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This Issue in the Journal

Tuberculosis epidemiology in New Zealand: 1995–2004
D Das, M Baker, L Calder

The epidemiology of tuberculosis in New Zealand is described, focusing on recent years. The steady decline in tuberculosis incidence in the four decades following the Second World War was halted in the mid-1980s. For the subsequent 20 years, the annual rate has been stable at around 10 cases per 100,000 people. About two-thirds of cases in New Zealand were born overseas. There is marked inequality in the incidence rate by ethnicity. Europeans have the lowest rate, while people of ‘Other’ ethnicity (Asian and non-Māori, non-Pacific, and non-European combined) the highest. Rates in Māori and Pacific people are in between. Tuberculosis outbreaks do occur occasionally and predominantly affect Māori and Pacific people. Tuberculosis mortality and case fatality rates are declining, presumably due to improved case detection and treatment. The paper dispels the myth (partly perpetuated by media coverage of occasional outbreaks) that tuberculosis incidence is increasing in New Zealand.

Why the tuberculosis incidence rate is not falling in New Zealand
D Das, M Baker, K Venugopal, S McAllister

This paper explores the reasons for the non-decline of the tuberculosis incidence rate in New Zealand in recent years. It shows that the main cause is the migration of tuberculosis-infected people to New Zealand from high-incidence countries and subsequent development of active disease in some of these people. The paper also shows that migrants are not spreading the disease locally to any significant degree. HIV/AIDS, multi-drug-resistant organisms, and animal tuberculosis are not significant contributors of (human) tuberculosis in New Zealand. Tuberculosis remains poorly controlled in Pacific and Māori communities. Further reductions in tuberculosis in New Zealand will depend on global and regional tuberculosis control; effective screening and follow-up of immigrants; rapid diagnosis and treatment of cases within New Zealand; and continuing efforts to reduce health disparities through such measures as better housing.

An outbreak of Legionella pneumophila suspected to be associated with spa pools on display at a retail store in New Zealand
Q Ruscoe, S Hill, T Blackmore, M McLean

A cluster of six cases of severe pneumonia in the Wellington region is described. Investigation involved epidemiological, environmental, and microbiological analyses. Three cases were confirmed as, or compatible with, Legionella pneumophila serogroup 2 infection. Exposure histories revealed that these three cases had visited the same store that was operating spa pools on display. Legionella pneumophila serogroup 1 was cultured from one of three pools. The pools were considered to be
the most likely source of infection in view of inadequate water chlorination. Public health risk intervention is described in what is the second known outbreak of Legionnaires’ Disease linked to operating spa pools on retail display in New Zealand; the first outbreak occurring in Auckland in 2002 in which one person died.

An Auckland regional audit of the nurse-led rheumatic fever secondary prophylaxis programme
S Grayson, M Horsburgh, D Lennon

The World Health Organization (WHO) recommends the use of regular benzathine penicillin injections (secondary prevention) as the first step towards preventing recurrent attacks of rheumatic fever. The Auckland Rheumatic Fever Register generates benzathine penicillin prescriptions for patients with rheumatic fever—and registered nurses (working from community nursing offices) deliver the 3-monthly prescriptions. Our study shows that these nursing services within the three Auckland DHBs achieve high levels of compliance with rheumatic fever secondary prevention. The nurses are flexible, innovative in tracking patients, and experienced in delivering a community-based programme—sometimes with the help of community health workers who are particularly valuable in engaging Māori and Pacific families where the highest incidence of rheumatic fever is found.

Influenza immunisation rate for 2005 and factors associated with receiving this vaccine in patients aged 65 years and over admitted to a general medical ward at Auckland City Hospital
E Curry, N Kerr, J Yang, S Briggs

Influenza immunisation results in a significant reduction in cases of influenza. This study found that 74% of patients admitted to a medical ward at Auckland City Hospital had received the influenza immunisation in 2005. We were not able to identify patient groups that had a low influenza immunisation rate. Reminding patients of the benefits of the influenza vaccine or offering this at the time of discharge from hospital at appropriate times of the year may increase the influenza immunisation rate of those recently hospitalised.
Tuberculosis in New Zealand: poverty casts a long shadow

Mark Thomas, Rod Ellis-Pegler

In an era concerned with the real or imagined threats from epidemics of “new” infectious diseases such as AIDS, SARS, and pandemic influenza, it is easy to forget the continued burden of disease caused by that “old” infectious disease, tuberculosis.

The white plague, as it was known in the past, killed famous names from this part of the world too. Robert Louis Stevenson (1894), Hone Heke (1909), and Katherine Mansfield (1923) are just some of the more notable examples.

In this issue of the New Zealand Medical Journal, Michael Baker and his colleagues remind us, in two articles,1,2 that tuberculosis has not gone away but instead continues to cause disease and death—disproportionately affecting the poor and recent immigrants.

These epidemiologic associations are not new. At the end of the nineteenth century, New Zealand was a favoured destination for British men with consumption, hopeful that a change in climate would improve their chances of survival. At that time, tuberculosis was the leading cause of death in the colony and each year killed approximately 100 of the 100,000 residents of Auckland.3

The migration to New Zealand of Europeans with tuberculosis during the nineteenth and early twentieth centuries has been followed by subsequent waves of migrants with high rates of tuberculous infection; from the Pacific Islands in the 1960s and 1970s, South East Asia in the 1980s, and Africa in recent years. The inevitable result of the introduction of tuberculosis to New Zealand by the early European settlers was transmission of the disease to Māori, followed by high rates of disease and death in Māori in particular.

Tuberculosis has always been a disease of poverty. Death rates due to tuberculosis in Māori men were 10 times those of non-Māori men as recently as the 1970s and 1980s,4 and the incidence of tuberculosis in Māori during the last decades remains approximately 10 times that in Europeans.1

The recent resurgence of tuberculosis in New Zealand, fuelled by immigrants from third world countries with high rates of infection,2 has also occurred in most other wealthy countries. In some countries, most notably the USA where the public health system and laboratory susceptibility testing were allowed to atrophy in the latter part of the 20th century, intersecting epidemics of tuberculosis and HIV infection led to explosive outbreaks of multi-drug resistant tuberculosis (MDR TB) (defined as resistance to at least rifampicin and isoniazid) in the most deprived members of society, such as prison inmates and homeless people.5

Fortunately this has not occurred in New Zealand. Overlapping systems based on clinician and laboratory reporting of tuberculosis have ensured relatively high notification rates. Reference laboratories in Auckland, Hamilton, and Wellington test the susceptibility of all Mycobacterium tuberculosis isolates in New Zealand.
Initial diagnosis and antimicrobial selection is predominantly in the hands of chest and infectious disease physicians, and a strong public health service has provided an effective supervision of outpatient treatments with anti tuberculous medicines. As a result, there has been only one case of locally generated MDR TB, and overall MDR TB constitute only 0.6% of all isolates. Nevertheless, MDR TB were causative (in 2005) in 2.0% of overseas-born cases.

Even more serious are the recent reports of extensive (the official term has been decreed “extensive” rather than “extremely”) drug-resistant M. tuberculosis isolates (XDR TB); bacteria which are not only MDR TB but also have resistance to at least three of the second line agents, viz. aminoglycosides, polypeptides, fluoroquinolones, thioamides, cycloserine, and para-aminosalicylic acid.

XDR TB have been found throughout most of the world, and in a recent evaluation constituted 4%, 19%, and 15% of MDR TB in the USA, Latvia, and South Korea respectively. Patients infected with such variants of tuberculosis do badly.

The recent report from Kwazulu-Natal in South Africa of an outbreak of 53 patients with XDR TB and (at least in the 44 tested) HIV infection—52 of whom died within an average of 25 days—has brought a brutal immediacy to these issues. Once again, intercepting epidemics of tuberculosis and HIV infection with appalling outcomes.

It is gently reassuring that there is no evidence of an important contribution to tuberculosis in New Zealand from those coinfected with HIV, but our clinical impression from Auckland, where we treat a large cohort of those with HIV infection, is that that may not continue to be true of 2005 and 2006 once evidence is available for those years.

While outbreaks of tuberculosis in New Zealand usually involve an average of 10 persons and almost exclusively occur in Pacific or Māori communities, the recent still-evolving epidemic in Palmerston North is atypical. There, about 1800 boys from Palmerston North Boys High School have been evaluated after the diagnosis of infectious tuberculosis in an immigrant boy.

Initial reports indicate 194 Mantoux-positive boys (i.e. ca. 11% infected), with 12 of them diagnosed as probable tuberculous disease and being treated. The remaining 182 Mantoux-positive boys will receive chemoprophylaxis. These are both very high infection and disease rates so there can be no rest from endless vigilance.

Improved control of tuberculosis in New Zealand will result from enhanced detection of infection in immigrants and a nationwide reduction in poverty. The new immigration protocols introduced late last year, which require stricter migrant and visitor health screening as Das et al outline, should produce a modest immediate reduction in the burden of disease.

Given the increasing risk of reactivation of latent tuberculous infection with increasing age, it will be decades before the full benefits will accrue, however. Indeed, we can expect continued tuberculous disease in recently arrived immigrants as they age over the next 30 to 40 years.

Finally, the doctor caring for a recent immigrant, Pacific, or Māori patient with fever, respiratory symptoms, and/or weight loss should remember the high rate of tuberculosis in such patients. Failing to enquire about past exposure to tuberculosis, ascribing illness to other causes of lung disease (most commonly pneumonia and
asthma), and failing to request a chest X-ray, were the most significant causes of delay in diagnosing patients with pulmonary tuberculosis in Auckland during the late 1990s.\textsuperscript{12}

Beware the recent Asian immigrant with fever and chronic illness or even the recent Asian immigrant who is afebrile and has just a single persistently enlarged cervical lymph node!

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HIV prevention in New Zealand—still room for improvement

Nigel Dickson, Oliver Davidson

When AIDS first appeared among men who have sex with men (MSM) in New Zealand in the 1980s, a comprehensive approach to prevention was initiated from within the gay community.¹ This not only informed MSM of the risks of HIV—and how to reduce them—but also successfully advocated for homosexual law reform that resulted in a more tolerant environment. These activities were supported by the Department of Health that also undertook its own campaign aimed at the wider population, and initiated changes in the law to allow the distribution of clean needles and syringes for injecting drug users.

These approaches showed appreciation that while individual behaviours are key to the spread of HIV, the right social and structural environment is necessary to enable an effective response. The actions were successful; in the early 1990s, the number of people being diagnosed with HIV was declining, and New Zealand was one of the first countries to experience a levelling of AIDS cases.² The latter occurred before the new era of treatment of HIV that started in 1996, when it became clear that appropriate antiretroviral therapy could have a major beneficial effect on the progression of HIV.

However, in 2005, more people were diagnosed with HIV in New Zealand than in any previous year, an extension of the trend noted since 2000. Indeed, from 2000 to 2005, the number of people diagnosed with HIV doubled.³ The increase was fuelled not only by more infections among MSM, but also infections among heterosexual men and women.

There is an important difference between the MSM and heterosexual groups with HIV. Nearly three-quarters of the MSM diagnosed in 2005 were infected in New Zealand, and most of the increase since 2000 was made up of infections acquired here. In contrast, most (88%) heterosexuals diagnosed in 2005 were infected overseas, and the recent rise is predominantly made up of people infected outside New Zealand.³

New Zealand is not alone in experiencing a rise in infections among MSM. In Western Europe, the number of diagnoses in this group increased between 2000 and 2004 by 45%.⁴ There have also been increases in the US and Australia.

In many countries, behaviour surveillance has shown that from the mid-1990s until recently, high-risk sexual behaviour among MSM increased.⁵ Internationally, while some have argued that there has been some relaxation in the culture of safer sex among MSM because of optimism about treatment,⁶ this alone does not appear to explain all the behaviour change.⁵ Two of the other factors that may have impacted on this increase over the past decade are more use of the Internet to find sexual partners, and of recreational drugs such as methamphetamine, that reduce inhibitions and increase sexual desire.⁷
While increases in sexual risk taking may well have played some part in the increasing number of new HIV infection among MSM in New Zealand, it is important to remember an underlying tenet of infectious disease epidemiology: prevalence drives incidence. Of particular relevance to the spread of HIV is that people are more infectious soon after infection, so an increase in the prevalence of recent infections has the potential to accelerate a further rise. Thanks to the new treatments, people are living longer with HIV, rather than dying of AIDS. Although this will impact on prevalence, and hence incidence, it must also be remembered that being on treatment makes people less infectious.

Other sexually tract infections increase the risk of HIV transmission. If the increase in people attending sexual health clinics with sexually transmitted infections reflects a rise among MSM, this may have contributed to HIV more spread in this group. In 2003, the Ministry of Health’s HIV/AIDS Action Plan was published. The continuing increase in HIV diagnoses makes it imperative that the implementation of this be examined, and action expedited if it has not occurred. In addition, consideration of new initiatives may be required.

The Action Plan highlighted the importance of societal attitudes to HIV, and people at risk, which are known to impact on the potential success of prevention strategies. Major efforts need to be made to ensure people at risk of HIV—whether infected or not—are not subject to stigma and discrimination. Where such barriers exist, uptake of measures needed to control HIV spread is inhibited. As one commentator put it, HIV should now be considered a “normal misfortune.”

Progress in de-stigmatisation, as promised in the Action Plan, should be reviewed in the light of research on how to monitor and manage this problem. In New Zealand, new migrant populations are particularly vulnerable, and some have been particularly affected by HIV. As well as addressing this issue, it is important to involve these groups in HIV prevention planning and delivery, as was investigated in the Mayisha Project in the UK.

We believe that the New Zealand control strategy should be more explicit about the importance of professionally delivered HIV testing. There are many good reasons why HIV should be diagnosed as soon after infection as possible. Diagnosis allows infected people not only to benefit from early effective care, but also reduces the risk of others being infected. Indeed, those diagnosed with HIV tend to reduce their risk behaviour, and treating individuals early reduces their infectivity. However the environment must be safe enough for people to be tested. The amnesty that was recently announced for Zimbabweans who came here before October 2004, which means that HIV infection will not now prevent them gaining New Zealand residency if this is sought soon, should be applauded.

A recent study confirmed that MSM most at risk are those having multiple partners, unprotected anal intercourse, and alcohol and drug use before sex. The results support the argument for promoting universal condom use during anal intercourse for MSM. They also show the need to acknowledge the role of multiple partners, because condoms will sometimes fail. We support the argument that reducing partner numbers should be given greater prominence in HIV prevention.
The New Zealand Primary Health Care Strategy emphasises prevention in primary care. General practice is where testing occurred most commonly among people diagnosed with HIV, and is likely to be where most testing takes place. It is therefore an important site for individualised health promotion. While many primary care practitioners may not yet be skilled as health promoters in this field, well designed education programmes can help staff address such sensitive issues. The newly formed primary health care organisations could have a crucial role in this area.

To conclude, there is an urgent need to reinforce HIV/AIDS prevention in New Zealand. While programmes should cover the entire range of people at risk, MSM continue to warrant specific consideration, as most new infections in New Zealand still occur in this group. However, there are others that also have specific prevention needs, particularly heterosexuals from high prevalence countries. Progress on the 2003 HIV/AIDS Action Plan must be reviewed, and new initiatives for prevention considered. While individual behaviour must be targeted, the success of these initiatives will only be maximised when full consideration is given to the social and psychological contexts in which people live.

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All change for the New Zealand laboratories

Ross Boswell, Andrew Tie

Since the 1950s, diagnostic laboratory testing for New Zealand patients has been fully funded by the Government with no charge to the patient. In community practice, laboratory tests have been divided into two categories: “schedule tests,” a list of about 80 specific tests listed in the Diagnostic Laboratory Schedule, and “non-schedule tests”—the remaining ever-expanding list of investigations.

Schedule tests are funded fee-for-service in most community laboratories, while non-schedule test specimens are commonly collected by those laboratories and transferred to a public hospital where the analyses are performed. The requirement for funding has been that the test is requested by a doctor (or in some circumstances a dentist or midwife), that the patient is not an inpatient in a public hospital, and that the test is for diagnosis and not for immigration, industrial, research, or insurance purposes.

New Zealand citizens, residents, and certain other categories of patient are eligible for funding but foreign patients, in general, are not.

Prior to 2003, the funding for schedule tests was administered centrally by the Health Department (later the Ministry of Health), then by Regional Health Authorities, and then by the Health Funding Authority. The funding for non-schedule tests was held by the applicable Hospital Board, then Area Health Board, then Crown Health Enterprise (CHE), and currently by the District Health Board (DHB). In most parts of New Zealand, that is the DHB within which the patient is domiciled—but in Greater Auckland the Auckland CHE and now Auckland DHB holds that funding for the whole region.

Because the funding for schedule tests was previously held centrally (except for the relatively brief experiment with four Regional Health Authorities), the domicile of the patient and the location of the laboratory were immaterial. Laboratories that were “recognised” to claim the laboratory benefit were able to claim for all of the qualifying work they did, and were able to operate regionally or even nationally.

Within this funding regime, community pathology laboratories grew to serve their populations and clinical practitioners. The funding schedule was seldom adjusted, but advances in technology and increases in test volumes and efficiency allowed laboratories to provide testing of increasingly high quality, both in technical terms and in terms of customer satisfaction, while remaining profitable. This profitability attracted commercial interest, and between 1985 and 2005 most community pathology practices in New Zealand ceased to be medical partnerships and became divisions of national or international corporations.

In 2003, the funding for community laboratory testing was devolved to DHBs. Since the laboratories’ operations in many cases extended beyond DHB boundaries, the funding was settled upon the DHB within whose district the laboratory headquarters lay. This produced anomalies where, for example, funding for some patients tested in Auckland was claimed from the Otago DHB. A complex system of “horse-trading”
grew up behind the scenes with DHBs agreeing to transfer funding according to the domicile of the patient.

In 2002, the incorporated society of DHBs, DHBNZ, commissioned a paper by Simon Terry Associates Ltd entitled *Options for Reform of Diagnostic Laboratory Services Markets*, (Reinhard Pauls) August 2002, which led them to consider the wisdom of continuing the existing arrangements where, when the community funding was transferred to them, most DHBs would fund separate hospital and community laboratories, and where in most cases they allowed competition between providers for community laboratory testing.

Because the competition was for market share, given a fee-for-service funding contract, it was perceived to drive up laboratory utilisation and therefore overall cost.

The outcome of this review has been the current community laboratory contracting round, involving most DHBs throughout the country. Solutions that have been sought are different according to the perception of local circumstances:

- In Otago-Southland, and in Nelson-Marlborough, a single company has been contracted to provide all testing in both community and public hospital practice, driving out both the previous DHB laboratories and the competing commercial laboratory. The Commerce Commission forbade a merger of the private providers.

- In Wellington and Hutt Valley, an initial proposal to award a sole provider contract to a consortium of DHB laboratories and a private provider was overturned, and the community contract awarded to a joint venture between the two private providers who previously served the region in competition, with Commerce Commission approval.

- In the greater Auckland region, the contract for private pathology services from mid-2007 has been awarded to a completely new provider, thus eliminating the current sole provider.

Pathology practices of decades’ standing have been terminated and disestablished. Private specialist practice is the target of specific intervention in at least one region (Wellington-Hutt Valley), where patients of private specialists will no longer have state funding for their pathology services.

Pathologists and their staff not surprisingly have vigorously challenged the processes and decisions, particularly those practices disadvantaged or eliminated. However, the real surprise to pathologists, and to administrators and boards, has been the unexpectedly high level of spontaneous public support and protest, exceptional for a clinical service with a normally low profile. Sceptics appear unconvinced that the savings expected will be better spent on other services by managers. Critics argue that estimates of transition costs are not available, or are ignored by contracting DHBs.

Legalistic process and devices such as probity reports have been used as a shield from criticism by DHB planners focused on reduction of cost and business risk. Clinical service consequences and clinical risk seem to have little influence in the final selection of providers.

Is the current furore merely the inevitable result of “kicking over the ant-heap,” or is there more substance to it? We believe that there is real cause for concern. These
changes are being implemented at local level with no national framework or oversight.

Workforce is a critical national issue in pathology as in other areas of health. New Zealand has a shortage of pathologists (a national workforce of about 200 equating to about 1:20,000 population, compared with Australia’s “severe shortage” at 1:15,000) and senior laboratory scientists. In Australia, UK, and in other countries there is strong demand for pathologists’ services. The changes in contracts that force them to change location or change employer will inevitably cause some of them to re-evaluate their options, and they will leave the country.

Compared with New Zealand, remuneration for pathologists in Australia is high, and employment more settled, so the trans-Tasman traffic is heavily biased in a westward direction. Pathologists and their practices contribute to medical care in a large number of unseen (but necessary) and mostly voluntary (non-contracted) activities. An incomplete list includes clinical review meetings, accreditation, quality assurance, research, professional society and college activities, government and hospital programmes and projects, peer reviews, advice to the Health and Disability Commissioner, and the training and examination of registrars.

When service reporting workloads reach the point where there is insufficient time or enthusiasm for optional tasks, pathologists will withdraw and retrench out of necessity. This will only become apparent after time, and re-engaging the disenfranchised will be very difficult.

The changes inevitably result in disruption of established clinical relationships. Referring doctors learn to know and trust the laboratory and pathologists on whose advice they rely, and patients with chronic disease know and trust the laboratory staff they visit regularly for blood tests. The costs in health terms of disrupting these clinical relationships appear to have been discounted in the rush to cut financial costs.

The changes result in clinical risk. This is particularly marked where service is to be transferred on a given date from one sole provider of service to another, as in Auckland. There is the prospect that a current contractor may be unable to provide service to the end of its contracted period, as its staff perceive the inevitability of its closure and seek employment elsewhere. There is the possibility that the new provider is unable to recruit and train staff, and set up premises, equipment, and procedures in time to assume the workload at the beginning of its contracted period.

The decision to provoke such a transition is indeed “courageous” (in the sense that Sir Humphrey Appleby used the word), and we should take careful note of the decision-makers so that if it is successful we will know where to sheet home the credit for success.

The changes open new possibilities for cost-shifting and service reduction, and may work against innovation. In all cases, the new contract is for a lower price than the contracts that it replaces, since the driver for change is cost reduction. Such a narrow commercial focus from the purchaser will inevitably lead to commercial responses from the providers: the service provided will be at the level specified in the contract, but it is unlikely to be better or broader.

In many cases the contracts appear to have been written so as to require the laboratory to bear or share the cost of increases in workload, so the laboratory is induced to
pressure referring practitioners to reduce the numbers of tests they order. That may be commercially rational, but it may also lead to underservicing and the loss of opportunity for early diagnosis and appropriate monitoring.

Balanced against these risks is the prospect of financial saving. We have been advised that the Auckland community contract is for $560 million over 8 years, and that it offers savings of $15 million per annum for this 8-year period.

Community laboratory testing accounts for about half of the cost of DHB-funded laboratory services in Auckland. Assuming that no cost-shifting occurs, we are then looking at a reduction of about 10% in laboratory costs, or about 0.5% in total DHB expenditure. While every little bit helps, it is hard to escape a conclusion that in pursuing this disruptive and risky course the DHBs may be penny wise but pound foolish.

What lessons are there from this situation? Firstly, it must be determined that in future no such wholesale change to a clinical service will be undertaken without a national policy framework, ensuring that issues of national significance are not lost in the drive to achieve local cost reductions. Secondly, it is imperative that clinical risks are carefully weighed and a sustainable benchmark of service estimated when financially-driven service changes are considered. Thirdly, relatively soon it will become apparent that defects in the terms of contracts allow or encourage behaviour that is not in the interests of patients or of the health system as a whole. These defects in approach should be exposed, so they may be avoided in the next round of contracting.

When the chickens come home to roost, many of the managers responsible for these changes will have moved on, leaving the profession to contend with what may be a far less attractive future for those who remain.

Indeed, the public may one day rue the decisions of administrators and board members to cheapen the service on their behalf without first establishing the case for change, without adequately weighing risk, and without a plan beyond a budget bottom line.

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Tuberculosis epidemiology in New Zealand: 1995–2004

Dilip Das, Michael Baker, Lester Calder

Abstract

Aims To describe the epidemiology of tuberculosis (TB) in New Zealand (NZ) for the 10-year period 1995–2004, and to place this in the context of long-term incidence trends.

Methods We calculated TB incidence rates since the early 1920s using published data. A more detailed analysis examined TB notification and laboratory data for the period 1995 to 2004 using population denominator data from the 1996 and 2001 Census. We calculated incidence rates by age, sex, ethnicity, place of residence, country of birth, and deprivation for the two 5-year periods: 1995 to 1999 and 2000 to 2004. We also calculated and compared TB case fatality and mortality rates for those periods. We described outbreaks by using TB outbreak reporting data.

Results The long-term decline in TB incidence in NZ halted in the mid-1980s, and in the last two decades, annual rates have stabilised at around 10 cases per 100,000. The average rate for 1995-2004 period was 10.3 per 100,000. The TB incidence rate in NZ is higher than that in Australia, USA, and Canada, and slightly lower than that in the UK. Within NZ there are marked ethnic differences in rates, with age-standardised incidence rates 10.5, 22.3, and 36.5 times higher in Māori, Pacific peoples, and people of Other ethnicity respectively than the rate in Europeans. Rates generally increase with age. Approximately two-thirds (64.6%) of people with TB were born overseas. TB case fatality and mortality rates in NZ are declining and are comparable to those in Australia, Canada, USA, and the UK. Twenty-four TB outbreaks, including 221 cases, were reported between mid-1996 and 2004.

Conclusions TB is not declining in NZ. The burden of disease is very unevenly distributed across the population with marked ethnic inequalities.

During the past several decades, developed countries made remarkable progress in tuberculosis (TB) control. Indeed, even before the advent of anti-TB drugs in the 1940s and 1950s, notification rates were declining. The availability of anti-TB drugs further accelerated the decline. However, the trend was halted in the mid-1980s and many developed countries experienced a rise in the incidence of TB.

In New Zealand (NZ), TB notification steadily declined after the Second World War and reached a nadir of 295 in 1988. In subsequent years, between 300 and 450 cases have been reported annually with a notification rate of around 10 per 100,000. This paper reports trends in the incidence of TB in NZ from the early 1920s and describes in detail the epidemiology of TB in NZ for the 10-year period from 1995 to 2004. Such information is essential to assess current TB prevention and control measures and identify areas where improvements can be made.
Methods

This analysis was largely based on anonymised TB surveillance and outbreak data from the Institute of Environmental Science and Research Ltd (ESR) as well as population data from Statistics NZ (Statistics NZ). In addition, TB crude incidence rates for the years 1922 to 1995 were calculated using numerator data from annual reports of the Director-General of Health and population information from NZ Yearbooks and more recently from Statistics NZ publications.

TB is a notifiable disease in NZ. Any medical practitioner diagnosing or suspecting a case of new or relapsed TB is required, under the Tuberculosis Act 1948, to notify the case to the local Medical Officer of Health (MOH). Local public health staff enter these TB notifications and additional laboratory data onto a national computerised database (EpiSurv). These data are centrally compiled at ESR.

For surveillance, the notification data are supplemented with TB laboratory data to:

- Identify cases that have not been notified; and to
- Provide additional information about each laboratory confirmed case, including typing information.

This report includes both ‘confirmed’ and ‘probable’ cases.

A confirmed case of TB is one that has been laboratory confirmed by one of the following:

- Positive culture for *Mycobacterium tuberculosis* or *Mycobacterium bovis*;
- Positive microscopic examination for acid fast bacilli when a culture has not been or cannot be obtained;
- Demonstration of *Mycobacterium tuberculosis* or *Mycobacterium bovis* nucleic acid in specimens;
- Histology strongly suggestive of TB.

A probable case of TB is one where there is no laboratory confirmation, but:

- There are symptoms and signs compatible with active TB, such as compatible radiology or clinical evidence of current disease, and
- Full anti-TB treatment has been started by a clinician.

We included in this analysis all TB cases meeting the above case definition, and notified between 1 January 1995 and 31 December 2004. This analysis excluded latent TB infections (LTBI) on treatment (i.e. Mantoux-positive but no evidence of active disease) but included relapsed/reactivated disease on treatment.

For the two 5-year periods (1995–1999 and 2000–2004), we analysed cases by district health board (DHB) of residence, age, sex, ethnicity and country of birth, and NZ indices of deprivation (NZDep96 and NZDep01), which are area based measures of socioeconomic deprivation constructed from 1996 and 2001 Census data respectively. We also calculated case fatality rate (percentage of TB cases who die of TB) and annual mortality rate (rate of TB deaths per 100,000 population per year).

Annual incidence rates were calculated by dividing the number of cases notified during the year by mid-year population (estimated and published by Statistics NZ) and expressing the result as cases per 100,000. If mid-year population estimates were not available for a particular variable (e.g. DHB of residence) or combination of variables, we used census data (1996 data for calculation up to 1999 and 2001 data subsequently) to calculate rates.

EpiSurv recorded self-reported ethnicity of cases. There was provision for indicating more than one ethnicity in the TB case report form and in the census form. We used Statistics NZ’s ‘prioritised ethnicity’ concept for both numerator and denominator. Prioritisation of ethnic group data assigns each person to just one ethnic group when a multiple response is given. In this analysis we used four prioritised ethnic groups: Māori, Pacific people (mostly of Samoan, Tongan, Niuean, or Cook Islands origin), ‘Other’ (Asian and non- Māori, non-Pacific, and non- European combined) and European (NZ and other European combined).

We age-standardised incidence rates to the NZ population age structure of the 2001 Census. Trend lines in TB incidence rates were generated by the least square method and were tested for statistical significance by the Chi-square test for trend. We used Microsoft Excel and EpiInfo (version 3.3.2)
software to analyse the data. We described outbreaks using outbreak-reporting data, which is also entered into EpiSurv.

**Results**

**TB incidence by year**—In the late 1920s and early 30s the TB incidence rate was declining. In the mid-30s, the trend was reversed and disease incidence peaked during the time of the Second World War with 2603 cases and a rate of 159.1 per 100 000 in 1943. The TB incidence rate then declined steadily for the following 40 years up to the late 1980s (Figure 1).

**Figure 1. Tuberculosis notification rate (crude rate per 100,000), New Zealand, 1922–2004**

Over the two decades from 1985 to 2004, between 300 and 450 cases were notified annually with a significant (p<0.001) increasing trend (Figure 2). The lowest number of notifications (295 cases) was in 1988. In spite of the increasing trend in the number of notified cases, the crude TB incidence rate has remained constant within a narrow range around 10 per 100,000 for these two decades (Figure 1). The crude rate for the 1995-2004 period was 10.3 per 100,000. The lowest incidence rate (8.5 per 100,000) was noted in 1997.

**TB incidence by District Health Board**—Table 1 in the Appendix shows the crude incidence rate of TB for each DHB for the two 5-year periods from 1995 to 2004. Some DHBs have consistently higher rates than the others. In the 1995–1999 period, DHBs with rates above 10 per 100,000 were Auckland (22.8), Counties Manukau (19.1), Hutt Valley (14.8), Capital and Coast (14.2), and Northland (10.2). In 2000–2004, DHBs with rates above 10 per 100,000 were Auckland (20.9), Counties Manukau (18.5), Capital and Coast (16.2), Hawke’s Bay (14.9), Waitemata (12.0), and Hutt Valley (11.4).
TB rates were consistently higher in overseas-born people than in NZ-born people in all DHBs and in both time periods. The rates in NZ-born were below 10 per 100,000 in all DHBs except in Counties Manukau in 1995–99 (10.8 per 100,000) and Hawke’s Bay in 2000–2004 (11.9 per 100,000). In Hawke’s Bay there was more than a three-fold increase in rate in the NZ-born between 1995–1999 and 2000–2004 (from 3.3 to 11.9 per 100,000). This increase was presumably due to a large outbreak in 2002 involving NZ-born people (mainly Māori).6

Figure 2. Number and trend in tuberculosis notifications, New Zealand, 1985–2004

[Graph showing the number of cases notified between 1985 and 2003 with a Chi-square test of trend: p<0.001]

**TB incidence by age and sex**—TB incidence by age groups has a bimodal distribution with higher rates in young adults and the elderly (Table 1, Figure 3). In the 1995–1999 period, rates were highest in the ≥70 group (20.3 per 100,000). However in the 2000–2004 period the highest rate was observed in the 20–29 years age group (18 per 100,000).

Comparison of the two 5-year periods (1995–1999 and 2000–2004) indicates that rates have risen significantly in adults in the age group 20–29 years (p<0.001). Rates have fallen significantly in 0–9 years (p<0.02), 50–59 years (p<0.05) and ≥70 years (p<0.001). In all other age groups, there are no significant changes in the incidence rates between the two periods.

In the whole 10-year period, sex was recorded for 99.2% (3743/3772) of cases. The proportion of cases in males (1923 or 51.4%) was similar to females (1820 or 48.6%).

**TB incidence by ethnicity**—Europeans have the lowest age-standardised annual incidence rates and people of Other ethnicity have the highest, with Māori and Pacific people in between (Table 1). Between the two 5-year periods, the age-standardised rates declined significantly in Europeans and in people of Other ethnicity, with no significant changes in rates in Māori and Pacific people. In the whole 10-year period,
the age standardised annual incidence rate for different ethnic groups were as follows: European - 2.0/100,000, Māori - 21.1/100 000, Pacific people - 44.8/100 000 and people of Other ethnicity - 73.1/100 000. This means that compared to European New Zealanders, Māori, Pacific and people of Other ethnicity had 10.5, 22.3 and 36.5 times higher risk of having TB respectively.

**TB incidence by age and ethnicity**—Figure 4 shows the age specific rates of TB for European, Māori, Pacific, and Other ethnic groups. Ethnicity and date of birth were not recorded for 133 and 6 cases respectively, and these records are excluded from the analysis. The actual number of cases and the age-specific incidence rates in different ethnic groups are shown in Table 2 in the Appendix. The higher rates of TB observed in the 20–29 year and 30–30 year age groups (Figure 3) are due to overrepresentation of people of Pacific and Other ethnicity in these age groups (Table 2 in the Appendix).

In all ethnic groups, the incidence rates generally rise with increasing age and are highest in people aged 70 years or over. However, in people of Other ethnicity high rates are also observed in young adults (20–39 years). When the two 5-year periods are compared, it is observed that in recent years the rates have significantly decreased in some age groups of European, Māori, and people of Other ethnicity. On the other hand, TB incidence has risen significantly in young Pacific adults (20–29 years old), which is contributing to a significant increase in the age-specific rate in 20–29 years in the total population (Figure 3).

**TB incidence by place of birth**—For the total 10-year period, country of birth was recorded for 89.3% (3367/3772) cases. Of these cases, 35.4% (1193) were born in NZ and 64.6% (2174) were born overseas. Figure 5 shows the number of TB cases with known country of birth (NZ-born and overseas-born) for the period 1995 to 2004. While the decreasing trend in the number of NZ-born cases is not significant (p=0.606), there is a significant (p<0.001) increasing trend in the number of cases in overseas-born people during this period. However, there was no significant increase in the incidence rate in overseas-born people between 1996 (31.7 per 100,000) and 2001 (32.3 per 100,000).

**TB incidence by NZ Index of Deprivation (NZDep)**—The incidence of TB generally increased with increasing deprivation, particularly for the most deprived decile (Figure 6). This pattern was more evident in the 2000–04 period where the average rate in the most deprived 20% of the population (19.7 / 100 000) was 3.7 times higher than in the least deprived (5.3 / 100 000). In the 1995–99 period, a large number of cases were notified from one particular area of low deprivation which resulted in a somewhat “U-shaped” distribution. During that period the average rate in the most deprived 20% of the population (15.5 / 100 000) was 1.4 times higher than in the least deprived (10.7 / 100 000).

**Death due to TB**—Of the 3772 cases notified during 1995–2004, 227 died from TB. This variable was not reported for 343 cases so they are excluded from the analysis. For the 1995–1999 period, the case fatality rate averaged 8.0% and mortality rate averaged 0.7 per 100,000. For the 2000–2004 period, the case fatality rate averaged 5.3% and the mortality rate averaged 0.5 per 100,000. Both the case fatality rate (p=0.01) and mortality rate (p=0.03) were significantly lower in the 2000–2004 period than in the 1995–1999 period.
Table 1. Incidence of tuberculosis by age, sex, ethnicity, and place of birth (NZ or overseas), New Zealand, 1995–2004

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<sup>1</sup>For the variable ‘age’, the rate, rate ratio, and the 95% CI are age-specific; <sup>2</sup>Census 1996, <sup>3</sup>Census 2001, <sup>4</sup>Age-standardised rate per 100,000, *Statistically significant difference, at the 95% CI in the rates between the two time periods.
Figure 3. Average annual incidence rate of tuberculosis by age group, New Zealand, 1995–1999 and 2000–2004

![Graph showing average annual incidence rate of tuberculosis by age group, New Zealand, 1995–1999 and 2000–2004.](image)

Figure 4. Incidence rate of tuberculosis by age group and ethnicity, New Zealand, 1995–1999 and 2000–2004

![Graph showing incidence rate of tuberculosis by age group and ethnicity, New Zealand, 1995–1999 and 2000–2004.](image)

Note that Y-axis scale is different for different ethnicities; Other=Asian and non-Māori, non-Pacific, and non-European combined.
Figure 5. Numbers and trends of tuberculosis cases in NZ-born and overseas-born people, New Zealand, 1995–2004

Figure 6. Tuberculosis incidence rate by NZ Index of Deprivation, New Zealand, 1995–1999 and 2000–2004
Figure 7 shows the TB case fatality and mortality rates and their trend in the period 1995–2004. The significant declining trend line for case fatality rate (p=0.014) results in a parallel decline in mortality rate (non-significant, p=0.057) over this period.

**Figure 7. Tuberculosis case fatality and mortality rates, New Zealand, 1995–2004**

**Outbreaks**—Outbreak information is recorded in two ways on EpiSurv: the outbreak reporting system and the case report form. An outbreak is defined as two or more cases that are linked by epidemiological investigation or DNA fingerprinting. A cluster of cases all living in the same household is not considered an outbreak. The outbreak reporting system recorded 24 TB outbreaks involving a total of 241 cases (including cases of LTBI) between June 1996 and the end of 2004. As an outbreak unfolds, the EpiSurv records get updated, though this process is not entirely complete. For example, in EpiSurv there are several outbreaks with only one case recorded, which by definition is not an outbreak.

Through the outbreak reporting system, nine outbreaks were reported from Auckland, six from Hawke’s Bay, three from Wellington and two from Wanganui in this period. Forty-seven percent of cases in outbreaks were Pacific people and 41% were Māori, whereas in non-outbreak situations their proportions were 16% and 15% respectively. Individual case report forms recorded 221 cases of TB disease (6.8%) as being part of outbreaks between June 1996 and December 2004.

**Discussion**

This review of TB epidemiology indicates that the incidence of TB in NZ has not declined over the past two decades. The moderate increase in case numbers over this period has been offset by an increase in population resulting in no net increase in the crude rate of disease. This finding is consistent with the results of a previous study,
which found a stable rate of bacteriologically and histologically confirmed cases for the period 1985–1990.7

NZ has a higher rate of TB than some other developed countries. Australia, Canada, and the USA all had annual incidence rates between 5 and 6 per 100,000 during the period 2000–2003.8 UK had a higher rate of around 12 per 100,000.8 The TB incidence rate declined in recent years in some of these countries. For example, USA had a gradual decline in incidence rate from 9.8 per 100,000 in 1993 to 5.1 per 100,000 in 2003.9 Canada has also had a gradual decline in the incidence rate from 7.2 per 100,000 in 1991 to 5.2 per 100,000 in 2002.10 However, the incidence rate remained stable in Australia11 (in the last 20 years) and the UK12 (in the last 15 years).

In terms of mortality, NZ rates are comparable to those of other countries. In NZ the average annual TB mortality rate declined from 0.7 per 100,000 in 1995–1999 period to 0.5 per 100,000 in 2000–2004 period. In the USA, the TB mortality rate was halved between 1993 and 2003 (from 0.6 per 100,000 to 0.3 per 100,000).9 In England and Wales, the TB mortality rate declined from 0.83 per 100,000 in 1993 to 0.74 per 100,000 in 2003.12

The TB case fatality rate in NZ (average 8.0% in 1995–1999 and 5.3% in 2000–2004) was comparable to that in Canada in 2001 (5.8%).10 The decline in mortality was mainly due to the decline in case fatality rate, as the TB incidence rate remained stable during this period.

Probably the most important trend over the 1995–2004 period was the diverging number of NZ- and overseas-born cases (Figure 5). While NZ-born cases are declining, overseas-born cases are increasing. However, the incidence rate in overseas-born people did not increase significantly between 1996 and 2001 (31.7 and 32.3 per 100,000 respectively). The increased number of overseas-born cases without a concomitant increase in the rate can be explained by a disproportionate increase in the size of the overseas-born population (an approximate 15% increase compared to about 10% for the total NZ population) between 1996 and 2001. The net effect is that NZ’s total TB incidence rate has remained static.

The contribution of immigration from high incidence countries, human immunodeficiency virus infection, multi-drug-resistant TB, and Mycobacterium bovis infection to total TB incidence in NZ is explored in detail in the following article in this issue of the Journal.13

Probably the most striking feature of TB epidemiology in NZ is the huge and persisting differences in incidence rate by ethnicity. For the whole 10-year period studied, the age-standardised incidence rates in Māori, Pacific people, and people of Other ethnicity are approximately 10, 22, and 36 times higher, respectively, than the rate in Europeans. This finding is consistent with previous observations.4,7,14 For all ethnic groups, TB rates reflect age cohort levels of infection and population socioeconomic status. Socioeconomic status is an important determinant of the disparity between European rates and those of the other groups. For Pacific people and people of Other ethnicity, pre-migration infection and subsequent development of disease is a major factor contributing to this disparity.

Cases occurring as part of reported outbreaks give an indication of ongoing disease transmission within NZ. The proportion of cases (6.8%) belonging to outbreaks is
lower than previously reported (10%). However, these proportions are likely to be underestimates, as several outbreaks had only one recorded case suggesting that outbreak reporting is incomplete. Māori and Pacific people are disproportionately affected by outbreaks, indicating social factors such as poor access to healthcare, delayed diagnosis and increased transmission due to overcrowding. There is some evidence that the incidence of TB is associated with overcrowding at the census area unit level. We will report on our investigation of this association in a future paper. As TB outbreaks are often prolonged, the outbreak information gets updated by recording outbreak number in the case report form of subsequent cases, which does not happen reliably. This mismatch has also been noted previously. There is a need to improve the outbreak information updating system in the individual case report form in EpiSurv and to standardise the case definitions so that cases of active disease can be distinguished from cases of LTBI.

This analysis has identified quite marked variations in TB burden between different DHB regions. An indication of local transmission is provided by the incidence rate in NZ-born cases, which effectively excludes the contribution from migration. Such rates were generally low across NZ. An exception was the Hawke’s Bay DHB, which was the only DHB where there was a significant rise in TB rate over the 10 years observation period (Table 1 in Appendix). This increase was explained by a large outbreak in 2002.

Surveillance data have inherent limitations for describing disease occurrence and distribution. Previous analyses have shown a degree of under-ascertainment (for TB in children this was 4% over the period 1 January 1992 to 30 June 2001, and in Otago in the early 1990s this was 33% for 1985-90 and 48.5% for 1991–92). The current surveillance system has measures to increase its sensitivity, including using both notification and laboratory data, availability of EpiSurv software in all public health services and dedicated clerical staff. The findings of this study are limited by incomplete data on some variables. For example, ethnicity and country of birth were not recorded in 133 (3.5%) and 405 (10.7%) of notified cases.

For calculation of rate by ethnicity, where there were multiple responses, we used the Statistics NZ prioritised ethnicity approach for both the numerator and denominator. However, it is possible that the numerator (surveillance data) and denominator (census data) were collected differently. In the census, ethnicity recorded is self-identified. EpiSurv is supposed to record client-identified ethnicity (including multiple responses if that is applicable). However, this may not happen consistently, particularly in a hospital setting. The health professional notifying the case might indicate only one ethnicity from his or her general knowledge of the patient or from the front sheet of the hospital record without specifically enquiring about multiple ethnicities the patient might belong to.

It has been repeatedly shown that hospital records are more frequently coded with sole rather than multiple ethnicities. These practices create a numerator-denominator bias which we cannot eliminate. The effect is to underestimate TB rates in Māori. This underestimation has been observed for other diseases as well.

In conclusion, this review of the epidemiology of TB in NZ paints a mixed picture of success in the prevention and control of this disease. Positive observations are the
Decline in incidence rate in European, in people of Other ethnicity, and the steady decline in case-fatality rate among reported cases.

Persistent ethnic disparities in TB risk remain a major concern. The main challenge to TB control in NZ, as in other developed countries, is the high global prevalence of infection in developing countries. This global disease burden manifests itself in a high prevalence of infection in migrants from these countries and is a further reminder of the urgent need to increase TB prevention and control at a global level. This analysis also demonstrates that ongoing TB transmission within NZ remains an import source of disease. The factors contributing to such transmission will be examined in detail in a future paper.

Disclaimer: Parts of this work are based on data and information provided by the Institute of Environmental Science & Research Limited (on behalf of the Ministry of Health) and Statistics New Zealand. However, the analyses, conclusions, opinions, and statements expressed herein are those of the authors, and not necessarily those of the Institute of Environmental Science & Research Limited, the Ministry of Health, or Statistics New Zealand.

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References:


### Appendix

Table 1. Tuberculosis number and incidence rate by District Health Board, New Zealand, 1995–1999 and 2000–2004

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¹Total includes cases whose birthplace status is unknown;  
²Annual incidence rate per 100,000.
Table 2. Tuberculosis number and incidence rate by age group and ethnicity, New Zealand, 1995–1999 and 2000–2004

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1 Average annual incidence rate per 100,000.

2 Age-standardised rate per 100,000, standardised to the NZ-population-age-structure of the 2001 Census.
Why the tuberculosis incidence rate is not falling in New Zealand

Dilip Das, Michael Baker, Kamalesh Venugopal, Susan McAllister

Abstract

Aims To assess the role of migration from high-incidence countries, HIV/AIDS infection, and prevalence of multi-drug resistant organisms as contributors to tuberculosis (TB) incidence in New Zealand (NZ) relative to ongoing local transmission and reactivation of disease.

Methods TB notification data and laboratory data for the period 1995 to 2004 and population data from the 1996 and 2001 Census were used to calculate incidence rates of TB by age and ethnicity, country of birth (distinguishing high and low-incidence countries), and interval between migration and onset of disease. Published reports of multi-drug-resistant TB for the period 1995 to 2004 were reviewed. Anonymous HIV surveillance data held by AIDS Epidemiology Group were matched with coded and anonymised TB surveillance data to measure the extent of HIV/AIDS coinfection in notified TB cases.

Results Migration of people from high-TB incidence countries is the main source of TB in NZ. Of those who develop TB, a quarter does so within a year of migration, and a quarter of this group (mainly refugees) probably enter the country with pre-existing disease. Rates of local TB transmission and reactivation of old disease are declining steadily for NZ-born populations, except for NZ-born Māori and Pacific people under 40. HIV/AIDS and multi-drug-resistant organisms are not significant contributors to TB incidence in NZ and there is no indication that their role is increasing.

Conclusion TB incidence is not decreasing in NZ mainly due to migration of TB infected people from high-incidence countries and subsequent development of active disease in some of them in NZ. This finding emphasises the importance of regional and global TB control initiatives. Refugees and migrants are not acting as an important source of TB for most NZ-born populations. Those caring for them should have a high level of clinical suspicion for TB.

It is well documented that tuberculosis (TB) incidence rates in the developed world rapidly declined after the Second World War. However, in the mid to late 1980s, the decline was halted and many countries experienced an increase in TB incidence rates.1

Occurrence of human immunodeficiency virus (HIV) infection and acquired immune deficiency syndrome (AIDS),1,2 emergence of multi-drug-resistant (MDR) organisms,1 and increased migration from high-incidence countries,1 have been implicated as causes for the TB increase.

New Zealand (NZ) also followed this declining trend in TB incidence up to the mid-1980s. Since then, the incidence rate has plateaued.3 TB in people who have emigrated from high-incidence countries has been thought to be one factor preventing a further decline in TB incidence in NZ. Specifically, inadequate screening of migrants who are subsequently shown to have active disease soon after arriving has been suspected to be one factor contributing to this pattern.4,5 However, it is important...
to assess the relative importance of other potential factors, notably the role of HIV/AIDS infection, increasing drug resistance (particularly MDR), and the potential decline in the effectiveness of local TB-control programmes. This paper uses data from several surveillance sources to analyse the relative importance of these factors in preventing the further fall in the TB incidence rate in NZ.

Methods

This paper is largely based on analysis of TB surveillance data for the years 1995–2004 and 1996 and 2001 Census data (see the preceding article for a description of these data sources).

Migration—The TB surveillance system records country of birth and, for migrants, their date of arrival in NZ. These data allow cases born in low TB incidence countries to be distinguished from those born in high-TB incidence countries (defined as all countries except Australia, Austria, Belgium, Canada, Czech Republic, Denmark, Finland, France, Germany, Greece, Holland, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, NZ, Norway, Slovakia, Sweden, Switzerland, the UK, and USA).

We calculated the crude incidence rate by migrant region and country (for countries contributing on average more than one TB case a year) using census data on country of birth to provide the denominator. We also examined the TB notifications of people born in low- and high-incidence countries according to the interval between time of arrival in NZ and the date of notification.

Transmission and reactivation in NZ—TB cases were divided into categories based on probable transmission setting and timing of infection.

This classification used a combination of:
- NZ-born versus overseas-born;
- Ethnic group; and
- Age group.

The age group classification split the population into those <40 years where infection is likely to be due to relatively recent transmission and those ≥40 where TB is more likely to be due to reactivation of infection acquired many years previously. In the older group, however, some recent transmission cannot be ruled out as evidenced by studies using genetic fingerprinting.

HIV/AIDS coinfection—Medical practitioners diagnosing a case of AIDS are required to notify this in a coded form to the local Medical Officer of Health (MOH). Nationally, AIDS notifications are compiled by the AIDS Epidemiology Group at the University of Otago, Dunedin. HIV infection is under laboratory-based surveillance. Two laboratories—one in Auckland Hospital and the other at the Institute of Environmental Science and Research Limited (ESR)—do the confirmatory ‘Western Blot’ test for HIV and report positive results in a coded form to the AIDS Epidemiology Group.

TB is an AIDS defining condition in HIV-positive persons. So, by definition, an HIV-positive person with TB also has AIDS. The AIDS Epidemiology Group matched the coded list of people with AIDS and HIV infections with the similarly coded list of TB notifications to detect people coinfected with HIV and TB.

AIDS is notified anonymously with a code using the first two letters of surname, first initial of given name, sex, day, month, and year of birth. We calculated the proportion of TB patients coinfected with HIV each year and examined the trend.

Multi-drug resistance—Three mycobacteriology laboratories (in Auckland, Wellington, and Waikato hospitals) test antimicrobial susceptibility of all Mycobacterium tuberculosis and Mycobacterium bovis isolated from human specimens in NZ. ESR matches these with TB case notifications and analyses the data to describe the distribution of TB-drug resistance in NZ. We reviewed reports for the period 1995 to 2001, and annual reports of anti-TB-drug resistance for subsequent years (2002-2004), to assess the role of multi drug resistance in local transmission of the disease.

Results

Migration—For the 10-year period 1995–2004, country of birth status (NZ or overseas) was reported for 89.3% (3367/3772) of notified cases. Of those for whom
country of birth status was known, 35.4% (1193) were born in NZ and 64.6% (2174) were born overseas.

Table 1 presents numbers and crude incidence rates of TB by region and selected countries (those with an average of more than one case per year). Most of the overseas-born cases were from three regions of the world—Asia, Africa, and Pacific Islands.

Countries (in descending order) contributing an average of more than 5 cases a year were India, China, Somalia, Samoa, Philippines, Tonga, Korea, Cook Islands, Vietnam, and Cambodia. Tuvalu contributed more than 5 cases per year for the period 2000–2004. Very high rates were observed in people born in Ethiopia (3209.9 per 100,000) and Somalia (1924.4 per 100,000) in the 1995–1999 period.

Among people born in high-incidence countries, the numbers of TB notifications was highest within the first year of arrival, and then decreased substantially in subsequent years (Figure 1). About a quarter (28.3% or 144 / 508) of people who were notified with TB within 1 year of migration from a high-incidence country were identified as having active TB within 2 months of arrival in NZ.

In contrast, very few people from lower-incidence countries were notified with TB within 1 year of arrival in NZ (Figure 2). Most of those who developed the disease did so 20 years or more after arrival (Figure 2).

Table 2 shows TB numbers and rates in NZ- and overseas-born populations according to broad age and ethnic groups. Compared to NZ-born people of Pacific and Other ethnicity, TB rates were much higher in their overseas-born counterparts. This difference indicates that exposure to TB overseas rather than local transmission is the predominant mode of acquiring TB in people of these ethnic groups.

Rates of TB in overseas born populations generally declined over the 10-year period, with the exception of Pacific people <40 years, where there was a slight increase.
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¹ Census 1996, ² Census 2001, ³ Annual incidence rate per 100,000.
Figure 1. Interval between migration and notification of tuberculosis in cases born in high-incidence countries, New Zealand, 1995-2004

![Interval between migration and notification of tuberculosis in cases born in high-incidence countries, New Zealand, 1995-2004](chart1.png)

Figure 2. Interval between migration and notification of tuberculosis in cases born in low-incidence countries, New Zealand, 1995-2004

![Interval between migration and notification of tuberculosis in cases born in low-incidence countries, New Zealand, 1995-2004](chart2.png)
## Table 2. Incidence of tuberculosis in New Zealand-born and overseas-born populations by ethnicity and age group, New Zealand, 1995-2004

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<td>3</td>
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<td>2</td>
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1 Census 1996, 2 Census 2001, 3 Annual incidence rate per 100,000, CI= Confidence interval. There were 270 and 182 TB cases, respectively, for the 1995–99 and 2000–04 periods that had missing values for either country of birth, ethnicity, or age. These cases were excluded. 4 This rate is used as the reference value for all rate ratios presented in this table.
Transmission and reactivation in NZ—In the NZ-born population (Table 2), TB incidence varied markedly by age group and ethnicity. In 2000–2004, rates ranged from <0.5 per 100 000 (in Europeans <40 years) up to 23.8 per 100,000 (in Pacific people <40 years). TB rates in NZ-born populations were generally higher in older age groups (≥40 years) except for Pacific people (where these rates were based on very small numbers of cases).

TB rates declined in most NZ-born populations except for Māori and Pacific people <40 years, in whom there were statistically non-significant increases between 1995–1999 and 2000–2004. Of particular note was the statistically significant decline in the TB rate for NZ born Europeans <40 years, which is the largest single sub-population identified in Table 2.

Multi-drug-resistant TB (MDR-TB)—MDR-TB is defined as TB resistant to at least isoniazid and rifampicin. Published reports on anti-TB drug resistance showed that MDR-TB is rare in NZ, with a total of 19 cases recorded in 10 years since national surveillance of anti-TB drug resistance began in 1995.\textsuperscript{10,11} Eighteen of the 19 MDR-TB cases identified were people born overseas and presumed to have acquired the MDR-TB overseas. The remaining MDR-TB case was also born overseas, but multi drug resistance appeared to have developed during treatment in NZ. There is no documented evidence that MDR-TB has been transmitted within NZ up to 2004.

HIV/AIDS coinfection—In the 10-year period (1995-2004), out of 3,772 notified TB cases, 45 (1.2%) were diagnosed with HIV infection. The annual proportion of TB cases with HIV showed a non-significant declining trend over this 10-year period (Figure 3).

Figure 3. Percentage and trend of tuberculosis cases coinfected with HIV, New Zealand, 1995–2004
Discussion

This analysis has shown that migration of TB-infected people from higher incidence countries is the dominant factor driving the incidence of this disease in NZ. By comparison, rates in most NZ-born populations are static or declining, indicating that local transmission is being effectively controlled for most population groups. This analysis has also shown that HIV/AIDS and TB drug resistance are not making a significant contribution to the burden of TB in NZ.

Amongst people migrating from countries classified as ‘high-TB incident’, rates of disease vary enormously. Large contributors (more than 5 cases per year) with particularly high rates include India, China, Somalia, Philippines, Vietnam, Cambodia and Korea (Table 1). A similar pattern has also been seen in Australia. However, this observation has some limitations. Denominators used to calculate the rates in Table 1 are from 1996 and 2001 Census data. They do not represent the actual number of people with specific country of birth in other years (other than 1996 and 2001). As the numbers of people born in some of the countries are small, a change in their number could have a large effect on the rates.

A quarter (508 out of 2036) of TB cases born in high-incidence countries were notified within 1 year of migration. This population of 508 included 144 cases (28.3%), who developed TB within 2 months of arrival. Most of these people are likely to be refugees who had active TB on their arrival in NZ. This assumption is supported by the findings of TB screening for quota refugees at Mangere Refugee Resettlement Centre. If 2% is taken as the prevalence rate of TB in newly arrived refugees, then NZ can expect to see 15 TB cases per year in the quota refugee population (750 refugees per year). Other groups that are not screened for TB before entry include short-term visitors, who may in a few instances have active TB on arrival in NZ.

Results of this analysis provide considerable reassurance that migrant populations are not acting as an important source of TB transmission to most NZ-born populations. The largest NZ-born population considered in this analysis, those of European ethnicity aged <40 years, experienced a significant decline in TB incidence over these two time periods. This is despite the rise in absolute numbers of TB cases in migrant populations from high incidence countries that occurred over this time period. This observation is consistent with the experience of other countries such as Australia, that has also found that TB transmission tends to occur within defined population groups.

The NZ-born populations that experienced modest, though non-significant, increases in TB rates were Māori and Pacific people <40 years. NZ-born Pacific people <40 have a significant burden of disease. There are several possible explanations for these observations, including: a potential decline in the effectiveness of local TB control measures; increased exposure to Pacific migrants with TB; increasing ease of transmission (from such factors as higher levels of household crowding); for Pacific people increased visiting to ancestral home countries; or a chance finding. These
possible contributing factors need further investigation to identify opportunities for improved prevention and control.

This study found that HIV is making only a small contribution to TB incidence in NZ. Woodhouse, by analysing the data from Auckland Hospital Infectious Disease Unit, reported an increasing incidence of HIV/TB coinfected cases from the 1985–95 period to 1996-2001 period. However, our study based on national data for a 10 year period, did not find any increasing trend either in the number or in the proportion of coinfected cases. The findings of this study (and also of the previous reports) indicate that HIV is an insignificant contributor to TB in NZ, unlike some other countries, and there is no indication that its contribution is increasing.

Drug resistance also does not appear to be making a significant contribution to TB transmission in NZ. Multi-drug resistance among TB isolates in NZ (0.6%) is low compared to the global median (1.7%), Australia (2.0%), United States (1.4%), and England and Wales (1.5%). Indeed, there is no indication that MDR-TB is increasing in NZ or is being transmitted.

One other potential source of TB infection in NZ is the large reservoir of *Mycobacterium bovis*-infected animals. A separate combined epidemiological and microbiological investigation has shown that these animal reservoirs are making only a small contribution to TB infection in humans (2.7% over the 1995 to 2002 period) and incidence from this source is not increasing.

One limitation of this study is the possible numerator-denominator bias in calculating incidence rate by ethnicity. Though prioritised ethnicity was used both for the numerator and denominator, it is possible that these were not collected in the same way. While ethnicity in the census (denominator) is self-reported, surveillance (numerator) data are more likely to contain health professional reported ethnicity. It is known that in comparison to census data, hospital records are more frequently coded with sole rather than multiple ethnicities. The implication for this is underestimation of the Māori rates.

In conclusion, the findings of this study clearly indicate that migration from high TB-incidence countries is the predominant source of TB in NZ, and this contribution is increasing over time. This source of TB is supplemented in a smaller way by transmission to young Pacific people, local outbreaks, and reactivation of latent infection in the NZ born population.

It is envisaged that (for economic, social, and humanitarian reasons) NZ will continue to accept immigrants (including refugees) from high-TB incidence countries in future. We therefore need to constantly identify approaches to improve the swift detection and treatment of TB in these populations. One area where improvements could be made is the systematic screening of family reunification refugees. For quota refugees, there is an organised system of screening soon after their arrival. The system for family reunification is not as well organised.

In 2005 the NZ Immigration Service introduced stricter migrant health screening. These measures included full medical certificate (including chest X-ray) for people wishing to stay in NZ for more than 12 months, temporary entry chest X-ray certificate for people wishing to stay for more than 6 months (but less than 12
months), and chest X-ray for international students and working holiday makers.\textsuperscript{21} The effects of these changes have yet to be seen.

At the time of entering NZ far greater numbers of immigrants have latent TB infection (LTBI) rather than active disease and LTBI is not detected by chest X-ray. So clinicians who are caring for immigrants and refugees (particularly from Asia, Africa and Pacific Islands) need to be very vigilant for features of TB. Early detection and treatment of cases improves clinical outcomes and minimises the further spread of disease.

Ultimately, TB needs to be seen as a regional and global health problem. Increasing aid and development assistance can contribute to preventing and controlling this health threat. Other countries, notably the United States, have also seen value in supporting TB control in neighbouring counties.\textsuperscript{22} NZ is well placed to support regional TB control efforts, and at the same time help to protect NZ from this disease.

\textbf{Disclaimer:} Parts of this work are based on data and information provided by the Institute of Environmental Science & Research Limited (on behalf of the Ministry of Health) and Statistics NZ. However, the analyses, conclusions, opinions, and statements expressed herein are those of the authors, and are not necessarily those of the Institute of Environmental Science & Research Limited, the Ministry of Health, or Statistics NZ.

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\textbf{References:}


An outbreak of *Legionella pneumophila* suspected to be associated with spa pools on display at a retail store in New Zealand

Quentin Ruscoe, Sarah Hill, Timothy Blackmore, Margot McLean

**Abstract**

**Aim** To investigate and characterise a cluster of six cases of severe pneumonia in the Wellington region notified to Regional Public Health in November 2003. And to describe the public health response to an identified subgroup of *Legionella* infections.

**Methods** The case definition was “a person admitted to Wellington or Hutt Hospital between 29 October 2003 and 9 November 2003 with severe pneumonia”. The cluster was initially investigated by interviewing patients to obtain histories of activities and exposures, and by reviewing medical notes. Medical teams sent further clinical specimens for testing (sputum for polymerase chain reaction [PCR], convalescent *Legionella* serology, and urine for *Legionella* antigen testing). Further investigation of *Legionella pneumophila* cases involved obtaining detailed exposure histories, environmental investigations at the suspect source of infection, and taking water and biofilm swabs for *Legionella* detection and serotyping.

**Results** Three cases from the cluster were confirmed as, or compatible with, *Legionella pneumophila* serogroup 2. With the other three cases there was evidence of infection with *L. longbeachae* (two cases) or respiratory syncytial virus (one case). Exposure histories for the *L. pneumophila* cases revealed that the three cases had visited a Lower Hutt retail outlet with operating spa pools on display. *Legionella pneumophila* serogroup 1 was cultured from one of three pools. All pools were positive for *Legionella* on direct fluorescent antibody testing.

**Conclusions** Although unproven, the display spa pools were considered to be the most likely source of *Legionella* infection in the three cases that had visited the retail outlet. Although *Legionella* isolated from the pools was a different serogroup from that identified in two cases, the pools were considered to be the most likely source of infection in view of inadequate chlorination of the waters. Public health intervention to address the immediate and longer-term health risks from the pools is described. This is the second outbreak of *Legionella pneumophila* linked to operating display spa pools in New Zealand and it appears to be the fourth recorded outbreak of Legionnaires’ disease associated with operating spa pools on display.

The Wellington Regional Public Health service (RPH) was alerted in November 2003 to the presence of six cases of severe pneumonia by the Infectious Disease team at Wellington Hospital. Over a short time, there were six severely unwell hospital inpatients with pneumonia of unknown aetiology in Wellington or Hutt Hospital, several of whom had required admission to intensive care. This clustering of severe pneumonia raised the possibility of a common microbiological agent and a possible common exposure amongst the affected individuals.
Methods

Investigation of a communicable disease cluster classically involves three types of inquiry: epidemiological, environmental, and microbiological. All these types of inquiry were used as the research on the cluster progressed.

Epidemiological investigation—Wellington RPH obtained a list of six patients in Wellington and Hutt Hospital who had severe pneumonia of unknown aetiology. The case definition was “person admitted to Wellington or Hutt Hospital between 29 October 2003 and 9 November 2003 with severe pneumonia”. RPH staff initially visited each patient, to review their medical notes, and obtain an initial history of any activities or exposures that had taken place in the likely incubation period. Because cases were extremely ill, the history could usually only be obtained from a partner or family member. Employment history, buildings or commercial premises visited, and social and sporting events engaged in (during the incubation period) were obtained. Information on laboratory investigations and their results was obtained.

Case numbers were too small to consider a formal case-control study to evaluate possible risk factors for disease. Instead, case histories were compared to look for any common exposures.

Repeat exposure histories were obtained from the cases in the final defined cluster (three cases) from the initial patient data collection, plus fuller interviewing of all those in this final defined cluster using RPH’s standard legionellosis case questionnaire in a face-to-face interview. Telephone follow-ups were later used to check aspects of exposure histories.

Laboratory methods—Specimens were tested using standard laboratory methods as follows:

- Nasopharyngeal samples for respiratory viruses by direct immunofluorescence and cell culture;
- Sputum samples for routine bacteriology, Legionella culture, and mycobacterial culture;
- Urine for Legionella antigen;
- Paired serology for Mycoplasma and Legionella; and
- Blood cultures for aerobic and anaerobic bacteria.

Legionella serology was performed using indirect immunofluorescence antibody testing (IFAT) of an initial screen of pooled strains. Reactive samples were then tested for individual members of the pool. Pool A (the only reactive pool) contained four strains of L. pneumophila serogroup 1, one strain each of L. pneumophila serogroups 2 and 3, and two strains of L. pneumophila serogroup 4.

Legionella PCR, detecting 16S ribosomal RNA gene sequences was performed according to the method of Jonas et al. The identity of PCR products was confirmed by automated sequencing.

Environmental investigations—The environmental investigation of an implicated site included site visits, review of documents, testing spa pool water temperatures, and measuring Free Available Chlorine Levels with a Hach DR 100 colorimeter (Hach Company, Loveland, Colorado).

Environmental samples for Legionella testing were obtained at the same time. Water samples (1 litre) were taken and filters and biofilm scums were swabbed aseptically from the three display spa pools at the retail premise. All samples were immediately transported to ESR Porirua for Legionella culture and direct fluorescent antibody detection of Legionella.

Results

Clinical and microbiological—Blood and sputum culture results were negative on all six patients, as was Legionella urinary antigen. One patient was found to have a distinct diagnosis of respiratory syncytial virus. Diagnosis of the remaining five cases was on the basis of serology.

Convalescent serology confirmed that three patients had been infected with L. pneumophila, and two were infected with L. longbeachae serogroup 1. Initially, sputum Legionella PCR results were negative on all but one patient. However, repeat PCR testing, repeated after the serology results were obtained, indicated that three patients had been infected with Legionella pneumophila.
Epidemiological—As serotyping indicated that the three cases of *L. pneumophila* developed antibody responses to *L. pneumophila* serogroup 2, this lead us to search for a common source of infection.

A summary of all the six cases in the cluster and their final diagnoses is shown in Table 1.

<table>
<thead>
<tr>
<th>Patient identification</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>82</td>
<td>52</td>
<td>57</td>
<td>65</td>
<td>74</td>
<td>55</td>
</tr>
<tr>
<td>Sex</td>
<td>M</td>
<td>M</td>
<td>M</td>
<td>M</td>
<td>M</td>
<td>M</td>
</tr>
<tr>
<td>Area of residence</td>
<td>Wellington</td>
<td>Upper Hutt</td>
<td>Mana</td>
<td>Lower Hutt</td>
<td>Upper Hutt</td>
<td>Upper Hutt</td>
</tr>
<tr>
<td>Severity of illness</td>
<td>Severe (hospital admission)</td>
<td>Life-threatening (ICU admission)</td>
<td>Severe (hospital admission)</td>
<td>Life-threatening (ICU admission)</td>
<td>Life-threatening (ICU admission)</td>
<td>Life threatening (ICU admission)</td>
</tr>
<tr>
<td>Legionella IFAT</td>
<td>1:64 to 1:256</td>
<td>1:128 to 1:1024</td>
<td>1:64 to 1:2048</td>
<td>1:128 to 1:2048</td>
<td>&lt;1:64 to 1:256</td>
<td>equation</td>
</tr>
<tr>
<td>Legionella serogroup</td>
<td><em>L. pneumophila</em> serogroup 1-4</td>
<td><em>L. pneumophila</em> serogroup 2</td>
<td><em>L. pneumophila</em> serogroup 2</td>
<td>NOT Legionella</td>
<td><em>L. longbeachae</em> serogroup 1</td>
<td><em>L. longbeachae</em> serogroup 1</td>
</tr>
<tr>
<td>Legionella PCR</td>
<td>Sputum, NP swab +ve</td>
<td>Sputum +ve</td>
<td>Sputum, blood, NP +ve</td>
<td>Nil specimens</td>
<td>NP -ve</td>
<td></td>
</tr>
<tr>
<td>Interpretation</td>
<td>Probable Lp sg 2</td>
<td>Lp sg 2</td>
<td>Lp sg 2</td>
<td>RSV</td>
<td><em>L. longbeachae</em> sg 1</td>
<td><em>L. longbeachae</em> sg 1</td>
</tr>
</tbody>
</table>

Note: All Legionella urinary antigen tests were negative; ICU=intensive care unit; IFAT = immunofluorescence antibody testing; Lp sg = Legionella serogroup; NP = nasopharyngeal; PCR = polymerase chain reaction; RSV IF = respiratory syncytial virus; +ve = positive; –ve = negative

No common elements were identified in the initial exposure histories obtained from all six cases. All cases were questioned about visits to any commercial buildings, and no commonalities were found. Case C was re-visited following discharge from hospital and confirmation of *Legionella* diagnosis, and the history was reviewed with the focus on links between the more tightly defined cluster.

He recalled visiting a Lower Hutt retail store that had operating spa pools on display, and specifically remembered having stopped to examine one of the display pools. Display spa pools have been previously associated with a *Legionella* outbreak in New Zealand, involving another branch of the same company in Auckland, as described in the Discussion.

The other two cases (A and B) in the cluster were re-contacted and asked specifically about any visits to the store in question. Both recalled having visited the store during their likely incubation periods (one had retained a purchase docket which confirmed the date of the visit), which further increased suspicion that the store in question was the source of *Legionella* infection.

Environmental—An initial visit was made to the Lower Hutt retail store which revealed that three display spa pools there were operating with heated water and air blower nozzles. They were located in a group in the centre of the retail shop. It was stated that the water in the display pools was maintained, and tested on a daily basis to
ensure adequate levels of free chlorine were present. There were no records available to confirm this.

Consequently, for each pool, RPH staff took water temperatures and measured Free Available Chlorine levels. The Free Available Chlorine levels in the three display spa pools were 0.15–0.8 mg/L, below the level recommended for bacterial control (2 mg/L). Water temperatures were 32–37°C, which would support growth of *Legionella* bacteria.

A written copy of the company’s water treatment procedures was provided later. These procedures did not specify the frequency of water treatment, but did require that water pH and Free Available Chlorine levels be tested and recorded on a daily basis. The staff confirmed that every store was expected to follow these procedures for their operating display pools. However, they were unable to show records of daily water testing for the Wellington store.

*Legionella pneumophila* serogroup 1 was cultured from all three samples taken from one of the pools. Samples from the other two pools were culture-negative but positive for *Legionella* antigen on direct fluoroscopic testing (Table 2).

**Table 2. Water test results from the display spa pools at the Lower Hutt retail store**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pool 1</th>
<th>Pool 2</th>
<th>Pool 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water temperature (°C)</td>
<td>32</td>
<td>36</td>
<td>37</td>
</tr>
<tr>
<td>Free chlorine (mg/L)</td>
<td>0.4</td>
<td>0.15</td>
<td>0.8</td>
</tr>
<tr>
<td>Microbiological culture</td>
<td><em>L. pneumophila</em> sg 1</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>Direct fluorescent antibody</td>
<td><em>Legionella</em> antigen detected</td>
<td><em>Legionella</em> antigen detected</td>
<td><em>Legionella</em> antigen detected</td>
</tr>
</tbody>
</table>

In view of these findings, the display spa pools were felt to be the most likely source of *Legionella* infection in the three cases that had visited the Lower Hutt store. *Legionella* isolated from the pools was of a different serotype to that identified in at least two of the cases (B and C).

The display spa pools were thought to be the most likely source of infection, since the chemical tests indicated the water was inadequately treated and microbiological tests confirmed the presence of *Legionella* bacteria. It is plausible that a different strain of *Legionella* was present in the pools at the time the cases were exposed 3 months prior to the water samples being taken. The store staff stated that the water had been changed in all three pools within the 3 months prior to RPH’s visit, but they had not been disinfected during this time.

**Control measures**—Further action addressed both the immediate and the longer-term health risks arising from the pools.

The store was requested to empty all three operating spa pools and thoroughly disinfect all pool surfaces (including pipes). A fourth display pool that had been present in the store during the cases’ exposure, and had subsequently been sold, was recalled by the store (it had not been operated since being dismantled and sold). The
store manager denied that any staff had been unwell or suffered from a cough or shortness of breath in the preceding few months.

This cluster was the second time a *Legionella* outbreak had been associated with one of this company’s stores. The previous outbreak was in Auckland and is mentioned in the Discussion. RPH advised the company should develop adequate water treatment and monitoring processes to address the potential public health risk posed by the display spa pools. The company responded by emptying all operating display spa pools, and subsequently deciding that they would no longer operate display spa pools in their stores.

The occurrence of two outbreaks within 2 years raised the possibility that other stores with display pools might also be a potential source of *Legionella* infection. A letter was sent to other spa pool retailers in the Wellington area, outlining the health risks of *Legionella* infection associated with inadequately treated spa pool water. The letter emphasised the importance of adequate water treatment.

**Discussion**

This investigation highlights the challenges of investigating a cluster of severe respiratory illness. Several factors were suggestive of a common source *Legionella pneumophila* outbreak involving all six of the identified patients. Further investigation found that there were three separate aetiologies for this temporal cluster of severe respiratory illness. This redefined the cluster of six cases into a series of three cases with a possible common organism and a common source, and three other unrelated cases of severe pneumonia.

This situation was identified on the basis of an unusual cluster of severe pneumonia in spring, in the absence of known outbreaks of other respiratory diseases. As is common with severe pneumonia, it was hard to establish a rapid microbiological diagnosis, and it was hard to obtain full histories until the patients recovered.

The rapid diagnosis of *Legionella pneumophila* can be difficult, and this presents challenges for the timely investigation of potential outbreaks. Urinary antigen testing is a sensitive and rapid test, however it only detects *Legionella pneumophila* serogroup 1.

Unfortunately, no respiratory samples yielded *Legionella* in culture, because it was not possible to obtain samples prior to starting antibiotics. Serological confirmation of legionellosis is slow—as both an acute and convalescent titre are required. Repeat interviews and follow-up calls on specific issues were needed before the most likely source of infection could be determined.

50 to 100 cases of Legionnaires’ disease occur in New Zealand each year, of which around 10 to 20 are due to *Legionella pneumophila*. The majority of these cases are thought to be sporadic.

*Legionella pneumophila* is ubiquitous in environmental water systems, including rivers, lakes, and streams. Under normal environmental circumstances, bacterial numbers are low, and the risk of infection in an immunocompetent individual is extremely small. However, bacterial proliferation is enhanced by warmer temperatures, high levels of sediment (particularly calcium and magnesium), and the presence of symbiotic microorganisms—conditions that often occur in artificial water
systems. And with increased bacterial numbers, aerosol particles from the infected water supply may act as a means of bacterial transmission to humans.

Outbreaks of Legionnaires’ disease have been associated with water systems in hospitals and cruise ships, cooling towers, mist machines, nebulisers, and spa pools. Legionella outbreaks are almost always associated with Legionella pneumophila. Within New Zealand, one previous outbreak had been attributed to a spa pool on display in a commercial store in Auckland almost 2 years previously.

The Auckland outbreak involved three cases with L. pneumophila serogroup 2, two of whom had visited the store in question; one of these cases later died.

Investigations undertaken by the Auckland RPH Service had found Legionella in a display spa pool at the Auckland store; this was found to be genotypically identical to the Legionella isolated from the three cases. The company owning the store had been required to disinfect the spa pool in question, and review the water treatment procedures used in its stores nationwide.

The close temporal clustering of the three cases described in the present paper raised suspicion of a possible common source of infection. These three cases had visited the store during the 10 days prior to becoming unwell; together with the evidence of bacterial contamination, this makes a strong case that a display spa pool was the likely source of infection.

Note that none of the three affected individuals who visited the store actually entered a spa pool at the store and none owned a spa pool at home. Legionnaires’ disease has previously been documented following very transient exposure to an infective source. This suggests that even a small number of bacteria is sufficient to cause disease in susceptible individuals. The production of aerosols from a contaminated water supply appears to be a significant contributing factor in bacterial transmission.

As far as we know, none of the staff working in the store developed Legionnaires’ disease, despite their regular proximity to the contaminated spa pools. We chose not to screen staff for serological evidence of Legionnaires’ disease (staff screening had been undertaken as part of the Auckland outbreak investigation, and no evidence of infection had been found).

Outbreaks of Legionella typically have a relatively low number of cases compared to number of people exposed: host factors including older age and smoking increase the susceptibility of some individuals to infection.

There is a theoretical risk that other spa pools may also act as sources of Legionella infection, both in commercial stores and in private homes. We are unaware of any cases of Legionnaires’ disease arising from contaminated spa pools in private homes; it may be that private owners are more vigilant than retail staff in changing and treating spa water.

The store we investigated did not specialise in spa pools, but rather acted as the retail outlet for an independent spa pool supplier. Staff in more specialised stores may have greater awareness of the potential for Legionella contamination in spa pool water. The Standard NZS 5826:2000 Pool Water Quality sets out chemical water quality criteria applicable to all spa pools.
It is recommended that spa pool manufacturers provide clear information on the risk of *Legionella pneumophila* proliferating in poorly chlorinated warm water, and outline the procedures required to avert this. Retailers should be made aware that customers and staff who walk close to a poorly maintained display spa pool may be at risk of legionellosis, and that this can cause a severe and potentially fatal illness.

Investigation of the above outbreak led to the identification and control of a potentially ongoing public health risk. This discovery highlights the importance of routine surveillance, vigilance, and effective communication on the part of the public health community, as well as effective liaison with clinical and laboratory colleagues.

We cannot categorically conclude that the three cases of *Legionella pneumophila* in this cluster were related. However, the investigation revealed a systematic failure in spa pool maintenance, and the investigation resulted in a corrective action recommendation. This maintenance deficit increased the likelihood and our suspicions that that these display spa pools were the source of the *Legionella* infections.

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**References:**


An Auckland regional audit of the nurse-led rheumatic fever secondary prophylaxis programme

Sarah Grayson, Margaret Horsburgh, Diana Lennon

Abstract

**Background** Rheumatic fever with ensuing rheumatic heart disease is considered to be a preventable chronic disease.

**Aim** To assess the compliance rates with the rheumatic fever secondary prophylaxis programme established through the Auckland Rheumatic Fever Register and managed by community nursing services in Auckland, New Zealand.

**Methods** An audit of the 1998 and 2000 Auckland Rheumatic Fever Register data was undertaken to establish the compliance rates of patients with the rheumatic fever secondary prophylaxis programme. The sample included all patients on the Auckland Rheumatic Fever Register during this time.

**Results** Results showed compliance rates across the three Auckland DHBs ranging from 79.9% to 100% for individual community nursing offices.

**Conclusion** A community-based nurse-led secondary prophylaxis programme for Rheumatic Fever heart disease is able to deliver excellent patient compliance levels. Secondary prophylaxis is the WHO-recommended cost effective first step to Rheumatic Fever/Rheumatic Heart Disease control. Community health workers have a key role to play in facilitating this compliance.

Rheumatic fever has largely disappeared in the developed world. In areas where rheumatic fever and its ensuing rheumatic heart disease still exists, the World Health Organization (WHO) recommends the use of secondary prevention (the most cost-effective measure) as the first step towards rheumatic heart disease control.

Regular benzathine penicillin reduces the risk of recurrent acute rheumatic fever by 87–96%. The attack rate of rheumatic fever after the triggering event of a group A streptococcal throat infection rises from 1 to 3% with the first attack to 25 to 75% with subsequent attacks.

Such recurrent acute rheumatic fever episodes compound the valvular damage produced by the initial episode, and increase chronic rheumatic heart disease severity.

In the greater Auckland region, a confirmed episode of rheumatic fever is notified to the Medical Officer of Health, and the patient is listed on the Auckland Rheumatic Fever Register and commenced on the rheumatic fever secondary prophylaxis programme. Community nursing services across greater Auckland deliver the rheumatic fever secondary prophylaxis programme.

The Auckland Rheumatic Fever Register was set up in 1981 to generate benzathine penicillin prescriptions for patients with rheumatic fever. Its purpose is to streamline parenteral benzathine penicillin delivery, and prevent further episodes of rheumatic
fever. This has been highly successful in Auckland and other parts of New Zealand. With a clinic-based oral penicillin prophylaxis programme in Auckland, recurrent attacks accounted for 20% of hospital admissions, reducing to 6% with a parenteral penicillin community nurse delivered programme at instigation, and more recently also seen in other parts of New Zealand.

The parenteral route per se has been shown to be more effective. Patients or caregivers sign a consent form to allow for the delivery of the secondary prophylaxis programme of intramuscular benzathine penicillin every 28 days.

Registered nurses, working from community (district) nursing offices, deliver (under delegated authority) 3-monthly prescriptions of parenteral benzathine penicillin. This may occur at school (the majority), home, or work, or at a community nurse-run clinic. Syringes are pre-filled, and patients remain on the programme at least for 10 years or until 21 years of age. Counties Manukau District Health Board also employ Maori and Samoan community health workers to facilitate education and compliance.

This study, undertaken as part of a Masters degree, aimed to determine the compliance rates (achieved by the community nursing offices in Auckland) of patients enrolled on the rheumatic fever secondary prophylaxis programme. It also aimed to identify factors impacting on these compliance rates.

Methods

This study analysed the previously uncollated data from patients on the Auckland Rheumatic Fever Register (1998 and 2000) and established levels of compliance with the secondary prophylaxis programme for the three Auckland District Health Boards (DHBs) and the individual community district nursing offices.

The study was undertaken as a project for a Masters degree as a one-off study. The years of study reflected the student’s availability and the knowledge that while data had not been analysed for these years there was anecdotal reports that compliance levels had deteriorated. There is no regular budgeted quality audit of this programme.

Ten community nursing offices (Table 1) each relate to geographic areas with the exception of an office operating from a Starship Hospital base. This office provides care for children across the Auckland District Health Board area. This sole paediatric office has a proactive approach to following up patients and ensuring they receive their injections. This may result in injections being given early, between 21 and 28 days.

Table 1. Greater Auckland District Health Board Community Offices delivering the Rheumatic Fever Secondary Prophylaxis Programme (1998–2000)

<table>
<thead>
<tr>
<th>District Health Boards (DHBs)</th>
<th>Community Offices</th>
</tr>
</thead>
<tbody>
<tr>
<td>Counties Manukau DHB</td>
<td>Orakau Rd</td>
</tr>
<tr>
<td></td>
<td>Howick</td>
</tr>
<tr>
<td></td>
<td>Manurewa/Papakura</td>
</tr>
<tr>
<td></td>
<td>Pukekohe</td>
</tr>
<tr>
<td>Auckland DHB</td>
<td>Starship</td>
</tr>
<tr>
<td></td>
<td>Waterview</td>
</tr>
<tr>
<td></td>
<td>Greenlane</td>
</tr>
<tr>
<td>Waitemata DHB</td>
<td>Waitakere Hospital</td>
</tr>
<tr>
<td></td>
<td>North Shore Hospital</td>
</tr>
<tr>
<td></td>
<td>Rodney Community Health</td>
</tr>
</tbody>
</table>

All the offices were included in the study, with the exception of the Rodney office, which had not collected data for the study period.
Numbers of patients on the Rheumatic Fever Register vary across the community nursing offices (in this study n = 11 to 205; mean = 56). Each nursing office records information on specially designed forms when prophylactic injections are given.

Patient forms were coded to record how many injections each patient received during the calendar year, how many injections were early or late, and the number of days injections were late. All the patients in the Auckland region currently on the benzathine penicillin prophylaxis regime for whom compliance data had been collected were included. A small number of compliance forms were not included, as data was poor or incomplete or the nursing office had not completed the forms. For 1998, there were 3 incomplete forms and for 2000 there were 10.

To calculate the average compliance rates for each community nursing office and DHB, the number of injections per patient for the year in question was divided by the total number of injections they should have received for that year and expressed as a percentage according to pre-set guidelines (Table 2). The individual patient percentages were then analysed to provide an overall mean and standard deviation for each office and DHB.

Table 2. Guidelines for assessing compliance data

- Each patient on the 28-day regime should receive 12–13 injections per year.
- Each patient on the 21-day regime should receive 17 injections per year.
- 12/12 means due to late start in January only 12 injections were possible for that calendar year (there are no exceptions to this, that is if patient starts before 20th January but only receives 12 injections their total will read 12/13 not 12/12).
- On time means injection given ≤5 days after due date of injection.
- Early means injection given ≥3 days prior to due date of injection. In exceptional circumstances only a benzathine penicillin injection may be given 7–14 days early, and the next injection due date then falls 28 days from the date of early injection (this guideline is approved by prescribing physicians for the Auckland Rheumatic Fever Register).
- Where a patient is new to the service their total will be out of the number of injections they should have received during the year. Compliance percentages will still apply but may be influenced by a shorter time for non-compliance to occur.

*Standard of care is benzathine penicillin every 28 days.*

Compliance was defined as:

- **Total compliance**—administration of all scheduled injections within predetermined time frames.
- **Partial compliance**—lateness of any injection delivery (see Table 2), or omission of one or more of the possible injections scheduled for the year omitted. Furthermore, patients who were receiving injections but elected at some point during the year of data collection to stop treatment for reasons not documented on data forms, and unknown to the healthcare teams, were recorded as falling into the partial compliers category.
- **Non-compliance**—missing all injections for the whole year.

In addition, a qualitative study using a semi-structured interview format was conducted with a sample of nine individual nurses from the different community nursing offices. These interviews conducted by the principal researcher (SG) allowed an understanding of any issues that may impact on the delivery of the programme to be gained. Interviews were transcribed, coded with a sequential number, and analysed using a general inductive approach.

The quantitative component of the study was conducted under the ethical approval obtained when the Auckland Rheumatic Fever Register was commenced in 1981. An approval letter from was obtained from the Chair of the Ministry of Health Auckland Ethics Committee.
In addition, approval to conduct the interviews was obtained from the University of Auckland Human Subjects Ethics Committee immediately prior to this study.

Results

In 1998 and 2000, both Counties Manukau DHB and Auckland DHB had an average fully compliant rate of approximately 96%. In 1998, Waitemata DHB had an average compliance rate of 92% falling to 86% in 2000 (Figure 1).

The numbers for Waitemata DHB are, however, small (1998: n=24; 2000: n=11). The sample size for Waitemata District Health Board is reduced by the non-inclusion of the Waitakere (for 1998) and Rodney (1998 and 2000) nursing offices in the final sample size due to the non-availability of data, thus making the rate for Waitemata District Health Board compiled from only one nursing office. The sample size for the Auckland DHB in 2000 was also reduced by the non-inclusion of one nursing office, due to the non-collection of data in 2000.

Figure 1. Full compliance rates (%) for the three Auckland District Health Boards

Rates for partially compliant patients varied. A patient may well receive all of the injections that are prescribed for them in one calendar year however they may have received them consistently late, thus allowing a time lapse that is far greater than recommended. Overall, the number of partially compliant patients in each office was low, with some patients appearing on multiple occasions.

When comparing the compliance rates of the rheumatic fever secondary prophylaxis programme, more variation is seen between the individual community nursing offices than between the cumulative rates for each DHB (Figure 2).
The compliance rates for the nine nursing offices range between 79.9 and 100%, with patient numbers for the individual offices ranging from n=11 to n=205. A few partially compliant patients may, however, distort the figures for those offices with low patient numbers.

Figure 2: Average full compliance rates (%) for Greater Auckland District Nursing offices

The sole paediatric-only community nursing office (Starship in Figure 2) achieved high compliance rates (97.9%–99.2%) with reasonably high patient numbers (n=69–92) in comparison to other nursing offices. Their proactive approach of following up patients at 21 days also resulted in 3% of injections being given early, between 21 and 28 days.

Interviews with the nurses identified that the factors appearing to impact positively on the delivery of the programme included the presence of community health workers actively involved in the programme. Other factors identified by the nurses ensuring...
success of the programme included communication from other services used by rheumatic fever patients (e.g. availability of outpatient appointment letters) and a part-time rheumatic fever resource nurse available in some offices who had an education role with dedicated time.

**Discussion**

Community nursing services within the three Auckland DHBs achieve high levels of compliance with the rheumatic fever secondary prophylaxis programme: 86–96% of the nurses delivering ~6,000 penicillin injections to 450 patients per year.

The community-based nurses are providing an efficient way to deliver the rheumatic fever secondary prophylaxis programme and to prevent the devastating long-term effects of rheumatic fever recurrences.

Cardiac transplantation due to severe rheumatic heart disease is an avoidable expense. In New Zealand, rheumatic fever is closely associated with low socioeconomic families who frequently move residence. Community-based nurses are flexible and able to be innovative in tracking patients. They are experienced in delivering a community-based programme (sometimes with the help of community health workers who are particularly valuable in engaging Maori and Pacific families where the highest incidence of rheumatic fever is found).

On-time and regular delivery of benzathine prophylaxis is essential. Indeed, missed or late injections lead to recurrences of rheumatic fever. Some countries advocate benzathine penicillin every 21 days, as penicillin levels up to 28 days can be unreliable. Although patients may receive all of their injections in one year in some instances, they may receive them consistently late. The actual time of penicillin coverage may therefore be poor so the risk of contracting a further Group A streptococcal infection is therefore increased.

While the community-based healthcare delivery data collected for this study was as complete as possible there were records that were incomplete. However, these were few and should not have greatly affected the final results. A limitation of the study is the fact that non-compliant patients who had no data form completed for them have not been included. In addition, patients and families were not interviewed.

Secondary prevention of second and subsequent attacks of acute rheumatic fever with benzathine penicillin is efficacious and cost-effective. This community nurse-led programme with a rheumatic fever register follows WHO guidelines and has led to reduced rheumatic fever recurrence rates with expected attendant reduced carditis as seen elsewhere. High compliance rates with parenteral penicillin are difficult to achieve but are vital for this health intervention.

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**Acknowledgements:** We thank the Community Nurses (District Nurses) who deliver the secondary prevention programme as well as Robyn Buchanan (Auckland Rheumatic Fever Register Coordinator).
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References:


Influenza immunisation rate for 2005 and factors associated with receiving this vaccine in patients aged 65 years and over admitted to a general medical ward at Auckland City Hospital

Elizabeth Curry, Nathan Kerr, Joseph Yang, Simon Briggs

Abstract

Aim To assess the influenza immunisation rate for 2005 in patients aged 65 years and over admitted to a general medical ward at Auckland City Hospital, New Zealand; to identify factors associated with receiving this vaccine; and to assess whether particular patient groups have a low influenza immunisation rate.

Method Consecutive patients aged 65 years and over admitted to two medical wards were surveyed. Demographic data, how recently patients had last seen their general practitioner (GP), whether patients had received an influenza vaccine reminder from their GP, and whether patients had received the influenza vaccine in 2005 were recorded. Logistic regression analysis was performed to investigate which variables were associated with receiving the influenza vaccine.

Results 148 of 200 (74%) patients who answered the questionnaire received the influenza vaccine. The variables found to be associated with receiving the influenza vaccine were whether patients had seen their GP in the last 6 months and whether patients had received an influenza vaccine reminder from their GP.

Conclusion Three-quarters of patients in this study received the influenza vaccine. We have not been able to identify patient groups that have a low influenza immunisation rate. Reminding patients of the benefits of the influenza vaccine or offering this at the time of discharge from hospital as autumn approaches each year may increase the influenza immunisation rate of those recently hospitalised.

Influenza immunisation has been shown to reduce cases of influenza by 60–70%; it is associated with a reduced risk of hospitalisation and mortality during influenza seasons. In 1997, the Ministry of Health commenced a free influenza immunisation programme for those aged 65 years and over. This was extended in 1999 to those aged under 65 years with certain chronic medical conditions.

We undertook this study to assess the influenza immunisation rate for 2005 in patients aged 65 years and over admitted to a general medical ward at Auckland City Hospital, as well as to identify factors associated with receiving this vaccine. If we were able to identify patient groups that had a low influenza immunisation rate, then strategies to increase this rate could be targeted at these groups.

Method

Consecutive general medical patients aged 65 years and over admitted to two general medical wards were asked to answer a questionnaire. The questionnaire assessed ethnicity, whether English was the patient’s first language, place of residence, number of hospital admissions in the last year, how recently
the patient had last seen their general practitioner (GP) (we excluded the most recent visit if this had resulted in the current hospital admission), whether they knew of the influenza vaccine, whether they received an influenza vaccine reminder from their GP, and whether they had received this vaccine in 2005. If they had not received the influenza vaccine then the reasons for this were documented. Interpreters were used when required.

To investigate which variables were associated with the probability of receiving the influenza vaccine, logistic regression analysis was used with immunised or not immunised as the binary outcome and age, gender, ethnicity, place of residence, whether English was the patients first language, whether the patient had been admitted to hospital in the last year and whether the patient had seen their GP in the last six months included as explanatory variables. A separate analysis was run including only those patients living in their own home with whether the patient had received an influenza vaccine reminder from their GP included as an additional explanatory variable. Statistical analysis was performed using SPSS v14.0 statistical software (SPSS Inc., Chicago, Illinois).

Ethical approval was granted by the Northern X Regional Ethics Committee.

Results

During an 8-week period in August and September 2005, 329 patients aged 65 years and over were admitted to the targeted wards. 200 (61%) patients consented to answer the questionnaire; 62 patients were unable to consent due to dementia, delirium or severe illness, 14 declined to answer the questionnaire, and 53 were discharged from hospital or died prior to questioning.

148 of the 200 patients (74%, 95% CI 68-80%) had received the influenza vaccine in 2005.

Table 1. Percentages of patients who received the influenza vaccine in 2005 for each of the explanatory variables included in the logistic regression analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Categories</th>
<th>Number</th>
<th>Number immunised</th>
<th>Percentage immunised</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)*</td>
<td>65 to 74</td>
<td>63</td>
<td>48</td>
<td>76 %</td>
<td>0.61</td>
</tr>
<tr>
<td></td>
<td>75 to 84</td>
<td>87</td>
<td>62</td>
<td>71 %</td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥85</td>
<td>50</td>
<td>38</td>
<td>76 %</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Female</td>
<td>97</td>
<td>68</td>
<td>70 %</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>103</td>
<td>80</td>
<td>78 %</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td>NZ European</td>
<td>139</td>
<td>107</td>
<td>77 %</td>
<td>0.34</td>
</tr>
<tr>
<td></td>
<td>Maori</td>
<td>5</td>
<td>3</td>
<td>60 %</td>
<td></td>
</tr>
<tr>
<td></td>
<td>European</td>
<td>25</td>
<td>16</td>
<td>64 %</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pacific Islands</td>
<td>19</td>
<td>12</td>
<td>63 %</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>12</td>
<td>10</td>
<td>83 %</td>
<td></td>
</tr>
<tr>
<td>Living in a residential care facility</td>
<td>Yes</td>
<td>20</td>
<td>13</td>
<td>65 %</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>180</td>
<td>135</td>
<td>75 %</td>
<td></td>
</tr>
<tr>
<td>English 1st language</td>
<td>Yes</td>
<td>163</td>
<td>124</td>
<td>76 %</td>
<td>0.40</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>37</td>
<td>24</td>
<td>65 %</td>
<td></td>
</tr>
<tr>
<td>Hospital admission in last year</td>
<td>Yes</td>
<td>113</td>
<td>88</td>
<td>78 %</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>87</td>
<td>60</td>
<td>69 %</td>
<td></td>
</tr>
<tr>
<td>Saw GP within last 6 months†</td>
<td>Yes</td>
<td>186</td>
<td>143</td>
<td>77 %</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>12</td>
<td>4</td>
<td>33 %</td>
<td></td>
</tr>
<tr>
<td>Received GP reminder‡§</td>
<td>Yes</td>
<td>85</td>
<td>73</td>
<td>86 %</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>90</td>
<td>61</td>
<td>68 %</td>
<td></td>
</tr>
</tbody>
</table>

*Age was entered into the logistic regression analysis as a continuous variable; †Data were not available for 2 patients; ‡Patients living in a long term care facility excluded; §Data were not available for 5 patients.
The percentages of patients who received the influenza vaccine for each of the explanatory variables included in the logistic regression analysis are shown in Table 1. The variables associated with receiving the influenza vaccine were whether patients had seen their GP in the last 6 months and whether patients had received an influenza vaccine reminder from their GP.

Fifty-two patients (26%) did not receive the influenza vaccine. Thirty-five patients actively chose against receiving this vaccine. The reasons given were concerns that the vaccine may cause influenza or significant side effects (n=22) and thoughts that immunisation was not necessary (n=11). No reason was recorded in two cases.

The reasons given by the remaining 17 patients for not receiving the influenza vaccine were no knowledge of the vaccine (n=7), forgetting to obtain the vaccine from their GP (n=8), or an inability to get to their GP (n=1). No reason was recorded in one case.

The ethnicity of the seven patients who had no knowledge of the influenza vaccine was Samoan (n=3), Rarotongan (n=2), European (n=1), and NZ European (n=1).

**Discussion**

The influenza immunisation rate in our study population (patients admitted to a general medical ward) in 2005 was 74%. While this is higher than the New Zealand-wide influenza immunisation rate of 61% in those aged 65 years and over in 2005, our study population is not representative of the general population.

Reasons for the higher rate in our study population could include an increased awareness that influenza may cause severe illness; and an increased contact with a GP, other doctor, or health professional who may recommend the influenza vaccine.

Patients who had seen their GP in the last 6 months or who had received an influenza vaccine reminder from their GP were more likely to have received the influenza vaccine. However identifying patients who had not seen their GP in the last 6 months or who did not receive an influenza vaccine reminder from their GP would not allow us to easily target a significant proportion of un-immunised patients.

Given the above findings, one way that hospital-based doctors may increase influenza immunisation rates in recently hospitalised patients is to remind un-immunised eligible patients of the significant benefits of this vaccine and of its availability free of charge from their GP. Alternatively, the influenza vaccine could be offered to these patients at the time of discharge from hospital at appropriate times of the year. This type of intervention has been shown to increase influenza immunisation rates elsewhere. Discussion with GPs, who are the primary providers of influenza immunisation, would need to occur if this intervention was considered.

The reasons given by the patients in this study for not receiving the influenza vaccine have been found in other studies. Ongoing efforts are needed to educate individuals of the benefits of the influenza vaccine and to address concerns regarding its safety.

This study has several limitations, including the small sample size, the study design (which could have resulted in inaccuracies due to impaired recall), and the low questionnaire response rate of 61%. Due to the small number of Maori and Pacific Islands people in this study, we were unable to assess whether there were differences in influenza immunisation rates in these groups of our study population. As already
stated, this study’s population of hospitalised patients is not representative of the general population; it may also not be representative of other hospital populations where patient demographics may differ.

While it is encouraging that three-quarters of patients in this study received the influenza vaccine, there is still an opportunity to increase this rate further. We have not been able to identify patients groups that have a low influenza immunisation rate. Reminding patients of the benefits of the influenza vaccine, or offering this at the time of discharge from hospital as autumn approaches each year, may increase the influenza immunisation rate of those recently hospitalised.

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**Acknowledgment:** We acknowledge and thank Joanna Stewart (Biostatistician, School of Population Health, University of Auckland) for her assistance.

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**References:**


Criteria for prioritising access to healthcare resources in New Zealand during an influenza pandemic or at other times of overwhelming demand

Michael Ardagh

Abstract

During overwhelming demand for resources, such as during an influenza pandemic, clinicians may be required to deny some patients access to a resource (for example ventilation, or hospital admission). However, no pragmatic guidance exists to help clinicians do this. This paper presents criteria for the prioritisation of access to resources during overwhelming demand. The criteria are in the form of eight questions related to the resource and the patients competing for it and are intended to be sufficiently comprehensive and sufficiently succinct to be useful to clinicians who might be required to make such decisions.

The emergence of a novel avian influenza virus (influenza A H5N1) has given impetus to planning for another influenza pandemic.

Ethical issues arising during planning for, and responding to, a pandemic include: a possible need to restrict personal freedoms (quarantining and border control); the extent of the 'duty of care' of healthcare workers who are putting themselves at risk of infection in the course of their duties; and the distribution of overwhelmed healthcare resources (which are likely to be depleted due to illness among the healthcare workforce).

Overwhelming of healthcare resources may also occur at other times of increased demand, such as after a natural or man-made disaster. This paper is concerned with prioritising access to healthcare resources at times of overwhelming demand, with a particular focus on an influenza pandemic.

Some useful publications about the incorporation of ethical values in pandemic planning (e.g. Stand on guard for thee. Ethical issues in preparedness planning for pandemic influenza. University of Toronto, 2005) have been published. Other publications give decision-making guidance during a pandemic such as The Siracusa Principles (which give guidance on the restriction of individual freedoms), and the work of Lo and Katz (which gives several general guiding principles about a variety of ethical issues during a pandemic).

Hick and colleagues, and Koenig and colleagues in an accompanying editorial, introduced discussion of the distribution of mechanical ventilators in an epidemic of respiratory disease. However, pragmatic guidance is limited and is variably relevant to the clinician ‘on the ground’, making decisions about real patients during a pandemic. In particular, there is a paucity of useful, succinct advice for clinicians who must prioritise access to overwhelmed resources such as emergency department cubicles, hospital beds, intensive care beds, and antimicrobial drugs, among others.
This paper will present a list of criteria, in the form of eight questions, to aid the clinician in prioritisation of patients for access to resources during a pandemic, or at other times of overwhelming demand. It is intended as a practical guide for clinicians and to be used to aid the application of overarching ethical values and processes for pandemic planning.¹

Although the focus of this paper is the context of an influenza pandemic, the criteria will have application to other situations where there is overwhelming demand for limited healthcare resources.

**Prioritisation criteria**

Eight questions are considered by clinicians who are required to prioritise access to a resource for one or more competing patients. Each question achieves relevance after answering the questions asked before. After some of the questions, the decision regarding access to the resource may be answered, otherwise the clinician proceeds to the next question.

- **Questions 1 and 2** ‘set the scene’ by verifying that there are people who ideally would access the resource in question, but there are too many people for the resource to accommodate.
- **Questions 3 and 4** seek to identify who might be taken out of the competition for the resource, without coming to harm, by accessing suitable alternative care, or by deferring access to care.
- **Question 5** seeks to expand the capacity of the resource so that more might be accommodated.
- **Question 6** accepts that, after alternatives are exhausted, those that can be deferred have been deferred, and the resource has been expanded, there are still too many worthy patients for the resource to accommodate. It then seeks to mitigate any harms that will ensue if patients miss out on accessing the resource.
- **Question 7** ‘ranks’ patients according to the ‘net benefit’ of access to the resource, and
- **Question 8** outlines methods for prioritisation if individuals cannot be ranked relative to each other.

A reference, or ‘wall chart’ summary, is reproduced in Tables 1 and 2.
Table 1. Criteria for prioritising access to resources during overwhelming demand

<table>
<thead>
<tr>
<th>Question Type</th>
<th>Description</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal threshold question</td>
<td>Does the patient meet the clinical criteria for access to the resource during normal times (that is, when there is not overwhelming demand for the resource)?</td>
<td>No – patient doesn’t access the resource. Yes – go to question 2.</td>
</tr>
<tr>
<td>Competition question</td>
<td>Are there other patients who meet the normal clinical criteria (as per question 1), who are competing for the same resource (which is currently insufficient to accommodate all of the patients who are competing)?</td>
<td>No – patient accesses the resource. Yes – go to question 3.</td>
</tr>
<tr>
<td>Alternative options question</td>
<td>Can any of the competing patients (including those who are already using the resource) have alternative care which, although perhaps not the first choice, will provide reasonably similar benefit to the patient and not cause significant harm due to accessing the alternative rather than the original choice?</td>
<td>Yes – appropriate patient(s) access the alternative care. No – for any remaining patients, go to question 3.</td>
</tr>
<tr>
<td>Deferability question</td>
<td>Can any of the competing patients have their access to the resource deferred to a future time when demand is likely to be less, without coming to significant harm.</td>
<td>Yes – defer access. No – for any remaining patients, go to question 4.</td>
</tr>
<tr>
<td>Expansion question</td>
<td>Can the resource be expanded to accommodate greater access, perhaps by redistribution of resources from services which are not experiencing overwhelming demand, or from services which can be deferred without significant harm to patients?</td>
<td>Yes – expand resource. No – for any remaining patients, go to question 5.</td>
</tr>
<tr>
<td>Mitigation question</td>
<td>After consideration of questions 1 to 5, there are still more patients needing to access the resource than the resource can accommodate. Are there any alternative options for any of the competing patients, which will mitigate the harms of missing out on the resource in question?</td>
<td>Yes – consider how effective the mitigating options will be, and go to question 7. No – go to question 7.</td>
</tr>
<tr>
<td>Ranking question</td>
<td>After consideration of questions 1 to 5, there are still more patients needing to access the resource than the resource can accommodate. Of those competing for the resource, (including those who are already using the resource) ‘rank’ them in order of perceived ‘net benefit’ of accessing the resource – that is, the sum of the estimated benefit of access to the resource and the harm of not accessing the resource. The ‘net benefit’ should also take into account any mitigation of harm arising from the options identified in question 6.</td>
<td>Can patients be ranked to clearly differentiate their net benefit of accessing the resource? Yes – those whose ‘net benefit’ ranks higher should access the resource before those whose ‘net benefit’ ranks lower. No – go to question 8.</td>
</tr>
<tr>
<td>Final question</td>
<td>If the competing patients cannot be differentiated in terms of ‘net benefit’ then fairness suggests they should access the resource according to who sought access first. If they cannot be differentiated on a ‘first come, first served’ basis then access can be determined by a process of equal and unbiased chance such as tossing a coin or use of a ballot.</td>
<td></td>
</tr>
</tbody>
</table>
Table 2. Notes to assist the application of the Priorisation Criteria

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a) <strong>Questions 4 and 5:</strong> If access to the resource in question is deferred or if other services are deferred provide whatever alternative care will mitigate symptoms, or manage the progression of the disease and arrange follow-up/review so that future access occurs or expedited access might occur if there are changes to circumstances.</td>
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<td>b) <strong>Question 7:</strong> Communication between clinicians, patients and other relevant parties will allow a patient centred perception of ‘net benefit’ to be brought to the deliberations.</td>
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<td>c) <strong>Question 7:</strong> Explicit, agreed criteria for measuring ‘net benefit’ are impossible to construct. Instead ‘ranking’ will be, most often, an uncertain estimate based on the application of the education and experience of clinicians, taking into account the many variables associated with the patients in question and the resource they are seeking to access. Ideally, senior clinicians should be involved in ‘ranking’ and what opportunities exist for discussion with the patients and with colleagues should be taken. An iterative interaction with the patients, to maintain a transparency of process and to allow emerging relevant factors to influence decisions, will contribute to trust in the process and fairness of the outcome. Collaboration with colleagues so that, if possible, agreement is reached, will minimise the risk of bias due to the personal perceptions of individual clinicians.</td>
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<td>d) <strong>Question 7:</strong> ‘Benefit’ may encompass contributions less directly related to the patient. For example, a contributor to the assessment of benefit might be that the patient has dependents who need this person restored to good health. The patient might be a health care worker and it could be a significant benefit to other patients to return this person to a depleted workforce as soon as possible. In addition, it may be considered beneficial to prioritise the care of health care workers who have caught the disease during their work to reinforce the principle of reciprocity – that we will look after those who put themselves at risk for us.</td>
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<td>e) <strong>All questions:</strong> Communication is a key contributor to a respectful, trusting relationship between carers, patients and their families. Application of the criteria will be made more palatable by transparency about the criteria and how they are being applied. The views of patients should be sought, particularly regarding net benefit of access, so that a patient centred perception of net benefit contributes to ranking, and to enhance the sense of trust and empowerment consequent to being heard. Knowledge of how a decision was made and an understanding that explicit and fair criteria were used will contribute to trust of the decision makers and acceptance of the decisions.</td>
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<td>f) <strong>All questions:</strong> An autonomous refusal of access when it is offered should be respected. However, a request for access when the patient does not warrant access according to the judicious application of these criteria, is insufficient to gain access.</td>
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**Question 1: Normal threshold question**—Does the patient meet the clinical criteria for access to the resource during normal times (that is, when there is not overwhelming demand for the resource)?

This question asks the clinician to consider if the resource in question would be accessed by the patient even if the resource was plentiful. The purpose of this question is to exclude those for whom the resource is not clinically indicated, prior to questions of prioritisation of patients (for whom the resource is clinically indicated) competing for the resource. For example, a patient with chronic airways disease, with poor exercise tolerance, may not be offered an Intensive Care Unit (ICU) bed for ventilation after an infective exacerbation of their disease, because an assessment by the clinicians, in consultation with the patient, is that ventilation would do the patient more harm than good.

This question addresses an important determinant of access to care and it is essential that it is not confused with the prioritisation decisions which follow. Patients, or other observers, should not perceive that access has been denied because of a rationing
decision when access was, in fact, denied on the basis of clinical determinants of what the resource had to offer the patient. Knowledge that access to the resource would not have been offered anyway, because it would not serve the best interests of the patient, should allow a refocusing of energies away from the denied resource to better alternatives.

If the patient does not meet the usual clinical criteria then, as will usually be the case, the patient will not access the resource. If the patient does meet the usual clinical criteria then the clinicians proceed to question 2.

**Question 2: Competition question**—Are there other patients who meet the normal clinical criteria (question 1), who are competing for the same resource (which is currently insufficient to accommodate all of the patients who are competing)?

This question confirms that there is insufficient resource for those who have the clinical indications to access it. If there is available resource, sufficient to accommodate the patient in question and without other patients competing, then the patient accesses the resource. If there are too many clinically appropriate patients for the resource to accommodate then the clinicians proceed to question 3.

**Question 3: Alternative options question**—Can any of the competing patients (including those who are already using the resource) have alternative care which, although perhaps not the first choice, will provide reasonably similar benefit to the patient and not cause significant harm due to accessing the alternative rather than the original choice?

This question asks the clinicians to consider other interventions, for which there may be less competition, which may serve the patient sufficiently well to be an alternative which will not disadvantage the patient significantly. For example, a patient with a bacterial pneumonia superinfection of influenza, who might normally be considered for intravenous antibiotics in a hospital bed (and hospital beds are the limited resource in question), may be able to be managed in a community setting with intravenous antibiotics. Or, a patient with hypoxia from a viral pneumonitis, who is competing for an ICU bed, may be able to be managed with non-invasive ventilation in a high dependency unit.

If there is a suitable alternative then the appropriate patient or patients should access the alternative care. For any remaining patients, the clinicians go to question 4.

**Question 4: Deferability question**—Can any of the competing patients have their access to the resource deferred to a future time when demand is likely to be less, without coming to significant harm?

Some patients may not need the resource immediately. For example, much ‘elective’ surgery could be deferred so that hospital and ICU beds are in less demand. Some ‘acute’ patients, for example some with fractures which need open surgical fixation, could be managed with more conservative means (a Plaster of Paris, for example) until competition for hospital resources is reduced.

If access to the resource in question is deferred or if other services are deferred (as might occur as a result of question 5, below), then alternative care should be provided to mitigate symptoms (for example, analgesia), or to manage the progression of the disease. Follow-up should be arranged so that future access occurs, or expedited access might occur, if there are changes to circumstances.
If access to the resource in question can be deferred, without significant harm to the patient, then it should be. For any remaining patients the clinicians should proceed to question 5.

**Question 5: Expansion question**—Can the resource be expanded to accommodate greater access, perhaps by redistribution of resources from services which are not experiencing overwhelming demand, or from services which can be deferred without significant harm to patients?

Resources from services which are less busy might be redistributed. For example, outpatient staff, and those mostly providing elective services, could be redeployed to augment ‘acute’ hospital services, fully staff intensive care beds and possibly change the function of some wards to provide a higher dependency (ICU step-down) function.

If this can be achieved then it should be. If there is no such redistribution, of relevance to the patients in question, then the clinicians should proceed to question 6.

**Question 6: Mitigation question**—After consideration of questions 1 to 5, there are still more patients needing to access the resource than the resource can accommodate. Are there any alternative options for any of the competing patients, which will mitigate the harms of missing out on the resource in question?

This question is not the same as question 3—the Alternative options question. That question was looking for alternatives which offer similar outcomes. Nor is it the same as question 4—the Deferability question. In that question the patient will come to no significant harm as a result of deferring access to the resource, although mitigation of symptoms and progression of the disease remain appropriate. This question comes at a time when it is conceded a patient (or patients) is going to be deprived of a resource they would normally access, and that this is likely to result in harm to them.

This question asks the clinician to explore options which will mitigate any harm. For example, a patient who would normally access in-hospital respiratory support cannot access it, but might be able to be given domiciliary oxygen. Or, a patient who is denied surgery due to (postoperative) ICU beds being used by victims of influenza, may have their pain managed with intravenous analgesics.

The clinicians now proceed to question 7. However, the relative benefits and harms of accessing or not accessing the resource will be considered taking into account the potential to mitigate the harms of missing out on the resource.

**Question 7: Ranking question**—After consideration of questions 1 to 5, there are still more patients needing to access the resource than the resource can accommodate. Of those competing for the resource, (including those who are already using the resource) ‘rank’ them in order of perceived ‘net benefit’ of accessing the resource—that is, the sum of the estimated benefit of access to the resource and the harm of not accessing the resource. The ‘net benefit’ should also take into account any mitigation of harm arising from the options identified in question 6.
Can patients be ranked to clearly differentiate their net benefit of accessing the resource?

Even in times with no extraordinary demands, those who seek health care need prioritisation. Those with acute healthcare demands, who present to hospitals in most countries of the Western world, will initially undergo some form of triage. This triage puts them in a queue for care mostly based on their need, or urgency, but also taking into account when they first tried to access care.

In general, those who are least urgent will not miss out, but they may have to wait. The philosophy of triage at this time is to allow access to everyone but to allow quickest access to those with the most urgent needs.

During a mass casualty incident (for example, an accident involving mass public transport, a natural disaster, or a terrorist attack), triage undergoes a change of emphasis. While urgency for care remains a key determinant of the make up of the ‘queue’, explicit attention is also paid to the patient’s ability to benefit.

The philosophy of triage takes on a utilitarian bent with a catch phrase of disaster triage being ‘the greatest good for the greatest number.’ A patient with severe head and other system injuries, who has some, but small, chance of acceptable survival may be triaged to the front of the queue under non-disaster conditions, but may be triaged to an alternative and less urgent type of care in a disaster.

It appears, therefore, that the richer the resource, relative to the demand, the more urgency, (or need), is a factor in prioritisation of acute care. The poorer the resource relative to the demand, the more ability to benefit influences prioritisation. However, discussions of the relative merits, and contributions, of need and benefit in prioritisation may seem academic to those who actually make triage decisions for acute care. For example, acute chest pain is one of the most common presentations to Emergency Departments (EDs).

Patients with whom the possibility of ischaemic cardiac chest pain is entertained by the triaging clinician will be triaged so that they are high in the queue. The reasons for a high triage ranking relate to the benefit of urgent defibrillation in case of life-threatening cardiac arrhythmias, and the benefit (decreased mortality and heart failure) associated with urgent reperfusion (thrombolysis or angioplasty) for those for whom it is indicated.

For the clinician, triaging patients presenting with acute chest pain, and in acute patients in general, the distinction between need and benefit is hard to make. Generally the more pressing the need (or urgency) for care the more benefit (or avoidance of harm) will be a consequence of timely care. Consequently, the term ‘benefit’ is used in these criteria to encompass any relevant contributors of need or urgency.

Furthermore, ‘benefit’ may encompass contributions less directly related to the patient. For example, a contributor to the assessment of benefit might be that the patient has dependents who will benefit from this person being restored to good health.

The patient might be a healthcare worker and it could be a significant benefit to other patients to return this person to a depleted workforce as soon as possible. In addition,
it may be considered beneficial to prioritise the care of healthcare workers who have caught the disease during their work to reinforce the concept of reciprocity—that we will look after those who put themselves at risk for us.\(^1\)

In addition to the apparent fairness of the concept of reciprocity, its explicit presence in planning and decision making will provide healthcare workers with a greater degree of comfort in continuing to provide care to infectious patients. Although they are taking on greater risk by doing so, they know they will be looked after should they, themselves, become unwell.

In the criteria, an assessment of ‘net benefit’ is made. This is the sum of the estimated benefit of access to the resource and the harm of not accessing the resource. The ‘net benefit’ should also take into account any mitigation of harm that can be achieved by alternative interventions should the patient not access the resource in question (question 6). Communication between clinicians, patients, and other relevant parties will allow a patient-centred perception of ‘net benefit’ to be brought to the deliberations.

Explicit, agreed criteria for measuring ‘net benefit’ are impossible to construct given the variables of context, the debated merits of different needs and benefits, and the difficulties of quantifying them. Instead ‘ranking’ will be, most often, an uncertain estimate based on the application of the education and experience of clinicians, taking into account the many variables associated with the patients in question and the resource they are seeking to access.

Ideally, senior clinicians should be involved in ‘ranking,’ and what opportunities exist for discussion with patients and colleagues should be taken. An iterative interaction with the patients, to maintain a transparency of process and to allow emerging relevant factors to influence decisions, will contribute to trust in the process and fairness of the outcome. Collaboration with colleagues so that agreement is reached, if possible, will minimise the risk of bias due to the personal perceptions of individual clinicians.

If patients can be ranked to clearly differentiate their net benefit of accessing the resource, then those whose ‘net benefit’ ranks higher should access the resource before those whose ‘net benefit’ ranks lower. If the patients cannot be ‘ranked’, then the clinician should proceed to question 8.

**Question 8: Final question**—If the competing patients cannot be differentiated in terms of ‘net benefit,’ then fairness suggests they should access the resource according to who sought access first. If they cannot be differentiated on a ‘first come, first served’ basis, then access can be determined by a process of equal and unbiased chance such as tossing a coin or use of a ballot.

If, after all of these considerations, there are competing patients, (none of whom has a good alternative care option, none of whom can be deferred, none of whom can have the harm of missing out mitigated any more, and all of whom seem to be equally worthy [in terms of net benefit]), then access to the limited resource should be on the basis of criteria which are fair, explicit, and predetermined.

It is generally accepted that, all else being equal, those who seek access first should get it. If there is still competition then access should be on the basis of ‘equal chance’ such as might occur with a toss of coin or a ballot.
Application of these criteria in a way which encourages respect for people

Communication is a key contributor to a respectful, trusting relationship between carers, patients, and their families. During a pandemic, ethical decision-making may be complicated by urgency to make decisions, (so that time for deliberation is short), and possibly, impaired patient decision-making capacity due to hypoxia or altered consciousness.

Prior public involvement in discussions about the criteria, and explicit communication of these criteria before they need to be applied, will ease their application during a pandemic. Similarly, real-time application of the criteria will be made more palatable by transparency about how access to a resource is achieved.

The views of individual patients seeking a resource should be sought, particularly regarding net benefit of access, so that a patient-centred perception of net benefit contributes to ranking, and to enhance the sense of trust and empowerment consequent to being heard.

Knowledge of how a decision was made, and an understanding that explicit and fair criteria were used, will contribute to trust of the decision-makers and acceptance of the decisions.

An autonomous refusal of access, when it is offered, should be respected. However, a request for access when the patient does not warrant access according to the judicious application of these criteria, is insufficient to gain access. Indeed, to allow access under these circumstances would be an injustice to any patients who meet the criteria but are denied access as a consequence of those who do not meet the criteria gaining access.

Summary

This paper presents criteria for the prioritisation of access to resources during overwhelming demand, such as during a pandemic. The criteria are in the form of eight questions related to the resource and the patients competing for it, and are intended to be sufficiently comprehensive and sufficiently succinct to be useful to clinicians who might be required to make such decisions.

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References:


Reversible posterior leukoencephalopathy associated with minimal change nephrotic syndrome

Jen Li Looi, Jonathan Christiansen

Case report

A 21-year-old Korean man was admitted with sudden onset of severe bitemporal headache and blurred vision followed by two generalised tonic-clonic seizures. Three weeks prior to admission, he had been investigated for peripheral oedema and a diagnosis of nephrotic syndrome made, with a 24-hour urine protein of 18 grams.

A renal biopsy confirmed minimal change disease, and prednisone 60 mg daily was commenced. On initial evaluation, he was alert (Glasgow Coma Score [GCS] 14/15), afebrile, and had a blood pressure of 124/58 mmHg.

The physical examination, including a detailed neurological assessment, was unremarkable apart from mild bilateral leg oedema. Investigations revealed leukocytosis (white blood cells [WBCs] 32.7×10^9/L) with neutrophilia (23.5×10^9/L).

Serum creatinine and electrolytes were normal, and serum albumin was 23 g/L. Blood cultures proved sterile. Chest X-ray and a non-contrast CT scan of the head were normal. An attempted lumbar puncture was non-diagnostic due to a traumatic tap. Intravenous ceftriaxone and acyclovir were commenced, but discontinued within 24 hours as he was afebrile. He remained normotensive, and his prednisone was withheld.

Magnetic resonance imaging (MRI) brain was performed and the patient had two further generalised tonic-clonic seizures immediately following the procedure. Intravenous phenytoin followed by maintenance oral phenytoin was administered. The MRI scan showed multiple areas of T2 and FLAIR hyperintensity involving the frontoparietal cortex, occipital cortices, and posterior temporal cortices bilaterally (Figure 1A). There was no evidence of venous sinus thrombosis or acute infarction. He had no further seizures and remained normotensive. His prednisone was restarted and he was discharged.

A follow-up MRI (Figure 1B) 2 months later showed complete resolution of the abnormalities. The clinical and radiological diagnosis was reversible posterior leukoencephalopathy.
Figure 1. MRI scan of the brain during the initial presentation (A), demonstrating focal areas of T2 and FLAIR hyperintensity involving the frontoparietal cortex, occipital cortices, and posterior temporal cortices bilaterally (arrows), which resolved entirely on convalescent imaging (B)

Discussion

Reversible posterior leukoencephalopathy (RPL) was first described by Hinchey et al in 1996, but has become increasingly recognised. RPL typically presents in the setting of accelerated hypertension or immunosuppressive therapy, although some patients are normotensive.

The pathophysiology of RPL is poorly understood, but two mechanistic theories have been proposed. The first postulates that a sudden increase in blood pressure
overcomes cerebral autoregulation leading to cerebral oedema. The failure of cerebral autoregulation at higher mean arterial pressures may lead to arteriolar vasodilation, capillary leakage, and ultimately extravasation of plasma in the extracellular space.

A second hypothesis suggests a sudden and severe increase in blood pressure produces vasoconstriction and cerebral ischaemia. This latter theory is supported by evidence from imaging studies, with vasoconstriction at cerebral angiography seen in conjunction with posterior white matter hypoperfusion. However the complete reversibility of the process seen in many patients is not consistent with oedema mediated by ischaemia and cell death.

Neither of these potential mechanisms for the development of RPL explain its occurrence in normotensive patients such as ours. Metabolic abnormalities including sepsis, electrolyte imbalance, fever, or renal failure may predispose to RPL in patients who are normotensive or with only mildly elevated blood pressure.

Our patient was afebrile with no evidence of sepsis, and his creatinine and electrolytes were normal. Immunosuppressive or cytotoxic drugs such as cyclosporine and tacrolimus are commonly associated with this syndrome, but less potent immunosuppressants such as steroids (e.g. prednisone) may also play a role.

The authors of a case report similar to ours implicate steroid use as the possible mechanism for RPL in a normotensive Korean woman with Down syndrome and nephrotic syndrome. Additionally, steroids have been linked with cortical blindness resulting from transient abnormalities of the occipital cortex, thus suggesting a common mechanism.

Several reported cases of RPL occurring in association with nephrotic syndrome have been linked to hypertension, high-dose intravenous methylprednisolone, or immunosuppressive drugs. Of note, however, are two patients with nephrotic syndrome, no steroid or immunosuppressant use, and mild hypertension, reported by Aksoy et al. The authors postulate that an increase in vascular permeability related to severe hypoalbuminaemia may be the pathogenesis of RPL in nephrotic syndrome. In our patient, the mechanism of development of RPL is unclear, but both steroid use and hypoalbuminaemia may be implicated.

This case highlights a potentially important association of RPL with either modest immunosuppressive doses of prednisone, or with nephrotic syndrome.

Although rare, the diagnosis of RPL should be considered in patients’ with intrinsic renal disease and a new onset of neurologic symptoms. Early MRI scanning should be undertaken in such cases.

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References:

Regulation of chicken contamination urgently needed to control New Zealand’s serious campylobacteriosis epidemic

Michael Baker, Nick Wilson, Rosemary Ikram, Steve Chambers, Phil Shoemack, Gregory Cook

Abstract

New Zealand’s campylobacteriosis epidemic reached a new peak in May 2006 with the annualised national notification rate exceeding 400 per 100,000 for the first time, the highest national rate reported in the literature. The epidemic is estimated to cause at least 1 fatality a year, >800 hospitalisations, and >100,000 cases in the community, and cost the New Zealand economy 75 million dollars per annum. There is overwhelming epidemiological and laboratory evidence that fresh chicken is the dominant source of human infection. The seriousness of this epidemic justifies rapid, decisive action to reduce human exposure to this pathogen.

There is good international evidence to support removal of fresh chicken from the food supply, with its reintroduction only when it can be shown to pose a very low risk to human health. Because freezing can substantially reduce Campylobacter levels, frozen chicken could be substituted to allow continued consumption of this popular food. Efforts to reduce Campylobacter colonisation of poultry flocks and contamination during chicken processing and distribution, along with continued consumer education, are important, but do not appear sufficient to control this epidemic in the short to medium term.

Imagine you were asked to investigate an epidemic of disease that was causing at least one death a year in New Zealand, putting over 800 people in hospital, causing in excess of 15,000 notified cases, and a further 100,000 non-notified cases. Also imagine that your epidemiological and laboratory investigation implicated a single food as the source for a large proportion of these cases. Imagine that systematic testing of this widely consumed food showed that it was routinely contaminated with the pathogen causing this illness, often with thousands of organisms on each piece in its pre-cooked state. Imagine you also found that cross-contamination in kitchens was very hard to prevent and likely to occur in thousands of instances each year. At this stage you would recommend the immediate withdrawal of this food from the market and recall of contaminated batches to prevent as many cases of disease as possible.

We do not need to imagine this scenario, as this is reality for New Zealand in 2006. This paper outlines the scale of New Zealand’s campylobacteriosis epidemic, the importance of fresh chicken as a major source of this infection, and why we consider that this food should be withdrawn from sale in New Zealand until it can be shown to pose a low risk to human health.
New Zealand’s campylobacteriosis epidemic

Our epidemic of campylobacteriosis reached a new high point in May 2006 when the annualised national notification rate exceeded 400 per 100,000 for the first time, based on 15,553 cases for the preceding 12 months. (see Figure 1)

Rates in New Zealand were already the highest reported by any country, being more than 3 times higher than in Australia and 30 times higher than the United States. Many other developed countries use surveillance methods that are as rigorous as those used in New Zealand, so there is no basis for concluding that we are just better at diagnosing and reporting this disease.

The health impact of campylobacteriosis now places it among New Zealand’s most important infectious disease problems. Campylobacteriosis caused more hospitalisations in 2005 than meningococcal disease, acute rheumatic fever, AIDS, and tuberculosis combined (based on principal diagnosis). The total number of campylobacteriosis cases in the community is conservatively estimated at 115,000 per annum (based on a widely used multiplier of 7.6 times the number of notified cases, although some countries have used multipliers as high as a 100 times).

Campylobacteriosis also kills. There is about one recorded fatality a year from the acute effects of this disease. Chronic effects include Guillain-Barré syndrome (GBS), the risk of which is greatly increased (by 77 to 100 times) in the 2 months following Campylobacter infection. GBS is fatal for 4–15% of patients and leaves around 20% disabled.

We also estimate that this epidemic is costing New Zealand about 75 million dollars a year, based on an update of a previously published costing estimate ($533 per case in 1999, updated using consumer price index movements to $632 in 2006 and applied to the increase in estimated cases).

Figure 1. Annual number of notifications (1980–2005) and hospitalisations (1995–2005) for campylobacteriosis in New Zealand

Source: Institute of Environmental Science and Research Limited (notifications) and New Zealand Health Information Service (hospitalisations, based on principal diagnosis).
What can be done to control this epidemic

The obvious control measure is to swiftly remove fresh chicken from the food supply and only reintroduce it when it can be shown to pose a very low risk to human health. Frozen chicken (or other processed forms with reduced contamination levels) could be substituted, in the interim, to allow continued consumption of this popular food. While this might appear a bold public health step, we think it is fully justified for the reasons outlined below.

Contaminated chicken meat has a central role as the source of the *Campylobacter* epidemic. Two separate New Zealand case-control studies of sporadic disease have specifically implicated this source. One of these was a large multi-centre study that found that chicken-related exposures could explain over half of the population attributable risk, more than all of the other risk factors combined. Other New Zealand epidemiological studies of sporadic campylobacteriosis and of outbreaks, often with supportive laboratory evidence, are also consistent with an important role for chicken as a risk factor.

The rise in campylobacteriosis over the last 25 years has coincided with a substantial increase in poultry consumption (Figure 2). Most of this increase has been in fresh chicken meat, which rose from 4 kg/person in 1981 to 30 kg/person in 2005, and now accounts for 80% of total production. Campylobacteriosis incidence is highly correlated with this increase in fresh chicken meat production (Spearman’s rank correlation coefficient $r_s = 0.98$, $p < 0.0001$).

**Figure 2. Annual production (tonnes) of fresh and frozen chicken meat in New Zealand (1981–2005)**

![Figure 2. Annual production (tonnes) of fresh and frozen chicken meat in New Zealand (1981–2005)](image)

**Source:** Statistics New Zealand quarterly survey of poultry producers. Note that because almost all NZ chicken production is destined for human consumption within NZ, and virtually no chicken is imported, these data correspond well to human consumption patterns.
Although human campylobacteriosis can come from a multitude of sources (contaminated food, water, or environments—or contact with infected humans or animals), no source except for chicken has been implicated in more than a few percent of cases in New Zealand.\textsuperscript{11,12,18} Overall notification and hospitalisation rates are higher in cities than in rural areas, which is also consistent with a largely food-borne illness rather than one from contact with contaminated environments, animals, or water.\textsuperscript{2}

For those living in rural areas, non-poultry sources of infection (such as direct zoonotic infection from farm animals) are likely to be relatively more important than for those living in cities.\textsuperscript{19} However, the rural population accounts for only 14\% of the total New Zealand population,\textsuperscript{2} and many of those will still consume and be infected from commercially produced chicken in the same way as those living in urban areas.

There is good evidence that withdrawing fresh poultry from the market would greatly attenuate this epidemic. There is a famous ‘natural experiment’ from Belgium where poultry was removed from the market for 4 weeks in 1999 because of a scare over dioxin contamination. The incidence of \textit{Campylobacter} infection in that population dropped by 40\% from the expected rate, and then returned to ‘normal’ when poultry was reintroduced.\textsuperscript{20}

In 2000, Iceland introduced an intervention similar to the one we are advocating. This intervention included testing chicken flocks and only allowing those that were ‘\textit{Campylobacter}-free’ to be sold as fresh chicken. The remaining contaminated chicken could only be sold frozen. This intervention was followed by a substantial decline in reported campylobacteriosis.\textsuperscript{21,22} This approach is also being used in Denmark and Norway.\textsuperscript{23} Evaluating the net effectiveness of this intervention has been complicated by the fact that it has been introduced with varying degrees of rigor, often along with other control measures.\textsuperscript{21}

Freezing chicken is not a perfect solution. While it does not eliminate \textit{Campylobacter} contamination, it has been found to markedly reduce levels of the organism in chicken meat (by 0.5 to >2.5 logs, or approximately a 3 to >300 fold reduction in contamination levels depending on the methods used).\textsuperscript{5,22–26} If introduced it would work in combination with current control measures, including cooking and careful food handling.

Quantitative risk assessment in Denmark suggests that a drop of about 100-fold in \textit{Campylobacter} contamination levels of fresh chicken is enough to reduce the risk to consumers by about 30-fold.\textsuperscript{2} Preliminary results from similar modelling carried out in New Zealand also shows that freezing poultry would considerably reduce the predicted number of human infections.\textsuperscript{27}

In addition, previous research in New Zealand has shown that using frozen chicken protects people from illness, presumably because people doing this are not handling and consuming fresh chicken.\textsuperscript{12} Applying this risk-reduction strategy in New Zealand could mean that instead of fresh chicken potentially causing 59,000 cases of illness each year, it might only cause 2000 cases (based on current incidence, using a conservative estimate of 50\% of cases attributable to chicken meat exposure,\textsuperscript{12} and a 2 log reduction in contamination through use of effective freezing\textsuperscript{5}).
Freezing, or using some other proven method to decrease contamination levels in fresh poultry, appears to be the only plausible way of reducing illness from this source in the short-term. The current approach of waiting for the poultry industry to lower contamination levels doesn’t appear to be working. Surveys of fresh chicken in New Zealand show that contamination is routine and appears to be at high levels (89% of 250 chicken meat samples collected in a national retail survey in 2003–4 were contaminated\(^{28}\)). Programmes to prevent poultry colonisation on the farm offer potential to reduce contamination levels, but may take years to implement.\(^{29}\) Reducing contamination in poultry slaughter houses has also proven difficult.\(^{30}\)

Educating those who prepare chicken at home and in restaurants to take precautions with handling chicken is important, but it is unlikely to significantly reduce infection rates because of the acknowledged difficulty of changing consumer behaviour,\(^{31}\) and because of the technical difficulties of preventing cross-contamination in the kitchen setting. Kitchen contamination studies show that preparation of meals with raw chicken results in cross contamination to hands, plates, chopping boards, utensils, and ready to eat food.\(^{32}\) Preparing fresh chicken also results in very widespread distribution of *Campylobacter* in the kitchen environment and this organism can be recovered from kitchen surfaces 24 hours later. Simple cleaning with hot water and detergent does not appear to be sufficient to clean these surfaces, and the use of a chlorine-containing disinfectant appears necessary.\(^{33}\)

Switching to frozen chicken will pose challenges for chicken producers and may be unpopular with some consumers, but is certainly not technically difficult to implement. Frozen chicken was the main form of retail chicken product in New Zealand up until the late 1980s and still accounts for about 20% of chicken sold in this country (Figure 2). The poultry industry could certainly be permitted to find alternative processing methods that achieved the same levels of microbial reduction as freezing. It will also be necessary to review and monitor the methods used to freeze poultry as some methods achieve much higher levels of organism reduction than others.\(^{22,25}\)

The requirement to achieve low *Campylobacter* contamination levels in chicken meat, through freezing or other methods, offers the huge advantage of rewarding producers who deliver safer food. Currently there is little or no financial incentive for industry to invest in methods to lower such contamination. These bacteria are invisible to consumers and restaurant staff, who cannot therefore assess (and potentially pay a premium for) ‘*Campylobacter*-free’ product to lower the risk to them and their customers.

Allowing producers who achieved low contamination levels to continue to distribute fresh chicken would give them a strong economic incentive to develop and implement methods to reach this goal. Given the very large cost of this epidemic, there would be a net economic benefit to New Zealand society from spending tens of millions of dollars on implementing such control measures.

As a major food producing and exporting country, we should be a world leader in controlling food-borne diseases like campylobacteriosis. Our current status at the top of the campylobacteriosis league table must be highly embarrassing for our critical food production sector.
Conclusions

Many of New Zealand’s important epidemics are difficult to control. The epidemics of diabetes, road traffic injuries, and some forms of cancer have complex origins and preventing them will take all of our collective ingenuity, not least because they involve sustained changes in human behaviour and extensive modifications to our environment. By contrast, the New Zealand campylobacteriosis epidemic is relatively easy to control because it can be greatly reduced by managing a single dominant source. There are parallels with the famous public health intervention of taking the handle off the public water pump to control the London cholera outbreak of 1854 (this is a slight simplification of historic events, but it illustrates the principle of controlling epidemics by identifying and managing their source).

Given this evidence, we call on the New Zealand Food Safety Authority to remove fresh chicken from sale until it shows Campylobacter contamination levels that are consistently below a specified maximum regulatory level. This change in policy could permit the continuing distribution of frozen chicken, which we suggest should be accompanied with large pictorial health warnings and instructions on each package about correct thawing and cooking.

We also recommend that these changes be accompanied by a rigorous evaluation programme to measure their impact on reducing the high health burden of campylobacteriosis in the New Zealand population. The poultry industry could assist by making the results of their extensive microbiological testing programme available to researchers. Evaluating interventions to reduce disease risk should take precedence over further research on Campylobacter transmission pathways and microbiology.

New Zealand has the opportunity to act decisively to control a serious public health problem. To not act would mean accepting the serious ethical, health, and economic consequences. Such inaction could only be justified if the arguments presented here can be convincingly refuted by sound evidence for alternative courses of action.

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Quality improvement in New Zealand healthcare. Part 7: clinical governance—an attempt to bring quality into reality

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Abstract

In this seventh and final article in the Series on quality improvement, we discuss clinical governance and its place in the New Zealand health sector. We describe it as requiring clinicians to accept transparent accountability, teamwork rather than individualism, a systems view and the need to share power with others in the clinical domain. In return, they must be given the autonomy to do the job they are trained for and the resources necessary for that job. Without this quid pro quo, clinical governance will not become a framework for clinicians to work effectively in healthcare organisations. However, with this recognition, it provides a sound basis for clinicians and managers to work together in contemporary healthcare organisations.

In the previous six articles in this Series, we have identified the need to improve safety and quality of care. We have examined how quality can be measured, monitored, and improved. We have also looked at the monitoring interests of the State and District Health Boards (DHBs) and the notion of patient-centred care.

This final article in the Series looks at how all this could come together in a system that places joint responsibility on clinicians and healthcare managers in a system frequently referred to as clinical governance.

The meaning of clinical governance

Clinical governance attempts to link clinical and managerial paradigms, and has been central to the health reform efforts in the United Kingdom since the Blair Government took office in the late 1990s. Leaders in medicine defined it at the time as:

“A framework through which NHS organisations are accountable for continually improving the quality of their services and safeguarding high standards of care by creating an environment in which excellence in clinical care will flourish.”

Clinical governance is about the clinical process and its governance. It is not a method for managers to govern or control doctors, nor is it a method for doctors to govern or control managers or other clinicians. To many of us, the word governance in this context has been misleading. It is quite different from governance in other contexts, such as corporate governance or the sort of governance that goes on in a DHB board meeting. It is not a top-down process, nor is it dominated by the financial imperatives that drive many other business management models.

Clinical governance depends on doctors and managers working together, each realising that they have the potential to gain from such collaboration.
The elements of clinical governance

The reason for investing in clinical governance is to improve quality of care. All else is subservient to this. However, this principle in itself causes a clash of ideologies between those who believe that each patient deserves the best care (typically doctors with a sense of “duty” towards their patients) and those who have to distribute funding and relate to a population (characteristically board directors and managers within an ethic of “utilitarianism”—the greatest good for the greatest number).

When clinical governance began within the NHS, its introduction was politically and financially supported by the Blair Government. At the same time, a refereed publication *The Journal of Clinical Governance* was launched and an agency was created for its promotion and development—The NHS Clinical Governance Support Team.

In New Zealand and Australia, clinical governance received less attention from the Government and DHBs, but nevertheless appeared on the agenda because of its promotion by interested doctors and academics. One of the latter, Pieter Degeling, has consistently identified four elements of the process as central to its implementation. These are set out in Table 1.

Essentially, this requires doctors to think differently about accountability and autonomy; to accept that their clinical work should be subject to scrutiny by their peers; that teams rather than individuals should be dominant in the delivery of healthcare; and further to accept that decision-making about clinical matters has resource implications that doctors need to manage.

Table 1. “Four elements”—their role in clinical governance

<table>
<thead>
<tr>
<th>Element</th>
<th>Doctors are called on to:</th>
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</thead>
<tbody>
<tr>
<td>Money</td>
<td>Accept the proposition that all clinical decisions have resource implications</td>
</tr>
<tr>
<td>Autonomy/accountability</td>
<td>Recognise the need to balance clinical autonomy with transparent accountability</td>
</tr>
<tr>
<td>The systems view</td>
<td>Support the systematisation of clinical work</td>
</tr>
<tr>
<td>Power sharing</td>
<td>Subscribe to the power sharing implications of team based approaches to clinical work</td>
</tr>
</tbody>
</table>

There will be elements in the above that doctors will be able to identify with. The four propositions may challenge traditional attitudes, beliefs, and behaviours of doctors who in response could ask, what are the managers and funders doing as their contribution to clinical governance? The four elements focus on doctors’ responsibilities primarily because clinicians take a central role in clinical governance by virtue of the fact that they do the clinical work. The attitude required of managers is their acknowledgment that the doctor is in charge of the decisions that result in cost and income for the organisation. Managers also need to facilitate good quality systems.

The question arises as to how clinical governance based on the propositions identified in Table 1 could be operationalised. An answer to this question can found in Degeling’s work reported in 2004. Here, it is proposed that hospitals and other
providers move away from the traditional quality silos (risk management, quality assurance, accreditation, adverse event reporting, and so on), which characterise existing healthcare organisations to a new structure which would be built around the work of groups of clinical staff who work within a particular service. In the management of heart failure, for example, this might include cardiologists, gerontologists, general physicians, general practitioners, clinical nurse specialists, nurse practitioners, practice nurses, dieticians, community nurses, smoking cessation educators, occupational therapists, psychologists, and possibly others.

Clinical governance accepts the multidisciplinary nature of much of modern medical endeavour. Supporters of clinical governance also advocate the use of clinical pathways, evidence-based clinical practice, and clinical audit backed up by appropriate resources to drive quality improvement.

We believe that there are several reasons why clinical governance has struggled to gain standing in New Zealand. We use the elements identified in the first column in Table 1 to elaborate these reasons, and then offer our approach to adopting a clinical governance agenda.

Money—While it is axiomatic that clinical decisions have resource implications, accepting this first proposition infers some responsibility on clinical staff and doctors, in particular to take the leadership role in the rationing process. This will be seen by doctors as acceptable only when the legitimate concerns of doctors on behalf of their patients are given standing. If managers do not support clinical staff when advocating reasonable positions, then doctors will not take difficult rationing decisions.

The recent decision of the Canterbury DHB (CDHB) to remove 5000 patients who had exceeded the 6-month waiting list limit (or were likely to) to satisfy Ministry of Health requirements is a case in point. In a letter from 74 of the Board’s 80 surgeons to the CDHB, it was accepted that healthcare funding was limited but they criticised the Ministry of Health’s waiting list policy that ranked certainty of operation as the prime driver in waiting list management. The letter went on to say that “the relative level of funding for the provision of healthcare for patients in our community and who require surgical care in particular is not adequate.”

CDHB have accepted that they could have handled the process better, especially ‘that it was a mistake not to involve the surgeons in the decision about who should be cut from the waiting list.’

Here we have a case where it appears that the Government wishes to give patients certainty (about having or not having an operation within 6 months) ahead of prioritising cases according to need. Furthermore, there is a financial penalty for those DHBs that do not comply with this MOH directive. Doctors have come to accept rationing, but not arbitrary rationing which fails to give priority to the most urgent cases. The case illustrates a lack of clinical governance and the resulting disaffection of doctors and patients.

Autonomy/accountability—The way in which the profession is seen to be adopting high clinical standards is through the use of accountability systems that stand up to scrutiny. Doctors practice in an open system that demands public levels of accountability against the backdrop of some members of the public favouring a culture of blaming and shaming. This means that managers, the Ministry of Health,
and others including the Health and Disability Commissioner must continue to support a no-blame culture and must support and fund strategies such as clinical audit that enable clinical professionals to be accountable. Doctors for their part need to participate in these programmes and take leadership roles in their management. Sound clinical governance should promote autonomy within such an accountability framework.

**The systems view**—Taking a systems view entails examining how care is delivered, how it can be redesigned to improve patient experience and outcomes, understanding the cause of errors and latent system weaknesses. It recognises that healthcare is a complex adaptive system.

James Reason has identified three key ‘symptoms’ of what he calls the ‘vulnerable system syndrome’ that predicts a high risk of error and failure. These are:

- Blaming front-line individuals.
- Denying the existence of systematic error, provoking weakness.
- Blinkered pursuit of productive and financial indicators.

By this definition, most of the large healthcare systems in New Zealand are vulnerable to systems failures, trapped in a never-ending cycle of reacting to problems (not training staff to prospectively identify and remedy deficiencies) and ignoring quality problems to focus only on financial bottom-lines.

A ‘systems’ view also focuses attention on the movement of patients across care settings—both horizontally (from one specialist or general practitioner to another) and vertically (from secondary care to primary care and vice versa). Doctors are central to this sort of systematisation and when given the opportunity and training, will take the lead in other types for example with root cause analysis. However, there are examples of doctors standing aside from efforts to systematise. For example, they often leave the development of clinical pathways to nurses and allied health professionals.

While managers are attracted to systems, they are in no position to lead their development in clinical settings because they lack the necessary knowledge. This is an area where doctors must increasingly take the lead and where doctors can learn from nurses.

**Power sharing**—The training, clinical knowledge, and experience of doctors makes them natural candidates to lead clinical teams. Indeed, historically, they have dominated other members of the healthcare workforce. Clinical governance requires all members of the clinical team to be able to participate as equals. If it is to gain traction in healthcare organisations, doctors will be required to share some of their power. They can do this by encouraging nursing and other colleagues to join key committees and other influential groups.
An approach to adopting a clinical governance agenda

We suggest that adopting clinical governance in New Zealand requires attention and energy on four fronts:

- Managers and doctors knowing what one another are talking about.
- Recognition on the part of funders and managers of the need for responsible funding of the service where clinical governance is sought.
- Recognition on the part of clinicians that the four items identified in Table 1 are legitimate.
- Provision of suitable structures, such as a clinical board and supporting infrastructure.

Communication—Doctors and managers need to work collaboratively in organisational settings. In dealing with one another, doctors and managers have to recognise that they use the same words but often mean different things when they use them. For example, doctors and managers frequently use the word “accountability.”

Accountability to the doctor means personal responsibility for their actions (in connection with a patient) and realising that any shortfall may lead to censure, legal action, and (at worst) patient harm. To a manager, however, the word is used in connection with resources. Managers view accountability in terms of living within budget, achieving output targets, and so on. Clinical governance requires these groups and those they represent to understand one another when they engage in conversation and to be aware of their different “languages”.

Funding—The key here is balance. If Governments are to adopt the position of the Secretary of State in the UK (which required managers to put financial management ahead of clinical objectives), then doctors will fail to engage with the four elements identified in Table 1. In contrast, if they adopt agendas that recognise responsible funding, they will be able to expect doctors to engage in responsible resource use.

The “four elements”—We consider that where doctors are employees in healthcare organisations they will recognise as legitimate the four elements identified in Table 1. Each is consistent with sound clinical practice and sound management practice.

Structural support—Clinical governance needs infrastructure. In some places it is given direction and standing by having a clinical board. Such a board can develop and support appropriate clinical policy.

Does clinical governance work?

If clinical governance is defined as we have described above, then the obvious question is whether it has been implemented anywhere and whether there is evidence that it improves patient care and outcomes.

Our response to this is that while it makes intuitive sense, it has not been subject to the sort of critical evaluation that doctors would find convincing. A review of the relevant literature suggests that most articles on this topic appeared before 2002.

Clinical governance has been described as building on existing processes for improvement, requiring leadership to integrate processes and drive change at the
highest level. However, investigation of the types of improvement methodologies used indicate limited application because of ambivalence among doctors about the impact of clinical governance. While there was often a lot of activity around setting up the structures for clinical governance, little has been reported on improved outcomes. In fact, clinical governance has struggled in most settings to gain the approval and commitment of doctors generally, and has been described as a yet another management tool to control clinicians and a further rehash of previous failed attempts.

Clinical governance might be better thought of as a system of management peculiar to health systems where those with the funds and those with clinical skills can work together. Perhaps the answer lies in improving relationships so that each views the other with trust and accepts that each has a valuable role in improving patient care with the judicious use of available funding.

**Conclusion**

For clinical governance to work, both doctors and managers have no alternative but to recognise that the values and objectives of each have standing. Doctors need to accept that every clinical decision they make carries an economic consequence that they are using up money for one person that will then not be available for others.

Managers need to value quality as well as financial risk management. They have to accept that doctors know more about what works for patients than they do. And they have to accept that the doctors make the decisions that cost money.

The way clinical governance operates in one locale may be different from the way it functions in another. What is clear is that irrespective of whether some activity is called clinical governance, the items identified above require appreciation and consideration. Even then, without adequate resources, clinical governance will founder.

The articles we have presented in this Series have touched on the separate but linked dimensions of healthcare quality—safe, timely, effective, efficient, equitable, and patient-centred care. They have identified how we can develop indicators or benchmarks—and properly measure, monitor, and improve the quality of care that we deliver. For quality improvement to be sustainable, it requires leadership from doctors with a continued commitment to work in multidisciplinary teams, and an interest in acquiring the skills of quality improvement.

It is also clear that patients want doctors to be more open about their decision-making, and they want doctors to include them in that process. Recognising that the patient view may be different (it may be less- or more-informed), is no less important.

**Conflict of interest:** No Conflict

**Author information:** Rod Perkins, Allan Pelkowitz, and Mary Seddon on behalf of EPIQ.

EPIQ is a School of Population Health Group (at the University of Auckland) with an interest in improving quality in healthcare in New Zealand.
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Golf as a preventive treatment for biliary colic

This was taken from a series of case reports written by Dr Martin and published in the New Zealand Medical Journal 1907, Volume 5 (21), p9.

Treatment.—During an attack of Biliary Colic, one must ease the pains. Morphia hypodermically is the speediest and most reliable. If Morphia fails, inhalation of Chloroform will succeed.

For the vomiting, Bi-carbonate of Soda one drachm to the pint of warm water, which gives the stomach something to act upon, and although it may be speedily expelled, it produces relief. Hot fomentations, leeching, draughts of warm water, antypyrin, all help to ease the condition.

The preventive treatment is important. One should endeavour to prevent stagnation of bile. Exercise is here commended. Best of all is horse exercise. Tennis, climbing hills, swimming, and rowing are good.

Golf is an excellent exercise and should be warmly advocated. The golfer swings and twists his body, and alternately contracts and releases his abdominal muscles. He one moment stoops to place his ball, and the next, with a violent rotary. abdominal and shoulder swing, sends it into the air. He crosses ditches and creeks, climbs up and down bunkers, and arrives at the last putting green hot and tired.

Could any bile stagnate during such a gymnastic cycle. One should always advise the liver patient to procure a quiet horse and purchase a pair of golf clubs.
Bilateral loin pain

Sujit Nair, Cherian George, Oliver Byass

A 36-year-old female presented with bilateral loin pain to accident and emergency. Her past medical history included depression and analgesic overdose.

**Figure 1. Plain radiograph**

**Figure 2. Excretory urogram:**
close-up AP view of both kidneys

**Figure 3. Excretory urogram:**
both kidneys and ureters
(AP view)

**Questions**—What does the excretory urogram show and what is the diagnosis?
**Answers**

The excretory urogram demonstrates bilateral ring-shaped filling defects, egg in cup appearance (small arrow), and hooks (dark arrow) which are characteristic of renal papillary necrosis.

Diagnosis—Bilateral chronic papillary necrosis.

Causes of bilateral chronic papillary necrosis include:

- Analgesic abuse.
- Diabetes mellitus.
- Recurrent urinary tract infections.
- Sickle cell anaemia.

Interstitial nephritis and papillary necrosis are characteristically seen in analgesic abuse. In early stages, the papillae swell and this is not radiologically demonstrable. In the later stages, the papillae shrink and slough off resulting hooks, spurs, egg in shell appearance, ring shadows, and ring-shaped filling defects (sloughed papilla) on the excretory urogram.

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Myocardial infarction—do Q waves matter?

A large (NZ) study involving over 15,000 patients with ST-segment-elevation myocardial infarction (STEMI) focussed on whether or not the presence of pathological Q waves in the cardiogram was important. Well it was—patients with initial Q waves had an absolute 30-day mortality just over 3% higher than patients without initial Q waves (10% vs 7%).

Patients in this study were treated with fibrinolytic therapy (aspirin and streptokinase) and randomised to either the direct-thrombin inhibitor bivalirudin or unfractionated heparin. As only the presence of Q waves affected outcome unfavourably, the authors recommend a more aggressive approach in these patients. An accompanying editorial agrees that primary percutaneous coronary intervention (PCI) would be the treatment of choice.


New first line treatment for hypertension

The British Hypertension Society and the National Institute for Health and Clinical Excellence (NICE)—the body that advises on use of treatments by the NHS in England and Wales—have recently reviewed their management guidelines for hypertension. In essence, they recommend dropping β blockers from the front line as head to head trials showed that β blockers were usually less effective than the other drugs in reducing major cardiovascular events, particularly stroke.

They now recommend the first choice for initial treatment should be either a calcium channel blocker or a thiazide-type diuretic in those aged 55 years or over—for patients under the age of 55, an angiotensin converting enzyme (ACE) inhibitor.

In both age groups, patients may end up taking all three classes of drug if monotherapy fails.

*BMJ* 2006;333:8

Paracetamol and liver enzymes

We know that paracetamol in overdose can cause lethal liver damage. But what about habitual use of therapeutic doses? An American group conducted a randomised study in which 145 healthy volunteers were randomised to three arms—4 grams of paracetamol daily (either alone or in combination with selected opioids) or a placebo.

More than a third of the volunteers taking paracetamol had serum concentrations of alanine aminotransferase over three times the upper limit of normal at some point during the 14 day experiment.

Well we have suspected that this would be the case—and now we know.

*JAMA* 2006;296:87–93
Acupuncture and knee osteoarthritis

Despite the popularity of acupuncture, evidence of its efficacy for reducing pain remains equivocal. A recently published study from Heidelberg studied over 1000 patients who had had chronic pain for at least 6 months due to osteoarthritis of the knee.

All were treated with physiotherapy and as-needed anti-inflammatory drugs. And they were randomised to traditional Chinese acupuncture, sham acupuncture (needling at defined nonacupuncture points), or conservative therapy. Both acupuncture and sham acupuncture were better than conservative measures—both over 50% success rate at 26 weeks compared with 29% for the control group.

Makes you think that sticking needles everywhere has a strong placebo effect?

Ann Intern Med 2006;145:12–20

Prescription drug abuse in the gym

Drug use for performance improvement by professional athletes is constantly in the news. But what about the amateurs in the gym or health club?

The authors of this paper did a survey of male and female health club attendees in Britain and observed significant increases in the use of the following drugs: diuretics (10%), thyroxine (10%), insulin (14%), clenbuterol (21%), tamoxifén (22%), human chorionic gonadotrophin (11%), growth hormone (24%), and ephedrine (44%).

And, they report that steroids were still the most abused drug. Even more astounding they quote an American study which reveals several trends in the non-medical use of steroids. Nearly four out of five users are non-athletes who take these drugs with the sole intention of improving physical appearance!

JRSM 2006;99:331–2
Alcohol excess may be overemphasised in gout treated in secondary care

Traditional images have depicted patients with gout as decadent, middle-aged men over-indulging in alcohol. Choi and colleagues have recently confirmed that alcohol intake is strongly associated with the development of gout in the general community, and that while beer confers greater risk than spirits, moderate wine intake does not increase the risk. Alcohol consumption has also been associated with flares of disease in patients with gout. It is likely that alcohol-related hyperuricaemia occurs due to reduced renal tubular urate excretion, and that beer confers additional risk due to the high purine content of this beverage.

We wished to determine whether alcohol intake is a significant contributor to gout in patients referred to secondary care. As part of an analysis of cardiovascular risk management in gout, a careful history regarding risk factors for gout was recorded. This history included a record of the number of standard drinks and type of alcohol consumed per week. Data were available from 100 consecutive patients with gout from outpatient clinics and inpatient assessments at Auckland and Counties Manukau District Health Boards, Auckland, New Zealand. All patients with gout met the Wallace criteria for diagnosis of gout.

Most of patients were men (78%). There were 49% patients of Pacific origin, 24% New Zealand Māori, 18% European New Zealanders, and 10% of other ethnicities. Median (range) disease duration was 10 (0.3–35) years, and 63% had tophaceous disease.

The majority of patients (75%) did not consume any alcohol. Of the 25 patients who did drink alcohol, 18 primarily consumed beer. There were 11 (11%) patients with weekly alcohol intake in excess of that recommended by the Alcohol Advisory Council of New Zealand (>14 standard drinks for women and >21 standard drinks for men). One patient (1%) had hepatic cirrhosis related to alcohol abuse.

These data indicate that within the secondary care gout population, alcohol intake is infrequent and that the vast majority of patients with severe gout do not consume excessive alcohol. We recognise that such cross-sectional analysis does not take into account the effect of previous alcohol intake, and it is conceivable that some of our patients may have reduced their alcohol intake following advice from health professionals. Furthermore, self-reporting of alcohol intake may under-represent true consumption.

We continue to advocate intensive assessment of lifestyle factors including alcohol intake in all patients with gout, and suitable management for those with excessive alcohol intake. However, we wish to emphasise that the perception of gout as a self-inflicted “shameful” disease that occurs principally due to alcohol excess is not warranted. Such perceptions may prevent presentation at an early stage of disease, when irreversible articular, renal, and cardiovascular damage can be avoided. It is likely that other factors, such as genetic risk, insulin resistance, and concomitant
medications, are far greater contributors to severe gout within our multicultural community.

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Regulations should ban the sale of cigarette pack covers of health warnings

For New Zealand to meet its obligations under the Framework Convention for Tobacco Control, further modifications of health warnings on cigarette packets are required. The Ministry of Health has already begun this process and a likely outcome is the adoption of graphic health warnings. This outcome is logical, given the strong scientific evidence base for such graphic health warnings.

In a 2003 article, two Australian authors described plans from the tobacco industry’s own internal documents suggesting that the provision of covers for packs bearing warnings may be used in the future to counter the impact of warnings on smokers. This “future” has arrived overseas with one tobacco company even having marketing on these pack sleeves (i.e. the Marlboro man on sleeves in Hong Kong). Closer to home, we recently purchased two cigarette-pack sized cardboard sleeves in Australia (Figures 1 and 2). These can be used by smokers to cover the graphic health warnings that are now legally required on Australian cigarette packs.

Figure 1. A cigarette packet “sleeve” marketed in Australia that can be used to cover the graphic warnings on a cigarette pack (version apparently more popular with females)

Figure 2. Another cigarette packet “sleeve” marketed in Australia that can be used to cover the graphic warnings on a cigarette pack (version apparently more popular with males)
There now appear to be moves to stop the use of these sleeves by the Federal Government legislation in Australia. However, to avoid this additional step in the future, New Zealand should ban all sleeves and other similar measures (such as stickers or other containers sold for this purpose) when the new graphic warnings are introduced. Ultimately, the best cure for chronically irresponsible tobacco industry behaviour in New Zealand is to adopt a new regulatory framework that removes the tobacco industry out of the driving seat.  

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Policymakers’ ignorance of New Zealand Government investment in tobacco companies

In a recent study, we explored the knowledge and attitudes amongst a small sample of policymakers about the tobacco-related policies. The aim of the study was to pilot the use of in-depth interviews of key informants for tobacco policy in the New Zealand setting.

Methods—10 interviewees were selected from:

- Politicians who currently had, or had previously had some role in tobacco policy in the past 10 years, or had commented publicly on tobacco policy in that period; and
- Senior government officials who were in a position to affect or comment on tobacco-related policies.

One of us (Sheena Hudson) interviewed a politician from each of five political parties; some holding or having held relevant ministerial portfolios, or holding shadow portfolios. The other five participants were managers or senior advisors from the Ministries of Health, Education and Social Development, whose roles involved or had involved them in legislation, advice to ministers, or policy implementation related to tobacco.

The audio-recorded interviews were on the basis of anonymity, and involved a semi-structured questionnaire format. They were conducted during May–August 2006.

Results—The interviewees’ comments indicated that they knew the general directions of tobacco company activities. They were sceptical of the motives of the tobacco companies’ attempts to be community or health minded, and believed that these companies were engaged in successfully selling a product that was harmful to health. All but one of the participants were clear in their opinion that promotion of tobacco to under 16 year olds still occurs in New Zealand.

However, all 10 interviewees appeared ignorant of the investment by the five Crown Financial Institutes (New Zealand Superannuation Fund, the Government Superannuation Fund, the National Provident Fund, the Accident Compensation Corporation [ACC], and the Earthquake Commission) in tobacco companies. They did not appear to have heard of, or have remembered, the media reporting of research that in December 2005 revealed the extent of this investment (e.g. 1–3). This was despite this media coverage detailing the New Zealand Government involvement in the announcement of the research, including the presence of the Minister of Finance as a speaker at the release of the report on this investment.4

Discussion—While the Minister of Finance has stated that Government has ‘clear and transparent policies for the holding and management of Crown financial assets’, 4 it appears that there is still a considerable need for him to inform Members of Parliament and government officials about investments which may prejudice New Zealand’s reputation. Since December 2005, there appears to have been no change in...
government policy on investment in tobacco companies (except for a statement of intent to change by ACC).

As the level of knowledge about such investment is so low, even amongst those closely concerned with tobacco-control policymaking, perhaps even wider action is needed. If the Minister of Finance wishes to be seen to have clarity and transparency about Crown investments, the New Zealand Government may also need to be much more proactive about informing the public about government investments. This is particularly so for investments such as these, which may prejudice New Zealand’s reputation, and which definitely endanger New Zealand and international public health.

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Protecting the mental health of lawmakers in New Zealand

Given the important decisions made by lawmakers, especially at times of national crisis, it is critical that their judgement is not impaired by excessive or chronic psychological stress, or attempts to relieve this by substance misuse. However, recent events in this country indicate that some lawmakers are subject to unacceptably high levels of stress. These include media and political intrusion into their own private lives and their families’ lives; and frequent personal verbal attacks in Parliament.

Experienced politicians have spoken frankly about the chronic and somewhat unpredictable stress associated with their roles. Furthermore, searches of media reports over recent years indicate that politicians take “stress leave”, take leave to repair damaged personal relationships, and seek treatment for alcohol problems. There has also been a conviction for driving while intoxicated, and the smell of alcohol on the breath of fellow politicians is sometimes the subject of Parliamentary debate (though these events may not necessarily reflect chronic alcohol misuse).

While some type of anonymous survey of occupational stress and substance misuse amongst politicians is probably desirable, the response rate is unlikely to be high for this busy group. Therefore we suggest a few simple ways in which the mental health of this particular occupational group might be improved. The first grouping of these responses can be considered to relate to work demands and “work–life balance”.

1. Enhancing the privacy of politicians with rules preventing disclosure of where they live and various private aspects of their and their families’ lives. The media could also adopt, or be required to adopt, guidelines on better respecting the privacy of politicians and their families. An independent body with statutory powers is needed to rule on whether reports on politicians’ private lives are relevant to the public interest.

2. Enhancing the management practices within the parties in Parliament so as to increase the level of control at work amongst MPs so that work demands are better matched to an individual’s experience and skill level. It is well established that high demand–low control in the occupational context poses risks to health.

3. Developing systems for increased opportunities for physical activity by politicians during their working week, given the evidence for physical activity being beneficial for reducing general anxiety.

4. Introducing rules that restrict the late night sittings of Parliament to only genuine emergency situations.

The second grouping of approaches relates to the management of relevant environmental factors.

1. The introduction of a statutory office, independent of Parliament, which would: (i) monitor and report on the environments in which New Zealand politicians operate; and (ii) make recommendations on changes which would promote better environments for the work of elected officials.
• The establishment of other systems for reducing the level of slander and abuse directed at politicians both in and outside Parliament. Within Parliament this could be done by having stricter rules that the Speaker of the House imposes, and harsher penalties for breaching these rules. Party leaders could also set, or be required to operate, guidelines so as to reduce the level of slander disseminated in newsletters, “blogs”, and radio talk shows by their own party members.

• Modifying the alcohol use culture in Parliament itself and perhaps reviewing its availability on the premises.

None of the actions we have suggested here are likely to endanger the transparency of our democracy if the appropriate systems are put in place. Indeed, these measures should be favoured by both the public and politicians on the grounds of protecting mental health and allowing Parliament to function more efficiently.

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References:
The ‘weighting’ list

If doctors can be said to have any views about anything at all, they are founded in the unswerving belief that the best things in life, whilst regrettably not free, are at least better paid for with government money.

I was accordingly not surprised to learn on September 25th that the New Zealand Medical Association had “welcomed” the health lifestyle “package of initiatives” of $67 million dollars announced by the Prime Minister, Helen Clark, on September 21st as part of the fight against obesity in children.

In the speech in Auckland announcing this low-calorie lolly scramble, code-named Mission-On, Ms Clark announced “a network of high-profile ambassadors” to spread the message. “From Edmund Hillary and Peter Blake,” intones our leader, “to our All Blacks and Olympians, our icons have been symbols of health and vitality…etc.”

“This programme gives GPs…the option of prescribing physical activity.” Now there’s a thought, but did we need—or ask for—some part of a $67 million dollar incentive scheme?

The advertising industry has been warned of new guidelines that will stop them promoting bad food choices for children. “Getting children and young people away from the TV and computer screens not only limits their exposure to advertising, but also means there is more time to be active,” says the PM.

Excellent sentiment, but read on. Websites, she promises, will be developed with “interactive features” providing “access to coaches, trainers, and virtual buddies…Radio and television programmes will be used to encourage children and young people to think about their lifestyles, to discuss the issues confronting them, and to make healthy food and physical activity choices.”

Get off the trampoline, my son, and come and watch the TV doco on fat kids. Then we’ll surf the net together if you will promise to sit quite still. Or listen passively to a dietitian on talk-back radio.

For the media, i.e. the people who run the country, Mission-On has to be good news, but, in the priorities battle, the New Zealand Medical Association’s uncritical endorsement is inexcusable.

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Survey of the Network of Youth Health Service Providers (NYHSP): affiliated to New Zealand Association for Adolescent Health and Development (NZAAHD)

The age group 10–25 years is the only population group in New Zealand in which the mortality rate hasn't improved.1

Primary healthcare services specifically for young people have been gradually developing over the last 10–15 years. School health services have been growing, alongside community “one-stop shop” services.

School health services have been (and still are) very variable across the country. Schools are providers of education services, but they also recognise that poor physical, mental, and emotional health is a barrier to educational achievement.

Under the Education Act of 1989, all schools have a responsibility to provide a safe physical and emotional environment for students. Most schools have policies around health issues, and some level of health service.

All schools are implementing the new health curriculum, although not all have dedicated health teachers. Nearly all have guidance counsellors and some have visits from public health nurses. Some have enrolled nurses on site and some have registered school nurses on site. Some have visits from Family Planning nurses and some from GPs. Some schools have wellness centres providing a variety of services from alcohol and other drug counselling and social work to physiotherapy and general practice care.

Some doctors are funded through the local Primary Health Organisation (PHO), and other schools fund the health service through their own resources. The Ministry of Health has recently published a booklet Improving the Health of Young People: Guidelines for school-based health care (June 2004). This details how primary health care professionals might work with schools to improve health outcomes for the students. It does not examine any holistic provision of health in the broad sense of whole school health promotion.

These advances are to be commended but there is a long way to go before there is a consistent, comparable, high standard service in all schools. In addition, the most high risk students, who often have the biggest health needs, leave school early. Students may need health care in the holidays when they are away from school. Young people between 16 and 24 still have developmental needs that are not well met by adult focused health care. These factors have led to the development of youth specific services in the community.

Sometimes these are called “one-stop shops”, but it is hard to define what makes up a “one-stop shop” as none of the current services provides everything. Most agree that there needs to be provision for physical, mental, emotional, and social health needs and they all work within a development paradigm, and involve young people in the running of the service.
History—Community youth health services started with the Wanganui Health Service and was then known as the Youth Advice Centre or YAC. It was started in 1994 by a Public Health Nurse who then drew in other services to do sessions at the Centre such as a local GP, the local Family Planning Association (FPA), and Sexual Health Service, the Child and Adolescent Mental Health Service (CAMHS), and Alcohol and Drug Service. The Youth Services Trust now runs it. The following year, the Youth Health Trust in Christchurch opened the 198 Youth Health Centre, which had a different model.

Total funding was negotiated with the Community Trust for start up funding and then a contract developed with the then Regional Health Authority (currently the District Health Board DHB). Staff included a 30 hour a week doctor, nurse, counsellor, social worker, and young people, in addition to administrative staff. All workers were employed by the Trust.

Since then, services have grown up throughout New Zealand, most of which follow either of the two aforementioned models or a mixture. In addition, services also have developed other contracts and sources of funding to make up for the deficiencies in the health funding. There are approximately 14 services from Auckland to Dunedin.

The Otago Youth Wellness Centre in Dunedin is different from the others as it started to provide services for young people who truant from school. The wrap around-service involves different agencies but not much in the way of health on site and referrals are made to outside health services. In Manukau, until very recently, the Manukau Youth Centre used to provide a drop-in youthwork service, but again referred out for health services. It has recently closed.

The Palmerston North one-stop shop provides a drop-in youthwork service, and has recently added in a health component. There are about four or five centres all planning to set up services at the present time around New Zealand.

In 2002, the Government published the Youth Health Action Plan to complement the Youth Development Strategy Aotearoa. The development of school and community youth-specific health services was among the many recommended actions. Funding for primary healthcare now flows from DHB to PHOs, and there has been more and more difficulty in funding not only new youth health services but also maintaining funding for current services.

The nature of the behaviour of young people when accessing health services means that the PHO model of funding is not a sustainable one for a small practice.\(^2\)

Method—To obtain information about the funding sources of current community youth-health services, a survey was performed by email with follow-up by phone or further email. Survey forms were sent to the 14 services in July 2005.
Nine services returned completed forms:

- 198 Youth Health, Christchurch
- Youth Services Trust, Wanganui
- Rotovegas Youth Health, Rotorua
- Café for Youth health, Taupo
- Evolve, Wellington
- Youth One Stop Shop, Palmerston North
- Vibe, Hutt Valley
- Kapiti Youth Support, Paraparaumu
- The Hub, Nelson

Five services did not respond:

- Manukau Youth Centre, Auckland (limited health services on site)
- Directions 200, Hastings
- Otago Youth Wellness, Dunedin (refer out to health services)
- Whai Marama Youth Connex, Hamilton (has no health services as yet)
- The Pulse, Whangarei (is not totally youth-specific but has a large emphasis on reaching young people)

Results—The services are open for between 30 and 50 hours a week, and provide free services for young people between 10 and 25 years, with some variation around the top and bottom age by 1–2 years. See Table 1 for a summary of responses.

Staffing—Five services have young people working there—of the four who don’t, one has volunteer young people at the centre and one has a youth advisory group.

Only one centre has no doctor, one has a doctor full time, and the rest have a doctor working part of the time the centre is open.

All but one of the services employs a nurse. The nurse was present full time except for one centre in which the nurse was part time.

Only four services have counsellors, most of whom are part time except one where there are two FTEs.

Five services have full time social workers, the rest have none

Services—Three services provide recreation programmes, and two are closely linked with other services that provide both art and recreation activities. In addition to primary health care, most provide health promotion and education, and some provide primary mental healthcare.

Four services provide outreach clinics to schools—one is a school for young parents.
Table 1. Summary of survey responses

<table>
<thead>
<tr>
<th>Youth health services</th>
<th>Number of contracts</th>
<th>Hours open</th>
<th>Nurse hrs</th>
<th>Doctor hrs</th>
<th>Peers hrs</th>
<th>Youth worker hrs</th>
<th>Counsellor hrs</th>
<th>Admin hrs</th>
<th>Arts recreation</th>
<th>Youth advisory</th>
<th>Outreach</th>
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<td>1+council</td>
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<td>42</td>
<td>7</td>
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<td>7</td>
<td>70.5</td>
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<td>27</td>
<td>19</td>
<td>80</td>
<td>40</td>
<td>Youth centre</td>
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<tr>
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<td>34.5</td>
<td>65</td>
<td>12</td>
<td>HP89</td>
<td>30</td>
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**Funding sources of contracts:** DHB, MOH, PHO, NCSP, CPU, Toi te Ora, Energy Trust, Rotary, Child Youth and Family (CY&F), Arts Council, Work and Income New Zealand (WINZ), Community donations, Council, Pub charities, Lottery, Public health.

**Funding sources**—All services had a contract with the MOH or DHB; four had additional contracts with their local PHO. All had additional sources of money from sources such as the City Council, Charitable Trusts, Pub Charities, etc. One had funding from WINZ and the Crime Prevention Unit, and two from CYF. The number of contracts ranged from 1 to 9, with an average of 4.

**Future development**—All services would like more funding. All would like to expand their hours, and those without counselling/mental health services see a need to provide them. Most would also like to expand the group work they do and to include parenting and anger management. Those that do not run arts or youth development programmes would like to be able to do so. Those without a social work service would like to be able to provide this service especially in the area of youth justice. Most services would like to be in a position to run more outreach clinics either to rural areas, schools, or youth justice facilities.

**Discussion**—A literature review of the outcomes of youth-specific services, both school and community based, has revealed very little research on this topic. A recent
review by Karen Mathias\textsuperscript{2} found 23 studies, which quantitatively evaluated youth-specific primary health care for 10–24 year olds with some measure of health outcome. Seventeen of the studies looked at access and utilisation, and all of them found high utilisation rates with enhanced access for the socioeconomically deprived and also at risk young women.

Studies looking at mental health show much improved access to primary mental healthcare, especially for young men,\textsuperscript{3,4} however there was no evidence for improved self-reported mental health status among clinic users. These studies also described significant reductions in emergency department use. None of the studies provided sufficient evidence to determine the effectiveness of youth-specific primary health services, and the review makes the point that more research is desperately needed in this area.

The Youth 2000 study\textsuperscript{5} was the first “snapshot” of health and development factors present in the lives of New Zealanders aged between 13 and 17, attending secondary school. Amongst many other self-report items contained in the survey healthcare utilisation was asked about. 83.4\% said they had a family doctor and approximately 50\% felt there were no barriers to obtaining healthcare.

The most common barriers to healthcare identified by the others were:

- Not wanting to make a fuss.
- Could not be bothered.
- Not feeling comfortable with the healthcare provider.
- Too scared.
- Worries about confidentiality.

Other factors identified in the Mathias Review included:

- Cost.
- Embarrassment.
- Distance to travel.
- Inconvenient times.
- Lack of cultural appropriateness.

Many of the youth-specific services in New Zealand did surveys of the young people in their area to find out what they wanted in terms of healthcare, two of which from Auckland and Wellington, have been published.\textsuperscript{6,7}

The youth-specific health service in Christchurch was evaluated in 1997 by Nicola Geddes.\textsuperscript{8} This was limited by lack of a comparison group, however the vast majority of attending young people found the service accessible, appropriate, and acceptable, with the most common reason for attending (77\%) being ‘no cost’ and 30\% saying that they would not have gone anywhere else if the service didn’t exist.

In Rotorua, the youth-specific service has been running for 3 years. Recently it has been having approximately 400 visits a month. In Rotorua, there is only one PHO so it is relatively simple to observe the numbers of young people attending. During the same time period there was no drop in the numbers of young people attending GPs.\textsuperscript{9}
A recent review of best practice in school clinics by Doone Winnard has found four critical success factors that best practice and evidence recommends. These success factors are:

- Wider engagement with school and community.
- Youth focus and participation.
- Delivery of high quality comprehensive care.
- Implementation of effective administrative/clinical systems and governance to support effective service delivery.

**Conclusion**

It has been shown that barriers to healthcare include many factors outlined above. Youth Specific health services have been set up in New Zealand to overcome these barriers. This survey shows that they have great potential. Standards and frameworks have been drawn up to enable them to be cost effective in their delivery of service. The survey also shows that services currently are patchy and often non-existent.

In September 2002, the Government released *Youth Health: A Guide to Action* (Ministry of Health: [http://www.moh.govt.nz/moh.nsf/wpg_Index/Publications-Youth+Health:+A+Guide+to+Action](http://www.moh.govt.nz/moh.nsf/wpg_Index/Publications-Youth+Health:+A+Guide+to+Action)). It had 10 goals to improve the health of young people. The provision of high-quality, youth-friendly, accessible health services was one of the goals. It is time to deliver services for young people that are consistent, drawn up to a national plan, and provided throughout the country in a way that will reduce the inequalities that currently exist.

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‘Nil by mouth’ post gastrointestinal surgery—is there any evidence?

Traditionally, the postoperative management of patients undergoing gastrointestinal (GI) surgery has been to keep them ‘nil by mouth’ (NBM) until the postoperative ileus resolves and bowel function resumes.\(^1\) The passing of flatus or the return of bowel sounds are typically taken as signs of bowel function returning. There is anecdotal evidence to suggest that this practice is commonplace in New Zealand. Clinical trials, however, have shown that the restriction of oral intake of nutrients is not based on sound statistical evidence. Early feeding is not only safe, but may be beneficial.

The restriction of early oral feeding has largely been secondary to concerns over its safety and tolerability, including the perceived risks of aspiration pneumonia and postoperative nausea and vomiting. Bisgaard et al suggests that concerns about anastamotic dehiscence and postoperative ileus may be significant factors in delaying an early resumption of an oral diet, with the notion that restriction of oral feeding gives the GI tract more time to heal and recover.\(^1\)

However, clinical trials do no support these concerns. Rather, there is evidence from several non-randomised and prospective randomised controlled trials (RCTs) that early oral feeding is safe and tolerable in patients undergoing colorectal surgery.\(^2-5\) There was no evidence for an increase in complications such as anastamotic dehiscence or aspiration pneumonia. In addition, these studies showed no increase in the duration of postoperative ileus or length of hospital stay, while some studies have shown a reduction in these outcomes. A RCT of early enteral feeding versus NBM in malnourished patients with peritonitis showed a reduction in the duration of postoperative ileus and major complication rates.\(^6\) Furthermore, a prospective randomised study by Stewart et al showed that early oral feeding post-colorectal resection led to an earlier resolution of postoperative ileus and reduced hospital stay.\(^7\)

A recent meta-analysis looking at 11 RCTs comparing any type of enteral nutrition (orally or via nasoenteric tube) within the first 24 hours following elective GI surgery versus traditional NBM management showed a reduction in the risk of infectious complications.\(^8\) This was independent of the patients’ nutritional status. This landmark study published in the *British Medical Journal* also provided evidence that early nutrition resulted in a statistically significant reduction in hospital stay and thus potentially in total hospital cost. There was also a trend towards reduction in anastomotic dehiscence, wound infection, aspiration pneumonia, intra-abdominal abscess and mortality. The authors’ concluded that ‘nil by mouth’ has no proven clinical benefit and that ‘early enteral feeding’ may be of additional benefit to the patient.

In summary, postoperative surgical outcome following GI surgery depends on a multitude of factors. There is evidence that starving patients in the early postoperative period has no clinical benefit. Early oral feeding has been shown to be safe and well
tolerated in numerous studies. There is Level I evidence to support early feeding—it may reduce the rate of infective complications and shorten length of hospital stay.

Nil by mouth management appears to be common in New Zealand, but in this modern era of evidence-based medicine, we as clinicians may need to review our practices and challenge ourselves to consider contemporary evidence-based treatments. Perhaps, as has been suggested, a larger multi-centre RCT is required before early postoperative feeding becomes the accepted norm.

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References:

**Beta agonists and asthma**

A recent letter\(^1\) in the *NZMJ* advocated prescribing long-acting beta agonists (LABA) with care. The letter cited the recent meta-analysis of LABA which concluded that LABA were shown to increase severe and life-threatening asthma exacerbations as well as asthma-related deaths.\(^2\) A previous meta-analysis of beta agonists concluded that regular beta agonist use for at least 1 week resulted in tolerance to their effects and poorer disease control compared to placebo. Regular use of beta agonist increased airway inflammation and increased asthma exacerbations.\(^3\) The meta-analysis commented on the development of receptor desensitisation and down-regulation along with rebound bronchoconstriction after sudden withdrawal of beta agonists.\(^3\) It was concluded that “to date no randomised trials (of beta agonists in asthma) have demonstrated a reduction in disease progression or in mortality.”\(^3\)

If short- and long-acting beta-agonists are associated with adverse outcomes such as increased airway inflammation; increased severe exacerbations of asthma, and increased deaths whilst lacking a convincing evidence base for use in chronic asthma, then perhaps we, as a profession, need to consider the apparent overuse of these therapies, and the related issue of continuing to ignore other promising approaches.

Are some of the latter overlooked because they are non-medication based? The Medical Council of New Zealand’s position statement regarding relationships between doctors and health-related commercial organisations acknowledges research showing that medical practitioners are influenced and biased by pharmaceutical company interactions.\(^4\)

The meta-analysis referred to above observed that if a study were funded or sponsored by a pharmaceutical company it was more likely to conclude that beta-agonists were helpful (73%) whereas only 10% of studies not declaring such support concluded that beta agonists were helpful.\(^3\)

It is our view that management of asthma is potentially improved by considering other perspectives on the problem. While “inflammation of the airways” has preoccupied mainstream understanding, perhaps it is not the whole answer. Konstantin Buteyko observed that patients with asthma hyperventilated and hypothesised that the dysfunctional breathing caused the asthma, rather than the conventional view of the asthma causing hyperventilation. Buteyko then went on to develop what was later called the Buteyko Breathing Technique (BBT) claiming a positive therapeutic effect.\(^5\)

The few published trials of BBT in adults with asthma have all found mean/median reductions in the order of 85% to 100% for beta agonist use and mean/median reductions of 40% to 50% for inhaled corticosteroid use whilst also decreasing symptoms and maintaining lung function.\(^6\text{-}^8\) In children with asthma, a small case series had mean reductions of 66% for beta agonists and 41% for inhaled corticosteroids.\(^9\) BBT advice regarding medication use is consistent with the advice of the New Zealand Guidelines Group: to use beta agonists only when necessary with early use of inhaled (and/or oral) corticosteroids.\(^10\)
While the mechanism of effect of BBT may not be accepted, or even thoroughly understood, it offers levels of impact that, if produced by a drug, would be adopted widely. Clearly, more research is urgently required before BBT or other breathing retraining approaches are incorporated, if appropriate, into the mainstream of asthma management to help reduce the mortality and morbidity resulting from beta agonist use.

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Bruce Duncan  
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Tairawhiti District Health, Gisborne

References:

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Duncan Harry James Shine

Duncan was born in the small New Zealand town of Putaruru in 1926. He grew up in a rural environment, but attended Mt Albert Grammar School in the big smoke of Auckland.

By all accounts, he was not an outstanding scholar. He was reasonably competent at sciences, but less so at English and Latin.

When offered a chance to attend a live performance of “Hamlet”, he stated that he should not have to pay for the privilege, but should instead be paid to go.

From school, he returned to the family farm, and it was then that he developed what would be a lifetime passion, not to mention a valuable skill, in mechanical engineering.

Too young for active service in WW2, Duncan enlisted in the RNZAF Engineering Corps, and was due to depart as part of the Japan Occupation Force, when a motorcycle accident spelled the end of a fledgling military career.

Duncan’s experiences as a consumer of medical services while recovering from this accident inspired him to train in medicine, and after taking the Medical Intermediate year at Auckland University (a very difficult year, due to the need to make up for deficiencies in his school education) he was accepted into the Otago Medical School, much to the pleasant surprise of his old school masters.

After graduation, Duncan worked mainly in General Practice in the Auckland area, before working his passage to England as a ship’s doctor. He worked for a year as a registrar in Anaesthetics obtaining his diploma and then 4 years as Medical Registrar at Oldham Hospital and Charing Cross Hospital, London, obtaining his Edinburgh membership and later fellowship before training in Oncology at Charing Cross and The Royal Marsden Hospitals. He gained both his diploma and fellowship in Radiation Oncology before returning to New Zealand.

Duncan met his wife Brenda in London in 1966. After his first marriage proposal was rejected for being insufficiently romantic, the second one was accepted, and they then returned to New Zealand, not by normal means, oh no, but in an adventurous manner, overland through the Middle East and Asia via Land-Rover.

Duncan’s initial consultant post in New Zealand was at Waikato Hospital, but after spending some time in Dunedin as a Visiting Specialist, he decided to move south, and took up a position as Radiotherapist at Wakari Hospital in Dunedin in 1970, an event which made the front page of the Otago Daily Times.

At that time, the Department was equipped with elderly Betatron and Telecobalt machines. For the next 22 years, frequently as the only Consultant in the Department, Duncan was the Director of Radiation Therapy Services. He was responsible for many
innovations in that time, including the introduction of chemotherapy in the early 1970s, and the recruitment of new staff and the use of innovative treatment techniques. He always had a passion for teaching, frequently done in his own time, and whole generations of Radiation Therapists owe much to this passion. His clinical ability was always held in high regard by colleagues.

He always drove an elderly Land-Rover (rebuilt more than once), and when, as happens on a few days each winter, Dunedin hill-suburb roads are impassable due to frost or snow, he would tour the city collecting staff, so the show could go on. His engineering skills were often put to good use attempting to fix the Betatron or Theratron before the arrival of the technicians. Periods of leave were often spent working in Papua New Guinea, to allow a colleague there some time off.

In 1990, Duncan’s vision and efforts were responsible for the opening of a new, state-of-the-art department at the Dunedin Hospital site, equipped with a new Linear Accelerator, with the old Betatron (no solid-state components here) finally consigned to a well-earned retirement.

He was also a keen traveller, frequently travelling rough through Asia and Europe, and on one occasion was observed—when the bus he was travelling in from Lhasa in Tibet to Kathmandu in Nepal was held up by major landslides and was likely to be stuck for a considerable period—to collect his pack and set off on foot to complete the journey.

Duncan continued to work until well past retirement age, initially in Dunedin until a suitable replacement could be recruited, and subsequently as a locum in Palmerston North and Wellington. He retired completely in 1997. His legacy remains, however, in the Dunedin Radiation Oncology Department, a modern facility that would not exist were it not for his foresight and energy.

Unfortunately, Duncan’s later years were blighted by illness, especially Parkinson’s Disease, multiple squamous carcinomas of the skin, and melanoma. Despite these illnesses, he remained positive and retained his mental powers.

Although I never had the opportunity to work with Duncan as a colleague, he was a pleasure to know and it was a privilege to be involved in his care in recent years.

Duncan is survived by his wife Brenda; children Fiona, Debbie, Richard, and Jonathan; and five grandchildren.

John North (Radiation Oncologist, Otago District Health Board) wrote this obituary.
Warren John Richard Muirhead

12 October 1927—25 July 2006

Warren died in New Plymouth after a short illness. He was born and educated in Dunedin. Initially he was a primary and secondary school teacher before he opted to do medicine.

He married Barbara Henderson (a physiotherapist) in 1953 and graduated from Medical School in 1959. While at University he won two NZ University Blues in athletics (sprinting and hurdling).

Following graduation, the family went to New Plymouth where he spent two house surgeon years at Base Hospital and then, because of his medical bursary, spent 1962 at the special area of Te Araroa on the East Coast north of Gisborne. From 1963 to 1969, he was in general practice in Gisborne with his brother Rod. He also gave anaesthetics at Cook Hospital Gisborne.

With some pressure from Basil James, he shifted his family back to Dunedin to do a 3-year course in psychological medicine at Ashburn Hall. He graduated from there at the end of 1972 with his DPM and then went to the Psychiatric Dept at Tauranga Hospital where he stayed until 1980. For the next 2 years Warren did GP locums, and one he often mentioned was in Perth. In 1982, he went to the Psychiatric Dept at New Plymouth Hospital and he worked there until his retirement in 1989.

Warren’s interests were wide and varied. He had a superb knowledge of the English language and read voraciously, particularly history and literature (but also good detective yarns). He was very interested in most of the other arts and played golf (deliberately and at his pace!!), croquet, and was also an occasional trout fisherman. Warren had many friends with whom he kept in contact. In their times of stress or illness, he was always available with advice and help.

Growing up in a family with a mother who was a music teacher he was encouraged to participate in this field. He was a very good boy soprano and sang in various choirs and in later years he became a very good tenor and was a member of the Taranaki Male Choir for several years.

Warren accepted his approaching death with equanimity telling me that he had had a great life, and a wonderful marriage.

He is survived by wife Barbara and their children Anthony, Michael, Ruth, and Diana.

Dr Peter Lay (New Plymouth), a friend and colleague to Warren for many years, wrote this obituary.
Coming Off Antidepressants: how to use – and stop using – antidepressants safely


This book has three purposes. The first is to detail the symptoms associated with antidepressant withdrawal. The second is to outline the frequency of these symptoms and what, in the author’s view, was a deliberate attempt by some pharmaceutical companies to minimise the extent of the problem. The third purpose is to give a practical 5-step programme for doctors and their patients to help avoid withdrawal symptoms.

The author is a psychiatrist at Harvard University Health Services and a clinical instructor in psychiatry at Harvard Medical School. The book has many examples of problems encountered in withdrawing patients from their antidepressants. These will be familiar to those who regularly prescribe antidepressants.

Withdrawal reactions when stopping antidepressants include dizziness, vertigo, nausea, headaches, tremor, electric ‘zap’ sensations, anxiety, crying, insomnia, and even suicidality. The rates vary and are strongly related to the half life of the drug, occurring in around 11% of patients in drugs with a long half life such as fluoxetine, to nearly 70% in short half life drugs, such as paroxetine and venlafaxine. The author details attempts to minimise these prevalence figures by drug companies and some academic psychiatrists. The examples are damming and should concern all clinicians.

The five steps advocated to stop antidepressant are entirely sensible clinical practice. Evaluating whether to stop, stopping slowly and monitoring withdrawal symptoms and re-evaluating at the end of stopping. They are practical, including advice on pill cutting and a checklist of antidepressant withdrawal symptoms.

This book is timely and reminds us of another hazard of casually prescribing antidepressants. It also introduces the potential of failing to recognise and diagnose withdrawal reactions and instead misdiagnosing a depressive relapse and resuming antidepressant treatment. It may be that some patients end up remaining on antidepressants to prevent withdrawal reactions rather than to treat ongoing depression. The book adds further weight to this under recognised problem.

Recommended.

Roger Mulder
Associate Professor of Psychological Medicine
Christchurch School of Medicine and Health Sciences