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This Issue in the Journal

A nutritional analysis of New Zealand military food rations at Gallipoli in 1915: likely contribution to scurvy and other nutrient deficiency disorders
Nick Wilson, Nhung Nghiem, Jennifer A Summers, Mary-Ann Carter, Glyn Harper

Amongst New Zealand soldiers in Gallipoli in 1915 there were reports of poor food quality and cases of scurvy. To further understand this problem, we analysed the foods in the military rations for 1915 using food composition data on the closest equivalents for modern foods. The nutrient analysis suggested that the military rations were below modern requirements for vitamins A, C and E; potassium; selenium; and dietary fibre. If military planners had used modest amounts of the canned vegetables and fruit available in 1915, this would probably have eliminated four of these six deficits. In summary, there is now both historical and analytic evidence that the military rations provided to these soldiers were nutritionally inadequate. These deficits are likely to have caused cases of scurvy and may have contributed to the high rates of other illnesses experienced at Gallipoli. Such problems could have been readily prevented by providing rations that included some canned fruit or vegetables (e.g., as manufactured by New Zealand at the time).

The Northland Emergency Meningococcal C Vaccination Programme
Clair Mills, Liane Penney

In 2011, there was a community outbreak of meningococcal C disease in Northland. This serious disease causes meningitis and septicaemia (blood poisoning) and can be rapidly fatal. An emergency vaccination programme was quickly implemented, aiming to vaccinate 85% of children and youth aged 1-<20 years, the highest risk age group. Over 32,000 children and youth were vaccinated (73%) in three months. “Walk in, no appointment needed” community clinics run out of mobile vans, shops and other sites were critical in ensuring good access and equitable coverage, especially for Maori families and youth. Although some general practices achieved very high coverage, there were significant inequities in general practice for children under 5 and teenagers. A range of services are needed to reduce access barriers.

Unanswered questions, the epidemiology of a community outbreak: meningococcal C disease in Northland, New Zealand, 2011
Clair Mills, Kerry Sexton, Philip Carter

In this study we describe the epidemiology of a community outbreak of Meningococcal C disease in Northland in 2011, and national trends in serogroup C disease in New Zealand. In 2011, the rate of group C meningococcal disease for the population in the Whangarei district aged less than 20 years was 27.6 cases per 100,000 population (6 cases) compared with 17.6 cases per 100,000 population under 20 years (8 cases) in the Northland District Health Board (DHB). All except one case
were under 20 years of age. 33% of these cases died. Nationally the rate of meningococcal C disease has fluctuated over the last decade, with an increasing trend apparent since 2007. There has been a noticeable increase over the last 3 years of group C cases infected with the C:P1.5-1,10-8 strain (including all of the Northland cases). This strain has also been associated with a higher case fatality rate (16% in the period 2007-2011). Meningococcal C disease in New Zealand, although still less common than group B, is poorly understood. The relationships between carriage, invasive disease and community outbreaks deserve greater study. Active monitoring of surveillance data is warranted to ensure timely funded introduction of the highly effective meningococcal C conjugate vaccine on to the national immunisation schedule when appropriate, given increasing disease rates, the high case fatality rate and significant Māori non-Māori inequities in disease incidence.

The epidemiology of acute rheumatic fever in Northland, 2002–2011
Audrey Robin, Clair Mills, Roger Tuck, Diana Lennon

We carried out an audit of Acute Rheumatic Fever cases in Northland in the period 2002-2011. This was to establish an accurate baseline against which we will be able to assess the impact of current prevention efforts (for example the school throat swabbing projects). Northland has very high rates and there are large inequalities in this illness: 95% of cases in Northland are in Māori children and young people, most between 5-15 years of age, and nearly 90% live in the least well off one-third (NZDep 8-10) of communities in our DHB. There appeared be a trend upwards in the number of cases in the period 2002-2011.

Nurse-led school-based clinics for skin infections and rheumatic fever prevention: results from a pilot study in South Auckland
Sarah Gray, Diana Lennon, Philippa Anderson, Joanna Stewart, Elizabeth Farrell

Rates of rheumatic fever and of hospitalisations for serious skin infections are high amongst New Zealand children. These conditions can lead to significant long-term sequelae if left undiagnosed and untreated but are potentially preventable. This paper looks at how acceptable and feasible it is for nurses to run clinics in low decile primary schools to diagnose and treat children with skin infections and streptococcal sore throats. This approach has the potential to reduce the number of children hospitalised for skin infections and to reduce the number of children developing rheumatic fever if it is run out on a larger scale.

Temperature management in haematology patients with febrile neutropenia: a practice survey
Robert Weinkove, Jennifer Clay, Catherine Wood

Infections are a common problem after chemotherapy, and can be life-threatening. Patients with infection after chemotherapy must be promptly treated with antibiotics. These patients often have high fevers. This paper is a survey of haematologists (blood specialists) in New Zealand, which asks how they manage fever in patients who have
had chemotherapy. The reason for asking this question, is that some researchers believe that having a high fever helps to eradicate an infection, and that measures to reduce fever (such as taking paracetamol) might be detrimental. However, we also know that paracetamol makes patients more comfortable. We found that the way clinicians manage fever among their patients is very variable, but that most do give their patients treatments to reduce their temperature. Nurses have a preference for intervening at a lower temperature than doctors do. The variability in clinical practice we found reflects the fact that there is no good evidence (from clinical trials) to guide us. In our survey, most clinicians supported the idea of a randomised trial to show whether lowering temperature during post-chemotherapy infections is beneficial or not.

The influenza pandemic of 1918–1919 in two remote island nations: Iceland and New Zealand
Jennifer A Summers, Nick Wilson, Michael G Baker, Magnús Gottfredsson

This article compares the epidemiological characteristics and public health interventions employed during the 1918-19 influenza pandemic by two remote island nations, from opposite sides of the globe: Iceland and New Zealand. Similar patterns of pandemic waves were found for both nations, although Iceland experienced a higher mortality rate overall and employed several public health interventions successfully.
Nutrition and disease: lessons learnt from Gallipoli

Geoffrey W Rice

The link between nutrition and good health is so fundamental that we take it for granted most of the time, vaguely hoping that variety means a balanced diet as we indulge ourselves in another rasher of breakfast bacon or that extra helping of pavlova with cream and kiwifruit.

The link between nutrition and deficiency-diseases is also pretty obvious. Everyone knows about scurvy and ascorbic acid, and Captain Cook’s careful attention to diet, which made his crews perhaps the healthiest of all those on long voyages of exploration in the 18th Century. That success was largely the result of observation and experiment, long before the science behind it was fully explained.

Historians interested in population increase in the past have long been aware of the complex interrelationship between nutrition and disease, but the links between nutrition and epidemic diseases have been more elusive. Many other factors come into the equation, which are often difficult or impossible to measure precisely.

In his 2004 doctoral thesis on 1840s Edinburgh, Neil MacGillivray attempted to show how poor nutrition made recent migrants to the city more vulnerable to outbreaks of typhoid and cholera, but found that overcrowding, poor sanitation and lack of potable water were probably just as important contributors to mortality. Stephan Curtis on the other hand examined the social and economic contexts of three scarlet fever epidemics in Sweden in the period 1860-90 and found a convincing correlation between food shortages from bad harvests, poor nutrition during pregnancy, and compromised immune-competence of young children. The impact of major epidemic diseases such as influenza, most obviously in the so-called ‘Spanish Flu’ of 1918–19, may well have been magnified by the vulnerability of some sections of the population from poor nutrition.

Svenn-Erik Mamelund’s detailed analysis of social class, household wealth and influenza mortality in Norway has shown that poorer people were probably more at risk of dying in this pandemic than better-fed neighbours, yet the evidence from New Zealand tends to suggest that infection was indiscriminate and that big strong men were more likely to die than skinny asthmatics, thanks to overreaction by their immune responses. As several doctors observed at the time, victims drowned in their own secretions.

An article in this issue of NZMJ by Nick Wilson and colleagues on the nutrition of New Zealand soldiers at Gallipoli in 1915 is a salutary reminder to historians that what people ate directly affected their state of health, and may therefore help to explain the outcome of larger events.

Far from home, the ANZACs (Australian and New Zealand army troops) on Gallipoli were supplied by the British Army, and front-line troops were expected to feed themselves on the standard fare of tinned ‘bully-beef’ and biscuit, tea and sugar, jam
and condensed milk. However, the heat and the flies made feeding a difficult business. Flies were especially attracted to jam, and as they may have been previously feeding on corpses, cross-infection was almost inevitable.

Lack of clean water and sanitation in the trenches meant that diarrhoea and dysentery were commonplace, for the better-fed officers as well as the troops. The lack of fresh fruit and vegetables, for months on end, caused serious deficiencies in vitamin A and vitamin C, which in turn caused night-blindness and scurvy.

These troops were seriously under-nourished, yet they showed remarkable resilience and bravery under appalling conditions. One wonders what they would have achieved if they had been fed on the steaks and ice-cream of a modern US army field-kitchen. On the other side of no-man’s land, the Turkish soldiers enjoyed a diet remarkably similar to that of the legions of ancient Rome: wheat bread, olive oil, nuts and dried fruits.

Gallipoli was a hastily-improvised campaign, and problems of supply had not been fully thought-through. Nor had the likelihood of heavy casualties. The British high command expected this to be a quick success, and had not planned for the evacuation of large numbers of wounded men.

The New Zealanders wounded at Gallipoli were scattered across more than 100 British military hospitals in the East Mediterranean and many lost touch with their families back home. The New Zealand government sent a cabinet minister, Heaton Rhodes, on a special mission to visit them and ensure that their delayed mail reached them from Egypt. As the months went by, supply problems on Gallipoli were resolved and the food improved somewhat, yet the importance of fruit and vegetables was not fully realised. Tinned fruit and peas from New Zealand would have made a significant difference, as the article in this issue argues, but the logistics of shipping and distance made this an impossible option at the time.

Lessons were learned, however slowly, and the New Zealanders in France in the later years of the First World War were far better fed than the ANZACs on Gallipoli. Colonel Stewart of the Canterbury Regiment recalled the ‘unfailing quantity and variety’ of the rations sent up to the line from well-organised quartermasters’ stores and kitchens.

Thanks to recent work by Rachel Duffett, much more is now known about the provisioning and nutrition of soldiers on the Western Front of Europe. The military approach to feeding was based on calories and the calculated energy values of various foods, as ‘fuel for the human motor’. The target of about 4000 calories per day was very similar to that adopted by most modern armies, but it was heavy in fats and carbohydrates.

The standard meat and potato stew made famous by its brand name ‘Machonochie’ was hard to digest, and as the body slowed gastric emptying to allow more time to digest the fat, troops suffered from constipation and that in turn led to bleeding piles.

Lack of ascorbic acid gave rise to the early stages of scurvy, most notably bleeding gums, loss of teeth and sore mouth, besides causing boils and impairing wound-healing.
The lessons of the past are easily forgotten in a world where few people read history books and prefer instead to rely on the History Channel or Time Team for their brief glimpses of the past. Today’s main concern in the field of nutrition and disease is the growing problem of obesity as a cause of chronic non-communicable diseases such as diabetes, cancer and cardiovascular illness.\(^\text{10}\)

The World Health Organization estimates that by 2020 two-thirds of the global burden of disease will be attributable to such chronic diseases, most of them strongly associated with diet. Yet as MacGillivray has shown for Edinburgh in the 1840s, many other factors may contribute towards disease mortality.

Poverty, poor housing conditions and inappropriate nutrition are still major issues for public health in some parts of New Zealand let alone the South Pacific and the Third World.

Competing interests: Nil.

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Meningococcal disease in New Zealand

Graham Mills, Anita Bell

Over the last two decades, New Zealanders have become very familiar with the devastating effects of meningococcal disease, specifically the meningococcal serogroup B epidemic.

Over the 15-year period from 1991 to 2006, almost 250 New Zealanders (80% younger than 20 years) lost their lives. We became familiar with Charlotte Lucy Cleverley-Bisman who survived the disease but with severe disabilities.

An effective public health tool, the “tailor-made” MeNZB vaccine, was developed to combat the deadly epidemic and an extensive campaign delivered the vaccine between 2004 and 2006. The vaccine effectiveness, based on two differing methodological approaches was subsequently shown to be 73% and 80% (consistent with the 70–87% effectiveness predicted based on other serogroup B vaccines in 2004).

Unvaccinated individuals were 3.7 to 6 times more likely to develop disease than vaccinated individuals. The good news was that the B epidemic came to an end. Yet despite the proven vaccine effectiveness, debate occurred as to whether the 200 million dollar vaccination campaign was money well spent. “Hindsight is always easier than the dreadful moment of decision” are words by the novelist Richelle Goodrich that remind us of the dilemma faced by health authorities in 2001 (which was subsequently shown to be the peak) as the epidemic continued to unfold.

2011 saw the Northland region of New Zealand face a smaller, but no less significant dilemma, this time caused by serogroup C meningococcal disease. During the winter of 2011, a significant number of cases of group C disease were reported in this region associated with three deaths.

The article by Mills and Penney in this edition of the NZMJ details the success of a rapidly implemented vaccination programme for cluster control of this mini-epidemic. The achievement of 34,000 vaccinated young people and an overall coverage of 73% in twelve weeks should be congratulated and shows what can be achieved with collaboration and focus. Fortunately, public health were able to call on a highly effective (estimated to be 97% in adolescents) vaccine already in existence to control this cluster.

This article is a timely reminder, even with the decline of the MeNZB epidemic, of the serious harm meningococcal disease can cause to affected individuals, families and communities. New Zealand rates, although more than five times less than the peak years, continue to be higher than other developed countries.

Meningococcal C has now overtaken serogroup B as the strain accounting for the majority of deaths in New Zealand (10 of the 13 deaths in 2011). Although public health units will continue to “fire-fight” when clusters of serogroup C disease reach predefined “threshold” levels, has the time come to now include meningococcal C
vaccine, a highly effective public health intervention, into NZ’s immunisation schedule? Cluster control by definition is more focused, but considerably more expensive per vaccinated person.

The introduction of such a vaccine into the routine immunisation schedule has been proven to be safe and effective with Australia introducing it into their schedule in 2003 following the highly successful experience of the UK in 1999. As a result they are both living with a much decreased burden of C disease than that which currently exists within New Zealand.

The clinical diagnosis of meningococcal disease is often fraught with difficulties as highlighted in the recent media reports surrounding the death of 4th year medical student Zachary Gravatt in 2009 from meningococcal C disease. Vaccine prevention is a pivotal part of the answer to prevent more lives being lost.

Competing interests: Nil.

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A nutritional analysis of New Zealand military food rations at Gallipoli in 1915: likely contribution to scurvy and other nutrient deficiency disorders

Nick Wilson, Nhung Nghiem, Jennifer A Summers, Mary-Ann Carter, Glyn Harper

Abstract

Background Amongst New Zealand soldiers in Gallipoli in 1915 there were reports of poor food quality and cases of scurvy. But no modern analysis of the military food rations has ever been conducted to better understand potential nutritional problems in this group.

Methods We analysed the foods in the military rations for 1915 using food composition data on the closest equivalents for modern foods. We compared these results with other plausible diets and various optimised ones using linear programming.

Results Historical accounts provide evidence for poor food quality supplied to these soldiers. The nutrient analysis suggested that the military rations were below modern requirements for vitamins A, C and E; potassium; selenium; and dietary fibre. If military planners had used modest amounts of the canned vegetables and fruit available in 1915, this would probably have eliminated four of these six deficits. The results from the uncertainty analyses for vitamin C (e.g., 95% uncertainty interval [UI]: 5.5 to 6.7 mg per day), was compatible with the range known to cause scurvy, but the UI for vitamin A intake was only partly in the range for causing night blindness. To indicate the gap with the ideal, an optimised diet (using foods available in 1915), could have achieved all nutrient requirements for under half the estimated purchase cost of the 1915 military rations.

Conclusions There is now both historical and analytic evidence that the military rations provided to these soldiers were nutritionally inadequate in vitamin C, and probably other nutrients such as vitamin A. These deficits are likely to have caused cases of scurvy and may have contributed to the high rates of other illnesses experienced at Gallipoli. Such problems could have been readily prevented by providing rations that included some canned fruit or vegetables (e.g., as manufactured by New Zealand at the time).

The upcoming centenary events concerning World War One (WWI) provide an opportunity to consider the historical lessons from this conflict—including those related to nutrition and military planning. Part of this conflict involved the New Zealand Expeditionary Force in a multi-country campaign on the Gallipoli Peninsula in Turkey. This campaign ran from April 1915 to January 1916, at which time a withdrawal from the peninsula occurred.\(^1\) There were an estimated 7991 casualties (57% of the NZ military personnel) and 2779 died.\(^2\) At least 200 of the deaths were from infectious diseases such as dysentery and typhoid\(^2\).
In addition, the official New Zealand medical history of WWI refers to reports of scurvy among the troops (in three places in the report\(^3\) (p66, 106, 123)). Furthermore, as Christopher Pugsley notes in his seminal account of New Zealanders at Gallipoli,\(^1\) (p346) most of these soldiers ended the campaign too sick and too weak to continue soldiering.

The importance of feeding an army properly has long been recognised as critical to its functioning. As Napoleon Bonaparte acknowledged: an army really does march on its stomach.\(^4\) (p274) Yet it is well known that military personnel have often suffered from nutritional deficiencies.

For example, in the American Civil War, scurvy was diagnosed in 47,000 Union troops and “…by physicians in the field from all theatres of action throughout the war”.\(^5\) Such cases of scurvy were often successfully treated with the provision of fresh vegetables. Scurvy was a major problem for both armies in the Crimean War\(^5\) and there was evidence for it in a number of other historical military settings such as Chinese garrisons and US military outposts.\(^6\)

Evidence of vitamin A deficiency (reports of “night blindness”) was also reported for American Civil War troops on both sides and especially during the last year of the war.\(^5\) Other work considered night blindness in an Austrian naval crew in 1857-59, which was successfully treated with the provision of ox liver.\(^7\) Similarly, in 1863, night blind soldiers in a Paris garrison were successfully treated with cod-liver oil.\(^7\)

Given this background, we examined historical accounts and performed a retrospective analysis of military rations provided to the New Zealand troops involved in the Gallipoli campaign in 1915.

Methods

**Historical context**—To provide context for food supply and consumption by military personnel in Gallipoli, we examined a number of documents written by contemporary authors,\(^3\) 8-10 and by subsequent historians and researchers\(^1\) 2 5 11-13 (including one of us\(^14\)).

**Scenario development**—Table 1 details the specific scenarios we considered which included replicating the actual military food rations as best as possible (Scenario “R-A”) but also to describe three possible alternatives. These were: (i) to slightly improve the amount of food variety in the 1915 rations by providing less corned beef and more fruit and vegetables (Scenario “R-V”); (ii) to achieve modern nutritional recommendations for the lowest cost (optimised dietary pattern, Scenario “R-O”); and (iii) to perform the latter but with more food variety (Scenario “R-OV”).

We suggest that it is very plausible that increased food variety and access to more vegetables and some fruit would have been desired by the soldiers (Scenario R-V), and that military planners could have considered this given prior evidence for problems with scurvy in the American Civil War, Crimean War and other preceding military situations (see Introduction). Nevertheless, it is acknowledged that the more optimised scenarios (R-O and R-OV) are completely hypothetical given the limitations of nutritional science in 1915.

In Table A1 (see Appendix 1) we list the actual rations as used by the New Zealand Expeditionary Force and documented on 13 April 1915.\(^3\) (p345) The “nearest equivalent” modern foods used in the various scenarios are detailed in Tables A2 and A3.

**Nutrient requirements and data used**—For the scenarios involving optimisation of nutrients (R-O and R-OV in Table 1) we generally used the modern day “estimated average requirements” (EARs) of nutrients per day for adult men which are based on values set for Australia and New Zealand.\(^15\) But we also considered current nutrient intakes for men from the New Zealand Adult Nutrition Survey (NZANS).\(^16\) Further details are provided in Table 3. For food composition data we used the 2012 “New Zealand food composition database” (http://www.foodcomposition.co.nz/foodfiles).
Food prices—To estimate the likely costs of the different scenarios, we used current 2011/12 food prices. These came from the official food price index (average monthly data for New Zealand in 2011)\textsuperscript{17} or when not available, supermarket prices (as detailed in Tables A2 and A3 in the Appendix 1).

Approach to mathematical modelling and uncertainty—For the nutrient optimisation we used the simplex algorithm to solve this linear programming problem (see Briend et al.\textsuperscript{18} for a detailed description of the latter). The scenarios were modelled in Microsoft Excel 2010 (Excel Solver, Simplex method). Details of the uncertainty analysis are in the Appendix 1.

Table 1: Specific nutritional scenarios used for the analyses of the actual military rations of 1915 and alternatives

<table>
<thead>
<tr>
<th>Name and aim of specific scenario</th>
<th>Additional details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scenario “R-A”: To replicate the actual military food rations</td>
<td>We used the nearest-equivalent modern foods, see Table A2 for further details of these rations. The modern food nearest equivalents we used are detailed in Table A3.</td>
</tr>
<tr>
<td>Scenario “R-V”: To slightly improve variety of the 1915 rations</td>
<td>In this Scenario we simulated a better attempt by the planners of military rations to approximate the typical NZ diet at the time i.e., more vegetables and fruit. So we halved the amount of corned beef (down to 227 g) and added: 1 cup each of the two cheapest canned vegetables (tomatoes [240 g], and peas [248 g] – both canned products were available in 1915, see Table A3 in the Appendix 1). 1 cup of the cheapest canned fruit (apricots with syrup [240 g], also available in 1915).</td>
</tr>
<tr>
<td>Scenario “R-O”: To achieve modern nutritional recommendations for the lowest cost (optimised dietary pattern)</td>
<td>To achieve this optimised diet with the type of dried and canned foods available at the time, while also: (i) achieving the same level of dietary energy as Scenario R-A; (ii) to ignore the modern recommendations on sodium levels (given the food preservation value of high sodium levels in a military field setting). See text and Table 3 for further details on the specific nutrient recommendations.</td>
</tr>
<tr>
<td>Scenario “R-OV”: As for Scenario “R-O” but with more variety</td>
<td>As for Scenario R-O above but with progressively lowering the maximum amount of the highest individual food item (by weight) until the variety in the original ration increased the number of foods reaching over 50 g per day to up to 10 different foods.</td>
</tr>
</tbody>
</table>

Results

Historical reports of food issues—That the majority of meals on Gallipoli consisted of corned beef (bully beef), hard biscuits and tea is especially significant to this study. For example, the first New Zealand historian of the campaign, Major Fred Waite, noted:

Food was always plentiful (except just after the Great Blizzard in November when stocks ran very low). Tinned meat, jam and hard biscuits and a mug of tea provided 99 per cent of the meals\textsuperscript{10}(p161) (see Figure 1).

On occasion (e.g., near the end of campaign\textsuperscript{2} (p267)) there were some improvements in the food provisioning. As one soldier remarked:

We get very well fed here, considering, but we do miss the fresh vegetables and fruit. We get plenty of rice, tea, sugar, biscuits and bully beef, jam, onions etc and an adequate supply of milk [tinned condensed] and bread. Once or twice we have had an egg each, and yesterday actually had a little fresh mutton served out.\textsuperscript{14}(p202)
Figure 1. Two signallers outside the Divisional Signal Office on the Gallipoli Peninsula (1915) enjoy a meal of bread and jam, washed down with a mug of tea. (The jam did provide a source of Vitamin C but it was not enough.)

Source: Used with permission from National Army Museum, New Zealand (Number: 1992.742)

An impression of some of the difficulties with food and water supply for the New Zealand military in Gallipoli is suggested by these commentators:

After the first two days the battalion had a quiet time in the Walker’s Ridge position. One of the greatest difficulties was in bringing up ammunition, water and food. The track up to the hill, 500 feet above the beach, was very narrow and steep, and exposed to sniping fire from the Turkish trenches; only small loads could be carried by each man, and each trip took a long time.

With all these discomforts, the exhaustion of labour, the strain of unceasing vigil and shell fire, the lack of nourishing food, and little sleep, there was always a shortage of water and the possibility of no water at all. One pint of water a day was the usual issue. (p38)

Other conditions may have limited food intake and palatability:

Owing to the annoyance of the flies some sections did not eat anything but a dry biscuit during the daytime. To eat biscuit and jam in the daytime a man had to keep moving the hand that held the food. (p37)

…. however palatable in a temperate climate, any form of tinned food becomes distasteful in a semi-tropical summer unless ice is available. … Not only was the clothing inadequate but the food and the feeding of the troops was unsatisfactory. (p113)

In commenting mainly on sanitation, the difficult circumstances for food preparation were described:

It is questionable whether any alterations in the dietary – and some were made – could have improved the sanitary situation. The baneful system of individual cooking, then prevalent, would have ruined any ration however good; every man cooked for himself, every dug-out became a midden of fly contaminated food and food refuse. (p114)
Figure 2. A precious water point at Anzac Cove, Gallipoli Peninsula (1915). Two quarts a day was the normal ration for New Zealand soldiers and it had to be used for all purposes. Most went to make tea. As one New Zealand soldier wrote: ‘water is worth its weight in gold here’.

Source: Used with permission from National Army Museum, New Zealand (Number: 2007.550)

Finally, Carbery\(^3\) (p113) appears to support a quote in the final report of the Dardanelles Commission, which stated that: “there was nothing actively injurious to health in the meat; but it was of poor quality, and, from being salt and stringy, it caused some intestinal irritation and so conduced to diarrhoea”.

The bulk of the food diet of the New Zealanders at Gallipoli was supplied via the United Kingdom through its Army Service Corps. Generally it was inferior in quality to similar food that originated from New Zealand. Most of the corned beef was supplied from Argentina and Fred Waite commented that it’s poor quality and the fact that it was served for meals three times a day, “becomes more than the constitution of a New Zealander can stand”.\(^10\) (p163)

When limited sources of food from New Zealand did arrive in at Gallipoli with the latest batch of Reinforcement drafts, it was much sought after and highly prized. This was especially true of the New Zealand army biscuit which was white, easy to eat and pleasant tasting. In contrast, British army biscuits were hard “beyond belief”. New Zealand soldiers at Gallipoli tended to nibble the edges of these hard slabs and throw their centres into No Man’s Land.\(^10\) (p162)
**Nutritional analyses**—The calculated weights of the foods in the different scenarios are shown in Table 2 and the nutrient analysis in Table 3. Of note is that the military rations (Scenario R-A) were far below modern nutritional requirements for vitamin C intake, around a third of the current estimated average requirement (EAR). They were also below the EARs for: vitamin A (33% too low), vitamin E (11% too low), potassium (36% too low), selenium (20% too low) and dietary fibre (36% too low), (Table 3).

Relative to modern requirements, the military diet was also excessively high in saturated fat (3.1 times too high) and sodium (5.2 times higher than the 2300 mg upper limit) (Table 3).

If the planners of military rations had made these rations somewhat more like the typical New Zealand diet in terms of modest amounts of vegetables and fruit (i.e., one cup each per day of peas, tomatoes and apricots used in our “Scenario R-V”), then this would have substantially improved the nutrient quality. In particular it would have eliminated the below EAR intakes of vitamins A, C and E and dietary fibre; improved intake of potassium, but would have made little change to selenium intake. It would also have partly reduced the high intakes of saturated fat and sodium, which is desirable from a health perspective.

If the relative food prices are roughly equivalent between now and 1915, then this alternative ration would have been achieved with only a minor increase in cost above the original ration (NZ$ 11.43 per day vs 11.03, Table 3). However, the higher weight (an extra 501 g/day) would have increased transport costs (Table 2).

In 1915 there was no possibility that a properly “optimised diet” could be developed as there was limited knowledge of nutrition and no capacity to apply mathematical techniques such as linear programming. Nevertheless, it is plausible that military planners could have made moderately more use of a variety of canned and dried foods that were available in New Zealand in 1915 and produced in other developed countries.

It is also of interest to demonstrate how far modern knowledge and methods can allow for optimal military rations to be formulated. Hence we “optimised” the military rations using canned and dried foods available in 1915, and found that only six foods would technically be required to meet all nutritional requirements: bread, flour, cheese, rolled oats, dried peas and canned tomatoes (Scenario R-O in Tables 2 and 3). Indeed, this ration would have been healthier in all of the dimensions shown in Table 3. Furthermore, this ration would have cost under half the cost of the original ration and would be a similar weight.

Expanding the variety of this optimised ration to include ten items (over 50 g each) as per Scenario R-OV would have probably resulted in additional cost compared to Scenario R-O. But this cost was still estimated to be likely to below that of the original ration (i.e., $9.54 vs $11.03 per day). However, the extra weight of the food (466 g/day) would have increased transport costs (Table 2).

The results in Table A3 (see Appendix 1) indicate that cheese was the likely major contributor to vitamin A intake in the rations. For vitamin C it was either jam, followed by dried potatoes (using the USDA food composition data); or corned beef followed by jam (using NZ food composition data).
In the uncertainty analysis (Table 5) the mean vitamin A intake was below half the EAR of 625 mcg and the 95% uncertainty interval (UI) never approached this requirement (using both food composition datasets). Similarly, for vitamin C intake with the mean below a quarter of the EAR and the upper limit of the 95%UI never exceeding a third of the EAR.

Table 2. Foods (with weights in g/day) included in the various daily dietary scenarios with some of these (in Scenarios R-O and R-OV) selected automatically in the optimisation process

<table>
<thead>
<tr>
<th>Food items</th>
<th>Scenario R-A (actual)</th>
<th>Scenario R-V (extra variety)</th>
<th>Optimised diets</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Food in the military rations of 1915 (descending amounts)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corned beef (canned)</td>
<td>454</td>
<td>227</td>
<td>0</td>
</tr>
<tr>
<td>Alternatives: Bread (white)</td>
<td>187</td>
<td>187</td>
<td>330</td>
</tr>
<tr>
<td>Or biscuit</td>
<td>150</td>
<td>150</td>
<td>0</td>
</tr>
<tr>
<td>Or flour (white)</td>
<td>150</td>
<td>150</td>
<td>585</td>
</tr>
<tr>
<td>Bacon</td>
<td>113</td>
<td>113</td>
<td>0</td>
</tr>
<tr>
<td>Cheese (cheddar)</td>
<td>85</td>
<td>85</td>
<td>110</td>
</tr>
<tr>
<td>Jam</td>
<td>85</td>
<td>85</td>
<td>0</td>
</tr>
<tr>
<td>Sugar (white)</td>
<td>85</td>
<td>85</td>
<td>0</td>
</tr>
<tr>
<td>Alternatives:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dried peas</td>
<td>19</td>
<td>19</td>
<td>8</td>
</tr>
<tr>
<td>Or dried beans</td>
<td>19</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>Or dried potatoes</td>
<td>19</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>Salt</td>
<td>14</td>
<td>14</td>
<td>0</td>
</tr>
<tr>
<td>Mustard</td>
<td>1.4</td>
<td>1.4</td>
<td>0</td>
</tr>
<tr>
<td>Pepper</td>
<td>0.8</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td><strong>Additional foods that could have been used in 1915 (see Appendix 1)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peas (canned)</td>
<td>–</td>
<td>248</td>
<td>0</td>
</tr>
<tr>
<td>Apricots (canned)</td>
<td>–</td>
<td>240</td>
<td>0</td>
</tr>
<tr>
<td>Tomatoes (canned)</td>
<td>–</td>
<td>240</td>
<td>353</td>
</tr>
<tr>
<td>Oats (rolled)</td>
<td>–</td>
<td>0</td>
<td>337</td>
</tr>
<tr>
<td>Peaches (canned)</td>
<td>–</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fish (canned)</td>
<td>–</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other**</td>
<td>–</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total food weight g/day</strong></td>
<td>1382</td>
<td>1883</td>
<td>1724</td>
</tr>
<tr>
<td><strong>Total foods ≥ 50 g (N)</strong></td>
<td>6*</td>
<td>11</td>
<td>5</td>
</tr>
<tr>
<td><strong>Total types of food (N)</strong></td>
<td>14</td>
<td>17</td>
<td>6</td>
</tr>
</tbody>
</table>

Notes:
* Six foods when considering the bread, biscuits and flour were describes as alternatives; and similarly for peas, beans or potatoes (see Table A1).
** Includes other canned food: pears, beans, sheep meat and milk powder.
Table 3. Results of estimated daily nutrient intakes for the various scenarios (replicated “actual” and more optimal daily diets)

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>EARS or other target values used as constraints in the optimisation modelling ([RDI] – for comparison)</th>
<th>Scenario R-A (“actual”)</th>
<th>Scenario R-V (extra variety)</th>
<th>Optimised diets</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>R-O</td>
</tr>
<tr>
<td>Price (NZ$ in 2011/12)</td>
<td>Not relevant</td>
<td>11.03</td>
<td>11.43</td>
<td>5.16</td>
</tr>
<tr>
<td><strong>Macronutrients</strong></td>
<td></td>
<td></td>
<td></td>
<td>R-V</td>
</tr>
<tr>
<td>Energy (kJ)</td>
<td>As per Scenario R-A* (except for R-V)</td>
<td>18,213</td>
<td>17,087</td>
<td>18,213</td>
</tr>
<tr>
<td>Protein, total (g)</td>
<td>≥52 [64]</td>
<td>193.0</td>
<td>157.0</td>
<td>160.6</td>
</tr>
<tr>
<td>Polysaturated fatty acids (g), (current intake in men from the NZANS**)</td>
<td>13.1 g (contributing 4.8% of dietary energy in modern NZ men)</td>
<td>9.6</td>
<td>8.4</td>
<td>13.1</td>
</tr>
<tr>
<td>Saturated fatty acids (g)</td>
<td>Using an upper limit of 12% of typical energy intake (i.e., 27.5 g for an intake of 11,450 kJ)</td>
<td>85.5</td>
<td>61.8</td>
<td>27.5</td>
</tr>
<tr>
<td>Sugars (g)</td>
<td>No limit</td>
<td>160.8</td>
<td>239.4</td>
<td>26.8</td>
</tr>
<tr>
<td>Dietary fibre (g)</td>
<td>≥30 (AI)</td>
<td>19.1</td>
<td>36.9</td>
<td>66.8</td>
</tr>
<tr>
<td><strong>Micronutrients</strong></td>
<td></td>
<td></td>
<td></td>
<td>R-V</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>≥840 [1000]</td>
<td>976</td>
<td>1,100</td>
<td>1,678</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>≥6 [8]</td>
<td>20.8</td>
<td>23.4</td>
<td>40.9</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>≥3800 (AI)</td>
<td>2,434</td>
<td>3,414</td>
<td>3,800</td>
</tr>
<tr>
<td>Selenium (mcg)</td>
<td>≥60 [70]</td>
<td>48.0</td>
<td>48.6</td>
<td>60.0</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>Upper limit not applied***</td>
<td>11,925</td>
<td>11,193</td>
<td>2,629</td>
</tr>
<tr>
<td>Vitamin A - total (mcg)</td>
<td>≥625 [900]</td>
<td>416.6</td>
<td>694.4</td>
<td>625.0</td>
</tr>
<tr>
<td>Thiamine (mg)</td>
<td>≥1.0 [1.2]</td>
<td>2.4</td>
<td>2.8</td>
<td>4.0</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>≥30 [45]</td>
<td>10.0</td>
<td>58.8</td>
<td>39.0</td>
</tr>
<tr>
<td>Vitamin D (mcg)</td>
<td>No limit</td>
<td>0.8</td>
<td>0.8</td>
<td>1.2</td>
</tr>
<tr>
<td>Vitamin E (mg)</td>
<td>≥10 (AI)</td>
<td>8.9</td>
<td>19.5</td>
<td>11.0</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>≥12 [14]</td>
<td>33.0</td>
<td>23.6</td>
<td>14.0</td>
</tr>
</tbody>
</table>

**Notes:**
Bolded values in the table show levels below current EARs.
AI – Adequate intake as defined by the NHMRC.15
RDI – Recommended dietary intake.15

* Given the rigours of the military environment, we used the dietary energy provided in the military rations for the optimised diets (i.e., 18,212 kJ). For comparison, 11,450 kJ is the estimated energy requirement (EER),15 averaged for four adult age-groups at the mid-range level of physical activity of 1.7 MJ/day. The latter relates to male weight sizes from 56.3 to 88.0 kg with a mid-range value of 71.3 kg. This compares to the mean enlistment weight of a New Zealand soldier of European ethnicity of 160 lbs or 72.6 kg.15 Nevertheless, the 18,212 kJ in Scenario R-A is equivalent to 4,350 calories which is slightly more than estimates for frontline British and German troops in WW1 (at 4,193 and 4,038 respectively), but less than for US troops (at 4,714).13

** New Zealand Adult Nutrition Survey (NZANS).16

*** The recommended upper limit for sodium of 2300 mg15 was not applied in our analyses as in the military situation, relatively higher sodium levels may have been more optimal for reducing food wastage from spoilage. Also losses of sodium via sweat during work in hot conditions might have increased sodium requirements – though probably still far below the estimated intake from the military rations as per Scenario R-A. Also at times of the year at Gallipoli it was also quite cold and so sodium losses via sweat would sometimes be fairly minimal.
Table 4. Food sources of two key vitamins (A and C) in the actual official military rations (using two differing food composition databases, ordered by vitamin C levels)

<table>
<thead>
<tr>
<th>Food items</th>
<th>Source of vitamin A (%)</th>
<th>Source of vitamin C (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NZ Food composition data</td>
<td>USDA data</td>
</tr>
<tr>
<td>Corned beef (canned)</td>
<td>23.3</td>
<td>0</td>
</tr>
<tr>
<td>Jam</td>
<td>0.1</td>
<td>0</td>
</tr>
<tr>
<td>Bacon</td>
<td>0</td>
<td>5.1</td>
</tr>
<tr>
<td>Dried potatoes</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>Biscuit</td>
<td>0</td>
<td>1.2</td>
</tr>
<tr>
<td>Dried peas</td>
<td>1.1</td>
<td>0.5</td>
</tr>
<tr>
<td>Cheese (cheddar)</td>
<td>75.0</td>
<td>93.0</td>
</tr>
<tr>
<td>Other#</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Notes:
* The USDA database reports zero vitamin C for most beef products, though some vitamin C is reported in raw ox liver (at 1.3 mg / 100 g) which could potentially contribute if the canning process in 1915 included parts of this or other organ meats.
** The USDA does report vitamin C in fresh raw pork (0.7 mg / 100 g) but not in bacon.
*** Uses the vitamin C data for dried potato from the NZ food composition database (given the very high value from likely vitamin C fortification in the USDA database for dried potato). See the Methods section for further details.
# Includes: bread, flour, dried beans, salt, mustard, pepper and sugar.

Table 5. Results with uncertainty intervals from 2000 iterations of the modelling around intakes of two key vitamins (A and C) in the official military rations in 1915 (Scenario R-A)*

<table>
<thead>
<tr>
<th>Statistics</th>
<th>NZ food composition data</th>
<th>USDA food composition data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Vitamin A (mcg)</td>
<td>Vitamin C (mg)</td>
</tr>
<tr>
<td>Mean</td>
<td>312.4</td>
<td>7.2</td>
</tr>
<tr>
<td>Median</td>
<td>312.7</td>
<td>7.2</td>
</tr>
<tr>
<td>95% Uncertainty interval [UI]*</td>
<td>291.4 to 332.4</td>
<td>6.5 to 7.8</td>
</tr>
<tr>
<td>99% UI*</td>
<td>283.3 to 338.1</td>
<td>6.3 to 8.0</td>
</tr>
</tbody>
</table>

* When considering the factors of: food wastage and the variability of the likely variation in nutrient concentrations in foods (including vitamin C in dried potato). See Methods for further details.

Discussion

Main findings and interpretation—The historical reports indicate the very limited variety of the military rations at Gallipoli and include additional factors that may have
impacted on food intake (e.g., supply to the front lines, harassment from flies, limited water supplies and low palatability of the food).

The nutritional analysis found that the military rations supplied at Gallipoli appeared to be deficient by modern nutritional standards (i.e., too low in: vitamins A, C, E, potassium, selenium and dietary fibre).

Furthermore, if the planners of military rations had made these rations somewhat more like the typical New Zealand diet in terms of modest amounts of canned vegetables and fruit (Scenario R-V), then this would probably have eliminated four of these deficits (i.e., including those for vitamins A and C). Other improvements would have been a reduction in the very high intakes of saturated fat and sodium. This higher variety diet (Scenario R-V) was estimated to only cost an estimated 40 cents per day more (albeit using modern pricing, i.e., $11.43 vs $11.03, Table 3).

The low vitamin C intake identified in this analysis (including in the uncertainty analysis) is entirely compatible with the medical reports of scurvy among the New Zealand soldiers at Gallipoli. It also fits with other evidence from Gallipoli: “…scurvy broke out among the Indian troops at Gallipoli, and among the Turkish troops taken prisoners, there were many well-defined cases of scurvy, beriberi, and pellagra”. Our estimated 95% UIs of 6.5–7.8 and 5.5–6.7 mg/day for vitamin C in the rations (depending on nutrient data and wastage of food – Table 5), fit with the evidence for clinical signs of scurvy in adults occurring with intakes of 7–8 mg/day or less.

But our estimates for vitamin C intake may still be overestimates since vitamin C content in foods would have been degraded by the drying and canning methods of the time, the long transport times of food to Gallipoli, and by cooking methods (e.g., prolonged heating and exposure to copper, iron or mildly alkaline conditions can destroy vitamin C; it can also be leached into water during cooking). Intake would have been impaired by reduced food intake from illness (widespread dysentery in these troops) and problems with food supply delivery to frontline troops (see above). Furthermore, tobacco was part of the rations supplied to the troops and plasma vitamin C concentrations can be reduced by 40% in male smokers. Finally, some individuals may have been particularly susceptible to scurvy as the more contemporary view is that it is a nutritional deficiency that is codetermined by genetic factors.

Our analysis also showed deficient vitamin A intake but nearly all the uncertainty analysis results gave higher levels than for causing night blindness from vitamin A deficiency (i.e. below 165 mcg/day as reported in some studies, though the lower bound of the 99% UI using USDA data was below this level). But our results are also possibly overestimates as the above reasons given for vitamin C intake around food supply and dysentery would also apply to lowering vitamin A intake and absorption.

So while we could find no medical reports of “night blindness” in these troops in the literature, it is plausible that some existed and was not reported (especially given past historical evidence for military populations with night blindness – see Introduction). Hence this deficiency could plausibly have increased injury risk if soldiers were more likely to be stumbling around at night (i.e., injury from enemy fire or falls).
Given the role of vitamin A in immune function and protecting against infectious disease mortality, the low intake could well have contributed to the high mortality rate from dysentery in the Gallipoli campaign.

Other nutritional morbidity could have arisen from the high sodium in the military rations (Table 3). This would have exacerbated thirst that occurred when frontline troops were short of water which was a common situation at Gallipoli. The hardness of the biscuits was also considered a problem and was reportedly the main reason for visits to army dentists.

Low dietary fibre in the military rations (especially when combined with water shortages), could have contributed to constipation and such related problems as haemorrhoids. But such problems would not be relevant at times when dysentery and typhoid were prevalent. Furthermore, the overall low variety and palatability of the food may have adversely impacted on military morale and mental health.

The comparison with “optimised” diets using foods available in 1915 is an artificial one as military planners in 1915 knew only about food costs and very little about nutrition. But this analysis does demonstrate the very large advances in modern knowledge combined with mathematical techniques and computerisation. That is only six foods (Scenario R-O) would be required to provide optimal nutrition and at under half the cost of the actual military ration in 1915. Even expanding the variety of foods beyond the military ration was cost saving when considering the purchase price (for 50 g+ items in Scenario R-OV).

**Study limitations**—As detailed above for vitamins A and C, our estimates of nutrient intake are probably overestimates for a range of reasons. But added to this is that the modern nutrient data of foods may not be fully comparable with the 1915 equivalents. For example, the modern food for which we used nutrient data for, may be more nutrient dense due to more efficient industrial scale harvesting and food processing. Modern canned food may have lower sodium levels and may reflect more modern preferences (e.g., with canned meat probably being less fatty).

Our estimates around the relative pricing of the different rations makes the crude assumption that relative prices between foods now are similar to those of 1915. Yet there have been many changes in agricultural and industrial techniques involved in food processing which may have influenced relative prices. We also do not consider how much a slightly heavier daily food ration in some scenarios might have impacted on transport costs by ship or from the beach depots at Gallipoli to the front lines.

The estimates used in the uncertainty analysis for food wastage (at 25%) are simplistic and could also be conservative. The uncertainty analysis also highlighted differences in the nutrient concentrations depending on the food composition database used (e.g., markedly so for vitamin C in canned corned beef). Given all these considerations, our nutritional analysis results should be considered as only “suggestive” and not definitive.

**The wider problem of poor planning**—The poor planning around low quality military food rations with lack of variety (no canned vegetables or fruit) and dominance of salty corned beef and limited water supplies could have been avoided if planners tried harder to better replicate the standard New Zealand diet at the time.
Furthermore, if military leaders and military medical personnel had appreciated the lessons from military history (e.g., the major problems of scurvy in the US Civil War⁵), then they may have ensured more fruit and vegetables were supplied to the troops.

It is possible that such risks were known by some, but that it was anticipated that the Gallipoli campaign would be short-lived and easily won. Certainly not one of the allied planners anticipated what followed. Instead of swift victory:

> There ensued stalemate and trench warfare perhaps even more ghastly than the Western Front, in which the stench of corpses rotting in the blazing sun of May, June and July blended in with the perfume of wild thyme, and the very narrowness of the front and rear areas added a dreadful claustrophobia.⁶

As the war progressed, there is considerable evidence on the Western Front that the New Zealand military tried to improve food quality (including provision of fresh vegetables).¹¹ Also New Zealand soldiers continually supplemented their diet by buying local produce particularly eggs and potatoes.

For the British Expeditionary Force as a whole, the quality and amount of food available improved considerably. Corned beef (bully beef), albeit varying in quality, was abundant. But as one soldier wrote to his parents: “We get plenty of tuck out here, bread, cheese and bacon and a butter issue twice a week and stew for dinner. It is a bit monotonous.”¹³ (p323) Monotonous yes, but it would have been more nutritious than the Spartan nature of the Gallipoli rations. This diet was also supplemented by regular food parcels from home and by purchasing food from local people.

Food and water supplies were just one of a number of planning problems at Gallipoli. Other problems included inadequate clothing,³ and lack of enough health services (including number of healthcare workers, medical supplies and hospital ships). Because of such problems during the Anzac landings and in the months that followed, “many did not get the surgery they needed”.

As a New Zealand historian acknowledged: “If there was one thing that showed our unpreparedness for war on a large scale, it was the neglect to anticipate accommodation for wounded.”¹⁰ (p990) Elliot also wrote in 1923 that “the high importance of hospital ships was not sufficiently recognised in the earlier stages of the Gallipoli campaign.”²²

Military planners were not anticipating such a prolonged and difficult campaign,²³ and all sides were surprised by the new scale and nature of industrialised warfare. But the major failure with Gallipoli may have been at the strategic level given the redundancy of the campaign when the outcome of WWI was always going to be determined on the Western Front.²³

The nutritionally inadequate food rations for the New Zealand soldiers on Gallipoli was symptomatic of this poor planning, and was a feature that is likely to have contributed to cases of scurvy and other nutrition-related health problems.
Competing interests: Nil.

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References:


Appendix 1

Additional methods

Approaches to uncertainty—Uncertainty analysis was performed around the levels of vitamins A and C in the official 1915 military rations using the software “@Risk for Excel” (version 5.7 Palisade, Sydney). We ran 2000 iterations of the model and calculated 95% and 99% uncertainty intervals (UI). The analysis considered uncertainty arising from:

- The variation in levels of these vitamins in food (we assumed that the levels of vitamins A and C followed a normal distribution with a standard deviation equal to 5% of the mean value). In addition to the “New Zealand food composition database”, we also considered the data in the US Department of Agriculture (USDA) database.  

- The exception to the above was that we excluded the USDA data for dried potato for vitamin C – as this was very high (at 81 mg/100 g and probably reflecting fortification of the product with vitamin C). Instead, for both the vitamin C estimates using NZ and USDA nutrient data, we applied loss estimates in the range of 82% to 96% (i.e., for the 95% uncertainty interval [UI], normally distributed). The former value represents an estimate for vitamin C loss from the drum-drying of potatoes (the same as the 82% loss for storage for 4.3 months at 25°C), and the latter value for the loss from preparation (i.e., reconstituting mashed potatoes and holding them 30 minutes on a steam table). Other work for modern convective drying of potato also reports substantive vitamin C losses.

- In the absence of good data on food wasted or lost in delivery to the battlefield, we just used the same point estimate (of 25%) for food waste from a modern UK study (Table 50 in the Report). Then we assumed a normal distribution and used ±10% around the point estimate for the 95% UI.

Nutrient analysis methods—In Tables A1-A3 are details of the military rations in 1915 and our assumptions around modern equivalents and plausible additions.
Table A1. Details of the daily 1915 military food rations and methods steps for further analysis

<table>
<thead>
<tr>
<th>Original specifications*</th>
<th>Equivalent used in our analysis (metric equivalents)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>“1¼ lbs, Bread or 1 lb. Biscuit or 1 lb. Flour.”</td>
<td>White bread (1.25 lb = 567 g) x 0.33</td>
<td>We assumed a third each of these three alternatives in the nutrient analysis. Of note was that in the early 1900s in NZ, white bread was preferred over brown (<a href="http://www.teara.govt.nz/en/food-and-beverage-manufacturing/6">http://www.teara.govt.nz/en/food-and-beverage-manufacturing/6</a>).</td>
</tr>
<tr>
<td>“3 ozs. Cheese.”</td>
<td>Cheddar cheese (3 oz = 85 g)</td>
<td>We used cheddar cheese as this was the major form produced by NZ (<a href="http://www.teara.govt.nz/en/agricultural-processing-industries/2">http://www.teara.govt.nz/en/agricultural-processing-industries/2</a>).</td>
</tr>
<tr>
<td>“½ lb. Jam”</td>
<td>Jam (3 oz = 85 g)</td>
<td>We used a mix-fruit jam in our analysis. Plum and apple, and apricot were the most common jams used in the rations.</td>
</tr>
<tr>
<td>“1¼ lbs. Fresh Meat or 1 lb. (nominal) preserved meat.”</td>
<td>Corned beef (1lb = 454 g)</td>
<td>We used corned beef (i.e., bully beef) as “fresh meat” was less commonly available in the military setting.</td>
</tr>
<tr>
<td>“4 ozs. Bacon.”</td>
<td>Bacon (4 oz = 113 g)</td>
<td>We used fresh bacon as food composition data on former heavily salted forms was not available. The bacon in the rations was quite fatty and was nicknamed ‘corporal’ bacon – for the one or two strips of meat through it.</td>
</tr>
<tr>
<td>“2 ozs. Peas, Beans or dried Potatoes.”</td>
<td>Dried peas (0.67 oz = 19 g)</td>
<td>We assumed a third of each of these 3 alternatives, but it is possible that in reality the cheapest item predominated: i.e., dried potatoes.</td>
</tr>
<tr>
<td>“½ oz. Salt”</td>
<td>Salt (0.5 oz = 14 g)</td>
<td></td>
</tr>
<tr>
<td>“1/20 oz. Mustard.”</td>
<td>Mustard (0.05 oz = 1.4 g)</td>
<td></td>
</tr>
<tr>
<td>“1/36 oz. Pepper.”</td>
<td>Pepper (0.028 oz = 0.8 g)</td>
<td></td>
</tr>
<tr>
<td>“3 ozs. Sugar.”</td>
<td>White sugar (3 oz = 85 g)</td>
<td></td>
</tr>
</tbody>
</table>

Notes:
* Included in the rations but which we excluded from our analysis were: “⅝ ozs. Tea.” (since it provides no dietary energy) and the following since they were declared “discretionary”: “1/10 gill Lime juice at discretion of G.O.C. on recommendation of S.M.O”; “½ gill Rum at discretion of G.O.C. on recommendation of S.M.O”.

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Table A2. Further details on the foods used to replicate the military rations for 1915 (as per those listed in column two of Table A1)

<table>
<thead>
<tr>
<th>Food items</th>
<th>Specific modern product used in this analysis</th>
<th>Amount (g) / day</th>
<th>Price per 100 g (NZ$, 2011/12)*</th>
<th>Comments (see also Table A1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bread</td>
<td>White sliced loaf (cheapest brand)</td>
<td>187</td>
<td>0.29 [FPI]</td>
<td></td>
</tr>
<tr>
<td>Biscuit</td>
<td>Cabin bread used for the nearest equivalent</td>
<td>150</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Flour</td>
<td>White (supermarket only)</td>
<td>150</td>
<td>0.14 [FPI]</td>
<td>The type of hard biscuit used in 1915 has no precise modern equivalent but for the nutrient analysis we used cabin bread.</td>
</tr>
<tr>
<td>Cheese</td>
<td>Mild cheddar (supermarket)</td>
<td>85</td>
<td>1.02 [FPI]</td>
<td></td>
</tr>
<tr>
<td>Jam</td>
<td>Supermarket brand (mixed fruit)</td>
<td>85</td>
<td>0.45</td>
<td></td>
</tr>
<tr>
<td>Corned beef (canned)</td>
<td>Supermarket brand corned beef</td>
<td>454</td>
<td>1.14</td>
<td></td>
</tr>
<tr>
<td>Bacon</td>
<td>Middle bacon product</td>
<td>113</td>
<td>1.52</td>
<td></td>
</tr>
<tr>
<td>Dried peas</td>
<td>Split peas (green)</td>
<td>19</td>
<td>0.34</td>
<td></td>
</tr>
<tr>
<td>Dried beans</td>
<td>Haricot beans</td>
<td>19</td>
<td>0.55</td>
<td></td>
</tr>
<tr>
<td>Dried potatoes</td>
<td>Instant potato (potato flakes)</td>
<td>19</td>
<td>1.13</td>
<td></td>
</tr>
<tr>
<td>Salt</td>
<td>Table salt</td>
<td>14</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>Mustard</td>
<td>Mild American style mustard</td>
<td>1.4</td>
<td>1.66</td>
<td></td>
</tr>
<tr>
<td>Pepper</td>
<td>Supermarket brand pepper black ground White</td>
<td>0.8</td>
<td>2.98</td>
<td></td>
</tr>
<tr>
<td>Sugar</td>
<td>White</td>
<td>85</td>
<td>0.20 [FPI]</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>1382.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes:
FPI = Food price index (average monthly data for 2011).17
* Prices from the online store of a major supermarket chain “Countdown”, unless otherwise indicated (March 2012). Lowest cost items were preferentially selected.
Table A3. Additional types of dry or canned foods that could have been included in military rations for 1915 and which were included in our further nutritional modelling (along with those itemised in Table A1)*

<table>
<thead>
<tr>
<th>Food category / items</th>
<th>Specific modern product used in the analysis</th>
<th>Price per 100 g (NZ$, 2011/12)**</th>
<th>Comments (availability in NZ at the time of WWI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cereals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oats (rolled)</td>
<td>Branded “porridge rolled oats”</td>
<td>0.41</td>
<td>In NZ, “by 1890 porridge (cooked rolled oats), introduced by Scottish immigrants, was common” (<a href="http://www.teara.govt.nz/en/food-and-beverage-manufacturing/5">http://www.teara.govt.nz/en/food-and-beverage-manufacturing/5</a>).</td>
</tr>
<tr>
<td>Fruit</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apricots (canned)</td>
<td>Supermarket brand apricot halves</td>
<td>0.27</td>
<td>Fruit canning was occurring in NZ in 1905 e.g., for apricots (<a href="http://www.teara.govt.nz/en/otago-region/6/5/2">http://www.teara.govt.nz/en/otago-region/6/5/2</a>). Around 1910 a canning factory in Hawke’s Bay made “various kinds of jam, canned peaches, pears, apricots, tomatoes, beans and peas, as well as pickles, spices, baking powder and lemon peel” (<a href="http://www.teara.govt.nz/en/food-and-beverage-manufacturing/4/1/1">http://www.teara.govt.nz/en/food-and-beverage-manufacturing/4/1/1</a>).</td>
</tr>
<tr>
<td>Peaches (canned)</td>
<td>Supermarket brand</td>
<td>0.42 [FPI]</td>
<td>See above.</td>
</tr>
<tr>
<td>Pears (canned)</td>
<td>Branded pear halves in juice</td>
<td>0.55</td>
<td>See above.</td>
</tr>
<tr>
<td>Vegetables***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tomatoes (canned)</td>
<td>Supermarket brand tomatoes diced</td>
<td>0.24</td>
<td>See above.</td>
</tr>
<tr>
<td>Peas (canned)</td>
<td>Branded “peas garden minted”</td>
<td>0.71</td>
<td>See above. (Price also adjusted as the drained weight is used in the nutrient analysis). As above for peas.</td>
</tr>
<tr>
<td>Beans (canned)</td>
<td>Branded “four bean mix”</td>
<td>0.83</td>
<td></td>
</tr>
<tr>
<td>Meat, fish and dairy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fish (canned)</td>
<td>Branded fish fillets smoked (NZ fish of unspecified species)</td>
<td>2.00</td>
<td>See above regarding canned fish. Price per 100 g has been adjusted to give the price for the drained weight (i.e., 200g /310 g). Nutrient analysis used canned tuna.</td>
</tr>
<tr>
<td>Milk powder (canned)</td>
<td>Supermarket brand skim milk powder</td>
<td>1.00</td>
<td>Canned milk powder was available e.g., Joseph Nathan and Company started making canned dried milk in 1904 at Bunnythorpe (<a href="http://www.teara.govt.nz/en/agricultural-processing-industries/2">http://www.teara.govt.nz/en/agricultural-processing-industries/2</a>).</td>
</tr>
</tbody>
</table>

Notes:
* Ignoring foods that could have potentially been purchased: (i) within the Mediterranean region e.g., from Greece: dried fruits, vegetables, olive oil; and (ii) on the international market e.g. canned sardines and canned salmon.
** Prices as per footnote for Table A1.
*** Canned baked beans and spaghetti in tomato sauce were not produced in NZ until the early 1930s.
The Northland Emergency Meningococcal C Vaccination Programme

Clair Mills, Liane Penney

Abstract

Aim This paper describes an emergency meningococcal C vaccination programme implemented in Northland, New Zealand in 2011. The programme aimed to reduce the impact of a meningococcal group C outbreak on the Northland population, through vaccination of 85% of children and youth 12 months to <20 years with a meningococcal serogroup C conjugate vaccine.

Method The emergency vaccination programme targeted an estimated population of 44,000 children and youth. Vaccinations were promoted and delivered by Northland District Health Board Public Health Nursing Service, Primary Health Organisations, General Practice, and Maori provider services, at schools, general practice clinics, via community clinics and outreach home-based vaccination services.

Results 32,410 children and youth were vaccinated. Overall coverage reached 73% (72% Māori, 75% non-Māori). Coverage differed across age, ethnic groups, school decile and geographic location. Vaccination coverage was highest for children 5 to <13 years at 84% for Māori and 81% for non-Māori. Coverage was lowest for the 17 to <20 year age group at 46% for Māori and 63% for non-Māori. In the pre-school population, 67% of Māori and 76% of non-Māori children 12 months to <5 years received vaccination. The 13 to <17 year age group reached 71% coverage for Māori and 70% for non-Māori.

Conclusion Equitable, high vaccination coverage is attainable in an emergency vaccination programme in New Zealand. However a range of service options, including community outreach, are necessary to reduce access barriers for some groups. The programme presented useful insights into what is possible with focussed attention to adapting services to meet diverse needs.

The Northland region of New Zealand has an estimated population of 158,000, of whom 32% identify as Māori. The median age of Māori is lower than non-Māori, and nearly half of the population under 20 years is Māori. There are small Pacific and Asian communities (together less than 5% of the population), with the remainder being European/Other.1

Many Northland communities experience high levels of socioeconomic disadvantage, with poor housing, higher unemployment and lower incomes than the New Zealand average. Māori are disproportionately over-represented in more deprived areas.1

During the winter of 2011, a community outbreak of meningococcal group C disease occurred across the Northland District Health Board (NDHB) region, predominantly affecting children and young people. By the end of December 2011, nine cases of meningococcal C had been confirmed, with three deaths.
The rate of group C meningococcal disease for the population in the Whangarei district aged less than 20 years was 27.6 cases per 100,000 population (6 cases) compared with 17.6 cases per 100,000 population under 20 years (8 cases) in the whole region - both higher than the community outbreak “threshold” rate defined by the Ministry of Health.\(^{2,3}\)

On 12 September 2011, the Ministry of Health recommended an emergency vaccination programme be implemented, offering free, single dose meningococcal C conjugate vaccine (Meningitec\(^{\circ}\)) to all children and young people in the NDHB area between the ages of 12 months and <20 years.

NDHB established a Steering Group on September 13\(^{\text{th}}\) to plan the delivery of the vaccination programme to an estimated 38,224 children and young people (85% of the target age group). The vaccination programme was phased in from 27 September and scaled up to reach the whole eligible population, as the required vaccine stock arrived in New Zealand in three stages. Completion of the vaccination programme was set for 16 December 2011.

**Method**

**Programme goal and target**—The goal of the vaccination programme was to reduce the impact of the meningococcal C outbreak in Northland, through vaccination of 85% of the child and youth population aged 12 months to <20 years with Meningitec\(^{\circ}\). The target 85% vaccination coverage was expected to be met equitably by age, ethnicity and geographic locality.

**Programme planning**—A Steering Group established from the point of programme approval enabled broad stakeholder engagement from primary care and public health services in programme planning and implementation oversight. Māori engagement involved Māori planning and funding expertise, a Māori provider and Whānau Ora Collective representative, and a Māori Nurse Specialist with a communicable disease background, as members of the Steering Group.

The Steering Group had 10 working days to plan the roll out of the programme with the first day of school-based vaccinations planned for September 27th. The urgency and short timeframe of the response was not only due to the severity of meningococcal disease, but also the altered timing of school holidays due to the 2011 Rugby World Cup, the need to vaccinate senior students before national school examinations, and limited initial vaccine availability.

Steering Group meetings were held daily, reducing to twice weekly and then weekly as the vaccination programme progressed. The meetings continued after completion of the vaccination programme to oversee coverage data reporting and analysis, options for utilisation of the remaining doses of vaccine, provider payment issues, and the programme evaluation.

**Programme design**—The programme was designed with three main approaches to reaching the eligible population and achieving 85% vaccination coverage:

- A school based campaign managed by the Public Health Nursing (PHN) service to reach students enrolled in schools.
- A General Practice (GP) campaign to reach under 5 year old children, home schooled children, out of school youth, and school enrolled children who preferred vaccination at their GP clinic.
- A community outreach campaign targeting out of school youth and Māori, but which could also provide vaccinations to all the eligible population from 12 months to <20 years. The targeting was in recognition of historically lower coverage for Māori and youth in previous vaccination programmes, both in Northland and nationally.\(^{4,5}\)

The community outreach campaign was managed by NDHB and supported in its operation by Māori Provider organisations. It utilised fixed locations such as a central shop in the main Northland city of Whangarei, space within Work and Income New Zealand offices, pharmacies, and Māori provider clinics.
Mobile units parked in main streets, shopping centre car parks, suburban streets and rural village locations, and visited Polytechnic and other training facilities. Fixed location clinics and mobile units were staffed by a range of personnel from Māori providers, the PHN service, and new fixed term appointments to the NDHB Public Health Unit specifically for the vaccination programme. Initially planned only for the school holidays in early October the community clinics were further extended when their success became apparent and populations or areas of low coverage were identified.

Communications—A comprehensive communication plan guided utilisation of a full range of media to raise public awareness of the disease outbreak and the immunisation campaign. Television, mainstream and iwi radio, print media, billboards, posters, flyers, windshield pamphlets, Facebook, Youtube, cinema advertising, and a range of different types of community gatherings communicated the key messages.

Kaimahi and youth health promoters “on the street” in the vicinity of community outreach clinics were key to engaging people in discussion and providing information to support vaccination. Healthcare provider organisations and individual health professionals across public health, primary health and secondary care also played important roles in the communication strategy, ensuring their clients were well informed of the disease, and were aware of protection through immunisation, eligibility for the vaccine, and how to access immunisation services.

Programme resources—Despite an environment of fiscal constraint, the NDHB was committed to funding services required to reach and vaccinate the eligible population once the vaccine had been purchased. The general funding approach was to monitor vaccination coverage on a weekly basis and commit the limited additional resources available as required to improve coverage.

Promotion of the vaccination campaign, co-ordination and delivery of immunisation services, were largely conducted by existing public health and primary health care teams. A small number of casual health promoters, registered nurses and data entry staff were recruited to increase capacity to deliver the programme. Existing teams managed the delivery of the programme by forfeiting other non urgent activity and working overtime when necessary.

Additional clinic rooms and mobile units were leased in order to provide community outreach clinics. Some clinic spaces and mobile units were also made available to the NDHB at no cost, by other health and social service providers.

The estimated total cost of the programme including vaccine, immunisation subsidy paid to General Practice and Māori provider organisations, additional staff and materials costs incurred by these primary care providers, all costs associated with the school and community clinics programmes, Primary Health Organisation (PHO) coordination costs, communications, and vaccination data management costs, was estimated at $3.2 million. This indicates an estimated programme cost of $98.73 per person vaccinated. The cost of the vaccine amounted to more than 50% of this cost.

Systems for coverage data collection, reporting and analysis—Vaccinations given in General Practice were recorded in the emergency vaccination programme area of Practice Management Systems, and this information was transmitted to the National Immunisation Register (NIR) using the emergency vaccination programme function. However the NIR data could not be viewed or queried due to the Ministry of Health’s delayed decision about its use and lack of consent obtained for participant “opt off”.

School-based and community outreach vaccinations provided by PHNs and Māori provider nurses were recorded on paper and entered into the Public Health Unit vaccination database. Weekly reports on vaccination coverage were generated by the Northland Primary Health Organisations and the Public Health Unit and supplied to the Steering Group. Weekly tracking of vaccination coverage by age group, ethnicity and geographic area informed ongoing programme activity.

Results

Community response—A high level of awareness of the meningococcal C outbreak and the vaccination programme was reported by evaluation interview participants and respondents to a Northland wide telephone reminder project. However, some people
had not heard about the programme and a few were unaware that free Meningitec® vaccinations would only be available for a limited period. There was minimal public questioning of the safety and efficacy of the vaccine, and there were reports of families choosing to have Meningitec® despite declining other vaccinations.

**Vaccination coverage**—A total of 32,410 of the estimated 44,000 eligible children and young people were vaccinated, an overall vaccination coverage rate of 73%.

**Place of vaccination**—Just over half of all vaccinations were given in schools, 38% were given in General Practice clinics and 10% in community outreach clinics. Community outreach clinics were most commonly utilised by Māori (59% of users). Sixteen to <20 year olds were the most common age cohort to utilise community clinics (37% of users).

These were the two target groups for community clinics. However, community outreach clinics were utilised by all age groups and all ethnic groups. Figure 1 below shows community clinic vaccinations increased Māori coverage by 9% and non-Māori coverage by 6%.

**Inequities in coverage**—Vaccination coverage differed across age groups, ethnic groups, school deciles and geographic locations.

**Age inequities**

- Vaccination coverage was highest for the 5 to <13 year age group at 84% for Māori and 81% for non-Māori.
- Non-Māori 12 months to <5 years coverage reached 76%.
- The 13 to <17 year age group reached 71% coverage for Māori and 70% for non-Māori.
- Vaccination coverage for Māori 12 months to <5 years was low at 67%.
• Vaccination coverage was lowest for the 17 to <20 year age group at 46% for Māori and 63% for non-Māori.

Inequities by school decile

• 68/158 (43%) of schools enrolling 32% of all students (and 54% of Māori students) in Northland are in deciles 1 & 2 [Decile 1 schools in New Zealand are the 10% of schools with the highest proportion of students from low socioeconomic communities (based on residential address of students), whereas decile 10 schools are the 10% of schools with the lowest proportion of these students].

• The overall vaccination consent form return rate was 82%, a lower return than that seen in routine school based vaccination programmes in Years 7 & 8 in Northland.

• There was a considerable range of coverage achieved across schools (from 21% to 98%) but little variance in mean coverage by school decile. Mean coverage in decile 1 & 2 schools was 58.8%, while it was 53.8% in the seven decile 9 schools in Northland (the least deprived - there are no decile 10 schools in Northland). The highest mean school coverage (59.6%) was in decile 3 & 4 schools.

Ethnic inequities

Ethnic inequities in coverage were most significant in two age groups.

• A large inequity of 17% lower vaccination coverage in Māori aged 17 to <20 years compared with non-Māori.

• Māori coverage was also lower (9%) in the 12 months to <5 year age group compared with non-Māori.

• Pacific under 5 year olds coverage was between Māori and NZ European, and Asian children under 5 years coverage was the highest at 90% (Figure 2).

Figure 2. Vaccination coverage 12 months to <5 years by ethnicity
Between general practices there were marked differences in the level of Māori:non-Māori inequities in the 12 months to <5 year age group. The greatest gap was a 35% higher non-Māori vaccination coverage compared with Māori in the under five cohort of one general practice, whilst in a small number of practices equitably high coverage (>85%) was achieved between Māori and non-Māori in this age group.

Higher Māori coverage than non-Māori in the largest age group (5 to <13 years) resulted in a relatively equitable overall coverage from 12 months to <20 years of 72% for Māori and 75% for non-Māori.

Geographic inequities—Geographic inequities were observed in the vaccination coverage of the 12 months to <5 year age group with coverage lowest in the following areas:

- Southern boundary of the Northland DHB region
- Dargaville and surrounding rural Kaipara
- Kaitaia, Kaikohe, Hokianga and Kerikeri
- Otaika, Tikipunga, Onerahi, and Kamo suburbs of Whangarei.

Across all age groups vaccination coverage was lowest at the Southern boundary of the Northland DHB region.

Discussion

The Northland emergency meningococcal C vaccination programme was planned and implemented in an extremely short timeframe. Achieving overall vaccination coverage of 73% of the 12 month to <20 year population in a twelve week programme, given historically low and inequitable immunisation coverage in Northland, is a significant achievement.

Routine well child immunisation coverage in Northland remains below 90% at two years of age, although Māori coverage is now similar to non-Māori. School immunisation programme coverage in Years 7 & 8 in Northland is less than 65%, and previous mass vaccination programmes such as the MeNZB programme have failed to achieve equitable coverage. However, in the meningococcal C programme, the combination of general practice, school programmes and community outreach clinics resulted in equitably high coverage in the 5-<13 year group.

There were pockets of excellence where some small geographic areas and general practice populations reached 85% coverage across all age and ethnic groups. However, as evidenced by vaccination coverage rates lower than the 85% target, and with significant inequities in coverage especially in the <5 years and 13 to <20 year age groups, further service developments are clearly necessary to achieve optimal coverage.

Based on ‘best practice’ examples, our broader programme evaluation, and evidence from New Zealand and international research, the following strategies are proposed for future programmes.

School programmes—High, equitable rates of vaccination consent form return are critical to achieving high coverage in school vaccination programmes. The routine
school based immunisation programmes in Northland (Boostrix® in Year 7 and Gardasil® in Year 8) are resourced sufficiently to enable follow up with families who have failed to return consent forms, resulting in consent form return rates of 85-100%.

This type of follow up was also important in achieving high school programme coverage in the MenZB campaign, but was not resourced for the Northland emergency meningococcal C campaign. Had it been, higher vaccination coverage rates—especially amongst school students over 13 years - are likely to have been achieved.

In-depth analysis of previous school consent form return and vaccination coverage rates in routine programmes, to identify individual schools which require additional systematic support, could also assist in improving overall school coverage rates.

Feedback from evaluation interview participants suggests engaging youth in communicating specific vaccination messages aimed at youth in schools could contribute to perceptions of appropriateness and acceptability, and also improve uptake of vaccinations by the older school students.

In addition, school vaccination programmes could enlarge access to vaccination for youth out of school and pre-school populations, if open to these groups, particularly in rural areas where schools may be much more accessible than general practice clinics.

Promoting the availability of vaccination at rural schools for their students’ pre-school or youth siblings should be considered for future vaccination campaigns.

**Community clinic services**—A full programme of community outreach clinic services should ideally be planned and funded from the commencement of future programmes, and include fixed and mobile clinics to cover urban and rural localities. In addition community clinics would be enhanced by:

- Development of authorised outreach vaccinator capacity within the primary care and Māori provider sectors to ensure an adequate supply of authorised vaccinators to deliver community outreach clinic services
- Better utilisation of Māori provider health promotion and vaccinator teams in the planning and delivery of community clinics
- Better use of local community knowledge to establish the optimal location of fixed and mobile clinics.
- Availability of community clinics at community events such as sporting and cultural festivals.

**General practice services**—To improve access to general practice vaccination services, strategies observed in general practices with the highest vaccination coverage rates would be applied uniformly. These included:

- A clinical leader or “champion” for immunisation services in every practice, who works with the whole practice team to reach agreement on a plan to achieve high vaccination coverage, to motivate the practice team to achieve vaccination coverage goals, and to monitor and report on vaccination coverage to the whole team.
• Telephone and/or text recall of all eligible patients to advise them of vaccinations due and to book an appointment. This could be undertaken by a practice team member or outsourced to a call centre. However, all call recipients should be given the opportunity to discuss vaccination with a nurse or doctor should they wish to, whether they are called by a call centre or a practice team member.

• Next of kin information utilised to follow up on patients who have changed contact details and are lost to follow up.

• Phone or text reminders of appointments for vaccinations, on the day or day before appointment.

• Systematic opportunistic vaccination, by ensuring all practice staff are primed to invite vaccination eligible patients attending the practice, and that vaccination eligible patients booked for another issue are flagged to be invited to be vaccinated.

• Systematic utilisation of PHO outreach vaccination services, other mobile nursing services, or a practice’s own home visiting services, to locate and vaccinate non-responders.

Many of these approaches, as well as the importance of well-informed and confident immunisation providers, have been shown in earlier New Zealand and international research to be associated with higher coverage at practice level.\(^9\text{-}^{12}\)

**Vaccination data management**—Quality vaccination coverage analysis requires leadership to plan and oversee data collection, recording, reporting, and analysis from programme commencement. Accurate and timely coverage data, by service, age, ethnicity and small geographic area, is critical to adapting strategies to maximise coverage during programme implementation. In future emergency responses, timely access to the National Immunisation Register to allow querying of individual vaccination status would be an important improvement.

**Conclusion**

The collaborative efforts of the Northland health sector enabled the timely delivery of a quality emergency vaccination programme to over 34,000 children and young people (73% of the target population) in twelve weeks. While a significant achievement, it has also provided opportunities to identify gaps and potential for improvements to routine immunisation services.

Equitable vaccination coverage cannot be achieved without implementing strategies to improve the availability of a range of accessible and appropriate services. Community clinics, with a “walk in, no appointment” approach, in a range of fixed locations and mobile units, provided an appropriate and accessible option for over three thousand children and young people (10% of the total vaccinated) who may not have otherwise received a Meningitec® vaccination.

General practice services remain an essential option for accessing vaccination, particularly for the pre-school population. General practice services are also the preferred option for some school aged children and youth. However meningococcal C vaccination coverage in Northland suggests general practice teams need to
continuously review and improve their processes in order to optimise access for their patients and reach high equitable vaccination coverage.

School based programmes demonstrate that equitable, high coverage can be achieved when vaccination services are taken to where children are, reducing barriers to access. However, school programmes also need to continuously review their systems and processes to ensure all students have equal opportunity to access vaccination services at school, particularly young adults.

The outstanding lesson for the Northland health sector is that a sharp focus on improving access for those who have previously been missed, and a will to innovate across all parts of the health sector is necessary, if immunisation coverage is to improve. Continuing to deliver vaccination services without system changes will not increase coverage or eliminate inequalities.

Competing interests: Nil.

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References:


Unanswered questions, the epidemiology of a community outbreak: meningococcal C disease in Northland, New Zealand, 2011

Clair Mills, Kerry Sexton, Philip Carter

Abstract

Aim We describe the epidemiology of a community outbreak of Meningococcal C disease in Northland in 2011, and national trends in serogroup C disease in New Zealand.

Methods Notification data from EpiSurv for all meningococcal C cases were analysed for 2011 for Northland and for the period 2001-2011 nationally.

Results In 2011, the rate of group C meningococcal disease for the population in the Whangarei district aged less than 20 years was 27.6 cases per 100,000 population (6 cases) compared with 17.6 cases per 100,000 population under 20 years (8 cases) in the Northland District Health Board (DHB). All except one case were under 20 years of age. The case fatality rate was 33%. Nationally the rate of meningococcal C disease has fluctuated over the last decade, with an increasing trend apparent since 2007. There has been a noticeable increase over the last 3 years of group C cases infected with the C:P1.5-1,10-8 strain (including all of the Northland cases). This strain has also been associated with a higher case fatality rate (16% in the period 2007-2011).

Conclusion Meningococcal C disease in New Zealand, although still less common than group B, is poorly understood. The relationships between carriage, invasive disease and community outbreaks deserve greater study. Active monitoring of surveillance data is warranted to ensure timely funded introduction of the highly effective meningococcal C conjugate vaccine on to the national immunisation schedule when appropriate, given increasing disease rates, the high case fatality rate and significant Māori non-Māori inequities in disease incidence.

Invasive meningococcal disease, caused by the Gram-negative diplococcus bacteria Neisseria meningitidis, is a serious illness commonly presenting as meningitis and/or septicaemia. The case-fatality rate in New Zealand over the last decade has varied from 4 to 10%.\(^1\)

The bacteria can be differentiated into groups according to the chemical and immunological properties of the capsular polysaccharide. The most common groups causing human disease are A, B, C, W135 and Y, with groups B and C most common in New Zealand.

During the 1990s through to 2011, the group B meningococcal disease strain B:4:P1.7-2,4 predominated nationally over other strains, reaching a peak of 200 cases per 100,000 population in 2001 in children aged less than 1 year.\(^1,2\)
Group B meningococcal disease has likewise predominated in Northland; only 7 confirmed cases of group C meningococcal disease were recorded in the 10-year period, from 2001 to 2010.

Group C is the second most common group causing disease in North America and Europe, and is playing an increasing role in Asia. Since 2002 it has been responsible for local outbreaks in China and plays a significant role in Singapore. Most cases of meningococcal C disease are sporadic but clusters, outbreaks and epidemics have been reported globally. Outbreaks of group C meningococcal disease usually resolve in 1-to-3 years. Northland experienced a community outbreak of meningococcal C disease in 2011.

Methods
Analysis of the Northland and national meningococcal disease cases is based on data recorded in the national notifiable diseases database, EpiSurv, and the national Meningococcal Reference Laboratory database as at 21 February 2012. Cases reporting multiple ethnicities were prioritised to a single ethnic group according to the following prioritised order: Māori, Pacific Peoples, Asian, Middle Eastern/Latin American/African, European or Other (including New Zealander). Rates were calculated using Statistics New Zealand mid-year population estimates, other than rates by ethnic group. Denominators by ethnic group for 2011 were estimated by applying the ethnic group proportions in the 2006 Census to the population estimates for 2011.

Results
Meningococcal C disease in Northland, 2011—The first case of invasive meningococcal disease in Northland in 2011 was notified on 10 July, 2011 and was typed as group C, serotype 2a, PorA 1.5-1,10-8. Unusually, three further cases of group C meningococcal disease followed within a month. In the period up to 21 December 2011, a total of 13 confirmed cases of invasive meningococcal disease were notified in Northland. Of these, four cases were group B meningococcal disease and nine cases were group C meningococcal disease (all C:2a:P1.5-1,10-8 strain).

The United States Centers for Disease Control and Prevention and the New Zealand Ministry of Health definition of a community outbreak is “three or more confirmed cases of the same serogroup (and serotype) within a 3-month period and an age-specific incidence or specific community population incidence of approximately 10 cases per 100,000 population, where there is no other obvious link between the cases”. The rates in Northland fulfilled the criteria for a community outbreak in those individuals younger than 20 years.

Of the nine cases of group C meningococcal disease, three cases were Māori and six cases were European or Other (including New Zealander). Eight cases were younger than 20 years of age. The rate of group C meningococcal disease for this age group was 27.6 cases per 100,000 population (6 cases) in the Whangarei district compared with 17.6 cases per 100,000 population under 20 years (8 cases) in the Northland District Health Board (DHB) (Table 1).

Three deaths occurred due to group C meningococcal disease. There were no epidemiological linkages between the cases in Northland.
Table 1. Comparison of group C meningococcal disease rates before the introduction of meningococcal C conjugate vaccine: Northland DHB, New Zealand, Australia, The Netherlands and the United Kingdom

<table>
<thead>
<tr>
<th>Location</th>
<th>Year</th>
<th>Meningococcal C disease rate per 100,000 population</th>
</tr>
</thead>
<tbody>
<tr>
<td>United Kingdom</td>
<td>1999</td>
<td>2.0</td>
</tr>
<tr>
<td>Australia</td>
<td>2001</td>
<td>3.5</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>2001</td>
<td>1.7</td>
</tr>
<tr>
<td>New Zealand</td>
<td>2011</td>
<td>0.7</td>
</tr>
<tr>
<td>Northland DHB (total population)</td>
<td>2011</td>
<td>5.7</td>
</tr>
<tr>
<td>Northland DHB (population aged &lt;20 years)</td>
<td>2011</td>
<td>17.6</td>
</tr>
</tbody>
</table>

**National trends in meningococcal C disease**—Since 2001, the number of group C meningococcal cases in New Zealand has fluctuated. There was a downward trend overall from 2001 to 2007, decreasing from 0.8 to 0.2 per 100,000 population (30 to 9 cases) over this period. Since 2007, the rate of group C disease has returned to around the 2001 level, with a rate of 0.7 per 100,000 population in 2011 (32 cases).

**Figure 1. Rate of group C meningococcal disease in New Zealand, 2001-2011**

The increase in group C disease since 2007 has principally been driven by a rise in the number of cases due to the C:P1.5-1,10-8 strain, the strain predominant in Northland in 2011. Disease due to other C strains has been steady at 4 or 5 cases per year over the last 5 years.

The C:P1.5-1,10-8 strain was first detected in New Zealand in 2001. However, routine typing to this level has only occurred since 2007, so the number of cases due to this strain pre-2007 is unknown. There has been a noticeable increase over the last 3 years with 24, 18 and 27 cases infected with the C:P1.5-1,10-8 strain in 2009, 2010 and 2011 respectively.
In 2011, the rate of disease due to this strain was highest in the 1 to 4 year age group (2.4 per 100,000, 5 cases) followed by the 15-19 year age group (1.9 per 100,000 population, 6 cases). The rate of disease due to the C:P1.5-1,10-8 strain was more than three times higher in Māori compared to the European or Other (including New Zealander) ethnic group (1.7 per 100,000 population (11 cases) compared with 0.5 per 100,000 population [15 cases]). Rates were highest in Northland DHB (5.7 per 100,000 population, 9 cases) followed by Waikato DHB (1.4 per 100,000 population, 5 cases).

One third (9) of the 2011 C:P1.5-1,10-8 cases died. However, the case fatality rate for this particular strain of group C disease has varied considerably from year to year, with a case fatality rate of 6% (1/18 cases) in 2010 and 40% in 2007 (2/5 cases). The average case fatality rate due to any group C meningococcal disease over the last 5 years is 16.3% (17/104 cases).

Discussion

Since 2007, rates of meningococcal C disease in New Zealand appear to be trending upwards. In addition, case fatality rates for group C meningococcal disease are substantially higher than for disease due to the predominant B strain (B:P1.7-2,4), resulting in a similar number of deaths from group B and C (16 versus 17) meningococcal disease in the last 5 years, despite the lower number of cases due to group C disease (approximately one third of the number due to group B disease).

Although the lack of routine sub-typing pre-2007 in New Zealand precludes definitive confirmation of the increase in the C:P1.5-1,10-8 strain, this is concerning especially given that all of the 2011 C:P1.5-1,10-8 strain isolates were determined through multi-locus sequence typing to belong to the ST-11 clonal complex. Group C strains from this clonal complex have been associated internationally with higher case fatality rates, and increases in disease due to such strains led to the introduction of group C meningococcal conjugate vaccine on to routine immunisation schedules in a number of European countries.\(^8,9\)

The reasons for changes in incidence and severity of disease caused by different meningococcal strains are poorly understood. The relationship between carriage and disease has not been fully elucidated.\(^10,11\) Although meningococcal carriage is thought to be common, and higher in teenagers, it varies considerably between settings.\(^12,13\) Carriage prevalence does not predict incidence of disease or outbreak occurrence.\(^11\) Carriage in most people results in the development of protective antibodies but during the carriage state, co-colonisation with other pathogenic and nonpathogenic bacteria may lead to genetic exchange resulting in the emergence of new meningococcal clones.\(^13,14\)

Capsule switching (for example from serogroup B to C), recombination events involving the \(\text{porA}\) gene and insertion and transformation of DNA have been described, but there remains little known about trends in genetic types and “hyper-responsive” (i.e. more invasive) lineages.\(^11,14\) There is very little difference between the genomes of carrier and invasive strains, suggesting that ”on/off” switching of genes may be an important determinant of pathogenesis.\(^15\)
Community carriage of group C strains and the relationship between carriage and transmission of meningococcal disease, including group C disease have not been well studied in New Zealand. A 2001 study estimated the overall carriage rate of \textit{N. meningitidis} among household contacts in New Zealand was 20.5\%. The lower rates of group C disease between 2004 and 2008 coincide with the years when a group B meningococcal disease vaccine (MeNZB) was in use as part of the New Zealand Meningococcal B Immunisation Programme.

It is possible that cross-protection from MeNZB™ vaccine against group C disease contributed to this reduction while the vaccine was in use (MeNZB was removed from the routine childhood immunisation schedule from June 2008). A recent MeNZB™ vaccine effectiveness study estimated the effectiveness of MeNZB™ vaccine against non-group B strain disease to be more than 50\%. However, the actual contribution of MeNZB vaccine to the observed reduction in group C disease is uncertain given that the effect of residual confounding could not be excluded.

The meningococcal C conjugate vaccine is very effective in reducing the disease burden, but also has a significant impact on carriage. Data from both the UK and the Netherlands have demonstrated that infants can be successfully protected by indirect protection (herd immunity). In the UK, carriage of serogroup C meningococci was reduced by 66\% 1 year after vaccination. There were no significant changes in carriage of other disease-associated serogroups.

Currently total population rates of group C disease in New Zealand are still significantly lower (0.7 per 100,000) than those in the United Kingdom (UK), other European countries and Australia at the time of introduction of the meningococcal C conjugate vaccine to the routine childhood immunisation schedule in those countries.

However, the 2011 Northland outbreak suggests that the relationships between carriage, invasive disease and community outbreaks in New Zealand deserve greater study. In addition, given the high case fatality rate, upward trend and significant inequities in meningococcal C disease in New Zealand, it is critical that active monitoring of surveillance data is undertaken to help determine appropriate and timely introduction of the highly effective conjugate meningococcal C vaccine on to the national immunisation schedule.


deserve greater study. In addition, given the high case fatality rate, upward trend and significant inequities in meningococcal C disease in New Zealand, it is critical that active monitoring of surveillance data is undertaken to help determine appropriate and timely introduction of the highly effective conjugate meningococcal C vaccine on to the national immunisation schedule.

**Competing interests:** Nil.

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**References:**

The epidemiology of acute rheumatic fever in Northland, 2002–2011
Audrey Robin, Clair Mills, Roger Tuck, Diana Lennon

Abstract

Aim An audit of rheumatic fever surveillance in Northland was carried out for the period 2002–2011. The aim of the audit was to establish the accuracy and completeness of surveillance of Acute Rheumatic Fever in Northland, and to provide a robust baseline for future comparison given current rheumatic fever prevention efforts.

Methods Cases of acute rheumatic fever (2002–2011) were identified and evaluated through auditing Northland hospital discharges, the Northland Rheumatic Fever secondary penicillin prophylaxis register and the national EpiSurv database. Cases were included in the audit if they met diagnostic criteria according to the 2008 Heart Foundation guidelines.

Results A total of 114 acute rheumatic fever cases met the audit criteria, an annualised incidence of 7.7/100,000 in Northland. 95% of all cases were Māori with a large disparity between Māori (24.8/100,000) and non-Māori (0.6/100,000). Acute rheumatic fever cases were strongly associated with living in high deprivation areas. This audit noted both under- and over-notification of acute rheumatic fever.

Conclusion Acute rheumatic fever rates in Northland Māori children aged 5–14 (78/100000) are similar to those seen in developing countries and nearly double the rates seen other New Zealand audits. The findings highlight the urgent need to address crowding, poverty and inequitable primary care access if rheumatic fever is to be eliminated.

Acute rheumatic fever (ARF) is a preventable disease associated with poverty, poor access to health care and crowding, and is now rare in most developed countries. It results from an abnormal autoimmune response to Group A streptococcal (GAS) pharyngitis in a susceptible individual. Repeated episodes of ARF can result in structural damage to the heart valves, or rheumatic heart disease (RHD).

This is an important cause of premature death and significant morbidity worldwide, and in Māori and Pacific communities in New Zealand. Rates of ARF in Northland have been historically amongst the highest in New Zealand, and disproportionately impact on Māori children.

The primary aim of this audit was to establish the accuracy and completeness of surveillance for ARF in Northland for the 10-year period 2002–2011 as a robust baseline for future comparison, given current prevention efforts. In addition, we aimed to identify patients with ARF who were not receiving best practice management as per the 2008 Heart Foundation guidelines (i.e. secondary penicillin prophylaxis and specialist follow up).
The population of Northland is estimated at 148,470, with 29% identifying as Māori. The Māori population is significantly younger than non-Māori (36% are aged less than 15 years, compared to 23% of non-Māori) and there are high levels of socioeconomic deprivation, unemployment and one-parent families in Northland, compared with the New Zealand population.

Methods

ARF cases (2002–2011) were identified and evaluated through auditing Northland hospital discharges, the RF register and the national surveillance EpiSurv database. All hospital discharges with Rheumatic Heart Disease (RHD) or ARF aged less than 35 years who were diagnosed during 2002–2011 were identified using the ICD-9 and ICD-10 coding systems (ICD codes 100, 101.0, 101.2, 101.8, 101.9, 102.0, 102.9). All patients on the RF register currently or ever receiving benzathine penicillin prophylaxis for ARF and RHD during the period 2002–2011 were reviewed, along with all Northland cases notified to EpiSurv from 2002–2011.

Cases were included in the audit if they were diagnosed with ARF and met criteria according to the 2008 Heart Foundation guidelines during the period 2002–2011, were resident in Northland and aged less than 35 years at the time of diagnosis.

All clinical notes were sought and reviewed of cases with an appropriate primary or secondary diagnostic code of ARF and RHD. All EpiSurv and RF register notes were reviewed. Information was extracted from case notes and entered into a standard data format. The modified Jones criteria (inclusive of echocardiographic detection of carditis in the absence of a clinical murmur as a major criteria) was used to determine ARF diagnosis of “definite”, “probable” or “possible”.

All “possible” cases were additionally reviewed by a paediatrician to ensure adequate diagnosis. Each case was geocoded according to their place of residence at the time of diagnosis, and deprivation status assigned using NZDep2006. Population statistics were obtained from Statistics New Zealand 2006 Census of population. Analysis was carried out by age and ethnicity.

Results

117 rheumatic fever cases (including six notified recurrences) were identified from EpiSurv. 13 cases were discarded on review, as they did not meet diagnostic criteria for acute rheumatic fever.

Of these, five were recurrences that had insufficient data and did not meet criteria for recurrence, and eight other ARF cases were excluded: five were not ARF and three were diagnosed outside of the area. One notified recurrence was actually a case of initial ARF incorrectly entered as a recurrence in EpiSurv.

157 RHD/ARF cases were identified from hospital discharge ICD coding to ensure RHD cases were not incorrectly coded as ARF and vice versa. Most were excluded as they were RHD rather than ARF, or were outside the audit criteria (for the period or age range). Ten cases met the criteria for ARF that were not duplicates with those on EpiSurv.

The RF register was compared with EpiSurv and 10 cases, all duplicates with those identified from ICD coding above, were identified that were not on EpiSurv. Therefore a total of 114 ARF cases (81 “definite”, 18 “probable” and 15 “possible”) met the audit criteria, an annualised incidence rate of 7.7/100,000 (~12 cases per year). The mean annual number of cases from 2002–2006 was 9.2 (range 7–12), while in the period 2007–2011 it increased to 13.6 per year (range 7–18).
95% of all ARF cases were Māori (n=108), with a large disparity between rates in Māori (24.8/100,000) and non-Māori (0.6/100,000). There were two cases in Pacific children. In the 5–14 age group where the highest rates and largest disparity were found, 94% were Māori (a rate of 78.0/100,000 compared with 4.6/100,000 for non-Māori).

60% (n=68) of cases were male and 40% (n=46) female, with ages ranging from 4–26 years; 85% (n=97) were aged 5 ≤15 years. The mean age was 11.4 years.

ARF cases were strongly associated with living in high deprivation areas and distributed across Northland (Figure 3). Over half (55%, 63 cases) resided in the most deprived decile (NZDep10) and 89.5% (102 cases) resided in NZDep deciles 8–10.
The majority of cases had a definite diagnosis (n=81, 71%) and were low risk (n=92, 81%). At diagnosis 97 (85%) had carditis, 48 (42%) polyarthritis, eight (7%) had chorea and 10 (9%) had erythema marginatum.
There were no cases of nodules recorded, but 48% had no data on the presence or absence of nodules. Of the 10 cases of erythema marginatum, only seven were definite, with one being noted on history and two recorded as “possible”. Three of the eight children presenting with chorea were NZ European (there were only four NZ European ARF cases in the period). The commonest presentation occurring simultaneously was carditis and arthritis (51 cases, 45%).

53 (46%) of the ARF cases gave a history of a preceding sore throat; 26 (23%) had a GAS positive throat swab, with only 16 (14%) having both a sore throat and GAS positive throat swab. There were no data on pharyngitis symptoms for 20 cases, and 21 (18%) had no data on GAS.

46 (40%) had both raised plasma antistreptolysin O titres (ASOT ≥480) and anti deoxyribonuclease B titres (antiDNAse B ≥660). 20 (18%) had raised ASOT only while 18 (16%) had only raised anti DNAse B titres. Of those with positive serological titres, 5 grew Group C and 1 Group G streptococci on throat swab. Of the remaining 30 (26%) with low titres or no titres documented, 9 (8%) had a GAS+ swab, 3 (3%) had chorea, 3 (3%) had rising or falling titres documented and in the remainder (15, 13%), ARF was considered the most likely clinical diagnosis.

Inflammatory markers (ESR and CRP) were both raised in 72 (63%) of ARF cases. 76 (67%) had a CRP >30 and 96 (84%) an ESR >50. In all cases where ESR levels did not meet criteria, none had a raised CRP; however for those that did not have a raised CRP, 14/22 (64%) had a raised ESR. Only two ARF cases had no data for both markers.

All ARF cases were risk allocated based on the severity of carditis as determined by the Heart Foundation guidelines. 92 (81%) were classified as low risk and 22 (19%) medium to high risk. In terms of post-diagnosis follow up, seven of the medium to high risk cases were not receiving best practice care as per HF guidelines, that is, being followed up by a cardiologist.

Of these seven, two have moved out of the region and care has been officially transferred, one did not receive cardiology follow up due to transport issues and four had no cardiology follow up organised. In addition, only one case was documented to have had a dental review in the preceding six months.

Ten ARF cases were not notified and were identified via ICD coding and the RF register (five definite, 3 probable and two possible cases); all have received/are receiving secondary penicillin prophylaxis. Four cases were identified as lost to follow up, and twelve (11%) ARF cases transferred out of the region during 2002–2011. Eight of these cases are now residing in Auckland.

**Discussion**

ARF rates in Northland Māori aged 5–14 (78.0/100000) are similar to those seen in developing countries and nearly double the rates seen in Auckland Māori children during 1993–1999 (41.2/100000) and in the Waikato, 1998–2004 (39.6/100000). Tairawhiti, a region with a similarly high proportion of Māori to Northland, has also documented high rates (total population 7.6/100,000 and 5–14 years 59/100,000 for Māori children 5–14years). Of additional concern, our audit indicates that cases in Northland show an increasing trend over the last 5 years.
As a result of improvements in socioeconomic conditions and primary health care access, ARF and RHD have almost been eradicated from developed nations around the world. High rates of ARF have been shown to be associated with socioeconomic deprivation.\textsuperscript{10} In Northland, the pattern of ARF cases correlates closely with socioeconomic deprivation, with 55\% of ARF cases in Northland living in the most deprived decile (NZDep10).

This audit noted both under- and over-notification of ARF. There were also gaps in the clinical reporting and follow up of ARF. This contributed to most recurrences being excluded, and a lack of data in some key clinical areas, as well as gaps in data on school attended at diagnosis. Lack of systematic use of diagnostic criteria as per the Heart Foundation Guidelines was also noticed.\textsuperscript{3}

Differentiating between major and minor manifestations in the diagnostic assessment was not always well done. Repeat streptococcal titres at 10–14 days were often not performed when initial titres were low. This is important as it is estimated that 50\% of the population are colonised with GAS and rising or elevated streptococcal titres are important to diagnose “definite” ARF.\textsuperscript{3} In addition, the clinical diagnosis of polyarthritis was commonly hard to distinguish from polyarthritis in clinical notes. The inability to weight-bear should prompt a diagnosis of arthritis.\textsuperscript{3}

Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are sensitive but non-specific tests that can be elevated in any inflammatory condition.\textsuperscript{11} The use of CRP in the diagnosis of ARF was noted as early as 1958.\textsuperscript{12} The audit suggests that ESR is more reliable as a positive minor manifestation in the diagnosis of ARF. In cases where the CRP was not raised, 64\% had an ESR >50, while in cases where the ESR was not raised there was no associated rise in CRP.

In the past ESR has been commonly used to monitor ARF. The current guidelines suggest all patients should also have a CRP checked.\textsuperscript{3} As CRP rises and falls faster than ESR this may be useful in uncomplicated cases of carditis to confirm the resolution of inflammation in those who have a prolonged elevated ESR.\textsuperscript{11} CRP could aid in determining the duration of bed rest in low risk patients.

The majority of ARF cases in this audit were low risk suggesting of ARF is being promptly detected in our setting. However the nearly 20\% who were medium/high risk require regular cardiology follow up and this was not always assured. Referral for regular dental review was also poorly documented. Those patients who were not benefiting from best practice have been identified and will be followed up by the Northland Public Health rheumatic fever team.

ARF is preventable. The audit findings highlight the urgent need to address crowding, poverty and inequitable primary care access if rheumatic fever is to be eliminated. Current school-based “sore throat” projects are important for primary prevention of ARF, by enhancing access to timely diagnosis and management of GAS pharyngitis. However, to reach the national goal of reducing ARF by two-thirds in 5 years, greater efforts—in Northland and nationally—will be required.\textsuperscript{13}

This will include improving housing quality and reducing crowding, addressing inequities in household incomes, reducing disparities in access to primary care and increasing awareness of the disease in those communities most affected.
Competing interests: Nil.

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References:

Nurse-led school-based clinics for skin infections and rheumatic fever prevention: results from a pilot study in South Auckland

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Abstract

Aim To assess the acceptability and feasibility of delivering targeted primary health care in a decile one primary school setting.

Method A pilot public health nurse (PHN)-led clinic was set up in a South Auckland primary school (roll ~ 400). The clinic was based on a previous sore throat clinic model with modifications aimed at improving programme feasibility and effectiveness. The timely identification and treatment of Group A Streptococcal (GAS) throat infections to prevent rheumatic fever (RF), and the prevention and treatment of four skin infections (cellulitis, impetigo, infected eczema and scabies) were the focus. The pilot ran for 15 weeks from April to July 2011. Evaluation included documentation review, key school and healthcare stakeholder interviews and parent questionnaires.

Results The consent rate was 92.2%. Of a total 722 throat swabs taken from 337 students, 94 were GAS positive. Ninety-eight assessments of skin conditions were completed at which 76 had a skin infection diagnosed, the most common infection being impetigo (n=46). Thirty-one skin infections were diagnosed in the first week of the pilot. PHN workload was high with a total of 539 phone calls, 137 home visits and 51 school-based parent consultations. The approach was highly acceptable to the majority of key stakeholders. Extrapolating pilot costs results in an estimated annual cost of $510 per student for the programme.

Conclusion It is likely to be both acceptable and feasible to take this model of delivering targeted primary health care to school aged children and use it on a larger scale. The complexity of providing this type of service should not be underestimated and it is essential that robust processes are in place to ensure smooth, safe running of such a programme. Long-term outcome evaluation will be vital to assess programme effectiveness.

In New Zealand many children in disadvantaged areas suffer from a high burden of disease that does not present to primary health care services.¹ In Counties Manukau District Health Board (CMDHB) there are persistent barriers to accessing primary health care that undoubtedly contribute to this lack of medical input.² The development of a primary school based, primary health care programme focussed in low decile schools is one approach that may increase access to primary health care services for children.

Nurse-led school clinics targeting rheumatic fever (RF) prevention have previously been run in South Auckland as part of a randomised controlled trial (RCT).³ These were found to be highly acceptable to the population, with high consent and retention...
rates and achieved a downwards trend in rate of RF, although this did not reach statistical significance. A subsequent meta-analysis of similar school and community programmes indicates a 60% reduction in rate of RF may be achievable through this type of approach.

Re-infection from non-treated household members may have limited the effectiveness of the RCT. An Australian study found that 43% of families who had a primary case of a GAS sore throat had at least one secondary case, which would support this hypothesis. A child in most decile 1 schools in South Auckland has a 1 in 200 chance of developing rheumatic fever by the end of year 8.

This pilot was undertaken to assess whether similar levels of acceptability could be achieved if the RCT clinic model was expanded to target other conditions in addition to RF and modified to try and increase both programme effectiveness and feasibility for a non-study setting.

The RCT identified a high burden of skin infections in enrolled students and recent research has found that skin infections are an increasing area of concern in New Zealand with the incidence of hospitalisation for serious skin infections in children almost doubling from 298.0/100,000 in 1990 to 547.3/100,000 in 2007. Over time there have been disproportionate increases in infection rates in Māori and Pacific children and children from highly deprived areas.

Serious skin infection is the most common medical condition hospitalised in school-aged children from CMDHB. It is reasonable to expect that timely treatment of minor skin infections will prevent progression to more severe infections that require hospital based intervention therefore the prevention and treatment of skin infections was added in this pilot. Cellulitis, scabies, impetigo and infected eczema were specifically targeted.

Methods

A ‘full primary’ (years 1-8), decile one school with a roll of approximately 400 and an ethnic composition of approximately two thirds Pacific and one third Māori, was chosen as the location for the pilot. The study ran for 15 weeks from April to July 2011.

In early 2011 a public health nurse (PHN) and whānau support worker (WSW), a lay worker trained in the recognition of skin infections and swabbing of sore throats, were recruited and students consented. A local General Practitioner was also identified to support the nurse with clinical queries beyond the scope of her practice.

Once the pilot commenced the WSW, working under the supervision and delegation of the PHN, visited each classroom daily to identify students that were symptomatic with a sore throat. Throat swabbing and diagnosis of GAS infection was undertaken as per the methodology used in the RCT. Students with skin infections were referred to the PHN for a full health assessment.

One modification to the RCT clinic model was that the choice of appropriate antibiotics for students diagnosed with GAS throat infections in the pilot was based on recommendations set out in the NZ Rheumatic Fever Guidelines. Oral amoxycillin once daily was therefore supplied by the PHN through standing orders in place of penicillin twice daily.

For skin infections medication choices were guided by specially developed evidence-based peer reviewed skin infection management guidelines. Sodium fusidate ointment was chosen as the topical treatment of choice as it is fully funded (permethrin for scabies). Cephalexin was supplied as the first line oral antibiotic, rather than flucloxacillin, due to relative palatability of the suspension and the fact it can be taken twice daily instead of three times daily.
Standing Orders for antibiotic use by PHNs in the school were ratified by the Middlemore Hospital Medicines Advisory Committee.

Other modifications included encouraging students and families to achieve good adherence to treatment rather than directly observing antibiotic therapy. Families were educated on the importance of completing the full course of antibiotics, students incentivised with sticker charts and families supported by regular phone calls from the PHN.

Assessment and treatment of household members of cases not attending the school (including preschoolers) was also offered, with view to reducing rates of re-infection and improving the health of the wider whānau. Home visits were offered both for families’ convenience and with view to enabling other risks to health (such as inadequate heating, overcrowding) to be identified and addressed.

Documentation was kept such that regular audits of clinical practice could be performed, any deficits remedied and pilot evaluation be carried out. Evaluation was a combination of formative, process and impact evaluation methodology, comprising of documentation review, key informant interviews and parent questionnaires. Outcome evaluation was not attempted due to the small size of the pilot and short time for which it was in operation.

Ethical approval was obtained from the Northern Regional Ethics Committee, Ref. NTX/10/09/097.

**Results**

A total of 434 students were documented as enrolled at the school for all or part of the pilot duration. Of these, 400 (92.2%) were consented into the programme. Eleven students did not return consent forms and 23 (5.3%) declined to take part.

**GAS sore throats**—A total of 722 throat swabs were taken from students of the school. Of these 94 were positive for GAS. Two swabs were re-swabs required due to a delay in treatment onset. Therefore there were 92/720 isolated occurrences of GAS (12.8 % positive). Two symptomatic pre-schoolers were swabbed of whom one had a positive and one a negative GAS result.

Figure 1 shows the number of sore throats swabbed each week. Excluding holidays, the mean number of throat swabs taken per week was 55.4 swabs. The average number of GAS positive swabs per week was 7.1 swabs.

Medication was provided by the PHN in 85/92 cases. The remaining seven students choose to seek medical treatment from their family GP. Sixty-nine of the 81 cases for whom self-reported antibiotic adherence was documented had ‘good’ adherence (full course completed), ten ‘intermediate’ (one dose missed or delayed) and two ‘poor’ (two or more doses missed).

A total of 337 students (84% of those consented) had at least one swab. The number of times each student was swabbed ranged from zero to eight with most students swabbed just once. Eleven students had two GAS positive swabs during the 15 week pilot.
Figure 1. Number of **Group A streptococcal positive**, ***“positive other”*** and negative swabs each week

![Bar chart showing number of swabs per week](chart.png)

* re-swabs excluded.
**“positive other” comprises Group G and Group C streptococcal infections (not treated).
***weeks 3 and 4 were the school holidays.

**Skin infections**—In total, 98 episodes of skin conditions in students were referred to the PHN. In 76 of these a skin infection was diagnosed. The most common infection seen was impetigo (n=46). The most common ‘other’ skin infection diagnosed was infected insect bites (n=6).

Of the 22 students who did not have a skin infection, 10 had non-infected eczema and 10 had other non-infective skin conditions. Two students were considered to be completely well.

Table 1 shows the complete list of skin condition diagnoses made by the PHN.

**Table 1: Skin conditions seen and assessed in the Pilot**

<table>
<thead>
<tr>
<th>Skin infections assessed and diagnosed</th>
<th>Number of students</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impetigo</td>
<td>46</td>
</tr>
<tr>
<td>Infected Eczema</td>
<td>4</td>
</tr>
<tr>
<td>Scabies</td>
<td>10</td>
</tr>
<tr>
<td>Cellulitis</td>
<td>3</td>
</tr>
<tr>
<td>Infected insect bites</td>
<td>6</td>
</tr>
<tr>
<td>Infected scratch or graze</td>
<td>3</td>
</tr>
<tr>
<td>Infected foreign body / suture line</td>
<td>2</td>
</tr>
<tr>
<td>Boil</td>
<td>1</td>
</tr>
<tr>
<td>Cold sore</td>
<td>1</td>
</tr>
<tr>
<td>Other skin infection not specified</td>
<td>3</td>
</tr>
<tr>
<td><strong>Total skin infections</strong></td>
<td><strong>79</strong></td>
</tr>
</tbody>
</table>
Other non-infected skin conditions assessed and diagnosed

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of Students</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eczema – non-infected</td>
<td>10</td>
</tr>
<tr>
<td>Insect bites – non infected</td>
<td>3</td>
</tr>
<tr>
<td>Grazes – non infected</td>
<td>3</td>
</tr>
<tr>
<td>Rash</td>
<td>2</td>
</tr>
<tr>
<td>Blister</td>
<td>1</td>
</tr>
<tr>
<td>Warts</td>
<td>1</td>
</tr>
<tr>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total non-infected skin conditions</strong></td>
<td><strong>22</strong></td>
</tr>
</tbody>
</table>

*Three students had more than one skin infection diagnosed at a single assessment
Two pre-schoolers were diagnosed and treated by the PHN for impetigo.

The number of weekly skin assessments ranged from a high of 40 in week 1 to a low of two in week 11 (Figure 2) (holidays excluded).

**Figure 2: Number of skin infections and non-infected skin conditions diagnosed each week**

The PHN supplied medication for skin infections to 56 students. Forty-five topical treatments were supplied (sodium fusidate 2% ointment or permethrin) and 16 courses of oral antibiotics (cephalexin). Five students required more than one medication. Twenty-four students were treated by their GP, either prior to self-reporting their skin infection to the WSW (n=6) or after (n=18). Three students with impetigo failed to respond to initial topical treatment and required transition to oral antibiotics.

A total of 83 students had a skin condition assessment (21% of those consented). Fourteen students were assessed twice or more of which 10/14 were diagnosed with two separate skin infections, 1/14 had three separate infections and 3/14 were...
diagnosed with one infection and one non-infective skin condition. Three students had multiple skin infections diagnosed at one assessment.

**Other conditions**—A total of 59/81 students with GAS throat infection and all 83 students assessed for a skin infection had their notes reviewed at the end of the pilot. Of these 142 students the pilot staff identified ten students with vision concerns and six with hearing concerns requiring referral to the school’s usual PHN or an optometrist.

**Costs**—Extrapolating pilot costs results in an estimated annual cost of $510 per student comprising $10 for consumables, $80 for diagnostic services and $420 for staffing costs.

**Workload**—The total number of phone calls and home visits made during the pilot were 539 and 137 respectively with an additional 51 parent consultations held at school. The highest numbers of phone calls and home visits made per week were 64 and 21 respectively. The highest number of parent consultations held at school was eight in any 1 week.

**Qualitative findings**—The key informants were universally supportive of the concept of the programme. Increasing the health knowledge and health literacy of all members of the school community was seen to be one of the most essential aspects of the programme, particularly by the school principal who observed that “the potential value in the clinic is immeasurable”.

Provision of free antibiotics by the PHN working under standing orders was also perceived to be a key element. It was universally felt to be both time saving to staff and empowering to families to encourage students and families to maintain good antibiotic adherence compared with directly observing therapy. The nursing team felt that this modification was useful for their professional development and provided them with a valuable opportunity to see the patient journey through from beginning to end. The relationship with primary care was seen as an area that needed strengthening.

Offering assessments to household members required intensive effort to organise and provide. Siblings were generally reported as asymptomatic or were unavailable for assessment even if home visits were undertaken. Many phone calls to families were made in the evenings and at weekends as these were the only times some parents could be contacted. The WSW’s familiarity with Samoan culture and language was found to be invaluable for this particular school community and especially important for home visits.

The opportunity to identify and address underlying housing related risk factors to health was not addressed during this pilot due to workload capacity issues but several key informants felt this modification could add much value from a public health perspective and that even if difficult to undertake the inclusion of this strategy should not be compromised because of cost or other such considerations.

Programme effectiveness was not formally assessed because of the small numbers involved in the study but the school principal considered there were fewer skin infections amongst the school students in term two 2011 compared with term two 2010. During the pilot a student at the school was diagnosed with acute rheumatic
fever (ARF). This student did not report any sore throat symptoms at school prior to their presentation with ARF symptoms and was therefore not throat swabbed in the programme prior to their ARF diagnosis. The working group reviewed the clinical records and found that pilot protocols had been fully adhered to in relation to this student but that this occurrence of ARF highlighted complexities of managing students who become unwell when absent from the school setting due to sickness or holidays.

A total of 37 parent questionnaires (approximately 14%) were returned to school. Of these 34 responded that they feel it is useful to have a PHN and WSW at school to treat children with sore throats and skin infections (two were ‘unsure’ and one replied ‘no’). The other overall themes in the responses were that parents liked the fact that the programme was free and convenient and that their children were seen in a timely fashion. Nearly half of the parent respondents indicated that they had learnt something new about sore throats or skin infections during the pilot.

Discussion

The pilot uncovered a high level of unmet health need amongst students, demonstrating the importance of a programme such as this for the child population in CMDHB. A total of 92 GAS sore throats in just 15 weeks and 31 skin infections in the first week of the pilot represent a significant workload and burden of disease in a school of approximately 400 students.

Notable also are the students identified with hearing and vision concerns who had not been referred to the routine PHN service. The average numbers of swabs taken and GAS positive 5 per week per student are consistent with those of the RCT.3 It is therefore likely that other decile one schools in South Auckland would have similar health need.

The evaluation findings show that this programme is highly acceptable to the majority of key stakeholders. Importantly the school principal’s feedback was very positive. The high consent rate and positive comments obtained from parent questionnaires also indicate that the programme is likely generally acceptable to parents/caregivers although non response bias limits the validity of this finding. A significant proportion of parents chose to take their children to the GP for treatment of skin infections. It may be useful to further explore the reasons underlying this tendency.

It is likely to be feasible to take this model of delivering targeted primary health care to primary school aged children and use it on a larger scale. The programme will need to be well resourced if quality is not to be compromised. Workload is a key issue for programme expansion.

Data collection was seen as time consuming. Providing a lap top computer to enable data to be entered on site at the school and during home visits could potentially resolve this if security issues can be addressed. It will be important to liaise early with diagnostic services to ensure that they have the capacity to manage the volume of throat swabs anticipated.

Although encouraging students and families to take responsibility for achieving good antibiotic adherence appeared acceptable and self-reported levels of antibiotic adherence were high, post treatment swabs were not performed so adherence to
medication could not be measured objectively. It is unclear whether a proportion of the 11 recurrent GAS infections were attributable to poor adherence to treatment rather than being true re-infections and this may need to be investigated further. Swabbing all household members (rather than just symptomatic ones) may be a consideration in regions such as this where RF is endemic.

During the pilot the PHN was unable to incorporate housing related health risk identification into her list of responsibilities due to workload capacity. Greater resourcing of the programme may be required to achieve this modification. The small numbers of household members being swabbed for sore throats and assessed for skin infections was disappointing. The reasons for this should be explored so that the full potential of this strategy can be realised.

The main strength of this approach is that it provides access to primary health care for certain targeted conditions of concern to children for whom there are well documented barriers to accessing primary health care at a population level. A focus on high risk low decile schools has the potential to reduce both the socioeconomic and ethnic disparities apparent in the child hospitalisation rates for the targeted conditions.

Additionally the incidence of rare sequelae such as post streptococcal acute glomerulo-nephritis and bone and joint disease may be reduced. While this pilot programme only included RF prevention and the identification and treatment of skin infections, it is likely that other health needs could be addressed through this model. Currently the feasibility of adding in injury prevention is being explored.

This pilot showed that school based clinics can deliver health care to children in a timely fashion, (particularly essential for infectious conditions) in a way that is convenient for them, and makes good use of frontline staff. The use of WSWs provides the dual benefits of assisting workforce development as well as strengthening cultural acceptability of the programme.

The ARF case highlighted the fact that it is possible, with a school-based programme, to miss students who become unwell during the holidays or who are absent from school for a prolonged period of time for any reason. Clear processes to cover holidays and to identify and follow up absent students need to be integral components of a school-based service. Involvement with GPs and PHOs is essential as this type of programme should be done in partnership with primary care services and not as a stand-alone initiative.

A wider roll out should be instigated with evaluation in mind so that the effectiveness and clinical safety of the programme can be assessed and monitored and the programme further modified as needs require. If multiple similar programmes are set up in New Zealand these should be standardised such that the results of all programmes can be combined to increase the statistical power to demonstrate any change in rates of rheumatic fever, hospitalisation of skin infections or other designated outcomes.
Competing interests: Nil.

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References:
Temperature management in haematology patients with febrile neutropenia: a practice survey

Robert Weinkove, Jennifer Clay, Catherine Wood

Abstract

Aim To assess the attitudes of clinicians to temperature management in haematology patients with febrile neutropenia.

Method An online scenario-based survey was circulated to consultant members of the New Zealand branch of the Haematology Society of Australia and New Zealand, to haematology advanced trainees, and to nursing representatives at each haematology department in New Zealand.

Results Eighty-eight responses were obtained, from 34 doctors and 54 nurses. Most respondents would advise a neutropenic patient to take paracetamol as needed for pain. Median temperature intervention threshold for an asymptomatic patient with febrile neutropenia was higher for doctors than for nurses (38.5 versus 38.0°C), despite considerable heterogeneity. Both groups indicated they would intervene at a median 38.0°C for a patient with rigors. Paracetamol was the preferred first-line cooling measure, with physical methods second-line, and pethidine third-line. All respondents favoured oral over intravenous or rectal paracetamol. Most believed a clinical trial of antipyretic treatment for febrile neutropenia was warranted, and indicated willingness to enrol their patients in such a study.

Conclusion This survey documents clinicians’ preferred temperature intervention thresholds and methods for haematology patients with neutropenic fever, and shows considerable variation in practice. Most respondents supported a trial of antipyretic management in febrile neutropenia.

Severe neutropenia is a risk factor for sepsis.1 Febrile neutropenia is commonly defined as a single fever of ≥38.3°C or a temperature of ≥38°C for at least 1 hour, in the context of a neutrophil count of <0.5×10⁹/L, or <1.0×10⁹/L with the expectation of a decline to <0.5×10⁹/L in subsequent days.2

Febrile neutropenia occurs in the majority of patients undergoing acute leukaemia induction and autologous and allogeneic stem cell transplantation,3,4 and carries a high mortality without prompt antibiotic treatment.1 Guidelines for the investigation and antimicrobial treatment of neutropenic fever have been published2,5,6.

Fever is a natural response to infection, and may be beneficial to the outcomes of sepsis: compared to 37°C, temperatures within the febrile physiological range inhibit in vitro growth of some bacteria,7 and enhance antimicrobial sensitivity.8 On the other hand, antipyretics such as paracetamol improve patient comfort,9 and may have favourable haemodynamic effects.10 The authors’ experience suggests that the attitudes of haematology clinicians to cooling measures in patients with...
neutropenic fever vary, with some encouraging, and others discouraging, the use of cooling measures.

We aimed to assess the attitudes of haematology clinicians in New Zealand to the management of fever in patients with febrile neutropenia using a practice survey.

Method

A survey asking respondents about their management approach to three clinical vignettes was designed. The first scenario was of a neutropenic patient at home asking their clinician whether they could use paracetamol as an analgesic: respondents were asked to state whether they would advise the patient to take paracetamol regularly, as needed, or to avoid paracetamol.

The second and third scenarios related to a patient with severe neutropenia and fever who has already commenced first-line antimicrobials: respondents were asked at which temperature they would intervene with cooling measures (with options in 0.5°C bands) if the patient were asymptomatic, or symptomatic with rigors. Respondents were then asked which physical or pharmacological cooling measures they would use, and whether paracetamol would be administered as needed or regularly, and via which route.

Respondents were asked at what time point they would use fever as a determinant of empiric antimicrobial change in a patient with febrile neutropenia. Finally, respondents were asked whether they felt a clinical trial of antipyretic treatment in febrile neutropenia was warranted, and whether they would be willing to enrol their patients in such a trial. The survey is in Appendix 1.

The survey was piloted in the authors’ own haematology department, and changes made to improve clarity, and add to response options. A link to the online survey was then emailed to all current medical and nursing members of the New Zealand branch of the Haematology Society for Australia and New Zealand (HSANZ), to all haematology trainees in New Zealand, and to a nursing representative at each haematology centre in New Zealand, for distribution to other nursing staff.

Survey responses were collected using an online survey tool (SurveyMonkey.com, Palo Alto, California, USA), exported to an Excel spreadsheet (Version 12.3.0, Microsoft Corporation, Redmond, Washington, USA), and analysed using Prism statistical software (Version 5.0d, GraphPad Software, La Jolla, CA, USA). All data were analysed using non-parametric statistical tests. The a priori subgroups of doctor and nurse professionals were analysed separately. Temperature thresholds (in 0.5°C bands) were treated as continuous variables for analysis. A probability value of p < 0.05 was considered significant. This online practice survey was classified as low-risk, and did not require formal ethical review according to the guidelines of the Central Regional Ethics Committee of New Zealand.

Results

Eighty-eight responses were received, from 20 consultant haematologists, 14 haematology trainee doctors, 18 senior nurses (charge nurses, clinical nurse specialists or nurse educators), and 36 ward or day unit nurses. Approximately 45 consultant haematologists and 25 haematology registrars are practising in New Zealand (Dr Bart Baker, personal communication), giving an overall response rate of 48% among doctors. The total number of nurses practising in haematology in New Zealand is not known. The number of respondents from each professional group, and duration of practice in clinical haematology, is given in table 1.
Sixty-eight percent of doctors and 44% of nurses indicated that they would allow a neutropenic patient to take paracetamol as needed for pain, and a further 15% of doctors and 7% of nurses would allow a neutropenic outpatient to take regular paracetamol. Nurses were significantly more likely than doctors to advise a neutropenic patient to avoid paracetamol (p < 0.05, Fisher’s exact test).

Figures 1A and 1B show the temperature thresholds at which respondents would intervene to lower temperature in an asymptomatic and a symptomatic patient with febrile neutropenia, respectively.

For asymptomatic patients, the temperature threshold for intervention varied widely between respondents, but the threshold was significantly higher for doctors than for nurses (median 39°C for doctors, 38.5°C for nurses; p<0.01, Mann Whitney test).

For symptomatic patients, the median intervention threshold was 38°C for both doctors and nurses (difference not significant). Considering all survey respondents, there was no significant correlation between years of experience in haematology and temperature intervention threshold for either symptomatic or asymptomatic patients (data not shown).

**Figure 1. Reported temperature intervention thresholds in severe neutropenia**
Fifty-five percent of respondents favoured paracetamol as the first line intervention to reduce temperature, with a further 44% selecting physical cooling measures. Forty-five percent of respondents favoured physical cooling as the second line intervention, with 30% selecting paracetamol. Sixty-eight percent of respondents selected pethidine as the third line intervention. Other measures, such as non-steroidal anti-inflammatory drugs (NSAIDS), cyclo-oxygenase 2 (COX-2) inhibitors, and other opiates, were favoured by fewer respondents. These data are presented in Figure 2.

Figure 2. Preferred temperature-lowering interventions in febrile neutropenia

Eighty-three respondents answered the question about frequency of paracetamol administration in inpatients with febrile neutropenia. Of these, seventy-two (87%) reported that they would administer or prescribe paracetamol only as needed, and three (4%) would prescribe or administer paracetamol regularly. Eight (10%) reported that they would never use paracetamol in this setting.

Regarding the route of paracetamol use in patients with febrile neutropenia, all 85 respondents to this question reported that they would use oral paracetamol as the first choice. Forty-nine respondents selected an alternative route of administration in case oral paracetamol could not be given, of which 39 respondents favoured intravenous paracetamol and ten favoured rectal paracetamol. Sixty-four percent of respondents to this question (54/85) reported that they would avoid rectal paracetamol in patients with neutropenic fever.

Among the 83 respondents who selected at least one choice of physical cooling method, the most frequently selected options were removal of clothes (83% of
respondents), provision of a fan (81%), tepid sponging (58%), and provision of a wet towel or flannel (53%). Fewer than ten percent of respondents selected the use of ice packs, cooling blankets or intravenous fluids for physical cooling.

Seventy-five respondents answered the question about time to change of antimicrobials. Of these, the majority stated that they would consider an antibiotic change at either 48 or 72 hours, with 39% (29 respondents) selecting each of these time points. A further 9% would change at 24 hours, 4% at 36 hours and 1% at 96 hours. Eight percent (6/75) indicated that they had no fixed time for antimicrobial change in this situation.

Sixty-nine percent of respondents (51/74) stated that they would be willing to enter their patients into a randomised study of antipyretic management in febrile neutropenia. A further 27% (20/74) were unsure. Three respondents to this question (4%) stated that they would not be willing to enter their patients in such a study. Asked whether there were specific groups of patients they would not be willing to enter into a randomised study, respondents nominated the following groups: elderly patients (3% of all respondents), children (3%), stem cell donors (5%), autologous stem cell transplant recipients (8%), allogeneic stem cell transplant recipients (18%).

Discussion

This practice survey reports the attitudes of haematology doctors and nurses to antipyretic treatment for patients with neutropenic fever.

The survey indicates that overall, most respondents would advise a neutropenic patient to take paracetamol as needed for pain, but that nurses were significantly more likely than doctors to advise patients to avoid paracetamol. In febrile neutropenia, thresholds for temperature-lowering interventions varied widely, but nurses reported they would intervene at a significantly lower temperature than doctors in a patient without rigors.

In a symptomatic patient, both professional groups would intervene at a median of 38.0°C. Most clinicians would use either paracetamol or physical measures as a first-line intervention, with a narrow preference for paracetamol. Physical measures were the favoured second-line, and pethidine was the favoured third-line, cooling intervention.

Oral paracetamol was preferred over the intravenous route, and most respondents would avoid the rectal route. Finally, the majority of respondents believed a clinical study of antipyretic treatment in febrile neutropenia was warranted, and would consider entering their patients in such a study.

To the authors’ knowledge, this is the first survey of temperature management in febrile neutropenia. In collaboration with the New Zealand branch of the HSANZ, we were able to survey nearly half of all haematology doctors working in New Zealand. The majority of respondents who commenced the online survey completed it, with 95% and 84% response rates to the penultimate and final survey questions, respectively. This study employed scenarios to assess clinical practice, an approach that has been validated in a variety of settings.11,12
Although we were able to calculate a response rate for doctors, the survey response rate among nurses is unknown due to a lack of a central register of haematology nurses in New Zealand, and a large number of haematology patients being cared for in mixed-specialty wards. Nonetheless, we believe the response rate among nurses to be lower than that among doctors.

Among both doctors and nurses, the voluntary nature of survey participation may result in bias, so the current findings may not be representative of all clinicians. Finally, reported preferences in a survey may not reflect actual clinical practice. We intend to address some of these points by undertaking an observational study of paracetamol usage among inpatients with febrile neutropenia.

The authors are not aware of any published surveys of fever management in neutropenic fever, but clinicians’ attitudes to fever have been investigated in other infection scenarios. In a review of fever management among critical care clinicians in Australia and New Zealand, doctors reported a significantly higher mean temperature intervention threshold than nurses (39.0°C vs 38.5°C), similarly to this study. The temperature intervention thresholds in the critical care study were higher than in the current study, possibly because many intensive care patients are sedated, so fever-related symptoms are less of a concern.

A study of fever management by paediatric junior doctors indicated a mean antipyretic treatment threshold of 38.6°C, with alternating aspirin and paracetamol as the favoured intervention. Very few respondents favoured non-steroidal anti-inflammatory drugs in the current study, possibly due to concerns about impairing platelet function in neutropenic patients, many of whom are also thrombocytopenic.

In a survey of fever management among Swiss paediatricians, a temperature threshold of 38.5°C was identified as a threshold for treatment, with the vast majority favouring paracetamol. In this study, the favoured routes of paracetamol administration were rectal for 18 month olds, and oral for older children. The widespread reluctance to use rectal paracetamol in the current study is likely to reflect concern about inducing bacteraemia in the neutropenic patient.

The role of antipyretics in the management of infection remains unclear. Observational studies have found that the absence of fever is associated with increased mortality in patients admitted to intensive care units with infection, and that the use of antipyretics is associated with increased mortality in septic, but not in non-septic patients.

A number of interventional studies support the notion that fever is an important physiological response to infection: in children with falciparum malaria, the administration of regular paracetamol was associated with delayed resolution of parasitaemia, and in healthy volunteers infected with rhinovirus, regular paracetamol administration was associated with a reduced antibody response and a prolongation of symptoms.

A single randomised trial comparing aggressive temperature control to permissive hyperthermia in patients with sepsis in intensive care found a trend towards reduced mortality in the permissive hyperthermia group. This finding is yet to be replicated.
The present study assesses clinician preferences regarding antipyretic treatment in febrile neutropenia using a scenario-based survey. This survey demonstrates a lack of clear consensus on thresholds for, and methods of, lowering temperature, which is understandable given the lack of evidence. Establishing the role of antipyretics in neutropenic fever would require a prospective randomised controlled trial, for which the majority of respondents to this survey indicate support.

**Competing interests:** Nil.

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**References:**


Appendix 1: Survey questions

1. What is your role in the management of haematology patients?
   - Consultant Haematologist (doctor)
   - Haematology Registrar
   - Other doctor in training
   - Clinical Nurse Specialist (in haematology and/or oncology)
   - Ward Nurse (in haematology or oncology)
   - Day Unit Nurse (in haematology or Oncology)
   - Other (please specify)

2. For how many years have you practised in haematology?

The following questions relate to clinical scenarios. Please indicate what your clinical practice would be.

3. A patient has just received chemotherapy which is expected to cause severe neutropenia, and is to be discharged from hospital. They ask if they can use paracetamol at home, for joint pain. What do you advise?
   - Yes, take paracetamol regularly for pain
   - Yes, take paracetamol as needed for pain, but avoid taking it regularly
   - No, do not take paracetamol
   - Other, please specify

4. A patient with fever and severe neutropenia following chemotherapy has already started first-line antibiotics. The patient is currently ASYMPTOMATIC. At what temperature would you intervene with medications or physical cooling measures?
   - 37.0, 37.5, 38.0, 38.5, 39.0, 39.5, 40.0, 40.5, 41.0, 41.5°C
   - I would not intervene at any temperature
   - Other (please specify)

5. A patient with fever and severe neutropenia following chemotherapy has already started first-line antibiotics. The patient complains of RIGORS and SWEATS. At what temperature would you intervene with medications or physical cooling measures?
   - 37.0, 37.5, 38.0, 38.5, 39.0, 39.5, 40.0, 40.5, 41.0, 41.5°C
   - I would not intervene at any temperature
   - Other (please specify)
6. You have decided to reduce the temperature of a patient with febrile neutropenia. What would be your first, second and third-line method(s) of reducing body temperature? If you would use more than one method at a time, please select all that apply.

<table>
<thead>
<tr>
<th>Variables</th>
<th>First line</th>
<th>Second line</th>
<th>Third line</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paracetamol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical cooling measures (e.g. fan, sponging, cooling blanket)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-steroidal anti-inflammatory drug (e.g. ibuprofen, diclofenac)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COX-2 selective inhibitor (e.g. celecoxib, paracoxib)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pethidine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Other (please specify)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

7. If you ever prescribe or administer paracetamol in febrile neutropenia, do you usually provide it regularly or as needed (PRN)?

- Regular paracetamol
- As needed (PRN)
- I never prescribe or administer paracetamol for febrile neutropenia

8. If you ever prescribe or administer paracetamol in febrile neutropenia, what route of administration do you prefer?

<table>
<thead>
<tr>
<th>Variables</th>
<th>First choice</th>
<th>Second choice</th>
<th>Third choice</th>
<th>I avoid this route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral paracetamol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intravenous paracetamol</td>
<td></td>
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</tr>
<tr>
<td>Rectal paracetamol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Comments:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If you ever recommend or apply physical cooling methods, which do you use (select all that apply)?

- Removal of clothes or coverings
- Tepid sponging
- Wet towel or blanket
- Fan
- Ice packs
In the planned clinical trial, we anticipate randomising patients with febrile neutropenia to receive paracetamol or placebo. Patients will start first-line antimicrobial therapy before paracetamol/placebo treatment. Patients will stop paracetamol/placebo before assessing the need for second-line antimicrobials. Antimicrobials can be changed at any time if culture results/antibiotic sensitivities or clinical features (e.g. hypotension, clinical deterioration) demand.

9. In the trial, patients would stop receiving paracetamol/placebo before a planned assessment of the need for second-line antimicrobials. At what time point would you empirically change to second-line antimicrobials in a patient with neutropenic sepsis and ongoing fever?

- After 24 hours (1 day)
- After 36 hours (1.5 days)
- After 48 hours (2 days)
- After 72 hours (3 days)
- After 96 hours (4 days)
- I do not have a fixed time before antimicrobial review
- Other (please specify)

10. Are there any groups of patients who you would NOT want to include in a randomized trial of paracetamol in febrile neutropenia? If not, why not (use comments box)?

- Elderly patients
- Autologous stem cell transplant recipients
- Patients having stem cell harvest
- Allogeneic stem cell transplant recipients
- Other patient groups / comments

11. Do you believe a clinical trial of antipyretic (paracetamol) use in febrile neutropenia is warranted?

- Yes
- No
- Unsure
12. Would you be willing to randomise your patients in a randomised trial of paracetamol for febrile neutropenia?

- Yes
- No
- Maybe

14. Would you like to add any further comments?
The influenza pandemic of 1918-19 in two remote island nations: Iceland and New Zealand

Jennifer A Summers, Nick Wilson, Michael G Baker, Magnús Gottfredsson

Abstract

Aim Nations varied in their experience of, and response to, the 1918-19 influenza pandemic. Island communities can provide unique opportunities to study the epidemiology of infectious diseases. We aimed to compare the epidemiology and public health response to this pandemic in two remote island nations, on opposite sides of the globe: Iceland and New Zealand (NZ).

Method Historical accounts in both nations were reviewed, along with recent analysis of the pandemics impact and course.

Results Marked similarities were noted in epidemic timing, failure of border control, shape of epidemic curves, and delayed use of public health interventions. However, amongst the exposed European populations, Iceland experienced a significantly higher mortality rate (830 vs 550 per 100,000) compared to NZ (rate ratio: 1.5, 95%CI: 1.4-1.6).

There is evidence that some public health measures in specific areas of both nations resulted in lower mortality rates. In particular, Iceland’s use of travel restrictions and ship quarantining, appeared to protect 36% of the population.

Conclusion The epidemiology of the 1918-19 influenza pandemic was fairly similar for the exposed European populations of Iceland and NZ. Nevertheless, major differences were the significantly higher overall mortality rate in Iceland and the success of Iceland’s use of travel restrictions.

Nations varied in their response to the 1918-19 influenza pandemic; however, certain epidemiological characteristics of this pandemic were similar in many locations.

Similar characteristics of the pandemic included waves of varying intensity, with the second wave generally causing the highest mortality; excess mortality amongst young adults; lower socioeconomic status associated with increased mortality risk; and some evidence, although conflicting, that rurality may have been protective.

Public health interventions included the use of quarantine, mass gathering restrictions, and closure of public facilities.

Island communities provide unique opportunities to investigate the epidemiology of infectious diseases. In this study, we aimed to compare the epidemiological characteristics of the 1918-19 influenza pandemic in two relatively large and geographically remote island nations, from opposite sides of the globe: Iceland and New Zealand (NZ).
Additionally, both nations predominantly had European populations in 1918, had some public health infrastructure, have good historical documentation and well-defined population data for 1918 (size and location).

Both nations were exposed to the first wave of the pandemic strain mid-1918 via ships with infected passengers, as neither country implemented full maritime quarantine prior to exposure. Therefore, we also compared the epidemiology of the pandemic and public health responses in the two countries.

Method

Data on pandemic influenza deaths and population figures in 1918 from Iceland were obtained from previously published work by one of the authors, along with other accounts and records (including Statistics Iceland data: www.statice.is). NZ’s pandemic mortality and population data were retrieved from historical records, along with historical accounts and more recent analyses.

Unlike Iceland, NZ has an indigenous population (Māori). But for comparability in this analysis, only deaths amongst the NZ European population were used, due to both the disproportionate mortality amongst the indigenous population and the higher quality of European based records. This restriction has led to a slight underestimate of the overall NZ population pandemic burden.

Given the strong evidence for successful isolation of the eastern and northern populations of Iceland during the pandemic’s second wave, we calculated the pandemic mortality only in the ‘exposed’ population, assuming an equal age-distribution in all regions of the country.

Results

Timing, mortality and geographical spread—Iceland, the smaller of the two nations in both population and landmass, is estimated to have had 484 pandemic-attributable deaths during 1918. These deaths occurred exclusively in the southern and western parts of the country. In contrast, the 1918 pandemic spread to almost all areas of NZ, mainly via coastal shipping routes and along national railway lines.

An estimated 6,000+ deaths occurred amongst New Zealanders of European descent. Iceland had a higher mortality rate (830 vs 550 per 100,000) amongst individuals of European ethnicity in the exposed population. This difference was statistically significant (rate ratio: 1.5, 95%CI: 1.4-1.6). Estimates for NZ suggest that 30-50% of the population was affected with symptomatic illness from the pandemic, whilst estimates in Iceland suggest an attack rate of 66% and as high as 80-90%.

The first pandemic wave was experienced by both nations between July and October 1918, and was reportedly mild. However, the second wave, commencing in late October 1918, exacted the largest mortality burden. This wave peaked in three weeks at roughly the same time (mid-November 1918) for both Iceland and the North and South Islands of NZ (Figure 1).

Iceland did not experience a noticeable third wave in 1919 (it is unclear for NZ); however, there is some evidence that previously unexposed populations in Olafsvik and Dyrholahreppur, Iceland experienced a severe influenza outbreak in mid-1921, clinically identical to the 1918-19 pandemic (unpublished diary and personal communication by Þórður Tómasson, Curator of Skogar Museum).
Age specific mortality, comparison between urban and rural areas—Both nations observed a “W-shaped” age distribution in mortality rates (Figure 2), characterised by relatively high pandemic mortality rates amongst young adults. However, the exposed Icelandic population experienced a higher rate per 100,000 in nearly all age-groups compared with NZ, most noticeably in the older population (60+ years). Reykjavik (Iceland’s capital) had a noticeably higher overall mortality rate (1700 per 100,000) compared with the four major NZ cities (range: 390 to 790 per 100,000). Lower mortality rates were generally experienced in rural areas of NZ (705 per 100,000 in cities/towns compared to 330 per 100,000 in rural areas); similar to Iceland, with a noticeably lower mortality experienced in rural areas (523 per 100,000) compared to the urban environment of Reykjavik. Crowded living conditions in Iceland and lower socioeconomic status in NZ were cited as risk factors during 1918.
Effects of travel restrictions, quarantine and other interventions—Few areas in NZ achieved any form of partial or full quarantine. One NZ town (population: ~1000) is noted as enacting a successful form of quarantine, resulting in lower mortality compared with surrounding areas. Iceland successfully introduced partial travel restrictions: a locally-initiated road patrol block on the main road leading to the northern part of the island, and a guard by a natural barrier of an unbridged glacial river, crossing the road to the eastern part. These measures were followed by ship quarantining which together, provided protection from the pandemic strain, sheltering 36% of the Icelandic population.

Interventions such as the closure of schools/shops and restrictions to mass gatherings differed between the countries. NZ’s response was varied, with social distancing policies between regions inconsistently implemented and introduced late during the pandemic period.

Conversely, Icelanders initiated more consistent interventions such as restrictions on mass gathering and closure of public facilities (notably publicly initiated), but most measures were delayed.

Community-based organisations in both nations contributed to the pandemic response, such as provision of food and supportive care. In post-pandemic years, changes to strengthen public health legislation were adopted in both NZ and Iceland.

Health services in both nations were stretched, with temporary/auxiliary hospitals created to deal with the pandemic cases. During 1918, Iceland had more doctors per head of population compared to NZ (80 vs 60 per 100,000), mainly due to one-third
of NZ doctors serving overseas as part of World War One. However, NZ did have an established nursing workforce (n=1,675), unlike Iceland.

**Discussion**

Influenza was a notifiable disease in Iceland before the pandemic, unlike NZ, which only initiated/implemented this requirement in the midst of the pandemic.

As both are relatively remote islands, requiring sea voyages of days to weeks to reach them in 1918, they had a potential geographic advantage compared with most other countries which have land borders. Yet, officials in both nations delayed in enacting responses or decided not to respond at all. In contrast, a few geographically isolated countries and areas did manage to exclude pandemic influenza in 1918-19, but only by very active border control policies.  

The relatively high mortality amongst young adults for both nations is consistent with other studies in various populations during the 1918-19 influenza pandemic. This distinct vulnerability for individuals aged 20-40 years suggests that host factors may have played a role in determining mortality risk for this particular pandemic influenza strain, although results are conflicting. Of note is the high mortality rate experienced by the elderly in Iceland.

There is evidence from both nations that deprivation may have played a role in determining mortality risk from pandemic influenza. Deprivation has long been cited as a mortality risk for infectious diseases, so this result is not surprising. Conversely, rurality is suggested as a protective factor for pandemic-related mortality risk in both Iceland and NZ.

Previous research has hypothesised that less exposure to the 1918-19 pandemic strain (eg, remoteness reducing risk of exposure to infected people), differing health care access, or various other socio-demographic variables (eg, less crowding) may have contributed to the observed protective effect of rurality in some populations. However, the results are conflicting, and more research is required in this area.

We can only speculate as to the possible reasons for the higher mortality and estimated morbidity rate in Iceland, particularly given the arguably more effectively implemented public health interventions employed compared to NZ. One possibility is the impact of seasonality, since in late 1918 Iceland was entering its winter season, while NZ was just entering its warmer summer months.

The NZ population may have had some form of residual protective immunity having just passed through its seasonal influenza period. Seasonality could have impacted on transmission levels with Icelanders spending more time indoors in close proximity to others; as well as being exposed to additional respiratory stressors such as indoor air pollution from cooking and heating.

Furthermore it can be speculated that different levels of pneumococcal colonisation amongst Icelanders and New Zealanders may have played a role. Potential nutritional/dietary variations may also conceivably have caused differences in immune responses between the populations of these two nations.

It is worth mentioning the element of latitude differences (NZ is closer to the equator than Iceland), which may correlate with less sun exposure in Iceland. Both the time of
year and latitude could have resulted in lower vitamin D levels (resulting from less ultraviolet light exposure) among Icelanders.

Low vitamin D levels have been suggested as a risk factor for increased mortality during the 1918-19 pandemic in a US study.\textsuperscript{16} Also there is some (albeit incomplete) more modern evidence for low vitamin D contributing to influenza risk.\textsuperscript{17} It has also been suggested that “annual light/dark cycle and mediated by the pattern of melatonin secretion, might account for many heretofore unexplained features of infectious disease seasonality”.\textsuperscript{18}

Furthermore, the winter conditions in Iceland may have favoured the transmission of influenza virus (since cold and dry conditions favour transmission\textsuperscript{19}) and also reduced ultraviolet light levels may have favoured virus survival (given evidence for sensitivity to this light\textsuperscript{20}).

Given global air travel and the difficulty containing spread of the 2009 influenza pandemic, island nations probably cannot fully rely on border control for protection from future pandemics.\textsuperscript{21} Although very small islands might benefit from travel volume reduction\textsuperscript{22}, larger islands will need other interventions.

Iceland’s successful use of ‘protective sequestration’ and local ship quarantine, sheltering one-third of the population, add to previous evidence to suggest that such approaches are worth considering in places where travel movements can be easily controlled.\textsuperscript{23} Furthermore, the late outbreak of influenza (probably of the same pandemic strain) in Iceland in 1921, reinforces the importance of careful monitoring of previously unexposed populations after a pandemic.

In summary, the epidemiology of the 1918-19 influenza pandemic was fairly similar for the exposed European populations of Iceland and NZ. Nevertheless, major differences were the significantly higher overall mortality rate in Iceland compared to NZ and the success of Iceland’s use of protective sequestration and localised ship quarantining which protected 36% of the population.

Competing interests: Nil.

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References:
Isolation of *Mycobacterium thermoresistible* from a mesh used in an incisional hernia repair

Niall Hamilton, Graeme Roadley

**Abstract**

We present a case of *Mycobacterium thermoresistible* infection from a hernia repair mesh, the first reported case of this infection in New Zealand.

*Mycobacterium thermoresistible* infection is rare, with only seven recorded cases in the literature. The presence of this isolate has implications for antibiotic regime and treatment duration. In this report we detail the case particulars and a brief summary of the previously documented cases.

The incidence of non-tuberculous mycobacterial disease has been increasing, with an widening variety of species isolated both worldwide and within New Zealand.1,2

*Mycobacterium thermoresistible* is an environmental species, isolated from dust and soil3, and has only been involved in a handful of infections in humans.

We describe the first case of *Mycobacterium thermoresistible* infection in New Zealand, the eighth reported case in the medical literature4.

**Case report**

The patient is a 46-year-old woman with a history of breast cancer. She had previously had a right mastectomy and TRAM reconstruction. Following this she developed an abdominal incisional hernia, and required two operations to repair this using a mesh.

She presented 4 weeks postoperatively with erythema and induration around the repair site. There was clinical evidence of a collection and 300 ml of pus was aspirated and cultured. There was no growth on trypticase soy agar with 5% sheep blood or chocolate agar following 5-day incubation at 37°C.

A large number of leucocytes were seen on Gram stain, but no organisms. Given this result, the agar plates were reincubated. The patient was empirically treated with IV gentamicin, cefuroxime and metronidazole as an inpatient with a significant clinical improvement. She was discharged after a 4-day course, before the final culture results had returned.

The agar plates grew a rapidly growing *Mycobacterium* species at day 7. While further identification was pending the patient presented again with similar symptoms. A CT scan of her abdomen showed evidence of infection around the hernia mesh, but no significant collection. She had a further week course of IV gentamicin, cefuroxime and metronidazole and was discharged on a 10-day course of amoxicillin-clavulanic acid.
The *Mycobacterium* species was confirmed as *Mycobacterium thermoresistible* using and Heat Shock Protein 65 (*hsp65*) gene analysis (assay as described by Kim et al\textsuperscript{5}) with 99% similarity (482 out of 485 nucleotides) of the polymerase chain reaction (PCR) product to the GenBank entry ([www.ncbi.nlm.nih.gov](http://www.ncbi.nlm.nih.gov)) for *M. thermoresistible*. Unfortunately the isolate was too slow-growing for accurate antimicrobial sensitivity testing.

The patient was admitted electively for removal of the infective mesh and was started on a 3-month course of ciprofloxacin, doxycycline and rifampicin postoperatively.

She remained well on completion of the antibiotic course with no further complications.

**Discussion**

*Mycobacterium thermoresistible* was first isolated from soil samples in 1966,\textsuperscript{4} and subsequently house dust, by Tsukamura in Japan.\textsuperscript{3} As the name suggests, it is able to grow up to temperatures of 52°C, distinguishing it from other biochemically similar *Mycobacterium* species like *M. gordonae*.

Human infection is rare, but is not restricted to immunocompromised hosts, with over half the reported cases occurring in patients with no significant background of immune deficiency (Table 1).

### Table 1. Summary of previous *M. thermoresitibile* case reports

<table>
<thead>
<tr>
<th>Case report</th>
<th>Method of identification</th>
<th>Isolate origin</th>
<th>Immunocompetent status</th>
<th>Antibiotic regime</th>
<th>Length of treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weitzman et al 1981\textsuperscript{6}</td>
<td>Culture and Biochemical profile</td>
<td>Lung tissue biopsy</td>
<td>Presumed immunocompetent</td>
<td>Rifampin, ethambutol, and streptomycin</td>
<td>Not specified</td>
</tr>
<tr>
<td>Liu, Andrews and Wright. 1984\textsuperscript{7}</td>
<td>Culture and Biochemical Profile</td>
<td>Lung tissue biopsy</td>
<td>Immunocompromised (hypogammaglobulinaemia)</td>
<td>Rifampin, ethambucil and streptomycin</td>
<td>Not specified</td>
</tr>
<tr>
<td>Neeley and Denning. 1989\textsuperscript{9}</td>
<td>Culture, biochemical profile and HLPC</td>
<td>Cutaenous nodule (thoracotomy scar site)</td>
<td>Immunocompromised (heart transplant recipient, diabetic)</td>
<td>Isoniazid, rifampin, ethambutol</td>
<td>Not specified</td>
</tr>
<tr>
<td>Wolfe and Moore. 1992\textsuperscript{10}</td>
<td>Culture, biochemical profile and high-performance liquid chromatography (HLPC)</td>
<td>Breast abscess (following mammoplasty)</td>
<td>Presumed immunocompetent</td>
<td>Rifampicin, ethambutol</td>
<td>16 months</td>
</tr>
<tr>
<td>Cummings et al 2000\textsuperscript{12}</td>
<td>Culture, biochemical profile and HLPC</td>
<td>Cutaenous lesion (hands)</td>
<td>Presumed immunocompetent</td>
<td>Doxycycline and levofloxacin</td>
<td>3 months</td>
</tr>
<tr>
<td>LaBombardi, Shastry, and Tischler. 2005\textsuperscript{11}</td>
<td>Culture, biochemical profile and HLPC</td>
<td>Prosthetic knee joint</td>
<td>Presumed immunocompetent.</td>
<td>Moxifloxacin and linezolid (doxycycline replacing linezolid)</td>
<td>8 months</td>
</tr>
<tr>
<td>Neonakis et al 2009\textsuperscript{8}</td>
<td>Culture, biochemical profile, 16S RNA and hsp65 polymerase chain reaction (PCR)</td>
<td>Sputum</td>
<td>Presumed immunocompetent (diabetic, chronic obstructive pulmonary disease)</td>
<td>Not treated as isolate thought to represent colonisation.</td>
<td>--</td>
</tr>
</tbody>
</table>
Three of the seven cases were pulmonary infections. This case report is the third reported postsurgical case that involves a foreign body.

A survey of non-tuberculous mycobacterium infections in New Zealand, showed a similar incidence to other developed countries, with 1.92 cases per 100,000 population in 2004. *Mycobacterium avium-intracellulare* complex (MAIC) was the most common isolate. Two.

There are no specific guidelines for the treatment of *M. thermoresistible*. The American Thoracic Society comments that the treatment of the less common non-tuberculous mycobacteria is based on previous cases. They recommend that any foreign bodies are removed and point out that *in vitro* antibiotic susceptibility testing often does not correlate well with the clinical response to the antimicrobials used.

The antibiotic regime for this patient was consistent with antibiotic choices in previous cases. As with the other cases, length of treatment was prolonged, but ultimately guided by resolution of clinical symptoms.

A high index of suspicion is needed for accurate diagnosis of the non-tuberculosis mycobacteria. With the advent of genetic profiling of mycobacterial species, it is likely that more cases of *M. thermoresistible* will be recognised.

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**References:**


Active tuberculosis (TB) with a negative interferon gamma release assay: failure of this test to rule out TB

Michael Maze, Lutz Beckert

Abstract
This report describes a patient with pulmonary tuberculosis in whom the interferon gamma release assay (IGRA) was negative at presentation. It became positive following treatment. This illustrates that this test cannot be used to rule out tuberculosis, even in a low incidence country.

Tuberculosis (TB) notification rates in New Zealand are around 7/100,000 with highest rates in those of non-European ethnicity.¹ Primary pulmonary infection is often asymptomatic with conversion of a tuberculin skin test or interferon gamma release assay (IGRA) the only evidence of infection.

Active tuberculosis refers to patients with replicating organisms.¹ The diagnosis of active tuberculosis can be difficult because of the low sensitivity of sputum microscopy and the prolonged culture required.²

IGRAs are increasingly being used as a screening test to rule out TB infection. A positive IGRA result does not distinguish between latent and active disease and importantly false negative results can occur.³–⁶

We describe a case of active tuberculosis associated with a negative IGRA at presentation that seroconverted following treatment.

Case report
A 29-year-old man of Indian origin presented with 7 weeks of an unproductive cough, unexplained weight loss and supraclavicular lymphadenopathy. He was born in India, lived in New Zealand since 2006 and had frequent visits to India. His sister-in-law was being investigated for active TB. He had no significant past medical history and HIV serology was negative.

Examination was unremarkable besides small non-tender supraclavicular lymphadenopathy on the right. His chest radiograph showed a 30mm opacity in the right upper lobe. His interferon gamma release assay (QuantiFERON-TB Gold in-tube assay, Cellestis Ltd., Victoria, Australia) was negative with a value of 0.23 IU/ml.

A computed tomography scan of his chest showed low attenuation centre consolidation in the right upper lobe with some associated ground glass and lymph nodes at the right hilum extending up the mediastinum to the root of the neck on the right hand side. These nodes showed low attenuation centres. He then underwent bronchoscopy with right upper lobe lavage as well as a fine needle aspirate of a supraclavicular node. Both were negative for acid-fast bacilli on Ziehl-Neeson stain.
Nucleic acid amplification with the GeneXpert (Cepheid, California, USA) detected *Mycobacterium tuberculosis* DNA. The organism was subsequently cultured and proved to be fully sensitive. He was treated with six months of standard combination anti-tuberculous therapy. His symptoms resolved and his chest radiograph normalised. His repeat IGRA was positive with a value of 0.86 IU/ml.

**Discussion**

New Zealand’s prevalence of TB is around 7-10/100,000; it is a low prevalence country but cases are concentrated in migrants. A highly sensitivity test is required to rule out tuberculosis in a low prevalence setting. IGRA indicates a cellular immune response to *Mycobacterium tuberculosis* antigens and therefore infection; they have been proposed as appropriate tests to rule out TB.

One study has shown the T-spot.TB to be more sensitive than QuantiFERON-TB Gold in-tube assay, but a recent meta-analysis showed a sensitivity of approximately 80% for both the QuantiFERON-TB Gold in-tube assay (Cellestis Ltd., Carnegie, Australia) and the T-Spot.TB (Oxford Immunotec Ltd., Abingdon, UK). This may be due to a window period (up to 22 weeks) from infection to IGRA conversion, immunosuppression such as HIV as well as impaired T cell activity from immune anergy caused by active TB infection. The low sensitivity of IGRA results in a low negative predictive value in situations such as this case when there is a high clinical suspicion of tuberculosis.

This case serves as a reminder that even in low prevalence countries such as New Zealand, a negative IGRA does not rule out active tuberculosis when the clinical suspicion is high. Current guidelines conclude that IGRA should not be part of routine investigation for active tuberculosis but contribute only supplementary information.

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**References:**

A timely reminder—rheumatic fever

Nikola Lilic, Priyanka Kumar

Abstract

Rheumatic fever is a disease diagnosed using the Jones criteria. The Jones criteria were designed using data from areas with a low prevalence of rheumatic fever. In New Zealand there is a high prevalence of rheumatic fever amongst Maori and Pacific peoples. A case is presented where a child of Samoan ethnicity is diagnosed and treated for rheumatic fever without fulfilling the Jones criteria. Evidence supporting the broadening of the diagnostic criteria in high prevalence areas is highlighted.

Rheumatic fever (RF) is a disease diagnosed using the Jones criteria. The criteria were initially published in 1944 and have been revised a number of times, the last revision being in 2002 by the American Heart Association.1,2 A case is presented here that highlights the limitations of the Jones criteria in high prevalence areas such as New Zealand.

Case report

A 6-year-old New Zealand-born Samoan boy presented with a 2-month history of migratory joint pain in the knees, ankles and wrists. His parents also reported that he had a sore throat 1-month prior. He had also experienced leg stiffness in the mornings. There was a history of possible fever the night before presentation without a history of trauma, rash or joint swelling. The arthralgia was treated with paracetamol syrup. No non-steroidal anti-inflammatory drugs were taken. The patient’s mother and two siblings had RF as children.

On examination he was afebrile with normal vital signs. All joints were non-tender with normal ranges of motion and the absence of clinical effusions. His cardiac examination revealed a 2/6 ejection systolic murmur loudest at the left sternal edge. He had bilateral cervical lymphadenopathy. The examination was otherwise normal.

Laboratory investigations revealed elevated antistreptococcal antibody titres; anti-streptolysin O titre (ASOT) was 711 IU/mL (normal <400) and anti-DNAse B was 1230 U/mL (normal <680). Erythrocyte sedimentation rate (ESR) was 95mm/hr. An electrocardiogram showed a PR interval of 0.2s (prolonged for his age). The transthoracic echocardiogram was normal.

Serological tests were performed to assess for alternative causes of arthralgia including Cytomegalovirus, Rubella, Epstein-Barr virus, Parvovirus, Mycoplasma, Yersinia and hepatitis; these were all negative.

A working diagnosis of RF was made and he was started on benzathine benzylpenicillin. He was followed up at the Rheumatic Fever Outpatient Clinic at 4 weeks where he was found to be symptom free. The transthoracic echocardiogram was once again normal and the ECG showed the PR interval to be 0.14s. The ESR was 17, ASOT 634 and Anti-DNAse 800.
Discussion

Using the Jones criteria this boy would not have been diagnosed with RF, as he had no major manifestations (see Figure 1). He did however have three confirmed minor manifestations: elevated ESR; prolonged PR interval; arthralgia and laboratory evidence of streptococcus infection.

The consulting paediatrician and infectious diseases specialist came to a clinical decision to treat this boy for RF given the high prevalence observed in Māori and Pacific peoples, his family history and the lack of an alternative explanation for his recent illness.

Less convincingly, the observed reduction in the PR interval and ESR at 4 weeks suggested that this was an acute event. Furthermore, the alternative causes of arthralgia, mainly viral infections, were excluded with serology as detailed above.

Figure 1. Jones criteria

![Jones criteria diagram](image)

Note: Diagnosis of RF requires: two major manifestations or one major and two minor manifestations.

The prevalence of RF in New Zealand Māori and Pacific peoples is approximately 40 to 100 cases per 100,000 per year, compared to less than 10 per 100,000 per year in European New Zealanders.3

It has been shown that the strict application of the Jones criteria in areas of high prevalence, such as Australia’s Northern Territory, will result in a substantial number of RF cases being missed.4

Another study in the Northern Territory found that 25% of Aboriginal patients diagnosed with RF with the main symptom being arthralgia, had no clinical evidence of arthritis.5 The authors went on to propose that polyarthralgia should be made a major criterion for probable RF.5

Without a gold standard test for RF diagnosis, clinicians practising in New Zealand have reason to consider broadening the diagnostic criteria when assessing people of Maori and Pacific descent given that the Jones criteria are based on USA data where RF prevalence is low. Broadening the criteria would increase the sensitivity and decrease the specificity of the criteria.

This would result in a greater number of patients being identified as having RF and therefore receiving treatment for the condition, thus decreasing the risk of cardiac
complications. However, it would also mean a greater number of patients without RF would be exposed to unnecessary penicillin treatment for a number of years.

The National Heart Foundation of New Zealand and the Cardiac Society of Australia and New Zealand have advocated broadening the criteria for populations with high prevalence but have not remarked on polyarthralgia specifically. However, it would also mean a greater number of patients without RF would be exposed to unnecessary penicillin treatment for a number of years.

Larger studies in high prevalence areas are needed to assess the adequacy of the Jones criteria in these populations.

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**References:**

An unusual complication of a pyrexia
Sibaji Phaujdar, Jayasri Phaujdar, Manish Saha

Clinical—A 6-year-old girl was brought into our emergency clinic for acute onset diffuse colicky pain in her abdomen. There was no history of diarrhoea or vomiting. She was having low-grade fever with rhinorrhoea for 2 days for which she was treated with antipyretic and nasal decongestant drugs for 1 day. Clinical examination was within the normal limit. Ultrasonography of whole abdomen was done but revealed no abnormality.

An erect abdominal radiograph was performed, which revealed small homogenous droplet-like opacities throughout her abdomen (Figure 1).

Routine laboratory investigations were normal.

Figure 1. Erect abdominal radiograph showing multiple droplet-like opacities throughout the abdomen

What is the abnormality and its management?
Answer and Discussion

Her parents could not find any unusual food intake in recent past, but on more specific enquiry the child admitted that she had accidentally swallowed something while measuring her oral temperature by clinical thermometer at home.

The thermometer was broken inside her mouth, by inadvertent pressure from her teeth, but she suppressed this fact from her parents. She was admitted for observation. General and systemic examination showed no signs of elemental mercury toxicity. Blood and urinary level of mercury and BUN values were normal. Her abdominal pain subsided with anti-spasmodic drugs and the patient was symptom-free after 24 hours. The patient was discharged in stable condition. In her follow-up visit after 1 week, a repeat X-ray abdomen was done, which revealed no such white opacities. Blood and urinary levels of mercury were measured again and were within the normal limit.

Human exposure of elemental mercury mostly occurs in academic institutes, home, healthcare facilities. Accidental exposure of mercury from broken medical thermometer is quite common in children below 6 years. Moreover, children are susceptible to toxic mercury vapour which is much heavier than air, accumulating preferentially in their breathing zone.

Absorption through the gastrointestinal tract and skin is quite low. Elemental mercury following ingestion was found to localise in appendix preferentially, occasionally culminating in acute appendicitis. But multiple case reports revealed no features of systemic toxicity after ingestion of mercury from a broken thermometer, though prophylactic medical (e.g. laxative) or surgical (e.g. appendectomy) measures were taken.

Active measures for decontamination in out-of-hospital exposure was mentioned in guideline suggested by E. M. Caravati et al; where they have also shown no therapeutic benefit of active decontamination (chelation) methods after ingestion of thermometer mercury.2

Routine clinical evaluation in an emergency medical service will suffice for “small spills” like from a broken thermometer.

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Lessons from the February 2011 Christchurch Earthquake for the training and preparation of Post Graduate Year 1 (PGY1) doctors

On 22 February 2011, a 6.3 magnitude earthquake struck Christchurch, New Zealand injuring 6659 people with the equivalent of a month’s worth of major trauma (150 cases) admitted through the Christchurch Hospital doors in just a few hours.¹ There was considerable distress and damage throughout the three hospitals.

This letter shares lessons learnt for informing prevocational (PGY1) training in New Zealand drawing on data from a larger study undertaken to document Prevocational House Officers (PGY1HO) experience of the earthquakes. This retrospective study used a mixed methods design² and is part of the Researching the Health Implications of Seismic Event group (RHISE).

There were 36 PGY1HOS employed at the Christchurch District Health Board (CDHB) at the time of the earthquake on 22 February 2011. There was a 72% response to the survey.

Immediately following the earthquake—There was confusion about where to go. 77% of respondents reported receiving no clear instructions from senior medical staff on what they should do immediately following the earthquake. 19% recall receiving immediate and clear instructions from a senior colleague.

During the first 3 days following the earthquake—73% reported being directly involved in the care of earthquake victims and work being emotionally challenging. Emphasis was placed on the early discharge of stable non trauma patients to free up beds for the incoming trauma cases. Supervision and leadership changed in a positive way, PGY1HO reporting that greater direction was provided by senior medical staff and constructive team work was seen across the specialties.

Impact professionally and personally was significant—Difficulties with work in the weeks following the earthquake were reported by 92%. This included stress from aftershocks, lack of sleep, patients’ distress and fear, the significant impact of poor living conditions, and problems with transport. The change of runs which occurs on a three monthly basis occurred one week after the earthquake and was seen as an added stressor by 14%.

Learning reported—54% described learning more about the process of emergency care and 19% said they developed new emergency clinical skills. Others reported that they had developed skills as to how to cope with the “unpredictability and stressfulness” of a crisis and grow from it as a person.

Issues consider in planning prevocational HOs orientation and training—Themes of management of willing helpers, alternative communication and teamwork with clear leadership emerged in this study (see also Ardagh¹).
For those of us involved in teaching PGY1 doctors there are some lessons to be shared:

- Firstly, for greater than 75% of PGY1HO to not know their responsibilities in a mass casualty incident is of concern. This could be rectified, by ensuring that during orientation the specific roles and responsibilities of PGY1HOs in such an event are outlined. Other recommendations from the HOs include designated meeting points for doctors at times of crisis and more trauma/disaster training for junior medical staff.

- The decision to change rotations was reported as stressful by the house officers. It disrupted established teams where communication patterns and trust had been established, limiting flexibility and responsiveness for some teams.

- Difficulties arose in dealing with frightened patients and at the same time not being able to communicate with one’s own family, and friends. We recommend that communication of the hospital plan at orientation includes forewarning about the personal impact of a disaster and stresses the importance of having a personal family plan for communicating and meeting in a disaster.

While the DHB did have and has a current disaster plan, improved training for junior doctors is required to ensure sure that they have a clearer understanding and awareness of disaster management at both a professional and personal level.

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References:


Decline in echocardiographic optimisation of cardiac resynchronisation therapy (CRT) devices at Christchurch Hospital

Cardiac resynchronisation therapy (CRT) is a treatment for patients with abnormalities in regional left ventricular activation, and is recommended in patients with severe heart failure (NYHA functional class III or IV), poor LV function with an ejection fraction of less than or equal to 35%, and a wide QRS (greater than or equal to 120 ms).

According to an ASE Consensus Statement published in 2008, “Echo plays an evolving and important role in the care of heart failure patients treated with biventricular pacing. Echo techniques potentially aid in patient selection for CRT prior to implantation and optimise settings afterward”.

Noting an apparent decline in echo optimisation referrals, we performed an audit of all CRT devices (both CRT-pacemakers and CRT-defibrillators) implanted at Christchurch Hospital between 2004 and February 2012. Data was collected relating to indication for implant, paced atrioventricular (PAV), sensed atrioventricular (SAV) and ventricle to ventricle (V-V) intervals at both implant, and at 6 week checks. Left ventricular ejection fraction (LVEF) data from pre-implant, 6 week check and latest echo reports was also obtained.

From a total of 243 patients, data was considered in quartiles according to presentation order. The first quartile presented between January 2004 and January 2007, the second March 2007 and January 2009, the third January 2009 and July 2010 and the fourth July 2010 and February 2012. Patient demographics were reasonably consistent with the mean age of patients ranging across quartiles from 63 to 67 years and the percentage of male patients ranged from 65 to 75.

Between the third and fourth quartiles, there was a dramatic reduction in the percentage of devices undergoing optimisation—respectively 60%, 68% and 62% of implants were optimised in the first three quartiles, with this dropping to 31% in the fourth quartile.

We also observed a steady decline in optimisations over the four quartiles where changes were made to CRT settings—37% of optimisations in the first quartile had changes made to CRT settings, 29% in the second, 24% in the third dropping to 20% in the fourth quartile.

In the patients that had changes made at optimisation, 72% had a change to the V-V interval and 47% had a change made to the PAV/SAV interval. In 29% of cases changes were made to both. There was no change in this pattern across quartiles.

We have found that at Christchurch Hospital, there has been a decline in the number of patients referred for echocardiographic optimisation of CRT and a decline in the percentage of these that have a change made as a result of echo optimisation.
Advances in CRT device technology and implant procedures leading to better optimisation at implant may be responsible. Levin et al have reported that for patients in sinus rhythm at CRT device implant, the interatrial conduction time measured at implant has a strong correlation with the echo derived optimal PAV and therefore this method could be used to program PAV intervals without the need for echo optimisation in these patients.\textsuperscript{2}

There has also been a growing awareness of the limitations of echocardiography in CRT optimisation. Recent data suggests that routine echo optimisation is not beneficial.\textsuperscript{3} In our practice it is now used in selected patients rather than in the majority.

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References:


Dear Editor,

I should be grateful for the advice of your readers, especially those with expertise in clinical microbiology, on a worrying matter affecting many communities in New Zealand. I am the former Whakatane Hospital Lab Manager who set up the Hepatitis Foundation of New Zealand. I now work as an advisor in Vietnam.

At one time, the Whakatane Hospital Laboratory handled all clinical laboratory specimens from the hospital and from GPs in the Eastern Bay of Plenty (EBOP). Our lab would have been highly profitable had we been paid at the same rates as were “private” labs. In the late 1990s a private laboratory was allowed to take over the GP work, with predictable and predicted effect on the hospital lab, which was not permitted to compete for the work. Specimens from most GPs were from then couriered on to Tauranga.

In 2010 the Bay of Plenty District Health Board (BOPDHB) awarded a contract to a Tauranga private laboratory to take over the running of the Whakatane Hospital Laboratory. A year later the Microbiology Department, whose IANZ audits frequently bettered that of the Tauranga labs, was “disestablished”.

Since then urgent microbiology specimens have been processed in Whakatane. “Routine” specimens are couriered to Tauranga. A problem was that the best microbiology scientist was amongst the staff laid off (he had argued against the downgrade), and others left; some resigning in protest. Thus the more pressing urgent work was left to a staff which had diminished in numbers and expertise. Another problem is that of deciding what is “urgent”.

In 2011, 30 of 31 Whakatane Hospital SMOs signed a memorandum to the BOPDHB, complaining about the laboratory downgrade and the lack of consultation. I was invited to lead a campaign seeking a return of a full onsite microbiology service, for both the hospital doctors and the GPs in EBOP communities.

Our principal concern is that all microbiology specimens from east of Whakatane, and perhaps 90% of specimens from the Whakatane district, arrive in Tauranga so late (>2 hours) that they fail to meet internationally accepted guidelines for processing such specimens. Many overseas laboratories refuse to accept late specimens because delicate organisms die off and others get smothered by harmless bacteria.

Lab scientists are taught that all specimens should be cultured within minutes of collection and not 2–26 hours later. We practised that basic standard when I ran the Whakatane Hospital Laboratory.

I recently consulted public hospital microbiologists in New Zealand, the United States and United Kingdom, and all agreed that it remains unacceptable practice to delay culture. Bearing in mind the time and effort required by patients and doctors to provide some of the specimens, the least one should expect is prompt handling and reliable reports.
I shall spare your readers a blow-by-blow account of our ongoing battle, during which our concerns and clear evidence was brushed aside by the DHB and Ministry of Health. A petition (Petition 2011/20 of Alexander Milne and 4,141 others) was presented to parliament’s Health Select Committee (HSC). Many doctors and other health workers had signed. I addressed the HSC on our submission, to which the BOPDHB had sent a rebuttal which was astonishingly inept and contained many errors of fact (see http://journal.nzma.org.nz/journal/126-1373/5627/response.pdf). The HSC then sought advice from the Ministry of Health, which contracted a private pathologist to provide an opinion.

The pathologist failed to consult any of the community or hospital doctors who had sent me reports regarding failings in our microbiology lab service (late reporting, inappropriate use of antibiotics, extended stays in hospital etc). The only GP member on the BOPDHB was ignored, as was myself despite the Ministry having full knowledge that I represented EBOP communities.

The “consultant” contacted only the provider and not the complaining doctors and patients, and advised the Ministry that the Eastern Bay of Plenty received a “best practice” microbiology service. Astonishingly, she attached an 11 page set of CDC guidelines (Manual of Clinical Microbiology) which contradicted her assertion on almost every page. The Ministry forwarded that advice to the Health Select Committee, whose majority membership then rejected our petition. Opposition parties presented opposing minority reports.

It would be wise to assume that this downgrade in standards applies New Zealand-wide, and is unlikely to be reversed. During my consultations with other New Zealand educators, a worrying revelation for me was that whilst medical laboratory scientists and pathologists are taught the importance of immediate culturing of all microbiology specimens, medical students are not taught this.

When this deficiency is addressed, doctors may be more alert to the fact that a report stating “no growth” does not mean that no pathogens were present when the specimen was taken. At this time, many NZ GPs and SMOs may assume that they receive a best practice clinical laboratory service when that cannot be the case.

I wrote to the Director General of Health in January 2013 invoking the Official Information Act, seeking an explanation for the Ministry’s bizarre advice, which swung the Health Committee against us. He failed to address our concerns, and deferred to his non-consulting pathologist and also the BOPDHB, as “this was an operational matter”.

I then reminded the DG that his Ministry was responsible for providing parliament’s Health committee with advice which endorsed the drop in standards by DHBs. I received another email from the DG today repeating that this is an operational matter. The Minister does not reply to our pleas.

The question is “Where does the buck stop when a DHB makes an operational decision which is demonstrably wrong?” In this case the consequences can be serious, and the claimed cost benefits are illusory. Do any of your readers have advice on dealing with a DHB which refuses to reverse a decision which is illogical and dangerous, and made without consultation with the users, both doctors and patients?
Also, do you consider that a 2–26 hour delay in handling specimens is acceptable, let alone “best practice”? In January 2013, Britain’s chief medical officer Dame Sally Davies warned her government that developing resistance to antibiotics was so serious an issue that it should be added to the British Government’s risk register of civil emergencies. New Zealand should be equally concerned, as this letter surely indicates.

I hope that you all agree that the first step we must take to tackle the problem of emerging bacterial resistance is to ensure prompt identification of pathogens and responsible use of antibiotics. We must strive for excellence and higher standards, and resist any downgrading of microbiology services.

I would be grateful for support from your Editorial Board and your readers.

Yours etc

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Molecular epidemiology of human tuberculosis in New Zealand and potential insights for disease control

Tuberculosis (TB) is a leading cause of infectious mortality worldwide, killing approximately 1.4 million people each year.1 In New Zealand during 2011, 308 new cases of TB were diagnosed including 2 cases of multidrug-resistant TB.2

After a peak in TB notifications in New Zealand of 142 cases per 100,000 in 1943,3 the incidence has decreased to the present 7 cases per 100,000.2,4 Much of the decline can be attributed to appropriate public health control measures and enacted legislation including the 1948 Tuberculosis Act.

Today, New Zealand is considered a low incidence country with respect to TB. However, this status can potentially mask significant disparities that exist in the incidence of TB between different ethnic groups in New Zealand. For example, disproportionately high rates of TB were observed, in both 2010 and 2011 among the Asian ethnic group [51.9/100,0004 and 40.6/100,000,2 respectively]. This means that among Asian persons in New Zealand, rates of TB are roughly comparable to the overall national rate in Brazil, one of 22 World Health Organisation designated high-burden countries for TB.1

In recent years in New Zealand, there has also been a marked change in the demographics of TB patients specifically with respect to the patient’s country of origin. The proportion of TB cases occurring in overseas-born persons has increased from 47.5% of cases in 19955 to 75.4% in 2011.2 Importantly, within this group, the highest numbers of cases occur within the first three years post entry into New Zealand.2 This indicates that a large proportion of overseas-born individuals may already be infected with Mycobacterium tuberculosis complex (MTBC) prior to their arrival in New Zealand. This deduction is supported by recent molecular epidemiological evidence. A strong association has been identified between the genetic lineage of the MTBC strain causing infection and the predominant lineage in the patient’s country of origin.6

Beyond the first year after arrival in New Zealand there is a sequential decrease in the rate of TB over subsequent years.7 In contrast, for patients originally from low-incidence countries, few cases occur within the first year of arrival and most occur after being in New Zealand for a period of over 20 years.7

In response to this, Immigration New Zealand introduced additional TB control measures in 2005. These include the requirement that persons who do not hold a passport from a list of low incidence TB countries and who plan to stay in New Zealand for 6 to 12 months, provide a chest X-ray certificate with their temporary entry class visa application.8 However, effective TB control not only requires measures to manage the number of overseas cases reaching New Zealand.

Some populations of New Zealand born persons also have disproportionately high rates of TB. Māori, for example, have an approximately six-fold higher rate of TB
compared to NZ Europeans.\textsuperscript{2,4} Interestingly, recent findings based on molecular TB typing methods show that Māori are more likely than Europeans to be infected with non-unique molecular strains.\textsuperscript{2,4} Clustering of non-unique molecular types has been seen also in the predominant genetic lineages of MTBC present in Māori cases.\textsuperscript{6} Patients sharing the same non-unique MTBC molecular type are likely to be connected epidemiologically by a shared chain of transmission. While further investigation is required to confirm this, the existing data suggest that there is a higher level of onward transmission of TB among Māori than among NZ Europeans. The median interval from onset of symptoms to commencement of treatment reported for TB cases in New Zealand in 2011 was 5 months.\textsuperscript{2} Delays in the diagnosis and treatment of TB of greater than 2 months increase the risk of spread of infection to household contacts, worsened severity of disease, and mortality.\textsuperscript{9} Conversely, early detection and treatment minimises the risk of transmission.\textsuperscript{10} Proposed measures to reduce detection times in New Zealand include community education on the symptoms of TB, heightened clinical awareness of TB among general practitioners, and earlier laboratory investigation of individuals in high risk groups.\textsuperscript{11} Further measures that have recently been introduced into routine use in New Zealand include rapid laboratory diagnostic technologies and eNotification of laboratory results. Expansion of the DOTS programme could also be considered with a view to reducing disease progression and the risk of ongoing transmission among high TB risk populations. While the level of investment required for each of these measures may vary, selective targeting of resources towards earlier diagnosis and treatment of TB among high risk populations in New Zealand seems likely to be a cost effective approach to reducing TB case numbers in New Zealand and decreasing the associated costs to the NZ healthcare system.

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Obituary (George Wilson) and Book Review (The Illness and Death of Napoleon Bonaparte)

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OBITUARY

It is with deep regret that we record the death of Dr George Wilson of Palmerston North. His death was the result of appendicitis. After practising in Edinburgh and Glasgow, Dr Wilson came to New Zealand in 1892, establishing himself in Palmerston, where he was known as a highly successful surgeon and physician. He took a keen interest in the affairs of our Association, and in everything that affected the welfare of the profession.

REVIEWS


This is a most interesting little book. The various doctors who attended Napoleon have left conflicting accounts of his illness, and Dr. Chaplin has therefore drawn his information directly from the Lowe papers in the British Museum.

Anything connected with Napoleon's life or death is a matter of absorbing interest. This book shows clearly that the cause of death was carcinomatous infiltration of an ulcer situated round the lesser curvature of the stomach.

The liver was found to be healthy at the post-mortem examination, much to the surprise of the French doctors who believed that Napoleon suffered from a complaint of the liver, the result of being imprisoned by the British in a tropical and unhealthy island. The apex of the left lung contained tuberculous cavities, and the bladder was diseased and contained calculi.

The diagnosis of cancer was made far too late, and this cannot be condoned, for even at the time of Napoleon's illness there was enough known on the subject of diagnosis of gastric cancer to justify a fairly early diagnosis in the case of the Emperor. The pathological specimens in the Museum of the Royal College of Surgeons in London, reputed to have been taken from the body of Napoleon, cannot be accepted as authentic.

It is evident that all whose official duty brought them into contact with the august patient of St. Helena had an exceedingly bad time, from Sir Hudson Lowe downwards.

Napoleon was a very refractory patient, but there is no doubt that he was a great sufferer.

We can recommend this book by Dr. Chaplin as being constructive and interesting.
Drug company gifts to medical students

Students in the United States have traditionally had a high level of exposure to drug industry sales representatives during their undergraduate medical education.

One survey of 8 US Medical Schools showed that over 90% of the students had attended a drug company sponsored lunch and had been the recipient of a non-educational gift. This report evaluates whether attending a medical school with an active policy that restricts gifts from the pharmaceutical industry influences subsequent prescribing behaviour?

The prescribing patterns of physicians graduating from the 14 schools with gift restriction policies before and after the introduction of the policy have been compared. The researchers conclude that “our study provides some preliminary evidence that exposure to a gift restriction policy during medical school may reduce the likelihood that a physician will prescribe newly introduced psychotropic medications over older alternatives within the same drug class.”


Endovascular treatment for acute ischaemic stroke

Intravenous recombinant tissue plasminogen activator (t-PA) is the standard treatment for acute ischemic stroke, but more than half of the patients treated do not recover completely. These researchers speculate whether endovascular therapy (intraarterial thrombolysis with recombinant tissue plasminogen activator (t-PA), mechanical clot disruption or retrieval, or a combination of these approaches) might produce better outcomes than intravenous t-PA.

They randomised 362 patients with an acute ischaemic stroke to receive endovascular therapy or intravenous t-PA. The media time from stroke onset to treatment was 3.75 hours for endovascular treatment and 2.75 hours for intravenous t-PA (P<0.001). Serious adverse effects were similar in each group. At 3 months 30.4% of the endovascular cohort and 34.8% in the intravenous t-PA cohort were alive without disability. They conclude that endovascular therapy is not superior to intravenous t-PA.

Another study in the same issue reports that combining endovascular therapy with intravenous t-PA is not better than intravenous t-PA alone.

Effects of body size and hypertension treatments on cardiovascular event rates

In previous clinical trials in high-risk hypertensive patients, paradoxically higher cardiovascular event rates have been reported in patients of normal weight compared with obese individuals. This paradox prompted these researchers to investigate whether the type of hypertension treatment affects patients’ cardiovascular outcomes according to their body size.

11,482 hypertensive patients had been randomised to treatment with single-pill combinations of either benazepril and hydrochlorothiazide or benazepril and amlodipine.

On the basis of their body mass index (BMR) the patients were classified as obese, overweight or of normal weight. Their conclusions were that “hypertension in normal weight and obese patients might be mediated by different mechanisms. Thiazide-based treatment gives less cardiovascular protection in normal weight than obese patients, but amlodipine-based therapy is equally effective across BMI subgroups and thus offers superior cardiovascular protection in non-obese hypertension.

Graham Lancelot Hill

Graham Lancelot Hill was born in 1939 in Dunedin, New Zealand, the son of Tom and Iris Hill. He was educated at Kings High School in Dunedin and went to Otago Medical School.

He went to Indonesia and Hong Kong as a missionary and later trained in Leeds, England, and Houston in the United States.

He returned to Auckland, New Zealand in 1980 as the Head of Surgery at the University of Auckland.

He retired to Wanaka in the South Island of New Zealand and more recently, due to failing health, he relocated to the town of his birth, Dunedin.

In 1296, long before the guilds became colleges, it was said that a great surgeon would have a temperate and moderate disposition.

That he would have well-formed hands, long slender fingers, a strong body, not inclined to tremble and with all his members trained to the capable fulfillment of the wishes of his mind. He would be of deep intelligence and of a simple, humble, brave, but not audacious disposition. He would be well grounded in natural science, and should know not only medicine but every part of philosophy…so as to be able to understand what is written, to talk properly and to support what he has to say by good reason. Graham fitted that description almost perfectly, except for the long slender fingers!

But who better to judge the man than his own patients and their families? Many still come to our clinics and speak so warmly of him, of his care and attention, his skill and wit. He made them feel important and special. At the bedside he would pull up a chair, gently take the hand, and speak in the way they understood. He was a master of reassurance, he instilled confidence, disarmed anxiety, encouraged those in the darkest places and brought hope tangibly into the room.

After a complex operation, his patients admired him even more when he would recount the complexity of the case, and I heard many times that ‘it was the most difficult of operations’ and ‘it took 10 years off my life’.

As a teacher of surgery, Prof (as he was known throughout New Zealand) was outstanding. His lectures and his informal tutorials were lucid and grounded and memorable. He had all the requirements-enthusiasm for his subject, ability to pitch at the right level, expert knowledge, a quick mind and a dash of humour. But he also had a certain gravitas, hard to replicate in today’s flatter world. You listened, were
inspired, and challenged. He drew a response from you. He encouraged deeper thought.

As a surgeon he was careful and considered. Very few are in a position to appraise a surgeon’s work because the work is so intensely private-inside an abdomen, inside a theatre, inside a hospital. Having worked opposite Prof, the master surgeon in his prime, it was like music and theatre, combined. There was a palpable rhythm, and there was purposeful melody line, and yes there were the occasional understated flourishes. But there was also an economy of motion, a steadiness of hand, deep concentration - and certainly no idle talk. His sharp dissection was meticulous and tissue planes parted before him as if on command.

His love of anatomy was infectious. His patience and persistence were legendary. He would stress that one should not go out the night before an operating list, but to prepare oneself well for the task ahead. He would speak of the need to be like a General, planning all the steps of an operation with the precision of a military campaign, and how to deal with the unexpected. He simply inspired you to do your best-by his own great example.

He understood the importance of those who had gone before, having been inspired himself by Allan Clark in Dunedin, Stanley Dudrick in Houston, Francis Moore in Boston, and John Goligher in Leeds.

It was Franny Moore who set the course of Graham’s surgical career-he saw first hand the way that a surgeon can be a bridge between the laboratory and the bedside, how research and clinical care can direct each other. Early on he committed to becoming a surgeon-scientist. And throughout his career he put enormous efforts into creating an environment in which both (science and surgery) could flourish.

He established the first colorectal unit in New Zealand, and through this delivered a national service for patients with complex colorectal disease. And he was a true pioneer in many areas of clinical surgery, including the physiology of ileostomy care, surgical stapling, highly selective vagotomoy, total parenteral nutrition, ileal J-pouch, enteric fistulae and extrafascial excision of the rectum.

Alongside this extra-ordinary contribution to the surgical craft he established the flagship body composition unit, which included the design and construction of the in vivo neutron activation facility, which has been the envy of the research community for decades and which continues to produce world class research. The validation of this facility took some courage, with a donated cadaver being measured before complete chemical analysis.

Through this facility he elegantly and convincingly demonstrated:

- The dramatic expansion of the extracellular fluid compartment in illness,
- The constancy of the fat free mass,
- The functional implications of body protein loss,
- And the effects of nutritional support.
He garnered critical international acclaim for this body of work and has been rightly known as the authority on the metabolic and body compositional effects of serious illness, and of the impact of malnutrition on surgical outcome.

Grahams inspiring life as a surgeon-scientist is now history, but fortunately a recorded history, in his book, subtitled ‘adventures in surgical research’ the breadth and depth of his surgical research is described. His patients were front and central-they were the reason for the research and their problems the focus. The book also describes his investment in young people, equipping and inspiring, sharing in discovery and in its application.

This was the vital ingredient that Professor Hill added to the environment of excellence in clinical surgery and surgical research. He selected and nurtured a whole succession of fellows. Graham said that one of his greatest joys was the training of young surgeons and to see them go on to succeed.

Research training was demanding under Professor Hill. He was exacting and set high standards in study design, data collection, analysis and presentation. He would frequently shred a protocol or presentation, but never the person. He was genuinely interested in personal development, and not just academic outputs.

Each Friday afternoon he used to drop by to find out what had been ‘discovered that week’ and how the research fellows’ lives were going. He advised on many matters—even how to change nappies-usually with sound common sense and substantial wisdom.

Wherever Graham worked-Dunedin, Indonesia, Leeds, Houston, and Auckland—he made a distinguished contribution, especially during his tenure as Head of Department at the University of Auckland.

His achievements, on paper, are impressive:

- Three academic degrees from the Universities of Otago and Leeds,
- Three College fellowships,
- Numerous visiting professorships, distinctions and awards, including the Louis Barnett Prize in 1972, the Moynihan Prize in 1978, and he was the James IV Traveller in 1982, John Mitchell Crouch Fellow in 1984 and a Hunterian Professorship in 1986.

In all he wrote 8 surgical books, 35 book chapters and published over 200 papers in the scientific literature and these papers are still consistently cited—over a decade since he retired, at almost 100 citations per year.

Two years after his early retirement at the age of 61 he was awarded the RACS Surgical Research Award, which was specifically created to recognize the distinguished lifetime contributions to research of a pre-eminent Australasian surgical scientist. And even wider recognition of his outstanding contributions to medicine came in 2009 when he was invested with the New Zealand Order of Merit (ONZM).
Graham Hill was a great man and a great surgeon. We will remember him for his passion to improve how we, as surgeons, do surgery, his passion for training young people as surgeon-scientists and his passion to improve patient care and outcome.

His leaves a phenomenal legacy, and we are, as a community of surgeons, deeply indebted to him. He belongs in the pantheon of surgeons. In reference to one of his own mentors Graham said that it was ‘a miracle and a privilege’ to be a surgeon. So too for Graham, in the deepest sense, for him it was a miracle—because surgical healing and wholeness comes from God and for him it was a privilege—because surgery and surgical research allowed him to participate in that process.

After retirement Graham involved himself in his local community in Wanaka, in the heart of the Southern Alps, the very place that he had begun his own Christian journey as a teenager. In particular his involvement in the local Presbyterian Church gave him great joy. He used his particular pastoral gifts to great effect and in the year prior to his passing he took on the mantle as the Moderator of the Presbyterian Synod of Otago and Southland.

Graham died on the 28 February 2013 after a long illness. He is survived by his wife Bartha and his three sons, Andrew, Philip and Douglas who are all Doctors, practicing in New Zealand.

John Windsor and Andrew Hill wrote this obituary.
Keith Edward Kibblewhite

Veteran Greymouth GP Dr Keith Kibblewhite has died, aged 85. Dr Kibblewhite, moved to Greymouth in 1954 to work for 6 months, and he never left.

He set up his own practice at his home in Cobden, where he lived with his wife and three children.

As a specialist physician he also worked at Greymouth Hospital and helped deliver hundreds of West Coast children over the years.

He rose to the top of his profession as President of the New Zealand Medical Association, and was also President of the West Coast Chamber of Commerce for many years.

Dr Kibblewhite closed his GP practice in 2006, and for the past 6 years has been a resident of Kowhai Manor rest home. The Greymouth Business and Promotions Association paid tribute to “Kibby” as he was affectionately known, acknowledging his dedication and contribution to the community.

“As well as a doctor, he did a lot of research. He was offered positions away, but always stayed,” chairwoman Ali Grooby said.

He never turned patients away if they were unable to pay. “If people did not have money, they could pay him with fish.” Because the surgery was at the front of the family home, his patients called on him any time of the day or night. “He did a lot more than people realised,” she said.

For a couple of years, Dr Kibblewhite was New Zealand’s only doctor fireman and he was honoured with a gold star in 1981, recognising his 25 years’ service to the Cobden Volunteer Fire Brigade.

He joined the brigade in April 1956 and had his first taste of action soon afterwards, when the brigade was called to the Holy Trinity Church, and his medical experience was called on at the same time when a fellow firefighter was hit by a jet of water at the fire scene.

Dr Kibblewhite died surrounded by his family and friends.

This obituary was written by Viv Logie and first appeared in the Greymouth Star. We appreciate the reprint permission.
Donald John Carr (Don) Horwood

20 June 1924–1 April 2013; MBChB MRCS, LRCP

Dr Don Horwood was born in Harrow, England, and after prolonged childhood illnesses interrupted his schooling he became determined to become either an engineer or a doctor, and despite humble beginnings, he persisted at his education; was admitted to and survived medical school through the war years; and went on to complete his clinical training at Charing Cross Hospital in London.

His starting salary in 1952 was 4 pounds 10 a week, 4 pounds of which the hospital kept for board and lodging, however during this time Don became interested and skilled in surgery and married his wife Ruth, who was a fellow student. Don and his young dentist wife moved to Uganda in 1955 where Don worked as a surgeon and district medical officer serving a population of 230,000 with two other doctors. It was during this time that Don undertook an operation which he later described as the high point of his career— with characteristic humility and compassion he described the procedure of gradually releasing the contractures caused by polio of a village boy, enabling him to walk for the first time in 8 years.

The feeling Don had seeing the elation and gratitude of the boy’s father never left him.

The Horwood family first came to New Zealand in 1963 where Don took up the role of Surgeon-Superintendent and part-time GP in Riverton, he also became Deputy Mayor, and learnt to fly, but the call back to Africa was very strong and in 1966 they moved back for Don to become a chief medical officer at Kilembe Copper Mine in Uganda. Don was unhappy with the treatment he saw here of the local miners, and after working out his year’s contract, looked around for the next adventure.

Canada beckoned, and Don’s life took another turn. As Zone Director for the Eastern Arctic, and later for the whole of the Yukon, Don was employed by the Federal Government to manage health services for vast regions of rural Canada, and although he reflected on this time regretting the lack of clinical work, and only occasional surgery he managed to perform, he performed with distinction and the responsibilities were huge.

In 1971 Don and Ruth decided to return to New Zealand. The Opotiki Lions organisation rang him and he was attracted to become the Surgical Superintendent to
Opotiki Hospital, a post he left in 1974, taking a full-time position as the town’s then only General Practitioner.

Don did not talk much about his life as a GP in Opotiki, reflecting that his life’s passion was surgery, I suspect his humility did not allow him to tell the stories that were too close to his home.

Locally he is remembered as a wonderful GP, who freely gave of his time, and energy, going out of his way to help families deal with poverty and disability; seeing a limping child in the street and stopping, making her take her to her mother, explaining the slipped epiphysis and arranging surgery to repair the damage; supporting families with budgeting advice, and setting up, along with his wife, a scholarship that still helps Māori women in tertiary education.

Don was a real gentleman, a dedicated rural physician, surgeon and father. He is survived by his wife Ruth, and two sons David and John.

Dr Jo Scott-Jones (GP in Opotiki) wrote this obituary.
Doctor Colenso, I Presume: missionary medical practice in midnineteenth century New Zealand

Ian St George. Published by the Colenso Society Inc. ISBN 978-0-9876604-1-1. Available from the Colenso Society at 32 Hawkestone St, Thorndon, Wellington 6011 (istge@yahoo.co.nz) for $10

This small soft-covered paperback book is an account of missionary medical practice in New Zealand in the midnineteenth century as illustrated by the work of Rev. William Colenso in the Wairarapa and Hawke’s Bay.

It contains 59 pages with black-and-white pictures. This book is somewhat difficult to follow and is not written in any conventional format. The book is written from a selection of writings of Dr Colenso. It is difficult to work out why these particular words were chosen. There are sections that can be followed and understood giving a small perspective of what life as a doctor may have been like for Dr Colenso. I enjoy medical history books, but I struggled with this book.

Frank Frizelle
Professor of Colorectal Surgery
Editor in Chief, New Zealand Medical Journal
Head of the Department of Academic Surgery, University of Otago, Christchurch, New Zealand.
My Journey: reflections on life from a cancer survivor


I am reviewing this book as it was sent to the NZMJ and five other people claimed to be too busy. This is a short book of poems written by an American author during her treatment for breast cancer. It covers different periods in her cancer journey from diagnosis and management to subsequent reflections on her experience.

I would like to say something positive about this book as we all recognise the suffering of many with cancer, so that when they write we assume they might do so to share the experience with others.

The book is a very personal account of her life and as such the voyagism may appeal to some, however I suspect most will find it not that interesting. It may have been cathartic to the author to write about their cancer experience however I am not certain their experience is that interesting to many others.

The poetry may appeal to some but didn’t do it for me. If this appeals to you perhaps the book is for you. The book is soft cover and short (116 pages), well bound and good page quality

“Bob”

You have always been here for me,
Though little is sometimes said.
Your subtle humour and your evenness and calm
Give me strength.
Through years of family,
Months of cancer treatment,
You have been by my side.
Did I thank you for your support?
In addition to your encouragement
You gave me freedom
To just be me.
Your steadfast love
Gives me inner strength,
The joy of sharing an
Everlasting love.

Frank Frizelle
Professor of Colorectal Surgery
Editor in Chief, New Zealand Medical Journal
Head of the Department of Academic Surgery, University of Otago, Christchurch, New Zealand.