Volume 5 Issue 2 ISSN 1176-2888

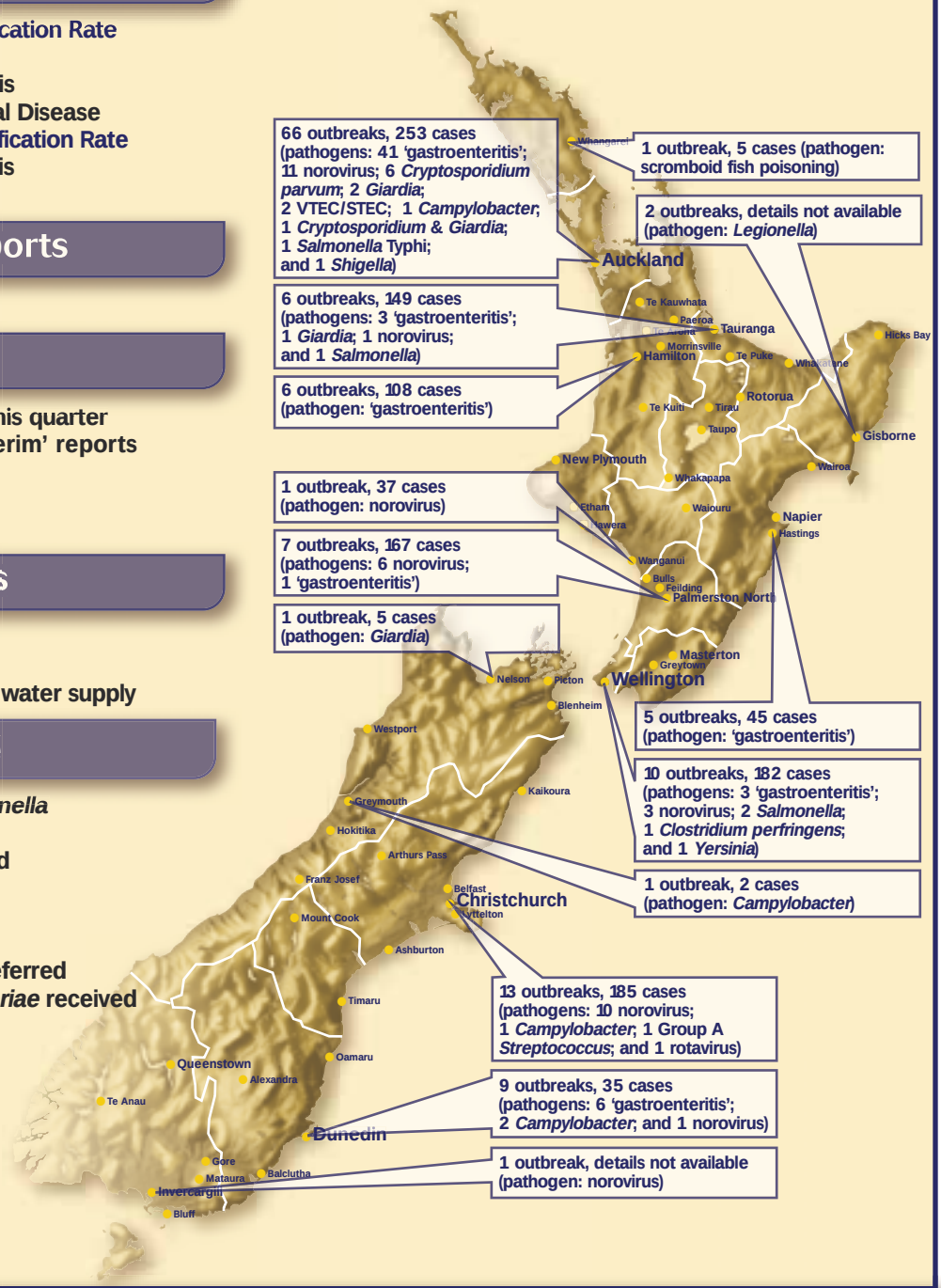
New Zealand Public Health Surveillance Report

June 2007: Covering January - March 2007

Contents & Highlights

This Quarter's Outbreaks

Notification and outbreak data in this issue are drawn from the January – March quarter of 2007. The outbreak map on this page consists of all outbreak information, final and interim. The total number of outbreaks and cases by region and outbreaks by pathogen are reported, as notified up to 10 April 2007.



The 2006 Annual Surveillance Summary and latest reports from STI Surveillance, Antimicrobial Resistance, Virology and Enteric Reference Laboratory are available at www.surv.esr.cri.nz

1. Editorial

Just a virus?

At the time of writing, the Ministry of Health (MOH) is about to embark on another Pandemic Influenza planning exercise, "Cruickshank". This coincides with the annual seasonal influenza vaccination campaign for eligible persons and health care workers (HCW). Despite efforts at encouragement, it is a real struggle to get HCW to accept the vaccine, and hospitals around the country embark on various campaigns to try and increase uptake.

Many HCW seem to regard influenza as a minor illness. Is this partly because we have gone too far with the message to not treat respiratory tract infections with antibiotics, emphasising that they may be caused by "just a virus"? The impact of seasonal influenza is clearly described in the annual ESR reports[1]. Every year at least 1% of the New Zealand population is affected by influenza-like illness (ILI) severe enough to result in a visit to a GP. All age groups, especially the very young, are affected. Patients with influenza are also admitted to hospital, with the winter peak in illness straining the hospital services to the extent that some have to move to emergency management-style planning each winter.

The early and severe peak in 2005 shown in the report only tells part of the story. A strain of influenza B affected children in particular, presumably because younger people were immunologically naïve to this strain[2]. Medical Officers of Health received reports of school closures, up to 20% absenteeism and even several sudden deaths. The most striking feature of this outbreak was how it took communities and health care workers by surprise. Hospital services became aware relatively late, which demonstrates that current surveillance activities for ILI could be expanded to allow rapid detection and outbreak responses. For example, ILI surveillance at Emergency and Paediatrics Departments could be introduced for this purpose.

Pandemic influenza planning includes stockpiling of oseltamivir by individuals and the MOH. Prior to H5N1, oseltamivir was rarely used by clinicians in New Zealand for management of influenza (Roche, personal communication). Anecdotally, it seems that some of the reluctance to prescribe reflects some people's views that it is not necessary to treat "just a virus", in spite of evidence demonstrating clear efficacy when started soon after onset of illness[3]. The data supporting empiric antibiotic use for infective exacerbations of bronchitis or chronic airways disease are far less clear.

So if influenza causes anything from mild to severe disease, but is treatable with amantadine or neuraminidase inhibitors, how will we use these drugs appropriately? The answer of course lies with diagnosis, another area of "influenza nihilism". Influenza can be diagnosed with point of care/near patient care rapid tests, immunofluorescence, viral culture and PCR. The choice of test depends on access to local laboratory facilities, but rapid tests are important when antiviral drugs are contemplated. Epidemiological information, including that required to select strains for future vaccines can only be obtained from viral cultures. The first step in diagnosis is the collection of appropriate samples, with nasopharyngeal or pernasal swabs being generally preferred. It is this author's experience that many doctors and nurses have not been adequately trained in specimen collection for respiratory virus diagnosis.

Prevention with vaccination is better than cure, and although we do well, we could certainly do better for over 65 year-olds and those with chronic medical illnesses. However, we fall well short with our HCW vaccination rates: for example at Capital and Coast DHB, we only manage around 30% staff coverage, with non-clinical staff having higher uptake rates than clinical staff. Perhaps the best reason for staff to be vaccinated is the benefit to their patients. There are four studies demonstrating benefit for patients who are cared for in institutions with good staff vaccination[4]. The most quoted study showed reduction in mortality: 20% of elderly patients in long-term care at non-vaccinating institutions had influenza detected at autopsy compared with 0% in those institutions with high levels of staff influenza vaccination[5]. In my opinion, this protection of patients is probably the key message that should be given to encourage HCW to accept the seasonal influenza vaccination, although others have proposed more draconian methods[6].

Our efforts so far to prevent, diagnose and treat seasonal influenza makes me wonder how we will do with pandemic 'flu'. I do hope however that pandemic planning will help improve the diagnosis, surveillance, treatment and vaccination of seasonal influenza. Such improvement would provide a dividend each year, which is not dependent on the "big bang" of a pandemic. Conversely, doing seasonal influenza better will enable us to cope better with a pandemic.

1 Lopez L, and Huang S. 2006. Influenza in New Zealand 2006, Report to the Ministry of Health. http://www.surv.esr.cri.nz/PDF_surveillance/Virology/FluAnnRpt/InfluenzaAnn2006.pdf

2 Shaw C. 2006. Influenza-like-illness in schools: A case study, Wellington 2005. Report to Regional Public Health.

3 Cooper N J, et al. 2003. Effectiveness of neuraminidase inhibitors in treatment and prevention of influenza A and B: systematic review and meta-analyses of randomised controlled trials. *BMJ 326*: 1235-1240.

4 Influenza Vaccination of Health-Care Personnel Recommendations of the Healthcare Infection Control Practices Advisory Committee (HICPAC) and the Advisory Committee on Immunization Practices (ACIP) Morbidity and Mortality Weekly Report 2006 Vol. 55. RR2.

5 Carman W, et al. 2000. Effects of influenza vaccination of health-care workers on mortality of elderly people in long-term care: a randomised controlled trial. *Lancet* 355: 93-97.

6 McLennan S, et al. 2007. The Health and Safety in Employment Act and the influenza vaccination of healthcare workers. *NZ Med. J.* 120(1250).

Invited contributor Tim Blackmore, Infectious Diseases Physician and Microbiologist, Capital and Coast DHB

2. Notifiable Disease Surveillance

The following is a summary of disease notifications for the January – March quarter of 2007 and cumulative notifications and rates calculated for a 12-month period (April 2006 – March 2007). For comparative purposes notification numbers and rates are presented in brackets for the same periods in the previous year. A robust method of constructing 95% confidence intervals is used to determine 'statistically significant differences' throughout this report unless otherwise stated [see Newcombe, R. G. and D. G. Altman. Proportions and their differences. In: *Statistics with Confidence*. 2000. BMJ Books. Bristol]. Data contained within this report are based on information recorded in EpiSurv by public health service staff up to 10 April 2007. As this information may be updated over time, these data should be regarded as provisional.

National surveillance data tables are available online (www.surv.esr.cri.nz).

Vaccine Preventable Disease

Haemophilus influenzae Type b

- *Notifications:* 5 notifications in the quarter (2006, 1); 13 notifications over the last 12 months (2006, 7) giving a rate of 0.3 cases per 100,000 population (2006, 0.2); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (0 cases); 3 notifications were aged under 5 years and 2 notifications were immunised

Mumps

- *Notifications:* 22 notifications in the quarter (2006, 9); 61 notifications over the last 12 months (2006, 59) giving a rate of 1.5 cases per 100,000 population (2006, 1.4); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (9 cases)

Pertussis

- *Notifications:* 107 notifications in the quarter (2006, 374); 853 notifications over the last 12 months (2006, 2,093) giving a rate of 20.6 cases per 100,000 population (2006, 51.1); a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (159 cases) and from the same quarter last year (374 cases)

Infectious Respiratory Diseases

Meningococcal Disease

- *Notifications:* 17 notifications in the quarter (2006, 31); 146 notifications over the last 12 months (2006, 206) giving a rate of 3.5 cases per 100,000 population (2006, 5.0); a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (40 cases) and from the same quarter last year (31 cases). Notifications were distributed by age as follows, 2 under 1 year of age; 4 (1-4 years); 1 (5-9 years); 1 (10-14 years); 3 (15-19 years) and 6 in the 20 and over category; 1 death was reported in this quarter

Enteric Infections

Campylobacteriosis

- *Notifications:* 4,648 notifications in the quarter (2006, 4,358); 16,163 notifications over the last 12 months (2006, 14,787) giving a rate of 390.5 cases per 100,000 population (2006, 360.8); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (4,398 cases) and from the same quarter last year (4,358 cases)

Gastroenteritis

- *Notifications:* 147 notifications in the quarter (2006, 325); 754 notifications over the last 12 months (2006, 693) giving a rate of 18.2 cases per 100,000 population (2006, 16.9); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly decrease from the same quarter last year (325 cases); note that this is not a notifiable disease *per se* except in persons with a suspected common source or with a high risk occupation, and the term 'gastroenteritis' provides a catch-all category for enteric diseases that are not notifiable and for syndromic reports that come through public health units, including direct reports from the public where the causative pathogen may never be known

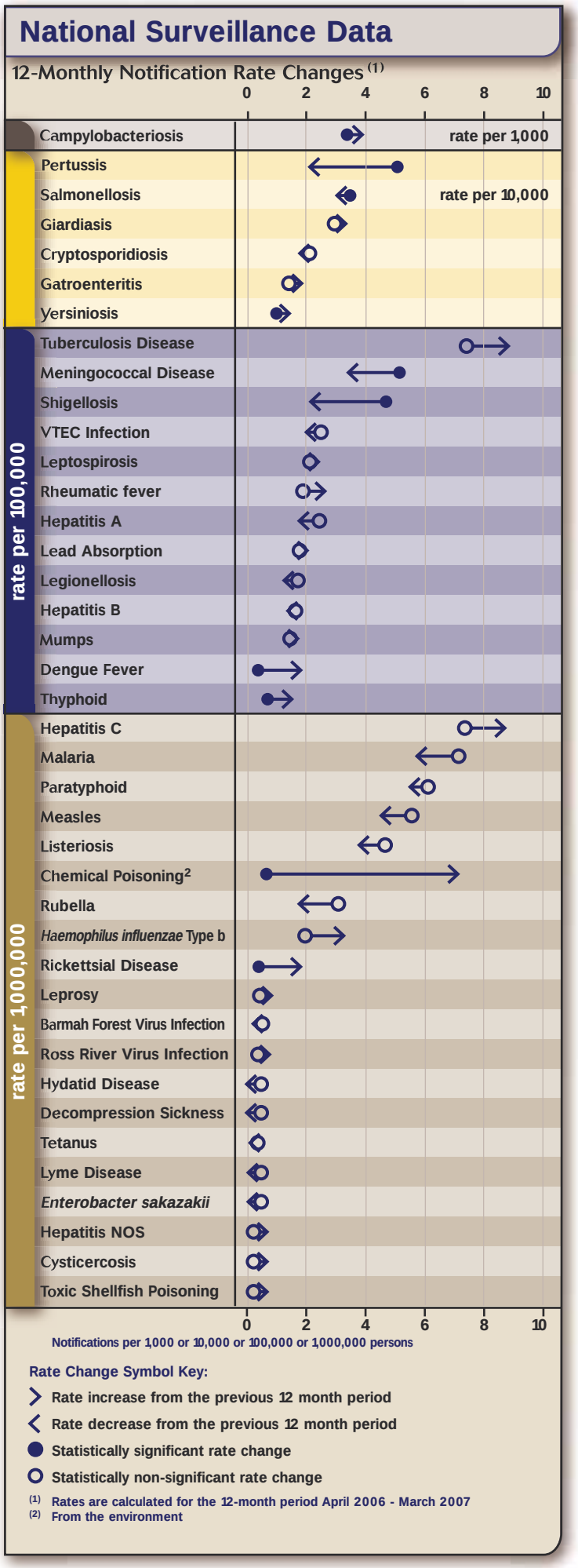
Salmonellosis

- *Notifications:* 374 notifications in the quarter (2006, 450); 1,259 notifications over the last 12 months (2006, 1,465) giving a rate of 30.4 cases per 100,000 population (2006, 35.7); a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (305 cases) and a statistically significant decrease from the same quarter last year (450 cases)

Shigellosis

- *Notifications:* 26 notifications in the quarter (2006, 41); 87 notifications over the last 12 months (2006, 196) giving a rate of 2.1 cases per 100,000 population (2006, 4.8); a statistically significant decrease

National Surveillance Data



continued...

Typhoid

- *Notifications:* 23 notifications in the quarter (2006, 8); 57 notifications over the last 12 months (2006, 28) giving a rate of 1.4 cases per 100,000 population (2006, 0.7); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (8 cases)

VTEC Infections

- *Notifications:* 31 notifications in the quarter (2006, 35); 83 notifications over the last 12 months (2006, 105) giving a rate of 2.0 cases per 100,000 population (2006, 2.6); not a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (16 cases)

Environmental Exposures and Infections

Chemical Poisoning

- *Notifications:* 2 notifications in the quarter (2006, 1); 29 notifications over the last 12 months (2006, 2) giving a rate of 0.7 cases per 100,000 population (2006, 0.0); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (24 cases)

Cryptosporidiosis

- *Notifications:* 212 notifications in the quarter (2006, 94); 854 notifications over the last 12 months (2006, 860) giving a rate of 20.6 cases per 100,000 population (2006, 21.0); not a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (325 cases) and a statistically significant increase from the same quarter last year (94 cases)

Giardiasis

- *Notifications:* 407 notifications in the quarter (2006, 316); 1,305 notifications over the last 12 months (2006, 1,221) giving a rate of 31.5 cases per 100,000 population (2006, 29.8); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (277 cases) and from the same quarter last year (316 cases)

Hepatitis A

- *Notifications:* 16 notifications in the quarter (2006, 63); 75 notifications over the last 12 months (2006, 98) giving a rate of 1.8 cases per 100,000 population (2006, 2.4); not a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly decrease from the same quarter last year (63 cases); all notifications were aged between 6 and 61 years, with 6 cases under the age of 16 years

Yersiniosis

- *Notifications:* 143 notifications in the quarter (2006, 114); 516 notifications over the last 12 months (2006, 419) giving a rate of 12.5 cases per 100,000 population (2006, 10.2); a statistically significant increase

New, Exotic and Imported Infections

Dengue Fever

- *Notifications:* 63 notifications in the quarter (2006, 4); 78 notifications over the last 12 months (2006, 12) giving a rate of 1.9 cases per 100,000 population (2006, 0.3); a statistically significant increase
- *Comments:* there has been a statistically significant increase from the previous quarter (9 cases) and from the same quarter last year (4 cases); 60 cases were laboratory confirmed, 1 case is listed as probable and 2 cases are under investigation; all cases were overseas during the incubation period; countries visited were Samoa, Cook Islands, Rarotonga, French Polynesia, Fiji, Australia, Indonesia, and India

Rickettsial Disease

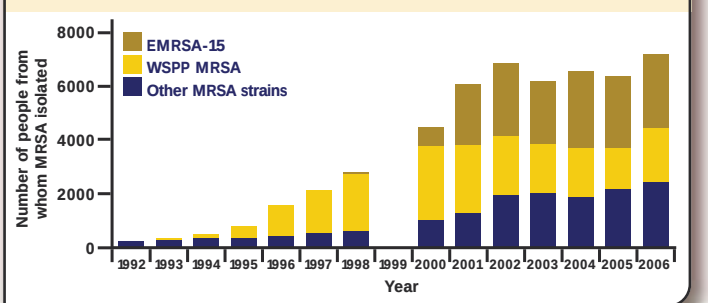
- *Notifications:* no notifications in the quarter (2006, 0); 7 notifications over the last 12 months (2006, 1) giving a rate of 0.2 cases per 100,000 population (2006, 0.0); a statistically significant increase

3. Other Surveillance Reports

Annual survey of MRSA, August 2006

ESR conducts annual one-month surveys of methicillin-resistant *Staphylococcus aureus* (MRSA) to provide information on the epidemiology of MRSA in New Zealand. The 2006 survey was conducted in August 2006. During that month, MRSA were referred from 593 people (579 patients and 14 staff) (Figure 1). This number of referrals equates to an annual incidence rate of 172 per 100,000 – an 11% increase on the 2005 rate of 155 per 100,000. There has been no significant ($p \leq 0.05$) change in the national incidence of MRSA over the last five years since 2002.

Figure 1 MRSA isolations, 1992-2006[a]



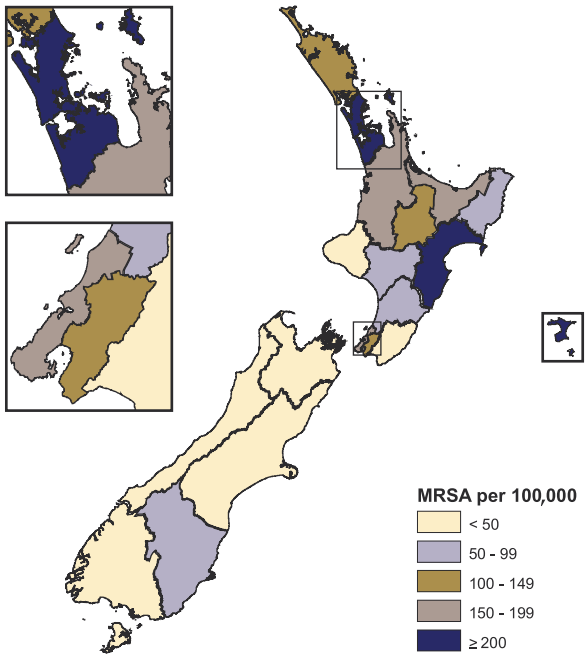
a Data between 1992 and 1998 based on continuous surveillance of all MRSA isolations. Data for 2000-2006 is annualised and based on one-month surveys conducted in these years. No survey was undertaken in 1999.

There are marked geographic variations in the incidence of MRSA in New Zealand (Figure 2). In 2006, the highest annualised incidence rates were in the Waitemata/Auckland/Counties Manukau (299.6 per 100,000), Hawke's Bay (263.0), Capital and Coast (189.2), Waikato (171.7), Bay of Plenty (157.0), Northland (144.3), Lakes (141.7) and Hutt (138.7) District Health Boards. Differences in screening policies may contribute to some of the apparent differences in incidence.

As has been the situation for the last six years, the two most commonly identified MRSA strains were the EMRSA-15 strain, which represented 38% of all isolates, and the WSPP MRSA, which represented 28% of isolates (Figure 1). Together these two strains accounted for two-thirds of MRSA. The prevalence of other strains was: AK3 MRSA strain, 3.7%; WR/AK1 MRSA strain, 3.0%; DN1 MRSA strain, 2.7%; AKh4 MRSA strain, 2.2%; and EMRSA-16, 1.0%. For a description of all these MRSA strains see www.esr.cri.nz/competencies/communicabledisease/MRSA+strains.htm

MRSA was reported as causing infection in 86% of the 421 patients for whom this information was provided. Among the 579 patients with MRSA, 49% were categorised as hospital patients and 51% as community patients. Patients were classified as hospital patients if they were in a healthcare facility (including residential-care facility) when MRSA was isolated or had been in a healthcare facility in the three months before MRSA was isolated. The majority of EMRSA-15 and AKh4 MRSA

Figure 2. Annualised incidence of MRSA by district health board, 2006



(65% and 85%, respectively) were isolated from hospital patients or staff, whereas most WSPP MRSA, AK3 MRSA, WR/AK1 MRSA and DN1 MRSA (73%, 64%, 78% and 69%, respectively) were isolated from people in the community. The age distribution of patients with the two most common strains was quite different, with EMRSA-15 being more frequently isolated from older patients and WSPP MRSA being more common in younger patients.

Overall, 34% of the MRSA were multiresistant, that is, resistant to ≥2 classes of antibiotics in addition to ß-lactams. The EMRSA-15 strain is invariably resistant to ciprofloxacin and often (62% in 2006) resistant to erythromycin, with inducible clindamycin resistance. The WSPP MRSA remain predominantly non-multiresistant, with only infrequent resistance to any antibiotics other than ß-lactams. The AK3 MRSA is usually only resistant to fusidic acid. The WR/AK1 strain is almost invariably resistant to fusidic acid and high-level mupirocin. The DN1 MRSA is typically resistant to ciprofloxacin and erythromycin.
The AKh4 MRSA is multiresistant to ciprofloxacin, clindamycin, co-trimoxazole, erythromycin, gentamicin and tetracycline.

For a more detailed report see www.surv.esr.cri.nz/PDF_surveillance/Antimicrobial/aMRSA_2006.pdf

Reported by Helen Heffernan, Communicable Disease Programme, ESR

4. Outbreak Surveillance

The following information is a summary of the outbreak trends for New Zealand, from data collected in the last quarter (January - March 2007). Comparisons are made to the previous quarter (October - December 2006), and to the same quarter in the previous year (January - March 2006). Note that the outbreak data in this section are notified to ESR by the Public Health Services.

General

- 129 outbreaks notified in this quarter (1173 cases)
- 59 are 'final' reports (707 cases); 70 are 'interim' reports (466 cases) that have yet to be finalised and closed

All following data pertain to final reports only.

- 12.0 cases on average per outbreak, compared with 12.7 cases per outbreak in the previous quarter (11.7 cases per outbreak in the same quarter of last year)
- 52 hospitalisations: norovirus (51 cases), and VTEC/STEC (1 case)
- no deaths

Pathogens

- 23 norovirus outbreaks (433 cases) during this quarter
- 20 'gastroenteritis' outbreaks (192 cases)
- 4 *Campylobacter* outbreaks (10 cases)
- 3 *Cryptosporidium parvum* outbreaks (7 cases)
- 3 *Giardia* outbreaks (10 cases)
- 2 *Salmonella* outbreaks (22 cases)
- 1 *Clostridium perfringens* outbreak (17 cases)
- 1 Group A *Streptococcus* outbreak (8 cases)
- 1 VTEC/STEC outbreak (2 cases)
- 1 *Yersinia* outbreak (6 cases)

Modes of Transmission

Note that reporting allows for multiple modes of transmission to be selected. In many instances no mode of transmission is selected for outbreaks notified to ESR, consequently, numbers may not add up to the total number of outbreaks reported.

- 30 person-to-person, from (non-sexual) contact with an infected person (including droplets): 19 norovirus (425 cases), 5 gastroenteritis (111 cases), 2 *Giardia* (8 cases), 1 *Campylobacter* (2 cases), 1 *C. parvum* (2 cases), 1 Group A *Streptococcus* (8 cases), and 1 *Yersinia* (6 cases)
- 11 foodborne, from consumption of contaminated food or drink (excluding water): 4 gastroenteritis (10 cases), 3 *Campylobacter* (6 cases), 2 *Salmonella* (22 cases), 1 *C. perfringens* (17 cases), and 1 VTEC/STEC (2 cases)
- 3 environmental, from contact with an environmental source (e.g. swimming): 2 norovirus (77 cases) and 1 gastroenteritis (27 cases)
- 4 other mode of transmission: 3 norovirus (via fomites, airborne) (98 cases) and 1 gastroenteritis (via fomites) (35 cases)
- 19 mode of transmission unknown: 11 gastroenteritis (71 cases), 4 norovirus (8 cases), 2 *C. parvum* (5 cases), 1 *Campylobacter* (4 cases), and 1 *Giardia* (2 cases)

Circumstances of Exposure/Transmission

Common 'settings' where exposure/transmission occurred or contaminated food/beverage was prepared for consumption are identified below. Note that multiple settings can be selected and in many instances no settings are selected in outbreaks notified to ESR.

- 16 home: 5 norovirus (18 cases), 4 *Campylobacter* (10 cases), 3 gastroenteritis (18 cases), 2 *Giardia* (8 cases), 1 *C. parvum* (2 cases), and 1 *Salmonella* (3 cases)
- 9 hospital (continuing care): 4 norovirus (85 cases), 4 gastroenteritis (138 cases), and 1 Group A *Streptococcus* (8 cases)
- 8 rest home: 7 norovirus (215 cases) and 1 gastroenteritis (27 cases)
- 4 hospital (acute care): 3 norovirus (72 cases) and 1 gastroenteritis (35 cases)
- 4 café: 1 *Campylobacter* (2 cases), 1 *C. perfringens* (17 cases), 1 gastroenteritis (4 cases), and 1 norovirus (6 cases)
- 4 takeaway: 3 gastroenteritis (6 cases) and 1 norovirus (2 cases)
- 3 childcare: 2 gastroenteritis (9 cases) and 1 *Yersinia* (6 cases)
- 1 camp: *Campylobacter* (4 cases)
- 1 community: *Salmonella* (19 cases)
- 3 'other setting' : 2 gastroenteritis (5 cases) (playground café) and 1 norovirus (37 cases) (military base)
- 13 outbreaks with no setting selected: 6 gastroenteritis (14 cases), 3 norovirus (6 cases), 2 *C. parvum* (5 cases), 1 *Giardia* (2 cases), and 1 VTEC/STEC (2 cases)

5. Outbreak Case Reports

Adenovirus type 8 outbreak

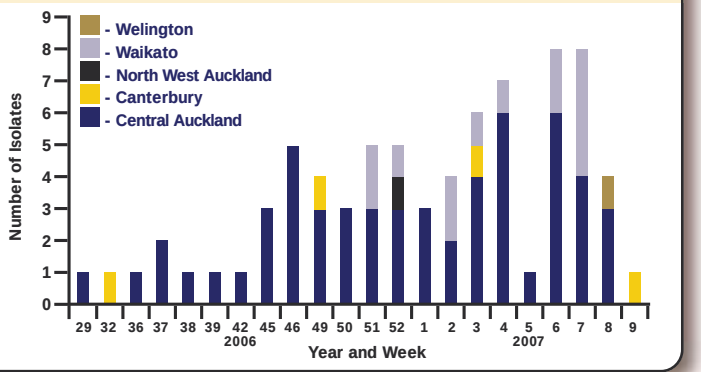
The New Zealand adenovirus laboratory network comprises four laboratories: one public health virology laboratory (ESR, Wellington) and three hospital virology laboratories in Auckland, Christchurch, and Waikato. The adenovirus surveillance is a year-round routine diagnostic surveillance for hospital in-patients and out-patients. Hospital laboratories report all adenovirus isolations and/or typing results weekly to ESR and these data are then made available nationally. Untyped or untypable adenoviruses are referred to ESR for identification.

Adenoviruses are DNA viruses comprising 49 serotypes that are classified into six subgenera (A-F). Infection with adenoviruses can produce a variety of clinical conditions including conjunctivitis. Adenovirus-associated conjunctivitis may be caused by a broad array of serotypes, whereas epidemic keratoconjunctivitis (EKC) has only been associated with serotypes 8, 19a, and 37 (subgenus D).

EKC has been recognised for a century as a nonpurulent conjunctivitis associated with a characteristic keratitis that can spread rapidly within a community[1]. EKC is a severe, painful, and highly contagious eye infection, which often causes outbreaks in settings where people seek ophthalmologic care[2]. The infection is frequently transmitted via the ophthalmologist's hands, contaminated instruments, or eye drops. EKC can last for up to four weeks and is often predominantly caused by adenovirus type 8 (Ad-8). In 1958, the first description of EKC due to Ad-8 was published by Jawetz and coworkers. The case was a merchant seaman who had traveled from the Far East to San Francisco and presented to an eye clinic with a severe conjunctivitis[3].

This is a short summary of the biggest Ad-8 outbreak reported in New Zealand. A total of 75 Ad-8 cases were reported from whom the specimens were collected from July 2006 to March 2007. The first case of Ad-8 occurred in a 2-month-old girl from Auckland in July 2006. The Ad-8 activity increased during the summer period from November 2006 to March 2007, with the highest number of reported cases occurring in the middle of February 2007 (8 cases each in weeks 6 and 7) (Figure 3). The majority of cases occurred in Central Auckland (56, 74.7%) with occasional cases reported in Waikato (13, 17.3%), Canterbury (4, 5.3%), North West Auckland (1, 1.3%) and Wellington (1, 1.3%). Of the 75 cases, 33 were male and 42 were female (M/F ratio 0.79:1). The median age was 40 years, ranging from 6 weeks to 82 years. The main clinical presentation of the patients was conjunctivitis.

Figure 3. Laboratory confirmed Adenovirus type 8 isolations, 2006-2007



1 Gottsch J D. (1996). Surveillance and control of epidemic keratoconjunctivitis. *Trans. Am. Ophthalmol. Soc.* 94: 539-587.

2 Vainio K, Borch E, and Bruu A L. (2001). No sequence variation in part of the hexon and the fibre genes of adenovirus 8 isolated from patients with conjunctivitis or epidemic keratoconjunctivitis (EKC) in Norway during 1989 to 1996. *J. Clin. Pathol.* 54(7): 558-561.

3 Jawetz E, Hanna L, Nicholas A, and Hoyt R. (1958). Some biological characteristics of adenovirus type 8. *Am J Hyg* 67(3): 276-285.

Reported by Sue Huang, Communicable Disease Programme, ESR

Pontiac Fever outbreak, Gisborne

Health Protection in Gisborne was notified of a suspected link between three hospitalised patients on 15 January 2007. The three patients had been admitted between 12 and 15 January, were all employees of the same horticultural nursery, had similar presentations (fever >39°C, photophobia, headache, lower back pain, joint pains, chills and sweats, and respiratory weakness) and the same onset date of 12 January.

Interviews were conducted and it was established they were part of a group of 10 workers involved in bagging up plants at the nursery during the preceding week and that several others of the group had also complained of similar symptoms.

Confirmation that *Legionella* DNA had been detected by PCR in the samples from the three patients was received during 16 January. With a short incubation period and the absence of pneumonia it was decided with the clinicians that the most likely diagnosis for these cases was Pontiac Fever.

A site visit was undertaken and samples of potting mix and other potential environmental sources were collected. Management were interviewed regarding systems, suppliers, and the use of personal protective equipment (PPE).

A hopper in the nursery was used in the bagging process. Large fadges of potting mix were loaded into the hopper from which the workers, standing only a few centimetres away, released smaller quantities of the potting mix into bags that contained small trees together with 'aerosolised compost'. No PPE was worn by the work group. The potting mix had been pre-mixed in Auckland where it had been treated with methyl bromide.

Seven further workers were interviewed, six of whom had similar symptoms and onset dates to the hospitalised cases with three developing a dry cough. All except one of the non-hospitalised cases had recovered after two days. Out of ten exposed workers, nine fell into the working case definition with symptoms suggestive of Pontiac Fever.

Upon the advice of the outbreak control team the nursery voluntarily closed the production room until the outcome of the environmental testing was known.

Samples from two of the three admitted patients were culture-positive for *Legionella longbeachae* serogroup 2, which was also isolated from the potting mix samples. No other *Legionella* species were isolated from the potting mix samples or from the other environmental samples collected.

The remaining in-patient case was positive from paired sera. Of the further seven exposed workers, two seroconverted against *Legionella longbeachae* serogroup 2, two showed low stable titres not diagnostic of a recent infection, and three were inconclusive due to no second sample.

The company has now introduced appropriate use of PPE and the workplace has been reopened. This outbreak investigation has highlighted again the implications for the use of compost in the workplace. An industry standard that includes suitable warnings and the need for the use of PPE are recommended. Other implications include the possibility of the under-diagnosis of non-pneumonic legionellosis as a cause of nonspecific viral type illness.

Reported by Dr Geoffrey Cramp, Public Health Physician, Alan Hall, Principal Health Protection Officer, Kate Sykes, Health Protection Officer, Tom Scott, Group Manager, Dr Bruce Duncan, Medical Officer of Health, Public Health Unit, Tairawhiti District Health, Gisborne

Norovirus contamination of a ski resort water supply

On 27 July 2006, Public Health South was alerted to a large gastroenteritis outbreak at Cardrona Ski Resort (CSR) through staff absenteeism 16 to 27 times higher than normal (3 small outbreaks were also self-reported on 26 and 27 July by recent visitors to CSR). The epidemic curve suggested either a common source outbreak or rapid person-to-person spread. Comprehensive information about the water and sewerage systems was sought and parts of the water and sewerage systems visualised but accurate plans were not available and inspection limited by the terrain. The restaurant, child-care and health-care facilities were also inspected. There was no obvious source for the outbreak, and so comprehensive outbreak control measures were implemented.

214 CSR staff (45%) completed a questionnaire, with 111 fitting the probable case definition of nausea (71%), vomiting (79%) or diarrhoea (67%) on or after 21 July. Staff who drank water at the staff cafeteria on 24 July, were twice as likely to develop acute gastroenteritis than those who did not (Relative Risk (RR) = 2.01 95% CI 1.53-2.81). The questionnaire did not differentiate between boiled water (from a hot water dispenser) and cold tap water. The RR for unboiled water is likely to have been higher. There were no other exposures with a statistically significant increased risk of illness. Four more staff members were subsequently identified from GP notification and laboratory results. In addition, 103 visitors are known to have developed acute gastroenteritis but the actual number is thought to be much higher.

The water supply for the ski field was described as coming from a natural spring/small lake uphill of the sewerage system, with filtration and UV treatment at the base facility prior to use. No recent problems with the treatment system or recent maintenance work on the sewage system was reported. However, following the microbiological results, it was determined that some water was being extracted from a stream downhill from the sewage storage pond, that this pond was unlined, and that water from the stream continued to be used for 3 days after the outbreak was first recognised. Later, it was found that on 22–23 July a septic tank had overflowed with the overflow likely to have reached the water extraction point in the stream.

Water samples taken on 27 July showed significant levels of *E. coli* (range 7.4 – 220 *E.coli*/100 mls) in water at the source, storage tanks and the main CSR building, and norovirus (NV) Genogroup I (GI) in water taken from the main building. Source stream water sampled on 3 August was also positive for NV GI. NV GI was detected in faecal specimens from 11 of the 31 cases who provided specimens (35%). In addition, cases of rotavirus (1 case), *Cryptosporidium* (4 cases) and *Campylobacter* (2 cases) were also identified.

This is New Zealand's largest waterborne NV outbreak and the first to be conclusively linked to a community water supply. Timely microbiological investigation and new laboratory techniques (ultrafiltration of water combined with NV quantitative real-time RT-PCR and DNA sequencing) greatly assisted the investigation. Several issues of concern arose from this outbreak: lack of notification to the Medical Officer of Health despite early involvement of a local general practitioner; slow disclosure of important environmental information; absence of a staff sickness policy and sick staff (including food-handlers) continued to work; insufficient hot water supplies; the unregistered drinking water supply was managed by an untrained person and had no risk management plan; and public health officials had no legislation with which to close CSR although the Territorial Local Authority could have closed the buildings under section 123c of the Building Act 2004.

NV GI is uncommon in New Zealand, accounting for less than 5% of identified outbreaks between 2004-2006 (G Greening, ESR, unpublished data). However, NV GI was responsible for another waterborne NV outbreak in New Zealand, which also occurred at a ski resort[1,2]. Other researchers have described the involvement of GI strains in waterborne outbreaks[3-5]. Many waterborne NV outbreaks have been associated with cold temperatures, showing that biological contaminants, including NV in human sewage can survive in cold alpine environments[1,5-7]. Faecal samples with NV GI have been shown to have 100 times less viral load than samples with GII[8], which may explain why secondary cases were not prominent in this outbreak.

1 Brieseman M, et al. 2000. A series of outbreaks of food poisoning? *NZ Med. J.* 113: 54-56.

2 Brieseman M. 1996. Outbreaks of Norwalk-like virus infections linked to contaminated water at ski field. *NZ Public Health Report* 3: 93.

3 Parshionikar S U, Cashdollar J, and Fout G S. 2004. Development of homologous viral internal controls for use in RT-PCR assays of waterborne enteric viruses. *J. Virol. Methods* 121: 39-48.

4 Häfliger D, Hübner Ph, and Lüthy J. 2000. Outbreak of viral gastroenteritis due to sewage-contaminated drinking water. *Int. J. Food. Microbiol.* 54: 123-126.

5 Maunula L, Miettinen I T, and von Bonsdorff C H. 2005. Norovirus outbreaks from drinking water. *Emerg. Infect. Dis.* 11: 1716-1721.

6 Anderson A D, et al. 2003. A waterborne outbreak of Norwalk-like virus among snowmobilers-Wyoming, 2001 *J. Infect Dis.* 187: 303-306.

7 Carrique-Mas J, et al. 2003. A Norwalk-like virus waterborne community outbreak in a Swedish village during peak holiday season. *Epidemiol. Infect.* 131: 737-744.

8 Chan M C, et al. 2006. Faecal viral load and norovirus-associated gastroenteritis. *Emerg. Infect. Dis.* 12: 1278-1280.

Reported by Derek Bell, Medical Officer of Health, Public Health South; and Joanne Hewitt, Communicable Disease Programme, ESR

6. Pathogen Surveillance

Unless otherwise reported, pathogen surveillance covers the January - March 2007 quarter.

Enteric Pathogens

The Enteric Reference Laboratory (ERL) is responsible for the confirmation of the following notifiable diseases *Salmonellae*, *Shigellae*, *Vibrio cholerae* O1 and VTEC.

Salmonella (ERL)

Human and non-human Salmonella isolate data are available at www.surv.esr.cri.nz/enteric_reference/enteric_reference.php

- 423 human and 170 non-human isolates were confirmed (2006: 497 and 343 respectively)
- 27 isolates *S.* Typhi confirmed (17 indicate recent overseas travel)
- cluster of 4 (phage type E1a) from South Auckland (January). No common source detected. PFGE profiles were quite distinct from any seen in New Zealand previously as well as 6 retrospective isolates from Tonga
- cluster of 3 (phage type E1a) from Wellington and 1 from Manawatu (February) linked to food imported from the Pacific Islands
- 18 isolates *S.* Typhimurium phage type 156 from Wellington/Hutt linked to a privately catered function

VTEC/STEC (ERL)

- 29 isolates laboratory confirmed (2006, 32)
- these include 3 separate family clusters of 2 cases each (North West Auckland, Waikato and Canterbury)
- PFGE analysis was performed on the isolates from North West Auckland as well as 2 separate isolates from the same area
- profiles obtained from the family cluster were indistinguishable from each other and were closely related to the 2 separate isolates which were also indistinguishable

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Norovirus (Norovirus Reference Laboratory)

- 58 confirmed norovirus outbreaks of which 40 (70%) occurred in rest home (31, 53.4%) and hospital (9, 15.5%) settings
- other outbreaks occurred in catered food settings, a military camp, a school camp and a camping ground
- majority of norovirus outbreak strains identified belonged to Genogroup II (49) although there were also 8 outbreaks associated with Genogroup I strains during this quarter
- predominant genotype was GII/4, accounting for at least 21 outbreaks, including 13 outbreaks in healthcare institutions. Other genotypes identified were GI/2, GII/2 and GII/8

Legionellosis and Environmental Legionella

- 19 legionellosis cases were laboratory identified, no deaths reported
- 17 laboratory-proven cases have been notified, with a further 7 notified cases not being laboratory-proven - 2 cases were notified without any laboratory samples being tested
- 1 outbreak was identified, associated with *L. longbeachae* serogroup 2 exposure of nursery workers, with 5 laboratory identified cases
- the remaining 14 laboratory identified cases were sporadic community acquired pneumonia cases
- 14 fitted the confirmed case definition and 5 fitted the probable case definition
- the 14 confirmed cases demonstrated either antibody titres >512 on two or more occasions (4 cases), or at least a four-fold rise in antibody titre by the legionella IFAT (6 cases), or a rising titre to at least 1024 (1 case), or were culture-positive (3 cases)
- the 5 probable cases demonstrated either stable antibody titres of 512 (3 cases), or a single antibody titre of ≥512 (1 case), or were urinary antigen positive (1 case)
- *L. pneumophila* serogroup 1 was identified as the causative agent in 5 cases
- *L. pneumophila* serogroup 5 was identified as the causative agent in 1 case
- *L. pneumophila* serogroup 10 was identified as the causative agent in 1 case
- *L. longbeachae* was identified in 8 cases, of which 5 were outbreak-associated
- *L. gormanii* was identified as the causative agent in 2 cases
- *L. micdadei* was identified as the causative agent in 2 cases
- Legionellae isolated from domestic water systems including spa pools (2), water blasters (1) and hot water systems (1) included *L. pneumophila* serogroups 1 and 8
- Legionellae isolated from industrial water systems including cooling towers included *L. pneumophila* serogroups 1 and 8, *L. anisa* and *L. quinlivannii*
- Legionellae isolated from composts and soils included *L. bozemanii*, and *L. longbeachae* serogroups 1 and 2

Respiratory Viruses

Influenza Virus

- 5 influenza viruses were reported from laboratory-based surveillance (2006, 7)
- 2 were identified as influenza A from Canterbury
- 3 were identified as influenza B, 1 from Canterbury, 1 from Hawkes Bay and 1 from Waikato. The influenza B from Waikato was further typed as B/Malaysia/2506/2004

Respiratory Syncytial Virus, Rhinovirus & Parainfluenza Virus

- 4 cases of respiratory syncytial virus were reported (2006, 7)
- 1 parainfluenza type 2 and 2 parainfluenza type 3 were reported (2006, 1)

Adenoviruses and Enteroviruses

Adenoviruses

- 125 adenoviruses were reported (2006, 47)
- 113 adenoviruses were serotyped as adenovirus type 1 (1), type 2 (4), type 3 (21), type 4 (2), type 5 (1), type 7 (2), type 8 (75), type 15/29 (2), type 21 (1), type 37 (1) and untypable (3). Adenovirus type 8 was the predominant serotype

Enteroviruses

- 31 enteroviruses were reported (2006, 35)
- 15 enteroviruses were serotyped as Coxsackie B5 (1), Coxsackie A9 (3), Coxsackie A16 (1), Echovirus 6 (3), Echovirus 18 (4), and Enterovirus type 71 (3)

Mycology

A table detailing the biannual summary of opportunistic mycoses and aerobic actinomycetes in New Zealand for the period July – December 2006 is available at www.surv.esr.cri.nz/surveillance/NZPHSR.php

Special Bacteriology

Listeria monocytogenes

- 2 isolates of *Listeria monocytogenes* from human cases were referred (for table of human *L. monocytogenes* cases giving more details see www.surv.esr.cri.nz/surveillance/NZPHSR.php)
- both cases were in adults who were elderly and had underlying illness

Corynebacterium diphtheriae

- 16 isolates of *Corynebacterium diphtheriae* were received for toxigenicity testing, typing and surveillance purposes
- 8 isolates were var. *mitis* strains and 8 were var. *gravis* strains; 1 var. *gravis* isolate was from blood of 21 year old male from Auckland; 15 isolates were from cutaneous sources, patients were aged between 9 and 46 years and came from Auckland (12), Christchurch (2) and Wellington (1)
- 1 isolate of *Corynebacterium ulcerans* was received from cutaneous source in 60 year old male (Christchurch), this species can harbour the diphtheria toxin gene
- all isolates were determined to be non-toxigenic by PCR examination for the toxin gene

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Contributions to this publication are invited in the form of concise reports on surveillance issues or outbreak investigations.

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