CONTENTS

Viewpoint

Clinical Correspondence

Letter

100 Years Ago in the NZMJ

Methuselah

Obituaries

In this Issue of the Journal

The epidemiology of rheumatic fever in the Tairāwhiti/Gisborne region of New Zealand: 1997–2009
Victoria Siriett, Sue Crengle, Diana Lennon, Mary Stonehouse, Geoffrey Cramp

Acute rheumatic fever (ARF) causes permanent heart damage in 30% of children who get it. It is preventable and very rare in better-off young New Zealanders. Rates of ARF in young Māori are similar to those in young Pākehā/NZ Europeans in the 1920s. This is most likely due to crowded housing, poor access to health care and low health literacy. Streptococcal sore throats if treated will no progress to ARF.

A national estimate of the hospitalisation costs for the influenza (H1N1) pandemic in 2009
Nick Wilson, Nhung Nghiem, Alisa Higgins, Giorgi Kvizhinadze, Michael G Baker, Tony Blakely

The 2009 influenza pandemic resulted in 1122 people being admitted to New Zealand hospitals (with a laboratory confirmed diagnosis for the pandemic strain). In this study, we aimed to estimate the hospitalisation costs borne by the New Zealand Government for this pandemic. We estimated the total average cost to the hospital sector in New Zealand of NZ$ 30.5 million (95% uncertainty interval: 22.3 to 39.5 million). In an additional cost-effectiveness analysis (using a hypothetical situation relating to no hospital care), the results were suggestive that hospital care was likely to be a relatively cost-effective means of preventing death from pandemic influenza (i.e., at around NZ$155,000 per life saved from pandemic influenza). In conclusion, these high hospitalisation costs for a relatively non-severe pandemic indicate the potential value of preventive measures (e.g., vaccination) and of investing in pandemic planning and other control measures to reduce person-to-person spread.

Haemophilus influenzae type b disease in Auckland children during the Hib vaccination era: 1995–2009
Bonnie Leung, Susan Taylor, Dragana Drinkovic, Sally Roberts, Phil Carter, Emma Best

Haemophilus influenzae type b is a known cause of life-threatening disease in young children. Immunisation against this infection has been fully funded in New Zealand since 1994. This study looked at the influence of immunisation on the number and outcome of this disease in children under 15 years in the Auckland region between 1995 and 2009. There has been a marked decrease in occurrence since immunisation. However, in children who were affected, the uptake of immunisation was poor. The focus should be on improving both the coverage of children having immunisations and to have them given on time. In this way, New Zealand can become even closer to eliminating this disease in the future.

The prevalence of *Helicobacter pylori* infection in Sherpa residents of the Upper Khumbu, an isolated community in eastern Nepal
Tshering W Sherpa, Kami T Sherpa, Garry Nixon, John Heydon, Emma Heydon, Susan Dovey

This study shows that the prevalence of the bacterium *Helicobacter pylori* (*H. pylori*) is high (70.5%) among the Sherpa residents of the Upper Khumbu region of Nepal. The burden of dyspepsia, gastritis and peptic ulcer disease is high in this community and almost all cancer related deaths in the past 2 years were secondary to stomach cancer. Infection with *H. pylori* has been proven to be one of the major contributing factors to the above-mentioned disorders and we believe that it is the same in our case. Up until now, no testing for *H. pylori* is done for patients visiting Khunde Hillary Hospital (the only primary healthcare centre for the region) with the above disorders and they are either treated symptomatically or empirically. The result of this study mandates that all patients should be tested for the presence of the bacterium and treated accordingly. While it may be more expensive in the short term it will be more cost effective in the long term by providing a cure for what are otherwise chronic problems.

Physical activity is not play: perceptions of children and parents from deprived areas
Amy Curtis, Erica Hinckson, Tineke Water

Children and parents from South Auckland were asked to talk about factors that influence children's participation in after school activities. Parents talked about barriers such as time, money and transport as well as the importance of community support and communication in creating safer communities and places for children to play. Children talked about friends and winning as primary motivators to participating in physical activity but this depended on whether children were overweight or healthy weight.

Imported malaria in Auckland, New Zealand
Anna E Camburn, R Joan H Ingram, David Holland, Kerry Read, Susan Taylor

Malaria cases diagnosed in Auckland over a one year period were seen in New Zealand born missionaries, New Zealand residents who had travelled to their home lands to visit friends and relations, new arrivals to New Zealand and refugees. No cases of malaria were diagnosed among New Zealanders who had travelled overseas as tourists. The rate of malaria in Auckland is higher than the national rate largely because of refugees with malaria.

Rheumatic fever: from disease targeting to child-centredness

Norman Sharpe

Rheumatic fever has featured regularly in this *Journal* over decades and yet has persistently defied control. It features again this month in a report from Siriett and colleagues[1] which indicates little change during the past decade in the incidence in Tairāwhiti/Gisborne region where it occurs principally in Māori children associated with living and schooling within high deprivation areas.

Rheumatic fever also persists elsewhere in the North Island with cases often clustered in well recognised high risk settings. Most cases occur in South Auckland where the rates are highest in Pacific children.

New Zealanders would like to think we all live in a fair society which gives everyone a chance to live a healthy life. It is a shameful and intolerable paradox that rheumatic fever, a third world disease, still exists in New Zealand where we generally claim a first world standard of preventive and clinical care.

Rheumatic fever is a conspicuous marker of inequity. It is but one of a number of infectious diseases for which hospitalisations have increased in New Zealand during the past 20 years with clear ethnic and socioeconomic inequalities in risk.[2] In the broadest sense, rheumatic fever is an indicator of child poverty and ill-health in New Zealand and how we value our children.[3] Comparison of the inequitable poverty rates between children and the elderly in New Zealand is also pertinent—our children do not have a universal "Super Gold Card".

There is a notable parallel between the regional incidence of rheumatic fever in Tairāwhiti and the recent mortality rates for ischaemic heart disease (IHD) in the same community. The most recent data[4] show that age-standardised death rates for IHD by DHB region in 2009 were highest in Tairāwhiti, where they were significantly above the national rate and almost twice the rate in Waitemata. Also significantly above the national rate for IHD were Lakes, Taranaki, Mid-Central, Whanganui and Wairarapa.

Similar wide regional variation was also demonstrated for all-cause mortality and most major causes of mortality. In addition to this regional variation, as is well recognised, IHD mortality and also that of other major causes, were significantly greater for Māori men and women than non-Māori. The national age-standardised rate for IHD was 71 per 100,000; for non-Māori 66 and Māori 128 per 100,000.

In response to these disparities there is also a parallel between the current actions being taken to control rheumatic fever on the one hand and those being taken to improve the management of ischaemic heart disease on the other. A national rheumatic fever programme is in progress with the primary emphasis and expenditure being directed towards the management of streptococcal sore throat in high risk settings.[5]

In the Midland region (centred on Waikato Hospital in Hamilton)—integrated with the work of the National Cardiac Clinical Network across all regions[6] and with excellent local leadership and teamwork—more timely and equitable access for management of patients with acute coronary syndromes is progressing well.

Rheumatic fever control has recently been targeted as part of the cross-government Better Public Services work programme[7] and this year Heart Health and Diabetes Checks for eligible adults have been mandated as a new national health target[8]. Both targets are the encouraging result of long-run advocacy leading to increased public and political understanding of the large existent inequalities and the need for urgency and priority for effective actions.

For success, we need to ensure that these targets and programmes are closely linked with and complemented by effective and enduring "upstream" interventions directed at primordial and environmental determinants of these diseases. The causal pathway for rheumatic fever is well understood and effective primordial prevention through a "whole of government" and "whole of community" response must be linked with the present primary and secondary prevention programmes.

The development of an effective vaccine for streptococcal disease which is on a distant horizon may eventually be realised. However this should not lessen the need to work "upstream" where the relevant determinants are child and family poverty, poor quality housing and overcrowding, as for the whole group of close-contact infectious diseases. For "non-communicable" ischaemic heart disease prevention and truly heart healthy children and adults throughout the lifecourse, these determinants remain highly relevant.

A recent collection of reports and recommendations related to child and family poverty in New Zealand is now engendering wide public support. The Advisory Group convened by the Child Health Commissioner[9] has recommended several key interventions for immediate and longer term improvement. It is important that these do receive public and political support and are translated into action. They must also be viewed in the broad context of the "upstream" determinants of health and the need for evidence-based comprehensive policies and programmes which place the child at the centre within their family/whānau and communities. Only in this way will effective prevention and greater health equity be achieved and sustained. Programmes targeted selectively on downstream sequelae only will be insufficient.

The recent public discussion which has built around the issues of child and family poverty and inequity suggests that as a society we think it is unfair that some families cannot afford the basics in life.

We occupy a very lowly position amongst the OECD group of nations in terms of child health and safety[10] and are now acknowledging an urgent need for transformation to a broad and comprehensive child-centred approach to policy making.

The OECD experience tells us that the greatest success in reducing poverty and inequity is achieved where there is greater commitment to progressive or proportionate universalism[11]. This simply means that there should be adequate support for all but more intensive provision for those with the greatest need. In New Zealand in recent decades we have actually seen the opposite, "disproportionate

universalism" with increasing relative inequalities. We have been going in the wrong direction despite good intentions.

These principles are not contentious amongst public health professionals but are not yet widely understood or agreed across our communities or by political representatives. The necessary transformation will require greater societal understanding and public support for enduring non-partisan policies which place the highest possible value on investment in our children.

Narrow targeting, a politically pragmatic response, may inadvertently reinforce the stigma of poverty. We need to find the right mix of broadly based universal and targeted approaches to support poor families, particularly those with young children.

Rheumatic fever is a visible and significant marker of child poverty. For success, targeting for control needs to be placed in the context of broadly based and balanced child and family/whānau-centred policies to guarantee a brighter future for all our children in all respects.

Competing interests: Nil.

Author information: Norman Sharpe, Medical Director, Heart Foundation, Auckland

Correspondence: Norman Sharpe, Medical Director, Heart Foundation, Auckland, PO Box 17-160, Greenlane, Auckland, New Zealand. Fax: +64 (0)9 5719190; email: NormanS@heartfoundation.org.nz

References:

1. Siriett V, Crengle S, Lennon D, Stonehouse M, Cramp G. The epidemiology of rheumatic fever in the Tairāwhiti/Gisborne region of New Zealand: 1997-2009 N Z Med J 2012;126(1365). http://journal.nzma.org.nz/journal/125-1365/5418
2. Baker MG, Barnard LT, Kvalsvig A, et al. Increasing incidence of serious infectious diseases and inequalities in New Zealand: a national epidemiological study. The Lancet 2012;379(9821):1112–9.
3. Hale M, Sharpe N. Persistent rheumatic fever in New Zealand – a shameful indicator of child health. N Z Med J 2011; 124 (1329). http://www.nzma.org.nz/journal/124-1329/4530/content.pdf
4. Mortality and Demographic Data 2009. http://www.health.govt.nz/publication/mortality-and-demographic-data-2009
5. Sharpe N. Rheumatic fever in New Zealand: from perennial failure to successful eradication. N Z Med J 2011; 124(1343). http://journal.nzma.org.nz/journal/124-1343/4887/content.pdf
6. National Cardiac Clinical Network http://www.nationalhealthboard.govt.nz/National-Cardiac-Surgery-Network.
7. Better Public Services http://www.health.govt.nz/about-ministry/our-priorities/better-public-services
8. National Health Targets http://www.health.govt.nz/new-zealand-health-system/health-targets/2011-12-health-targets
9. Expert Advisory Group on Solutions to Health Poverty http://www.occ.org.nz/data/assets/pdf_file/0011/9857/FINAL_Issues_and_Options_Paper.pdf
10. OECD. Comparative child well-being across the OECD. In: Doing Better for Children. 2009 http://www.oecd.org/dataoecd/19/4/43570328.pdf
11. OECD. Child poverty in OECD countries: lessons for developing countries, perspectives on social policy in international development. Issue No 2, June 2012.

The epidemiology of rheumatic fever in the Tairāwhiti/Gisborne region of New Zealand: 1997–2009

Victoria Siriett, Sue Crengle, Diana Lennon, Mary Stonehouse, Geoffrey Cramp

Abstract

Aim To establish Acute Rheumatic Fever (ARF) rates within the Tairāwhiti District Health Board (1997-2009) to identify communities for primary prevention programmes.

Method ARF cases (1997–2009) sought by audit of Gisborne Hospital admissions, penicillin prophylaxis lists and the EpiSurv notifiable disease database.

Results ARF rates (n=44 cases) during 1997 to 2009 (7.6/100,000) with a continuing significant disparity between Māori (n=40, 15.2/100,000) and non-Māori, (n=3, 1.1/100,000). One case was Pacific. This disparity was marked in school-aged children (5–14 years: Māori 59/100,000 vs non-Māori 8/100,000). Over 80% of ARF cases demonstrated heart damage (18% moderate, 20% severe and 8% requiring heart surgery). ARF cases were strongly associated with living and schooling within high deprivation areas

Forty ARF cases were enrolled in 13/21 Gisborne schools, 4/18 East Coast schools and 2/17 western rural schools. (No school for 8 cases). When assessed as a percentage of school rolls there were no discernable differences between primary, intermediate and secondary schools. Of the 44 cases, 35 (80%) resided in areas of NZDep06 score 8–10 (most deprived).

Conclusion Very high ARF rates were recorded in the 1960's; the continuing burden of ARF in Māori children indicate a strong requirement for primary prevention strategies. Progress has plateaued in the last 20 years.

Acute Rheumatic Fever (ARF), a preventable disease triggered by a group A streptococcal (GAS) pharyngitis, is associated with long-term heart damage.

It is recognised that a single ARF episode can lead to Rheumatic Heart Disease (RHD) where heart valves can be damaged to a level that valve repair or replacement is required due to progressive heart failure. At least 50% of those who have had ARF may suffer long-term RHD[1] which not only places a significant burden on the healthcare sector but causes an average of 145 deaths per year in New Zealand.[2]

It is known that the majority of ARF cases can be prevented with antibiotic treatment of a sore throat caused by a group A streptococcal infection, clearly indicating that ARF is a preventable disease.[3]

While ARF has been virtually eliminated in other developed countries, this cannot be said for New Zealand where Māori and Pacific children and young people endure high rates of ARF (40–100/100 000), up to 100 times higher than in other developed countries.[1,4,5]

In New Zealand European children the rate is 1/100,000, highlighting a significant disparity between Māori and non-Māori. Compounding this inequality, Māori are over 7 times more likely to have a RHD-related death but only twice as likely to receive heart valve surgery than non-Māori.[2]

The Tairāwhiti region of New Zealand has a high proportion of Māori at 47.1%, with 75% of children being Māori.[6] Furthermore, census findings indicate significant deprivation within the region.[7] The significance of this is the strong correlation between deprivation and the incidence of ARF.[4,8] Evidence of high rates of ARF in the region have been documented since the 1960's,[9] though more recent systematic epidemiological evaluation of ARF has been limited.

This study was performed to establish current rates of ARF in the Tairāwhiti DHB region and to identify areas where future ARF primary prevention programmes may be targeted in the region.

Methods

Potential ARF cases within the Tairāwhiti region were acquired from the following sources:

- A retrospective audit within the Tairāwhiti DHB using clinical records of all Gisborne Hospital admissions with a diagnosis of ARF during 1997–2009. Cases diagnosed prior to 2000 or after this time were identified using the ICD-9 and ICD-10 (Codes 100, 101.0, 101.2, 101.8, 101.9, 102.0, 102.9) coding systems respectively.[10]
- A listing of patients currently receiving benzathine penicillin prophylaxis for ARF maintained by the Tairāwhiti district health (TDH) Well Child Team.
- Accessing the EpiSurv database (ESR national database for the surveillance and control of notifiable diseases) to identify cases listed as ARF from 1997 to 2009.

All ARF and recurrent cases were included if they were identified from at least one of these sources, while under-reporting of ARF within the Tairāwhiti region was also assessed. The 1992 Jones criteria adapted for NZ use (echocardiographic detection of regurgitation in the absence of an audible heart murmur could exceptionally be a major criterion) were used to categorise the ARF cases as definite, probable, and possible.[11]

Recurrent cases required a definable new ARF episode with supportive evidence of a recent GAS infection.[12] A geographical cluster analysis was performed by classifying each case according to DHB and Census Area Unit (CAU) at the time of diagnosis. Population denominator data and New Zealand Index of Deprivation (2006) (NZDep06) data were obtained from the Ministry of Health 2009 PHO enrolment demographics and the 2006 Statistics New Zealand Census of population and dwellings respectively.[6,7]

The data were then analysed to determine the population rates of ARF within the Tairāwhiti region from 1997–2009. The cases were then organised by age, ethnicity and CAU to determine disparities between the various groups. The one ARF case in a Pacific child was grouped with Māori for calculation of rates.

Results

During 1997–2009, a total of 44 ARF cases of all ages (36 definite, 2 probable, 4 possible and 2 recurrent cases) were diagnosed within the Tairāwhiti region according to the 1992 Jones Criteria. Cases sourced from hospital discharge data and Public Health prophylaxis data bases matched those found from the Episurv database. The mean age of individuals presenting with ARF was 11 years of age, (range 4 to 24 years) (Figure 1).

Figure 1. The age at presentation of ARF cases in Tairāwhiti from 1997–2009 (n=44)
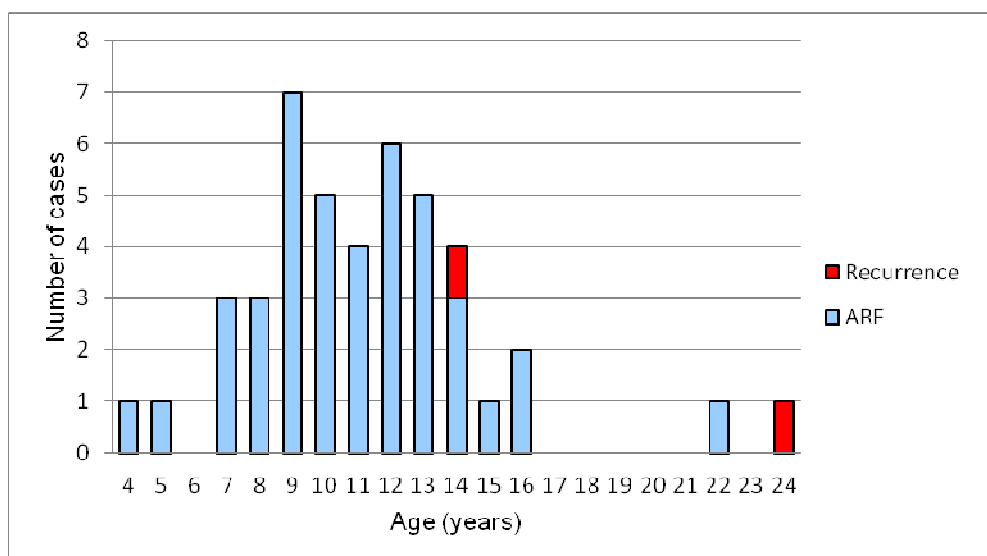
A significant disparity was observed between Māori and non-Māori with 90.9% (40 cases) stating they were of Māori ethnicity, while only 6.8% (3 cases) were NZ European and 2.3% (1 case) was of Pacific ethnicity. Within the Tairāwhiti region an annual total population incidence (1997–2009) of 7.6/100,000 for ARF was observed. However, Māori were found to have an incidence of 15.2/100,000 compared to 1.1/100,000 for non-Māori, illustrating a significant disparity between the ethnic groups. This disparity was accentuated in the 5–14 year age band with an incidence of 59/100,000 in Māori/Pacific children compared to 8/100,000 in non-Māori children (Figure 2).

Figure 2. The incidence of ARF/100,000 by age and ethnicity in Tairāwhiti from 1997–2009 (n=44). (Pacific case included in total)
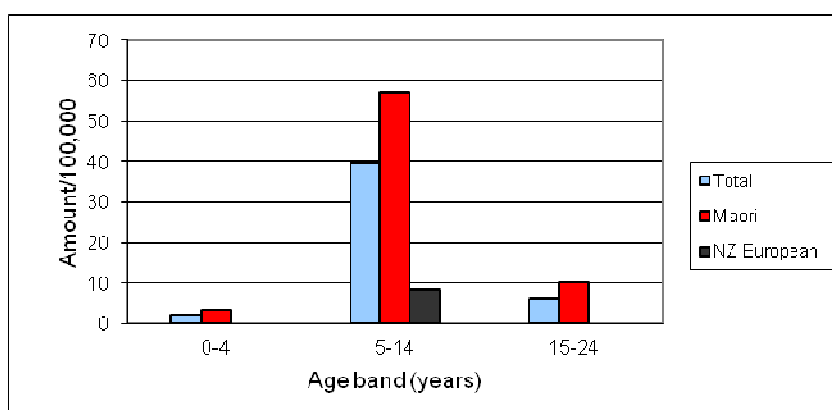
Eighty per cent (35/44) of cases had carditis at diagnosis and 70% (34/44) of cases had polyarthritis. No cases presenting with nodules were observed and only four cases (9%) presented with chorea. Three of the four chorea cases occurred in 1999. The majority of cases presented with raised ESR and streptococcal serology (anti streptolysin O and/or anti DNase B antibodies), while raised ESR levels (39/44) were more frequently observed than raised CRP levels (28/44).

Normal ESR levels were observed in only 3 cases (7%), all of whom presented with chorea and carditis. Only 4 cases (4/38, 11 %) presented with positive GAS culture following a throat swab. The commonest presentation was carditis and polyarthritis simultaneously (43%) followed by symptoms of carditis alone (20%). No cases with Erythema Marginatum were described.

The most frequent type and severity of cardiac involvement (figure 3) was mild mitral regurgitation (MR) occurring in 55% (20 cases) of the cases presenting with carditis, followed by mild aortic regurgitation (AR) (33% or 12 cases) and left ventricular dilation (22% or 8 cases). A total of 3 cases (8%) presented with congestive heart failure and required surgery for mitral valve repair. All 3 of these cases were under the age of 10, with the youngest being 4 years of age.

Figure 3. The type and severity of cardiac valve involvement in ARF cases in Tairāwhiti from 1997–2009 (n=36 patients)
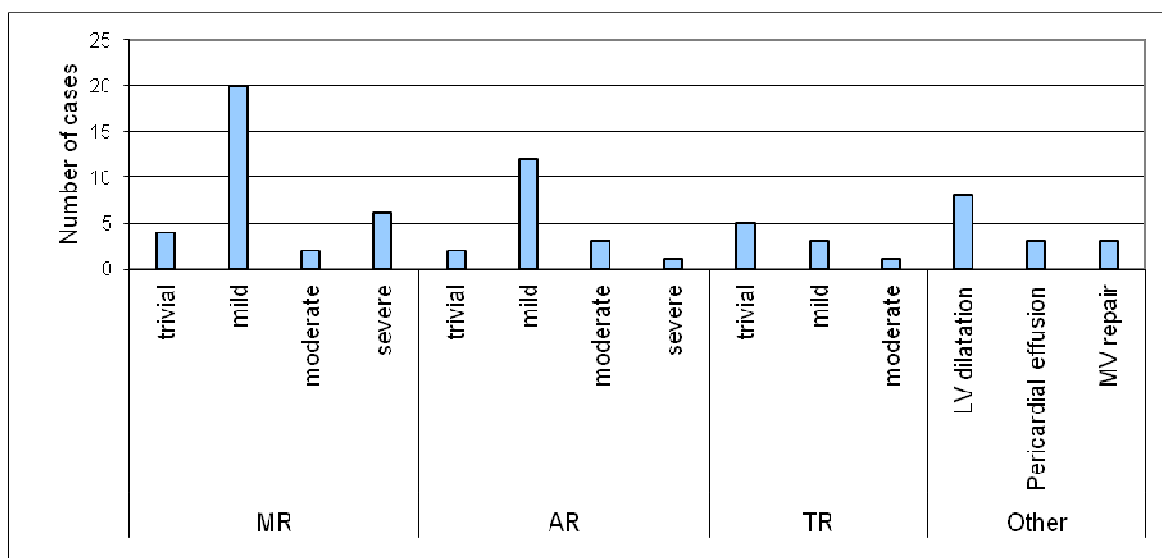
MR=mitral regurgitation, AR=aortic regurgitation, TR=tricuspid regurgitation, LV=left ventricular, MV=mitral valve.

An analysis of the number of valves affected in each carditis case indicated that MR alone occurred in 46% of the cases (15 cases), while MR with AR was observed in 22% (8 cases) and MR, AR and TR occurred in 14% (5 cases). Overall assessment for

RHD indicated 18% of carditis cases demonstrated severe RHD, while 20% had moderate and 61% had mild RHD.

The home address at the time of diagnosis for each ARF case within the Tairāwhiti DHB was classified according to CAU to determine any areas of high ARF incidence. This analysis showed cases were spread out over a number of CAUs, however one CAU (North Kaiti) within Gisborne city contained 20% of the ARF cases. Of the 44 cases, 35 (80%) resided in the areas with a deprivation score of 8–10 according to the NZDep06 scoring[7] (Figure 4).

Figure 4. The number of ARF cases in Tairāwhiti from 1997–2009 shown by NZDep06 scoring (n=44)
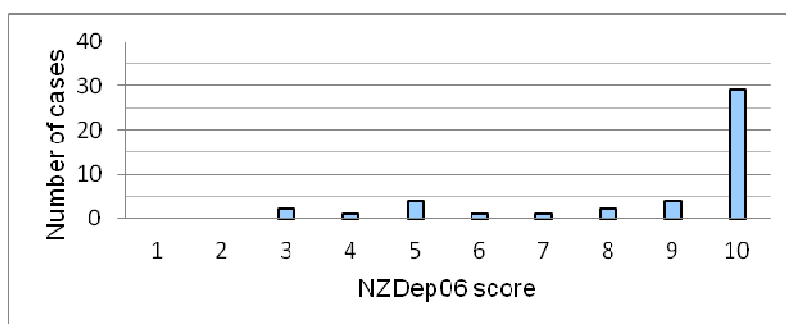
NZDep06 score of 10 indicates the highest possible level of deprivation

Of the 44 ARF cases, 40 cases were enrolled in schools within the Tairāwhiti region (Table): 26 cases in Gisborne schools (within 13 of the 21 Gisborne schools), 4 in coastal schools (within 4 of the 18 coastal schools), 2 in western rural schools (within 2 of the 17 western rural schools) and 8 cases with no schooling details. In total, 19 schools were associated with ARF cases with minimal differences between primary, intermediate and high schools as well as location (i.e. Gisborne, coastal or the western rural area of the district) when assessed as a percentage of individual school rolls. However, affected schools were associated with areas of high deprivation and high Māori enrolment.

Cases grouped by geographic area and age group in the Table suggest all Tairāwhiti schools serving the primary and intermediate school age group are close to or over the threshold suggested in the National Heart Foundation guideline (>50/100,000) for school clinic eligibility (www.heartfoundation.org.nz).

Table 1. Acute Rheumatic Fever rates (5–14y) in Tairāwhiti Schools (1997-2009)

Locality	Cases	Number of Schools	School of Population	Rate per 100,000
Gisborne City	18	10	3,115*	44/100,000[⊠]
East Coast	4	4	399[+]	77/100,000
West Rural area	2	2	283°	54/100,000
Total	24		3,797	49/100,000

Key:

[+]	yr 1–13
°	yr 1–8
*	yr 1–8
[⊠]	school A 75/100,000 (5–10 years)
	school B 104/100,000 (5–10 years)

Discussion

From 1958 to 2009 there have been variable methods for the recording of cases of ARF in the Tairāwhiti region. During 1958–1973, 223 ARF cases were reported, of which 77 (35%) were recurrent cases.[9] Incidence was not reported. During 1974–1984 the number of cases dropped to 77 with 14 (18%) being recurrent cases.

More recent studies have reported the total population incidence of ARF (n=36 notified cases) in the Tairāwhiti region at 13.1/100,000 during 1995–2000[15] and 9.7/100,000 in 1996–2005 (n=47 cases from hospital discharge data).[16] In the current study, 44 ARF cases were reported using all available data sources and including notified and hospitalised cases (4.5% being recurrent cases). This equates to a total population annual incidence of 7.9/100,000 suggesting that the incidence of ARF has changed very little in the past decade.

As ARF is now rare in the non-Māori population in this DHB the current incidence of 15.1/100,000 for Māori would more accurately reflect the burden , starkly demonstrating that ARF within the region remains unacceptably high and highlighting the disparity in incidence of ARF between Māori and non-Māori. This finding is consistent with past studies.[4,9,16]

Higher rates of ARF have been shown by others to be associated with socioeconomic deprivation.[4,8] Approximately 60% of the Tairāwhiti region was scored at NZDep06 8–10 (more deprived). Eighty percent (35/44 cases) resided in these areas, though 20% occurred in 1 census area unit (NZDep06 score). Furthermore, schools associated with ARF cases were also correlated with areas of high deprivation and high Māori enrolment.

A significant finding was the extent of cardiac involvement during ARF, with 82% (36 cases) presenting with some degree of carditis. Of these, 20% presented with severe RHD while 8% required mitral valve repair. The long term implications of these outcomes mean a substantial burden is placed on an individual, their family, and the healthcare system.

Retrospective studies of DHB records may underestimate the true prevalence of RHD. The usual presenting symptoms and signs of ARF in childhood such as arthritis may be ignored or misdiagnosed as injury even in a population at high risk of ARF as a

result of inadequate public health messages, limited parent/family health literacy or insufficient degree of suspicion by healthcare personnel.

An unpublished study of echocardiographic screening for RHD of 685 students in 5 schools in the TDHB area found 8 previously unknown cases (defined as definite and probable) of RHD (1.61%), many of whom had mild MR with an inaudible murmur. In addition a further 19 cases had possible RHD (2.77%) with valvular echographic changes of mostly mild MR. The natural history of subclinical echographic changes of RHD without presentation with ARF is unknown.

The observed correlations of ARF with Māori ethnicity, and living and schooling in high deprivation areas identifies high risk groups which could be targeted for future primary prevention programs. Although evidence for significant reductions in ARF incidence from school-based throat swabbing programs have yet to be substantiated, a recent meta-analysis reported a reduction in ARF cases by approximately 60% if streptococcal pharyngitis were treated with a 10 day course of oral penicillin in a school and/or community-based programme.[17,18]

Current ARF prevention guidelines outline the management of a school clinic sore throat project.[13] The steps include raising local awareness of the importance of treating sore throats, engagement of identified high risk schools for a commitment to participation, conveying ARF information to families and gain necessary consent, designing the project methodology (including frequency of swabbing and testing resources), establishing treatment strategies (i.e. school-based antibiotic delivery or from family doctors) and carrying out contact tracing for GAS positive students.

It is clear from this study that a significant need for primary ARF prevention remains within the Tairāwhiti region. Furthermore, community and school-based prevention efforts should be targeted towards the identified high risk groups of Māori within the 5–14 year age group.

Throat swabbing resources and sore throat education needs to be expanded to facilitate the prevention of ARF. This will enable the reduction of the burden associated with this disease to individuals, their families and to the healthcare system, as well as lead to the reduction of inequalities within New Zealand communities.

Author information: Victoria Siriett, Medical Student, The University of Auckland, Auckland; Sue Crengle, Te Kupenga Hauora Māori, School of Population Health, The University of Auckland, Auckland; Diana R Lennon, Professor of Population Child & Youth Health, Paediatrics, The University of Auckland, Auckland; Mary Stonehouse Paediatrician, Gisborne Hospital; Geoffrey Cramp. Medical Officer of Health, Tairāwhiti District Health Board (Tairāwhiti DHB), Gisborne

Correspondence: Diana Lennon, Professor of Population Child & Youth Health/Paediatrician in Infectious Diseases, Community Paediatrics, Department of Paediatrics: Child & Youth Health, The University of Auckland, Tamaki Campus, School of Population Health, Private Bag 92019, Auckland, 1142, New Zealand. Fax: +64 (0)9 3035932; email: d.lennon@auckland.ac.nz

References:

1. Lennon D. Acute Rheumatic Fever, in Feigin & Cherry Textbook of Pediatric Infectious Diseases, R.D. Feigin, et al., Editors. 2009, Saunders: Philadelphia. p. 419-434.

2. Robson B and Harris R. Hauora: Māori Standards of Health IV. A study of the years 2000-2005. 2007, Te Ropu Rangahau Hauora a Eru Pomare: Wellington.

3. Catanzaro FJ, Rammelkamp Jr.CH, and Chamovitz R. Prevention of rheumatic fever by treatment of streptococcal infections. New England Journal of Medicine. 1958;259(2):53-7.

4. Craig E, Jackson C, Han DY. Monitoring the health of New Zealand children and young people: indicator handbook. 2007, Paediatric Society of New Zealand, New Zealand Child and Youth Epidemiology Service: Auckland. p. 218.

5. Milne, R. J., D. R. Lennon, et al. (2012). "Incidence of acute rheumatic fever in New Zealand children and youth." Journal of Paediatrics and Child Health: no-no. Published online: http://dx.doi.org/10.1111/j.1440-1754.2012.02447.x

6. Ministry of Health, PHO Enrolment Demographics for 2009. Available from: http://www.moh.govt.nz/moh.nsf/indexmh/phcs-pho-enrolment

7. Statistics New Zealand. New Zealand Census of People and Dwellings 2006.

8. Zaman MM, Yoshiike N. Socio-economic deprivation associated with acute rheumatic fever. A hospital-based case-control study in Bangladesh. Paediatric and Perinatal Epidemiology, 1997. 11(3): p. 322-32.

9. Frankish J.D. Rheumatic fever on the East Coast, North Island. New Zealand Medical Journal. 1974;80(520): 48-51.

10. International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10). Geneva, Switzerland: World Health Organization;1992.

11. Atatoa-Carr P, Bell A , Lennon D. Rheumatic fever diagnosis, management, and secondary prevention: a New Zealand guideline. New Zealand Medical Journal.2008. 121(1271): 59-69.

12. Spinetto H, Lennon D, Horsburgh M. Rheumatic fever recurrence prevention: A nurse-led programme of 28-day penicillin in an area of high endemnicity. J Paediatr Child Health. 2011;47(4): 228-234.

13. The National Heart Foundation of New Zealand, Cardiac Society of Australia and New Zealand. Evidence-based, best practice New Zealand Guidelines for Rheumatic Fever: 3. Proposed rheumatic fever primary prevention programme at www.heartfoundation.org.nz 2006.

14. Frankish JD. Rheumatic fever prophylaxis: Gisborne experience. New Zealand Medical Journal. 1984;97(765):674-675.

15. Thornley C, McNicholas A, Lennon D et al. Rheumatic fever registers in New Zealand. New Zealand Public Health Report. 2001;8(6):41-4.

16. Jaine R, Baker M, Venugopal K. Epidemiology of acute rheumatic fever in New Zealand 1996-2005. J Paediatr Child Health. 2008;44(10):564-71.

17. Lennon D, Stewart J, Farrell E, et al. School-Based Prevention of Acute Rheumatic Fever: A Group Randomized Trial in New Zealand. Pediatric Infectious Disease Journal. 2009;28(9):787-794.

18. Lennon D, Kerdemelidis M, Arroll B. Meta-Analysis of Trials of Streptococcal Throat Treatment Programs to Prevent Rheumatic Fever. Pediatr Inf Dis J. 2009;28(7):e259-e264.

A national estimate of the hospitalisation costs for the influenza (H1N1) pandemic in 2009

Nick Wilson, Nhung Nghiem, Alisa Higgins, Giorgi Kvizhinadze, Michael G Baker, Tony Blakely

Abstract

Aim To estimate the hospitalisation costs borne by the New Zealand Government for the influenza pandemic in 2009 (with uncertainty).

Methods Data were derived from national and local New Zealand studies, and from a combined Australia and New Zealand study on intensive care unit (ICU) use and costs. Probabilistic sensitivity analysis was performed (2000 iterations).

Results We estimated the total mean cost to the hospital sector in New Zealand of NZ$30.5 million (95% uncertainty interval (UI): 22.3 to 39.5 million) [US$14.8 to 26.3 million]. The mean cost per capita was NZ$7.01. In an additional cost-effectiveness analysis (using a hypothetical counterfactual relating to no hospital care), the results were suggestive that hospital care was likely to be a relatively cost-effective means of preventing death from pandemic influenza.

Conclusions These high hospitalisation costs for a relatively non-severe pandemic indicate the potential value of preventive measures (e.g., vaccination) and of investing in pandemic planning and other control measures to reduce person-to-person spread.

The 2009 influenza pandemic in New Zealand had a significant nation-wide impact[1][2] including on the hospital sector.[3] One Australasian study,[4] considered the impact of the 2009 pandemic on intensive care unit (ICU) admissions and a related study costed these admissions at over A$65 million, for both Australia and New Zealand collectively.[5] This costing study did not, however, separate out the cost estimates for New Zealand and did not calculate costs for hospitalised cases not admitted to ICUs.

We therefore aimed to expand on this work to provide best estimates of such hospitalisation costs for the New Zealand setting for the 2009 pandemic.

Methods

We took a healthcare provider perspective, i.e., that of the New Zealand Government which fully funds public hospitals. New Zealand data for the year 2009 included 1508 hospitalisations for influenza, a four-fold increase on the number in the preceding year.[3] Most of these people (n=1122) were admitted to hospital with a primary diagnosis of "pandemic influenza A(H1N1) 2009". The dominant role of the pandemic strain in 2009 in causing influenza in this year also comes from virological surveillance data,[6] and from two local hospital studies.[7,8]

Other national data used were on intensive care unit (ICU) admissions from an inception-cohort study by the ANZIC Influenza Investigators which collected data on all ICU admissions for pandemic influenza A (H1N1) cases in both Australia and New Zealand.[5] A Wellington based study was used to provide additional data on length-of-stay in hospital.[7]

We explored the use of national costing data from the Ministry of Health, but this did not allow for clear enough separation of ICU and non-ICU costs. Therefore we used the Australasian cost estimates,[5] along with the New Zealand length-of-stay data (see Table 1).

We applied gamma distributions for length-of-stay and for mean cost-per-person-per-day in ICU and non-ICU settings, and conducted probabilistic sensitivity analysis using the software "@Risk for Excel" (version 5.7 Palisade, Sydney). We applied purchasing power parity adjustments to produce cost results in NZ$ for 2009.[9]

Results

We estimated the mean total ICU cost to be NZ$9.9 million and the mean total non-ICU hospitalisation cost to be NZ$20.6 million for the 2009 pandemic (Table 1). That is, a total mean cost to the hospital sector of NZ$30.5 million (95% uncertainty interval (UI): 22.3 to 39.5 million). The mean cost per capita was NZ$7.01.

While we have focused on performing a cost-of-illness study, a simplistic and hypothetical cost-effectiveness analysis can also be considered. That is, we assumed the counterfactual of "no hospital care" (e.g., as if hospital services were completely overwhelmed during a pandemic) and that this lack of care resulted in 100% of the year 2009 ICU cases dying and 10% of non-ICU hospitalised cases dying.

Given such assumptions, this would suggest that hospital care has a relatively high cost-effectiveness in the order of NZ$155,000 per life saved from pandemic influenza [i.e., NZ$30.5 million / ((102 in ICU – 16 [who died based on the ANZIC Influenza Investigators data]) + (1122 – 11 [who died[3]]) × 10%)].

Given that the median age of hospitalised cases was 26.7 years and only 2.0% (5/49) of all pandemic-attributable deaths were among those under age 65 years,[3] the cost-effectiveness of hospital care in preventing years-of-life-lost, would probably be very favourable. However, this benefit is hard to calculate precisely in the population hospitalised with pandemic influenza given the relatively high levels of co-morbidity in this population,[3] and hence lower than average life expectancy.

Table 1. Details of input parameters and results of the probabilistic sensitivity analysis for estimating hospitalisation costs in New Zealand attributable to the 2009 influenza pandemic (n=2000 iterations using @Risk)

Key parameter (2009 influenza pandemic related)	Data inputs	Details and approaches to modelling uncertainty
Input data – ICU admissions		
Number of ICU admissions in NZ in the period 1 June 1 to 31 August 2009 (ANZIC Influenza Investigators database)	N=102*	–
Days stay in ICU in NZ (ANZIC Influenza Investigators database)	Median=5 days Mean=12.41 days (standard deviation [SD]=14.80)	To provide for population level variation we calculated the standard error (SE) of the mean (SE=1.47) and used this in our analysis. Based on the distributional pattern for both Australia and NZ data (Figure 1 in Higgins et al[5]), we applied the distribution with the best fit (gamma) for length-of-stay in ICU (alpha=71.74, beta=0.17).
Mean cost-per-person-per-day in ICU for H1N1 cases (2009 Australian dollars)[5] [A$63,298 / mean days (8.4)=A$7,535].	A$7,535 per day (NZ$7,787)	We used a gamma distribution with a SD of approximately ± 20% of the mean (i.e., alpha=25.00, beta=311.50).

Key parameter (2009 influenza pandemic related)	Data inputs	Details and approaches to modelling
Time in hospital (outside of the ICU)		
Days stay in hospital before or after ICU, ie, for those (n=88) for whom data were available (ANZIC Influenza Investigators database)	Median=5 days Mean=10.86 days (SD=19.23)	To provide for population level variation we calculated the SE of the mean (SE=1.90) and used this in our analysis. We applied the same type of distribution as for the ICU data (gamma) (albeit different alpha=32.57, beta=0.33).
Mean cost-per-person-per-day (based on data in an Australasian study[5] in 2009; and generated by subtracting the total mean hospital cost for these patients from the total mean cost of the ICU stay and then dividing by the mean days spent in hospital (outside of the ICU)). [(A$85,359 – A$63,298) / (15.5 days – 7 days)=A$2595]	A$2595 per day (NZ$2,682)	We used a gamma distribution with a range of approximately ± 20% of the mean (alpha=25.00, beta=107.27).
Other hospital admissions (non-ICU)		
Number of hospitalisations in NZ (2009)[3] with a primary diagnosis of "pandemic influenza A(H1N1) 2009"	N=1122	–
Mean days stay in hospital (based on published data from a Wellington, NZ study[7] (where mean duration of admission was 6.1 days, for range 0–24 days).**	Mean=6 days, SD=3 days	We derived estimates from the published data (Figure 2 in Verrall et al[7]) to calculate the SE of the mean (SE=0.2). We used a gamma distribution with alpha=916.00 and beta=0.01.
Mean cost-per-person-per-day. Given the absence of NZ data we used the data for hospital cases calculated for the Australasian study[5] above.	A$2,595 per day (as per the above cost-per-day estimate)	Of note is that this cost estimate might be an over-estimate as it is derived from those who had been particularly ill (i.e., had previously been in ICU).
Results	**Median [Mean] (NZ$)**	**95% Uncertainty interval (NZ$)**
ICU – cost-per-person	95,000 [97,000]	59,000 – 146,000
ICU – total costs	9,653,000 [9,857,000]	6,060,000 – 14,923,000
Hospital (not-ICU) – cost-per-person	17,000 [17,000]	11,000 – 23,000
Hospital (not-ICU) – total costs	20,488,000 [20,626,000]	13,890,000 – 28,177,000
Total hospital costs (ICU + non-ICU)	30,204,000 [30,483,000]	22,250,000 – 39,525,000
Total hospital costs (ICU + non-ICU) per capita (NZ population in 2009)[10]	6.95 [7.01] per capita	5.12 – 9.09 per capita
Total hospital costs (ICU + non-ICU) in US$	US$20,124,000 [20,310,000]	US$14,825,000 – 26,335,000

Notes:

* Other work has reported a higher estimate (n=119)[3], but this was for a longer time period. The ANZIC Influenza Investigators dataset involved carefully identifying all transfers (using initials, date-of-birth, day of discharge from one ICU and day of admission to another etc), and only included them as one ICU admission.

** A slight limitation with these NZ data are that they include 19 cases (8% of the total) who were admitted to "intensive care or high dependency units for at least 1 night".

Discussion

These cost estimates are the first we know of for all hospitalisations from the 2009 influenza pandemic at a country-level. They suggest a significant extra cost to the health sector from even a relatively non-severe influenza pandemic (compared to previous influenza pandemics for New Zealand[11–13]). Nevertheless, these estimates are still likely to be underestimates of the true costs to the hospital sector given that

the calculations in the Australasian study[5] did not include certain cost items (e.g., "blood products" even though usage of these was relatively high for patients treated with extracorporeal membrane oxygenation [ECMO]).

Furthermore, we have not included the costs associated with disruption to normal hospital operations e.g., as elective surgical procedures were cancelled as ICU space became very constrained in some New Zealand and Australian hospitals.[14]

A more sophisticated analysis would also consider a wider range of locality-specific factors (e.g., actual New Zealand data on hospital costs, national level data on length-of-stay, and correlations between length-of-stay and average daily cost). Nevertheless, at the ICU level, the experience for patients in Australia and New Zealand appeared to be fairly similar, with similar rates of ECMO use (7% in Australia compared with 8% in New Zealand), invasive ventilation (64% compared to 53%) and case-fatality proportions (16% for both groups).

A wider health system perspective would consider costs for emergency departments, primary care, and the public health sector. Societal costing would consider the contribution of morbidity, premature death, absenteeism from work and educational settings, and impacts on the tourism industry. Given the sudden and unpredictable nature of such pandemics, there is a case for further study of these costs – to help determine the appropriate scale of pandemic planning and preventive measures.

In summary, this analysis provides initial estimates (with uncertainty estimates) of the hospitalisation costs to the New Zealand Government during the first wave of the 2009 influenza pandemic. But given the relatively non-severe nature of this pandemic, it is likely to provide only an approximate lower bound cost for this sector from new influenza pandemics in the future.

Much more complete costing studies are probably warranted in this and other countries to guide future decision-making around investment into influenza pandemic planning. Nevertheless, these high cost estimates indicate the potential value of further work on preventive measures (e.g., vaccination) and of investing in pandemic planning and other control measures to reduce person-to-person spread.

Author information: Nick Wilson[1], Nhung Nghiem[1], Alisa Higgins[2], Giorgi Kvizhinadze[1], Michael Baker[1], Tony Blakely[1]

1. Department of Public Health, University of Otago, Wellington, New Zealand
2. Australian and New Zealand Intensive Care Research Centre, School of Public Health and Preventive Medicine, Monash University, Melbourne, Australia

Clinical Trials Group for access to data. June Atkinson provided assistance in exploring the value of Ministry of Health costing data.

Correspondence: Associate Professor Nick Wilson, Department of Public Health, University of Otago – Wellington, Box 7343, Wellington South, New Zealand. Fax: +64 (0)4 3895319; email: nick.wilson@otago.ac.nz

References

1. Baker MG, Wilson N, Huang QS, Paine S, Lopez L, Bandaranayake D, et al. Pandemic influenza A(H1N1)v in New Zealand: the experience from April to August 2009. Euro Surveill. 2009;14:ArticleId=19319.

2. Baker M, Huang Q, Bandaranayake D, Mackereth G, McArthur C, Paine S, et al. The influenza A(H1N1) pandemic in New Zealand during 2009 [Poster presentation 217]. 2010 International Conference on Emerging Infectious Diseases. Atlanta, Georgia 11-14 July. 2010.

3. Pandemic Influenza Mortality and Morbidity Review Group. Report for the Minister of Health from the Pandemic Influenza Mortality and Morbidity Review Group. Wellington: Ministry of Health, 2010.

4. Webb SA, Pettila V, Seppelt I, Bellomo R, Bailey M, Cooper DJ, et al. Critical care services and 2009 H1N1 influenza in Australia and New Zealand. N Engl J Med. 2009;361:1925-34.

5. Higgins AM, Pettila V, Harris AH, Bailey M, Lipman J, Seppelt IM, et al. The critical care costs of the influenza A/H1N1 2009 pandemic in Australia and New Zealand. Anaesth Intensive Care. 2011;39:384-91.

6. Centers for Disease Control and Prevention. Surveillance for the 2009 pandemic influenza A (H1N1) virus and seasonal influenza viruses - New Zealand, 2009. MMWR Morb Mortal Wkly Rep. 2009;58:918-21.

7. Verrall A, Norton K, Rooker S, Dee S, Olsen L, Tan CE, et al. Hospitalizations for pandemic (H1N1) 2009 among Maori and Pacific Islanders, New Zealand. Emerg Infect Dis. 2010;16:100-2.

8. Dee S, Jayathissa S. Clinical and epidemiological characteristics of the hospitalised patients due to pandemic H1N1 2009 viral infection: experience at Hutt Hospital, New Zealand. N Z Med J. 2010;123:45-53.

9. OECD. StatExtracts PPPs and exchange rates: OECD, 2011.

10. Statistics New Zealand. National Population Estimates: December 2009 quarter. Wellington: Statistics New Zealand, 2012. http://www.stats.govt.nz/browse_for_stats/population/estimates_and_projections/NationalPopulationEstimates_HOTPDec09qtr.aspx

11. Rice G. Black November: The 1918 influenza pandemic in New Zealand. Christchurch: Canterbury University Press, 2005.

12. Davis L. Report on the 1957 pandemic of influenza (to the World Health Organization, Geneva). Wellington: Division of Public Hygiene, Department of Health, 1958.

13. Wilson N, Telfar Barnard L, Summers J, Shanks G, Baker M. Differential mortality by ethnicity in 3 influenza pandemics over a century, New Zealand. Emerg Infect Dis. 2012;18:71-77.

14. Drennan K, Hicks P, Hart G. Impact of pandemic (H1N1) 2009 on Australasian critical care units. Crit Care Resusc. 2010;12:223-9.

Haemophilus influenzae type b disease in Auckland children during the Hib vaccination era, 1995–2009

Bonnie Leung, Susan Taylor, Dragana Drinkovic, Sally Roberts, Phil Carter, Emma Best

Abstract

Aim To characterise *Haemophilus influenzae* type b (Hib) invasive disease in the era of Hib vaccination, in children of the greater Auckland region of New Zealand.

Method Identification of sterile site culture positive Hib via the Auckland hospital laboratories databases and national laboratory surveillance database in the time period; 1995 to 2009.

Results There were a total of 26 cases in the Auckland Region. Over the 15-year period, the annual incidence of invasive Hib disease was 0.61 per 100,000 (95% CI: 0.4–0.9) for children aged under 15 years and 1.65 per 100,000 (95% CI: 1.1–2.5) for children aged under 5 years. Ninety-two percent were under 5 years and 54% were under 1 year. Sixty percent of the children were of Māori and Pacific ethnicity. The predominant diagnosis was meningitis, accounting for 15 cases (60%). There were no fatalities. Forty-eight percent of affected children were completely unimmunised with the Hib vaccine which has been fully funded on the National Immunisation Schedule since 1994.

Conclusion Since the introduction of the Hib vaccine, the disease rates have greatly reduced in the Auckland region. Although ethnic disparities have improved amongst the cases that occur, immunisation rates in cases are low and infants remain most at risk. Current emphasis on intensifying immunisation programmes to achieve higher vaccination rates and timeliness of delivery will help in efforts to achieve elimination of the disease in New Zealand.

Haemophilus influenzae (Hi) is a Gram-negative coccobacillus that exists as one of six distinct capsulated strains or as a non-encapsulated strain. The encapsulated serotype b (Hib) is a recognised cause of life-threatening invasive infection in children under 5 years. Prior to vaccination in New Zealand, Hib caused 95% of Hi invasive disease in infants and children.[1]

In Auckland, disease rates were reported as 43 per 100,000 in children under 5 years, with rates in Pacific and Māori children even higher (57 per 100,000).[2,3] Over a quarter of these cases occurred in the first 6 months of life. Hib was the major pathogen of bacterial meningitis in infants and young children in Auckland prior to vaccination.[3]

New Zealand has had effective immunisation against Hib since 1994 as part of the National Immunisation Schedule. Since then there has been a 90% reduction in incidence of Hib disease in children aged under 5 years.[1] Immunisation reduces the frequency of asymptomatic colonisation of Hib, but in the unimmunised child, severe

Hib invasive disease may still occur. We reviewed the cases of Hib disease in the paediatric population in Auckland after the implementation of the Hib vaccine over the 15-year period of 1995 to 2009.

Method

All cases of invasive Hib disease occurring in children under 15 years from the Auckland Region, between first of January 1995 through to end of December 2009 were ascertained and reviewed.

Hib cases were included if there was a laboratory confirmed isolate of Hib from a sterile site. Study investigators at the Auckland District Health Board, Counties Manukau District Health Board and Waitemata District Health Board laboratories searched their databases and cross referenced with the Environmental Science and Research (ESR) national database to ensure all cases were identified.

Paper and computer scanned medical records were reviewed. Typing information was provided by ESR. From the end of 1997 all invasive Hi isolates referred to ESR were further tested by polymerase chain reaction for the presence of capsular gene and type b capsule. Hib became a notifiable disease in 1996 and notification data along with isolate typing was provided from the ESR national reference laboratory.

Cases included in the analysis were from the Auckland Region. One case was excluded as the child was not resident in the Auckland Region at the time of illness onset.

Changes to the National Immunisation Schedule regarding the Hib vaccine were reviewed over the period from 1994 through to 2009. In 1994, the Hib vaccine was first introduced as a component of the quadrivalent vaccine (DTwPH). This was changed to the polyribosylribitol phosphate outer membrane protein (PRP-OMP) Hib vaccine in 2000 as it offered more antibody protection after early doses, enabling better protection of young infants.

In 2008, a hexavalent polyribosylribitol phosphate tetanus toxoid (PRP-T) Hib vaccine was introduced which involves a primary course of three doses and a booster at 15 months. [4]

Immunisation status was categorised in the following way; Complete, Partial, Missed and Unimmunised. Children who received all doses of the Hib vaccine including the booster were categorised into "Complete". Those that were up to date for their age group but too young to receive all doses and booster were categorised into "Partial".

Children that had received a Hib vaccine but missed other age-appropriate doses were categorised into "Missed". Finally children that had not received any immunisations were categorised into "Unimmunised".

Rates were calculated using Statistics New Zealand estimated resident population denominators. Each case's address at diagnosis was used to assign 2006 census area unit codes enabling an area-based NZ Deprivation Index 2006 decile to be assigned. Data were compiled and analyzed using SPSS version 16.0.

Ethical approval was obtained from the Northern Regional Ethics committee in January 2010.

Results

A total of 26 paediatric cases of *Haemophilus influenzae* type b (Hib) disease were identified in the Auckland Region between 1995 and 2009. Complete clinical records were available for 25 of 26 cases.

The annual incidence over the 15-year time period, was 0.61 per 100,000 for children aged under 15 years and 1.65 per 100,000 for children aged under 5 years. The incidence of invasive Hib in the Auckland Region did not vary significantly over time during this period. However it is significantly less than the pre-vaccine rate reported for the Auckland Region for 1981–1987 (Figure 1).[3]

During 1995–2009, the highest rates were observed in children aged less than 6 months (5.2 per 100,000), and rates in children less than 1 year remained significantly

higher than rates in children aged 3 to 14 years (Table 1). The majority of cases were aged less than 1 year and all but 2 cases were aged less than 5 years.

Figure 1. Incidence of invasive *Haemophilus influenzae* type b disease in children aged 0–14 years, Auckland Region 1981–87 (pre-vaccination period) and 1995–2009
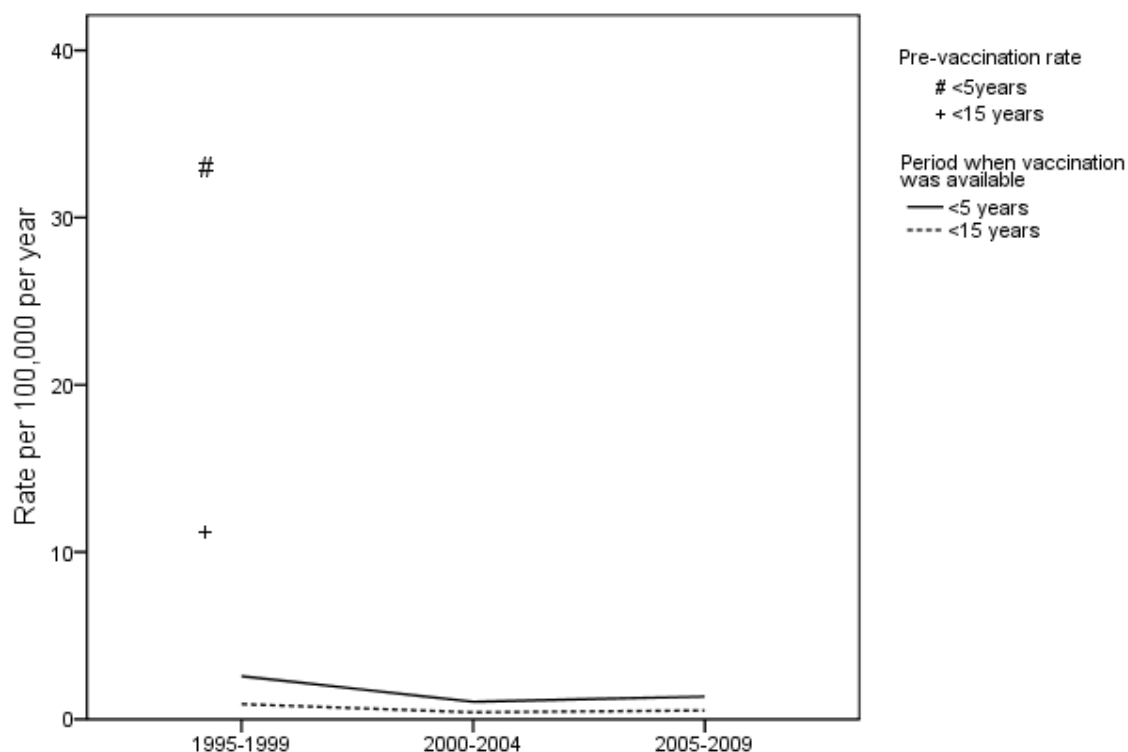
There were no significant differences in incidence by gender, ethnicity and New Zealand Deprivation Index (NZDep) at their home residence (Table 1).

Table 1. Sociodemographic data of Auckland children with *Haemophilus influenzae* type b disease, 1995–2009

Age	Cases (N)	Rate per 100,000	95% CI
< 6 months	8	5.17	2.46- 10.47
6-12 months	6	3.88	1.59-9.76
1-2 years	5	0.86	0.31-2.09
3-14 years	7	0.21	0.09- 0.44
Gender	Cases (N)	Rate per 100,000	95% CI
Male	12	0.55	0.30-0.97
Female	14	0.67	0.39-1.15
Ethnicity	Cases (N)	Rate per 100,000	95% CI
Māori	9	1.13	0.57- 2.21
Pacific	6	0.74	0.30- 1.66
European & Other	10	0.38	0.20- 0.71
NZDep			
Decile	Cases (N)	Rate per 100,000	95% CI
1-7	14	0.53	0.31 -0.91
8-10 (most deprived)	11	0.67	0.36- 1.22

Amongst cases of invasive Hib disease only 3 (12%) children were completely immunised (Table 2). There were 15 cases of meningitis (60%). Eight of these children were unimmunised, three had missed immunisations, two were partially immunised and two completely immunised.

The median age of children with Hib meningitis was 1.9 years. Other presentations included pneumonia which accounted for five cases (20%), epiglottitis in two cases (8%), bacteraemic sepsis, septic arthritis and facial cellulitis involving one case each (4%).

Seven (28%) children had significant underlying conditions, including one with congenital heart disease, two with syndromic developmental delay (Down's syndrome, Wolf-Hirschhorn Syndrome), one with motor development delay, one with dysplastic kidney and two with recurrent pneumonia.

Of these seven children, six were unimmunised; two due to parental choice. Five of the children were seeing tertiary services prior to Hib disease and only one was fully immunised.

Table 2. Immunisation status and sequelae of invasive *Haemophilus influenzae* type b disease in children aged 0–14 years, Auckland Region 1995-2009

Immunisation status	Number of Hib vaccines received	N (%)	Sequelae after Hib disease N (%)
Unimmunised	No Hib vaccines received	12 (48%)	2 (8%)
Missed	At least one Hib vaccine received but missed other age-appropriate doses	6 (24%)	2 (8%)
Partial	Up to date for their age at presentation but too young to receive all doses	4 (16%)	0 (0%)
Complete	All doses of the Hib vaccine including the booster received	3 (12%)	1 (4%)

Complete treatment data was available for 23 cases. Fourteen cases used cefotaxime alone or followed by amoxycillin, cefuroxime or amoxycillin/clavulanate. The median duration of treatment was 10 days (range of 5 to 57 days). Amoxycillin resistance was present in two of the Hib isolates. One isolate also had intermediate resistance to rifampicin but all were susceptible to ceftriaxone, cefuroxime, co-trimoxazole and amoxycillin/clavulanate.

Of the 26 isolates, two were nontypable by conventional typing and were identified by polymerase chain reaction to have both the capsule and type b gene present. Both of these cases had clinically consistent findings of invasive Hib disease which included septic arthritis in a 6-month-old unimmunised infant and facial cellulitis in a 3-month-old infant who had received one Hib vaccine.

There were no fatalities, although five children had sequelae after the Hib disease. Four children with meningitis had significant neurological consequences including paresis, delayed motor development and hearing impairment requiring cochlear implants. The child who presented with septic arthritis had a knee effusion for a month. Two of these children had missed immunisations and two were unimmunised.

Discussion

In this retrospective study of invasive Hib disease during the era of Hib vaccination in children between 1995 and 2009, 26 cases were identified in the greater Auckland Region. The overall incidence over the 15-year time period was 0.61 per 100,000 for children aged less than 15 years and 1.65 per 100,000 for children aged less than 5 years.

This is markedly reduced from rates of 14 per 100,000 for under 15 years and 41 per 100,000 for under 5 years in the pre-vaccination period of 1981 to 1987 in Auckland[3] and indicates that elimination of Hib disease in New Zealand is achievable. Meningitis was the predominant diagnosis with other classic and life-threatening presentations of Hib also observed.

The decrease in the incidence of invasive Hib disease in the Auckland Region is consistent with prior New Zealand literature observing a 90% reduction of disease in children under 5 years since the vaccine was introduced in 1994.[1] *Haemophilus influenzae* type b immunisation is postulated to prevent at least 80 cases of meningitis and 30 cases of epiglotittis every year in children under 5 years in New Zealand.[5]

Our observed Auckland rate is consistent with national surveillance data from 1997 to 2005, which indicates mean annual rate of Hib disease of 0.77 per 100,000 (standard deviation 0.42) children under 15 years.[6]

Hib disease rates in Auckland compare favourably with those reported in Australia and other developed countries. Since the introduction of Hib immunisation in Australia in 1993, incidence rates for the period from 1997 to 2000 were reported as 1.7 cases per 100,000 in children under 5 years.[7]

Similar reductions in Hib disease have been seen in other countries with routine Hib immunisations such as the United States (1.4 per 100,000 children under 5 years) and United Kingdom (1.8 per 100,000).[8,9] However significantly higher rates of invasive Hib disease are still observed amongst Australian Aboriginal children despite high vaccine coverage (6.7 per 100,000 in children under 5 years).[7]

More recently, Australian national Hib disease rates have declined further to 0.5 per 100,000 in children aged under 5 years, whilst still remaining higher in the Northern Territory (1.2 per 100,000).[10]

Although 60% of our cohort were Māori and Pacific, the small number of total cases is likely to have meant ethnic differences in Hib incidence were not statistically significant. This is in contrast to the pre-vaccine period in New Zealand where Māori and Pacific children had higher rates of invasive disease and were younger at presentation.[2] However, one of the potential benefits of immunisation is to eliminate Hib invasive disease and reduce ethnic health inequalities which may otherwise be difficult to address with other health interventions. The same improvement across ethnic groups was also observed in the New Zealand meningococcal B campaign.[11]

Our review demonstrates young children continue to be the age group most at risk with 56% of cases aged less than 1 year, consistent with the pre-vaccine data in the Auckland Region where 60% of Hib cases were infants.[3]

Of the 14 cases aged less than 1 year, only one was completely immunised. Five children were unimmunised (36%) and the other 8 had either partial or missed immunisations. Current governmental health targets are important in working toward not only achieving 95% of children being fully immunised by the age 2 years, but also timely immunisation, particularly of infants.[12]

Delayed infant immunisation is a risk for acquisition of Hib in indigenous communities leading to persistent carriage.[13] The higher rate of Hib disease enduring in Auckland in light of Australia's improved national rate demonstrate carriage and transmission still occur in our young and unimmunised children. Efforts to eliminate invasive Hib disease should mean that each case be regarded as a sentinel event and indicative of ongoing Hib transmission within a reservoir, whether it be a family or a community with poor health access.[14]

Of the seven children with pre-existing medical conditions, six were unimmunised; two by parental choice. Of the five children seen by tertiary services prior to the episode Hib disease, only one was fully immunised. This highlights an avenue of missed opportunity where encouraging adequate immunisation during consultations for other medical conditions would benefit children at increased risk.

Prior to the introduction of immunisation, Hib invasive disease had a case fatality proportion of 5 to 10%.[8] Furthermore, survivors of Hib meningitis had a 15 to 30% risk of long-term neuro-developmental impairment.[8] Although no fatalities were observed in this study, sequelae occurred in 20%. Following with meningitis, consequences were severe and long standing.

Amongst invasive Hib isolates, 85% were susceptible to amoxycillin. Although national susceptibility of all invasive Hi isolates demonstrates increasing amoxycillin resistance over the past decade, our small numbers in this review did not show trends of increasing amoxycillin resistance.[15] Current empiric therapy for paediatric meningitis usually includes third generation cephalosporins to which all Hib isolates remain susceptible.[16]

This study also demonstrated the impact of vaccination at both at the population and individual level. Although immunisation has reduced the overall incidence of Hib within the Auckland Region, for the individuals that had invasive Hib, poor immunisation uptake was apparent. Only 12% were fully immunised and almost half were unimmunised. In most cases, the reason for the child being unimmunised was not specified in medical records.

Limitations include the fact that hospital admission and mortality data were not searched for International Classification of Diseases (ICD) codes consistent with invasive Hib. A prior New Zealand Hib disease review identified a very small number of additional cases using ICD9 codes.[5] Hospital admission and mortality data are currently coded using ICD10 codes which are unable to distinguish Hib from other capsular Hi or nontypable cases without chart or laboratory data review. Thus the number of additional cases of true Hib missed by our review of both national and local laboratory databases is likely to be very small.

One of the strengths of this review was laboratory database search including sterile and non sterile site paediatric *Haemophilus influenzae* isolates. This enabled detection and inclusion of cases such as those nontypable by conventional methods but detected by polymerase chain reaction and shown to be consistent clinically.

Conclusion

In Auckland, Hib is now a rare disease following effective Hib immunisation and New Zealand is moving toward elimination of the disease. However opportunities to improve immunisation still exist. Immunisation rates are poor amongst those with Hib invasive disease and young children remain most at risk.

Opportunities for immunisation of children should be encouraged, particularly amongst those with prior specialist medical contact. Furthermore, emphasis should be placed on implementing existing immunisation programmes to further raise uptake rates and timeliness of immunisation.

Author information: Emma J Best, Paediatric Infectious Diseases Consultant, Senior Lecturer, Department of Paediatrics, University of Auckland and Department of Infectious Diseases, Starship Children's Health, Auckland; Bonnie Leung, Trainee Intern, University of Auckland; Susan Taylor, Clinical Microbiologist, Middlemore Hospital, Counties Manukau District Health Board, Otahuhu, Auckland; Dragana Drinkovic, Clinical Microbiologist, Waitemata District Health Board, Takapuna, Auckland; Phillip Carter, Principal Scientist, Institute of Environmental Science and Research (ESR), Wallaceville, Wellington; Sally A Roberts, Clinical Head of Microbiology Department, Auckland District Health Board, Auckland

Correspondence: Dr Emma Best, Senior Lecturer, Department of Infectious Diseases, Starship Children's Health, Auckland District Health Board, Park Road, Auckland, New Zealand. Fax: +64 (0)9 3078977; email: ebest@adhb.govt.nz

References:

1. New Zealand Immunisation Handbook 2011. Ministry of Health: Wellington; 2011, p155–58.

2. Lennon D, Voss L, Gillies M, et al. Epidemiology of invasive Haemophilus influenzae (Hi) disease in New Zealand (1978-87). Paediatric Research. 1989;25:183A.

3. Voss L, Lennon D, Gillies M. Haemophilus influenzae type b disease in Auckland children 1981-87. N Z Med J. 1989;102:149-51.

4. Reid S. Evolution of the New Zealand childhood immunisation schedule from 1980: a personal view. N Z Med J. 2006;119.

5. Wilson N, Wenger J, Mansoor O, et al. The beneficial impact of Hib vaccine on disease rates in New Zealand children. N Z Med J. 2002;115.

6. New Zealand Public Health Observatory; Notifiable Disease, Counts and rates by age group. [accessed 29 May 2012]. Available from http://www.nzpho.org.nz/NotifiableDisease.aspx

7. Horby P, Gilmour R, Wang H, et al. Progress towards eliminating Hib in Australia: an evaluation of Haemophilus influenzae type b prevention in Australia, 1 July 1993 to 30 June 2000. Commun Dis Intell. 2003;27:324-41.

8. Bisgard KM, Kao A, Leake J, et al. Haemophilus influenzae invasive disease in the United States, 1994-1995: near disappearance of a vaccine-preventable childhood disease. Emerg Infect Dis. 1998;4:229.

9. Peltola H. Worldwide Haemophilus influenzae type b disease at the beginning of the 21st century: global analysis of the disease burden 25 years after the use of the polysaccharide vaccine and a decade after the advent of conjugates. Clin Microbiol Rev. 2000;13:302.

10. Wang H, Deeks S, Glasswell A, et al. Trends in invasive Haemophilus influenzae type b disease in Australia, 1995-2005. Commun Dis Intell. 2008;32:316-325.

11. Lennon D, Reid S, Stewart J, et al. Reducing inequalities with vaccines: New Zealand's MeNZB vaccine initiative to control an epidemic. J Paed Child Health. 2011; Oct 14

12. Targeting Immunisation: increased immunisation. [accessed 20 December 2011]. Available from http://www.health.govt.nz/publication/targeting-immunisation-increased-immunisation

13. Singleton R, Bulkow LR, Levine OS et la. Experience with the prevention of invasive Haemophilus influenzae type B disease by vaccination in Alaska; the impact of persistent oropharyngeal carriage. J Pediatr. 2000;137:313-320

14. Millar EV, O'Brien KL, Levine OS, et al. Toward elimination of Haemophilus influenzae type B carriage and disease among high-risk American Indian children. American Journal of Public Health. 2000;90(10):1550.

15. Antimicrobial susceptibility of invasive Haemophilus influenzae, 2010 [accessed 20 December 2011]. Available from http://www.surv.esr.cri.nz/PDF_surveillance/Antimicrobial/HIN/HIN_2010.pdf

16. Voss L, Nicholson R. Meningitis. Auckland: Starship Children's Health Clinical Guideline; 2009 [accessed 10 July 2011]. Available from: http://www.starship.org.nz/assets/Uploads/Starship-Hospital-Content/Health-Professionals/Clinical-Guidelines/Meningitis.pdf

The prevalence of *Helicobacter pylori* infection in Sherpa residents of the Upper Khumbu, an isolated community in eastern Nepal

Tshering W Sherpa, Kami T Sherpa, Garry Nixon, John Heydon, Emma Heydon, Susan Dovey

Abstract

Aim To determine the prevalence of *Helicobacter pylori* (*H. pylori*) among Sherpa residents of the Upper Khumbu region of Nepal and to test for associations between presence of *H. pylori* infection and lifestyle and health measures.

Method Written questionnaires were used to collect data from 383 individuals in randomly selected households in three villages of the region. Early morning stool samples were tested immediately for the presence of *H. pylori* antigen using standard rapid diagnostic Pylori strips. A descriptive data analysis was performed to estimate overall prevalence and its association with age, sex, dyspepsia, smoking, alcohol intake, diet, and medication use.

Results The overall prevalence of *H. pylori* in the study sample was 70.5%. The prevalence was high in all the three villages of Thame, Kunde and Fortse. Prevalence was high in all age groups, including a high prevalence of 78.1% in children aged <10 years. The presence of *H. pylori* was not significantly associated with any of the lifestyle and health measures collected, including dyspeptic symptoms, medication, smoking, alcohol intake and dietary factors like salt, smoked food, fruit/vegetable and pickle consumption.

Conclusion The overall prevalence of *H. pylori* in Upper Khumbu is high with the infection being acquired early in the first decade of life. This lifelong infection may explain the very high incidence of gastric cancer in this community. The rate of infection is not dependent on individual variables including demographic, social and dietary factors.

The upper Khumbu, located in the eastern Himalayas of Nepal, where many residents live above 3400 meters, is one of the highest permanently inhabited regions of the world. It is home to around 3300 indigenous Sherpa people. The region is isolated, more than a week's walk from the nearest road-end. Kunde Hillary Hospital was built by Sir Edmund Hillary in 1966 and is the primary medical centre for the Upper Khumbu area.

Both dyspepsia and gastric cancer appear to be particularly prevalent in this Sherpa community. According to hospital records, dyspepsia accounted for more than 12% (1590) of the total (12,904) outpatient consultations made in the 2 years, July 2007 to July 2009. Over the same period 9 patients died of histologically proven gastric cancer, a mortality rate of 269 per 100,000 per annum (confidence interval (CI): 131.4, 530.2) in this Sherpa population.

By comparison, the International Agency for Research on Cancer (IARC) estimates the age-standardised worldwide mortality rate for gastric cancer as 10.3 per 100,000.[1] There were no recorded deaths from other forms of cancer over the same period. Anecdotal evidence obtained from the resident doctors suggests that dyspepsia is much more common in the Sherpa community than in other ethnic groups in the region.

Current practice at Kunde Hillary Hospital is to treat all cases of dyspepsia with Histamine H2 receptor blockers and move to proton pump inhibitors should initial treatment fail. Triple therapy (omeprazole, clarithromycin and amoxicillin) for *Helicobacter pylori (H. pylori)* eradication is reserved for patients who present with upper gastrointestinal (GI) bleeding. Currently no testing for *H. pylori* is undertaken. Patients with symptoms and signs suggestive of stomach cancer are referred to the capital city, Kathmandu, for endoscopy and further management.

We postulated that this high incidence of upper GI disease could be explained by a correspondingly high prevalence of *H. pylori* and that determining this could guide treatment strategies. The link between *H. pylori*, chronic gastritis, peptic ulcer disease and malignancies including MALT lymphoma and gastric cancer is well established.[2–6]

The Eurogast study group found an approximate six-fold increase in the incidence of gastric cancer in populations with 100% *H. pylori* infection compared to a population with no infection.[5]

Infection with *H. pylori* is distributed worldwide but its prevalence is especially high in developing countries.[2,4,9] The variation in *H. pylori* prevalence between different communities is mostly due to differences in the incidence of the bacterial infection during childhood.[11]

In developing countries the infection is usually acquired during childhood and has been linked to low socioeconomic status, poor sanitation and dietary factors[9,11-13,15,16]. We aimed in this study to investigate whether *H. pylori* prevalence in the Upper Khumbu was high enough to suggest a change in clinical practice, and whether *H. pylori* was related to particular lifestyles of the Sherpa people in the region.

Methods

Sampling method and sample size—The Upper Khumbu region has a population of 3,335 (digital Himalaya Census of Nepal), with people living in small seasonal settlements and larger permanent villages scattered throughout the region.

Only Sherpas were included in the study. Members of other ethnic groups form a transient population group who live and work in the Khumbu, mostly during the trekking seasons. They are not registered in the local population databases. Only Sherpa households are included in the databases that were used to find participants. Participants were drawn from three of the largest villages – Thame (population 299), Fortse (population 322) and Kunde (population 292). These three villages provide a reasonable representation of the Upper Khumbu both geographically and socioeconomically.

A sample size of 384 was established, based on the sample size formula: $n= Z^2 \ast P\,(1-P)/d^2$ where n = *sample size, Z = Z statistics for level of significance*, P = *expected prevalence or proportion* and d = *precision*[17]. This was calculated using a *H. pylori* prevalence assumption of 50% with a 95% confidence interval and a precision of 5%. Households, rather than individuals, were randomly selected for practical and cultural reasons.

All households in the three villages were allocated specific numbers and the numbers were randomly selected using an online sample generator. All members of the selected households were included in

the study. 33, 32 and 35 households were needed from Thame, Kunde and Fortse, respectively, in order to give the desired sample size of 128 participants from each village.

The size of families living in selected houses ranged from one to nine members with an average of four. Individuals who were absent at the time of data collection and not expected to be home during the nine months study duration were excluded from the study.

Questionnaire—The *de novo* written questionnaire was piloted on 30 patients visiting Kunde Hillary Hospital. The data collection was undertaken by the community medical assistants employed by Kunde Hillary Hospital and the principle investigator. Written informed consent was sought from all eligible candidates and from the family heads in the case of children.

The questionnaires contained the following information: 1) demographics including age and sex, 2) medical history of upper GI disorders including dyspepsia, upper GI bleeds and stomach cancer, 3) medication intake in the form of antacids, histamine H2-receptors antagonists and proton-pump inhibitors, 4) family history of gastric cancer and history of gastric surgeries, 5) dietary history including salt, smoked food, fruit and vegetable and pickle intake, and 6) history of alcohol, cigarette and chewing tobacco consumption.

Ethical approval was obtained from the Nepal Health and Research Council (NHRC).

Households were visited to explain the study, seek consent and collect data. Study participants were given clean, leak proof plastic containers without preservatives and were asked to bring two loops of early morning stool samples to the villages' clinic laboratory. Participants were asked to stop taking antibiotics and proton pump inhibitors for four weeks and to stop taking H2 receptor blockers and antacid for two days prior to providing a sample. The stool samples were stored in icepacks and study tests were performed on the day of stool collection.

***H. pylori* stool antigen test—**Most *H. pylori* prevalence studies have been based on serological testing. However, the sensitivity and specificity of serology is low and serology tests do not distinguish between past and current infection.[18,19]

Endoscopy and biopsy followed by culture is considered the gold standard for establishing *H. pylori* infection but this can be performed only in specialized centres and is not practical for population based studies. The 13C-urea breath test (UBT) has a high sensitivity and specificity but is expensive and difficult to perform.[20] In a resource limited setting like ours the stool antigen test appeared to be a valuable alternative. The stool antigen test is highly sensitive and specific.

A systemic review of 43 studies in 2001 found that the sensitivity, specificity, positive predictive value and negative predictive value (weighted mean) were, 92.4 %(95% CI: 91.0%, 93.0%), 91.9% (CI: 91.0%, 92.0%), 92.1% (91.0%, 93.0%), and 90.5% (90.0%, 91.0%) respectively[21]. In view of its high diagnostic accuracy and ability to determine current infection, the stool antigen test can be a favoured approach in prevalence studies. Moreover the test is non-invasive, cost effective and simple to perform, and therefore was considered most appropriate for use in our study.

Commercially available test kits manufactured by Coris Biocept were used in accordance with the manufacturer recommendations. According to the manufacturers specifications the kits have a sensitivity of 97.1% and specificity of 96.7%. It is a spot test that is based on the homogeneous membrane system technology with latex microspheres.

The stool samples were diluted with 0.5 ml of buffer (HC) solution (Saline solution buffered to pH 7.5 with Tris-EDTA, NaN_3 (<0.1%), a detergent, and charged proteins) until a homogenous solution was obtained. The test strip was then dipped into the faecal suspension and allowed to stand for up to 10 minutes. The interpretation of the test depends on the appearance of coloured lines on the strip (one green line = negative; one green line AND one red line = positive; no line = invalid). Invalid tests were repeated again for reconfirmation.

Statistical analysis—The statistical analysis used SPSS (Statistical Package for the Social Sciences) and Stata 11 software.[22] Comparison of the variables used Pearson's Chi-squared test, Fisher's exact test for smaller values and a test for trend. Logistic regression analyses were run with age, sex, heartburn history, family history of gastric cancer and village (± dietary variables) in the models, run both taking into account household clustering and without clustering.

Participants who requested it were provided with the results of their test.

Results

The total sample size was 383; 174(45.4%) males and 209(54.6%) females. No one refused to join the study and only one person was dropped from the study (failure to provide a stool sample). Overall 270 (70.5%) participants were positive for *H. pylori*. There was no significant difference in prevalence between sexes.

The sample was divided into age groups with 108(28.2%) children under 19 years, 201 (52.5%) adults aged 19 to 59 years and 74(19.3%) elderly over 60 year old. The prevalence (95% CI) in these three age groups was 72.2% (62.6, 80.2), 70.7% (63.7, 76.6) and 67.6% (55.6, 77.7) in children, adults and elderly respectively, as shown in Figure 1.

Children aged <10 years had higher *H. pylori* prevalence than other age groups (78.1%) but none of the differences between age groups reached statistical significance (p-value for trend = 0.509). *H. pylori* prevalence in Fortse, Thame and Kunde was 62.2%, 72.7% and 76.6 % respectively. The difference between the villages was statically significant (p=0.034).

Figure 1. *H. pylori* results according to age group

% H. pylori positive
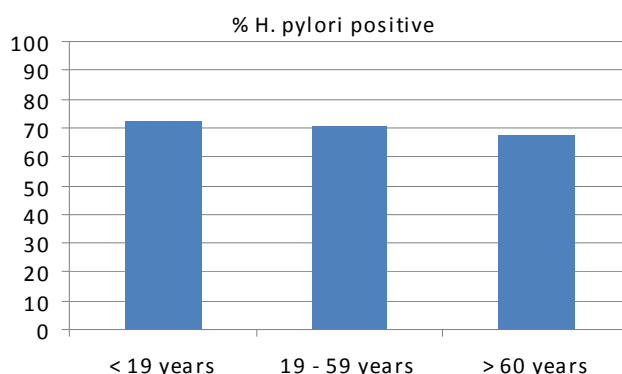
A history of dyspepsia was reported by 154(40.20%) participants, 15(3.91%) had a history of upper GI bleeding, and 18 (4.93%) had a family history of stomach cancer. No statistically significant association was identified between *H. pylori* infection and dyspepsia, upper GI bleeding or family history of stomach cancer, as shown in Table 1. Likewise there was no statistically significant association between *H. pylori* and diet, tobacco use or alcohol consumption.

Table 1. Association of medical history, lifestyle factors and dietary factors with *H. pylori* infection

Medical and lifestyle variables	Total	N (%) *H. pylori* positive	P value
History of dyspepsia			
-No	229	167(72.9)	0.204[a]
-Yes	154	103(66.9)	
History of upper gastrointestinal bleeding			
-No	368	261 (70.9)	0.363[a]
-Yes	15	9 (60.0)	
Family history of stomach cancer			
-No	365	260(71.2)	0.155[a]
-Yes	18	10(55.6)	
Smoking status			
-Never smoked	368	259(70.4)	0.937[b]
-Ex-smoker	2	2(100.0)	
-Current smoker	13	9(69.2)	
Chewing tobacco use			
-No	371	263(70.9)	0.348[a]
-Yes	12	7(58.3)	
Alcohol consumption			
-Never	247	178(72.1)	
-Social	110	75(68.2)	0.237[b]
-Daily	19	14(73.7)	
-Heavy drinker[d]	7	3(42.9)	
Dietary variables	**Total**	**N (%) *H. pylori* positive**	**P value**
Smoked food intake			
-Low	381	269(70.9)	0.504[c]
-High	2	1(50.0)	
Salt intake			
-Low	335	235(70.2)	0.694[a]
-High	48	35(72.9)	
Fruit/vegetable intake			
-Low	344	239(69.5)	0.194[a]
-High	39	31(79.7)	

[a]Pearson's Chi^2 test; [b] Test for trend; [c]Fisher's exact test (if cell count <5).

[d]Daily consumption starting in the morning.

Discussion

We found in this study that the prevalence of H. pylori infection in the Upper Khumbu region of Nepal was 70.5%. The methods used for this study may be unusually robust in that samples were collected from almost 100% of Sherpa people living in a random sample of households of remote villages, using a test that is specific for active disease.

In most other prevalence studies the samples groups have been selected by other means (e.g. blood donors) and tested using serology, which does not differentiate between active and past infection.

H. pylori prevalence is known to be related to poor socioeconomic status, household crowding, education, hygiene and sanitation.[12,13,15,16] These are all common in isolated

rural communities in developing countries. H. pylori prevalence in this study was higher than that found in a rural village of Nepal,

Kyotang in 1998 (41.5%)[23] but it is comparable to the prevalence found in isolated rural communities in other parts of the world. A prevalence of 69% was observed among residents of a rural community in the Columbian Andes.[15] Similarly, high prevalence's of 72.3% and 91% were found in a rural Beninese population and an indigenous, rural community in Western Australia, respectively.[24,25]

A surprising result from this study was the very high prevalence amongst children - at least as high as it was in older age groups. Once acquired, H. pylori infection usually persists in a chronic form. While most infections in developing countries are acquired in childhood the prevalence in children is still usually lower, gradually increasing as age advances.[9,11,23]

Studies in children of Peru[26] and Pakistan[16] revealed a prevalence of 50% and 47% (respectively) - considerably lower than our figure of 78%. However a very high *H. pylori* prevalence, similar to that shown here, was found in school children of Iran[14] and children aged less than 8 years in Bangladesh.[27]

A study of Japanese American men provides evidence that the early acquisition of *H. pylori* infection is associated with an increased incidence of gastric cancer and gastric ulcers later in life.[10] The early acquisition and long duration of *H. pylori* infection may explain the very high incidence of gastric cancer and chronic gastritis in the Sherpas of the Upper Khumbu.

These results mandate a change in practice for the management of dyspepsia in the area. When the prevalence of *H. pylori* in the community is greater than 30% authorities recommend a 'test and treat strategy'.[18,28,29] Instead of treating patients empirically with H2 blockers, patients presenting with dyspeptic symptoms in the Upper Khumbu should be tested for *H. pylori* and if positive treated with triple therapy to eradicate the infection. There is also evidence suggesting that *H. pylori* eradication in high risk populations can reduce the incidence of gastric cancer.[5–7]

A new treatment strategy has the potential to reduce the burden of upper GI disorders including gastric cancer. While it may be more expensive in the short term it may be cost effective in the long term by providing a cure for what are otherwise chronic problems. The Himalayan Trust is looking at funding options. The incidence of upper GI disorders in the Khumbu should be monitored long term to evaluate the effectiveness of this strategy.

Competing interests: Nil.

Author information: Tshering W Sherpa, **job??**, Kunde Hillary Hospital, Soulukhumbu, Nepal; Kami T Sherpa, **job??,** Kunde Hillary Hospital, Soulukhumbu, Nepal; Garry Nixon, Department of General Practice and Rural Health, University of Otago, Dunedin, New Zealand; John Heydon, **job??**, Department of General Practice and Rural Health, University of Otago, Dunedin, New Zealand; Emma Heydon; Department of Public Health and Primary Care, University of Cambridge, Cambridge, England; Susan Dovey, Associate Professor, Department of General Practice and Rural Health, University of Otago, Dunedin, New Zealand

Correspondence: Tshering Wangdi Sherpa, Kunde Hillary Hospital, C/O: The Himalayan Trust, PO Box 224, Kathmandu, Nepal. Email: tshering_kums@hotmail.com

References:

1. Ferlay, J., et al., Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008. Int J Cancer, 2010. 127(12): p. 2893-917.

2. Taylor, D.N. and M.J. Blaser, The epidemiology of Helicobacter pylori infection. Epidemiol Rev, 1991. 13: 42-59.

3. Malfertheiner, P., et al., Current concepts in the management of Helicobacter pylori infection--the Maastricht 2-2000 Consensus Report. Aliment Pharmacol Ther, 2002. 16(2): 167-80.

4. World Gastroenterology Organisation Global Guideline: Helicobacter pylori in Developing Countries. J Clin Gastroenterol, 2011. 45(5): 383-388.

5. An international association between Helicobacter pylori infection and gastric cancer. The EUROGAST Study Group. Lancet, 1993. 341(8857): 1359-62.

6. Wong, B.C., et al., Helicobacter pylori eradication to prevent gastric cancer in a high-risk region of China: a randomized controlled trial. JAMA, 2004. 291(2): 187-94.

7. You, W.C., et al., Randomized double-blind factorial trial of three treatments to reduce the prevalence of precancerous gastric lesions. J Natl Cancer Inst, 2006. 98(14): 974-83.

8. Chiba, N., et al., Treating Helicobacter pylori infection in primary care patients with uninvestigated dyspepsia: the Canadian adult dyspepsia empiric treatment-Helicobacter pylori positive (CADET-Hp) randomised controlled trial. BMJ, 2002. 324(7344): 1012-6.

9. Ford, A.C. and A.T. Axon, Epidemiology of Helicobacter pylori infection and public health implications. Helicobacter, 2010. 15 Suppl 1: 1-6.

10. Blaser, M.J., P.H. Chyou, and A. Nomura, Age at establishment of Helicobacter pylori infection and gastric carcinoma, gastric ulcer, and duodenal ulcer risk. Cancer Res, 1995. 55(3): 562-5.

11. Pounder, R.E. and D. Ng, The prevalence of Helicobacter pylori infection in different countries. Aliment Pharmacol Ther, 1995. 9 Suppl 2: 33-9.

12. Santos, I.S., et al., Prevalence of Helicobacter pylori infection and associated factors among adults in Southern Brazil: a population-based cross-sectional study. BMC Public Health, 2005. 5: 118.

13. Shi, R., et al., Prevalence and risk factors for Helicobacter pylori infection in Chinese populations. Helicobacter, 2008. 13(2): 157-65.

14. Alborzi, A., et al., Prevalence of Helicobacter pylori infection in children (south of Iran). Diagn Microbiol Infect Dis, 2006. 54(4): 259-61.

15. Goodman, K.J., et al., Helicobacter pylori infection in the Colombian Andes: a population-based study of transmission pathways. Am J Epidemiol, 1996. 144(3): 290-9.

16. Jafri, W., et al., Helicobacter pylori infection in children: population-based age-specific prevalence and risk factors in a developing country. Acta Paediatrica, 2010. 99(2): 279-82.

17. Naing, L., T. Winn, and B.N. Rusli, Practical issues in calculating the sample size for prevalence studies. Arch OrofacSci, 2007. 1: 9-14.

18. Elwyn, G., et al., Which test is best for Helicobacter pylori? A cost-effectiveness model using decision analysis. Br J Gen Pract, 2007. 57(538): 401-3.

19. McNulty, C., et al., Test and treat for dyspepsia--but which test? BMJ, 2005. 330(7483): 105-6.

20. Koletzko, S., et al., Isotope-selective non-dispersive infrared spectrometry for detection of Helicobacter pylori infection with 13C-urea breath test. Lancet, 1995. 345(8955): 961-2.

21. Gisbert, J.P. and J.M. Pajares, Diagnosis of Helicobacter pylori infection by stool antigen determination: a systematic review. Am J Gastroenterol, 2001. 96(10): 2829-38.

22. StataCorp, Stata Statistical Software: Release 11, 2009, StataCorp LP: College Station, TX.

23. Kawasaki, M., et al., Seroprevalence of Helicobacter pylori infection in Nepal: low prevalence in an isolated rural village. Eur J Gastroenterol Hepatol, 1998. 10(1): 47-50.

24. Aguemon, B.D., et al., Prevalence and risk-factors for Helicobacter pylori infection in urban and rural Beninese populations. Clin Microbiol Infect, 2005. 11(8): 611-7.

25. Windsor, H.M., et al., Prevalence of Helicobacter pylori in Indigenous Western Australians: comparison between urban and remote rural populations. Med J Aust, 2005. 182(5): 210-3.

26. Begue, R.E., et al., Dietary risk factors associated with the transmission of Helicobacter pylori in Lima, Peru. Am J Trop Med Hyg, 1998. 59(4): 637-40.

27. Mahalanabis, D., et al., Helicobacter pylori infection in the young in Bangladesh: prevalence, socioeconomic and nutritional aspects. Int J Epidemiol, 1996. 25(4): 894-8.

28. New Zealand Guidelines Group., Management of dyspepsia and heartburn. Evidence-based best practice guideline2004, Wellington, N.Z.: New Zealand Guidelines Group.

29. Lassen, A.T., et al., Helicobacter pylori test-and-eradicate versus prompt endoscopy for management of dyspeptic patients: a randomised trial. Lancet, 2000. 356(9228): 455-60.

Physical activity is not play: perceptions of children and parents from deprived areas

Amy D Curtis, Erica A Hinckson, Tineke C A Water

Abstract

Aims To explore the perceptions of primary school aged children (n=9) and parents (n=21) from areas of socioeconomic deprivation in New Zealand in order to determine the factors which influence healthy and overweight children's after school activities.

Method We held focus groups with children, utilising photo-voice prompts for discussion. Focus groups and semi-structured interviews were also conducted with parents. Content analysis of data was undertaken.

Results Both children and parents described physical activity and play as different constructs; physical activity was considered as an organised activity and play was identified as fun. Parents perceived that time, money and transport were all barriers to children participating in physical activities after school. Parents explained that children's enjoyment of a particular activity as well as self-esteem influenced whether or not children participated in physical activity. Community support and communication were also identified as important in creating safer communities and places to play for children.

Conclusion When developing after school community activity programmes, the emphasis should be on active play rather than physical activity.

Overweight and obesity in children is a global concern.[1] Consequences of childhood obesity include developing hypertension, dyslipidaemia, chronic inflammation, increased blood clotting, hyperinsulinaemia, Type II diabetes and glucose intolerance.[2] Obesity can result from low physical activity and/or high levels of sedentary behaviour.[3]

Current recommendations suggest that children should participate in a minimum of 60 minutes moderate-vigorous physical activity and a maximum two hours of recreational screen time activities per day.[4,5]

Children living in areas of socioeconomic deprivation have higher rates of obesity than children in more affluent communities.[1,6]

While there is no evidence to suggest that children from areas of deprivation engage in less daily physical activity than children from higher socioeconomic backgrounds,[7] there is evidence that these children participate in more sedentary behaviours than their more affluent peers.[6]

Interventions generally aim to increase physical activity levels,[8] although these have demonstrated variable success [9]. In order to successfully implement after school activity programmes in areas of socioeconomic deprivation, it is important to

understand the perceptions of children and parents around the barriers to and facilitators of physical activity.

Current research that has focused on children's engagement in physical activity from deprived areas suggests that social disorder and lower neighbourhood safety are strongly linked with decreased physical activity.[10]

Studies have shown that body awareness, which manifests as self-consciousness in overweight children,[8] may be a prime reason for overweight children's low participation levels in physical activity and higher engagement with sedentary activities. These factors have not been explored in detail, especially in deprived areas in New Zealand where little work has been carried out on children's perceptions of their physical and sedentary behaviours.

The purpose of this study was to determine which factors influence children from areas of socioeconomic deprivation to engage in after school activities. Findings will provide a basis for developing future after school physical activity programmes in these areas.

Method

Nine children (age range ~8–12 years old) and 21 parents (age range ~ 31–43 years old) participated in the study. Participants were recruited from a government subsidized school-holiday programme (9 parents and 9 children) and intermediate schools (12 parents) in South Auckland (80% response rate).

South Auckland encompasses the socioeconomically deprived areas of Manukau and has a relatively high proportion of Maori (17%) and Polynesian (25%) families, with just under half of the population identified as New Zealand European and Asian (15%).[11]

Of the 23 eligible schools (intermediate years, state, coed[ucational], and low decile) in the Manukau region, 14 schools were randomly selected. Decile rating for each school was either 1 or 2. Decile rating (1–10) indicates the extent to which the school draws its students from low to high socioeconomic communities. Decile 1 includes the schools with the highest and Decile 10 the lowest proportion of students from low socioeconomic communities.[12]

Once schools and holiday-programme centre were randomly selected, the principal of the school and manager of the centre were emailed information regarding the study. Consent to access the schools and holiday programmes to recruit participants was provided by school principals and holiday-programme centre management.

An information pack and letters inviting children and parents to participate in the study were sent home with children. Informed consent was obtained from parents and assent from children to participate in focus groups. Parents also consented (and children assented) for their children's height and weight to be measured. A purposeful sample of convenience was used; those who agreed to participate were selected. Ethics approval was obtained by AUT University's Ethics Committee. Participant characteristics are presented in Table 1.

Table 1. Participants' characteristics in means ± standard deviation (SD) and percentages

Participant characteristics	Children (n=9)	Parents (n=21)
Age (years)	9.3±1.2	37±5.6
Height (cm)	140.9±9.7	–
Weight (kg)	36.8±11.9	–
BMI	17.8±3.9	–
Gender		
Male	67%	9.5%
Female	33%	90.5%
Ethnicity		
NZ European	52%	
Pacific Island	28%	
Maori	10%	
European Other	7%	
Asian	3%	

Children's height and weight were measured using a portable stadiometer (Design No. 1013522, Surgical and Medical Products, Seven Hills, Australia) and digital scales (Model Seca 770, Seca, Hamburg, Germany) according to the ISAK protocols (International Society for the Advancement of Kinanthropometry).[13]

Body mass index (BMI) was calculated as weight (kg) divided by squared height (m^2) and, using international BMI cutoffs,[14] children were categorised into overweight and healthy weight groups.

Both children's focus groups were held at the holiday-programme centre. Focus groups were mixed gender but children were separated into overweight (four children) and healthy weight groups (five children). Children remained unaware of the focus group segregation. During focus groups, children were asked to discuss their perceived barriers and facilitators to participation in physical and sedentary activities after school.

During focus groups a "photovoice" approach was utilised to ensure data trustworthiness. Prior to each focus group, children were given disposable cameras to photograph places where they were active or played. They were instructed not to photograph people or events that would jeopardise their safety and only the activity and/or the place the activity was held. All children's photographs were printed prior to each focus group.

Children were asked to describe their photographs and explain to the researcher their meaning. We expected the children to be valid and reliable informants as they were over the age of 7.[15]

One parent focus group (seven participants) was held at the schools and the remaining parents were interviewed over the phone. Nine of the parents were associated with the child participants. Parents were asked to comment on their perceived facilitators and barriers of their child's participation in physical and sedentary activities after school and any solutions to the barriers encountered.

Interviews and focus groups were audio recorded and transcribed by the researcher (AC). Children's data were grouped according to their body size. A content analysis of the transcripts was undertaken with common statements coded and categorised. The different views of overweight children, healthy weight children and parents were reported separately. Categories were cross checked for credibility by the other two authors.

Results

Data were analysed in relation to the factors which influence children's after school physical activity behaviours and the solutions to increasing such activity. The categories identified are presented in Figure 1. Participants' quotes are presented in Table 2.

Figure 1.
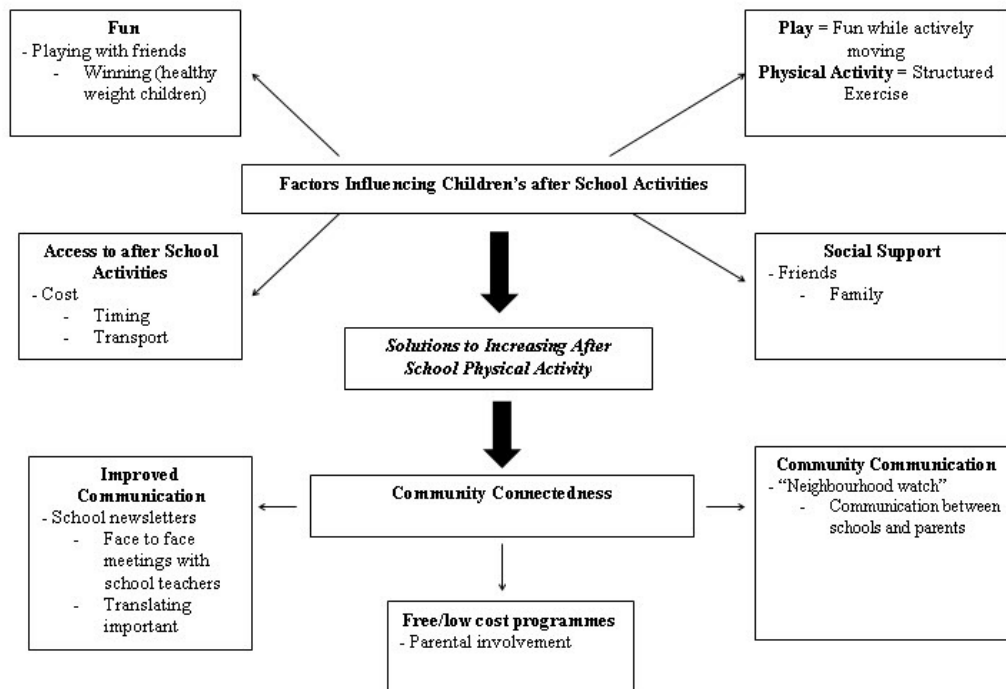
Parents recognised the importance of making physical activity fun for children to ensure their continued engagement. If their child did not feel the activity was fun, or if their child's self-esteem was affected because they were unable to keep up with their peers, that child would be less willing to engage in physical activity.

Many parent comments also illustrated the importance, as they perceived it, of encouraging "the right attitude" rather than "the right aptitude" to facilitate their child's engagement in physical activities.

Most children identified that fun was the main reason for engaging in physical activities. Children emphasised the enjoyment of the adrenaline rush and speed (e.g. bouncing high on a trampoline or going fast on a bike) when moving their bodies. However, for healthy weight children, competing was important and was in fact linked to their self-esteem. These children perceived themselves to be "good at the activities" they participated in and able to succeed in a competitive environment.

In this study participants generally perceived physical activity and play differently. Although participants recognised that both physical activity and play involved being active, physical activity was seen as a structured activity that should be undertaken for a certain time period every day, whereas play was seen as an unstructured activity that involved having fun.

Table 2 Perceptions on children's physical and sedentary activities: parent and children quotes

Categories	Parent	Child
Fun	*"… (their child will participate in) whatever's fun (for them) at the time…"* *"…having the right attitude and letting the kids enjoy what they are doing rather than the competitive side like winning"*	*"… cos most of the time I win."* (healthy weight child) *"When I jump I go really high."* *"Cos it's fun when you go fast"*
Physical Activity vs Play	*"(Physical activity means) …something that you do for a minimum of twenty minutes at a time…."* *"(Play means) probably when they go outside and have a water gun fight, play tag."* *"(Play means) like just letting loose and having fun."*	*"(Physical activity means) um anything that gets your heart rate going."* *"(Play means) …just have a little bit of, just have fun I guess…your friends are involved"*
Access to After School Activities	*"…money is a barrier…"* *"…too much money…."* *"…the major one (barrier) would be working with me…"* *"Cos we are split and in that situation it's really hard…"* *"…I don't want them walking to school and they have to cross a really busy road…"*	*"My mum works most of the time…"* *"…It's just too far away…"*
Social Support	*"…social interaction as well…always been outgoing and like talking to people…"* *"…enjoyable because we (the child's parents) are there…"*	*"Not really cos there's no one to play with."* (overweight child) *"(I'd like to) play with my dad more…cos he doesn't get home till like six thirty."* (overweight child)
Community Communication	*"… communication between the school and parents can help…to raise awareness and encourage them to participate because maybe they just don't know what's going on"*	
Improved Communication	*"…having good communication with your neighbours and knowing each other"*	
Free/low Cost Programmes	*"there are a lot of parents… (who will) take credit for the school and achievements without putting in the time and effort"*	

For children play was not just about 'fun activities' but also involved others, particularly friends. The perception that physical activity is something structured (in order to be beneficial) seemed to distance participants from engagement. There was a strong perception that physical activity was "*good for you*", rather than "*being fun*", and this perception seemed to be a barrier to children becoming involved in physical activity.

All participants commented that cost was a barrier to physical activity. For parents on limited incomes, competing demands (such as food and shelter) were a priority over structured after school activities. The high cost of structured after school physical activities prevented most parents from enrolling their children into these activities. Money constraints, rather than any unwillingness to engage in structured after school activity, was the main barrier.

Free structured after school activities were usually held at times when parents were working. All parents commented on work commitments (whether both parents were working or they were a single parent) impacting on their ability to safely transport children to and from structured after school activities.

Children tended to agree with their parents that work commitments were a major barrier. Many parents felt it was unsafe for their child to walk or cycle to after school activities due to built environment risks, including busy roads and distance to destination. Children likewise recognised safety and distance as barriers to active transport. Parents preferred to drive their children to their structured after school activities, however this was conditional on work commitments. Further discussions revealed that, for these families, there was no way of overcoming this barrier.

Social support influenced children's participation in after school physical activities. Children who reportedly engaged in less physical activity or sport were more likely to engage in sedentary activities, including TV watching, playing play station, "Lego" and "dolls". These children also reported not having family members to play with (either sibling or parent) because they were a single child or their parent(s) worked and it was these children who were more likely to be overweight.

Increased parental or family involvement seemed to facilitate participation in physical activity and parents were aware their involvement was both important and added enjoyment. Parents discussed the physical and social benefits of engagement in physical activities not only for the child, but the whole family.

It was evident from the data that community connectedness was perceived by parents as the factor most likely to increase children's participation in after school physical activity. Many parents commented that improving communication between the school and parents through newsletters, email, "one-on-one" meetings and having important messages translated in languages spoken throughout the community would all increase community connectedness.

Communication was perceived as crucial for creating safer communities and enabling parents to trust others to care for their children. Parents discussed having a "neighbourhood support coordinator" who would email important updates to all parents in the neighbourhood. Neighbours could also communicate important information to households without computers. Parents believed increased communication would help connect families and parents in the neighbourhood, and enable car pooling to structured after school activities and play dates.

Many parents also commented on the importance of communication between school and parents to ensure safer communities. Some parents discussed that receiving newsletters from schools informing them of "stranger danger" was a beneficial strategy to reinforce safety in the neighbourhood.

Parents discussed the importance of free or low-cost programmes to enable more families to participate. Most parents felt that community support and involvement were necessary to keep the cost of the programmes to a minimum. Parents believed that some members of the community would be willing to facilitate the programmes weekly, but they understood that not all parents would be in a position to volunteer.

Discussion

For children and parents physical activity was seen as a 'structured' activity, whilst play was considered as fun. A sense of enjoyment encouraged children's engagement in activities, structured or otherwise, and was enhanced by the presence of friends.

Our results agree with others who found that games and unstructured activities are considered more exciting or "fun" compared to structured activities and a primary motivator to participate. [16]

 Although children are more likely to participate in activities they perceive as "fun", many interventions to date have tended to include structured exercises with variable success. [9] While interventions focusing on active play have proved successful at increasing physical activity, [17] practitioners do not view play as physically demanding or able to provide children with the same benefits of structured exercise.

Nonetheless, children have been found to engage with more moderate to vigorous physical activity from active play during recess than from structured exercise in physical education classes. [17] Therefore, a focus on active play may be just as, if not more, beneficial in increasing children's daily physical activity levels.

Consistent with previous studies, cost, timing of activities and safety were the main barriers preventing children's participation in after-school structured activities. [18, 19] These barriers were compounded by parents' busy work schedules, and therefore ability to transport their children to and from activities, and children not able to transport themselves due to concerns around safety of the neighbourhood. Safe access to spaces to play also impacts on where children play.

Children in socioeconomically deprived neighbourhoods report playing in friends' backyards, whereas children from more affluent neighbourhoods report playing in parks / playgrounds. [20] This can be explained by children in socio-economically deprived areas having to travel two and a half times the distance to parks / playgrounds than those children living in less deprived areas. [20]

Offering supervised school playgrounds for children to play in after school and during weekends may provide a possible solution to concerns around safe access. [21] Providing a supervised playground has successfully increased children's physical activity levels, [21] however the sustainability and feasibility of paying supervisors needs to be considered.

The current study found that supportive neighbourhood environments are required to overcome the barriers of cost and safety. [22] Parents believed that afterschool programmes should be community based and supported by community residents and schools to ensure programme sustainability and a positive community environment.

Good communication was seen as key to achieving this and the use of newsletters, email notifications and meetings with the community and school was seen as a way of

promoting an awareness of safety issues within the community. Consultation with communities has been shown to be effective in promoting safety and trust, essential when developing community programmes. [22] Lack of engagement with the wider community could explain the lower success rate of some children's activity programmes.

Family support was highlighted as a factor that influenced participation in physical activity in this study. Children whose families members who engaged in playing games with them were more physically active and tended to be in the healthy weight range compared to those children who were overweight. Factors such as the importance parents attach to outdoor play, role modelling and parental encouragement are positively correlated with children's participation in outdoor physical activity. [23, 24]

Research around health promotion and intervention in the area of childhood obesity, where physical activity is a component, has shown that a family centred approach is more likely to be effective in the long term. [25] Parents in this study viewed "participation" and "fun" as more important than "competition" and "winning" when encouraging children's engagement in physical activity.

Earlier studies have found that feeling good and having fun were more important to parents and children than winning and medals [26] and furthermore pursuits were more likely to be enjoyable when parents encouraged experimentation with many different activities rather than pressuring their child to compete in one activity. [26, 27]

There are many children's physical activity interventions which focus on one pursuit, for example, soccer, aerobics, walking groups or swimming. [9] These interventions are likely not to have provided participants with the opportunity to explore different activities and discover the physical activity they most enjoy or are at which they are most successful. Therefore, activity programmes that include a wide selection of games, activities and sports are more likely to encourage participation.

While overweight children identified the presence of friends as being a primary motivator to participating in physical activity, for healthy weight children winning is the motivation. Healthy weight children appeared to have higher self-esteem and perceived themselves as being good at their activities.

Conversely, overweight children did not discuss competition or winning. In previous studies, overweight children reported body-consciousness and sweating during exercise, with muscle soreness after physical activities and inability to perform movements as ably as their peers. [8, 28]

Overweight children have often reported feelings of being stigmatized; although they wanted to play, they believed their peers did not want to play with them. [28] These feelings further contribute to low self-esteem levels. As self-esteem and self-efficacy have been positively associated with children's physical activity, [8] it would seem that exclusion, body image issues and functional inability impair overweight children in their desire to engage in physical activities. [28]

Long term interventions need to focus on inclusion and participation, tailored to the child's ability and be mindful of weight discrimination against overweight children.

There are limitations to this study. First, the sample size of each group was small and participants' views may therefore not adequately represent the views of others in the Auckland area. This study does, however, provide a 'snapshot' of perceptions that may be transferred to other groups. Secondly, the majority of parents participating in this study were mothers and therefore results reflect the perceptions of mothers to a greater extent than the perceptions of fathers.

Competing interests: Nil.

Author information: Amy D Curtis, Master of Health Science candidate; Erica A Hinckson, Associate Professor in Physical Activity**;** Tineke C A Water, Senior Lecturer; Centre for Physical Activity and Nutrition, Centre for Child Health, Auckland University of Technology, Auckland

Correspondence: Dr Erica Hinckson, Centre for Physical Activity and Nutrition, Centre for Child Health, Auckland University of Technology, Private Bag 92006, Auckland, New Zealand. Fax: +64 (0)9 9219746; email: erica.hinckson@aut.ac.nz

References:

1. Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. Obes Res. 2004;5(1):4-85.
2. Kumanyika S, Jeffrey R, Morabia A, Ritenbaugh C, Antipatis V. Obesity prevention: the case for action. Int J Obes Relat Metab Disord. 2002;26:425-436.
3. Nagel G, Wabitsch M, Galm C, et al. Determinants of obesity in the Ulm Research on Metabolism, Exercise and Lifestyle in Children (URMEL-ICE). Eur J Pediatr. 2009;168:1259-1267. doi:10.1007/s00431-009-1016-y.
4. American Academy of Pediatrics Committee on Public Education. Children, adolescents, and television. Pediatrics. 2001;107(2):423-425.
5. Ministry of Health. Physical Activity. New Zealand: Ministry of Health; n.d.
6. Ministry of Health. A portrait of health: key results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health,; 2008.
7. Sallis J, Prochaska J, Taylor C. A review of correlates of physical activity of children and adolescents. Med Sci Sports Exerc. 2000; 32(5):963-975.
8. Weintraub D, Tirumalai E, Haydel F, et al. Team sports for overweight children. Arch Pediatr Adolesc Med. 2008;162(3):232-237.
9. Brown T, Summerbell C. Systematic review of school-based interventions that focus on changing dietary intake and physical activity levels to prevent childhood obesity: an update to the obesity guidance produced by the National Institute for Health and Clinical Excellence. Obes Res. 2009;10:1120-1141.
10. Molnar B, Gortmaker S, Bull S, Buka S. Unsafe to play? Neighborhood disorder and lack of safety predict reduced physical activity among urban children and adolescents. Am J Health Promot. 2004;18(5):378-386.
11. Manukau City Council. City Statistics. Auckland: Manukau City Council, 2009.
12. Ministry of Education. Education counts [cited 2012 1st May], Available from: www.educationcounts.govt.nz
13. Norton K, Olds T. Anthropometrica: a textbook of body measurement for sports and health courses. Sydney: University of New South Wales Press Ltd; 1996.
14. Cole T, Flegal K, Nicholls D, Jackson A. Body mass index cut offs to define thinness in children and adolescents: international study. Br Med J. 2007;335(7612):194-197.
15. Faux SA, Walsh M, Deatrick JA. Intensive interviewing with children and adolescents. West J Nurs Res. 1988;10(2):180-94.

16. Fitzgerald E, Bunde-Birouste A, Webster E. Through the eyes of children: engaging primary school-aged children in creating supportive school environments for physical activity and nutrition. Health Promot J Austr. 2009;20(2):127-132.

17. Berntsen S, Mowinckel P, Carlsen K, et al. Obese children playing towards an active lifestyle. Int J Pediatr Obes. 2010;5:64-71.

18. Booth M. What proportion of Australian children are sufficiently active? Med J Aust. 2000;173: 6-7.

19. Hesketh K, Waters E, Green J, Salmon L, Williams J. Healthy eating, activity and obesity prevention: a qualitative study of parent and child perceptions in Australia. Health Promot Int. 2005;20(1):19-26. 10.1093/heapro/dah503

20. Veitch J, Salmon J, Ball K. Children's active free play in local neighborhoods: a behavioral mapping study. Health Educ Res. 2008;23(5):870–879. 10.1093/her/cym074.

21. Farley T, Meriwether R, Baker E, et al. Safe play spaces to promote physical activity in inner-city children: results from a pilot study of an environmental intervention. Am J Public Health. 2007;97(9):1625-1631.

22. Berkwits M, Inui T. Making use of qualitative research techniques. J Gen Intern Med. 1998;13(3):195-199. 10.1046/j.1525-1497.1998.00054.x.

23. Cleland V, Timperio A, Salmon J, et al. Predictors of time spent outdoors among children:5-year longitudinal findings. J Epidemiol Community Health. 2010;64:400-406. 10.1136/jech.2009.087460.

24. Sallis J, Prochaska J, Taylor W, Hill J, Geraci J. Correlates of physical activity in a national sample of girls and boys in Grades 4 through 12. Health Psychol. 1999;18(4):410-415.

25. Luttikhuis OH, Baur L, Jansen H, et al. Interventions for treating obesity in children (Review). United States: John Wiley & Sons, Ltd;2008.

26. McCullagh P, Matzkanin K, Shaw S, Maldonado M. Motivation for participation in physical activity: a comparison of parent-child perceived competencies and participation motives. Pediatric Exercise Science. 1993;5:224-223.

27. Allender S, Cowburn G, Foster C. Understanding participation in sport and physical activity among children and adults: a review of qualitative studies. Health Educ Res. 2006;21(6):826-835.

28. Lee P, Lai H, Chou Y, Chang L, Chang W. Perceptions of exercise in obese school-aged children. J Nurs Res. 2009;17(3):170-178.

Imported malaria in Auckland, New Zealand

Anna E Camburn, R Joan H Ingram, David Holland, Kerry Read, Susan Taylor

Abstract

Aim To describe the current malaria situation in Auckland, New Zealand.

Method We collected data on all cases of malaria diagnosed in Auckland from 1st October 2008 to 30th September 2009. Enhanced surveillance was arranged with all hospital and community haematology laboratories in the region. Laboratories notified us when a diagnosis of malaria was made. After obtaining informed consent the patient was asked about their travel, prophylaxis taken and symptoms. Laboratory results were collected.

Results There were 36 cases of malaria in 34 patients. Consent could not be obtained from two patients so data is from 34 cases in 32 patients. (One patient had *P.falciparum* then later *P.vivax*, the other had *P.vivax* and relapsed.) There were 24 males and 8 females with a median age of 21 years (range 6 months to 75 years). Eleven of the 32 were New Zealand residents. 8 of these 11 had travelled to visit friends or relatives (VFR) while 3 were missionaries. In this group 6 had *P.falciparum*, 4 *P.vivax* and one had both. Twenty-one of the 32 were new arrivals to New Zealand: 11 refugees and 10 migrants.

Conclusion Malaria in Auckland is seen in new arrivals and VFR travellers, not in tourist travellers.

Rates of malaria have fallen in a number of endemic countries in recent years[1] and a corresponding drop in the number of imported cases has been seen in several high income countries.[2,3] Rates of malaria in travellers to India,[4] Asia[5] and Africa[6] have fallen.

Our aim was to describe the current malaria situation in Auckland, New Zealand.

Methods

We conducted a prospective observational study of all cases of malaria diagnosed in Auckland over a 12 month period (1 October 2008–30 September 2009). Ethics approval was obtained from the Northern Y Ethics Committee.

Enhanced surveillance was set up with all hospital and community haematology laboratories in the region, with the laboratories requested to notify the principal investigators or the treating infectious diseases physician whenever a case of malaria was diagnosed.

Following written informed consent, each study participant was interviewed. A closed ended questionnaire was used to record information which described their resident status in New Zealand, their travel history, whether any prophylaxis was taken and clinical symptoms present at the time of diagnosis.

Laboratory results from blood tests collected at the time the diagnosis of malaria was made were also reviewed. No patient was asked to have further blood tests or additional investigations.

Results

During the 12-month period of the study 36 cases of malaria were reported to us by the haematology laboratory services in Auckland. Consent could not be obtained from two of the cases, so data collected are from 34 episodes of malaria diagnosed in 32 patients. One patient was initially diagnosed with *P.falciparum*, and presented 5 months later with *P.vivax* – presumably contracted during the same trip to Papua New Guinea. Another patient was diagnosed with *P.vivax*, but did not complete eradication treatment with primaquine and presented with relapsed infection.

Among the 34 episodes of malaria *P.falciparum* was diagnosed in 18 and *P.vivax* in 16 patients. Of the 32 patients 24 were male and 8 were female. The median age at the time of diagnosis was 21 years (range 6 months to 75 years).

Malaria in New Zealand residents—Only 11 of the 32 patients (29%) were New Zealand residents. Three of these were New Zealand born, and acquired their infection whilst travelling to malarious areas as missionaries. The remaining 8 patients were all "migrants", but living permanently in Auckland and acquired malarial infection whilst returning to their country of origin to visit friends and relatives.

The countries of acquisition were Sudan, Kenya/Uganda, Nigeria, India, Thailand and Papua New Guinea. The median age of the New Zealand residents with malaria was 32 (14–64) years. One patient had *P.falciparum* followed 5 months later by *P.vivax*. Seven cases were *P.falciparum* and 5 were *P.vivax*.

Those with *P.falciparum* developed symptoms a median of 3 days after returning to New Zealand (0 to 26 days) although one had symptoms before returning home. Those with *P.vivax* developed symptoms a median of 43 days (10-274) after their return.

Table 1. Episodes of malaria diagnosed in Auckland by traveller type, country visited and species

Country	*P.vivax* NZers	New Entrants	*P.falciparum* NZers	New Entrants	Refugees	Total
Papua New Guinea	2		2			4
Thailand	1					1
India	2	10	1			13
Pakistan		1				1
Uganda					11	11
Uganda/Kenya			1			1
Sudan			1			1
Nigeria			2			2
	5	11	7		11	34

Table 2. Rates of imported malaria in travellers from Auckland

Number with malaria	Number of short-term travellers*	Rate/1,000,000/mth residence in malarious areas†	VFR with malaria	Number of VFRs	Rate/1,000,000/mth for VFRs	Number with malaria
India	3	9728	62	3	5939	101
Nigeria	2	99	4,040	2	25	16,000
PNG	3	950	630	1	68	2,941
Sudan	1	17	11,764	1	17	11,764
Thailand	1	9830	20	1	1441	138

Notes:

Numbers of travellers obtained from departure records for 12 month period from October 2008 (Short-term NZ traveller departure totals , Statistics New Zealand)

* Assumes one-third of New Zealand travellers are from Auckland

† Assumes average length of stay in malarious areas is two weeks

Data for one patient not included as they had visited Kenya and Uganda.

Table 2 shows the cases of malaria among travellers from Auckland who visited one country, short term (<12 month) New Zealand departures to those destinations and VFR departures to those destinations in the year to September 2009. Rates of malaria are very high for travellers to Nigeria and Sudan, high for Papua New Guinea and low for India and Thailand. Rates in VFRs to those countries are even higher.

Chemoprophylaxis was taken by 6 of the New Zealand residents, with all reporting only partial (50% on average) compliance. None of them continued the prophylaxis after their return to New Zealand. Four had taken doxycycline and 2 could not recall what they had taken.

All developed symptoms of fever, with 8/12 also reporting shaking chills/rigors and 7/12 reported gastro-intestinal symptoms (abdominal pain, nausea or vomiting). Headache was reported by 6/12, and myalgia orarthralgia by 5.

In 11 of the cases the diagnosis was made by examination of a peripheral blood smear preparation, and 1 case (*P.falciparum*) was diagnosed by an immunochromatographic (rapid antigen) test.

All were treated with appropriate therapy; the 7 cases of *P.falciparum* were treated with quinine/doxycyline or quinine/clindamycin and of the 5 cases of *P.vivax* 3 were treated with chloroquine/primaquine and 2 with mefloquine/primaquine. All patients in this group were admitted to hospital except 2 with *P.vivax*. No patient required intensive care but the one patient who had *P.falciparum* followed by *P.vivax* had marked anemia on both occasions and required transfusion. She had used artemisin-based combination therapy self treatment two weeks before presenting with *P.falciparum.*

Malaria in new entrants—Another 11 cases of malaria were diagnosed in 10 patients who all arrived in New Zealand as "new entrants", i.e., they were new arrivals who had come to New Zealand to study (8 of the patients) or as part of a family migrating to New Zealand permanently (2). In this group, 9 were migrants from India, 1 from Pakistan, and all were diagnosed with *P.vivax*. None had taken antimalarial prophylaxis. The median age of this group was 21 years (3–75). They presented a median of 138 days (1–327) after arriving in New Zealand. All reported

fevers. Shaking chills were reported in 10, headache in 6, gastro-intestinal symptoms in 5 and myalgia in 2.

In all 11 cases the diagnosis was made by peripheral blood smear examination. All received treatment with chloroquine, but only 9 cases reportedly received eradication therapy with primaquine. As mentioned previously, 1 patient who did not take the primaquine prescribed because he couldn't afford it represented 2 months later. Seven of these 11 patients were admitted to hospital for a mean of 1.6 days (1-3).

Malaria in refugees—The third subgroup comprised 11 refugees, who were all diagnosed through the Auckland Refugee Centre. Four of the 11 reported their country of origin to be Congo, but all 11 had arrived from Uganda. Only 1 of these patients was symptomatic (reporting long standing abdominal discomfort), and 2 were screened for malaria due to the clinical finding of splenomegaly. The remaining 8 cases were all screened for malaria on the basis of close relationship with an index case. The median age of this group was 12 years (6 months -34 years).

All patients in this group were diagnosed with *P.falciparum*, and again the diagnosis was made by peripheral blood smear examination. Three received quinine and doxycycline, 2 received quinine and clindamycin and 6 were prescribed 2 doses of mefloquine. Two refugees were admitted to hospital and 2 were seen but not admitted for the initiation of their treatment. The remainder received all their treatment at the Auckland Refugee Centre.

Discussion

Although rates of imported malaria have decreased recently in a number of countries, it remains an important diagnosis with an associated morbidity and potential mortality. During the study period a total of 36 cases of malaria were diagnosed in Auckland. With an estimated 1.4 million residents, this equates to 2.5 cases of imported malaria per 100,000 people per annum. This is higher than the national rate of 0.9 per 100,000 in 2008 and 1.2 in 2009.[7] This higher rate is partly explained by the cases seen in quota refugees as they are screened in Auckland prior to settling around the country.

In a similar study by our unit in 1993 43 cases were diagnosed in Auckland.[8] Other New Zealand rates prior to the introduction of laboratory based reporting in 2008 are unreliable as cases are underreported by clinicians.[9]

Despite New Zealanders' passion for travel, and thousands of Kiwis visiting malarious areas each year (for example in the twelve months to September 2009 11,599 New Zealanders gave Indonesia as their main destination, 17,299 Malaysia, 9,737 Philippines, 8,323 Vietnam and 11,721 Vanuatu) [10], not a single case was diagnosed in a New Zealand tourist traveller.

Nor were any of our 1130 defence personnel deployed overseas over the study time period [11] diagnosed with malaria after returning to Auckland. Their deployments included Malaysia, Timor, Pakistan and the Solomon Islands.

Of concern is the number of cases of malaria diagnosed in New Zealand residents with family ties in malarious areas who travelled to visit friends and relatives. Elevated malaria risk in this group is well described.[12] The reasons are multiple and

include longer duration of travel, more rural travel and less use of preventive strategies because of lack of awareness , financial constraints and provider factors.[13] This is a group to which further education and appropriate prophylaxis strategies should be targeted.

New Zealand is a popular destination for foreign students, with large numbers from South East Asia and India. Accordingly it can be expected that malaria will continue to be diagnosed in this patient group, and GPs and physicians alike should ensure they take a thorough travel history and request a malaria screen when seeing a "New Entrant" patient presenting with fever.

Malaria continues to be diagnosed in our refugee population. Of note 8of the 11 patients diagnosed in this group were completely asymptomatic. This implies that all refugees from a malarious area should be screened as part of their immigration "work up". It is recommended that all refugees are screened for malaria on arrival in Australia.[14]

At the time of the study quinine was standard first line therapy for *P.falciparum* malaria in our region however now artemesinin based treatment is often used.

Malaria prevention advice for many travellers continues to be important and should include the need to avoid mosquito bites from sunset to sunrise. Use of clothing, repellents and permethrin impregnated bed nets should be emphasised.

Malaria risk remains high and widespread in many of the malarious parts of Africa, Papua New Guinea and Solomon Islands. Thus most travellers to these regions should use prophylaxis with mefloquine, doxycycline or malarone.

For many other destinations malaria rates vary from high to low and the need for prophylaxis thus needs to be individualised depending on the regions a traveller is visiting and their planned activities. This makes knowledge of risk areas vital when giving pretravel advice. (Useful maps can be seen on sites such as www.fitfortravel.nhs.uk). For example while parts of Cambodia have high rates of malaria the risk to a traveller only visiting Phnom Phen, Siem Reap and Angkor Wat is low and prophylaxis unnecessary. Similarly many travellers visiting only the high altitude areas of Bolivia and Peru do not need malaria prophylaxis.

Author information: Anna Elinder Camburn, Haematology Registrar; R Joan H Ingram, Infectious Diseases Physician, Auckland City Hospital; David Holland, Infectious Diseases Physician, Middlemore Hospital; Kerry Read, Infectious Diseases Physician, North Shore Hospital; Susan Taylor, Microbiologist, Middlemore Hospital, Auckland

Correspondence: R Joan H Ingram, Infectious Diseases Physician, Auckland City Hospital, Park Rd, Auckland, New Zealand. Fax: +64 0(9) 3074940; email: joani@adhb.govt.nz;

References:

1. World Malaria Report 2008, WHO
2. Imported malaria cases and deaths, United Kingdom: 1990 – 2009 http://www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1195733773780

3. Arboviral diseases and malaria in Australia, 2007/08: Annual report of the National Arbovirus and Malaria Advisory Committee
http://www.health.gov.au/internet/main/publishing.nsf/content/cda-cdi3302b.htm

4. Schmid S, Chiodini P, Legros F et al. The risk of Malaria in Travelers to India. J Travel Med 2009;16:194-9

5. Behrens R, Carroll B, Hellgren U et al. The incidence of malaria in travelers to South-East Asia: is local malaria transmission a useful indicator? Malar J 2010; 9:266

6. Behrens R H, Carroll B, Smith V, Alexander N. Declining incidence of malaria imported into the UK from West Africa. Malar J. 2008:7:235.

7. Notifiable and Other Diseases in NZ: 2009 Annual Surveillance Report Population and Environmental Health Group Institute of Environmental Science and Research Limited April 2010

8. 8.Shew R , Wong C, Thomas MG, Ellis-Pegler RB Imported malaria in Auckland in 1993 NZMJ 1995;108:380-2

9. Kriechbaum AJ, Baker MG The epidemiology of imported malaria in New Zealand NZMJ 1996;109:405-7

10. http://www.stats.govt.nz/infoshare/SelectVariables.aspx?pxID=67dcde70-71e9-4fab-b4a6-239c313db11f

11. Personal communication 2011David Redfern, New Zealand Defense Force

12. Leder K, Tong S, Weld l et al Illness in Travelers Visiting Friends and Relatives: A review of the GeoSentinel Surveillance Network CID 2006;43:1185-93

13. Chiodini J The standard of malaria prevention advice in UK primary care Travel Med and Infect Disease 2009;7:165-68

14. Murray R, Davis J, Burgner D The Australasian Society for Infectious Diseases guidelines for the diagnosis, management and prevention of infections in recently arrived refugees Med J Aust 2009;190:421-425

The 2009 influenza pandemic: a review of the strengths and weaknesses of the health sector response in New Zealand

Nick Wilson, Jennifer A Summers, Michael G Baker

Abstract

Introduction To inform future pandemic planning and disaster response, we aimed to review the literature on the health sector response to the influenza A (H1N1) 2009 pandemic in New Zealand in 2009.

Methods We searched PubMed and Google Scholar along with the websites of government agencies for the period 1 April 2009 to 20 May 2012.

Results In 2009, 18% of the New Zealand population had evidence of infection from the pandemic strain, 1122 people were hospitalised (with pandemic influenza as the primary diagnosis), 102 of those hospitalised were treated in intensive care units (ICU), and there were an estimated 49 pandemic-attributed deaths. At the severe end of the disease spectrum (ICU admissions and mortality), the health burden was significantly worse for Māori and Pacific peoples.

The available evidence (including various estimates of low case-fatality risk relative to other high income countries), is consistent with the overall response in the public health, primary care and hospital sectors being fairly successful. Nevertheless, a number of likely weaknesses were identified, including a relative lack of: (i) a detailed review of the epidemiology and health sector response; (ii) sophisticated analytic studies to identify risk factors (e.g., using case-control studies); (iii) studies on pandemic vaccine uptake and public acceptability; and (iv) evaluation of the health protection messages that were used in campaigns and in media releases from health authorities.

Conclusions There appear to have been both strengths and weakness in the New Zealand health sector's response to the 2009 influenza pandemic. Nevertheless, it is probably still worthwhile to address some of the omissions to inform future pandemic and natural disaster planning and preparations.

It is important to consider the lessons from the influenza A (H1N1) 2009 pandemic for New Zealand (NZ), given the likely occurrence of future such pandemics. These lessons may inform how this country could further upgrade its national and local pandemic plans and also develop its surveillance system for infectious diseases in general (for which there is significant scope for improvement[1]).

Such lessons may also be relevant to other pandemic diseases such as a re-emergence of a more infectious form of SARS or even disease associated with bioweapons, albeit probably a very low risk for NZ.[2] Finally this pandemic, although involving a virus strain of relatively low virulence,[3] can be considered a type of "natural disaster". As such it may provide lessons for dealing with other natural disasters and civil defence emergencies.

Some of these events are likely to become more prevalent with global climate change (e.g., severe storms and flooding events).

Three years following a new influenza pandemic is a good point to reflect on the health sector response at a country-level. This is a time when relevant local research has often been published. Some review work to date has included an official document that had reported and summarised selected aspects of the health burden of the 2009 pandemic for New Zealanders.[4] But the scope of this work was largely limited to the mortality burden. Two other reviews have considered studies from the Southern Hemisphere, but these dealt with fairly selected aspects of the NZ data.[5,6]

We therefore aimed to review the literature on the 2009 pandemic relating to NZ so as to: summarise its impact; to make comparisons with the 1918 pandemic; and to detail the strengths and weaknesses of the health sector responses (including public health, primary care and hospitals).

Methods

We searched PubMed and Google Scholar along with the websites of government agencies (Ministry of Health and ESR – a Crown Research Institute). The search period was from 1 April 2009 to 20 May 2012, and the search terms included "influenza" and "2009" and "Zealand". We identified a total of 54 relevant PubMed-indexed articles and letters. We purposefully excluded from the above total the following: case reports (n=2); international studies/reviews that contained NZ data, but with this not being new data (n=6); research related to subsequent waves in 2010 (n=4); and research on certain highly specialised intensive care unit (ICU) issues (n=4).

We organised the findings by first summarising the health impact of the pandemic and to provide context, followed by a structured comparison of key parameters in 2009 along-side those relating to the 1918 pandemic. For organising the published literature we used the following headings: national epidemiology, local epidemiology, surveillance systems, key epidemiological parameters, risk factors, screening and self-diagnosis and response to influenza-like illness, behavioural responses and risk communication, laboratory diagnostics and virological studies, health services response and clinical management, and immunisation. From our interpretation of literature and the apparent gaps, we then attempted to extract the possible strengths and weaknesses of the health sector response.

Results

Summary of the health impact—New Zealand was one of the first countries to experience the 2009 pandemic,[7] which was characterised by a short and abrupt "epidemic curve" with evidence of moderate infectivity.[8] Relative to past influenza pandemics to reach NZ,[9,10] this one involved a pandemic strain of relatively low virulence and case-fatality risk. It was much less severe than what had been anticipated and planned for in pandemic preparations which were relatively advanced in the NZ setting.[11] The key epidemiological features of the 2009 pandemic are summarised in Table 1 and compared to the much more severe 1918 pandemic (which was also the last H1N1 pandemic to affect NZ, the 1957 pandemic being H2N2, and 1968 being H3N2). Most striking was the much lower cumulative proportion dying in the 2009 pandemic.

For the 2009 pandemic, a national serosurvey estimated that 18.3% of New Zealanders were infected with the pandemic virus during the first wave (when adjusting for baseline immunity from testing of stored sera from before the pandemic and with age and ethnicity standardisation to the NZ population) (Table 1).[12]

Table 1. Key epidemiological parameters and features of the 2009 influenza pandemic in New Zealand (Wave 1) and comparison with those for the 1918 pandemic

Key parameter/ feature	2009 pandemic – wave 1 (95%CI)	1918 pandemic	Comment and sources
Reproduction number	1.25 (1.07–1.47)	1.3–3.1 in a military setting; 1.2–1.8 in community settings	For the 2009 pandemic: based on work by Roberts and Nishiura,[15] (see Table 2 for other studies). For the 1918 pandemic: estimates in military[16] and community settings.[17] The reproduction number estimates for NZ in 1918 fit within worldwide estimates of between 1.1–5.4.[18-21]
Generation time (days)	2.38 (mean result)	Estimated between 2–4 days	For the 2009 pandemic there was variation in the modelled results: "generation time is biased downwards during the beginning of the epidemic".[15] For the 1918 pandemic,[17] this was based on previous generation time modelling.[22 23]
Cumulative infection	18.3% (serological evidence)	Unknown for NZ	For the 2009 pandemic.[12] For the 1918 pandemic: worldwide estimates suggest between 25-50% cumulative infection.[24 25]
Cumulative proportion with symptomatic illness	12.2%	Estimated: 33%–50%	The estimate for the 2009 pandemic was for the total population (with another 6.1% having asymptomatic illness).[8] For the 1918 pandemic this was for the total NZ population.[9]
Cumulative proportion hospitalised	26 per 100,000	Unknown for NZ	For the 2009 pandemic[8] and with 10.6% of these hospitalised cases being admitted to ICUs.[4]
Cumulative proportion dying	1.38 per 100,000	550 per 100,000 (European) 4230 per 100,000 (Māori)	For the 2009 pandemic.[4] For the 1918 pandemic,[9] but excluding deaths of New Zealanders overseas. The case-fatality risk was: around 0.005% of infected cases,[8] 0.01% of symptomatic cases,[8] and 0.5% (16/3179) of laboratory-confirmed cases.[27]
Ethnic gradient for hospitalisation and mortality	Elevated for Māori and Pacific peoples	7.3 (times higher burden for Māori[9])	Māori and Pacific peoples had higher rates of ICU admissions than the NZ European population[4] and similarly for both populations for age-standardised mortality (e.g., for Māori vs Others (non-Māori and non-Pacific, mainly European) the rate ratio was 2.6, (95%CI: 1.3–5.3).[14]
Socioeconomic gradient (mortality)	Suggestive evidence	Suggestive evidence	For the 2009 pandemic there appeared to be a deprivation gradient for mortality (39% of the cases in the 2 most deprived deciles) – but this was not adjusted for age, sex or ethnicity.[4] For the 1918 pandemic in Auckland, the mortality rate in "poor" suburbs was higher than "well-to-do" suburbs (RR=1.42; 95%CI: 1.10–1.82, p=0.004**) but there was no age, sex or ethnicity adjustment.

Notes: * Insufficient data were generally available for similar comparisons with the other influenza pandemics of the 20th century that reached New Zealand (though some ethnicity comparisons for the 1918 pandemic have been performed[14]).

** Risk ratio calculated from tabulated data in a thesis (p169).[26] Other work by Rice[9] indicates some variation in mortality rates by occupational group (Table 10.2, p226), but it is difficult to interpret these results e.g., there was no age/sex-standardisation and some denominators may have been influenced by differential involvement by occupation in the overseas war effort (e.g., many of the country's health professionals were overseas). The socioeconomic analysis by Rice (Table 10.4, p233) was also constrained (e.g., no rate calculations).

Children and adolescents (aged 5–19 years) had the highest total seroprevalence (46.7% – not adjusted for pre-pandemic immunity). Prevalence was also relatively high for Pacific peoples (49.5%) and for Māori (36.3%), compared to the "Other" (mainly European) ethnic group (25.9%).

For the year 2009 there were an estimated 1508 hospitalisations for influenza, a four-fold increase on the number in the preceding year.[4] Most of these people (n=1122) were admitted to hospital with a primary diagnosis of "pandemic influenza A (H1N1) 2009". Of these admissions, 102 occurred to ICUs. These ICU admissions were significantly higher for both Māori and Pacific peoples compared to European New Zealanders.[4] Other risk factors identified for ICU admission included: pregnancy, body mass index >35, and having pre-existing asthma or another chronic pulmonary disease.[13]

An official Mortality Review Group reported that there were 49 deaths due to H1N1 infection in 2009.[4] Significantly elevated age-standardised mortality rates for both Māori and Pacific peoples were apparent.[14] There was also some evidence of a socioeconomic gradient with 39% of those dying having an area deprivation score of either 9 or 10 (the most deprived two deciles), compared with the expected 20% of the population. Of those dying, 86% had at least one comorbid or associated condition e.g., obesity (74%), morbid obesity (56%) and respiratory disease (49%).

In addition to the above summarised data, many additional aspects of the pandemic and the health sector response have been considered in other studies (Table 2).

Table 2. Published work on to the 2009 pandemic in New Zealand (to 20 May 2012)*

Topic area	Aspects covered
National epidemiology	A detailed initial description of the epidemiology in NZ was published in August 2009[27] (with this information subsequently compared to that of other Southern Hemisphere countries[7]).
Local epidemiology	These studies included one for South Auckland using a capture-recapture method,[28] and studies of hospitalisations at Hutt Hospital[29] and for the Wellington region.[30]
Surveillance systems	The surveillance systems used in NZ for the pandemic were described in 2009[27][31] along with the potential for more innovative approaches (e.g., "Google flu trends"[32]) and summarised as part of a wider surveillance review.[1]
Key epidemiological parameters	The reproduction number of the new pandemic was calculated in several analyses[33][15][34-36] with additional commentaries.[37][38] The NZ data has also been used to suggest the utility of a forecasting system that could be used in real time at the early stages of a pandemic.[39] Specific data on transmission risk came from an airline setting[40] and a tour group study.[41] Other epidemiological studies (see above) provided further information e.g., on estimating the community burden and the case-fatality risk. An international study included a focus on age distribution data from NZ.[42]
Risk factors	Several of the epidemiological and ICU studies detailed elsewhere in this Table included risk factor data (i.e., relating to age, ethnicity, obesity and chronic conditions). In addition, two other studies considered pregnancy as a risk factor (including the increased risk for Māori and Pacific women who were pregnant).[43][44]
Screening and self-diagnosis and response to influenza-like illness	A study of screening at the border (Auckland airport), suggested a very low sensitivity of this intervention at 5.8% (95%CI: 2.3%–14.0%).[45] However the screening system used was a relatively passive one that relied on self-identification of symptoms by the in-coming travellers. Another study investigated self-diagnosis behaviour and found that those who thought they had been infected with H1N1 in 2009 were no more likely to be seropositive than those who did not.[46]

Topic area	Aspects covered
Behavioural responses and risk communication	A study in mid-2010 involved eight focus groups (including Māori, Pacific peoples, parents of children, and vulnerable people with chronic conditions).[47] This work identified a range of issues around risk perception, risk communication, response to pandemic-specific health messages, and community-level approaches. One conclusion was "the problem with a 'one size fits all' pandemic warning strategy that risks antagonising and distancing communities and thereby reducing trust in agencies and the likelihood that advice will be followed." Hand hygiene behaviours of the public were studied in a hospital entrance setting,[48-50] along with respiratory hygiene behaviours in a range of public places (with low use of the recommended behaviours).[51]
Laboratory diagnostics and virological studies	Diagnostic methods were studied[52-54] along with studies around the sensitivity of the pandemic strain to oseltamivir.[55 56]
Health services response and clinical management	Health professional bodies provided guidelines,[57] opinion leaders provided advice,[58 59] and a review on pandemic-related psychological issues was published.[60] A study described how "a range of Canterbury agencies worked together in a coordinated health-led response" and how the response meant that "health care services were not overwhelmed".[61] The key lesson was "the importance of preparing and working together across the sector". Other work reported on the response of nurses in a community mental health residential facility[62] and how a hospital managed diagnosis and care of healthcare workers at risk of H1N1 infection.[63] The experience for ICUs and data on admissions was particularly well studied, often in combination with Australian ICU data.[13 64-67] Even the paediatric ICU[68] was covered. Some work focused on the use of extracorporeal membrane oxygenation in treatment.[69-71]
Immunisation	The effectiveness of the 2009 seasonal influenza vaccine against the pandemic strain was studied.[72] Adverse events following use of the trivalent influenza vaccine in children have also been investigated.[73]

Discussion

Probable strengths of the health sector response

The NZ health sector appeared to invest considerable effort on initial containment measures with case identification, isolation, contact tracing and the provision of antivirals. There is no strong evidence that such measures may have slowed the initial spread. Nevertheless, one argument was that "the considerable interval without reported cases during May (before the epidemic accelerated in June) provides some suggestive evidence for the success of the containment measures".[27] However, it is possible that this apparent lack of cases simply reflected the difficulty detecting spread of the pandemic virus during the early establishment stages, as was observed in parts of Australia.[74]

Further work could compare the time scales for NZ with other OECD countries and in the context of the relatively low reproduction number of this pandemic strain.[15] Nevertheless, the vigorous public health response was probably justified on precautionary grounds given that little was known about the new pandemic strain at the time. In addition, the relatively intensive response provided valuable experience in demonstrating that such a response is highly demanding in terms of human resources and requirements for antivirals.

Health agencies such as the Ministry of Health appeared to be extremely active in providing information to the public, sometimes with multiple media releases per day and having detailed website information. Regional health agencies were also active with their own campaigns and website information.[61]

The impact of the public health messages and information is not clear, but these could have facilitated the generally helpful response (from a public health perspective) of ongoing media coverage of the pandemic. There was also some evidence that New Zealanders responded to infection control messages with primary care consultations being reportedly down for other illnesses during the pandemic in Canterbury.[61] There was also some limited evidence that hand hygiene practices improved during the pandemic period,[50] even though observed respiratory hygiene was fairly poor during the pandemic.[51]

The Mortality Review Group noted the "considerable logistical challenge for laboratories and primary and secondary health services."[4] Yet despite this limitation, there are various indications that the health sector performed fairly well, at least relatively to other countries:

- Firstly, the overall extremely low case-fatality risk (CFR) at 0.005% for all infected cases in NZ,[8] was less than the 0.01% estimated in a systematic review for high-income countries.[75]

- Secondly, the CFR for symptomatic cases in NZ was lowest for both groups of 0-17 year olds and 18-64 year olds) or second lowest (0-17 year olds) in a six study comparison (including Denmark, Netherlands, UK and USA [n=2 studies]; albeit with some overlapping confidence intervals).[76]

- Thirdly, the CFR for laboratory-confirmed cases was only 0.5% (Table 1), which compares to that of 1.1% (95% CI: 0.0-3.0%) from a systematic review (using 33 reports from high-income countries).[75] This NZ result was also second lowest in a review of studies in Southern Hemisphere countries.[5]

- Fourthly, the CFR for those admitted to hospital and to ICUs was relatively low and also (for ICU admissions), the same as in Australia i.e., 16% (16/101) compared to 16% (105/643) for Australia (Personal communication, Lisa Higgins, Australian and New Zealand Intensive Care [ANZIC] Research Centre, Australia). These proportions were both lower than those for California at 25%.[77]

There were also signs of effective management in the NZ setting whereby hospital work loads were re-organised to ensure capacity was maintained (e.g., postponing elective surgery). Indeed, this was seen with Australasian-wide reductions in admissions to ICUs associated with elective surgery during 2009.[66] Clinicians also actively explored intensive treatment options e.g. in the use of extracorporeal membrane oxygenation (Table 2).

The health sector also appeared to work successfully to ensure that appropriate vaccines were made available prior to winter 2010. Initially a monovalent vaccine to provide protection against pandemic A (H1N1) 2009 was supplied, though this had low uptake,[78] and shortly became redundant with the availability of trivalent vaccines.[79] The latter provided additional protection against seasonal influenza A (H3N2) and influenza B in addition to pandemic A (H1N1) 2009.

These vaccines appear to have reasonable public acceptability and their introduction attracted little adverse media publicity in NZ. This situation is noteworthy for several reasons. Firstly, the pandemic strain had relatively low virulence. Secondly, the

vaccination programme had some remarkable features, including administration to pregnant women and children (with targeting based on ethnicity and residence in deprived areas). Use of one of these trivalent vaccines (Fluvax) was also associated with highly publicised adverse events in Australia (an increase in reported febrile convulsions in Western Australia).[80]

Subsequent research in NZ indicated a significantly higher frequency of fever following administration of Fluvax(® CSL Biotherapies) compared with Vaxigrip(® Sanofi Pasteur).[73]

Probable weaknesses in the responses

No overall review of the response—As of June 2012, there had been no detailed review of the epidemiology and health sector response to the 2009 pandemic in NZ. Any internal documents on the Ministry of Health's review of its pandemic performance have not been made available on its website and the national response has not been considered in the light of review work by WHO on the international response.[3]

All this information would collectively assist with pandemic plan revisions and re-assessing the value of previous pandemic planning exercises (e.g., "Exercise Cruickshank"[81]). Ideally this work might also have been supplemented with more extensive research on risk factors and containment measures (see below), and relating to populations suffering the highest burden such as Māori and Pacific peoples (i.e., expanding on the one qualitative study[47]). There could also have been a review by the relevant parts of the Parliament such as the Health Select Committee and/or the Māori Affairs Select Committee.

Some examples of additional issues (not discussed further below) for which some information should ideally have been reported nationally, include: the impacts on cancelling of elective surgery, the impacts on pharmaceutical use (antivirals and antimicrobials), and the impacts on laboratory and hospital staff workloads.

Limited analysis of the effectiveness of border control and containment measures—Probably the most distinctive element of NZ's pandemic plan is a major focus on border control and containment ("*Keep it out, Stamp it out*"). These measures consumed a great deal of resources during the planning phase and the pandemic itself (up until the switch to the mitigation phase on 22 June 2009, eight weeks after H1N1 was first detected on 25 April). However, there has been just one study on border control issues,[45] and the impact of containment was limited to the study of a tour group.[41] Yet NZ was relatively well positioned for more detailed studies given the extensive PCR testing of potential cases in the early stages of pandemic spread. This applied research could potentially have informed the use of in-the-field isolation measures and anti-viral prophylaxis.

No sophisticated analytic studies on risk factors for poor outcomes—Such work in the form of a case-control study of hospitalised cases could have substantially improved on the risk factor data from descriptive studies. Such work was conducted elsewhere for this pandemic e.g., in Canada[82 83] and Australia.[84] Furthermore, national research funding agencies appeared to be relatively slow in making funds available for pandemic-related research (though the Health Research Council of New Zealand

did make useful funding available in a special funding round late in 2009, the *Influenza A (H1N1) Rapid Response Research Initiative*).[85]

Lack of studies on pandemic vaccine uptake and acceptability—We could only identify one qualitative study that touched on vaccination in the pandemic context.[47] Yet information on public (and health professional) vaccine uptake and acceptability could help inform future decision-making around the provision of vaccination against pandemic influenza. One barrier to research on influenza vaccine in NZ is that administering this vaccine is not currently recorded on the National Immunisation Register. This appears to be an important information system deficit (albeit one that is in the process of being amended as of early 2012).

No evaluation of the public health messages—The hygiene and other messages used in mass media campaigns by the health sector were not formally evaluated, and information is limited to the modest amount of data from one qualitative study.[47] While some behavioural data around hygiene practices were collected,[48-51] this work was not specifically designed for campaign evaluation. Yet to appropriately inform future health sector investment in such messages and campaigns, evaluation of effectiveness and cost-effectiveness seems critical. Similarly, there was no research on how the media responded and yet such work can potentially inform how the health sector engages the media as was done for SARS in NZ.[86]

No economic impact assessment—Studying the economic impacts on the health sector, education sector and economy (tourism impacts, absenteeism from school and work) would help inform future decision-making around pandemic control. The cost of unused monovalent pandemic vaccine and expired stockpiled antivirals could also be documented. Some work identified the relatively high ICU costs for Australasia, but this work did not separate out NZ data.[87] NZ work, outside of the review period of this study, has also started to consider hospital costs associated with the pandemic.[88]

Conclusions

The available evidence (including the low case-fatality risk relative to other countries), is consistent with a successful overall response to the 2009 influenza pandemic by the public health, primary care and hospital sectors. Nevertheless, we suggest there were a range of "weaknesses", albeit mainly omissions in the post-pandemic period. It could be argued that some of these "weaknesses" might be justified in terms of limited resources and may reflect NZ having a relatively poor record of funding research (compared to some other OECD countries).

The pandemic was "predominantly of seasonal intensity",[89] and so could be seen as providing a relatively weak test of NZ's pandemic response capacity. Given the huge potential impact of more virulent pandemics in the future, we need to learn as much as possible from this recent experience. It is probably still worthwhile to address some of the omissions identified in this review and consider more the lessons detailed in the international literature.[3 90] Such knowledge is important for informing future pandemic and natural disaster planning and preparations.

Author information: Nick Wilson, Associate Professor; Jennifer A Summers, PhD Candidate; Michael G Baker, Associate Professor; Department of Public Health, University of Otago, Wellington

Correspondence: Dr Nick Wilson, Department of Public Health, University of Otago Wellington, PO Box 7343 Wellington South, New Zealand. Email nick.wilson@otago.ac.nz

References:

1. Baker MG, Easther S, Wilson N. A surveillance sector review applied to infectious diseases at a country level. BMC Public Health 2010;10:332.

2. Wilson N, Lush D. Bioterrorism in the Northern Hemisphere and potential impact on New Zealand. N Z Med J 2002;115:247-51.

3. World Health Organization. Report of the Review Committee on the Functioning of the International Health Regulations 2005 and on Pandemic Influenza A (H1N1) 2009, (WHA ref A64/10). Geneva: World Health Organization, 2011. http://apps.who.int/gb/ebwha/pdf_files/WHA64/A64_10-en.pdf

4. Pandemic Influenza Mortality and Morbidity Review Group. Report for the Minister of Health from the Pandemic Influenza Mortality and Morbidity Review Group. Wellington: Ministry of Health, 2010.

5. Falagas ME, Koletsi PK, Baskouta E, et al. Pandemic A(H1N1) 2009 influenza: review of the Southern Hemisphere experience. Epidemiol Infect 2011;139:27-40.

6. Van Kerkhove MD, Mounts AW, Mall S, et al. Epidemiologic and virologic assessment of the 2009 influenza A (H1N1) pandemic on selected temperate countries in the Southern Hemisphere: Argentina, Australia, Chile, New Zealand and South Africa. Influenza Other Respi Viruses 2011;5:e487-98.

7. Baker M, Kelly H, Wilson N. Pandemic H1N1 influenza lessons from the southern hemisphere. Euro Surveill 2009;14:pii=19370.

8. Baker M, Huang Q, Bandaranayake D, et al. The influenza A(H1N1) pandemic in New Zealand during 2009 [Poster presentation 217]. 2010 International Conference on Emerging Infectious Diseases. Atlanta, Georgia 11-14 July. 2010.

9. Rice G. Black November: The 1918 influenza pandemic in New Zealand. Christchurch: Canterbury University Press, 2005.

10. Davis L. Report on the 1957 pandemic of influenza (to the World Health Organization, Geneva). Wellington: Division of Public Hygiene, Department of Health, 1958.

11. Wilson N, Baker MG. Comparison of the content of the New Zealand influenza pandemic plan with European pandemic plans. N Z Med J 2009;122:36-46.

12. Bandaranayake D, Huang QS, Bissielo A, et al. Risk factors and immunity in a nationally representative population following the 2009 influenza A(H1N1) pandemic. PLoS One 2010;5:e13211.

13. Webb SA, Pettila V, Seppelt I, et al. Critical care services and 2009 H1N1 influenza in Australia and New Zealand. N Engl J Med 2009;361:1925-34.

14. Wilson N, Telfar Barnard L, Summers J, et al. Differential mortality by ethnicity in 3 influenza pandemics over a century, New Zealand. Emerg Infect Dis 2012;18:71-77.

15. Roberts MG, Nishiura H. Early Estimation of the Reproduction Number in the Presence of Imported Cases: Pandemic Influenza H1N1-2009 in New Zealand. PLoS One 2011;6:e17835.

16. Sertsou G, Wilson N, Baker M, et al. Key transmission parameters of an institutional outbreak during the 1918 influenza pandemic estimated by mathematical modelling. Theor Biol Med Model 2006;3:38.

17. Nishiura H, Wilson N. Transmission dynamics of the 1918 influenza pandemic in New Zealand: analyses of national and city data. N Z Med J 2009;122:81-86.

18. Andreasen V, Viboud C, Simonsen L. Epidemiologic characterization of the 1918 influenza pandemic summer wave in Copenhagen: implications for pandemic control strategies. J Infect Dis 2008;197:270-8.

19. Mills CE, Robins JM, Lipsitch M. Transmissibility of 1918 pandemic influenza. Nature 2004;432:904-6.

20. Chowell G, Ammon CE, Hengartner NW, Hyman JM. Transmission dynamics of the great influenza pandemic of 1918 in Geneva, Switzerland: Assessing the effects of hypothetical interventions. J Theor Biol 2006;241:193-204.

21. Ferguson NM, Cummings DA, Fraser C, et al. Strategies for mitigating an influenza pandemic. Nature 2006;442:448-52.

22. Nishiura H, Wilson N, Baker M. Quarantine for pandemic influenza control at the borders of small island nations. BMC Infect Dis 2009;9.

23. Carrat F, Vergu E, Ferguson NM, et al. Time Lines of Infection and Disease in Human Influenza: A Review of Volunteer Challenge Studies. Am J Epidemiol 2008;167:775-785.

24. Frost W. Statistics of influenza morbidity. Public Health Rep 1920;35:584-597.

25. Crosby AW. Epidemic and Peace: 1918. Westford: Greenwood Press, 1976.

26. Bryder L. The 1918 Influenza Epidemic in Auckland [Unpublished MA Thesis, University of Auckland], 1980.

27. Baker MG, Wilson N, Huang QS, et al. Pandemic influenza A(H1N1)v in New Zealand: the experience from April to August 2009. Euro Surveill 2009;14:ArticleId=19319.

28. Jackson G, Thornley S. Burden of novel influenza A virus (H1N1) in Auckland and Counties Manukau District Heath Boards (July 2009): a capture-recapture analysis. N Z Med J 2009;122(1301):66-9.

29. Dee S, Jayathissa S. Clinical and epidemiological characteristics of the hospitalised patients due to pandemic H1N1 2009 viral infection: experience at Hutt Hospital, New Zealand. N Z Med J 2010;123:45-53.

30. Verrall A, Norton K, Rooker S, et al. Hospitalizations for pandemic (H1N1) 2009 among Maori and Pacific Islanders, New Zealand. Emerg Infect Dis 2010;16:100-2.

31. CDC. Surveillance for the 2009 pandemic influenza A (H1N1) virus and seasonal influenza viruses – New Zealand, 2009. MMWR 2009;58:918-21.

32. Wilson N, Mason K, Tobias M, et al. Interpreting Google flu trends data for pandemic H1N1 influenza: the New Zealand experience. Euro Surveill 2009;14:pii=19386.

33. Nishiura H, Wilson N, Baker MG. Estimating the reproduction number of the novel influenza A virus (H1N1) in a Southern Hemisphere setting: preliminary estimate in New Zealand. N Z Med J 2009;122(1299):73-7.

34. Paine S, Mercer GN, Kelly PM, et al. Transmissibility of 2009 pandemic influenza A(H1N1) in New Zealand: effective reproduction number and influence of age, ethnicity and importations. Euro Surveill 2010;15:pii=19591.

35. Opatowski L, Fraser C, Griffin J, et al. Transmission Characteristics of the 2009 H1N1 Influenza Pandemic: Comparison of 8 Southern Hemisphere Countries. PLoS Pathog 7:e1002225.

36. Hsieh YH. Pandemic influenza A (H1N1) during winter influenza season in the southern hemisphere. Influenza Other Respi Viruses 4:187-97.

37. Nishiura H, Roberts MG. Estimation of the reproduction number for 2009 pandemic influenza A(H1N1) in the presence of imported cases. Euro Surveill 2010;15:pii=19622.

38. Kelly P, Mercer G, Glass K, Paine S. Author's reply: Estimation of the reproduction number for 2009 pandemic influenza A(H1N1) in the presence of imported cases. Euro Surveill 2010;15:pii=19623.

39. Roberts MG. Epidemic models with uncertainty in the reproduction number. J Math Biol 2012;[e-Publication 5 May].

40. Baker MG, Thornley CN, Mills C, et al. Transmission of pandemic A/H1N1 2009 influenza on passenger aircraft: retrospective cohort study. BMJ 2010;340:c2424.

41. Mardani J, Calder L, Laurie K, et al. Limited novel influenza A (H1N1) 09 infection in travelling high-school tour group. Influenza Other Respi Viruses 2011;5:47-51.

42. Karageorgopoulos DE, Vouloumanou EK, Korbila IP, et al. Age distribution of cases of 2009 (H1N1) pandemic influenza in comparison with seasonal influenza. PLoS One 2011;6:e21690.

43. ANZIC Influenza Investigators and Australasian Maternity Outcomes Surveillance System. Critical illness due to 2009 A/H1N1 influenza in pregnant and postpartum women: population based cohort study. BMJ 2010;340:c1279.

44. Knight M, Pierce M, Seppelt I, et al. Critical illness with AH1N1v influenza in pregnancy: a comparison of two population-based cohorts. BJOG 2011;118:232-9.

45. Hale MJ, Hoskins RS, Baker MG. Screening for Influenza A(H1N1)pdm09, Auckland International Airport, New Zealand. Emerg Infect Dis 2012;18:866-8.

46. Jutel A, Baker M, Stanley J, et al. Self-diagnosis of influenza during a pandemic: a cross-sectional survey. BMJ Open 2011;1:e000234.

47. Gray L, MacDonald C, Mackie B, et al. Community responses to communication campaigns for influenza A (H1N1): a focus group study. BMC Public Health 2012;12:205.

48. Murray R, Chandler C, Clarkson Y, et al. Sub-optimal hand sanitiser usage in a hospital entrance during an influenza pandemic, New Zealand, August 2009. Euro Surveill 2009;14:pii=19331.

49. Manning S, Barry T, Wilson N, Baker M. Update: follow-up study showing post-pandemic decline in hand sanitiser use, New Zealand, December 2009. Euro Surveill 2010;15:pii: 19466.

50. Manning S, Barry T, Baker M, Wilson N. Hand hygiene practices at a hospital entrance after the 2009 influenza pandemic: observational study over one year. N Z Med J 2011;124(1334):111-114.

51. Barry T, Manning S, Lee MS, et al. Respiratory hygiene practices by the public during the 2009 influenza pandemic: an observational study. Influenza Other Respi Viruses 2011;5:317-20.

52. Peacey M, Hall RJ, Bocacao J, Huang QS. Diagnostic assay recommended by the world health organization for swine origin influenza a (h1n1) virus cross-reacts with H5N1 influenza virus. J Clin Microbiol 2009;47:3789-90.

53. Hall RJ, Peacey M, Huang QS, Carter PE. Rapid method to support diagnosis of swine origin influenza virus infection by sequencing of real-time PCR amplicons from diagnostic assays. J Clin Microbiol 2009;47:3053-4.

54. Ducatez MF, Sonnberg S, Hall RJ, et al. Genotyping assay for the identification of 2009-2010 pandemic and seasonal H1N1 influenza virus reassortants. J Virol Methods 2010;168:78-81.

55. Hall RJ, Peacey MP, Ralston JC, et al. Pandemic influenza A(H1N1)v viruses currently circulating in New Zealand are sensitive to oseltamivir. Euro Surveill 2009;14:pii=19282.

56. Peacey M, Hall RJ, Sonnberg S, et al. Pandemic (H1N1) 2009 and seasonal influenza A (H1N1) co-infection, New Zealand, 2009. Emerg Infect Disease 2010;16:1618-20.

57. Cheng AC, Dwyer DE, Kotsimbos AT, et al. Summary of the Australasian Society for Infectious Diseases and the Thoracic Society of Australia and New Zealand guidelines: treatment and prevention of H1N1 influenza 09 (human swine influenza) with antiviral agents. Med J Aust 2009;191:142-5.

58. Jennings LC. Influenza A(H1N1)09: another public health risk to New Zealand. N Z Med J 2009;122(1298):11-6.

59. Thompson C, Rolls S. Keeping alert to changes during the pandemic. Nurs N Z 2009;15:30.

60. Johal SS. Psychosocial impacts of quarantine during disease outbreaks and interventions that may help to relieve strain. N Z Med J 2009;122:47-52.

61. Williams D, Begg A, Burgess K, et al. Influenza H1N1 2009 in Canterbury: a case study in pandemic response co-ordination. J Prim Health Care 2010;2(4):323-9.

62. Hughes FA. H1N1 pandemic planning in a mental health residential facility. J Psychosoc Nurs Ment Health Serv 2010;48(3):37-41.

63. Raymond NJ, Berry N, Blackmore TK, et al. Pandemic influenza A(H1N1)2009 in hospital healthcare workers in New Zealand. Infect Control Hosp Epidemiol 2012;33:196-9.

64. Webb SA, Seppelt IM. Pandemic (H1N1) 2009 influenza ("swine flu") in Australian and New Zealand intensive care. Crit Care Resusc 2009;11:170-2.

65. Kotsimbos T, Waterer G, Jenkins C, et al. Influenza A/H1N1_09: Australia and New Zealand's winter of discontent. Am J Respir Crit Care Med 2010;181:300-6.

66. Drennan K, Hicks P, Hart G. Impact of pandemic (H1N1) 2009 on Australasian critical care units. Crit Care Resusc 2010;12:223-9.

67. Harrigan PW, Webb SA, Seppelt IM, et al. The practical experience of managing the H1N1 2009 influenza pandemic in Australian and New Zealand intensive care units. Crit Care Resusc 2010;12:121-30.

68. Yung M, Slater A, Festa M, et al. Pandemic H1N1 in children requiring intensive care in Australia and New Zealand during winter 2009. Pediatrics 2011;127:e156-63.

69. Buckley E, Sidebotham D, McGeorge A, et al. Extracorporeal membrane oxygenation for cardiorespiratory failure in four patients with pandemic H1N1 2009 influenza virus and secondary bacterial infection. Br J Anaesth 2010;104:326-9.

70. Davies A, Jones D, Bailey M, et al. Extracorporeal Membrane Oxygenation for 2009 Influenza A(H1N1) Acute Respiratory Distress Syndrome. JAMA 2009;302:1888-95.

71. Sidebotham D. Extracorporeal membrane oxygenation--understanding the evidence: CESAR and beyond. J Extra Corpor Technol 2011;43:P23-6.

72. Jefferies S, Earl D, Berry N, et al. Effectiveness of the 2009 seasonal influenza vaccine against pandemic influenza A(H1N1)2009 in healthcare workers in New Zealand, June-August 2009. Euro Surveill 2010;16:pii=19761.

73. Petousis-Harris H, Poole T, Booy R, Turner N. Fever following administration of two inactivated influenza vaccines--a survey of parents of New Zealand infants and children 5 years of age and under. Vaccine 2011;29:2933-7.

74. Kelly HA, Mercer GN, Fielding JE, et al. Pandemic (H1N1) 2009 influenza community transmission was established in one Australian state when the virus was first identified in North America. PLoS One 2010;5:e11341.

75. Khandaker G, Dierig A, Rashid H, et al. Systematic review of clinical and epidemiological features of the pandemic influenza A (H1N1) 2009. Influenza Other Respi Viruses 2011;5:148-56.

76. Dawood FS, Iuliano AD, Reed C, et al. Estimated global mortality associated with the first 12 months of 2009 pandemic influenza A H1N1 virus circulation: a modelling study. Lancet Infect Dis 2012;[E-publication 26 June].

77. Louie JK, Jean C, Acosta M, et al. A review of adult mortality due to 2009 pandemic (H1N1) influenza A in California. PLoS One 2011;6:e18221.

78. Bandaranayake D, Jacobs M, Baker M, et al. The second wave of 2009 pandemic influenza A(H1N1) in New Zealand, January-October 2010. Euro Surveill 2011;16.

79. Ministry of Health. Influenza. In: Immunisation Handbook. Wellington: Ministry of Health, 2011. http://www.health.govt.nz/system/files/documents/publications/15influenza.pdf

80. Armstrong P, Dowse G, Effler P, et al. Epidemiological study of severe febrile reactions in young children in Western Australia caused by a 2010 trivalent inactivated influenza vaccine. BMJ Open 2011;(30 May). doi: 10.1136/bmjopen-2010-000016.

81. Ministry of Health. Report on Exercise Cruickshank. Wellington: Ministry of Health, 2007. http://www.health.govt.nz/publication/report-exercise-cruickshank

82. Zarychanski R, Stuart TL, Kumar A, et al. Correlates of severe disease in patients with 2009 pandemic influenza (H1N1) virus infection. CMAJ 2010;182:257-64.

83. Gilca R, De Serres G, Boulianne N, et al. Risk factors for hospitalization and severe outcomes of 2009 pandemic H1N1 influenza in Quebec, Canada. Influenza Other Respi Viruses 2011;5:247-55.

84. Ward KA, Spokes PJ, McAnulty JM. Case-control study of risk factors for hospitalization caused by pandemic (H1N1) 2009. Emerg Infect Disease 2011;17:1409-16.

85. Wilson N, Baker M, Jennings L, et al. A design fault in New Zealand's health research funding system exposed by pandemic influenza. N Z Med J 2009;122(1304):121-2.

86. Wilson N, Thomson G, Mansoor O. Print media response to SARS in New Zealand. Emerg Infect Dis 2004;10:1461-4.

87. Higgins AM, Pettila V, Harris AH, et al. The critical care costs of the influenza A/H1N1 2009 pandemic in Australia and New Zealand. Anaesth Intensive Care 2011;39:384-91.

88. Wilson N, Nghiem N, Higgins A, et al. A national estimate of the hospitalisation costs for the influenza (H1N1) pandemic in 2009. N Z Med J 2012;125(1365). http://journal.nzma.org.nz/journal/125-1365/5430

89. Kelly HA. A pandemic response to a disease of predominantly seasonal intensity. Med J Aust 2010;192:81-3.

90. Leung GM, Nicoll A. Reflections on pandemic (H1N1) 2009 and the international response. PLoS Med 2010;7.

Celebrating 50 years of polio elimination in New Zealand: but inadequate progress in eliminating other vaccine-preventable diseases

Nick Wilson, Michael G Baker

Abstract

New Zealanders can now reflect on and celebrate 50 years of polio elimination in this country. This success was followed by eliminating two other infectious diseases, brucellosis and hydatids, and an imported potential disease vector, the southern saltmarsh mosquito. However, this country has made inadequate progress in eliminating several other vaccine-preventable diseases. These include measles, mumps, and rubella, which are priority candidates for elimination, and potentially Hib disease and rotavirus infection.

To achieve such successes almost certainly requires that the country: (i) builds national leadership for elimination goals; (ii) develops detailed plans; (iii) continues recent successes in enhancing routine vaccination coverage; (iv) introduces rotavirus vaccine into the childhood immunisation schedule; and (v) strengthens surveillance and research (on such questions as the cost-effectiveness of new vaccines, measures to enhance uptake, and effective border controls to reduce the risk of disease importation).

For 50 years now (since 1 April 1962), New Zealand has been free of transmission of wild-type polio virus infection. The end of this disease was particularly sudden with cases declining from 214 notifications in an outbreak in 1961 (with seven deaths) to only five cases in early 1962.[1]

The end coincided with mass immunisation campaigns with the new Sabin (oral) vaccine in 1961 and 1962. The coverage in the vaccination campaign running from April to June 1962 was approximately 95% of the child population up to school leaving age.[2] In the subsequent 50 years there have only been occasional cases of vaccine-associated paralytic poliomyelitis (VAPP), four of which have been laboratory-confirmed.[3] However, there is no longer a vaccination-related risk in New Zealand as in 2002 the national immunisation schedule was changed to replace oral polio vaccine with inactivated polio vaccine (the Salk vaccine).[3]

At a regional level, the Western Pacific Region, of which New Zealand is a part, was declared polio-free in 2000,[3] the consequences of which further lower the risk of disease importation into New Zealand. Even so, health authorities remain appropriately vigilant by maintaining surveillance of acute flaccid paralysis and keeping polio on the notifiable disease schedule of the Health Act.[4]

In this viewpoint article we go beyond the polio success to briefly consider the benefits of disease elimination, the additional vaccine-preventable diseases (VPDs) that could be eliminated in New Zealand, and the actions which are probably needed to achieve such elimination goals.

Disease elimination and eradication internationally

Infectious diseases are one of the few human health threats that have the potential to be entirely eradicated (along with some forms of injury and poisoning where the external cause can be removed from the environment). *Eradication* refers to the cessation of disease transmission at a global level based on the total absence of human cases and the lack or removal of natural reservoirs or other sources of infection. The term *elimination* is generally used to describe the cessation of transmission within a country or region (though "regional eradication" is sometimes also used for the latter). Disease *control* refers to the use of interventions that restrict the circulation of an infectious agent beyond the level that would occur without any such interventions, and unlike the other terms, should be qualified by specifying the level of control achieved.[5]

Disease eradication offers the huge potential advantage that might arise from no longer having to invest resources in prevention measures, as has been seen with global smallpox eradication and more recently with the globally eradicated cattle disease rinderpest.[6] In contrast, elimination and control imply a need for continuing interventions and surveillance, as is still currently the case with polio (i.e., which remains endemic in several countries, albeit at generally declining levels).

Only some diseases are candidates for global eradication or regional or national elimination at the current time. At a technical and biological level, eradication usually requires an effective, practical and affordable intervention; the availability of accurate diagnostic tests that can detect levels of infection that can lead to transmission; and that "humans are essential for the life-cycle of the agent, which has no other vertebrate reservoir and does not amplify in the environment".[7] In addition to biological feasibility, disease eradication requires adequate public health infrastructure, sufficient funding, and sustained political/societal will.[8]

The International Taskforce on Disease Eradication has repeatedly published (and updated) a list of diseases that they consider are candidates for eradication.[9] Their current list of eradicable and potentially eradicable diseases consists of: poliomyelitis, dracunculiasis (Guinea worm disease), measles, mumps, rubella, lymphatic filariasis and taeniasis/cysticercosis (pork tapeworm disease). They also list a number of other diseases, such as hepatitis B, where elimination is conceivable in the future.

Vaccine-preventable diseases (VPDs) that could potentially be eliminated from New Zealand

Polio is the only disease to have been eliminated in New Zealand as a result of mass immunisation. The other elimination successes for brucellosis and hydatids, involved a mix of public health and veterinary measures.[10] Another elimination success was from environmental control measures i.e., eliminating the imported southern saltmarsh mosquito (a potential mosquito vector for Ross River virus).[10]

Measles, mumps and rubella—Of the diseases identified for global eradication, measles is currently receiving the greatest attention. A recent supplement of the *Journal of Infectious Diseases* was devoted to this question and concluded that measles eradication is biologically feasible so the challenges are logistical, political

and financial.[11] Achieving global eradication is the disease control scenario that modelling indicates is the most cost-effective.[12]

There has been regional elimination of both measles and rubella in the Americas,[8] with this status persisting even though occasional outbreaks associated with imported cases occur (e.g., in the US in 2011[13]). Also there were zero cases of measles in eight European countries in 2010,[14] though recent outbreaks have involved much of Europe.[15] In New Zealand's own region (the World Health Organization's [WHO] Western Pacific Region), measles elimination is a 2012 target.[16]

WHO defines measles elimination as "the absence of endemic measles transmission in a defined geographical area (e.g., region) for ≥12 months in the presence of a well performing surveillance system."[17]

Following the last national epidemic of measles in 1997,[18] New Zealand had relatively few cases of measles for a decade but then in recent years there have been notable outbreaks. There were 248 notified cases in 2009, 48 in 2010, and 597 in six outbreaks in 2011[19] and additional outbreaks in early 2012 (e.g., n=69 cases for January to April).[20] The incidence of measles in New Zealand was the highest reported in the Western Pacific Region of WHO in 2009 (NZ and Vietnam both reported rates of 59.3 cases / million)[21] and again in 2011 (the NZ rate was 135.7 cases / million).[22]

The size of these outbreaks in New Zealand probably reflects the inadequate levels of vaccination coverage historically (with older child and youth age cohorts with suboptimal coverage) and suggests that the country will need to do more to achieve elimination status. As noted by the WHO, "Countries with ongoing measles virus transmission in 2012—including China, Malaysia, New Zealand and Singapore— likely will need to supplement routine immunisation with special activities to interrupt measles virus transmission in 2012."[22]

Hib disease—The progress in some European countries (e.g., zero cases in Greece, Iceland and Malta in the last 2 years of 2005/06 for one data source[23]), suggests that elimination of *Haemophilus influenzae* type b (Hib disease), may be feasible at the country-level. Furthermore, the herd immunity effect from vaccination appears notable for this disease.[24] Since New Zealand introduced vaccination against Hib disease, there have been impressive reductions in the disease burden.[25,19] So potentially only modest further improvements in childhood vaccination coverage in New Zealand might be needed to achieve elimination.

Rotavirus infection—This is a VPD where infant vaccination appears to have large herd immunity effects and helps to protect older unvaccinated children and young adults.[26] This suggests scope for national elimination, though global eradication may not be technically feasible because of zoonotic transmission and animal reservoirs.[27] In 2009, the WHO recommended inclusion of rotavirus vaccination in all national immunisation programmes.[28] While there is favourable evidence from a cost-effectiveness study for New Zealand,[29] rotavirus vaccine has not yet been added to the routine childhood immunisation schedule in this country.

Other VPDs—Other VPDs will become candidates for elimination in the future, as new vaccines and other control measures become available. A potential example is varicella (chicken pox). Previous modelling work has suggested that vaccination

against varicella might result in elimination at the country-level (e.g., in Germany[30]). Furthermore, in the US there has been near elimination of deaths from varicella with the one-dose programme and "with the current two-dose programme, there is potential that these most severe outcomes of a vaccine-preventable disease could be eliminated".[31] Nevertheless, the issues around vaccination benefits and costs are very complex. For example, while a study found a combined vaccination policy to be cost-effective (i.e., both varicella and zoster vaccination options),[32] it concluded that: "Policy makers should be aware of the potential negative benefits in the first 30–50 years after introduction of a childhood varicella vaccine. This can only be partly mitigated by the introduction of a herpes zoster vaccine. They have to decide how they value the potential benefits beyond this time to consider childhood vaccination cost-effective."[32]

Given such complexities, aiming for varicella elimination is probably too uncertain and a more appropriate goal for New Zealand may be to focus only on disease burden reduction by adding a suitable vaccine to the childhood immunisation schedule and carefully monitoring experience elsewhere.

Vaccine introduction has previously been recommended for New Zealand by the Immunisation Technical Forum, by various paediatricians,[33] and by immunisation experts.[34] Work indicating favourable cost-effectiveness for varicella vaccination in New Zealand has been done,[35] though ideally this could benefit from updating.

Potential actions to progress VPD elimination in New Zealand

There are a number of actions that New Zealand could take to progress the elimination of those VPDs listed above:

- **National leadership and planning—**National-level political and health leaders should ideally articulate VPD elimination goals in political manifestos, in strategic documents and in the media. Having detailed plans with defined elimination goals is critical, as was seen for the recent elimination of the southern saltmarsh mosquito.

- **Enhancing routine childhood immunisation coverage—**The current New Zealand Government has made raising routine childhood immunisation coverage one of its health priorities and has been making good progress towards this.[36 37] It might be enough to continue on the track of recent improvements in immunisation service delivery (as recently detailed[37]). But if after a few more years there is not good evidence that measles is about to be eliminated, then other options include: (i) Considering supplementary measles vaccination for older children; (ii) Generating media coverage for a national measles elimination goal (and potentially for selected other VPDs); (iii) Running paid mass media campaigns to promote vaccination uptake. Such media campaigns could address the barrier of "public misconception of seriousness" for measles,[9] and potentially any public concerns around vaccine safety; (iv) Possibly further incentivising vaccination providers to enhance service delivery.

- **Introducing routine rotavirus vaccination—**While updating a previous cost-effectiveness study[29] on this oral vaccination for the New Zealand setting

would be optimal, there is probably enough information for prudent policy makers to act now with this introduction. Some of the additional information has been recently cited by Reid[34] and relates to the likely reductions in vaccine price, the prior cost-effectiveness work not considering full benefits to caregivers (aspects of the loss of income for caregivers associated with caring for sick children), and the emerging evidence for improvements in the timeliness of coverage for other scheduled vaccinations when rotavirus vaccination is used. In addition, the previous cost-effectiveness work was considered conservative by its authors[29] and as they stated it didn't consider potential benefits of herd immunity and benefits for preventing nosocomial transmission. Finally, the potential benefit of reducing the seasonal burden of rotavirus outbreaks on health service functioning (as detailed for the UK[38]), was not accounted for.

- **Possible activities at the border—**When measles vaccination coverage for children exceeds 95% in New Zealand, supplementary activities at the border could be considered. That is, all children entering or departing could be required to show documentation of measles vaccination. This need was illustrated by the recent well documented importation of measles cases into Auckland.[39] Nevertheless, such moves need to consider the various limitations identified from previous New Zealand experience with border control activities for infectious disease control in humans.[40,41]

- **Strengthening surveillance capacity—**Comprehensive surveillance is critical to support all stages of disease elimination, from describing the incidence and distribution of disease, identifying individual cases for control measures, monitoring the coverage of interventions such as vaccinations, and finally for confirming successful disease elimination (as is currently illustrated with acute flaccid paralysis for polio in New Zealand[4] and elsewhere). There is considerable potential to improve New Zealand's infectious disease surveillance system to support elimination and control objectives.[42]

- **Research—**To guide decisions around the potential for rotavirus elimination in New Zealand, there needs to be more research on zoonotic and environmental transmission to humans in this country. For example, rotavirus has been detected in surface waters used as drinking water sources in New Zealand.[43]

Action in these areas is compatible with the current government's interest in improving routine childhood immunisation coverage (as one of six health priorities).[36] They are also generally compatible with the international obligations the country has agreed to for measles elimination.[16] But most importantly, elimination of these additional VPDs would be beneficial for improving child health, reducing health inequalities, and lowering costs for New Zealand's health care services.

Competing interests: Although we do not consider it a competing interest, we note that both of the authors have previously done contract work for the Ministry of Health (MoH) or have been advisors to the MoH on infectious disease epidemiology and control issues.

Author information: Nick Wilson, Associate Professor, Department of Public Health, University of Otago, Wellington; Michael G Baker, Associate Professor, Department of Public Health, University of Otago, Wellington

Correspondence: Dr Nick Wilson, Department of Public Health, University of Otago Wellington, PO Box 7343 Wellington South, New Zealand. Email nick.wilson@otago.ac.nz

References:

1. Statistics New Zealand. The New Zealand Official Year-Book, 1965. http://www3.stats.govt.nz/New_Zealand_Official_Yearbooks/1965/NZOYB_1965.html
2. Statistics New Zealand. The New Zealand Official Year-Book, 1963. http://www3.stats.govt.nz/New_Zealand_Official_Yearbooks/1963/NZOYB_1963.html
3. Ministry of Health. Immunisation Handbook 2011. Wellington: Ministry of Health, 2011.
4. Chambers ST, Dickson N. Global polio eradication: progress, but determination and vigilance still needed. N Z Med J. 2011;124(1337):100-4.
5. Miller M, Barrett S, Henderson DA. Control and Eradication (Chapter 62). In: Jamison DT, Breman JG, Measham AR, et al (editors). Disease Control Priorities in Developing Countries. 2nd edition. Washington (DC): World Bank; 2006.
6. Normile D. Animal science. Rinderpest, deadly for cattle, joins smallpox as a vanquished disease. Science. 2010;330:435.
7. Dowdle WR. The principles of disease elimination and eradication. Bull World Health Organ. 1998;76 Suppl 2:22-5.
8. Dowdle WR, Cochi SL. The principles and feasibility of disease eradication. Vaccine. 2011;29 Suppl 4:D70-3.
9. International Task Force for Disease Eradication. Disease considered as candidates for global eradication by the International Task Force for Disease Eradication. Atlanta, GA. The Carter Centre, 2008. http://www.cartercenter.org/resources/pdfs/news/health_publications/itfde/updated_disease_candidate_table.pdf
10. Wilson N, Lush D, Bell P, Wyber R. Lessons for zoonotic disease and vector eradication in New Zealand from past successes [Poster P1-370 at the World Congress of Epidemiology, Edinburgh, 7-11 August 2011]. http://www.otago.ac.nz/wellington/otago024538.pdf
11. Moss WJ, Strebel P. Biological feasibility of measles eradication. J Infect Dis. 2011;204 Suppl 1:S47-53.
12. Levin A, Burgess C, Garrison LP, Jr., et al. Global eradication of measles: an epidemiologic and economic evaluation. J Infect Dis. 2011;204 Suppl 1:S98-106.
13. CDC. Measles: United States, January--May 20, 2011. MMWR. 2011;60:666-8.
14. Steffens I, Martin R, Lopalco P. Spotlight on measles 2010: measles elimination in Europe - a new commitment to meet the goal by 2015. Euro Surveill. 2010;15:pii=19749.
15. Moss WJ, Griffin DE. Measles. Lancet. 2012;379:153-64.
16. World Health Organization. WHO Regional EPI targets: eliminate measles and control hepatitis B by 2012. Fifty-sixth session of the WHO Regional Committee for the Western Pacific, 19 to 23 September, 2005.
17. World Health Organization. Monitoring progress towards measles elimination. Wkly Epidemiol Rec. 2010;85:490-4.
18. Mansoor O, Blakely T, Baker M, et al. A measles epidemic controlled by immunisation. N Z Med J. 1998;111:467-71.
19. Institute of Environmental Science and Research Ltd (ESR). Notifiable and Other Diseases in New Zealand: Annual Report 2011. Porirua, ESR, 2012.
20. ESR. Notifiable disease surveillance. N Z Public Health Surveillance Report 2012;10(2):3.

21. Sniadack DH, Mendoza-Aldana J, Jee Y, et al. Progress and challenges for measles elimination by 2012 in the Western Pacific Region. J Infect Dis. 2011;204 Suppl 1:S439-46.

22. Regional Office for the Western Pacific - World Health Organization. Special topic - Measles elimination and importations in the Western Pacific Region. Measles-Rubella Bulletin. 2012;6:9-10.

23. Ladhani S, Slack MP, Heath PT, et al. Invasive Haemophilus influenzae Disease, Europe, 1996-2006. Emerg Infect Dis. 2010;16:455-63.

24. McVernon J, Ramsay ME, McLean AR. Understanding the impact of Hib conjugate vaccine on transmission, immunity and disease in the United Kingdom. Epidemiol Infect. 2008;136:800-12.

25. Wilson N, Wenger J, Mansoor O, et al. The beneficial impact of Hib vaccine on disease rates in New Zealand children. N Z Med J. 2002;115:U122.

26. Lopman BA, Curns AT, Yen C, Parashar UD. Infant rotavirus vaccination may provide indirect protection to older children and adults in the United States. J Infect Dis. 2011;204:980-6.

27. Midgley SE, Hjulsager CK, Larsen LE, et al. Suspected zoonotic transmission of rotavirus group A in Danish adults. Epidemiol Infect. 2012;140:1013-7.

28. World Health Organization. Rotavirus vaccines: an update. Wkly Epidemiol Rec. 2009;84:533-40.

29. Milne RJ, Grimwood K. Budget impact and cost-effectiveness of including a pentavalent rotavirus vaccine in the New Zealand childhood immunization schedule. Value Health. 2009;12:888-98.

30. Wutzler P, Neiss A, Banz K, et al. Can varicella be eliminated by vaccination? Potential clinical and economic effects of universal childhood varicella immunisation in Germany. Med Microbiol Immunol. 2002;191:89-96.

31. Marin M, Zhang JX, Seward JF. Near elimination of varicella deaths in the US after implementation of the vaccination program. Pediatrics. 2011;128:214-20.

32. van Hoek AJ, Melegaro A, Gay N, et al. The cost-effectiveness of varicella and combined varicella and herpes zoster vaccination programmes in the United Kingdom. Vaccine. 2012;30:1225-34.

33. Walls T, Wilson E. Has the time come for a universal varicella (chicken pox) vaccination in New Zealand? N Z Med J. 2010;123(1326):22-5.

34. Reid S. The further and future evolution of the New Zealand Immunisation Schedule. N Z Med J. 2012;125(1354):86-99.

35. Scuffham P, Devlin N, Eberhart-Phillips J, Wilson-Salt R. The cost-effectiveness of introducing a varicella vaccine to the New Zealand immunisation schedule. Soc Sci Med. 1999;49:763-79.

36. National Heath Board and Ministry of Health. Targeting Immunisation. Wellington: Ministry of Health, 2011. http://www.nationalhealthboard.govt.nz/our-priorities/health-targets

37. Turner N. The challenge of improving immunization coverage: the New Zealand example. Expert Rev Vaccines. 2012;11:9-11.

38. Morgan C, Adlard N, Carroll S, Parvataneni L. Burden on UK secondary care of rotavirus disease and seasonal infections in children. Curr Med Res Opin. 2010;26:2449-55.

39. Centers for Disease Control and Prevention (CDC). Notes from the field: Multiple cases of measles after exposure during air travel--Australia and New Zealand, January 2011. MMWR. 2011;60:851.

40. Priest PC, Duncan AR, Jennings LC, Baker MG. Thermal image scanning for influenza border screening: results of an airport screening study. PLoS One. 2011;6:e14490.

41. Hale MJ, Hoskins RS, Baker MG. Screening for Influenza A(H1N1)pdm09, Auckland International Airport, New Zealand. Emerg Infect Dis. 2012;18:866-868.

42. Baker MG, Easther S, Wilson N. A surveillance sector review applied to infectious diseases at a country level. BMC Public Health. 2010;10:332.

43. Williamson WM, Ball A, Wolf S, et al. Enteric viruses in New Zealand drinking-water sources. Water Sci Technol. 2011;63:1744-51.

Shewanella algae causing lower limb soft tissue infection in New Zealand

Bonnie Leung, Richard Meech, Nicky Lau, Robert Cunliffe

Abstract

A 59-year-old man presented to Tauranga Hospital (Tauranga, New Zealand) with lower limb soft tissue infection growing *Shewanella algae* isolated from blood and skin after fishing in seawater. This is the first published report of this marine organism causing infection in New Zealand.

Case report

A 59-year-old recreational fisherman—with a background of chronic heart failure, mild lower limb oedema and superficial skin abrasions—sustained a laceration to the lateral aspect of his right leg from the lid of a fish bin. Over the next 12 hours he developed an acute cellulitis of his right leg which was empirically treated with flucloxacillin by his GP.

He was then admitted to hospital after 2 days of fevers, rigors and severe leg pain. The blistered skin was bright red, swollen and had a malodorous sero-sanguineous discharge (likened to the smell of "rotting fish").

On examination, he was tachycardic, with a blood pressure of 132/88 mmHg and temperature of 38.6°C. There were two 1×1 cm superficial infected wounds on the right posterior calf, diffuse superficial blistering and several bullae on the lateral aspect of the leg (Figure 1).

Figure 1. Lateral right leg on admission (left) and at discharge (right)
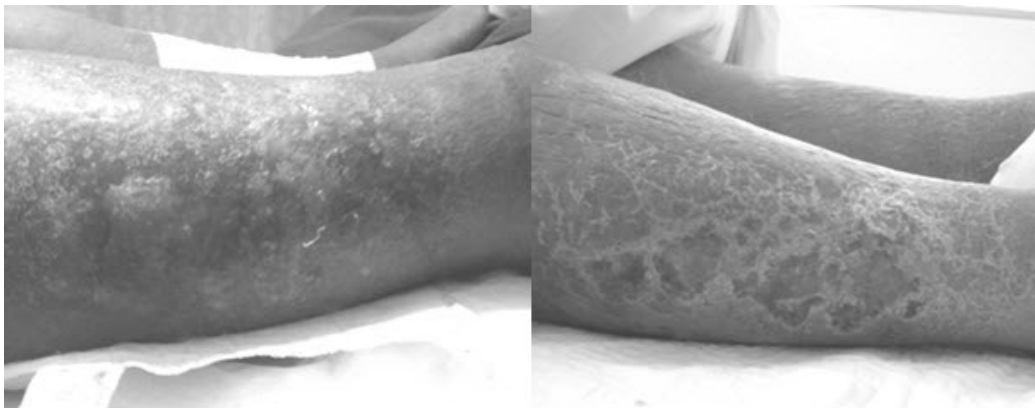
Investigations showed leukocytosis (14.7×10^9/L), neutrophilia (13.2×10^9/L) and a raised C-reactive protein (197 mg/L). Lower limb skin swabs grew *Shewanella algae* (*S. algae)* [Figure 2] and group B beta-haemolytic streptococci.

Blood cultures were positive for *S. algae* sensitive to amoxycillin, cefuroxime, cotrimoxazole and gentamicin.

Figure 2: Yellow-brown *S. alga*e colonies on MacConkey agar (top half), and characteristic mucoid appearance on blood agar (bottom half)
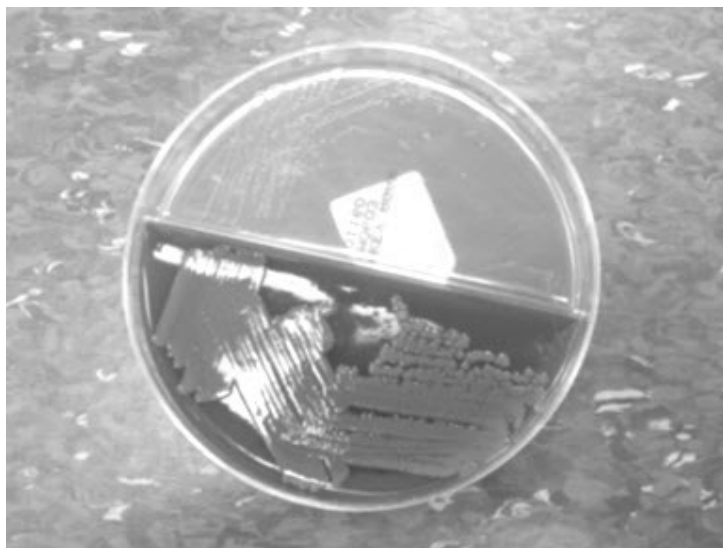
He was treated for a total of 20 days, with piperacillin/tazobactam and then amoxycillin when sensitivities were obtained. Oral clindamycin was added in the last week to broaden the antibiotic cover because improvement was slow to progress. However, surgical intervention was not required.

Discussion

Shewanella algae are motile, oxidase-positive and non-fermentative organisms found in marine environments, more frequently in warmer climates and during summer. It produces hydrogen sulphide and volatile amine compounds, mainly trimethylamine, which cause the characteristic pungent smell of rotting fish.[1]

First described in 1990, the species is a rare human pathogen that causes skin and soft tissue infections, often with bacteraemia.[2,3] Isolates found in this case were sensitive to amoxycillin, but had a poor response to flucloxacillin because *S. algae* is a Gram-negative bacillus. *S. algae* colonies on MacConkey agar are characteristically yellow-brown.[4] In contrast to *S. putrefaciens*, they have a mucoid appearance and are weakly haemolytic on blood agar.[4] Most isolates from human disease are now thought to be *S. algae*, rather than *S. putrefaciens*.[1]

S. algae can be found as a coloniser or a component of mixed flora which may make it difficult to distinguish the clinical significance.[4] In this case, *S. algae* were pathological as it was grown in blood culture.

Tsai et al identified a total 23 cases of soft tissue infections in the published English literature up to 2008; 10 cases (43%) were associated with bacteraemia. Comorbid conditions found in this group were chronic leg ulcers in the majority of cases (52%), steroid use (17%) and liver cirrhosis (9%). Two patients died of complications of septicaemia.[3] Patients with peripheral vascular disease, diabetes or receiving immunosuppressive medication may be at increased risk of *Shewanella* soft tissue infection.[5,6]

Case reports indicate that *Shewanella* infections should be treated aggressively with appropriate antibiotics along with surgical debridement when necessary.[4] Delay in treatment has been associated with an increased risk of morbidity and mortality.[4]

Author information: Bonnie Leung, Trainee Intern, Faculty of Medical and Health Sciences, University of Auckland; Richard Meech, Infectious Disease Physician, Tauranga Hospital; Nicky Lau, General Medicine Registrar, Tauranga Hospital; Robert Cunliffe, Gastroenterologist, Tauranga Hospital, Tauranga

Correspondence: Dr Robert Cunliffe, Tauranga Hospital, Private Bag 12024, Tauranga, New Zealand. Fax: +64 (0)7 5782649, email: Robert.Cunliffe@bopdhb.govt.nz

References:

1. Holt HM, Gahrn-Hansen B, Bruun B. Shewanella algae and Shewanella putrefaciens: clinical and microbiological characteristics. Clinical microbiology and infection. 2005;11(5):347-52.
2. Simidu U, Kita-Tsukamoto K, Yasumoto T, Yotsu M. Taxonomy of four marine bacterial strains that produce tetrodotoxin. International journal of systematic bacteriology. 1990;40(4):331-6.
3. Tsai MS, You HL, Tang YF, et al. Shewanella soft tissue infection: case report and literature review. International Journal of Infectious Diseases. 2008;12(6):e119-e24.
4. Goyal R, Kaur N, Thakur R. Human soft tissue infection by the emerging pathogen Shewanella algae. The Journal of Infection in Developing Countries. 2011;5(4):310-2.
5. Dominguez H, Vogel BF, Gram L, Hoffmann S, Schaebel S. Shewanella alga bacteremia in two patients with lower leg ulcers. Clinical Infectious Diseases. 1996;22(6):1036-9.
6. Papanaoum K, Marshmann G, Gordon LA, et al. Concurrent infection due to Shewanella putrefaciens and Mycobacterium marinum acquired at the beach. Australasian Journal of Dermatology. 1998;39(2):92-5.

Ptosis and diplopia as initial manifestation of Guillain-Barré syndrome

Sandjay Pandey, Manmohan Mehndiratta

Abstract

In this case report a 58-year-old Guillain-Barré syndrome patient in India presents with ptosis and diplopia. Cranial nerve examination revealed bilateral restricted abduction and ptosis as shown in the associated video clip.

Oculomotor weakness in Guillain-Barré syndrome (GBS) is uncommon. Rarely patients of GBS have been described with severe ptosis.[1] We describe a patient of GBS who presented with ptosis and diplopia.

Case report

A 58-year-old gentleman presented with ptosis of both eyes and diplopia for 3 days and quadriparesis with dysphagia for 2 days. His symptoms worsened rapidly and was admitted in neurology intensive care unit. There was no history of snake bite.

On examination he was afebrile, pulse rate was 76/minute, blood pressure was 126/80 mmHg and there was no autonomic dysfunction. He was conscious and obeying to commands. Cranial nerve examination revealed bilateral restricted abduction and ptosis (see Figure 1 video). Pupils were normal. Motor power was grade 2/5 (MRC scale) in upper limbs and grade 3/5 in lower limbs. All deep tendon reflexes were absent. Nerve conduction study was suggestive of acute inflammatory demyelinating polyneuropathy.

Figure 1. Video showing ptosis and bilateral abduction restriction

View this video at http://journal.nzma.org.nz/journal/125-1365/5424/video.avi

Cerebrospinal fluid examination revealed cytoalbuminological dissociation (no cell, protein 161 mg/dl). Anti Gq 1b antibody was negative. Repetitive nerve stimulation and Tensilon tests were normal. Magnetic resonance imaging of brain was normal. Patient was given intravenous immunoglobulin for 5 days (0.4 gm/kg/day). He started improving by day 16 of his illness. His ptosis improved and quadriparesis also recovered. He was discharged on day 23 of admission. He recovered fully in 6 weeks.

Discussion

Clinical course, nerve conduction study, negative anti Gq 1b antibody, negative RNS and tensilon tests, cytoalbuminological dissociation and good response to treatment are characteristic of GBS in our patient. However ptosis and diplopia as presenting

symptom is very unusual. Rarely GBS patients may have opthalmoplegia during course of their illness.

In a study, 9.9% patients of GBS had ophthalmoplegia.[2] In some patients a restricted type of pharyngeal-cervical-brachial variant of GBS is seen who does not progress.[3] In another study out of 92 consecutive patients of GBS, 8 had severe ptosis without ophthalmoplegia.[1] None of the patients developed other signs of oculomotor weakness. Regional variant of Guillian Barré syndrome presenting as isolated abducent nerve palsy has been reported.[4]

Reason of ophthalmoplegia has been debated in GBS but it seems to be due to minor changes in intraorbital pressure leading to change in eye position.[5] Ophthalmoplegia is a consistent feature of Miller Fisher syndrome which is characterised by ataxia and positive anti Gq 1b antibody. Rarely myasthenia gravis and GBS have been described in the same patient.

Autoimmunity plays an important role in both diseases due to molecular mimicry between infectious agent and self antigen.[6] Common infectious agent producing cross-reacting antibodies against myelin of peripheral nerve and AChR are considered to be the cause in this condition. But in our patient negative Tensilon test and normal RNS ruled out the possibility of neuromuscular junction involvement. Cases with unilateral internal ophthalmoplegia without external ophthalmoplegia have also been described.[7]

Author information: Sandjay Pandey, Manmohan Mehndiratta, Department of Neurology, GP Pant Hospital, Delhi, India

Correspondence: Dr Sandjay Pandey, Department of Neurology, RN. 507, GB Pant Hospital, Delhi, India. Email: sanjaysgpgi2002@yahoo.co.in

References:

1. Ropper AH. Unusual clinical variants and signs in Guillain-Barré syndrome. Arch Neurol. 1986 Nov; 43(11):1150-2.

2. Castaigne P, Brunet P, Nouailhat F. Clinical investigation of inflammatory polyradiculoneuritis in France. Rev Neurol (Paris). 1966 Oct;115(4):849-72.

3. Nagashima T, Koga M, Odaka M, Hirata K, Yuki N. Continuous spectrum of pharyngeal-cervical-brachial variant of Guillain-Barré syndrome. Arch Neurol. 2007 Oct;64(10):1519-23.

4. Tatsumoto M, Odaka M, Hirata K, Yuki N. Isolated abducens nerve palsy as a regional variant of Guillain-Barré syndrome. J Neurol Sci. 2006 Apr 15;243(1-2):35-8.

5. Jampel RS, Haidt SJ. Bell's phenomenon and acute idiopathic polyneuritis. Am J Ophthalmol. 1972 Jul;74(1):145-53.

6. Kizilay F, Ryan HF Jr, Oh SJ. Myasthenia gravis and Guillain-Barré syndrome occurring simultaneously in the same patient. Muscle Nerve. 2008 Apr;37(4):544-6.

7. Hakamada S, Watanabe K, Hara K and et al. Guillain-Barré syndrome presenting with signs of ophthalmoplegia. Brain Dev. 1980;2(4):407-9.

Worms, not germs

Christopher J Hopkins, Vinu M Abraham

Abstract

We present a case of a recent immigrant from India with 8 weeks of respiratory symptoms, eosinophilia, and diffuse reticulonodular opacity on chest X-ray. Further investigation revealed the cause to be tropical filarial pulmonary eosinophilia, for which he was successfully treated. We discuss the clinical features, investigation, and aetiology of this condition, which should be considered in patients from endemic areas.

We present a case of tropical filarial pulmonary eosinophilia.

Case report

A 27-year-old male, originally from India, was referred by his general practitioner to the Internal Medicine service with an 8-week duration of cough, producing occasional yellow sputum in the mornings. This was associated with chest pains, dyspnoea and wheezing. He had lost weight, but denied fevers or night sweats. Several courses of antibiotics were ineffective.

He denied any past medical history, in particular he had never suffered from asthma nor any other respiratory illnesses. He denied previous tuberculosis or known exposure. He was not taking regular medications.

He had been in New Zealand for 6 months. An immigration screening chest radiograph showed no evidence of pulmonary tuberculosis. He was a light smoker.

Physical examination was normal, with no lymphadenopathy, chest signs, hepato-splenomegaly or skin changes. Initial investigations identified elevated white blood cell count with a marked eosinophilia at 8.6×10^9/L. C-reactive protein was mildly elevated at 11 mg/L, and his electrolyte, renal and liver profile were all normal. The chest radiograph on presentation is shown in Figure 1.

Due to the suspicion of pulmonary tuberculosis, he was admitted into respiratory isolation. Sputum microscopy revealed eosinophils, but no acid-fast bacilli. Routine and mycobacterial cultures were negative. Serum IgE levels were grossly elevated at 2779 kU/L (normal range <100 kU/L). *Aspergillus* precipitins, *Aspergillus*-specific IgE enzyme allergosorbent testing, and serum anti-neutrophil cytoplasmic antibody were negative**.** Morning cortisol was within normal range. Stool microscopy was negative for ova, cysts and parasites. A peak flow diary did not demonstrate a diurnal pattern of bronchospasm. He was unable to perform lung function tests.

Serum Quantiferon Gold assay for *Mycobacterium tuberculosis* was "Positive". He underwent a high-resolution CT scan of the lungs, shown in Figure 2.

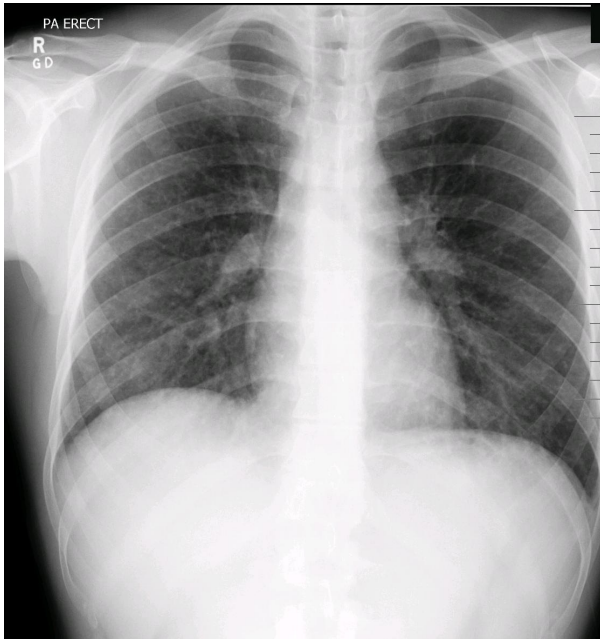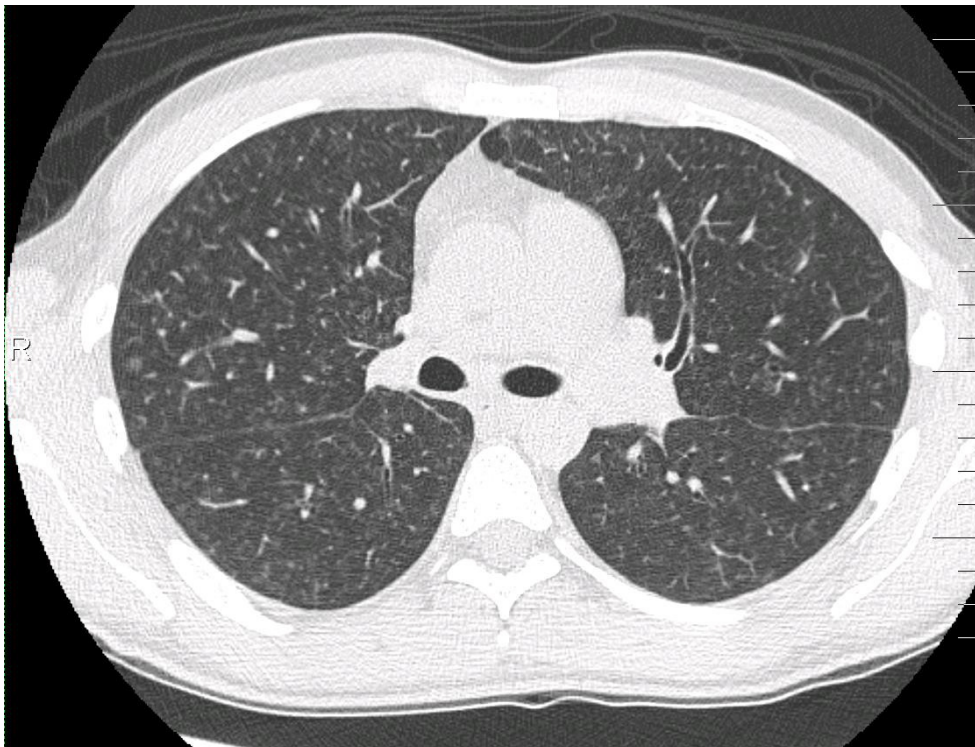**Figure 1. Chest radiograph – diffuse reticulonodular opacity**

Figure 2. High resolution CT scan of the chest – widespread miliary pattern of small nodules

Radiology reported a suspicion of miliary tuberculosis. On bronchoscopy the macroscopic appearance of the airways was normal, and multiple broncho-alveolar lavage samples were sent for analysis. These demonstrated 90% eosinophils, as shown in Figure 3. The samples were negative for acid-fast bacilli, parasites and malignant cells.

Figure 3. Cytology preparation of broncho-alveolar lavage – demonstrating 90% eosinophils
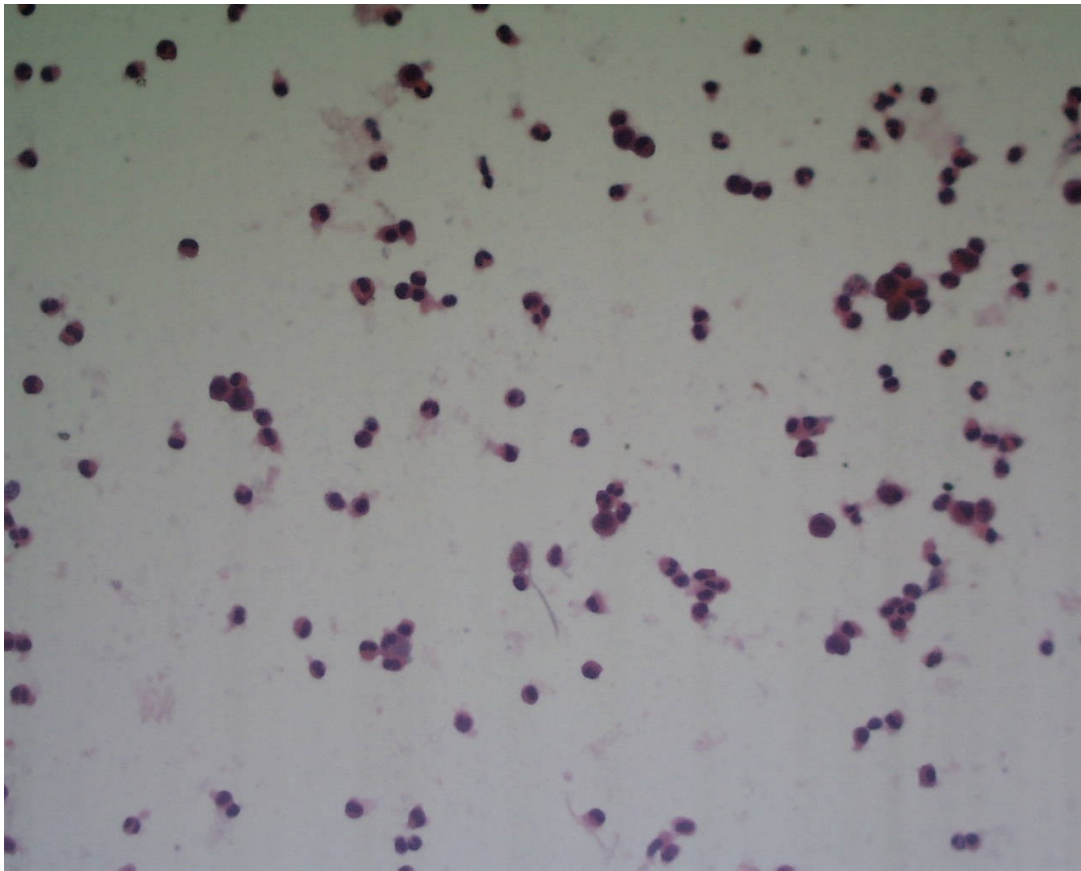
Serology for *Filaria* species was positive, with a signal-to-cutoff ratio of 10.0 (greater than 3.2 is considered positive). He received 2 mg/kg diethylcarbamazine thrice daily for 14 days, and on follow-up he reported a complete resolution of all symptoms. A repeat chest radiograph showed marked improvement. The diagnosis of *tropical filarial pulmonary eosinophilia* (TFPE) was therefore confirmed.

Discussion

TFPE is a clinical syndrome of paroxysmal non-productive cough, wheeze, occasionally weight loss, lymphadenopathy and low-grade fevers in association with

marked peripheral eosinophilia. Key laboratory features include markedly elevated serum IgE, high titre anti-filarial antibodies, and intense eosinophilia on broncho-alveolar lavage. Chest imaging usually reveals fine miliary lesions and increased bronchovascular markings. Lung function tests reveal a mixed pattern, and reversibility with bronchodilators.[1]

The causative organisms are the mosquito-transmitted filarial helminth species, *Wuchereria bancrofti*, *Brugia malayi* or *Brugia timori*. These have overlapping distributions, and collectively they are endemic in most tropical regions. They cause lymphatic filariasis, which has numerous clinical manifestations, rarely including TFPE. The syndrome is characterised by a hypersensitivity response to the blood-borne micro-filariae, which become opsonised and deposited in lung and reticulo-endothelial tissues. Pulmonary interstitial infiltrates result, eventually causing pulmonary fibrosis if untreated.[2]

Patients from tropical countries presenting with respiratory symptoms are often appropriately investigated for a suspicion of pulmonary tuberculosis. However, in this case the marked eosinophilia prompted a broader differential diagnosis, and investigations that led to the diagnosis and successful treatment of tropical filarial pulmonary eosinophilia.

Author information: Christopher J Hopkins, Medical Registrar, Department of Medicine, Taranaki Base Hospital, New Plymouth; Vinu M Abraham, Consultant Physician (Internal Medicine/Pulmonologist), Department of Medicine, Taranaki Base Hospital, New Plymouth

Correspondence: Dr Chris Hopkins, Department of Medicine, Taranaki Base Hospital, David Street, Private Bag 2016, New Plymouth 4620, New Zealand. Fax: +64 (0)6 7537721; email: Christopher.Hopkins@tdhb.org.nz

References:

1. Weller PF, Leder K. Tropical filarial pulmonary eosinophilia. In: UpToDate. v19.3 [cited 2011 Dec 8]. http://www.uptodate.com
2. Cottin V, Cordier J. Eosinophilic lung diseases. In: Mason RJ, editor. Murray and Nadel's Textbook of Respiratory Medicine. 5th ed. St Louis: Saunders; 2010. p. 1469-95.

Red eyes and red ears

Bipul Baibhav, Satya Kurada

Clinical—A 71-year-old woman presented with progressive redness, pain and swelling of both eyes and ears along with polyarthragias of 1 week duration. A month prior she had transient hearing loss. Physical examination revealed swollen erythematous pinnae with sparing of the lobules (Figures 1 and 2). Eye exam showed bilateral diffuse scleritis and uveitis (Figure 3).
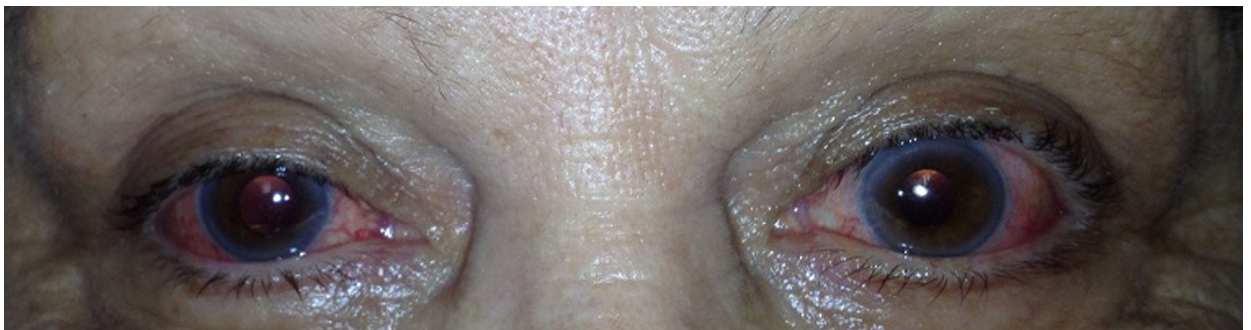
Figure 1. Swollen erythematous pinna with sparing of lobule

Figure 2. Swollen erythematous pinna with sparing of lobule

Figure 3. Bilateral diffuse scleritis and uveitis

Laboratory data revealed an erythrocyte sedimentation rate (ESR) of 67 mm/hr, C-reactive protein (CRP) of 129.6 mg/L and an anti cyclic citrullinated peptide (anti–CCP) of 60 units. A clinical diagnosis of *relapsing polychondritis* (RPC) was made. There was marked improvement in her symptoms in less than 24 hours after initiating steroids. Three months later she remains asymptomatic on steroids.

Discussion—RPC is a rare autoimmune disorder affecting primarily cartilaginous structures but can involve other tissues which have biochemical or immunological similarity. Eyes, blood vessels and the heart can be involved, all of which are also rich in proteoglycans. RPC can also involve the inner ear and cause hearing loss.

Markers of inflammation such as ESR and CRP are commonly elevated in RPC. RPC can be associated with other autoimmune disorders such as rheumatoid arthritis, systemic lupus erythematosus and Sjogren's syndrome.

The level of anti-CCP was elevated in our patient, which is very specific for rheumatoid arthritis. This case illustrates that RPC may signal the onset of rheumatoid arthritis in an elderly patient.

Author information: Bipul Baibhav, Attending Physician; Satya Kurada, Physician; Department of Medicine, Rochester General Hospital, Rochester, NY, USA

Correspondence: Bipul Baibhav, MD, Department of Medicine, Rochester General Hospital, 1425 Portland Ave, Rochester NY 14621, USA. Fax: +1 (0)585 9222908; email: bipul.baibhav@rochestergeneral.org

WHO's "Clean Care is Safer Care" campaign: why hasn't New Zealand joined?

More than 165 years after the Hungarian physician Dr Ignaz Semmelweis observed that simply washing hands could drastically reduce high rates of maternal death during childbirth, half of practitioners still do not regularly wash their hands before seeing patients.[1] Healthcare-associated infections remain a major global issue for patient safety, with hundreds of millions of patients around the world affected each year. The prevention of healthcare-associated infections was chosen by WHO Patient Safety as the theme of its 1st Global Patient Safety Challenge "Clean Care is Safer Care" launched in October 2005.[2]

The International Society for Quality in Health Care's annual conference, ISQua, was held this year in Geneva on 22-24 October 2012, with over a 1000 delegates from 70 countries around the world. Professor Didier Pittet, Director of Infection Control at the University of Geneva Hospitals and External Programme Lead of the WHO's "Clean Care is Safer Care" campaign, demonstrated on a world map how 129 WHO countries or areas have made formal statements pledging their support to implement actions to reduce health care-associated infection within their countries and to share results and learning internationally.[3]

It came as a great to surprise to see that New Zealand was one of only a hand full of countries that have not signed this pledge. Indeed, I was so surprised I wrote to Hand Hygiene New Zealand,[4] a collaboration between the Auckland District Health Board and the Health Quality & Safety Commission, to confirm and ask why New Zealand had not joined. I was advised that the matter had been raised with the Ministry of Health but that little progress was being made in getting the Minster of Health to sign the WHO pledge.

The Hand Hygiene New Zealand programme is an important measure, and as Sally Roberts and colleagues demonstrated in their May 2012 *NZMJ* article,[5] some progress is being made in improving compliance with hand hygiene. However, it is clear further efforts are required. By not signing the WHO pledge and supporting this international initiative to reduce health care-associated infections and improve patient care, the Minster of Health is sending a negative message to healthcare workers and the public of New Zealand. The Minster should be seen to be supporting quality initiatives in healthcare by showing strong leadership from the top.

Stuart McLennan
Research Associate
Institute for Biomedical Ethics, University of Basel
Basel, Switzerland
s.mclennan@unibas.ch

References:

1. Institute of Medicine. Best Care at Lower Cost: The Path to Continuously Learning Health Care in America. Washington, DC: The National Academies Press, 2012.
2. World Health Organisation. Clean Care is Safer Care. http://www.who.int/gpsc/country_work/en/. Accessed 25 October 2012.
3. World Health Organisation. Clean Care is Safer Care: Support from countries or areas worldwide - Pledges to combat health care-associated infections. http://www.who.int/gpsc/statements/en/index.html. Accessed 25 October 2012.
4. Hand Hygiene New Zealand. http://www.handhygiene.org.nz/. Accessed 25 October 2012.
5. Roberts SA, Sieczkowski C, Campbell T, Balla G, Keenan A. Implementing and sustaining a hand hygiene culture change programme at Auckland District Health Board. NZMJ, 125(1354); http://journal.nzma.org.nz/journal/125-1354/5181/. Accessed 25 October 2012.

Obituary, Dr Closs

Published in NZMJ 1912

Dr. Closs, who died at Featherston on Monday, February 5th, 1912, left Dunedin to spend his usual holiday with Mr. Tringham, the well-known Wairarapa station holder.

He was suddenly seized by gastro-enteritis and quickly succumbed. His wife learned of his serious illness only on Monday morning, and left Dunedin by the second express, too late to be with her husband before the end. The deceased was born in Glasgow about 62 years ago, and was brought to New Zealand at an early age. He was educated at the University of Otago, and in Edinburgh, where he graduated M.B., Ch.M., and some years later took his degree of M.D. He was for many years a lecturer on clinical surgery in the medical school, and was till the date of his death an honorary surgeon to the Dunedin Hospital. He was generally acclaimed a most skilful surgeon and physician of experience and ripe judgment.

Outside the practice of his profession he was a man of many activities; was Deputy District Grand Master of the Grand Lodge of Otago and Southland (English Constitution) from 1904 to 1907, and formerly president of the Otago Lawn Tennis Club.

At one time he took all active interest in racing, and his horse "Hero" won the Hunt Club Cup at the Hunt Club meeting at Forbury. The deceased gentleman leaves a widow but no children. He has only one brother alive, the Rev. Mr. Closs, of Christchurch.

Are shift workers at greater risk of vascular events?

Disruption of circadian rhythm may predispose shift workers to vascular events. This systematic review from Canada attempts to clarify this hypothesis by analysing data from 34 studies. Their results indicate that shift work was associated with myocardial infarction (risk ratio 1.23) and an increased risk for coronary events (risk ratio 1.24). The relative risk for ischaemic stroke was 1.05. However, shift work was not associated with an increased rate of mortality, whether vascular cause specific or overall. Although the relative rates are modest they note that on a population basis they are important. On the basis of the Canadian prevalence of shift work of 32.8%, the population attributable risks were 7.0% for myocardial infarction, 7.3% for all coronary events, and 1.6% for ischaemic stroke.

BMJ 2012;345:e4800.

Parkinson's disease – does physiotherapy help?

The question addressed in this study is how effective is physiotherapy in treating patients with Parkinson's disease? Results from 29 trials were reviewed. The reviewer's definition of physiotherapy interventions included general physiotherapy, exercise, treadmill training, cueing, dance, and martial arts. The trials reviewed were relatively small, and most assessed the effect of physiotherapy intervention versus no intervention over a short period of time (<3 months). The results were that compared with no intervention, physiotherapy has short term benefits in Parkinson's disease. However, the reviewers point out that in a long term disease such as Parkinson's disease, future research should investigate the effect of therapy over a much longer period.

BMJ 2012;344:e5004.

Hyponatraemia related to mortality in patients with heart failure

Hyponatraemia has been associated with reduced survival in patients with heart failure and reduced ejection fraction. This meta-analysis sets out to define whether hyponatraemia also increases mortality in those with a preserved ejection fraction (≥50%). Twenty two studies including 14,766 patients were reviewed. 1618 of the patients were hyponatraemic (serum sodium <135mmol/L) and they fared significantly worse in terms of mortality compared with those who were not hyponatraemic. At 3 year follow-up 21% of the hyponatraemic cohort had died whereas only 16% of those with a normal sodium had succumbed. The authors conclude that further work is needed to determine if correction of hyponatraemia translates into clinical benefits.

European Journal of Heart Failure 2012;14:1139-46.

C-reactive protein, fibrinogen, and cardiovascular disease prediction

The authors of this review point out that there is debate about the value of assessing levels of C-reactive protein (CRP) and other biomarkers of inflammation for the prediction of first cardiovascular events. They have analysed data from 52 prospective studies that included 246,669 participants without a history of cardiovascular disease to investigate the value of adding CRP or fibrinogen levels to conventional risk factors for the prediction of cardiovascular risk. Patients who had previously had a myocardial infarction, angina or a stroke were excluded. Data concerning conventional cardiovascular risk factors - age, sex, smoking status, diabetes, systolic blood pressure, levels of total and high density lipoprotein cholesterol were available as were baseline levels of CRP, fibrinogen (or both). The authors report that information on CRP and fibrinogen was valuable and significantly improved the predictive value of the conventional risk factors. Assuming that appropriate treatments (eg statins) would be used they predict that over 10 years one cardiovascular event for every 400-500 patients screened might be prevented.

N Engl J Med 2012;367:1310-20.

Radiofrequency ablation as initial therapy in paroxysmal atrial fibrillation

The aim of this trial was to compare the long-term efficacy of an initial strategy of radiofrequency catheter ablation with an initial strategy of antiarrhythmic drug therapy in a larger population of patients with paroxysmal atrial fibrillation. They randomly assigned 294 patients with paroxysmal atrial fibrillation and no history of antiarrhythmic drug use to an initial treatment strategy of either radiofrequency catheter ablation (146 patients) or therapy with class IC or class III antiarrhythmic agents (148 patients). At 3,6,12,18 and 24 months after the initial treatment patients were reviewed by 7 day Holter-monitor recordings. The authors report no significant difference between the treatment groups in the cumulative burden of atrial fibrillation over a period of 2 years. Three cases of cardiac tamponade occurred in the ablation group and there was one death from a procedure related stroke in the ablation group.

N Engl J Med 2012;367:1587-95.

Robert Bohdan Mikolaj (Bob) Ravich

MBChB (Otago), FRACP; 1937–2012

Bob Ravich, general physician and clinical haematologist, practised and taught medicine at the highest level. He integrated a prodigious knowledge of human anatomy, physiology, psychology and empathy with current and breaking medical science, practising the 'art' of medicine with discernment and perspicacity. Proficient in lateral thinking, he became the doctors' doctor; the person to whom other doctors referred difficult diagnostic problems.

To the majority of patients he became a friend; a trusted partner who accompanied them on their medical journeys giving them the confidence that he knew where they were going and would travel with them whether the outcome was cure, remission or death.

It was not unusual for him to sit with a patient in their last hours to calm them in their final transition.

Although academia was not his passion, he lectured, tutored, examined and mentored many students throughout his career and was in his element when teaching the 'art' of medicine—active listening, careful observation, eliciting confirmatory physical signs. He inspired many, imbuing them with a sense of purpose and excellence in practice that contributes significantly to his medical legacy.

Born in Gdansk, Poland in 1937, he and his mother escaped in 1939, travelling through the incoming German army to eventually reach the comparative safety of England. He finally met his father at the age of 8 on the Glasgow Railway Station.

The family emigrated to New Zealand post WWII and Bob graduated in Medicine, MBChB (Otago) in 1962. He undertook his residency training in Christchurch Hospital, New Zealand, staying on as Senior Medical Registrar in 1966 and Senior Registrar in Haematology in 1967.

Bob moved to Sydney with his wife Chris and their young family in 1968 when he was appointed Senior Research Fellow in Haematology at Sydney Hospital and Honorary/Visiting Physician in 1970. He became a Fellow of the Royal Australasian College of Physicians in 1972.

Bob established a Private Practice as a Consultant Physician in Clinical Haematology/General Medicine in Macquarie Street in 1971, despite warnings from some colleagues that, as he was not a graduate of Sydney University, this was unlikely to succeed. It did.

He moved the Practice to St Leonards in 1978 when he was appointed Visiting Medical Officer (VMO) at the then brand new Royal North Shore Hospital and

eventually to Crows Nest in 2004 to be closer to the Mater Hospital. On his retirement in 2006 he sold the Practice to colleagues as a productive and dynamic business with an ethos of excellence in patient care and the art of medicine.

Although a professed atheist, Bob shared a focus on compassionate patient care with the Sisters of Mercy, his relationship with the Mater Hospital growing over time and ranging from his initial appointment in 1972 as Honorary/Visiting Physician in Clinical Haematology/Oncology and General Medicine, to serving as a Director on the Board from 1993-2001.

In 1994 he conceived and founded the Department of Cancer Medicine at the Mater, chairing it until he stepped down in 2001. This Department was structured to provide total and multidisciplinary care to the patient with cancer, from the time of diagnosis to cure or death.

Without Bob's vision and drive the Mater would not be the centre of excellence in cancer care that it is today. The existence of the Department of Cancer Medicine and the Chemotherapy Cottage where it was initially centred, laid the foundation for the future development of the Patricia Ritchie Centre for Cancer Care and Research and the establishment of the Poche Centre to house the Melanoma Institute of Australia.

In the latter years of his life, Bob had the opportunity to guide the distribution of significant financial donations to both medicine and conservation. He undertook this with the same deliberation and attention to detail that he applied to his own medical work, directing funds to specific projects within organisations such as the NSW Leukaemia Foundation, the Institute for Emerging Infections and Biosecurity at the University of Sydney, and BirdLife Australia.

Bob was an insatiably curious, cultivated and creative person, more inclined to lead from behind than from the front; to encourage others and eschew the limelight—and all was permeated with a robust sense of humour and of the ridiculous. Outside medicine he had a broad range of diverse interests. He read widely—everything from Bulfinch's Mythology to Latham's Quarterly—assembling an extensive library of Science Fiction along the way. His great love of music resulted in another library described by his daughter as 'iTunes before its time'.

His love and respect for nature, shared with his wife Kate, led them to purchase a newly covenanted Private Forest Reserve on King Island, Tasmania, in 2004, in part as a contribution towards the preservation of biodiversity. His love of and fascination with language inspired him to learn a new word everyday—something he continued up until the end. He was the ultimate pragmatist with a number of his own dictums that he lived by: "If a thing is worth doing, do it to excess" encapsulated his passionate embrace of life and, most recently as he faced his own death, "Life without death is meaningless".

Bob died peacefully at his home in Sydney on 1 September 2012, surrounded by his family. He is survived by his wife Kate, children Stephen, Joanna and Katie and six grandchildren. As well as his family, his legacies embrace medicine, conservation and a positive influence on the lives of many.

Colleagues Prof Bruce Robinson, A/Prof Fran Boyle, and wife Kate Ravich wrote this obituary.

David Cranleigh Thomson Bush

MBChB, DA(Eng), FANZCA

David Bush died in Christchurch on 5 July 2011 aged 84 years.
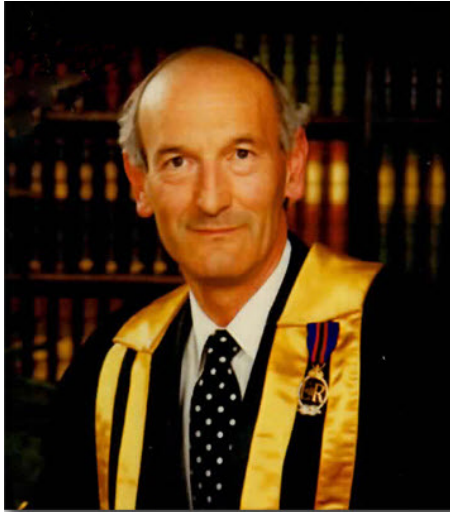
David was born in Wellington and grew up on a farm in the Awatere valley—an isolated area in the upper South Island, and as a boy always wanted to be a farmer.

However his father had other ambitions for him and sent him to secondary school at Christ's College Christchurch where he was a Prefect and gained his school colours for shooting.

He also did well enough academically to obtain a place at Otago University and then onto the medical school, graduating with his MBChB in1952.

Following graduation he did his house-surgeon years at Christchurch Hospital and then took the 6-week boat trip to England as the ship's doctor. In the UK he continued his anaesthesia training, obtaining the D.A. while working in Whittington Hospital.

He returned to Christchurch to finish his anaesthesia training and having obtained his FFARACS took up a post as a consultant anaesthetist. At this time he also met, and after a 3-week courtship proposed to, his wonderful wife Nan.

David's initial interests included paediatric anaesthesia and he anaesthetised a lot of children with great skill in his early career.

He eventually dropped some sessions at Christchurch Hospital and developed a very busy private practice. His surgeons recall him as being very meticulous in his work—a great attribute for an anaesthetist-and very caring to his patients. He was also very willing to come in at all hours for emergencies.

David was also very highly regarded by the nursing staff at all the hospitals he worked in. He was forever the polite gentleman, always calling the nurses "Sister" never by their Christian name, and was known for his impeccable manners. He was also much liked for his habit of calling into the wards at the end of a busy day in theatre to check that all was well and offer to reinsert any IVs.

Although quite conservative by nature he did have quite an innovative side. When the IRD (tax department) decreed that only vans could be claimed as work vehicles he took the back seats out of his racey yellow Mitsubishi and turned it into a van! Early on in his private career he recognised that the backless theatre stools, which were the only seating provided in theatre at that time, were not good for the anaesthetist's

posture during long cases. To solve this problem, he installed the "Bush Chair of Anaesthesia" in the theatre he used most. This was a padded swivel chair with arms which made extended plastic surgery cases a lot more comfortable. On the clinical side the PACU nurses recall that he was one of the first Christchurch anaesthetists to prescribe IV rather than IM analgesia for his patients in the recovery unit. This was unusual at the time.

Another of David's contributions to anaesthesia in Christchurch was the encouragement of younger anaesthetists starting out in private practice. He was a great mentor and even arranged with his surgeons to hand over some of his lists to help them get started. He would also very generously put on a luncheon at his local restaurant for our annual meeting.

Away from the operating theatre, David had a wide range of interests. Encouraged by his school friend, radiologist Shailor Weston, he joined the Royal Naval Reserve where he served as a Medical officer for over 15 years, reaching the rank of Surgeon Lieutenant Commander and receiving the Volunteer Reserve Decoration for his work. His interest in things nautical also extended to his owning various boats which were used for family holidays on the Southern Lakes where he also headed for winter skiing. He was a keen golfer and bridge player as well as a model train enthusiast-using left over orthopaedic plaster-of-paris for his scenery.

Along with his all his hobbies and very busy work schedule David, in conjunction with his colleague Bill Pryor somehow managed to co-author the third and fourth editions of Bill's book "A Manual of Anaesthetic Techniques". These were published in the late sixties and early seventies and being a very practical tome, proved popular with the junior anaesthetic staff at that time. The fourth edition even managed a Spanish translation! David also contributed the chapter "Anaesthesia for Major Oral surgery " in Bill's other book on anaesthesia for dentistry.

Having worked incredibly hard over the years David retired in his early sixties determined to leave anaesthesia practice while still "on top of his game". This he certainly achieved. He had several years of very happy retirement with Nan who had not only looked after the family during David's very busy career but had also been his secretary-making herself constantly available to answer the phone to surgeons and patients. There were no practice rooms or cell phones in those times!

He was absolutely devastated when Nan was diagnosed with Motor Neurone Disease and when she predeceased him his health unfortunately deteriorated rather rapidly. However even in the rest-home, afflicted with the cruel symptoms of Alzheimer's, his hallmark impeccable manners never deserted him. He continued to greet family and friends, whom he no longer recognised, with a wonderful smile and ever polite greeting. Unfortunately his good manners did cause occasional problems. When David was moving around the home Zimmer frame pile-ups were a common problem with David always insisting that the ladies went through the doorways first!

David will always be remembered as a very principled man, a true gentleman, a skilled anaesthetist who gave great care to a huge number of patients and great service to his surgeons, a very generous colleague and a very devoted husband, father and grandfather—greatly missed.

Dr Peter Pryor of Christchurch provided this obituary.