Volume 4 Issue 2 ISSN 1176-2888

# New Zealand Public Health Surveillance Report

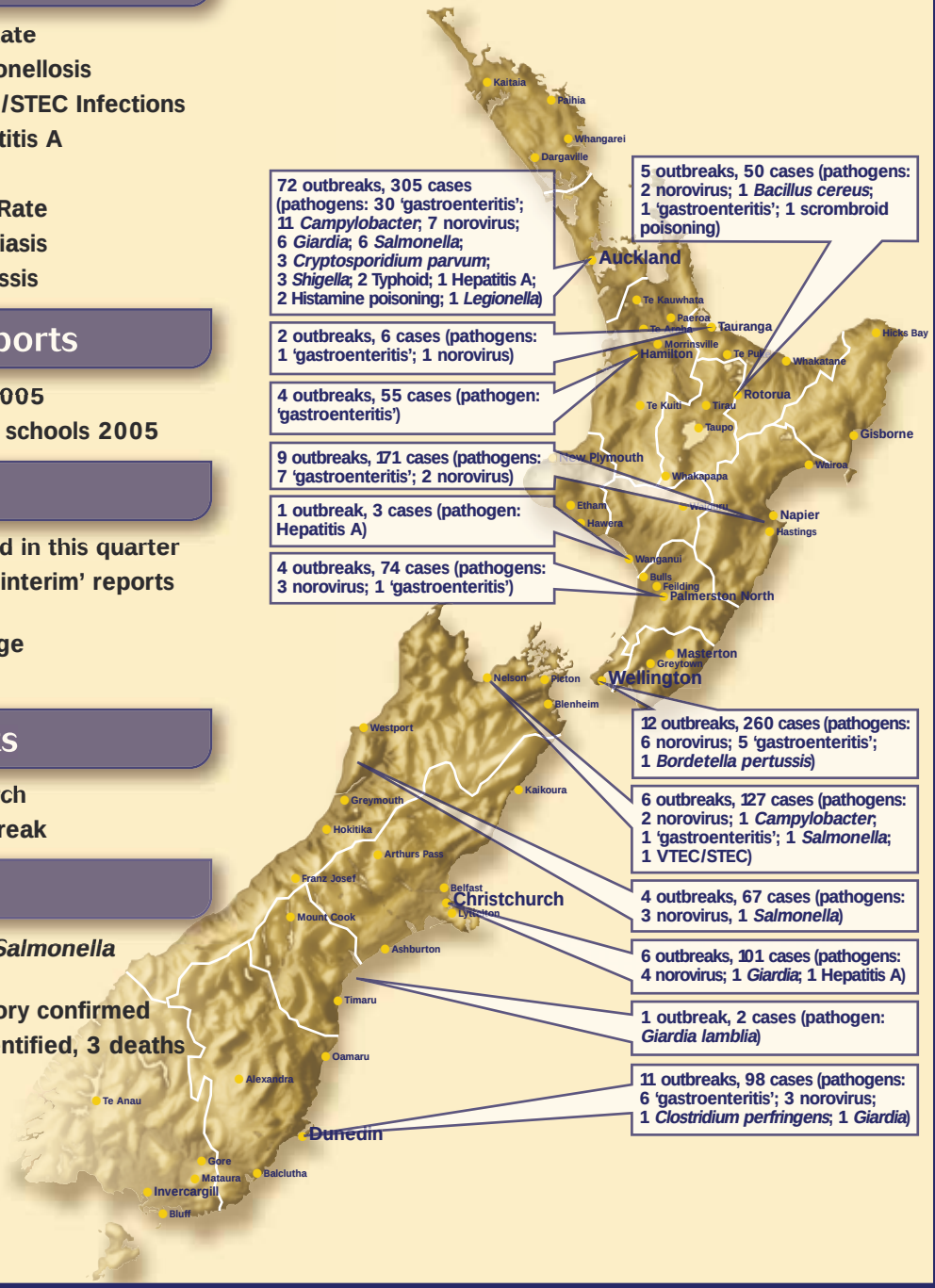
## June 2006

## Contents & Highlights



### This Quarter's Outbreaks

Notification and outbreak data in this issue are drawn from the January – March quarter of 2006. 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 2006.




The 2004 Environmental Health Indicators Report, and annual reports for 2005 for Surveillance of Notifiable and other diseases and STIs, Antimicrobial Resistance, Virological Surveillance, and the Enteric Reference Laboratory are now available at www.surv.esr.cri.nz

# 1. Editorial

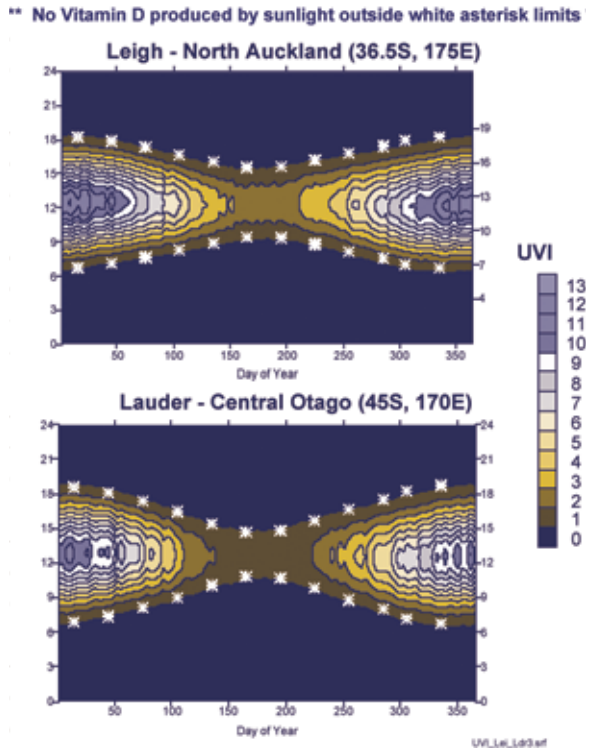
## UV exposure and vitamin D production

The importance of vitamin D for good health has been extensively reported in the recent health literature, with papers relating low vitamin D status to a wide range of disorders beyond the original issue of Rickets. Two New Zealand studies found low vitamin D levels in a significant number of participants, implying that several groups are at risk of insufficiency or even deficiency.[1,2] The implication for public health advice may be that many people should spend more time in the sun, but this message seems difficult to reconcile with the ongoing need to warn against the risk of excessive UV exposure. UV damage causes glaucoma, basal and squamous cell carcinomas, and potentially deadly melanoma. In New Zealand, there are approximately 67,000 new cases of skin cancer per year, with treatment costs exceeding $30 million per year.[3]

We believe it is important that health professionals understand the wide range in UV intensity with time of day, season, and latitude. Advice to the public on UV intensity uses the internationally accepted UV Index (UVI). In New Zealand the peak UVI in summer can exceed 12, but peak values in winter are less than two. Figure 1 shows that there is a strong latitudinal and seasonal dependence in the periods when UV damage can occur (UVI > 3) and when vitamin D can be produced (between the asterisks). Periods of vitamin D productivity were calculated using a web-based tool described by Engelsen et al.[4] Throughout the country, exposure to the midday sun should be avoided over the summer, especially for the 5-hour period centred on solar noon (e.g., from 11 am to 4 pm). However, if the same advice is heeded in the winter, then no vitamin D will be produced. In the south of the country in the middle of winter, vitamin D production is limited to three hours around solar noon. To a reasonable approximation, vitamin D can be produced only when the UVI is one or more. To produce enough vitamin D, most people should expose skin to the sun around midday in winter, but only in the morning or late afternoon in summer. The optimum exposure periods satisfying both requirements are in the brown regions of the plot, where $1 \leq \text{UVI} \leq 3$. Where UVI is less than three, skin damage normally occurs only after exposure periods greater than an hour.

The summer-winter contrast must not be confused with temperature. The tendency to seek shade in hot weather is a helpful but inadequate cue to UV safety, as we cannot directly sense UV intensity. New Zealand suffers extreme incidence of skin cancer partly because periods of high UVI occur even in cool temperatures when we welcome the sun's warmth. With an onshore breeze the air temperature on a fine day at Dunedin's St Kilda beach in January might be only 18°C, as it might also be at Takapuna beach in July, but the UV intensity would be more than five times greater at St Kilda because the sun is high. Temperature also complicates the question of adequate skin exposure for vitamin D requirements. Total vitamin D synthesis is a product of UV intensity, duration, and skin area exposed, as well as other factors. Cold temperatures may make it impractical to make enough vitamin D during winter in the south of New Zealand.

**Figure 1 Calculated seasonal and diurnal variation in UV index for sites near the north and south of New Zealand**




Note: The asterisks mark the limits of the day for vitamin D production. Because of printing limitations, the standard UVI colours have not been used.

Reported by Richard McKenzie, Ben Liley, and Paul Johnston, National Institute of Water & Atmospheric Research, Lauder, Central Otago

1 Rockell, J.E., Green, T.J., Skeaff, C.M., Whiting, S.J., Taylor, R.W., Williams, S.M., Parnell, W.R., Scragg, R., Wilson, N., Schaaf, D., and Fitzgerald, E.D. (2005) Season and ethnicity are determinants of serum 25-hydroxyvitamin D concentrations in New Zealand children aged 5 to 14 y, *J. Nutrition* 135: 2602-2608.

2 Rockell, J.E., Skeaff, C.M., Williams, S.M. and Green, T.J. (In press 2006) Serum 25-hydroxyvitamin D concentrations of New Zealanders aged 15 years and older. *Osteoporosis Int.*

3 O'Dea, D. (2000) The costs of skin cancer to New Zealand. Report to the Cancer Society of New Zealand, 1 Feb 2000.

4 Engelsen, O., Brustad, M., Aksnes, L., Lund, E. (2005) Daily duration of vitamin D synthesis in human skin with relation to Latitude, Total Ozone, Altitude, ground cover, aerosols and cloud thickness. *Photochemistry and Photobiology* 81: 1287-1290.

# 2. Notifiable Disease Surveillance

**The following is a summary of disease notifications for the January - March quarter of 2006 and cumulative notifications and rates calculated for a 12-month period (April 2005 - March 2006). 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 2006. 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

### Hepatitis B

- *Notifications*: 17 notifications in the quarter (2005, 8); 69 notifications over the last 12 months (2005, 33) giving a rate of 1.8 cases per 100,000 population (2005, 0.9); statistically significant increase
- *Comments:* All cases were aged 18 years or older

### Meningococcal Disease

- *Notifications*: 31 notifications in the quarter (2005, 51); 206 notifications over the last 12 months (2005, 335) giving a rate of 5.5 cases per 100,000 population (2005, 9.0); statistically significant decrease

• *Comments*: there has been a statistically significant quarterly decrease from the same quarter last year (51 cases). Notifications were distributed by age as follows, 5 under 1 year of age, 5 (1-4 years), 1 (5-9 years), 3 (10-14 years), 7 (15-19 years), and 10 (20+ years). No deaths were reported in this quarter

### Pertussis

• *Notifications*: 349 notifications in the quarter (2005, 1,000); 2,069 notifications over the last 12 months (2005, 4,160) giving a rate of 55.4 cases per 100,000 population (2005, 111.3); statistically significant decrease

• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (642 cases) and from the same quarter last year (1,000 cases). The data indicates that we are in the tail end of the current epidemic that began in 2004. However, case numbers have not yet decreased to the average number reported (74.5 cases per month) between the two most recent pertussis epidemics (i.e. from mid-2001 to mid-2004)

## ENTERIC INFECTIONS

### Campylobacteriosis

• *Notifications*: 4,346 notifications in the quarter (2005, 3,407); 14,775 notifications over the last 12 months (2005, 11,846) giving a rate of 395.3 cases per 100,000 population (2005, 317.0); statistically significant increase

• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (4,648 cases) and a statistically significant increase from the same quarter last year (3,407 cases)

### Gastroenteritis

• *Notifications*: 314 notifications in the quarter (2005, 189); 682 notifications over the last 12 months (2005, 1,277) giving a rate of 18.2 cases per 100,000 population (2005, 34.2); statistically significant decrease

• *Comments*: there has been a statistically significant quarterly increase from the previous quarter (105 cases) and from the same quarter last year (189 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*: 451 notifications in the quarter (2005, 367); 1,467 notifications over the last 12 months (2005, 1,094) giving a rate of 39.3 cases per 100,000 population (2005, 29.3); statistically significant increase

• *Comments*: there has been a statistically significant quarterly increase from the previous quarter (375 cases) and from the same quarter last year (367 cases)

### Shigellosis

• *Notifications*: 41 notifications in the quarter (2005, 28); 196 notifications over the last 12 months (2005, 135) giving a rate of 5.2 cases per 100,000 population (2005, 3.6); statistically significant increase

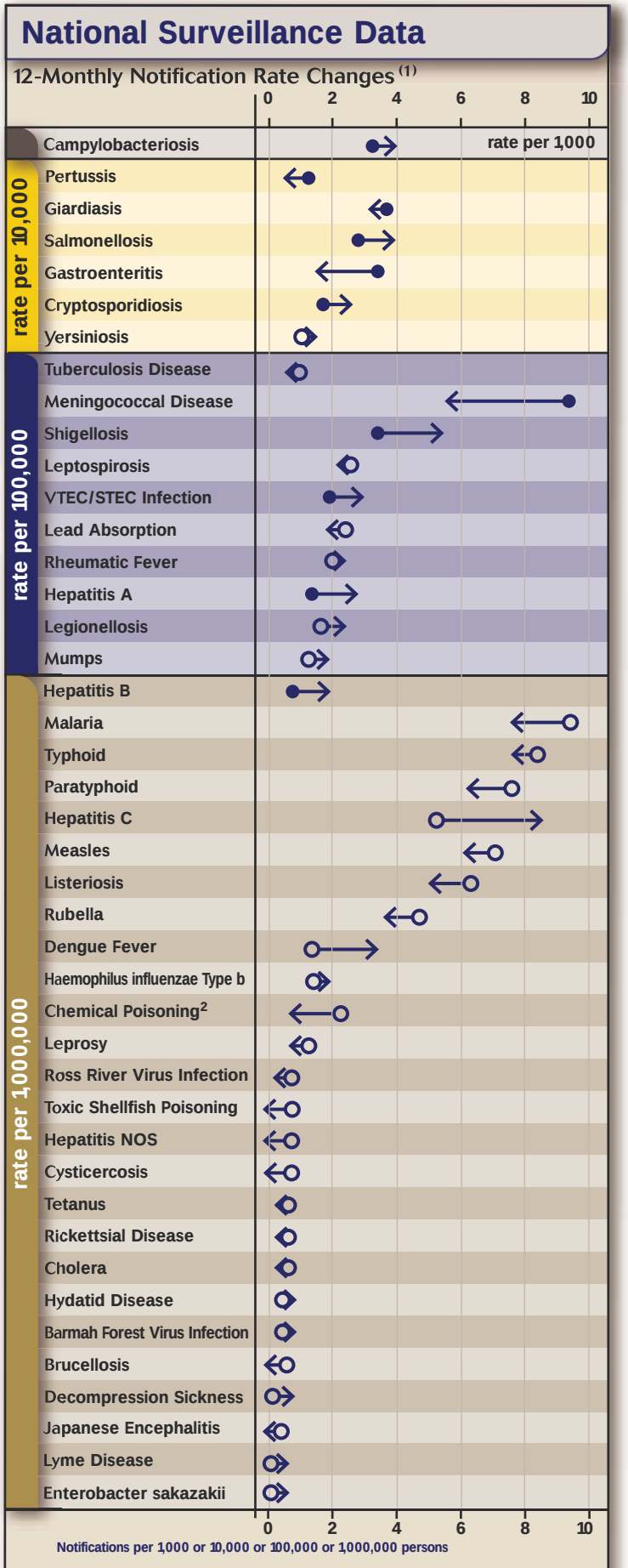
• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (93 cases)

### VTEC/STEC Infections

• *Notifications*: 36 notifications in the quarter (2005, 22); 106 notifications over the last 12 months (2005, 76) giving a rate of 2.8 cases per 100,000 population (2005, 2.0); statistically significant increase

• *Comments*: there has been a statistically significant quarterly increase from the previous quarter (19 cases)

## National Surveillance Data




**Rate Change Symbol Key:**

- Rate increase from the previous 12 month period
- Rate decrease from the previous 12 month period
- Statistically significant rate change
- Statistically non-significant rate change

(1) Rates are calculated for the 12-month period to the end of this quarter.
(2) From the environment

*continued...*

## ENVIRONMENTAL EXPOSURES AND INFECTIONS

### Cryptosporidiosis

- *Notifications*: 94 notifications in the quarter (2005, 123); 860 notifications over the last 12 months (2005, 678) giving a rate of 23.0 cases per 100,000 population (2005, 18.1); statistically significant increase
- *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (362 cases) and from the same quarter last year (123 cases)

### Giardiasis

- *Notifications*: 315 notifications in the quarter (2005, 326); 1,220 notifications over the last 12 months (2005, 1,392) giving a rate of 32.6 cases per 100,000 population (2005, 37.2); statistically significant decrease

### Hepatitis A

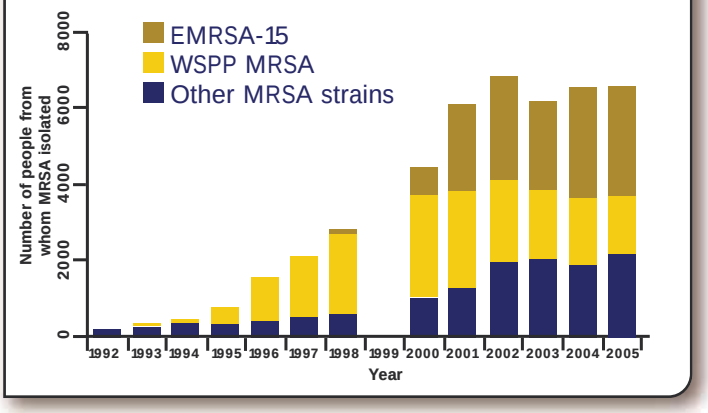
- *Notifications*: 64 notifications in the quarter (2005, 16); 99 notifications over the last 12 months (2005, 47) giving a rate of 2.6 cases per 100,000 population (2005, 1.3); statistically significant increase
- *Comments*: there has been a statistically significant quarterly increase from the previous quarter (17 cases) and from the same quarter last year (16 cases); 25 cases were aged under 16 years

# 3. Other Surveillance Reports

## Annual survey of MRSA, August 2005

ESR conducts annual one-month surveys of methicillin-resistant *Staphylococcus aureus* (MRSA) to provide information on the epidemiology of MRSA in New Zealand. The 2005 survey was conducted in August 2005. During that month, MRSA were referred from 530 people (513 patients and 17 staff). This number of referrals equates to an annual incidence rate of 170 per 100,000 - similar to the 2004 rate of 175 per 100,000 (Figure 2). There are marked geographic variations in the incidence of MRSA in New Zealand (Figure 3). In 2005, the highest annualised incidence rates were in the Auckland (368 per 100,000), Bay of Plenty (182), Waikato (151), Hawke's Bay (151), Capital and Coast (107), and Northland (103) District Health Boards. Differences in screening policies may contribute to some of the apparent differences in incidence.

**Figure 2. MRSA isolations, 1992 - 2005[a]**

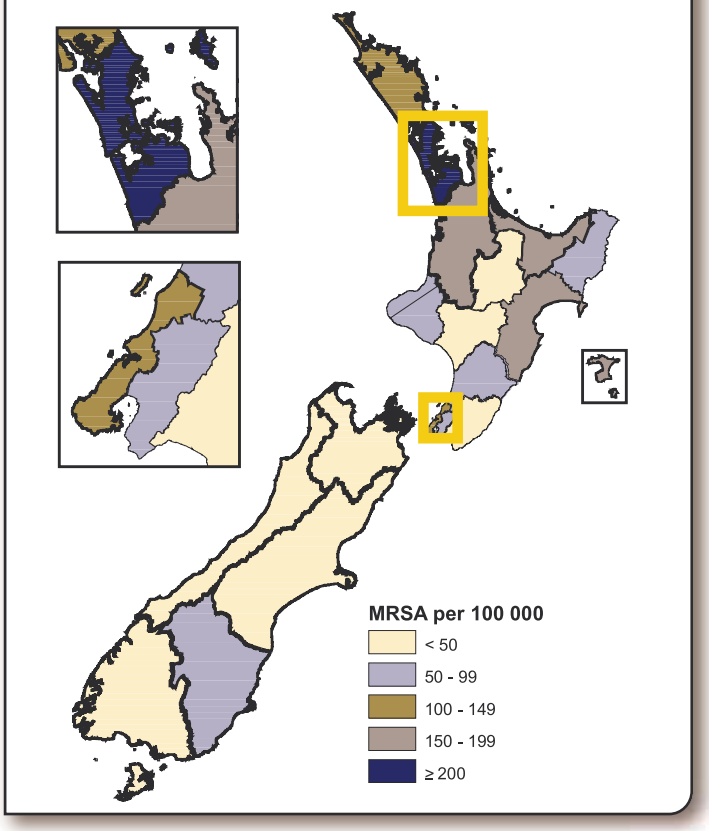



[a] Data between 1992 and 1998 based on continuous surveillance of all MRSA isolations. Data for 2000-2005 are annualised and based on one-month surveys conducted in these years. No survey was undertaken in 1999.

The majority of the MRSA isolates were the EMRSA-15 strain (42%), WSPP MRSA strain (24%), WR/AK1 MRSA strain (5%) or AKh4 MRSA strain (5%). The proportion of MRSA that were the non-multiresistant, community WSPP MRSA decreased again in 2005 – a trend evident since 2000, when the healthcare-associated EMRSA-15 strain emerged and spread in hospitals and residential-care facilities in some parts of the country (Figure 2).

MRSA was reported as causing infection in 77% of the 366 patients for whom this information was provided. Among the 513 patients with MRSA, 51% were categorised as hospital patients and 49% 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 (74% and 69%, respectively) were isolated from hospital patients or staff, whereas most WSPP MRSA and WR/AK1 MRSA (70% and 72%, 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.

**Figure 3. Annualised incidence of MRSA by district health board, 2005**




Overall, 46% 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 (70% in 2005) 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 WR/AK1 strain is almost invariably resistant to fusidic acid and high-level mupirocin. The AKh4 MRSA is typically multiresistant to ciprofloxacin, clindamycin (constitutive resistance), co-trimoxazole, erythromycin, gentamicin and tetracycline.

For a more detailed report see www.surv.esr.cri.nz/PDF_surveillance/Antimicrobial/aMRSA_2005.pdf

**Reported by Helen Heffernan, Communicable Disease Programme, ESR**

# Influenza-like illness in Wellington schools 2005

In late May 2005 an outbreak of viral illness commenced in Wellington (this same illness was seen throughout the entire country). The illness mainly affected school-aged children, and there was considerable media attention due to high levels of absenteeism from schools and a small number of child deaths. Regional Public Health conducted a survey of all schools in Wellington and the Hutt Valley in the last week of May 2005 in order to gauge the extent and causes of the illness. This survey had a 63% response rate (139 schools of 220 in the region). Table 1 shows the level of illness reported at schools.

**Table 1 Level of illness in schools in Wellington and Hutt**

| Level of illness | Number of schools (Percent) |
|---|---|
| Not aware of any illness | 12 (9%) |
| Few extra cases compared to normal | 42 (30%) |
| Some extra illness | 42 (30%) |
| Large amount of extra illness | 38 (27%) |
| Not answered | 5 (4%) |
| Total | 139 |

Other findings included:

(1) High levels of absenteeism, with 25 schools (18%) recording absenteeism above 20%, and 42 schools (30%) reporting absenteeism of 10-20%.

(2) 69 schools (50%) reported that the sickness was increasing.

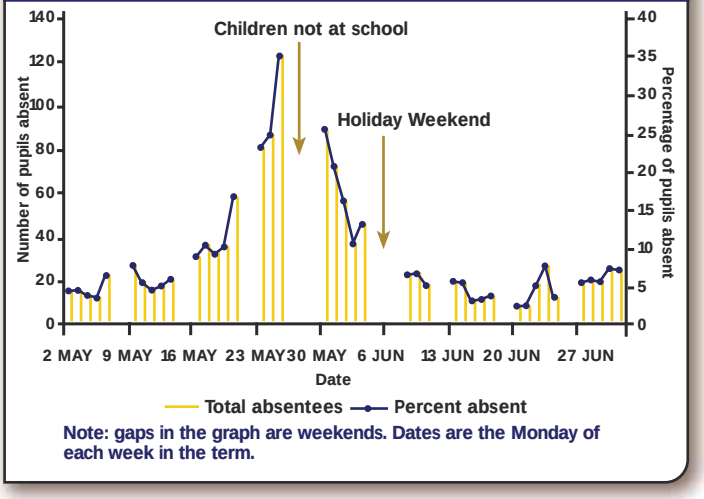
(3) 97 schools (70%) indicated they had absent staff.

(4) 76 schools (55%) reported that ill children had both respiratory and gastrointestinal symptoms.

(5) Illness was widespread with schools in all Territorial Local Authority districts affected.

Figure 4 shows absenteeism over the period of the outbreak (term 2 2005) in School A in Wellington. This school has 350 pupils and covers years 1-8. This school was severely affected and provides an example of how an outbreak can spread through a school.

**Figure 4. Total and percent of pupils absent Term 2 2005, School A**




Note: gaps in the graph are weekends. Dates are the Monday of each week in the term.

Absenteeism rates were between 5-8% in the first two weeks of term, however in week 3 (starting 16 May 2005) absenteeism increased from 9 to17% over the course of the week. By week 4, absenteeism was above 20%, resulting in the school asking children not to come for the last 2 days of that week. By week 5, absenteeism was declining but did not come down below 10% until week 6. Attack rates could not be specifically calculated, but were probably above 30%. The outbreak lasted about a month with a two-week peak in illness.

The national influenza GP sentinel surveillance system showed a simultaneous increase in influenza-like illness (ILI). The increase in ILI was largely due to Influenza B/HongKong/330/2001-like,[1,2] and samples from ill children in Wellington also showed influenza B/HongKong/330/2001-like virus.[1] Results from the survey suggested there were a large number of children presenting with gastrointestinal symptoms, often in combination with respiratory symptoms. There was one confirmed norovirus outbreak in a school in the region. However it is common for children infected with influenza B to have gastrointestinal symptoms.[3]

Although a large number of children were affected, adults were not affected to the same degree. It was hypothesised, at the time of the outbreak, that children were disproportionately affected because of a lack of immunity to the B/HongKong/330/2001-like virus. The genetic antecedent of this particular virus has not circulated in great numbers in New Zealand since 1987, thus the current cohort of children would not have immunity.[4] There were also high consultation rates for ILI in children aged 1-4 years in 2005, consistent with immunologic naivety to B/HongKong/330/2001-like virus.[5] However Influenza B made up 32% of the circulating strains in 2002, with B/HongKong/330/2001-like virus being typed in 90% of the Influenza B isolates,[6] so the explanation above does not entirely clarify why 2005 was such a severe year for children.

Similar phenomena have occurred in other places; the 2002/03 influenza season in Texas and in the 2005/06 influenza season in England.[7,8] In both these outbreaks of Influenza B/Hong Kong/2001-like, children were disproportionately affected with high school absenteeism and, in Texas there were widespread, unprecedented, school closures.[7,8] In Texas, the lack of previous exposure to this virus was also thought to be the reason for the severity of this event in children.[7]

School based surveillance for infectious diseases, including influenza, is sometimes suggested as a possible adjunct to sentinel surveillance. Advantages of school-based surveillance are:

(1) Simplicity - schools are required by the Ministry of Education to keep attendance records on their pupils, thus a surveillance system utilises secondary data, which simply needs to be collated appropriately and passed on.

(2) Timeliness - daily reports allow the system to be almost 'real time', overcoming issues related to a weekly surveillance system.

(3) Potential for intervention - children are thought to be a sentinel population in terms of influenza and have a role in spreading influenza through the remainder of the community.[9-11] Thus detecting an outbreak early in this population would theoretically allow interventions to prevent spread of influenza through the rest of the population.

School-based surveillance programmes have been implemented in a number of places, although only a small number have been evaluated.[12-15] Most of the evaluations conducted have been less than robust, the most comprehensive evaluation concluded that school absenteeism data are too noisy to use for surveillance for emerging infections.[12] There were a large number of days with no data (i.e. weekends and holidays), and twenty percent of data are unusable due to 'normal' levels of pupil absenteeism on certain days being so high they would automatically generate a positive signal (all Mondays and Fridays had to be discarded along with days at either end of holidays). Ultimately this type of surveillance was judged to generate too many false positive signals, which would have been too resource intensive to investigate.[12]

There are a number of issues that need to be clarified if school based surveillance for early detection of infectious disease outbreaks were to be considered in New Zealand. These include:

(1) Determining what are 'normal' levels of absenteeism in New Zealand schools. There is currently no national database of school absenteeism and understanding is limited to national audits carried out by the Ministry of Education every few years.[16-18]

*continued...*

(2) What is the sensitivity and specificity of school based surveillance for various illnesses? Most studies have not considered these issues thoroughly, and the one study that did suggested that sensitivity and specificity were poor.[12]

(3) Practical issues also need to be considered. For example the majority of schools in New Zealand utilise paper-based school rolls,[18] often aimed at looking at an individual child's absence during a week. Transferring these data to a useable format for surveillance may take significant effort.

(4) How much value would school based surveillance add to the existing GP sentinel surveillance system? The 2005 outbreak was detected by the existing surveillance system, so the costs and benefits of a new system need to be carefully weighed up.

The 2005 influenza season disproportionately affected school aged children in Greater Wellington (and nationally). While this was an unusual event for New Zealand,[19] international experience suggests it is not unprecedented.[7,8] This outbreak promoted Regional Public Health to consider the evidence around school based surveillance and strengthened the links between the education sector and Regional Public Health which are useful for our ongoing pandemic planning.

For list of references see - www.surv.esr.cri.nz/surveillance/NZPHSR.php

**Reported by Caroline Shaw, Public Health Medicine Registrar, Margot McLean, Medical Officer of Health, Jill McKenzie, Public Health Medicine Registrar, Regional Public Health, Hutt Valley DHB**

# 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 2006). Comparisons are made to the previous quarter (October - December 2005), and to the same quarter in the previous year (January - March 2005). Note that the outbreak data in this section are notified to ESR by the Public Health Services.**

## General

- 137 outbreaks notified in this quarter (1319 cases)
- 76 are 'final' reports (942 cases); 61 are 'interim' reports (377 cases) that have yet to be finalised and closed

*All following data pertain to final reports only.*

- 12.4 cases on average per outbreak, compared with 6.8 cases per outbreak in the previous quarter (10.8 cases per outbreak in the same quarter of last year).
- 13 hospitalisations: norovirus (10 cases), gastroenteritis (3 cases)
- no deaths

## Pathogens

- 25 norovirus outbreaks (594 cases) during this quarter
- 23 'gastroenteritis' outbreaks (264 cases)
- 8 *Campylobacter* outbreaks (23 cases)
- 8 *Salmonella* outbreaks (19 cases)
- 6 *Giardia* outbreaks (22 cases)
- 2 *Cryptosporidium parvum* outbreaks (5 cases)
- 2 Histamine poisoning outbreaks (4 cases)
- 1 *Clostridium perfringens* outbreak (9 cases)
- 1 *Shigella* outbreak (2 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.

- 44 person-to-person, from (non-sexual) contact with an infected person (including droplets): 21 norovirus (581 cases), 12 gastroenteritis (234 cases), 4 *Salmonella* (11 cases), 3 *Giardia* (12 cases), 2 *Campylobacter* (9 cases), 1 *C. parvum* (3 cases), and 1 *Shigella* (2 cases)
- 27 foodborne, from consumption of contaminated food or drink (excluding water): 8 gastroenteritis (25 cases), 7 *Campylobacter* (18 cases), 4 norovirus (96 cases), 4 *Salmonella* (10 cases), 2 Histamine poisoning (4 cases), 1 *C. perfringens* (9 cases), and 1 *Giardia* (2 cases)
- 5 environmental, from contact with an environmental source (e.g. swimming): 5 norovirus (158 cases)
- 3 waterborne, from consumption of contaminated drinking water: 1 *Campylobacter* (2 cases), 1 *C. parvum* (2 cases), and 1 *Salmonella* (2 cases)
- 2 zoonotic, from contact with an infected animal: 2 *C. parvum* (5 cases)
- 13 mode of transmission unknown: 6 gastroenteritis (19 cases), 2 *Giardia* (8 cases), 3 norovirus (10 cases), and 2 *Salmonella* (4 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.

- 20 home: 5 *Salmonella* (13 cases), 4 norovirus (22 cases), 3 *Campylobacter* (9 cases), 3 *Giardia* (12 cases), 2 gastroenteritis (5 cases), 1 *C. parvum* (2 cases), 1 Histamine poisoning (2 cases), and 1 *Shigella* outbreak (2 cases)
- 16 rest home: 11 norovirus (286 cases) and 5 gastroenteritis (172 cases)
- 13 café: 6 gastroenteritis (41 cases), 3 *Campylobacter* (7 cases), 1 *C. perfringens* (9 cases), 1 Histamine poisoning (2 cases), 1 norovirus (2 cases), and 1 *Salmonella* (2 cases)
- 5 continuing care: 3 norovirus (92 cases) and 2 gastroenteritis (66 cases)
- 3 acute care: 2 norovirus (46 cases) and 1 gastroenteritis (7 cases)
- 2 camp: 1 gastroenteritis (12 cases) and 1 norovirus (50 cases)
- 2 farm: 1 *C. parvum* (2 cases) and 1 norovirus (6 cases)
- 2 takeaway: 1 gastroenteritis (2 cases) and 1 Histamine poisoning (2 cases)
- 2 hotel/motel: 1 *Campylobacter* (5 cases) and 1 gastroenteritis (24 cases)
- 2 workplace: 1 gastroenteritis (24 cases) and 1 *Giardia* (6 cases)
- 1 childcare centre: gastroenteritis (5 cases)
- 1 community: norovirus (11 cases)
- 1 other food outlet: gastroenteritis (2 cases)
- 1 school: norovirus (89 cases)
- 5 'other setting': 2 overseas acquired: 2 *Giardia* (4 cases); 1 holiday home: norovirus (5 cases); 1 town hall: norovirus (74 cases); and 1 wedding hall: gastroenteritis (8 cases)
- 11 outbreaks with no setting selected: 4 gastroenteritis (10 cases), 3 norovirus (7 cases), 2 *Salmonella* (4 cases), 1 *Campylobacter* (2 cases), and 1 *C. parvum* (3 cases)

# 5. Outbreak Case Reports

## Hepatitis A outbreak in Christchurch

Following the notification of a case of hepatitis A on 27 December 2005 to Community & Public Health, Canterbury District Health Board, further cases were notified in the first 10 days of January 2006. Initial impression was that there were 10 cases in three unrelated clusters. A food source (possibly berries) was at first suspected. A standard questionnaire and follow up procedures were used to this point.

A Hepatitis Outbreak Group was established on 10 January 2006 to oversee the management of the outbreak. Media publicity and notification of other interested parties was made at this stage. A case of hepatitis notified in Wellington on 11 January 2006 was found to have had Christchurch associations.

However, as the information on the cases accumulated it became apparent on 12 January 2006, that there was a common feature in the form of links to children who attended a day care centre. A further link with the same day care centre was established between a family of three cases that were notified in early December 2005 as having developed Hepatitis A after returning from Fiji.

A decision was made to use Hepatitis A vaccine as a control measure. Accordingly arrangements were made to hold a vaccination clinic at the childcare centre involved. It was decided to offer immunisation to all children and the families and staff involved at the centre and others who could be identified as close contacts. To this end the majority of health protection, health promotion and administrative staff who had returned to work after the Christmas break were involved in following up cases, identifying contacts and providing information to individuals and families involved.

On 17 January 2006, 352 doses of vaccine were administered at the centre. A further 174 doses were administered subsequently to those who were unable to be present at the initial clinic. The second dose of the vaccine has been arranged for those who accepted the first.

As a consequence of the follow up further cases were identified. From 1 December 2005 to 1 February 2006, a total of 32 cases of hepatitis A were notified in Canterbury, all but two of which had some association with the childcare centre or cases associated with it. This included 12 children and one staff member from the centre and 3 children and 14 adult family members or contacts of the children. Additional cases identified in Taranaki and the Wellington region were also associated with the centre. No further cases associated with the outbreak have been notified since 1 February 2006, which would indicate that the preventive measures put in place have been effective. Although the childcare centre was at the centre of the outbreak there were clearly a number of situations where inadequate hygiene in other settings contributed to the transmission.

Follow up and control measures were complicated by the fact that pre-school children were either asymptomatic or showed only vague symptoms. This meant that it was not possible either to identify exclusion periods using the normal criteria or to be sure whether the administration of immunoglobulin was timely – hence the choice to use vaccine.

The outbreak raises the question as to whether the risk of hepatitis A warrants the immunisation of all childcare workers against the disease. This has been recommended in this setting and overseas experience would suggest that wider usage could be warranted.

The heavy usage of staff in this outbreak indicates that the workload in outbreak or pandemic situations should not be underestimated. The ease with which the disease spread in a number of social settings also indicates that the importance of hand washing hygiene in the community is grossly underestimated and this does not bode well for a pandemic situation.

**Reported by Melvin Brieseman, Medical Officer of Health, Community & Public Health, Canterbury District Health Board**

## Suspected foodborne illness outbreak

On 27 February 2006, a suspected foodborne illness outbreak was notified to the Hawke's Bay Public Health Unit involving seven members of a party of 14 from three different households. A case was defined as a person who had gastro-intestinal illness with similar symptoms who attended a common event. The common event was consumption of a meal at the same restaurant in Napier, on the evening of 9 February 2006. The notification was made by one of the cases. The seven cases were three teenagers 15, 16 and 18 (from the same household), one 20 year old (from the same household but lived off the property), a 50 year old (who stays on his own), and a 51 and 56 year old couple from the same household. The cases were interviewed by telephone on 28 and 29 February 2006 and the ESR case report forms were completed. No faecal or food samples were taken because of the time delay of the notification, so it has not been possible to determine the cause of their illness. Five of the seven cases started with the symptoms of nausea and vomiting on Saturday 11 February 2006, followed by diarrhoea, stomach cramps, fever and lethargy. It was hypothesised, that food from the restaurant (namely Chicken, Seafood, and Pavlova) caused vomiting, diarrhoea, fever, abdominal cramps and lethargy in seven cases within 31 to 64 hours after consumption.

The owner and chef of the restaurant were interviewed on the telephone. The receipt, storage, preparation and serving of the above three foods appears to be carried out in a safe and hygienic manner. A verbal report from Napier City Council's Environmental Health Officer, was that the premises is kept in good condition.

The incubation period and the symptoms are an indication that the food from the restaurant (namely Chicken, Seafood, and Pavlova) may have caused the illness. No other reported or confirmed cases were notified and no other complaints about the food or the premise have been received. The cause of this outbreak therefore cannot be confirmed.

**Reported by Gray Bamber, Health Protection Officer, Hawke's Bay Public Health Unit, Hawke's Bay District Health Board**

# 6. Pathogen Surveillance

**Unless otherwise reported, pathogen surveillance covers the January - March 2006 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

- 497 human and 343 non-human isolates were submitted to ERL (2005: 401 and 153 respectively)
- 5 household cases *S.* Typhimurium phage type 160, Canterbury
- 11 cases *S.* Typhi which includes 3 contacts and 1 household cluster of 2 cases, the latter recent travel to India
- Uncommon phage type *S.* Typhimurium 195 isolated from stitchbirds on Tiri Tiri Matangi Island

### VTEC/STEC (ERL)

- 32 laboratory confirmed cases of *E. coli* O157: H7 (2005, n=19) includes 1 family cluster of 4 and 1 of 2 cases
- 1 laboratory confirmed case of *E. coli* O113: H21 presenting with HUS
- 1 laboratory confirmed case of *E. coli* O91: H21 presenting with bloody diarrhoea

*continued...*

### Other (ERL)

- 2 non-toxigenic strains of *E. coli* O113: H6 isolated from blood cultures from 2 neonates

### Norovirus (Norovirus Reference Laboratory)

- 55 confirmed norovirus outbreaks were reported to the NRL
- 31 (56.4%) outbreaks occurred in March, 17 occurred in the Auckland district but outbreaks were also reported from most health districts
- 34 (61.8%) outbreaks occurred in rest homes (25) and hospitals (9). Catered settings featured in 7 outbreaks, and home settings were identified in 5 outbreaks
- 2 outbreaks occurred in school settings, 1 in a military base and 1 at a camping site during the summer holiday break
- 1 outbreak of 9 cases was associated with consumption of imported Korean oysters. Norovirus was identified in the oysters as well as in specimens from 2 cases. Genotyping of norovirus strains from oysters and cases is in progress
- Genotyping is not complete for this quarter but to date the trend indicates that the GII/1,4,8 group is once again becoming predominant in 2006. This group of norovirus strains was responsible for a large increase in norovirus outbreaks in New Zealand during 2004

## LEGIONELLOSIS AND ENVIRONMENTAL LEGIONELLA

- 17 legionellosis cases were laboratory identified in the first quarter
- 15 lab-proven cases have been notified, with a further 4 notified cases not being laboratory-proven
- 1 outbreak was identified in this quarter, associated with *L. pneumophila* serogroup 1 contamination of roof catchment rain water tanks, with 4 lab-proven cases
- 3 deaths were associated with legionellosis
- the remaining 13 lab-proven cases were sporadic CAP cases
- of the 17 cases identified, 11 fitted the confirmed case definition and 6 fitted the probable case definition
- the 11 confirmed cases demonstrated either antibody titres >512 on two or more occasions (6 cases), or at least a four-fold rise in antibody titre by the legionella IFAT (3 cases), or a rising titre to at least 512 (1 cases), or culture-positive (1 case)
- the 6 probable cases demonstrated either stable antibody titres of 512 (1 case), or a single antibody titre of $\geq$512 (5 cases)
- *L. pneumophila* serogroup 1 was identified as the causative agent in 10 cases
- *L. pneumophila* serogroup 4 was identified as the causative agent in 1 case
- *L. longbeachae* was identified in a further 5 cases
- 1 infection was caused by *L. micdadei*
- *L. pneumophila* serogroup 1 was isolated from 4 different domestic water supplies associated with cases of legionellosis
- Legionellae isolated from industrial water systems including cooling towers included *L. pneumophila* serogroups 1, 5, 6, & 8, *L. anisa* and *L. feeleii* serogroups 1 & 2

## RESPIRATORY VIRUSES

### Influenza Virus

- 7 influenza viruses were reported from laboratory-based surveillance (2005, 6)
- 5 were influenza A, 1 as A/California/7/2004 (H3N2), 1 as A/New Caledonia/20/99 (H1N1) and 3 yet to be subtyped

### Respiratory Syncytial Virus, Rhinovirus & Parainfluenza Virus

- 7 cases of respiratory syncytial virus were reported (2005, 10)
- 11 rhinoviruses were reported (2005, 10)
- 1 parainfluenza type 3 virus was reported (2005, 6)

## ADENOVIRUSES AND ENTEROVIRUSES

### Adenoviruses

- 47 adenoviruses were reported (2005, 74)
- Adenovirus type 3 and type 4 were the predominant serotypes
- 45 adenoviruses were serotyped as adenovirus type 1 (3), type 2 (1), type 3 (13), type 4 (13), type 5 (2), type 8 (4), type 11 (1), type 15 (1), type 29 (4), type 37 (1) and untypable (2)

### Enteroviruses

- 35 enteroviruses were reported (2005, 60)
- Coxsackie B5 was the predominant serotype
- 14 enteroviruses were serotyped as Coxsackie B1 (1), Coxsackie B2 (2), Coxsackie B3 (1), Coxsackie B5 (3), Coxsackie A4 (1), Coxsackie A10 (1), Coxsackie A12 (1), Echovirus 25 (1), Echovirus 27 (1), Enterovirus type 71 (1) and untypable (1)

## SPECIAL BACTERIOLOGY

### *Listeria monocytogenes*

- 7 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)
- 6 cases were in adults with underlying illnesses and/or were elderly
- 1 case was 4 year old female, no risk factors identified

### *Corynebacterium diphtheriae*

- 13 isolates of *Corynebacterium diphtheriae* was received for toxigenicity testing, typing and surveillance purposes
- 2 isolates were var. *gravis* strains from blood of endocarditis patients, males aged 26 years and 62 years from Hamilton and Auckland respectively
- 1 isolate was var. *gravis* strain from ear discharge of 10 year old male from Christchurch
- 10 isolates were var. *mitis* strains from cutaneous sources, patients were aged between 4 and 65 years and came from Auckland (7), Christchurch (2) and Wellington (1)
- all isolates were non-toxigenic by PCR examination for the toxin gene

New Zealand Public Health Surveillance Report is produced quarterly by ESR for the Ministry of Health and may be downloaded in PDF format from www.surv.esr.cri.nz



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

Please send contributions to: Scientific Editor, New Zealand Public Health Surveillance Report, ESR, PO Box 50-348, Porirua, Wellington, New Zealand. Phone: (04) 914 0700; Fax (04) 914 0770; Email: survqueries@esr.cri.nz