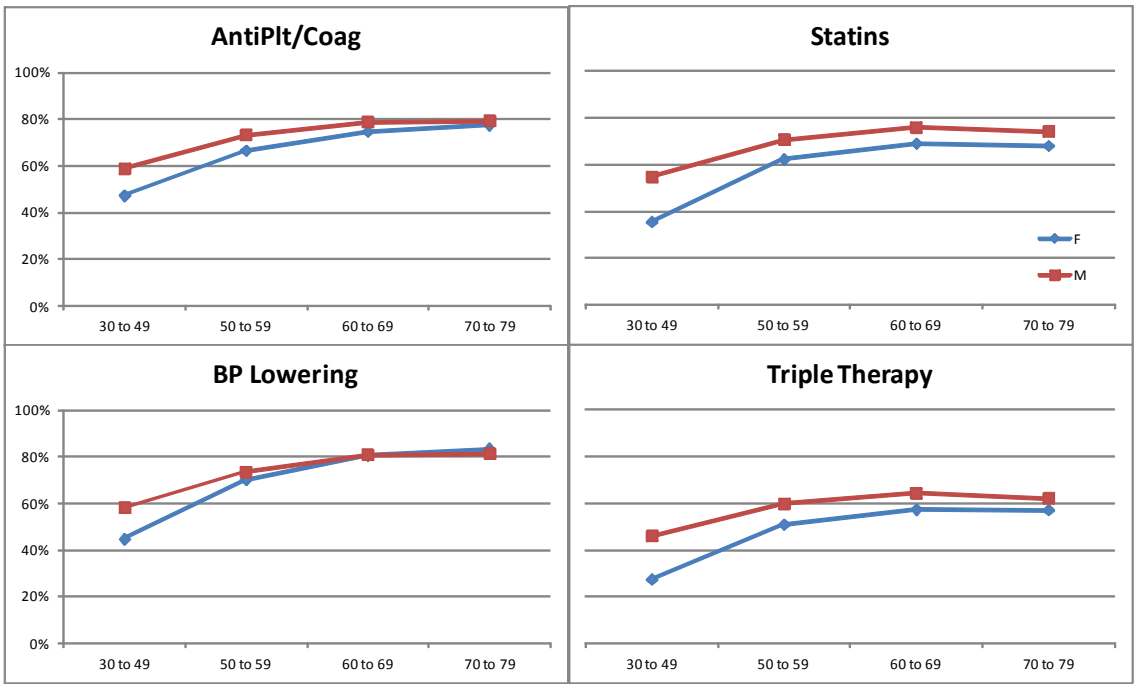
Triple therapy maintenance for women ranged from 27% for those <50 years to 57% for those >60 years, and for men from 46% in those <50 years to 64% in those 60ï 69 years. Overall maintenance on BP-lowering agents was similar for men and women, but statin use (64% vs 72%) and antiplatelet/anticoagulant use (72% vs 76%) was lower for women, with the difference being most marked in younger women. This resulted in a lower rate of triple therapy in women (54% vs 61%) and a nearly 20% absolute difference in those <50 years (27% vs 46%).

Table 2. Adequate maintenance on medications by age and gender

Gender	Age band (years)	Total	AntiPlt/Coag*	Statins	BP-lowering	All three
Female	30 to 49	2534	47%	35%	45%	27%
	50 to 59	4597	67%	62%	70%	51%
	60 to 69	8526	75%	69%	81%	57%
	70 to 79	12691	77%	68%	83%	57%
Female total		**28348**	**72%**	**64%**	**77%**	**54%**
Male	30 to 49	5024	59%	55%	58%	46%
	50 to 59	11533	73%	71%	74%	60%
	60 to 69	19738	79%	76%	81%	64%
	70 to 79	21613	79%	74%	81%	62%
Male total		**57908**	**76%**	**72%**	**78%**	**61%**
GRAND TOTAL		**86256**	**75%**	**70%**	**77%**	**59%**

* Antiplatelet/anticoagulant.

Figure 2. Adequate maintenance on medications by gender



Adequate CVD prevention medication maintenance by ethnicity and deprivation score (Figure 3 and Appendix 3; Table 3)—Compared with other ethnic groups, young Indian men, and to a lesser extent Pacific women were more likely to be maintained on triple therapy.

Younger MŪori, Pacific and European/Other patients had much lower maintenance rates than their older counterparts. Except for the small cohort of Pacific women <50

years, women of different ethnic groups had similar rates in each age band, but European/Other and Pacific men rates were slightly higher than MŪori men.

Adequate maintenance on triple therapy for men was similar across quintiles of deprivation for all age bands. The maintenance rate for women living in the most deprived areas (55%, Quintile 5) was slightly higher than for those in least deprived areas (50%, Quintile 1) and this difference was evident in each age band.

Figure 3. Adequate maintenance on triple therapy by ethnicity

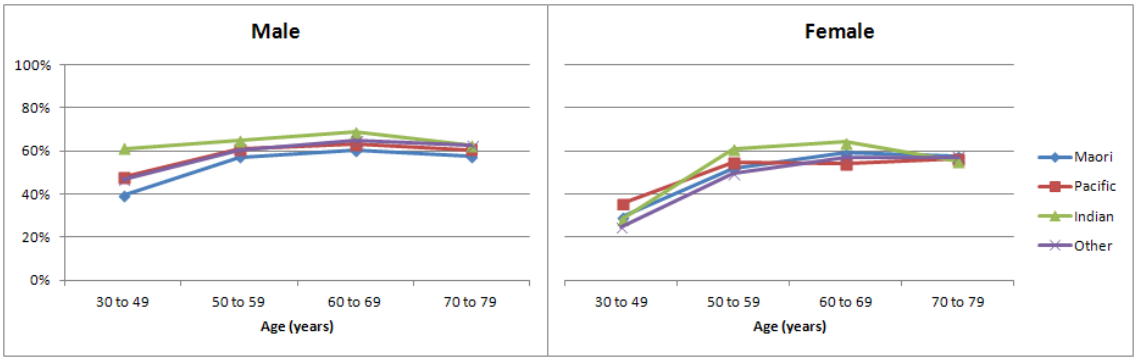


Table 3. Adequate maintenance on triple therapy by index of deprivation (NZDep2006)

Gender	Age band	NZDep2006 1	2	3	4	5	Grand total
Female	**30 to 49**	26%	23%	29%	26%	29%	27%
	50 to 59	45%	50%	47%	54%	54%	51%
	60 to 69	55%	55%	58%	57%	60%	57%
	70 to 79	53%	57%	58%	56%	59%	57%
Female total		50%	53%	54%	54%	55%	54%
Male	**30 to 49**	47%	48%	44%	46%	46%	46%
	50 to 59	59%	61%	58%	61%	60%	60%
	60 to 69	64%	64%	66%	65%	64%	64%
	70 to 79	62%	63%	62%	62%	61%	62%
Male total		61%	62%	61%	61%	60%	61%
GRAND TOTAL		**58%**	**59%**	**59%**	**59%**	**58%**	**59%**

Adequate maintenance by cardiac network region (Table 4)—There is a south to north gradient in maintenance of triple therapy with the highest rate (61%) in the Southern region and a slightly lower rate in the Northern region (57%) with intermediate rates in the centre of the country. This gradient is similar for each age band.

Table 4. Regional variation in adequate maintenance on triple therapy

Gender	Age band	Northern	Midland	Central	Southern	Grand total
Female	30 to 49	26%	28%	30%	27%	27%
	50 to 59	51%	49%	52%	53%	51%
	60 to 69	56%	58%	57%	60%	57%
	70 to 79	54%	58%	58%	59%	57%
Female total		**51%**	**54%**	**54%**	**56%**	**54%**
Male	30 to 49	45%	44%	49%	48%	46%
	50 to 59	59%	59%	60%	62%	60%
	60 to 69	62%	65%	65%	67%	64%
	70 to 79	61%	61%	62%	65%	62%
Male total		59%	61%	61%	64%	61%
GRAND TOTAL		57%	59%	59%	61%	59%

Table 5. Independent predictors of adequate maintenance on triple therapy

Variables	Multivariate adjusted RR
30ï 49y	0.78 (0.76ï 0.80)
50ï 59y	0.96 (0.95ï 0.98)
60ï 69y	Reference
70ï 79y	0.96 (0.95ï 0.97)
Male	Reference
Female	0.90 (0.88ï 0.91)
European/Other	Reference
Maori	0.97 (0.96ï 0.99)
Pacific	1.04 (1.01ï 1.06)
Indian	1.11 (1.08ï 1.15)
NZDep06 deciles 1ï 2	0.97 (0.96ï 0.99)
3ï 4	0.98 (0.97ï 1.00)
5ï 6	0.99 (0.97ï 1.00)
7ï 8	0.99 (0.97ï 1.00)
9ï 10	Reference
Northern Region	Reference
Central	1.02 (1.00ï 1.03)
Midland	1.02 (1.00ï 1.04)
Southern	1.06 (1.04ï 1.07)

Multivariate analysis—After adjustment, the strongest independent predictors of lower maintenance was age with age group 30ï 49 years (associated with 22% lower maintenance compared with 60ï 69 year olds), female sex (10% lower rate), and Indian ethnicity (11% higher maintenance than European/Other patients.

Pacific patients were slightly more, and MÜori patients slightly less, likely to be maintained on triple therapy than European/Other patients. Patients living in the least

deprived areas were slightly less likely, and those living in the Southern region slightly more likely, to be maintained on triple therapy throughout 2011.

Discussion

In this national cohort of New Zealanders aged 30 to 79 years in 2010, who had been hospitalised in the previous 10 years with myocardial infarction, ischaemic stroke or CVD procedure, 59% were adequately maintained on triple therapy during 2011. In individual medication classes, maintenance on BP-lowering (77%) and antiplatelet/anticoagulants (75%) was higher than for statins (70%). There were important variations in medication maintenance by age, gender and ethnicity.

Young people were significantly less likely than older patients to be maintained on all drug classes. Women of all age groups were less likely to be maintained on a statin than their male counterparts, and younger women were less likely to receive antiplatelet/anticoagulants or BP-lowering agents than younger men.

Compared to other ethnic groups Indian patients were most likely to be maintained on triple therapy, while there were minimal differences between the other ethnic groups and by deprivation.

People living in the Southern region of New Zealand had 6% higher rates of triple therapy than in the Northern region with rates in the Central and Midlands regions intermediate between the Northern and Southern regions.

Comparisons with other studies—Most previous studies of secondary prevention medication utilisation have reported on specific categories of CVD such as acute coronary syndrome, coronary heart disease or stroke.[14,15] A major advantage of using national hospitalisation data is that we were able to identify a comprehensive population-based cohort of patients who had been hospitalised for the wider group of CVD diagnoses for which statins, BP-lowering agents and antiplatelet/anticoagulants are recommended. Many prior studies report medication adherence relating to continued prescribing or dispensing after initially starting a medication. Our approach, like some other studies[12,13] was to use all patients with presumed atherosclerotic CVD as the denominator for dispensing, since in the absence of contraindications or intolerance, triple therapy is recommended for all these patients.

Because of this, our dispensing rates capture both those who were never initiated on a medication, as well as those who did not have medications consistently dispensed. There are a wide range of methodologies for reporting medication adherence in the literature.[20] These include use of patient self-report, prescribing, dispensing records and pill counting.

Adherence is typically reported by some measure of persistence. For single agents this is typically a Medication Possession Ratio (MPR) calculated from the number of days supplied divided by the number of days in the study period. This method is technically challenging when there are multiple drugs in a class and when adherence to multiple agents is reported simultaneously. In New Zealand most patients receive 3-month prescriptions, so in this study we chose a simpler approach by defining adequate maintenance as being dispensed medications in at least 3 of the 4 quarters in the year.

Lower dispensing of statins compared with other drug classes—A lower rate of statin dispensing compared with blood pressure-lowering drugs or antiplatelet/anticoagulants was observed in this study. Prior large observational studies have reported approximately 10% intolerance due to the statin-specific side effects of myalgia and myopathy,[21,22] and real or perceived intolerance to statins may explain these findings.

Further research is needed to better understand this observation given the substantial benefits of statins in this high risk population. We are unaware of any good data regarding intolerance/contraindication rates to the overall classes of blood pressure lowering drugs or antiplatelet/anticoagulants in the CVD population as a whole.

Whilst some patients will be intolerant to individual blood pressure and antiplatelet/anticoagulant drugs, because of the greater range of agents to choose from within each class, the intolerance rates for the class as a whole are likely to be lower than for statins. In addition we specifically excluded patients with haemorrhagic stroke who have an absolute contraindication to antiplatelet/anticoagulants.

Age differences in medication dispensing—Patients <50 years were approximately 20% less likely than their older counterparts to be dispensed secondary prevention medication.This is of particular concern as high risk young patients have the most to gain from secondary prevention. Our findings are consistent with those from a recent Spanish registry study which reported lower rates of secondary prevention prescribing in young patients after an acute coronary syndrome.[14] However it is in apparent conflict with the conclusions from recent meta-analyses of adherence to either statin[23] or triple therapy agents,[15] which reported no effect of age on adherence in secondary prevention cohorts.

One possible explanation for this difference is that, as discussed above, most adherence studies take as their denominator population only those patients who have been started on a particular medication; those who never start a medication or are intolerant are not included. We therefore need to understand whether the young are more likely to be intolerant to medication side effects or less likely to be started on medication and the reasons for this. However, even when initiated on medication, younger patients may be less likely to maintain long-term therapy.

High risk behaviours are important contributors to premature CVD and are probably also associated with poor medication maintenance. In a recent local study young patients with an acute cardiac event were more likely to smoke, be obese and have hyperlipidaemia than their older counterparts[24] and other studies have shown that patients with healthy lifestyle behaviours are also more likely to take their tablets.[20]

The dispensing rate was highest in the 60-69 year age band and fell slightly for those in the 70-79 year old group. Other studies which, unlike ours, have included patients aged >80 years have reported markedly lower adherence in that age group.[13,18,25] With increasing age there is an increasing proportion of people with multiple comorbidities for whom secondary prevention may be less beneficial. Nevertheless secondary prevention is clearly beneficial for a substantial proportion of elderly patients and a challenge for future work will be identifying those without limiting comorbidities, to enable reporting of dispensing rates in those elderly who will continue to benefit.

Gender differences in medication dispensing—Dispensing of statins for women was 8% lower than men in absolute terms (64% vs 72%, respectively). Whilst this gap was seen in all age bands, for women aged <50years the difference was 20%. Young women were also less likely to be dispensed antiplatelet/anticoagulants or BP-lowering medication but no gender difference was observed in older patients. This finding contrasts with two meta-analyses of statin and combination CVD secondary prevention therapy which found no effect of gender on adherence.[15, 23] However, a more recent meta-analysis, specifically focussed on ethnicity and gender, found that women were 10% less likely to be adherent to statin therapy.[26] Gender differences in medication tolerance might contribute to this difference. However, observational studies of statin intolerance[21, 22] have not found a significant overall difference between men and women, although in one study there was a significantly higher rate of side effects for women in the subgroup without diabetes.[22]

Other data shows very elderly women are at higher risk of side effects,[27] which may explain some of the gender difference in statin use in the 70-79y age band, but not for younger women for whom the gap is greatest. Other established non drug-intolerance reasons for lower dispensing in women include a misconception that women are at lower risk and womenôs caregiver responsibilities as a barrier to looking after their own health.[26]

In addition there will be a small number of CVD cases included in our cohort, that are not clearly related to atherosclerosis and so triple therapy would not have been recommended. Some of these, such as spontaneous coronary dissection and apical ballooning syndrome occur predominantly in women, and may contribute to the lower observed dispensing rates, particularly in the younger age bands where atherosclerotic CVD events are less frequent.

Ethnicity and socioeconomic differences in medication dispensing—Indians were approximately 10% more likely to be dispensed triple therapy than other ethnic groups once age, gender, deprivation and region were adjusted for. After multivariate adjustment dispensing for MŪori was only 3% lower than for European/Others.

In contrast, a recent international meta-analysis[26] reported that non-white patients were 10% less likely to be adherent to statins compared with white patients. In a recent whole of New Zealand data-linkage study of maintenance of statin therapy up to 3 years after hospitalisation with an acute coronary syndrome, Indians and NZ European/Others had very similar statin maintenance.[28]

This discrepancy raises the possibility that the Indian cohort differs from other ethnic groups perhaps in the distribution of CVD diagnostic type and/or date of diagnosis. This requires further investigation.

Patients living in more deprived areas of New Zealand are just as likely, if not more likely, to be consistently dispensed secondary prevention therapy compared with those from less deprived areas. This differs from studies in other countries where adherence to CVD prevention medication has been shown to be lower using various indicators of lower socioeconomic status.[23,29,30]

In this study we used a well validated area-based measure of relative deprivation so it is unlikely that the absence of a socioeconomic gradient is due to the measure used. It is more likely that the consistency is due to our comprehensive public health system

spanning primary and secondary care that is supported by well-disseminated national CVD prevention guidelines.[31]

At the time of this study there was a $3 co-payment with each prescription item. Since then the co-payment has been increased to $5. It will be important to repeat the current analysis a year after introduction of this increased fee to determine the extent to which the co-payment impacts on socioeconomic disparities.

Limitations—This study will not have identified all patients with atherosclerotic CVD living in New Zealand in 2010. In particular those with their last CVD hospitalisation over 10 years ago, those diagnosed only in primary care or overseas without being hospitalised in New Zealand, and those who only had a private hospital procedure are not included.

Because our aim was to identify a cohort highly likely to have atherosclerotic CVD for whom triple therapy is recommended we deliberately excluded events/procedures where there may be higher levels of diagnostic inaccuracy (e.g. unstable angina and TIAs) or non-atherosclerotic aetiology. Despite including only ñhardò atherosclerotic CVD ICD-10 coded events there will still be a small percentage of patients with a non-atherosclerotic aetiology, such as myocardial infarction due to spontaneous coronary dissection or coronary embolus, in whom triple therapy may not be clearly indicated.

In addition, dispensing is an indirect measure of whether patients actually take their medication, although the definition of adequate treatment maintenance used (dispensing in at least 3 of the 4 quarters of the year) makes it likely patients are taking their medication. A prior study has reported high concordance between medication dispensing and pill counts and concluded that the rate at which patients are dispensed medication is usually consistent with the rate they consume them.[32] However, it is possible there are systematic differences between demographic subgroups in the relationship between dispensing and drug consumption. For this study we did not use drug dosage information.

Other studies have shown that patients are often prescribed doses lower than those used in the clinical trials.[33] The national pharmaceutical dataset used for our study does include drug dose information and further work is needed to understand population subgroup differences in optimal dosing of secondary prevention medications.

Another limitation of this study is that it doesnôt take account of the reduced number of possible prescriptions that could be dispensed to people who died or who spent long periods in hospital during 2011. Adequate medication maintenance was defined as being dispensed in 3 of the 4 quarters of 2011 so patients who died before the third quarter could not meet the criteria.

Clinical implications—Although CVD mortality rates have continued to decrease in New Zealand, the under-utilisation of inexpensive secondary prevention medication documented in this study provides an opportunity to further improve outcomes. The biggest opportunity for improvement is in younger adults and in women. In New Zealand non-MŪori non-Pacific people have a 2 to 3 fold lower age-specific coronary artery disease mortality rate compared to MŪori and Pacific peoples.[34]

Higher utilisation of evidence-based therapy including medication and revascularisation in these high-risk population groups could help close this gap. In our multivariate analysis MŪori were only slightly less likely to receive triple therapy, but the disproportionate numbers of young MŪori and Pacific patients with CVD means that targeting these younger people for preventative treatment should be advocated.

What dispensing rates should we aim for?—The percentage of patients in this cohort who have medication contraindications or intolerance cannot be precisely defined. The best indicator of potential medication maintenance for chronic coronary disease probably comes from the Courage trial where, after 5 years, 93% were maintained on statins and 94% on aspirin.[35] Several other factors impact on optimal targets.

Our cohort is based on hospitalised CVD diagnoses and although only events that were highly likely to be due to atherosclerosis were included it is inevitable that there will be a small number of patients with non-atherosclerotic CVD who may not have an indication for triple therapy. In observational registries a statin intolerance rate of around 10% has been reported.

A further data source available to us is the best achieved rates in various New Zealand population subgroups. For example 60ï 69 year old men on the West Coast achieved 79% statin maintenance and 71% triple therapy maintenance. Taking these factors into account the National Clinical Cardiac Network has proposed an aspirational target of 85% for statins and 70% for triple therapy.

What can we do?— Making the gaps in evidence-based practice visible to patients, health providers and health administrators is a critical first step. The data has been made available to the public and medical community in an interactive mapping tool as part of the HQSCs New Zealand Atlas of Health Care Variation (http://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/cardiovascular-disease/).

One recommendation from a national consultation process (see accompanying paper in this issue of the Journal by Grey et al.) was that dispensing data should be provided to PHOs broken down by individual practices, to be used as part of local quality improvement processes. The first set of practice-based reports was circulated in 2013 in the Northern region by the NDSA. Regular updates of the data are also needed to support change. The NDSA is updating this data annually and the Northern region is tracking change in key demographic subgroups on behalf of the Northern and National Cardiac Networks.

A multifactorial approach to improve maintenance of triple therapy dispensing which encompasses patients, medical staff, systems of care, quality improvement processes and health information technology is required. Electronic practice registers and population management tools may identify high risk CVD patients but need to be used consistently to ensure those with the most to gain are receiving treatment and that treatment is equitable and appropriately proportioned according to CVD risk.

Many practices have population management tools which allow them to identify which high risk patients have been prescribed medication, but there is more that can be done with electronic systems such as sending reminders when prescriptions are due to be refilled. Simplification of dosing regimens through the use of polypills could

also improve dispensing rates. More intensive intervention maybe useful in some population groups including targeted coverage that is well supported by social systems, addressing financial barriers, health literacy programmes, and navigation support.[36]

Competing interests: Nil.

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Funding: This research was co-funded by the Health Quality and Safety Commission, the Auckland Medical Research Foundation (AMRF #1110022) and the Health Research Council (11/800). Dr Wells was supported in part by the Stevenson Foundation. Corina Grey is funded by a Health Research Council Clinical Research Training Fellowship (11/814).Dr Riddell is funded by the Health Research Council (11/800).

Acknowledgements: The authors wish to thank Richard Hamblin and Catherine Gerard at the Health Quality &Safety Commission for their support in developing this atlas. We also thank the Northern District Support Agency (NDSA) and the Northern Regional Cardiac Network for their role in initiating CVD indicator development for New Zealand.

In addition we thank the team at Analytical Services, National Health Board, for the provision of the routine data used to develop this cohort, Alan Kerr who reviewed the atherosclerotic procedure codes and Chris Wiltshire at Enigma Solutions Ltd for encrypting National Health Identifiers.

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Appendix 1. ICD-10 codes used to identify patients with ischaemic CVD

CLINICAL CODE	DESCRIPTION
3317800	Repair of ruptured aneurysm in neck
3318100	Repair ruptured intra-abdominal aneurysm
3350000	Carotid endarterectomy
3350600	Innominate endarterectomy
3350601	Subclavian endarterectomy
3350900	Aorta endarterectomy
3351200	Aorto-iliac endarterectomy
3351500	Aorto-femoral endarterectomy
3351501	Ilio-femoral endarterectomy, bilateral
3351800	Iliac endarterectomy
3352100	Ilio-femoral endarterectomy, unilateral
3352400	Renal endarterectomy, unilateral
3352700	Renal endarterectomy, bilateral
3353000	Coeliac endarterectomy
3353001	Superior mesenteric endarterectomy
3353300	Coeliac &supr mesenteric endarterectomy
3353600	Inferior mesenteric endarterectomy
3353900	Endarterectomy of extremities
3354200	Extended endarterectomy deep femoral art
3354800	Patch graft of artery using vein
3354801	Patch graft art usg synthetic material
3354802	Patch graft of vein using vein
3354803	Patch graft vein usg synthetic material
3355100	Procurement vein fm limb f patch graft
3355400	Endarterectomy w art byps prep f anstms
3270000	Carotid bypass using vein
3270001	Carotid-carotid bypass using vein
3270002	Carotid-subclavian bypass using vein
3270003	Carotid-vertebral bypass using vein
3270004	Aorto-subclavian-carotid bypass usg vein
3270005	Carotid bypass using synthetic material
3270006	Carotid-carotid bypass usgsynthcmatrl
3270007	Carotid-vertebral bypsusgsynthcmatrl
3270008	Carotid-subclavian bypass synthcmatrl
3270009	Aorto-carotid bypsusgsynthc material
3270010	Aorto-carot-brachial bypass synthcmatrl
3270011	Aorto-subclavn-carot bypass synthcmatrl
3270300	Resection carotid artery w re-anstms
3270800	Aorto-femoral bypass usgsynthc material
3270801	Aorto-femoro-femoral bypass synthcmatrl
3270802	Aorto-iliac bypass using synthetic matrl
3270803	Aorto-ilio-femoral bypsusgsynthcmatrl
3271200	Ilio-femoral bypass using vein
3271201	Iliofemoral bypass usgsynthc material
3271500	Subclavian-femoral bypsusgsynthcmatrl
3271501	Subclavian-bifemoral bypass synthcmatrl
3271502	Axillo-femoral bypass usgsynthcmatrl
3271503	Axillo-bifemoralbypsusgsynthcmatrl

3271800	Ilio-femoral crossover bypass
3271801	Femoro-femoral crossover bypass
3273000	Mesenteric bypass usg vein single vessel
3273001	Mesenteric bypssynthcmatrl, sglvesl
3273300	Mesenteric bypass usg vein mult vessels
3273301	Mesenteric bypssynthcmatrl, multvesl
3273600	Other proc on inferior mesenteric artery
3273900	Femoral art bypass usg vein above knee
3274200	Femoral art bypass usg vein below knee
3274500	Femor art bypsven, tibl/&peroneal art
3274800	Femoral art bypsusgven w in 5cm ankle
3275100	Fermoral art bypssynthcmatrlabv knee
3275101	Fermoral art bypssynthcmatrlblw knee
3275102	Femor art synthcbypstibl&/peronl art
3275103	Femor art synthcbyps w in 5cm ankle
3275400	Femoro-femoral bypass usg composite gft
3275401	Femoro-popliteal bypsusg composite gft
3275402	Femor to tibl/peronl art bypscompstgft
3275700	Femoral artery sequential bypsusg vein
3275701	Femoral art sequential bypssynthcmatrl
3276300	Other arterial bypass using vein
3276301	Other arterial bypsgftusgsynthcmatrl
3276302	Subclavian vertebral bypass using vein
3276303	Subclavian axillary bypass using vein
3276305	Aortocoeliac bypass using vein
3276306	Aortofemoropopliteal bypass using vein
3276307	Ilioiliac bypass using vein
3276308	Poplitealtibial bypass using vein
3276309	Aortosubclavian bypass usgsynthcmatrl
3276310	Subclaviansubclavianbyps, synthcmatrl
3276311	Subclavianvertebral bypass synthcmatrl
3276312	Subclavianaxillary bypass, synthcmatrl
3276313	Axilloaxillary bypass usgsynthcmatrl
3276314	Axillobrachial bypass usgsynthcmatrl
3276316	Aortocoeliac bypass usg synthetic matrl
3276317	Aortofemoropoplitealbypssynthcmatrl
3276318	Ilioiliac bypass using synthetic matrl
3276319	Poplitealtiblbypsusgsynthc material
3305000	Replace popliteal aneurysm using vein
3305500	Replace popliteal anrysmusgsynthcgft
3307500	Repair of aneurysm in neck
3308000	Repair of intra-abdominal aneurysm
3310000	Replace carotid artery aneurysm w graft
3311200	Replace suprarenal AAA with graft
3311500	Replace infrarenal AAA with tube graft
3311800	Replace infrarnl AAA bifurgft iliac art
3312100	Replace infrarnl AAA bifurgftfemor art
3312400	Replace iliac art aneurysm w graft, unil
3312700	Replace iliac art aneurysm w graft, bil
3313000	Exc& rep visc art aneurysm, diranstms
3315100	Replace ruptured suprarenal AAA w graft
3315400	Replace ruptdinfrarenal AAA w tube gft

3315700	Replace ruptdinfrarnl AAA w iliac graft
3316000	Replace ruptdinfrarnl AAA w femor graft
3316300	Replace ruptd iliac art aneurysm w graft
3530306	Perc transluminal balloon angioplasty
3530307	Open transluminal balloon angioplasty
3530400	PTCA, 1 coronary artery
3530401	Open TBA of 1 coronary artery
3530500	PTCA, multiple coronary arteries
3530501	Open TBA mult coronary arteries
3530906	PTA perc w stenting, single stent
3530907	PTA perc w stenting, multiple stents
3530908	Open TBA w stenting, single stent
3530909	Open TBA w stenting, multiple stents
3531000	Perc ins trnslml stent, sglcoron artery
3531001	Perc ins multtrnslmlstntsglcoron art
3531002	Perc ins >=2 trnslmlstntcoron arteries
3531003	Open ins trnslml stent single coron art
3531004	Opn ins multtrnslmlstntsglcoron art
3531005	Opn ins multtrnslmlstntcoron arteries
3531200	Perc peripheral artery atherectomy
3531201	Open peripheral artery atherectomy
3531500	Perc peripheral laser angioplasty
3531501	Open peripheral laser angioplasty
3863700	Reop recon occluded coronary artery
3845619	Othintrathorproc arteries heart wo CPB
3849700	Coron art byps using 1 saph vein graft
3849701	Coron art byps using 2 saph vein grafts
3849702	Coron art byps using 3 saph vein grafts
3849703	Coron art bypsusg>=4 saph vein grafts
3849704	Coron art bypsusg 1 other venous graft
3849705	Coron art bypsusg 2 other venous grafts
3849706	Coron art bypsusg 3 other venous grafts
3849707	Coron art bypsusg>=4 oth venous grafts
3850000	Coronary artery bypass, using 1 LIMA gft
3850001	Coronary artery bypass, using 1 RIMA gft
3850002	Coron artery bypass usg 1 radial art gft
3850003	Coron art bypsusg 1 epigastric art gft
3850004	Coron art bypsusg 1 other arterial gft
3850300	Coronary artery bypass, using 2 LIMA gft
3850301	Coronary artery bypass, using 2 RIMA gft
3850302	Coron artery bypass usg 2 radial art gft
3850303	Coron art bypsusg 2 epigastric art gft
3850304	Coron art bypsusg>=2 oth arterial gft
3850500	Open coronary endarterectomy
3850700	Left ventricular aneurysmectomy
3850800	L ventricular aneurysmectomy w ptchgft
3850900	Repair of ventricular septal rupture
9020100	Coron art bypsusg 1 other matrlgft NEC
9020101	Coron art bypsusg 2 other matrlgft NEC
9020102	Coron art bypsusg 3 other matrlgft NEC
9020103	Coron art bypsusg>=4 other matrlgft
9022900	Other endarterectomy

9023000	Embolectomy/thrombectomy of other artery
I210	Acute transmural MI of anterior wall
I211	Acute transmural MI of inferior wall
I212	Acute transmural MI of other sites
I213	Acute transmural MI of unspecified site
I214	Acute subendocardial MI
I219	Acute myocardial infarction unspecified
I220	Subsequent MI of anterior wall
I221	Subsequent MI of inferior wall
I228	Subsequent MI of other sites
I229	Subsequent MI of unspecified site
I230	Haemopericardium current comp foll ac MI
I231	ASD as current comp following acute MI
I232	VSD as current comp following acute MI
I233	Rupt card wall wohemopericrdfoll ac MI
I234	Rupt chordae tendineae comp foll ac MI
I235	Rupt papillary muscle comp foll ac MI
I236	Atrlthromb auric append ventric w ac MI
I238	Other current complication foll acute MI
I240	Coronary thrombosis not resulting in MI
I248	Other forms of acute IHD
I249	Acute ischaemic heart disease NOS
I252	Old myocardial infarction
I255	Ischaemic cardiomyopathy
I630	Cereb infarct dt thrombosis precereb art
I631	Cereb infarct dt embolism precereb art
I632	Cereb infarct dtocclusprecereb art NOS
I633	Cereb infarction dt thrombosis cereb art
I634	Cereb infarct dt embolism cerebral art
I635	Cereb infarct dt occlusion cereb art NOS
I636	Cereb infarct dtcntrlventhrombnonpyo
I638	Other cerebral infarction
I639	Cerebral infarction unspecified
I64	Stroke not spec haemorrhage or infarct
I650	Occlusion & stenosis vertebral artery
I651	Occlusion and stenosis of basilar artery
I652	Occlusion and stenosis of carotid artery
I653	Occlus stenos mult&bilprecereb artery
I658	Occlusion & stenosis othprecereb artery
I659	Occlusion & stenosis precereb art NOS
I660	Occlusion stenos middle cerebral artery
I661	Occlusion & stenosis ant cerebral artery
I662	Occlusion & stenosis post cereb artery
I663	Occlusion & stenosis cerebellar arteries
I664	Occlus& stenosis mult&bilcereb art
I668	Occlusion & stenosis other cerebral art
I669	Occlusion & stenosis cerebral artery NOS
I693	Sequelae of cerebral infarction
I694	Sequelae of stroke not haem or infarct
I698	Seqoth/unspec cerebrovascular dis
I7021	Atheroscl artery extrem w intermit claud
I7022	Atheroscl artery extrem w rest pain

I7023	Atherosclerosis artery extremity w ulcer
I7024	Atherosclerosis artery extrem w gangrene
I713	Abdominal aortic aneurysm ruptured
I714	Abdo aortic aneurysm wo rupture
I739	Peripheral vascular disease unspecified
I740	Embolism & thrombosis abdominal aorta
I741	Embolism & thrombosis oth/unspec aorta
I742	Embolism &thromb arteries uppextrem
I743	Embolism &thromb arteries lower extrem
I744	Embolism &thromb arteries extrem NOS
I745	Embolism and thrombosis of iliac artery
I748	Embolism & thrombosis other arteries
I749	Embolism & thrombosis unspecified artery
E1052	Type 1 DM w perphangiopathy w gangr
E1452	Unspec DM w perphangiopathy w gangr
Z951	Presence of aortocoronary bypass graft
Z955	Presnc coronary angioplasty implgft
Z958	Presncoth cardiac vascular impl graft
Z959	Presnc cardiac vascular implgft NOS

Appendix 2. CVD pharmaceuticals – statins

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Statins	Statins	1063	Fluvastatin	106301	Cap 20 mg	cap
Statins	Statins	1063	Fluvastatin	106302	Cap 40 mg	cap
Statins	Statins	1137	Atorvastatin	113701	Tab 10 mg	tab
Statins	Statins	1137	Atorvastatin	113702	Tab 20 mg	tab
Statins	Statins	1137	Atorvastatin	113703	Tab 40 mg	tab
Statins	Statins	1137	Atorvastatin	113725	Tab 80 mg	tab
Statins	Statins	2513	Simvastatin	251301	Tab 10 mg	tab
Statins	Statins	2513	Simvastatin	251302	Tab 20 mg	tab
Statins	Statins	2513	Simvastatin	251303	Tab 5 mg	tab
Statins	Statins	2513	Simvastatin	251304	Tab 40 mg	tab
Statins	Statins	2513	Simvastatin	251325	Tab 80 mg	tab
Statins	Statins	2780	Pravastatin	278002	Tab 10 mg	tab
Statins	Statins	2780	Pravastatin	278003	Tab 20 mg	tab
Statins	Statins	2780	Pravastatin	278025	Tab 40 mg	tab
Statins	Statins	3853	Ezetimibe with simvastatin	385325	Tab 10 mg with simvastatin 20 mg	tab
Statins	Statins	3853	Ezetimibe with simvastatin	385326	Tab 10 mg with simvastatin 40 mg	tab
Statins	Statins	3853	Ezetimibe with simvastatin	385327	Tab 10 mg with simvastatin 80 mg	tab
Statins	Statins	3853	Ezetimibe with simvastatin	385328	Tab 10 mg with simvastatin 10 mg	tab

Appendix 2. CVD pharmaceuticals – anti hypertension

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Anti-HT	Beta blockers	1001	Acebutolol	100101	Tab 400 mg	tab
Anti-HT	Beta blockers	1001	Acebutolol	100102	Cap 100 mg	cap
Anti-HT	Beta blockers	1001	Acebutolol	100103	Cap 200 mg	cap
Anti-HT	Thiazides	1005	Acebutolol with hydrochlorothiazide	100501	Tab 200 mg with hydrochlorothiazide 1	tab
Anti-HT	Beta blockers	1029	Alprenolol	102901	Tab long-acting 260 mg	tab
Anti-HT	ACE inhibitors	1031	Trandolapril	103101	Cap 0.5 mg	cap
Anti-HT	ACE inhibitors	1031	Trandolapril	103102	Cap 1 mg	cap
Anti-HT	ACE inhibitors	1031	Trandolapril	103103	Cap 2 mg	cap
Anti-HT	Other AHT	1050	Amiloride	105001	Tab 5 mg	tab
Anti-HT	Other AHT	1050	Amiloride	105004	Oral liq 1 mg per ml	ml
Anti-HT	Thiazides	1053	Amiloride with hydrochlorothiazide	105301	Tab 5 mg with hydrochlorothiazide 50 r	tab
Anti-HT	Angiotensin II antagonists	1061	Losartan	106101	Tab 50 mg	tab
Anti-HT	Angiotensin II antagonists	1061	Losartan	106102	Tab 12.5 mg	tab
Anti-HT	Angiotensin II antagonists	1061	Losartan	106105	Tab 50 mg with hydrochlorothiazide 12	tab
Anti-HT	Angiotensin II antagonists	1061	Losartan	106125	Tab 100 mg	tab
Anti-HT	Angiotensin II antagonists	1061	Losartan	106126	Tab 25 mg	tab
Anti-HT	Beta blockers	1094	Atenolol	109401	Tab 50 mg	tab
Anti-HT	Beta blockers	1094	Atenolol	109402	Tab 100 mg	tab
Anti-HT	Thiazides	1095	Atenolol with chlorthalidone	109501	Tab 100 mg with chlorthalidone 25 mg	tab

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Anti-HT	Thiazides	1095	Atenolol with chlorthalidone	109502	Tab 50 mg with chlorthalidone 12.5 mg	tab
Anti-HT	Thiazides	1116	Bendrofluazide	111601	Tab 2.5 mg	tab
Anti-HT	Thiazides	1116	Bendrofluazide	111602	Tab 5 mg	tab
Anti-HT	ACE inhibitors	1127	Cilazapril with hydrochlorothiazide	112701	Tab 5 mg with hydrochlorothiazide 12.5	tab
Anti-HT	Angiotensin II antagonists	1254	Candesartan	125401	Tab 4 mg	tab
Anti-HT	Angiotensin II antagonists	1254	Candesartan	125402	Tab 8 mg	tab
Anti-HT	Angiotensin II antagonists	1254	Candesartan	125403	Tab 16 mg	tab
Anti-HT	Angiotensin II antagonists	1254	Candesartan	125425	Tab 32 mg	tab
Anti-HT	Thiazides	1282	Chlorothiazide	128201	Tab 500 mg	tab
Anti-HT	Thiazides	1282	Chlorothiazide	128202	Oral liq 50 mg per ml	ml
Anti-HT	Thiazides	1290	Chlorthalidone	129001	Tab 25 mg	tab
Anti-HT	Other AHT	1317	Clonidine	131702	TDDS 2.5 mg, 100 mcg per day	patch
Anti-HT	Other AHT	1317	Clonidine	131703	TDDS 5 mg, 200 mcg per day	patch
Anti-HT	Other AHT	1317	Clonidine	131704	TDDS 7.5 mg, 300 mcg per day	patch
Anti-HT	Other AHT	1318	Clonidine hydrochloride	131801	Tab 150 mcg	tab
Anti-HT	Other AHT	1318	Clonidine hydrochloride	131808	Tab 25 mcg	tab
Anti-HT	Thiazides	1367	Cyclopenthiazide	136701	Tab 0.5 mg	tab
Anti-HT	Other AHT	1604	Hydralazine	160401	Tab 25 mg	tab
Anti-HT	Other AHT	1604	Hydralazine	160402	Tab 50 mg	tab
Anti-HT	Other AHT	1604	Hydralazine	160403	Inj 20 mg per ml, 1 ml	inj
Anti-HT	Thiazides	1643	Indapamide	164301	Tab 2.5 mg	tab
Anti-HT	Beta blockers	1699	Labetalol	169901	Tab 50 mg	tab
Anti-HT	Beta blockers	1699	Labetalol	169902	Tab 100 mg	tab

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Anti-HT	Beta blockers	1699	Labetalol	169903	Tab 200 mg	tab
Anti-HT	Beta blockers	1699	Labetalol	169904	Tab 400 mg	tab
Anti-HT	Thiazides	1801	Methyclothiazide	180101	Tab 5 mg	tab
Anti-HT	Thiazides	1805	Methyldopa with hydrochlorothiazide	180501	Tab 250 mg with hydrochlorothiazide 1	tab
Anti-HT	Other AHT	1806	Methyldopa	180601	Tab 125 mg	tab
Anti-HT	Other AHT	1806	Methyldopa	180602	Tab 250 mg	tab
Anti-HT	Other AHT	1806	Methyldopa	180603	Tab 500 mg	tab
Anti-HT	Beta blockers	1817	Metoprolol succinate	181701	Tab long-acting 47.5 mg	tab
Anti-HT	Beta blockers	1817	Metoprolol succinate	181702	Tab long-acting 95 mg	tab
Anti-HT	Beta blockers	1817	Metoprolol succinate	181703	Tab long-acting 190 mg	tab
Anti-HT	Beta blockers	1817	Metoprolol succinate	181725	Tab long-acting 23.75 mg	tab
Anti-HT	Beta blockers	1818	Metoprolol tartrate	181801	Tab 50 mg	tab
Anti-HT	Beta blockers	1818	Metoprolol tartrate	181802	Tab 100 mg	tab
Anti-HT	Beta blockers	1818	Metoprolol tartrate	181803	Tab long-acting 200 mg	tab
Anti-HT	Beta blockers	1838	Nadolol	183801	Tab 40 mg	tab
Anti-HT	Beta blockers	1838	Nadolol	183802	Tab 80 mg	tab
Anti-HT	Calcium channel blockers	1863	Nifedipine	186301	Tab long-acting 10 mg	tab
Anti-HT	Calcium channel blockers	1863	Nifedipine	186302	Tab long-acting 20 mg	tab
Anti-HT	Calcium channel blockers	1863	Nifedipine	186303	Tab long-acting 30 mg	tab
Anti-HT	Calcium channel blockers	1863	Nifedipine	186304	Tab long-acting 60 mg	tab
Anti-HT	Calcium channel blockers	1863	Nifedipine	186305	Cap 5 mg	cap
Anti-HT	Calcium channel blockers	1863	Nifedipine	186306	Cap 10 mg	cap

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Anti-HT	Beta blockers	1912	Oxprenolol	191201	Tab 40 mg	tab
Anti-HT	Beta blockers	1912	Oxprenolol	191202	Tab 80 mg	tab
Anti-HT	Beta blockers	1912	Oxprenolol	191204	Tab long-acting 160 mg	tab
Anti-HT	Beta blockers	1989	Pindolol with clopamide	198901	Tab 10 mg with clopamide 5 mg	tab
Anti-HT	Beta blockers	1991	Pindolol	199104	Tab 5 mg	tab
Anti-HT	Beta blockers	1991	Pindolol	199105	Tab 10 mg	tab
Anti-HT	Beta blockers	1991	Pindolol	199106	Tab 15 mg	tab
Anti-HT	Beta blockers	2060	Propranolol	206001	Tab 10 mg	tab
Anti-HT	Beta blockers	2060	Propranolol	206002	Tab 40 mg	tab
Anti-HT	Beta blockers	2060	Propranolol	206005	Tab 160 mg	tab
Anti-HT	Beta blockers	2060	Propranolol	206006	Cap long-acting 160 mg	cap
Anti-HT	Beta blockers	2169	Sotalol	216901	Tab 80 mg	tab
Anti-HT	Beta blockers	2169	Sotalol	216902	Tab 160 mg	tab
Anti-HT	Beta blockers	2266	Timolol maleate	226601	Tab 10 mg	tab
Anti-HT	Thiazides	2293	Triamterene with hydrochlorothiazide	229301	Tab 50 mg with hydrochlorothiazide 25	tab
Anti-HT	Calcium channel blockers	2317	Verapamil hydrochloride	231701	Tab 40 mg	tab
Anti-HT	Calcium channel blockers	2317	Verapamil hydrochloride	231702	Tab 80 mg	tab
Anti-HT	Calcium channel blockers	2317	Verapamil hydrochloride	231703	Tab 120 mg	tab
Anti-HT	Calcium channel blockers	2317	Verapamil hydrochloride	231708	Cap long-acting 120 mg	cap
Anti-HT	Calcium channel blockers	2317	Verapamil hydrochloride	231709	Tab long-acting 120 mg	tab
Anti-HT	Calcium channel blockers	2317	Verapamil hydrochloride	231710	Cap long-acting 240 mg	cap
Anti-HT	Calcium channel	2317	Verapamil	231711	Tab long-acting 240 mg	tab

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
	blockers		hydrochloride			
Anti-HT	Calcium channel blockers	2398	Felodipine	239801	Tab long-acting 5 mg	tab
Anti-HT	Calcium channel blockers	2398	Felodipine	239802	Tab long-acting 10 mg	tab
Anti-HT	Calcium channel blockers	2398	Felodipine	239803	Tab long-acting 2.5 mg	tab
Anti-HT	Beta blockers	2514	Celiprolol	251401	Tab 200 mg	tab
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252801	Tab 30 mg	tab
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252802	Tab 60 mg	tab
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252803	Cap long-acting 90 mg	cap
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252804	Cap long-acting 120 mg	cap
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252805	Cap long-acting 180 mg	cap
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252806	Cap long-acting 240 mg	cap
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252807	Tab long-acting 180 mg	tab
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252808	Tab long-acting 240 mg	tab
Anti-HT	Calcium channel blockers	2528	Diltiazem hydrochloride	252809	Cap long-acting 120 mg (twice per day)	cap
Anti-HT	ACE inhibitors	2708	Enalapril with hydrochlorothiazide	270804	Tab 20 mg with hydrochlorothiazide 12	tab
Anti-HT	ACE inhibitors	2711	Enalapril	271101	Tab 5 mg	tab
Anti-HT	ACE inhibitors	2711	Enalapril	271102	Tab 10 mg	tab
Anti-HT	ACE inhibitors	2711	Enalapril	271103	Tab 20 mg	tab

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Anti-HT	ACE inhibitors	2770	Cilazapril	277001	Tab 0.5 mg	tab
Anti-HT	ACE inhibitors	2770	Cilazapril	277002	Tab 2.5 mg	tab
Anti-HT	ACE inhibitors	2770	Cilazapril	277003	Tab 5 mg	tab
Anti-HT	Calcium channel blockers	2771	Isradipine	277101	Cap long-acting 2.5 mg	cap
Anti-HT	Calcium channel blockers	2771	Isradipine	277102	Cap long-acting 5 mg	cap
Anti-HT	Calcium channel blockers	2771	Isradipine	277103	Tab 2.5 mg	tab
Anti-HT	ACE inhibitors	2772	Quinapril	277201	Tab 5 mg	tab
Anti-HT	ACE inhibitors	2772	Quinapril	277202	Tab 10 mg	tab
Anti-HT	ACE inhibitors	2772	Quinapril	277203	Tab 20 mg	tab
Anti-HT	Calcium channel blockers	2793	Amlodipine	279301	Tab 5 mg	tab
Anti-HT	Calcium channel blockers	2793	Amlodipine	279302	Tab 10 mg	tab
Anti-HT	ACE inhibitors	2794	Benazepril	279401	Tab 5 mg	tab
Anti-HT	ACE inhibitors	2794	Benazepril	279402	Tab 10 mg	tab
Anti-HT	ACE inhibitors	2794	Benazepril	279403	Tab 20 mg	tab
Anti-HT	ACE inhibitors	2795	Lisinopril with hydrochlorothiazide	279501	Tab 20 mg with hydrochlorothiazane 12	tab
Anti-HT	ACE inhibitors	2797	Lisinopril	279701	Tab 5 mg	tab
Anti-HT	ACE inhibitors	2797	Lisinopril	279702	Tab 10 mg	tab
Anti-HT	ACE inhibitors	2797	Lisinopril	279703	Tab 20 mg	tab
Anti-HT	ACE inhibitors	2806	Perindopril	280601	Tab 4 mg	tab
Anti-HT	ACE inhibitors	2806	Perindopril	280602	Tab 2 mg	tab
Anti-HT	ACE inhibitors	2840	Captopril with hydrochlorothiazide	284001	Tab 25 mg with hydrochlorothiazide 15	tab
Anti-HT	ACE inhibitors	2840	Captopril with	284002	Tab 50 mg with hydrochlorothiazide 25	tab

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
			hydrochlorothiazide			
Anti-HT	ACE inhibitors	2841	Captopril	284101	Tab 12.5 mg	tab
Anti-HT	ACE inhibitors	2841	Captopril	284102	Tab 25 mg	tab
Anti-HT	ACE inhibitors	2841	Captopril	284103	Tab 50 mg	tab
Anti-HT	ACE inhibitors	2841	Captopril	284106	Oral liq 5 mg per ml	ml
Anti-HT	ACE inhibitors	2841	Captopril	284107	Oral liq flavour free 5 mg per ml	ml
Anti-HT	ACE inhibitors	3749	Quinapril with hydrochlorothiazide	374925	Tab 10 mg with hydrochlorothiazide 12	tab
Anti-HT	ACE inhibitors	3749	Quinapril with hydrochlorothiazide	374926	Tab 20 mg with hydrochlorothiazide 12	tab
Anti-HT	Beta blockers	3772	Carvedilol	377225	Tab 6.25 mg	tab
Anti-HT	Beta blockers	3772	Carvedilol	377226	Tab 12.5 mg	tab
Anti-HT	Beta blockers	3772	Carvedilol	377227	Tab 25 mg	tab

Appendix 2. CVD pharmaceuticals – antiplatelet/coagulants

Indicator Flag	Drug Category	Chemical Code	Chemical Name	Formulation Code	Formulation Name	Units
Antiplatelet/Coag	Warfarin	2331	Warfarin sodium	233101	Tab 1 mg	tab
Antiplatelet/Coag	Warfarin	2331	Warfarin sodium	233103	Tab 2 mg	tab
Antiplatelet/Coag	Warfarin	2331	Warfarin sodium	233104	Tab 2.5 mg	tab
Antiplatelet/Coag	Warfarin	2331	Warfarin sodium	233105	Tab 3 mg	tab
Antiplatelet/Coag	Warfarin	2331	Warfarin sodium	233106	Tab 5 mg	tab
Antiplatelet/Coag	Rivaroxaban	3924	Rivaroxaban	392425	Tab 10 mg	tab
Antiplatelet/Coag	Dabigatran	3937	Dabigatran	393725	Cap 75 mg	cap
Antiplatelet/Coag	Dabigatran	3937	Dabigatran	393726	Cap 110 mg	cap
Antiplatelet/Coag	Dabigatran	3937	Dabigatran	393727	Cap 150 mg	cap
Antiplatelet/Coag	Aspirin	1087	Aspirin	108701	Tab dispersible 300 mg	tab
Antiplatelet/Coag	Aspirin	1087	Aspirin	108702	Tab 300 mg	tab
Antiplatelet/Coag	Aspirin	1087	Aspirin	108705	Tab EC 300 mg	tab
Antiplatelet/Coag	Aspirin	1087	Aspirin	108725	Tab 100 mg	tab
Antiplatelet/Coag	Clopidogrel	3860	Clopidogrel	386025	Tab 75 mg	tab

Appendix 3. Table: Adequate maintenance on triple therapy by ethnicity

GENDER	Age Band	Māori	Pacific	Indian	Other	Grand Total
Female	30 to 49	29%	36%	28%	25%	27%
	50 to 59	52%	55%	61%	49%	51%
	60 to 69	59%	54%	64%	57%	57%
	70 to 79	58%	57%	55%	57%	57%
Female Total		52%	52%	57%	54%	54%
Male	30 to 49	39%	48%	61%	47%	46%
	50 to 59	57%	61%	65%	60%	60%
	60 to 69	61%	64%	69%	65%	64%
	70 to 79	58%	61%	63%	63%	62%
Male Total		55%	60%	65%	62%	61%
Grand Total		54%	57%	63%	59%	59%

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

Construction and use of mapping techniques to describe the geographical distribution of medication dispensing for the secondary prevention of atherosclerotic CVD in New Zealand: VIEW-2

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Abstract

Background The Health Quality & Safety Commission (HQSC) is developing *Atlases of Healthcare Variation in New Zealand.* We were invited to create and map the sociogeographic distribution of medication dispensing patterns among people with atherosclerotic cardiovascular disease (CVD).

Methods We developed two interactive online atlas ótemplatesô demonstrating geographical variations in CVD medication dispensing using InstantAtlas. Each template provides stratified results in tabular, graphical and map format. In the óStandardô template proportions were mapped according to standard deviations from the mean, while the óAdvancedô template used a óheat-mapô classification to show DHB-level variations. Furthermore, we added commentaries describing the variations present and provided questions of potential interest for users.

Results The Standard template was best suited for DHB-level variations relative to a specific age, gender, or ethnic group but less effective for showing comparisons between two groups (e.g. MŌori and Pacific).

By contrast, the fixed legend and óradar plotô in the Advanced template highlighted variations in medication dispensing rates geographically and sociodemographically more effectively.

Conclusions Geographical mapping of unwarranted variations in effective care requires careful consideration. Our experience from developing this CVD atlas is documented here for developers of other components of the *Atlas of Healthcare Variation in New Zealand*.

Unwarranted variation in healthcare, defined as delivery of care that is not consistent with a patientôs preference or related to a patientôs underlying illness[1], can differ dramatically between clinicians, health care organizations, regions and countries. This variation can be described through pictorially mapping geographical differences in health care as pioneered by the influential Dartmouth Atlas of Healthcare[2] project (www.dartmouthatlas.org).

The Dartmouth group produced online atlases investigating variations in American health and healthcare using a statistically robust approach that is accessible to clinicians, researchers and the general public. Due to the increased availability and quality of electronic health records supporting the development of cohorts from routine health databases in recent years, countries such as Italy, Germany, England,[3]

Australia and New Zealand,[4] have been able to follow suit and investigate their own national geographical differences in health care.

Categorisation of unwarranted variation can carried out according to variation in preference-sensitive care, supply-sensitive care and effective care.[5] Preference-sensitive care involves trade-offs where there are (at least) two valid alternative treatments available (e.g. back surgery or conservative therapy for a herniated disc). Supply-sensitive care relates to the availability of hospitals and specialist care; the more health services allocated per capita to a given population, the greater the frequency of use.

Lastly, effective care relates to a therapy or intervention that has proven effectiveness for health outcomes and where there are no significant trade-offs. One example of effective care is the use of statins, anticoagulant/antiplatelet, and blood pressure-lowering medications (also known as triple therapy) for patients who have sustained a heart attack, stroke or other atherosclerotic cardiovascular (CVD) events.

In 2011 we were asked by the Health & Quality Safety Commission (HQSC) to construct an Atlas depicting the extent of social and geographical variation in the dispensing of the triple therapy medications among the New Zealand resident population aged 30ï 79 years with existing CVD. This would form a part of the newly developed *New Zealand Atlas of Health Care Variation*. The purpose of this Atlas was to stimulate questions, and in a shared community domain, debate why the depicted variations exist.

In this paper we briefly describe the construction of the CVD cohort from routine national health databases and then the use of mapping techniques and software functionalities to develop the online CVD atlas for the dispensing of evidence-based triple therapy. Furthermore, in this paper, we document our experience from developing this CVD atlas to guide others in their development of subsequent *Atlases of Healthcare Variation in New Zealand*.

Methods

The New Zealand Atlas of Healthcare Variation: atherosclerotic CVD Management ï cohort definition. Anonymised data from national hospitalisation, primary health organisation (PHO) enrolment, mortality and pharmaceutical dispensing datasets were used to identify all New Zealand residents aged 30-79 years, still alive in the year 2011 and in contact with the health system between 2009 and 2011, who had been discharged from hospital between 01/01/2001 and 31/12/2010 with atherosclerotic CVD. Stratifying covariates were age, gender, ethnic group (MÜori, Pacific, Indian, Other) and NZ deprivation index categorised into quintiles of deprivation. The data linkage processes, definitions of ischaemic CVD, ICD codes and covariates are described in detail in the accompanying paper by Kerr et al,[6] and in summary at http://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/cardiovascular-disease/.

We used each patientôs most recent interaction with the publicly-funded health system (i.e. GP visit, pharmaceutical dispensing, laboratory testing, in-patient and out-patient service) between 1 January 2009 and 31 December 2010 to identify the Census Area Unit representing their residential address. In addition, the CAU was used to identify the District Health Board (DHB) and National Cardiac Network Region the patient lived in. While analyses by area-level deprivation (e.g. NZDep2006)[7] are possible (and presented in the accompanying paper by Kerr et al[6]), presenting deprivation results at the DHB level is not appropriate. Deprivation indices are typically designed for small geographic areas (e.g. neighbourhoods) and the larger administrative areas such as DHBs likely mask pockets of neighbourhoods whose deprivation circumstances are significantly better or worse than the average for a given DHB.[8]

Measuring medication dispensing proportions—A person was considered to be adequately maintained on atherosclerotic CVD preventive medications (antiplatelet/anticoagulant, blood pressure lowering drug, statin or all three medications [triple therapy]) regularly if they had been dispensed these in at least three out of the four quarters of 2011.

We calculated the proportion of the population in each DHB consistently dispensed these medications and the corresponding 95% Confidence Intervals (95% CIs), by age group, gender and ethnicity using Stata version 12. [9] In some DHBs where the denominator for particular strata were small (e.g. the Pacific population in the West Coast), extreme values such as 0% or 100% were possible, for which CIs could not be calculated. Therefore, in all of our analyses, the denominator population was defined as the population+1, which enabled the calculation of 95% CIs for all data combinations.

In this study, the 95% Confidence Intervals were used to determine whether the proportion of medication dispensing in a given DHB was significantly different to the national average. The inclusion of confidence intervals in this study also acknowledges that we excluded the small proportion of patients that were admitted to a private hospital for an ischaemic event.

Geographical scale—Since 2000, there have been 21 DHBs responsible for the health of their local populations. In May 2010, the Southland and Otago DHBs amalgamated to form the new Southern DHB, reducing the number of DHBs across the country to 20. In this study, however, we kept the Otago and Southland DHBs separate because the two regions have quite different socio-demographic structures.

In 2009 the National Clinical Cardiac Network of New Zealand was formed. This Network groups adjacent DHBs into four service regions: Northern (Northland, Waitemata, Auckland, Counties Manukau); Midland (Waikato, Lakes, Bay of Plenty, Tairawhiti, Taranaki); Central (Hawke's Bay, MidCentral, Whanganui, Capital and Coast, Hutt Valley, Wairarapa, Nelson Marlborough); and Southern (West Coast, Canterbury, South Canterbury, Otago, Southland).

Instant Atlas software—Consistent with existing *Atlases of Healthcare Variation* published in New Zealand[4] we used InstantAtlasÊ software.[10] This software integrates data from GIS and Excel to produce an interactive atlas in either HTML or Adobe Flash format, according to a pre-defined map template. For example, the *Single Map Template* allows users to present their data as a single map in conjunction with a table and one or more graphs (e.g. bar graph, pie chart, time series plot, box-and-whisker plot).

The *Area Profile* template also combines a single map and table but users can choose between vertical or horizontal bar charts or radar plots. A horizontal bar chart can demonstrate medication dispensing for different subpopulations, for example by ethnicity, and indicate how a particular DHB is performing relative to the cardiac network regionôs average. By contrast, the concentric circles underpinning the Radar plot can be used to show medication dispensing proportions for every age, gender and ethnic group.

Users can modify the design and configuration of their InstantAtlas projects, but it is not possible to amalgamate functions in the Area Profile template such as a radar plot with the Single Map template. All of the elements in an InstantAtlas project are linked, so when a user selects data from the map, corresponding information in the table and graphs are also selected.

The numerators (number of people consistently dispensed medications), denominators (total number of people with atherosclerotic CVD), proportions and CIs calculated in Stata for each age band, gender and ethnicity were imported into InstantAtlas for online mapping. Our standard template comprised a Single Map template, integrating a single DHB-level map, table, bar chart of medication dispensing proportions by gender, age-group and ethnicity.

We used the HQSCôs style guide for the standard template, which presents data for a particular gender, ethnicity or age group as the standard deviation (SD) from the mean. The Standard Deviation legend in InstantAtlas classifies DHBs according to the number of SDs the medication dispensing proportion lies above or below the national mean, *for the medication and population sub-group currently being displayed.* The calculation of the SDs for display purposes is based purely on the distribution of the medication dispensing proportions, *not* the raw numerator and denominator values.

The Standard Deviation legend is calculated separately for each population group (e.g. MĀori, Pacific), which ensures that óoutliersô will be seen for each map according to the light-grey to dark-blue colour ramp. Following the HQSCôs style guide, those DHBs shaded light-grey (i.e. Ó1 SD below the mean) are defined as ólowestô in the legend, while those DHBs with medication dispensing proportions Ó1 SD

above the mean are defined as ôhighestõ. However, caution is required when interpreting variations in levels of medication dispensing between population groups.

We recommend referring to the bar charts and the graphical depiction of confidence intervals above each bar to determine whether the regional variation seen on the map is likely to be statistically significant.

The Advanced template developed for this study was derived from the Area-Profile template and combines the single DHB-level map with a table, bar chart, commentary and a radar plot. We also included additional design features such as a button to switch the display between the radar plot and the tabular data, or the inclusion of comparison areas in which users can compare variations in the proportion of people taking different medications in their DHB with the national or cardiac network regional patterns.

Whereas the legend (proportion dispensed and standard deviation) of the standard template changes every time a user chooses another variable of interest, the Advanced template uses a fixed ôheat-mapõ legend comprising four classes (Ò55%, 56-65%, 66-75% and >75%) to indicate medication dispensing proportions. The salmon-to-blue colour scheme (lowest category to highest category) used for the heat maps enables users to compare rates between age or ethnic groups in their DHB in relation to clinical best practice.

Results

Population distribution—The epidemiology of CVD medication dispensing patterns is described in the accompanying paper by Kerr et al[6] in this issue of the *NZMJ*.

In brief, we identified 86,256 patients aged 30-79 years who were survivors of an atherosclerotic CVD event in 2000-2010, of whom 57,908 (67%) were male, 29,138 (34%), 17,702 (21%), 20,317 (24%) and 19,099 (22%) lived in the Northern, Midlands, Central and Southern Cardiac Regions.

Patients of ñOtherò ethnicities accounted for 79% of the participants (most of whom were New Zealand European). MŪori comprised the second-largest group (10,958 patients, 13% of cohort), followed by Pacific peoples (4,845 patients, 6% of cohort) and Indians (1,993 patients, 2% of cohort).

Standard template format maps—Figure 1 outlines the layout of the standard template and maps the geographical distribution of using triple therapy dispensing by DHB as an example. The Data Explorer (Figure 1a) allows the user to display the proportions of people dispensed the therapy of interest (triple therapy, statins, BPL, anti-coagulant/antiplatelets, or on both statins and BPL), by gender, age group (30-39, 40-49, 50-59, 60-69 and 70-79 years) or ethnicity (MŪori, Pacific, Indian, Other Asian, or European and Others [NZEO]). This interactive standard template is published as part of the HQSCõs *New Zealand Atlas of Healthcare Variation* (http://www.hqsc.govt.nz/assets/Health-Quality-Evaluation/CVD_standard/atlas.html).

The map (Figure 1b) shows the variation in triple therapy medication dispensing using the standard deviation classification technique. The HQSC style displays DHBs using a light grey-dark blue colour ramp, with lighter shades depicting areas with lower levels of medication dispensing (Figure 1c).

The elements of the atlas are all linked, so that when a user hovers their mouse over a DHB on the map, that DHB is simultaneously highlighted in green on the map, bar chart (Figure 1d), and the table (Figure 1e). Similarly, clicking a particular bar on the graph or DHB on the map selects the data and is displayed in purple.

Given the relatively low populations of Pacific and South Asian patients in our cohort, some cells in the data tables have very few patients. Therefore, we suppress medication adherence results for DHBs where there are fewer than 10 residents. In these situations, we assign a distinct colour to the affected DHBs and remove the data from the bar graphs. The corresponding table reports the population as <10 and the prevalence is not published.

The legend (Figure 1c) is also interactive, so clicking on the darkest shade of blue (i.e. Ó1SD from the mean) selects the corresponding DHBs on the map, and the corresponding bars on the extreme right hand side of the bar graph. Note that the legend (Figure 1c) only provides labels for the Ó1SD below the mean (ñlowestò) and Ó1SD above the mean (ñhighestò) and is consistent for all therapy/demography combinations. The mean is calculated for the current display (i.e. triple therapy for all persons) and apportions the data range (54.1% to 65.6%) into 6 groups with a bandwidth of 0.5 SD from the mean.

The advantage of the bar graph (Figure 1d) in the standard template is its ability to show the proportions of medications dispensed and the corresponding 95% confidence intervals by DHB and in relation to the national average for the therapy/strata of interest. Similarly, the standard template includes the DHB-level results as a table (Figure 1e) to report the proportions but also to provide the CVD patient population in each DHB for the map of interest.

In Figure 1, the West Coast DHB is shaded purple on the map and bar graph highlighting the highest proportion of the population consistently dispensed triple therapy (65.6%). Figure 1e informs the user that there were only 719 CVD patients aged 30-79 living in the West Coast DHB in 2011.

To further add context to the CVD Atlas, we provide the users with a commentary (Figure 1f) that provides a description of the data currently being mapped, a summary of what the key patterns in the map are, and a selection of questions that these maps prompt. For example, as triple therapy among all persons is part of the gender strata in the Atlas, we prompt users to consider why more men than women are dispensed triple therapy, what the userôs DHB can do to address this difference and what can be done to improve dispensing in CVD patients in the userôs cardiac network region.

Separate commentaries were prepared for each therapy/demographic combination and will refresh, for example when users change the display from triple therapy among all persons to triple therapy among MŪori.

Additional functionality in the standard template include a Data button (Figure 1g) to toggle the display between the Data Explorer and a pie graph that allows users to select a wedge of data, which updates the display on the map and bar graph.

Whereas the Methodology button (Figure 1h) provides users with a PDF document describing our methodology, the User Guide button (Figure 1i) links to the HQSC website and the generic information regarding the atlas.

Whilst DHB-level variations can be displayed separately for any therapy/demography sub-group in the cohort, these cannot be displayed simultaneously. As a result, this display facilitates an understanding of the variation between regions for that particular

therapy/demography group, but not a comparison according to other demographic parameters e.g. MŪori vs. European, young vs. old, men vs. women.

In addition, because the standard deviations are derived for the therapy/demography sub-group of interest they cannot be used to make statistical comparisons with dispensing in other sub groups, and the use of standard deviations to show variability, whilst useful to understand statistically significant regional variation, does not emphasize the absolute magnitude of this variation, or how far from ideal targets each DHB is.

Advanced template format maps—An important aspect of this project was to engage with the sector to obtain feedback from our target audience. The stakeholder feedback (reported in detail in the accompanying paper by Grey et al.,)[11] noted some limitations of the standard template, including the inability to simultaneously visualise variability across demographic parameters beyond just DHB of residence, and use of the standard deviation classification approach which does not allow users to easily draw comparisons between all age/gender/ethnic groups for a particular DHB or cardiac network region. To address these concerns we developed the óAdvancedô template.

Figure 1. Screenshot of the 'Standard' template, displaying the geography of triple therapy medication dispensing by DHB. This template is available at http://www.hqsc.govt.nz/assets/Health-Quality-Evaluation/CVD_standard/atlas.html

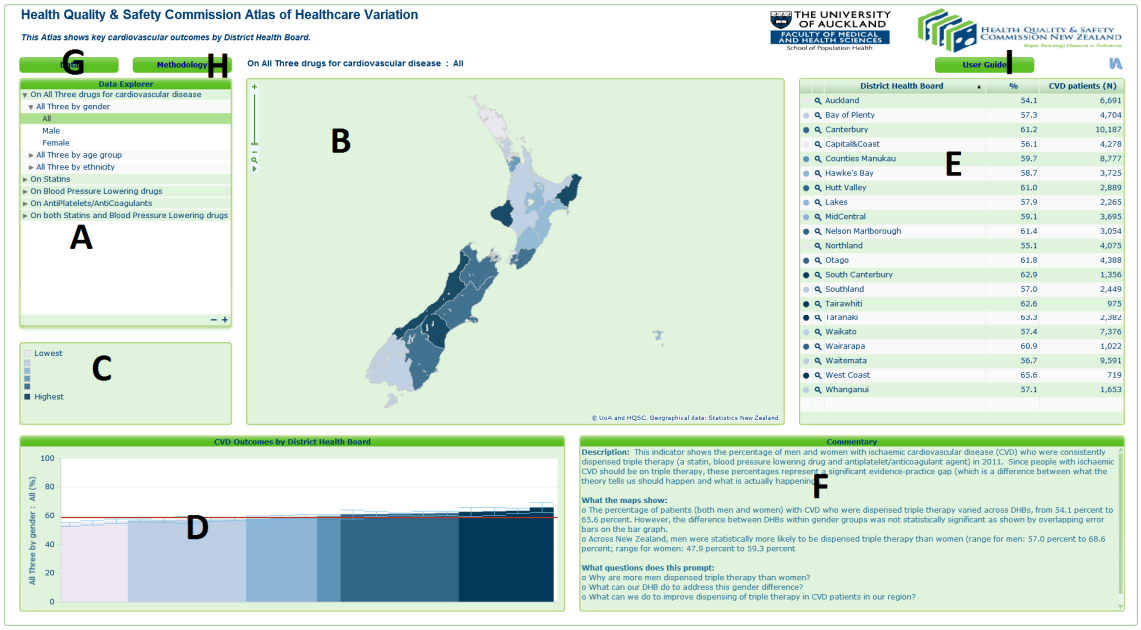


Figure 2 demonstrates the configuration of our advanced template, using geographical variations in triple therapy dispensing among the population aged 60-69 years as an example. The Atlas is available for interactive evaluation at the HQSCôs website:

http://www.hqsc.govt.nz/assets/Health-Quality-Evaluation/CVD_advanced2/atlas.html

While the functionality of the Data Explorer and bar graph in the advanced template are identical to those in the standard template, a number of modifications were made following stakeholder feedback to improve interpretation of the patterns.

First, we implemented the ‘heat-map’ legend (Figure 2a), which described variation using four groups (<55%, 56-65%, 66-75% and >75%) and is consistently applied across each drug therapy or demographic characteristic the user selects.

The legend also includes an option for users to display the outline of the four National Cardiac Network regions, but all data remains mapped at the DHB level. The location of the Chatham Islands was moved from its correct locality (some 700 km off the east coast of the South Island) to an inset close to Hawkes Bay DHB (the DHB to which these islands belong). This enabled a more compact DHB map displayed at a larger map scale than the standard template (Figure 2b).

To facilitate the comparison of variability between gender, age and ethnic groups more easily we included the radar plot to provide a summary of triple therapy medication dispensing proportions (or each medication class separately) for each demographic group for one or more selected DHBs. For example, Figure 2c shows demographic variations in medication dispensing for the Northland (purple) and West Coast (blue) DHBs, in addition to the national levels (red).

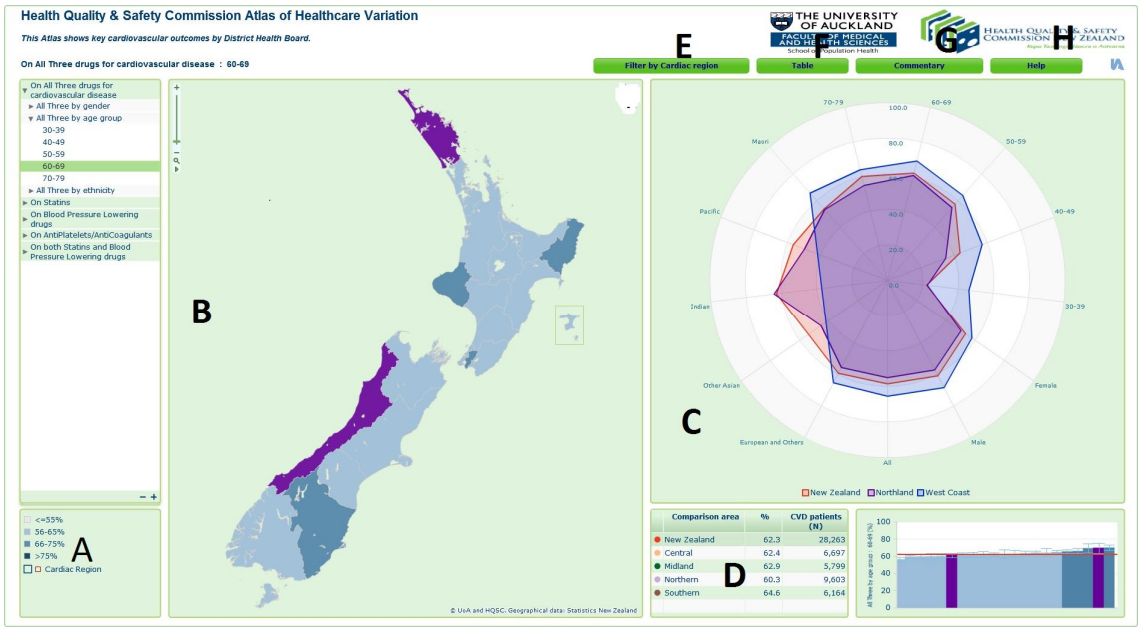
Overall, the West Coast DHB has slightly higher levels of medications dispensed than both the Northland DHB and the national average for the total population, males, females, each age group, MŪori and European/other ethnic groups. However, the radar plot highlights the low numbers of Pacific, Indian and Other Asian ethnicities in the West Coast DHB area.

By contrast, with the exception of the Indian population, the Northland DHB has dispensing proportions consistently lower than the national average. Another pattern evident from the radar plot is the low proportion of the population aged 30-39 years consistently dispensed triple therapy.

While Figure 2c compares the selected DHBs with national dispensing patterns, users can choose relevant Cardiac Network regions for comparison, from the comparison area box (Figure 2d). Furthermore, we have also included the ability to filter the map display (Figure 2e), radar plot and bar graph by cardiac network region (Figure 2f) for a more focused analysis of the regional variations.

In the advanced template we maximised the area dedicated to the geographic map and radar plot at the expense of the table and commentary that were included in the standard template. However, we have included these elements as buttons so the display can toggle between the radar plot and the table (Figure 2f) and the commentary (Figure 2g). Users also have access to the HQSC Atlas help pages (Figure 2h).

Figure 2 Screenshot of the 'Advanced' template, displaying the geography of triple therapy medication dispensing among the population aged 60–69 years by DHB. This template is available at http://www.hqsc.govt.nz/assets/Health-Quality-Evaluation/CVD_advanced2/atlas.html



Discussion

This study is the first in New Zealand to examine the geographical variations in CVD medication patterns, particularly in relation to national CVD guidelines. The online Atlas is available in an interactive format that can inform clinicians, policy makers and the public.

We stress that the HQSCôs vision for the Atlas of Variation project is NOT to create league tables that rank the performance of DHBs for a particular health outcome. Rather, the Atlas aims to highlight healthcare variations in a manner that provides benchmarks for other DHBs, and potentially provide an opportunity for others to gain knowledge related to best practice.

While the Standard layouts readily provide users with maps of medication dispensing for different health determinants, the radar plots in the Advanced layouts provide a multi-determinant perspective of different medication adherence trends within and between DHBs. The development of CVD indicators were dependent on the availability and completeness of routine health databases and the ability to link these databases using an individualôs encrypted national health identifier (NHI) to follow their cardiovascular health interactions with primary and secondary care providers.

The accuracy of our analysis is limited by the precision of data in the national health datasets, and while over 98% of the population are identifiable using the NHI,[12] the inclusion criteria for this study may be subject to misclassification error. We defined

our cohort according to patients hospitalised for (and who survived) an atherosclerotic CVD event between 2000 and 2010, while the dispensing patterns relate to 2011.

We excluded events such as unstable angina and Transient Ischaemic Attacks (TIAs), where the diagnosis may not be as certain and so CVD triple therapy is less clearly indicated and we have not captured diagnoses made in primary care, or by private providers, or overseas. In addition, our definition of consistently being on medication refers to a patient being dispensed medication in at least three of the four calendar quarters in 2011, and does not capture the purchase of medications (e.g. aspirin) over the counter. However, we acknowledge that dispensed medication is not a direct indicator of whether a patient actually takes medication daily.

Socioeconomic position is undoubtedly a key determinant of CVD health and a small-area measure of deprivation (NZDep2006) was included in our dataset, but excluded from the Atlas because there was considerable heterogeneity within large administrative districts such as DHBs and the National Cardiac Network regions.

We used InstantAtlas™ to develop our online atlas, which facilitates an effective web mapping solution for developers not proficient in specific GIS software. The Flash-based software creates templates of data that users can investigate, and the presentation of our results in the Standard Layouts is comparable to layouts used in the NHS Atlas of Variation for England.[3]

At present, we provide users with maps of medication dispensing for one variable at a time (e.g. one age, gender, or ethnic group) and although the software can display data for many strata and variables in the radar plots, grouping data by two variables such as ethnicity and age (e.g. MŪori aged 60-69) for mapping geographically would have resulted in small sample sizes and produced unstable estimates of dispensing, particularly in the less populated DHBs.

The configuration of our standard template may be misleading, especially if users do not recognise that the dispensing patterns for one group (e.g. MŪori) may be quite different to another (e.g. Indians). Using the standard deviation method to map these variables will ultimately result in the data being presented using the same colour-ramp (light-grey to dark blue), even though the range of values observed for the MŪori and Indian populations differs substantially.

As the proportions of medication dispensing are so different between some population strata, we do not recommend comparing maps in the Standard Template atlas, but the advanced templateôs fixed legend overcomes this limitation. A further limitation of this study is that our configuration of the Atlas restricts users from interrogating the data for different geographical scales or for sub-populations, such as MŪori aged 50-69 years, which is a particular strength of the Dartmouth Atlas.[2]

This research provides a benchmark against which clinicians and other health care professionals can measure DHB or National Cardiac Regional performance in the future. It is intended that these maps be used as part of quality improvement initiatives to optimise CVD secondary prevention in New Zealand. Future maps will provide time series analyses of dispensing performance over time to track change.

Research investigating unwarranted variations in effective care requires comprehensive patient-level risk factor and outcome data. Our research was only

possible due to the completeness of the National Health Identifiers maintained by the Ministry of Health. Further improvements to these data, such as improving access to electronic health records will enable the development of further atlases of unwarranted variation in healthcare.

Competing interests: Nil.

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Funding: This research was co-funded by the Health Quality & Safety Commission, the Auckland Medical Research Foundation (AMRF #1110022) and the Health Research Council (11/800). Dr Wells has a Stevenson Foundation Fellowship in Health Innovation and Quality Improvement. Corina Grey is funded by a Health Research Council Clinical Research Training Fellowship (grant number 11/814).

Acknowledgements: The authors wish to thank Richard Hamblin and Catherine Gerard at the Health Quality & Safety Commission for their support in developing this atlas. We also thank the Northern District Support Agency (NDSA) and the Northern Regional Cardiac Network for their role in initiating CVD indicator development for New Zealand. In addition we thank the team at Analytical Services, National Health Board, for the provision of the routine data used to develop this cohort, and Chris Wiltshire at Enigma Solutions Ltd for encrypting National Health Identifiers.

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

Stakeholder engagement for the New Zealand Atlas of Healthcare Variation: cardiovascular disease secondary prevention: VIEW-3

Corina Grey, Sue Wells, Daniel J Exeter, Grant Hanham, Jinfeng Zhao, Andrew J Kerr

Abstract

Aims As part of the Health Quality & Safety Commissionôs *Atlas of Healthcare Variation in New Zealand*, sociodemographic and regional differences in drug management for people with cardiovascular disease (CVD) were mapped. The aim of stakeholder engagement was to obtain feedback regarding interpretation, presentation and use of the Atlas data.

Methods Feedback was obtained through surveys, one-on-one interviews and presentations at various meetings of clinicians, managers and researchers with an interest in CVD. Presentation and utility of the Atlas data for frontline quality improvement was explored.

Results 28 stakeholders completed one-on-one feedback and over 100 attended meetings where the Atlas data were presented. Differences in dispensing by medication type, age, gender and ethnicity were thought to be related to diagnostic accuracy or the behaviour of prescribers or patients. Stakeholders found a funnel plot of the variation in triple therapy dispensing among general practices to be the most useful method of presentation, as it enabled practitioners to benchmark against peers, highlight areas for improvement, and monitor their progress over time.

Conclusion Stakeholder engagement has informed the interpretation of findings and the formatting of the Atlas data in a way that would potentially lead to improvements in the quality of patient care.

An ćevidence-practice gapô is a discrepancy between care defined as best practice from high-quality evidence and care provided in usual clinical practice.[1] Substantial care gaps have been noted in the primary and secondary prevention of cardiovascular disease (CVD) in New Zealand and elsewhere.[2,3]

In accompanying articles, Kerr et al. and Exeter et al. describe current gaps and variations in the evidence-based treatment of patients with established CVD throughout New Zealand and the mapping approach used for these data.

These analyses were conducted as part of a collaborative project between the Health Quality & Safety Commission (HQSC), the Northern District Health Board Support Agency (NDSA) and the School of Population Health for the HQSCôs *Atlas of Healthcare Variation in New Zealand* (henceforth referred to as the *Atlas*).

While some variation in healthcare is expected, high levels of variation for treatments with a robust evidence base may indicate systematic differences in practice between different organisations or in the care of certain population groups.[1]

There is no facility in New Zealand for the regular reporting of care gaps for patients with established CVD at a national level. Clinicians should be aware of their own prescribing patterns but have little knowledge of medication use at a regional level or by population subgroup.

Researchers have been criticised for failing to disseminate healthcare practice-related findings to providers and health services in a meaningful, accessible way that can be used to improve healthcare delivery.[4,5] The *Atlas* therefore provided a unique opportunity to report key findings to clinicians and other stakeholders to raise awareness of care gaps for CVD patients, and to obtain feedback to ensure meaningful data interpretation and presentation.

This paper outlines the process and outcomes of stakeholder engagement for the CVD management component of the *Atlas*. The ultimate goal of the project was to actively engage stakeholders to help inform the interpretation and presentation of the Atlas data in order to better support improvements in health services and frontline care.

Methods

Data analysis for the New Zealand Atlas of Healthcare Variation: CVD management— Development of, and key findings from, the CVD management section of the Atlas are described in detail elsewhere (Kerr et al; Exeter et al) and are available at http://www.hqsc.govt.nz/atlas

Anonymised data from national hospitalisation, Primary Health Organisation (PHO) enrolment, mortality and pharmaceutical dispensing datasets were used to identify all New Zealand residents aged 30ï 79 years, alive in 2011, who had been discharged from hospital between 01/01/2001 and 31/12/2010 with ischaemic CVD.

A person was considered to be taking a relevant medication (antiplatelet/anticoagulant, blood pressure lowering drug, statin or all three medications [triple therapy]) regularly if they had been dispensed it in three out of the four quarters of 2011. Ethical approval was obtained from the Multi-region Ethics Committee (MEC/11/EXP/078).

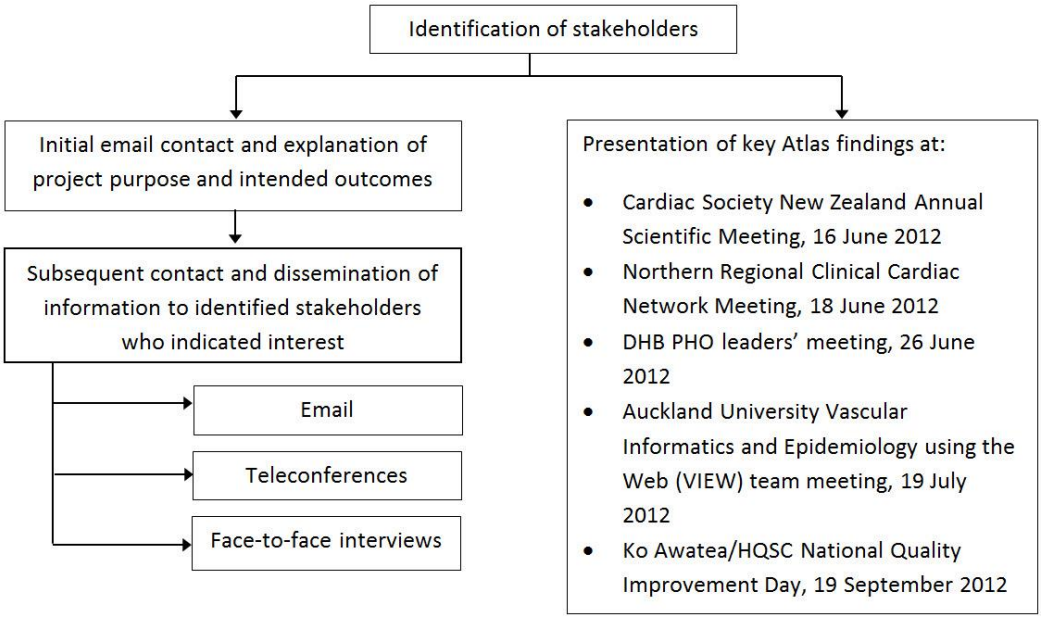
Data analysis identified four major differences in dispensing by medication type, age, gender and ethnicity (see Kerr et al. for more detail). These findings are summarised in Table 1.

Table 1. Differences in preventive medication dispensing for patients with CVD

Variables	Results	Proportions dispensed triple therapy
Medication type	Fewer people were dispensed statins compared to other CVD drugs.	
Age	Fewer younger people were dispensed medications.	30ï 49 years 40% 60ï 69 years 62% 50ï 59 years 57% 70ï 79 years 60%
Gender	Fewer women were dispensed medications, particularly statins.	Women 54% Men 61%
Ethnic group	Compared to Europeans, a higher proportion of Indians, and a lower proportion of Maori and Pacific people were dispensed triple therapy.	European/Other 60% Maori 54% Pacific 57% Indian 63%
Socioeconomic status	There were no significant differences in medication dispensing by socioeconomic status.	

Process of stakeholder engagement—The stakeholder engagement process is outlined in Figure 1. All people working in the health sector with an interest in inequalities and/or CVD were considered potential stakeholders. Stakeholders included: regional leaders and members of the National Clinical Cardiac Network (including all New Zealand cardiologists); Public Health Physicians from Regional Public Health Units, DHB Funding and Planning Units, NDSA and Pharmac; PHO chief executives and locality managers; General Practice (GP) liaison officers; epidemiologists and other researchers; chairs, directors or key contacts for the New Zealand Medical Association GP Council, Royal New Zealand College of General Practitioners, GP Nursing Alliance, National Heart Foundation, Te Ora, Pasifika Medical Association and consumer advocacy groups.

Figure 1. Flowchart showing the steps taken in the stakeholder engagement process



Over 100 stakeholders were identified and contacted via email to inform them of the *Atlas* and the purpose of stakeholder engagement. Three weeks after initial email contact, those that had indicated interest were sent documents and instructions on how to provide feedback.

The documents included a presentation outlining the aims, methods, key findings and maps for the Atlas, an accompanying document with 14 feedback questions, and a Technical Document (for reference only) describing the methods and results in greater detail. Stakeholders were able to provide feedback via email, teleconference or face-to-face interview. All feedback was anonymised.

Stakeholder feedback questions and analysis of data—The feedback questions covered three domains related to the Atlas: interpretation of results, format of presentation and data use. The interview schedule is shown in Table 2.

The content of the stakeholder feedback was analysed by domains using a General Inductive approach to qualitative data analysis and organised into emergent themes.[6]

Table 2. Interview schedule

Data Interpretation

1. Do you have any comments regarding the differences in dispensing of statins compared to antiplatelets/anticoagulants and blood pressure lowering drugs? Does this seem consistent with what you see in your practice?
2. Why do you think there are age differences in the dispensing of CVD medications?
3. Why do you think there are gender differences in the dispensing of CVD medications?
4. Why do you think there are higher proportions of Indians dispensed CVD medications (especially triple therapy) compared to other ethnic groups?
5. What do you think about there being no socioeconomic differences in dispensing?

Data Presentation

6. What do you think of the Instant Atlas? Is it useful?
7. Do you like the colours/layout/setup?
8. Is there anything you think is missing or unnecessary in the map?
9. Any suggestions for improvement?
10. Does the funnel plot convey useful information? Why/why not?

Data Use

11. Do you think practices would want to know/should know where they lie on the funnel plot? Do you think practices should know where other practices lie on the funnel plot?
12. At what level should this information be aimed at? (PHO / Practice / Provider)
13. How do you think the information could be used to achieve or feed into existing quality improvement processes?
14. These reports are all based on non-patient identifiable data. It would be technically possible, on request from the GP, to provide lists of patients who are not being dispensed prevention medication. Do you think this would be useful for a practice based quality improvement program? What privacy safeguards would need to be in place? Other comments?

Results

Over 100 individuals from health-related organisations in the public and private sector were invited to participate in the feedback process. Of these, 40 indicated interest and 28 completed feedback.

The group that completed feedback questions comprised 20 clinicians and/or other healthcare workers (seven primary, seven public health, five secondary and one pharmacy), four managers from DHBs, PHOs and Pharmac and four health researchers.

In addition, the results of the data analysis were presented to, and feedback compiled from, 25 people at the June meeting for the Northern Clinical Cardiac Network (cardiologists, general practitioners and hospital managers), 15 people at a DHB PHO Leadersô meeting (General Practitioners, PHO and DHB Managers and Clinical Directors), 10 people at Auckland University (epidemiologists, pharmacists and analysts with a special interest in CVD), 10 people at a workshop at the Ko Awatea National Quality Improvement Day (hospital managers, chief analysts and health spokespeople from throughout New Zealand and Samoa), and approximately 100 people at the Annual Scientific Meeting for the Cardiac Society of Australia and New Zealand.

Data interpretation—The main themes of the stakeholder responses to the data interpretation domains are summarised in Table 3.

Data presentation—Stakeholders were shown a map of triple therapy management and asked to critically assess its layout and usefulness. Overall, 45% of stakeholders agreed the map was useful, 25% disagreed, and 30% had no opinion.

Many people felt that the data presented on the map were easier to interpret as graphs and tables. The most common reason for disliking the map was that it seemed to highlight differences between regions that were not statistically significant.

Many stakeholders felt that having a fixed number of variation categories (five per map for each population subgroup) created an illusion of regional differences when none really existed. Instead, many preferred to have shades of colour representing categories that were consistent for all maps to better enable across-group comparisons.

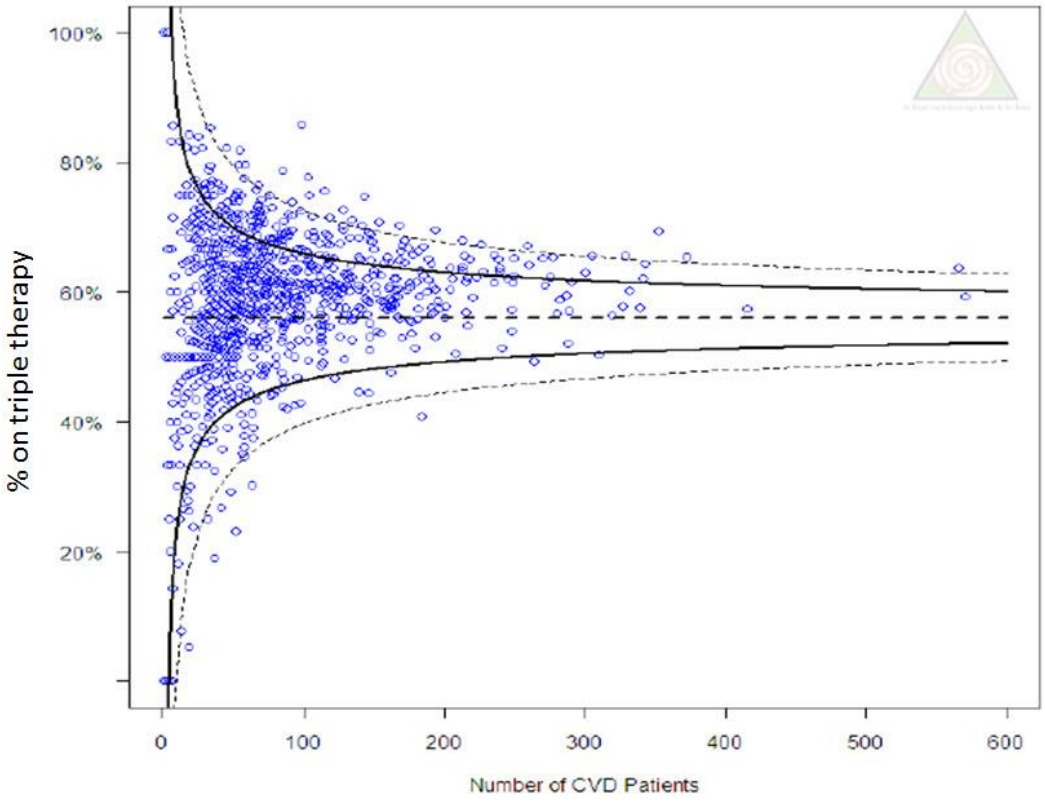
Use of data—To identify how data could be used by health practitioners to improve dispensing of medications, stakeholders were shown a funnel plot (Figure 2) of the variation in triple therapy dispensing among all GPs throughout New Zealand and asked to assess its usefulness. Each dot on the funnel plot represents a practice.

The proportion of patients on triple therapy is shown on the y-axis and the number of CVD patients per practice on the x-axis. The central dotted line shows the mean percentage of people on triple therapy and the other two lines represent the 95% and 99% confidence intervals, respectively.

Table 3. Main themes of stakeholder responses to questions regarding differences in medication dispensing by population group/medication type

Differences in dispensing (see Table 1):	Prescribers' behaviour	Patients' behaviour	Diagnostic accuracy
Medication type	Treating risk factors (e.g. blood pressure) rather than absolute risk.	Opting for ólifestyle changesô in preference to taking medications to lower lipid levels.	
	Still believing that statins need Special Authority.	Not realising that statins are a lifelong treatment.	
		Responding to negative publicity about possible side effects of statins.	
Age	Perceiving younger people as being at lower risk of a recurrent event.	Younger people being generally less engaged with the health sector, so there is less opportunity to be prescribed medications.	Diagnosis of ischaemic CVD is less accurate in younger people.
	Treating risk factors, which are more likely to be ónormalô in younger people.	Younger people tend to recover faster after a CVD event and may not see a need to take medications.	
		Older people tend be more accepting of the fact that they have an illness and be more compliant.	
		Younger people may be less aware that their condition is severe and impacts on mortality.	
		Younger people may be more likely to opt for ólifestyle changesô in preference to taking lifelong medications.	
Gender	Perceiving the CVD risk of women to be less than that of men.	Women may perceive more medication side effects (especially from statins).	Younger women are more likely to have non-atherosclerotic reasons for CVD events than men.
	Being more reluctant to treat women of childbearing age with statins and blood pressure-lowering drugs.	Women may be more open to lifestyle changes.	
		Women may be busy looking after others and not prioritise their own health.	
Ethnic group	Being more aware of the fact that Indians are a group at high risk of CVD and diabetes.	Indians may be more motivated to take medications due to: A heightened awareness of the effects of CVD in the community A culture where taking long-term medications is more acceptable High levels of family support	

Figure 2. Funnel plot showing the variation in triple therapy dispensing by general practice



Clinicians and primary care managers found the funnel plot particularly useful. Five major uses of the plot were identified and are outlined in Table 4.

Table 4. Uses of a funnel plot showing variation in triple therapy dispensing by general practice

• Benchmark achievement against peers of similar size • Know what is possible and normal in a population • Highlight where we could do better • Identify high performing practices and pinpoint factors related to their success • Monitor progress

Stakeholders agreed that individual practices should be told their own position on the funnel plot, but not the position of other practices.

There was an overwhelming sense that the funnel plot should be used to motivate practices to ñdo betterò rather than ñname and shameò. Stakeholders emphasised the need to re-publish funnel plots at regular intervals to monitor progress and create feedback loops.

When stakeholders were asked whether unencrypted data (with ethical approval) should be used to provide lists to primary care providers of patients potentially missing out on therapy, 50% agreed, 23% disagreed, and 27% were undecided.

Stakeholders who disagreed were concerned about the potential breach of individual privacy rights. Stakeholders who agreed felt that, with the right governance structures in place, if non-anonymised lists of patients were sent to PHOs to disseminate to individual practices, this would benefit both providers and patients.

Smaller PHOs were seen to gain the most, because their limited resources often meant that there was not enough time, staff or software available to identify individual patients easily.

Discussion

In this paper, we have described a process of stakeholder engagement undertaken as part of the *New Zealand Atlas of Healthcare Variation (CVD Management),* a project which aims to improve the quality of long-term CVD management. We obtained individual feedback from 28 clinicians, healthcare managers and researchers, and received input at five different meetings (attended by over 150 people) to assist with data interpretation, presentation and utility.

This feedback led to extra maps being created to reflect more consistent categories of medication dispensing and to better enable across-group comparisons (see Exeter et al. for further detail). The ideas generated around data use are also being explored further, and as a result of feedback, data atlases will be published on a regular basis.

One of the key recommendations from stakeholders was that DHBs and PHOs should be able to access clear and concise summaries of medication dispensing by sociodemographic factors for their region. This would enable better targeting of interventions to those groups currently missing out on treatment.

We therefore developed radar plots of medication dispensing for each DHB region. These plots are a graphical method of displaying multivariate data on a two-dimensional chart, and enable users to rapidly identify sub-populations of CVD patients receiving the lowest levels of treatment in their region. See the accompanying paper by Exeter et al. for further information. These radar plots are available as an advanced function on the web-based HQSC maps: http://www.hqsc.govt.nz/assets/Health-Quality-Evaluation/CVD_advanced2/atlas.html

Stakeholdersô responses to questions regarding the reasons for differences in dispensing by age, gender and ethnicity provided insight into potential barriers to medication use for different patient groups. These responses suggest that the observed variations may stem, in part, from prescriber and patient misperceptions about individual risk and the role of medications for secondary prevention.

There is currently a paucity of data on how best to convey information about CVD risk to patients with pre-existing CVD. However, the few studies that have been conducted suggest that numerical and graphical presentation of risk over a short timeframe (<10 years) can lead to more accurate risk perceptions in patients and encourage acceptance of treatment.[7]

While these discussions are important in all patients, our results indicate that clinicians may need to spend more time communicating these messages to patients who are younger (particularly <50 years), women, and those of Maori or Pacific ethnicity.

Actively engaging stakeholders with data from the *Atlas* enabled people to interpret the results with reference to their everyday work. While awareness of new evidence is a prerequisite to changing practice, evidence suggests that there are still large differences between what clinicians know and what they do.[8] Clinicians face barriers to following guideline recommendations other than knowledge, including a lack of motivation and/or self-efficacy and competing promotional influences.[1] The stakeholder engagement process shared many features in common with strategies that have been recommended to overcome such barriers, including face-to-face educational outreach,[1] audits and feedback with comparison to local peers.[9]

Stakeholder engagement is thought to be an important part of any project aimed at quality improvement in healthcare.[10] However, few studies outlining processes for, and outcomes of, stakeholder engagement appear in the medical literature. Those that have been published tend to focus on reducing medical errors in hospitals.[11, 12]

To our knowledge, this is the first paper to describe a process of stakeholder engagement in relation to secondary prevention of CVD. One of the strengths of this project was its interactive nature, which benefited both the research team and participants. Data analyses were enriched by insights provided by those at the frontline of healthcare. Their shared experiences may help develop interventions that address challenges that patients and prescribers face in the management of CVD.

Benefits of the engagement process for stakeholders included being provided with an opportunity to ensure that *Atlas* data was interpreted in a manner relevant to their daily practice and to have input into how data was presented and whether unencrypted data should be used for quality improvement.

A limitation of the project was the low individual response rate. While every effort was made to increase the participation rate, the project had a very short time frame (four months). However, with a sampling frame targeted to people involved in policy, practice and frontline quality improvement, data saturation was reached after the third meeting we attended. We also acknowledge that engagement did not extent to patients or other members of the public, media or politicians. This should be done in the next stage of evaluation. Our aim for this project was to optimise its use for health professionals and health sector managers.

The *Atlas* data provide an important description of the current state of the secondary prevention of CVD in this country. Based on feedback from the stakeholder engagement process, these data will also motivate practitioners to improve the treatment of their CVD patients and identify potentially excluded population groups.

Stakeholders were clear that regular, up-to-date reports were needed to monitor progress and maintain motivation to increase levels of CVD treatment. A particular recommendation was that dispensing data at the practice level should be provided to PHOs, so that it could be used for quality improvement. A commitment has therefore been made to produce these reports annually and ensure they are widely disseminated.

The first set of practice-based reports was circulated in the Northern region in 2013 by the NDSA. The 2012 *Atlas* therefore provides a baseline measure against which success of quality improvement efforts throughout the country can be monitored. We intend to incorporate updated data into the *Atlas* in the future.

Competing interests: Nil.

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Funding: This research was co-funded by the Health Quality & Safety Commission, the Auckland Medical Research Foundation (AMRF #1110022) and the Health Research Council (11/800). Dr Wells was supported in part by the Stevenson Foundation. Corina Grey is funded by a Health Research Council Clinical Research Training Fellowship (grant number 11/814).

Acknowledgements: The authors thank Richard Hamblin and Catherine Gerard at the Health Quality & Safety Commission for their support in developing this Atlas; stakeholders throughout New Zealand from primary care, secondary care and public health for contributing to this project; Geeta Gala and Helen McKenzie at the Northern District Support Agency (NDSA) for their assistance in this project; Northern Regional Cardiac Network for their role in initiating CVD indicator development for New Zealand; the team at Analytical Services, National Health Board, for the provision of the routine data used to develop this cohort; and Chris Wiltshire at Enigma Solutions Ltd for encrypting National Health Identifiers.

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

Why are we failing with the epidemic of obesity and other chronic diseases? A further look at aetiopathogenesis

William Ferguson

Abstract

It is timely to be addressing this question: in some respects medicine and the delivery of healthcare has driven itself slowly to an impasse, whilst the biological sciences that should underpin our understanding of healthcare are undergoing a revolution. That there could now be seriously conflicting scientific opinion over the role of saturated fats in the aetiopathogenesis of coronary heart disease is a terrifying indictment of our limited understanding of the role of the modern diet in disease.

Currently there remains a substantial disconnect between the new directions indicated by the biological sciences, and our entrenched views concerning treatment of the epidemic chronic diseases such as obesity. As the new knowledge unfolds it seems likely that a better understanding of diet in all respects will move to centre stage as we endeavour to solve these problems.

The last decade has seen the field of epigenetics emerging from the fog, and with it entirely new biological structures for understanding the aetiology of disease.[1,2]

This development has thrown open an important window upon our understanding of the mechanisms by which diet, nutrition, and many other environmental factors influence gene expression and ultimately health and disease.[3] The specialty within which the study of epigenetics has found immediate relevance has been that of fetal development andð more specificallyð the developmental origins of adult disease.[4]

It increasingly appears that the fetus is the ócanary in the coalmineô: maternal physiology and biochemistry, and anything that influences it, can potentially be transfused into lifelong patterns of gene expression and consequent risk for metabolic disease.[5,6] From the moment of conception the physiological attributes of the expected environment are being used to program changes in chromatin structure and expression of the genome, through the intermediary of even the most subtle nuances in the maternal microenvironment. The major window of this epigenetic imprinting takes place up until the time of birth.

The clustering comorbidities of obesity, diabetes, cardiovascular disease, and even mood disorder are becoming epidemic in the developed world. Questions are now being asked about the relationship between the surge of these disorders and the modern western diet, as well as other aspects of the environment that coexist with these pathologies.

Consider the example of obesity, which is clearly a multifactorial condition. Many different potential genetic, epigenetic, environmental, nutritional, and lifestyle factors all potentially contribute to an apparent variety of developmental trajectories that lead to this condition.

Rather than reflecting fixed change in the genome itself, the explosion of the epidemic of obesity and metabolic disease suggests factors that are environmental and epigenetic. Molecular epidemiology has failed to find any single strong identifiable genetic links; instead, there is an association with a broad range of genes subject to many single nucleotide polymorphisms. Generically-applied dietary and lifestyle advice to address obesity that takes no account of this heterogenicity is of limited value. Unfortunately, some past public health advice in this respect has not been good.

The plethora of animal and human studies which highlight many different facets of developmental programming known to influence adult obesity have recently been well reviewed.[7] For example, it now appears from both animal and human studies that the macronutrients in a pregnant womanôs diet can influence the incidence of obesity in the later life of her offspring.[8,9]

Manipulating the macronutrient component of the maternal diet in animal studies (in which diet can be rigorously controlled) has been shown to induce obesity in the offspring.[10,11] Thus the quantity, the quality, the composition, and the timing of nutritional interventions in animal models have all been shown to potentially influence adult obesity. If there are aspects of the maternal diet that either augment or attenuate the transgenerational transmission of obesity risk, then it is urgent that these issues be clarified.

To add yet another layer of complexity, it has been demonstrated that changes in 1-carbon metabolism may provide a broad amplification or reduction of the risk generated by these macronutrient modifications.[12] In animal studies, the modification of DNA methylation by methyl donor supplementation has been shown to prevent transgenerational amplification of obesity in the well worked Agouti mouse model of genetic predisposition to obesity.[13,14] Restriction of folate, vitamin B12, and methionine from the peri-conceptual diet of sheep-induced obesity in the offspring.[15] The influence of diet upon epigenetics extends beyond the provision of macronutrients and methyl groups.

There are some 25,000 bioactive compounds in the human diet, such as bioflavanoids, that also directly influence many aspects of epigenetic regulationð although their role, if any, with obesity is unknown. Furthermore, the fetus may be especially sensitiveð on account of its open window of epigenetic ówork in progressôð to the consequences of endocrine disrupting chemicals, now known to include obesity.

In another environmental-dietary nutrient type interaction, bisphenol A-induced epigenetic changes were shown to be negated by additional methyl group supplementation during gestation.[16] Thus diet, in terms of its macronutrient composition, the availability of methyl groups, and its xenobiotic content have all been shown to influence epigenetic mechanisms relevant to fetal programming and obesity.[17]

Increasingly, we see that a single environmental, genetic, or epigenetic input may lead to many diseases, and a single disease may have many inputs. Thus, environmental inputs as diverse as psychosocial stress, toxic exposure, and nutrient deficiency are capable of programming the pre-natal hypothalamic-pituitary axis in a manner that increases vulnerability to disease processes as diverse as diabetes, obesity,

cardiovascular disease, and depression. These epigenetic influences are now understood to be transferable to a subsequent generation.

To illustrate this, a lot of very disparate lines of research have converged on epigenetic modification of the expression of the glucocorticoid receptor. Thus, psychosocial stress (both during pregnancy and in the neonatal period) has been shown to induce differential methylation in the promoter region of the glucocorticoid receptor.

In animal studies, these changes were reversible with methionine, the precursor of the methyl donor S-adenosyl methionine.[18,19] It has since been shown that partner violence in pregnancy and childhood maltreatment also increases methylation of the glucocorticoid receptor promoter region.[20,21] However, epigenetic effects in the same receptor have also been demonstrated to be a target of modifications in the protein content of the diet in pregnancy,[22] again reversible with dietary methyl supplementation.[23]

Thus, glucocorticoid receptor sensitivity seems to be a key point at which the hypothalamicï pituitary axis (HPA) reactivity is set or programmed by aspects of the intrauterine milieu. Meaney[24] proposed that ñthe HPA axis is both a target for environmental influences and a mediator of the relationship between early life events and health in adulthood.ò

Apparently, anything the fetus translates as stressð be it nutritional, psycho-social, or due to toxic exposureð is translated into an integrated set of physiological responses centred around altered HPA-axis functioning, including heightened stress response, increased central nervous system corticotrophin releasing factor (CRF), and adaptations centred around increased production and storage of energy.[25]

The broad environmental inputs into fetal HPA axis programming are all ultimately translated (at least in terms of GR receptor function) into a simple binary code of HPA-axis functioning, which manifests across multiple endocrine, metabolic, and central nervous systems. When dysregulated, this seems to be associated with that familiar cluster of epidemic comorbidities in adult life.

A ómoney launderingô effect occurs by which it becomes impossible to tease out the diverse environmental inputs (apparent risk factors) from the adult disease outcomes. Thus, for example, in terms of glucocorticoid receptor methylation, prenatal dietary effects on methylation pertinent to obesity may be relevant to the aetiology of lifelong depression, and factors relevant to depression might also affect obesity. There will be a summation of effects, and this will occur across many receptors and many genes yet to be studied as closely. It is also clear that these epigenetic effects can be transmitted to a second generation.[26]

It now appears that, in many instances, causality may be better conceived as a network, and that these networks are not constrained to a single organ system; nor is the understanding of them contained in a single medical specialty-based silo of knowledge.[27,28] The increased recognition of the protean effects of underlying inflammatory mechanisms and methylation imbalances across a wide spectrum of diseases are two examples of an evolving understanding of pathophysiology.

Interest is now focusing on ‘intermediate patho-phenotypes’ or ‘endophenotypes’ (a term co-opted recently within the field of biological psychiatry). This relates to underlying mechanisms of disease, often underpinned by genetic or epigenetic variations, and often brings a commonality to disorders that have seemed otherwise unrelated in terms of existing diagnostic classifications.

As we dig deeper into the aetiology and pathophysiology of these chronic conditions, current diagnostic groupings of diseases start to look less relevant, and increasingly the molecular signatures of disease are providing better information relevant to treatment and prognosis than histopathology.[29] The unexpected benefits of statins– by virtue of their anti-inflammatory actions on patients with chronic obstructive pulmonary disease (COPD) and in the prevention of malignancy-related thromboembolic disease, and the diverse influence of omega-3 fatty acids and vitamin D across a range of disease processes– also illustrates aspects of this emerging complexity when we consider treatment.

The chronic diseases that the health system is failing to address, such as obesity and treatment resistant depression, are all subject to significant genetic influence from dozens, if not hundreds, of genes. These genes are subject to a multitude of single nucleotide polymorphisms, the expression of which is associated with a broad range of effects that can be cross-referenced to lists of seemingly unrelated diseases.

Downstream of this biological investigators are now working with vast transcriptomic, proteomic, and metabolomic datasets in individual health and disease which contain useful functional information and which again create new groupings of previously unrelated disorders.[30] Targeted profiling of these polymorphisms and other diagnostic processes that identify relevant intermediate patho-phenotypes has the potential to lead to more individualised care for patients within these broad diagnostic categories. Such profiling can enable very important differences to be made in the provision of advice to patients concerning pharmacological agents, diet, nutrients, lifestyle, and even exercise.

Fundamentally, it informs the ability to individualise treatment in order to achieve optimum therapeutic efficacy. It also holds the promise of an improved ability to prevent disease, and a roadmap towards the application of public health genomics has been proposed.[31] But this approach cannot easily be assimilated into the trusted methodologies of evidence based medicine– particularly not the randomised controlled trial (RCT), which studiously and philosophically avoids the individualisation of treatment. Thus, work in these areas currently exists only on the fringe of mainstream medicine.

The RCT aims, by weight of numbers, to systematically eliminate individual differences. Thus, an effective intervention for a small undefined sub-group within a heterogeneous study population may be concealed. The more that individuality is probed by the RCT methodology, the smaller the numbers and the weaker its power to discern an effect. The ability of that RCT to ‘see’ an effect in a vast heterogeneous population will depend entirely on its ability to precisely identify and group small numbers of patients in some of the new ways alluded to above.

Thus the current pervading medical paradigm sees the acknowledgement of the biological reality of individuality as problematic– at the very moment that the

biological sciences are making sense of it. In trying to treat these new epidemics we risk being like a shoe salesman trying to fit the same shoe size to every customer because we have evidence pertaining to the size of the average foot. To move forward with these diseases there must first be a necessary mind shift to accepting that bigger and bigger trials with single treatment or intervention modalities is not going to provide us with the answers, or certainly not with the speed which is now required.

To ñdeliver the right treatment to the right patient; and the right prevention to the right populationò[32] there are three inescapable conclusions. The first is the necessity of a better understanding of the emerging role of diet in all of the mechanisms alluded to above. The second is that dietary interventions must be first targeted both pre pregnancy, during pregnancy and in the first postnatal year. The third is that ultimately, more effective treatment can only be delivered by interventions better individualised and targeted to specific underlying mechanisms of disease, not just treatments allotted by traditional diagnostic categorisation.

Competing interests: Nil.

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

Cephalic tetanus complicating geriatric fall

Josie McCabe, Tessa La Varis, Deborah Mason

Abstract

We report a case of cephalic tetanus which initially presented with acute lower motor neurone facial weakness. Tetanus is a rare diagnosis in the developed world but sporadic cases do occur. People born before 1960 in New Zealand are less likely to be immune. Judicious use of human tetanus immunoglobulin (TIG) and immunisation prevents the development of tetanus following injury and should always be considered in the elderly who are less likely to have immunity.

An independent 87-year-old lady with a history of falls and seronegative arthritis, treated with hydroxychloroquine, presented to hospital with a 1-day history of left facial weakness. Fourteen days previously she lacerated her leg whilst gardening and received a single tetanus vaccination. Seven days prior to presentation she fell again, sustaining a left forehead laceration that was sutured.

On admission she was hypertensive (210/70 mmHg), had left lower motor neurone facial weakness and dysphagia with normal limb neurology. CT head showed small vessel ischaemic change. She developed bilateral facial weakness and trismus. The clinical presentation was unusual and a diagnosis of post-immune polyradiculitis involving multiple cranial nerves (polyneuritis cranialis) secondary to tetanus toxoid injection or herpetic infection was considered.

An inability to manage oral secretions necessitated sedation and intubation on day three. MRI brain and CSF examinations were normal, nerve conduction studies and electromyelography of limbs showed normal motor conduction and no evidence of denervation. Debridement of the head wound revealed a 6 cm^2 area of necrotic muscle. Treatment with metronidazole and human tetanus immunoglobulin was commenced following a clinical diagnosis of cephalic tetanus.

Tetanus is caused by *Clostridium tetani* a spore-forming organism ubiquitous in the environment. Under anaerobic conditions the spores germinate and release tetanospasmin, a powerful neurotoxin, which binds irreversibly to the motor neurone then migrates by retrograde axonal transport to the neuromuscular junction where the light chain cleaves synaptobrevin. This prevents inhibitory control of alpha motor neurones leading to tetany and disrupts autonomic controls causing autonomic instability.[1]

Cephalic tetanus is characterised by paralysis of one or more cranial nerves (thought to be due to reduced acetylcholine levels at motor end plate), presence of trismus and a cranial source of tetanus spores, usually a wound. Cephalic tetanus is a clinical diagnosis which can mimic a variety of conditions including stroke and vestibular neuritis.[2,3]

Immunisation and improved wound care have made tetanus a rare disease in the developed world. Tetanus toxoid immunisation became universal in New Zealand in

1960; people born prior may have never received the full course of three immunisations.

Immunity is expected to last 20 years after a primary course and booster, immunisation status should be reviewed in adults at ages 45 and 65.[4] It is recommended that anyone with an unclear immunisation history treated for a tetanus-prone wound (penetrating, older than 6 hours or contaminated) receives a course of three immunisations plus intramuscular human tetanus immunoglobulin (TIG) at a dose of 250ï 500 IU.[4]

TIG acts by binding the tetanus toxin before it binds to neurones. The only documented tetanus immunization this patient received was 2 weeks prior to presentation. There is some evidence suggesting that hydroxychloroquine treatment may reduce the immune response to vaccination.[5]

Modern intensive care has improved survival but mortality remains between 12ï 38%.[6,7] Poor prognostic markers include a short incubation period, rapid progression of symptoms, cephalic or neonatal tetanus, autonomic instability and no history of prior immunisation.[7,8]

Tetanus in developed countries is now predominantly a disease of older adults. In New Zealand there were 21 tetanus cases in 2001ï 2012, of which 9 were born prior to 1960.[4] The elderly are less likely to have received full immunisation or booster in the preceding 20 years; in serological studies the older population have lowest levels of immunity.[9]

Most clinicians will never encounter clinical tetanus, however sporadic cases do occur and a high index of clinical suspicion is required to make the diagnosis. Judicious use of TIG and immunisation prevents the development of tetanus following injury and should always be considered in the elderly who are less likely to have immunity.

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

An unusual case of painful ophthalmoplegia

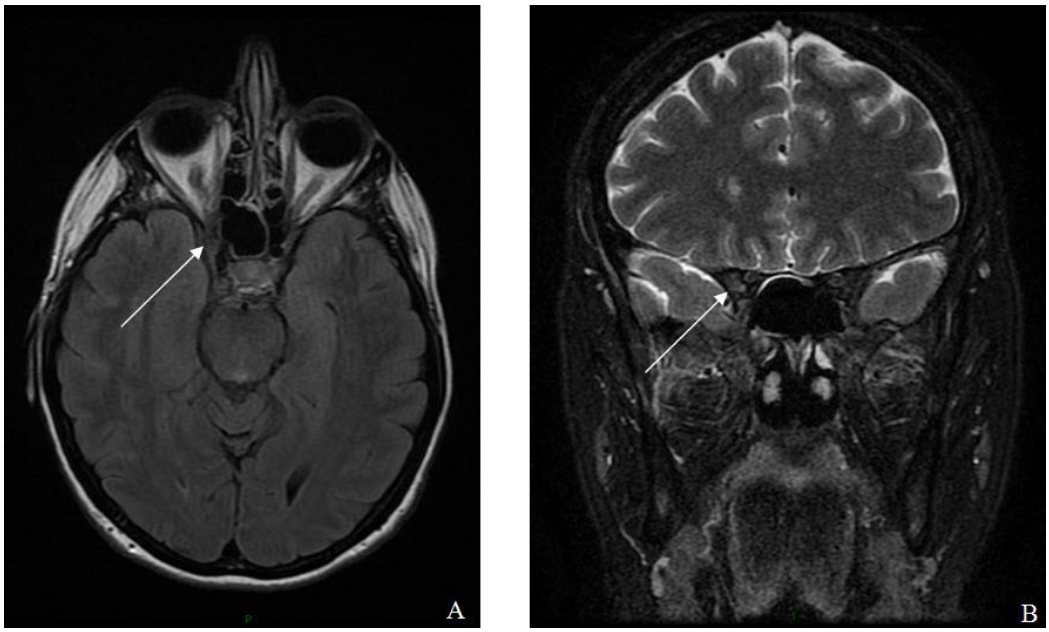
Verona E Botha, Kay F Evans

Clinical A 50-year-old female was referred to our eye clinic with a 1-week history of progressively worsening horizontal diplopia and pain in the right maxillary region. She was started on antibiotics the day before for a right upper molar dental abscess, but was otherwise healthy. Her visual acuity was 6/6 in both eyes. She had no abduction of the right eye. Due to this right abducens nerve palsy she was referred for an MRI, which revealed no cause for her symptoms.

On review the following week she had worsening diplopia, paraesthesias in the V1 distribution and right periorbital pain. On examination she had an ongoing right abducens palsy, but had also developed a pupil-sparing occulomotor palsy. Blood and CSF workup were normal. Urgent CT angiography excluded an intracerebral aneurysm and cavernous sinus thrombosis.

A repeat MRI was performed, obtaining the images below.

Figure 1. MRI of the brain (A=axial view. B=coronal view). Showing an asymmetric enhancement in the right mesiotemporal region extending to the cavernous sinus



What is the diagnosis?

Answer—*Tolosa-Hunt syndrome*.

Discussionð Tolosa-Hunt syndrome is a painful inflammatory ophthalmoplegia with cranial nerve palsies localising to the region of the cavernous sinus, superior orbital fissure or orbital apex.[1ï3] It is a rare condition, with incidence estimated at 1 per 1,000,000.[4] It can present at any age[1,4] and shows no gender predilection.[1]

Presentation is usually with unilateral periorbital pain, and motor cranial nerve palsies that may involve the occulomotor, trochlear and/or abducens nerves.[1,2] The pupil may be involved.[1] The optic, trigeminal and facial nerves may also be affected.[1] Nausea may be present, but no other systemic involvement occurs.[1]

The differential diagnosis is wide, as it includes any inflammatory, vascular or neoplastic lesion affecting the cavernous sinus, superior orbital fissure or orbital apex.[1] Therefore Tolosa-Hunt remains a diagnosis of exclusion,[1] as highlighted in the International Headache Societyôs revised criteria for Tolosa-Hunt.[2,5]

Leukocytosis and a raised erythrocyte sedimentation rate has occasionally been reported.[1] CSF examination is usually unremarkable, but may show slightly raised protein levels.[1] MRI is helpful in excluding other causes, but may also show an area of abnormal tissue in the region of the cavernous sinus,[1,2] as was the case in our patient. Biopsy is only considered in cases of rapidly progressive neurological deficits not responding to treatment or persistent abnormalities.[4]

The mainstay of treatment is steroids and a treatment trial may be diagnostic.[1] Our patient improved dramatically after the initiation of steroids, with an improvement in pain and ptosis after just one dose and slower resolution of the ophthalmoplegia. It is important to bear in mind other conditions that may also improve with steroids and can mimic the condition, including sarcoidosis, vasculitis, lymphoma and even neoplasms.[5]

The course of the condition may be unpredictable with recurrences common within months to years of the initial presentation.[1] Our patient had a relapse on the contralateral side when steroids were weaned, requiring an increase in treatment with slower tapering. Residual cranial nerve palsies may persist.[1] Focal radiotherapy has shown some efficacy in cases where relapse has occurred.[6]

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

Response to Professor Richmond's viewpoint article on the End of Life Choice Bill

We write in response to the 4 July 2014 viewpoint article *A critical analysis of the End of Life Choice Bill 2013*.[1] The results (opinions of Richmond who is an active opponent of voluntary euthanasia), and the conclusions, do not stand up to scrutiny.

How Richmond comes to the conclusion that a request for medical help in dying from a competent terminally ill patient or person suffering from an irreversible unbearable illness (e.g. motor neurone disease), requiring two written requests in writing, and examined by 2 medical practitioners with a set of requirements, and strict reporting conditions can be interpreted as óvirtually any person over the age of 18 can receive euthanasiaô is beyond belief!

Loneliness, poor family relationships, ótired of livingô and his long list of possibilities simply do not qualifyð they are not irreversible. Depression needs some comment: most depression is treatable and does not qualify. In very severe depression disordered thinking will rule out a person. There may be an occasional person with intractable longstanding depression who may qualify, but a psychiatrist would be involved. There is only one person who knows whether suffering is unbearableð the patient, e.g. end-stage respiratory failure and breathlessness, inability to move or swallow.

Richmond fails to understand the vast difference between irrational suicide and a medical practitioner assisting someone to have a good death, a person who is going to die anyway, or has unbearable irreversible suffering.

The rules are quite clear regarding medical practitioner involvementð if they do not want to be involved, they have that right without even having to explain their reasons. They are at complete liberty to advise against voluntary euthanasia (VE), and furthermore, as in overseas cases may certify that the person does not meet the criteria. That is completely different from forgery or fraudulent behaviour which perverts the patientôs wishes where they meet the requirements of the Act.

Every applicant has to be competent, make two written requests spaced by at least 7 days, be assessed in full by two doctors, and meet the criteria in the Act. The assertion that there is minimal protection against VE without consent or request is bizarre.

The assertion that reporting of events can easily be circumvented is also strange given that two different doctors are involved and there would be penalties for that. In fact, rather than being the least regulated practice in medicine there are detailed procedures and stringent reporting, far more than in other branches in medicine.

In contrast, what reporting of end-of-life procedures and care exists at the moment? Precisely none, except for the usual completion of records in the patient notes and a death certificate. One can undergo palliative sedation (where the patient is anaesthetised with drugs) and have fluids and food stopped without any outside

reporting. A New Zealand study has already shown that doctors and nurses do hasten death purposely in end-of-life situations.[2]

It is noteworthy that Quebec in Canada has just passed a law called ñAn Act respecting end-of-life careò which is very similar to that intended in Maryan Streetôs ôEnd-of-Life-Choice Billô. One significant addition is in Section 3 (3): ñend-of-life careò means palliative care provided to persons at the end of their lives, including palliative sedation, and medical aid in dying. Reporting requirements include palliative sedation as well as medical aid in dying. The Act may be easily accessed online.[3]

The information used to inform the passing of the Act resulted from a 2-year study by a Select Committee of the National Assembly of Quebec, reported 2012. The Select Committee addressed many issues including palliative care, and the practical and ethical issues involved in VE.

Because they were hearing submissions from a lot of opponents of VE, they visited the jurisdictions where VE was legalised, and talked to opponents and supporters. A summary paper of each jurisdiction is included in their report and contradicts many of the assertions made by David Richmond.

Anybody interested in the truth on the subject should read this document.[3]

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

Falling through the cracks: New Zealand prostate cancer survivors' experiences and views regarding PSA testing

Prostate cancer (PCa) is the most common male cancer in many countries.[1] The 5-year survival rates for PCa are rising, with this thought to reflect improvements in early detection via prostate-specific antigen (PSA) testing and improved outcomes from treatment options.[2] However, there are also risks associated with PSA testing, such as misdiagnosis and harmful side-effects from various treatments that may outweigh any potential benefits, especially for older men.[3]

The aim of this study was to identify and examine PCa survivors' experiences and views regarding PSA testing.

Participants were 8 PCa survivors aged 60 years and older (65.0±6.5 years) who had taken part in a larger qualitative study that examined perceived quality of life and perceived barriers and facilitators for physical activity post diagnosis.[4,5]

Table 1 provides information on participant characteristics.

Table 1. Participant characteristics

Variables	Number (%)
Age (years)	65.0±6.5
Time since diagnosis (years)	1.4 ±0.8
Gleason scores	
6	1 (13%)
7	4 (50%)
9	3 (38%)
Previous prostate-related surgery	5 (63%)
Previous chemotherapy treatment	0 (0%)
Previous radiation treatment	0 (0%)

Note: Data for the Gleason scores, prostate-related surgeries, chemotherapy and radiation treatment includes the absolute number of participants and in parentheses, the percentage of participants. Percentages do not necessarily add to 100% due to rounding errors.

Participants took part in a 70-minute focus group which was audiotaped. Responses were analysed using an inductive thematic approach.[6] Three main themes emerged:

Theme 1: Bad camp and good camp

This theme was related to the concept of two viewpoints that participants referred to as the ódbad campô and ógood camp.ô The ódbad campô was perceived to consist of GPs who followed the Ministry of Health guidelines, which do not support screening asymptomatic men. The ógood campô was perceived to consist of GPs who monitor their middle-aged to older-aged patientsô PSA levels via annual blood tests, and who typically refer their patients to a urologist for further investigation if their PSA exceeds a certain threshold level.

The following quotes illustrate how participants viewed the concept of the ó*bad camp*ô:

> ñThere is two camps. There is the camp that is simply listening to the Government guidelinesé which is effectively discouraging men to go to specialists because radical surgery has got its problems, and they want to minimise that.ò
>
> You've got men's lives being played with on the basis that the statistics say that between...PSA of 3 and let's say 12, 95% haven't got cancer. You've got this 5% falling through the cracks, and I happened to be one of them.ò

The following quotes illustrate how participants viewed the concept of the ó*good camp*ô:

> ñMy doctor.....as soon as he noticed it rising he sent me through to a ... (urologist)é so that was fantastic, he must have been in the good camp.ò
>
> ñMy doctor sent me for blood tests every 12 monthsé he monitored the PSA until it got to 5, I think. He sent me off to the specialist. So I was very fortunate.ò

Theme 2: Early detection

This theme was related to participantsô views on PSA testing as a form of early detection. The following quotes demonstrate these points:

> ñWhy not have a form of screening early so that the surgeons can then present more than one option, as opposed to one option.ò
>
> ñIt might be false, but too bad.ò

Theme 3: Publicity and awareness

This theme related to participants conveying the view that GPs as a group were not providing adequate information to their patients regarding prostate health, which was in contrast to the growing public awareness of prostate health issues through the media.

These perceptions are conveyed in the following quotes:

> ñThere is more publicity given to prostate cancer now.ò
>
> ñAnd this is the travesty! I'm surprised that the doctors, the GPs were not a little bit more progressive and proactive in getting the message across to people.ò

PCa survivors in the present study conveyed that men should receive more balanced information from their GPôs regarding both the risks and benefits of PSA testing. Previous studies[7ï9] that have examined both the views of asymptomatic men and survivors regarding PSA testing, found that men in general were mostly in favour of PSA testing.

In most cases these men felt that such a test would be beneficial in regard to early detection and treatment options. However, the majority were largely unaware of any risks associated with such screening. The men in our study also seemed unaware of any possible adverse events relating to the outcomes PSA testing.

The National Health Committee's 2011[10] inquiry into early detection and treatment of PCa may be a catalyst for Government-based guidelines change regarding patient education and primary care practice for PCa detection and treatment in New Zealand.

Recommendations include providing GPs with relevant information and support in the form of best-practice, including encouraging GPs to advise men who have a strong family history of PCa to have PSA tests from the age of 40 years onwards.

While this study only included responses from a small number of men, the aim was to provide an avenue for survivorsô to voice and discuss their experiences and views regarding a salient, controversial issue.

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Editorial: Outbreak of WW1

Published in NZMJ August 1914;13(54):287.

SINCE the last issue of this journal probably the most devastating war of all time has broken out, and the time has gone by for any academic discussion on the futility and absurdity of war.

We are far removed from the main theatre of hostilities, and it may be that the red tide will never roll far south; but no matter what happens as loyal sons of the Empire, our wish is similar to the cry of Cato, *"Ceterum censeo Carthaginem esse delendam"*ð for the rest, we vote that the power of Germany must be destroyed.

As Britons, we deplore that a great Power should have driven us reluctantly to the stern arbitrament of the sword, but we believe our hands are clean. The blood that will be shed is on the heads of the war party that fashioned an army and fleet for aggression, and called on High God to witness that they are the instruments of His destiny.

Great and manifold have been our own national sins, and, in humility, we beseech the same omnipotent Throne to crown our arms with victory, and send us speedily peace. The sacrifice of a hecatomb of lives, the loss of treasure, the cessation of scientific and commercial progress, the shrieks of widows and the cries of orphans, and Hell in a world created for nobler purposesð these are but a few of the fruits of war.

Perhaps, some sleepless night, the War Lord of Europe may conjure up the ghosts of soldiers, who will have died for him, filing past in thousands, and casting upon him from Eternity an earnest and upbraiding look; but to the ordinary mind, even of one who has seen with his own eyes some of the horrors of war, the concentrated miseries of this campaign can only be very inadequately conceived.

The blatant frenzy of a noisy jingoism will not carry us through the troublous times ahead, but the Homeland, in quietness and confidence, has faced the storm, and here in New Zealand we will not be behind in calmness, in determination, and loyalty.

Thousands of our own profession will serve under the flag, and we doubt not they will maintain and increase the great traditions of the Royal Army Medical Corps, and our medical brethren in New Zealand are sacrificing their practices on the altar of patriotism.

It will become the duty of every division of our Association to safeguard the interests of those of our members who go to the wars, and something-of this kind has been already accomplished in Wellington.

It is within a year of the centenary of Waterloo, and it says little for the progress of mankind that the same scenes are now to witness a yet more terrific holocaust:

> "And Ardennes waves above them her green leaves,
> Dewy with Nature's tear-drops, as they pass,
> Grieving, if aught inanimate e'er grieves,
> Over the unreturning brave,ð alas!
> Ere evening to be trodden like the grass
> Which now beneath them, but above shall grow
> In its next verdure, when this fiery mass
> Of living valour, rolling on the foe,
> And burning with high hope, shall moulder cold and low."

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Paracetamol and serious skin reactions

Paracetamol is widely used as an analgesic and antipyretic. This is because it is effective and safe if used appropriately. It is known that accidental or intentional overdose may cause very serious liver damage. Because of its safety profile it may be bought over-the-counter in pharmacies and supermarkets.

It is less well known that it can rarely cause very serious skin reactions. In 2013 the US Food and Drug Administration (FDA) issued a drug safety communication warning that paracetamol can, in rare cases, cause serious skin reactions, also known as Severe Cutaneous Adverse Reactions (SCARs); these include Stevens Johnson Syndrome, toxic epidermal necrolysis, acute generalised exanthematous pustulosis, and erythema multiforme.

Prescriber Update 2014;35:28.

Adverse drug events causing hospital admission

Adverse drug events (ADE) cause a significant burden to individual patients and healthcare systems.

Studies in Europe and Australia have found up to 22% of hospital admissions are caused or contributed to by ADE. This prospective study from Christchurch seeks to elucidate the New Zealand situation.

All patients (336) admitted under a general medical team over a 20-week period were reviewed. 28.8% (96) patients were identified as having ADE. In 65 patients (19.3%) the ADE was the cause of their admission. In 31 patients (9.2%) the ADE contributed to their admission. If intentional overdoses and recreational drug use were excluded, ADE patients were significantly older at 72.4 years than non-ADE patients.

Vasodilators, psychotropic drugs and diuretics were the commonest culprits. The most frequent problems were postural hypotension, vasovagal syncope, intentional drug misuse and dehydration.

Internal Medicine Journal 2014;44:633ï 638.

Impact of antibiotics on growth in children in low and middle income countries

In a recent abstract (*NZMJ*, 6 June 2014) we noted that the use of antibiotics as growth promoters in livestock has recently come under critical scrutiny. This meta-analysis looks at trials concerning the use of antibiotics as growth promoters in prepubertal children in low and middle income countries.

The researchers found 10 studies involving more than 4000 children. They report that antibiotic use increased height by 0.04cm/month and weight by 23.8g/month. The conclude that evidence for these trials shows that antibiotic use in prepubertal children from undernourished populations leads to clinically relevant growth gains, particularly for weight.

An editorial commentator notes that the researchers speculate the effects may operate through reduction in subclinical infections and beneficial effects on intestinal microbiota. He also notes that such usage may have serious consequences to the individuals and to global populations through the emergence of bacterial resistance.

BMJ 2014:348:g2267 & g2624.

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Desmond Alexander McQuillan

MBChB, DA(RCS,RCP), FFARACS, MRCGP, Dip Obst, FANZCA;1925–2014

Desmond (Des) McQuillan was born in Auckland, New Zealand on 19 August 1925 and died in Auckland on 28 March 2014.

Des, who was an only child, attended Bayfield Primary School and Sacred Heart College Auckland, where he excelled at cricket, rugby and tennis as well as academics. From his early teenage years he decided to study medicine and he completed his medical studies at Otago University Medical School, Dunedin, in 1950.

In December 1949 in Wellington he married Judith Foden, a home science graduate, whom he had met while at university. After house surgeon years at Palmerston North Hospital (1950) and Wairau Hospital, Blenheim (1951), Des, as a consequence of Department of Health bursary bonding obligations, was given the choice of placement as a General Practitioner in either Ruawai or Rawene in Northland.

He chose Ruawai where he was the only doctor for a large area from south of Dargaville to Matakohe. He travelled many thousands of miles annually on the Kaipara District's notorious unsealed roads. Patient isolation meant at times rides on horseback or boat were required. After a few years he acquired his own runabout boat to visit isolated patients and experience catching the then well stocked Kaipara harbour fish resources. Concern for his patients having to travel out of district for X-rays saw him acquire and operate his own X-ray machine and plastering facilities.

As a sole practitioner servicing the local rural community during the peak baby boom years, obstetrics became a significant part of his practice. The two local maternity hospitals his patients attended were in Te Kopuru and Paparoa. The quickest way to the hospital at Te Kopuru, just south of Dargaville, was by car ferry across the Northern Wairoa River. Night deliveries thus involved disturbing the local ferryman with whom he developed a great cooperative relationship.

The opening of a new hospital in Dargaville saw Des kindle his interest in anaesthetics as he attended to a weekly afternoon theatre clinic. Throughout his time in Ruawai he greatly appreciated the collegial support from professionals in Dargaville, especially Dr Maurice Matich to whom he entrusted his wife's obstetric care.

Des was a great GP, competent, kindly and well integrated into the local community. The large number of MŪori families that he served were devoted to him.

After 13 years Des, Judith and by this time their 4 children left Ruawai for Auckland where Des worked as an anaesthetics registrar at the Auckland Hospital Boardôs Auckland, National Womenôs, Greenlane, and Middlemore Hospitals. In 1965 he decided to further pursue his interest in anaesthetics which required a shift for the family to the UK. While completing his speciality exams he worked at hospitals in London and Shotley Bridge Hospital in County Durham. During his time in the UK he was a very enthusiastic and eloquent spokesman for the beauty and charm of his homeland and encouraged others to visit, some of whom stayed in New Zealand.

In 1967 on his return to New Zealand, Desô interest in obstetric anaesthesia led him to take up a position as a senior anaesthetist at National Womenôs Hospital (NWH), Auckland. These were the days of international renown for National Women's and Des' colleagues included Professors' Liggins, Liley and Bonham.

In 1969 Auckland University offered a Diploma of Obstetrics and Des (who claimed he got the job of delivering many a baby because of the late arrival of the obstetrician) was one of the inaugural successful recipients. He later helped with the tutoring for this diploma.

His special interest was epidural anaesthesia for which he perfected the technique both left- and right-handed. Des and Dr Ian Hutchison (Hutch) popularised epidural anaesthesia for obstetrics at NWH to such an extent the workload risked being unsustainable at the staffing level of the time. Hence he and Hutch also explored the possibilities of using a neuroleptic technique for pain relief in labour. Des continued with this research trialling various drugs over the next few years and in early 1976 he presented his findings to the World Conference in Anaesthesiology in Mexico.

Des became Senior Anaesthetist-in-Charge at National Womenôs Hospital in 1973 and continued in this position until August 1976, when at the age of 50 he suffered a stroke while at work in the NWH operating theatre. The left-sided paralysis from the stroke prevented him from working in anaesthetics again but he did return to doing some part-time GP work. Des was not one to complain but he did have a number of health setbacks in his life including hepatitis during his matriculation year, meningitis (twice), pneumonia, broken vertebrae and a congenital kidney condition.

Des was a great human being as well as a doctor. He was what one would call a real kiwi bloke with all the positive things that implied: simplicity, a strong sense of equality, openness, compassion for the underdog and the sick, and a humble, well concealed competence as a doctor.

His well-stocked medical bag went with him everywhere. Even when holidaying at his favourite camping site at Tauranga Bay, Northland he held a free daily clinic and responded to many an emergency. Outside of medicine he was involved with tennis, playing for Otago and Northland, rugby playing and coaching, and lawn bowls, spending time as the president of Auckland Bowling Club.

In his later years grandchildren became a major interest for him and he was immensely proud of all their sporting and academic achievements. He was also very proud of discovering his MŪori heritage in the early 1990s and took great pleasure in meeting his MŪori relatives, attending the National MŪori Tennis Championships with some of his grandchildren and visiting the marae in Northland, which ironically is very near Rawene where he had originally been offered a GP position.

In retirement he continued to follow the new trends in medicine, read the latest ANZCA magazine from cover to cover and took an interest in politics which included putting a submission in for the review of the MMP parliamentary system in New Zealand.

Des died from a ruptured abdominal aortic aneurysm. Fully aware of the situation he was in over his last few hours he remained the caring family patriarch. Des is survived by his wife Judith, children Janet, Michael, Helen and Linda, 7 grandchildren and one great grandchild.

This obituary was written by familyð Linda O'Rourke, Michael McQuillan, Helen Birse, Janet Sweeneyð with contributions from Professor John Werry and Dr Glyn Richards.

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Professional Misconduct (Med10/152P)

Dr Suresh Kumar Vatsyayann

Charge—On 7ï 11 and 14ï 17 February 2011, the Health Practitioners Disciplinary Tribunal (the Tribunal) considered a charge laid by a Professional Conduct Committee against Dr Suresh Kumar Vatsyayann (The Doctor), medical practitioner of Hamilton.

The charge alleged The Doctor was guilty of professional misconduct in that he:

1. permitted the enrolment of patients at the clinic without the patient being aware of the enrolment and /or without the patient giving informed consent to the enrolment.
2. permitted or was responsible for allowing a consultation with a patient to be undertaken in the same room as another patient which breached the privacy of the patients concerned.
3. permitted or was responsible for allowing his wife who was registered and/or unqualified to:
    - carry out cervical smears;
    - remove intrauterine devices;
    - give vaccinations;
    - give Depo-Provera injections;
    - conduct herself in ways so as to represent she was a qualified nurse, including allowing other staff to represent the same.
4. was responsible for inaccurate clinical records which showed that he had provided treatment or performed a clinical procedure when he had not.

Finding and First Penalty Decision—The Tribunal found each of the four particulars were established and that each individually constituted professional misconduct. The Tribunal found that there was a very serious pattern of multiple breaches, which could only be described as grave.

On 21 April 2011 the Tribunal reconvened for a penalty hearing. The Tribunal ordered cancellation of The Doctorôs registration. The Tribunal further ordered that The Doctor be censured and pay costs of $256,000.

Appeal—The Doctor appealed both the Tribunal finding and the penalties imposed by the Tribunal to the High Court. The appeal decision was delivered on 25 May 2012. The Court dismissed the appeal relating to the Tribunalôs finding of professional misconduct and the censure of The Doctor by the Tribunal. The Court

allowed the appeal relating to cancellation of the appellantôs registration and payment of costs and referred the matter of penalty back to the Tribunal for a rehearing. The Court ordered that its order allowing the appeal relating to cancellation and costs was not to take effect until the day of the Tribunal penalty rehearing.

Final Penalty Decision—On 13 September 2012 the Tribunal issued the final penalty decision which ordered that The Doctorôs registration should be cancelled and he pay costs of $150,000.

The full decision relating to the case can be found on the Tribunal web site at www.hpdt.org.nz
Reference No: Med10/152P

THE NEW ZEALAND MEDICAL JOURNAL
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Professional Misconduct (Med10/170D)

Dr Suresh Kumar Vatsyayann

Charge—On 9ï 11 May, 30 Mayï 1 June and 31 Octoberï 2 November 2011, the Health Practitioners Disciplinary Tribunal (the Tribunal) considered a charge laid by a Professional Conduct Committee against Dr Suresh Kumar Vatsyayann (The Doctor), medical practitioner of Hamilton.

The charge alleged The Doctor was guilty of professional misconduct in that he:

1. failed to adequately diagnose the cause for iron deficiency anaemia in his patient;
2. failed to adequately monitor and/or manage treatment for iron deficiency anaemia in his patient;
3. failed to adequately follow up signs of pathology in his patient;
4. was responsible for inaccurate clinical records which showed that he had provided treatment or performed a clinical procedure when he had not;
5. diagnosed gastritis without performing an abdominal examination on the patient or auscultating his patient; and/or
6. failed to adequately document care of his patient.

Finding—The Tribunal found, in regard to particular 1, although technically The Doctor failed to make an adequate diagnosis it was of the view that the failure was not sufficient to justify disciplinary intervention. The Tribunal found particulars 2, 4 and 5 were not established on the facts of the case. The Tribunal found in regard to particular 6 that while the documentation of his care of his patient was not adequate and amounted to misconduct it was not of such severity as to justify disciplinary sanction.

The Tribunal found particular 3 was established. The Tribunal considered the failures on The Doctorôs part amounted to malpractice, negligence and have brought or were likely to bring discredit to the medical profession.

Penalty—The Tribunal ordered that the following conditions will apply should he ever seek to commence practice as a medical practitioner for a period of three years from the commencement of practice (on the assumption that his name is properly on the register) and having had at his cost a psychological assessment as directed and approved by the Medical Council before he resumes practice as a medical practitioner:

1. undergo such ongoing clinical psychologist treatment and assistance and other rehabilitation steps as are required by the Medical Council;
2. practise in a group practice approved by the Medical Council, which practice must include a vocationally registered medical practitioner; and

3. practise under a supervision plan approved by the Medical Council and under the supervision of a supervisor approved by the Medical Council, the cost of the supervision to be met by The Doctor.

He was ordered to pay costs of $106,190.

Appeal—The Director of Proceedings appealed against two aspects of the Tribunalôs decision. First, the Director challenged the Tribunals finding that The Doctor did not diagnose gastritis. Secondly, the Director argued that the Tribunal was wrong to find that The Doctorôs failure to adequately document his care of the patient did not justify a disciplinary sanction. The Director further sought an order increasing the overall penalty imposed.

On 5 October 2012 the Court issued a judgment that dismissed the Directorôs appeal and The Doctorôs cross appeal.

The full decision relating to the case can be found on the Tribunal web site at www.hpdt.org.nz
Reference No: Med10/170D