Volume 5 Issue 3 ISSN 1176-2888

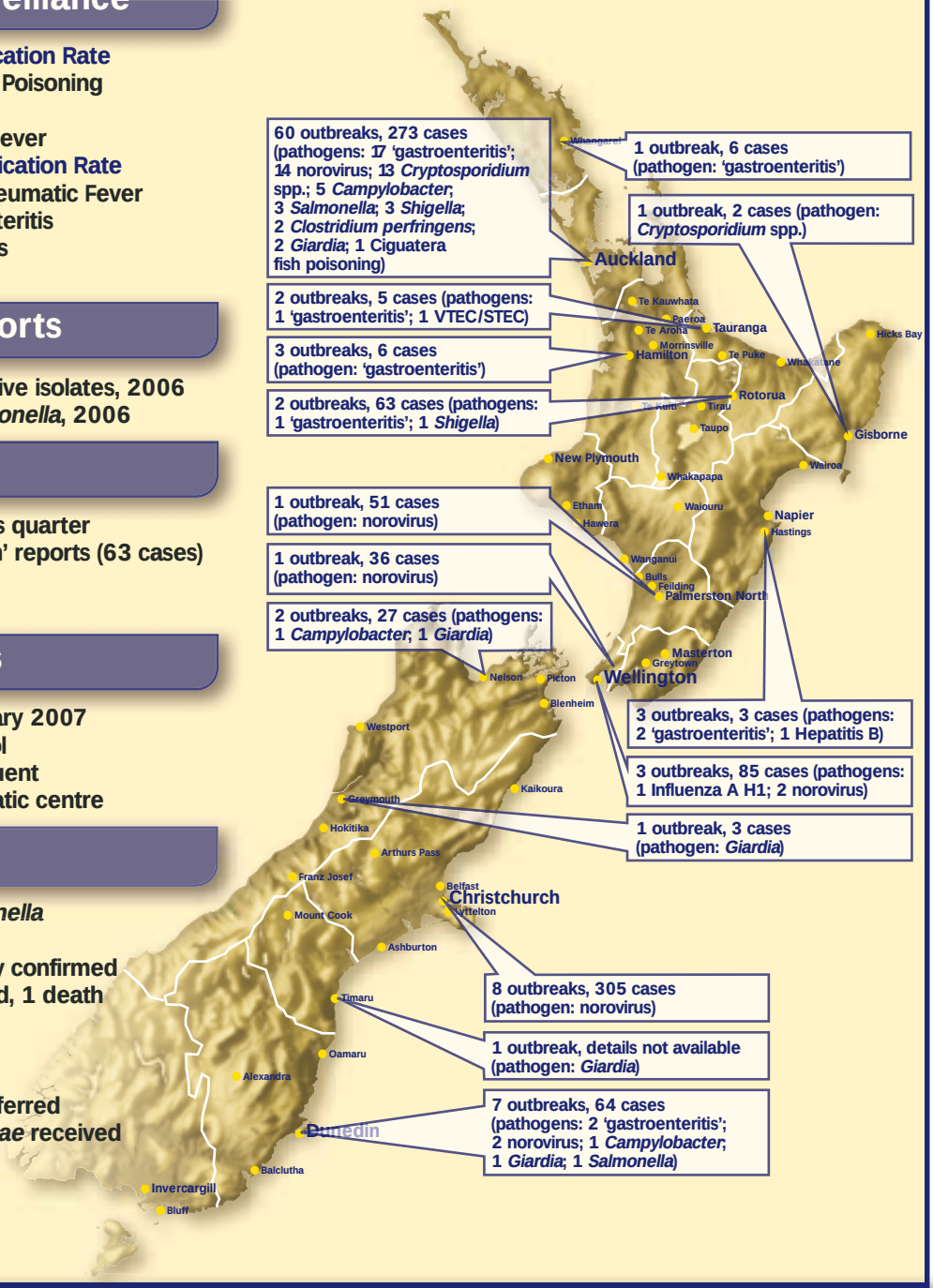
New Zealand Public Health Surveillance Report

September 2007: Covering April - June 2007

Contents & Highlights

This Quarter's Outbreaks

Notification and outbreak data in this issue are drawn from the April – June 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 9 July 2007.



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

1. Editorial

The National Centre for Biosecurity and Infectious Disease - Wallaceville

A collaborative effort of agencies specialising in infectious animal and human disease and in biosecurity, The National Centre for Biosecurity and Infectious Disease - Wallaceville (NCBID - Wallaceville) will significantly enhance New Zealand's capability to respond rapidly to infectious disease and other aberrant events. The Wallaceville site in the Upper Hutt Valley, Wellington region, has a proud tradition originating in 1905 when the Chief Veterinary Officer, John Anderson Gilruth, moved there with his staff. The division had previously been housed in (or rather on) Parliament Buildings and, latterly, at the Public Health Laboratory courtesy of the Medical Officer of Health[1]. Gilruth himself also worked part-time for the then Department of Public Health. The development of NCBID - Wallaceville therefore returns to a past close collaboration between animal and human health laboratories. This is a deliberate, strategic move in an era when emerging threats to human health involve viruses or bacteria of animal origin that have the capacity already to infect humans or can evolve to cause serious human illness. Zoonotic organisms account for many of the infectious diseases introduced to New Zealand over the past 20 years.

The Centre comprises the Ministry of Agriculture and Forestry's Wallaceville Investigation and Diagnostic Centre, the AgResearch Infectious Disease Diagnostic team, Agriquality's Animal Diagnostics unit, and both laboratory scientists and aberrant event investigation specialists from ESR. They are supported by the Ministry of Agriculture and Forestry, the Ministry of Health, the Ministry of Research, Science and Technology through various funding mechanisms, and commercial and other activities consistent with the goals of NCBID - Wallaceville.

NCBID's primary role is to provide a centralised coordination and emergency response site for disease outbreaks, biosecurity issues, and also chemical and biological threats and events. For example, NCBID - Wallaceville would play an important role in any response to a pandemic influenza threat. It will enhance collaboration among animal and human health researchers, investigators and diagnosticians, assist in the identification of research and development needs, and enhance the application of research results to achieve applied biosecurity and infectious disease control outcomes that help to protect New Zealand and New Zealanders from biosecurity risks[2].

NCBID-Wallaceville includes the highest biocontainment laboratory in New Zealand (physical containment 3+ or PC3+) which is necessary for the investigation of suspected cases of diseases such as SARS or foot and mouth. ESR has also commenced construction of new PC 2 laboratories to support access to the PC3+ facility and office space for outbreak investigators and scientists. This will eventually be joined by a seminar facility.

Collaboration between animal and human health scientists is one important benefit from co-location. The skills and equipment required for the laboratory diagnosis of human and animal infectious diseases are similar. Collaboration between epidemiologists and laboratory scientists is another key benefit. A number of joint and independent research initiatives are already well under way with on-site and other partners. Staff of NCBID - Wallaceville organisations, along with other partners, are currently examining the microbiological or epidemiological profiles of diseases such as Salmonella, Avian Influenza, vector borne diseases e.g. Ross River Virus disease and Murine Typhus, bovine tuberculosis, and Johne's disease of deer and cattle. NCBID - Wallaceville partners are also actively exploring ways in which they will be able to support each other in emergency responses.

Links with the health sector are essential for both the operational response to human infectious disease threats and for the conduct of ongoing research to support responses. The NCBID - Wallaceville partners are seeking health sector and research collaborators to work with as the Centre develops. Funding from Research and Education Advanced Network New Zealand (REANNZ) will support the development of a research clinical microbiology laboratory network based on the Kiwi Advanced Research and Education Network (KAREN) which will assist with this. Enhanced links with those involved in aberrant disease investigations will be supported by ongoing skill development and post-graduate education programmes as well as the exchange of timely information on aberrant disease events.

NCBID – Wallaceville is an exciting new collaboration essential to the development of science to strengthen New Zealand's capability to respond to aberrant events.

1 Tenquist JD. 1990. *Wallaceville Veterinary Laboratory – An anecdotal history.* MAF Technology.

2 NCBID Governance Committee 2006. *NCBID- Wallaceville Vision.* NCBID Management Committee.

Virginia Hope, ESR Programme Manager for NCBID

2. Notifiable Disease Surveillance

The following is a summary of disease notifications for the April – June quarter of 2007 and cumulative notifications and rates calculated for a 12-month period (July 2006 – June 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 9 July 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

Pertussis

- *Notifications*: 73 notifications in the quarter (2006, 261); 665 notifications over the last 12 months (2006, 1,859) giving a rate of 16.1 cases per 100,000 population (2006, 45.4); a statistically significant decrease
- *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (107 cases) and from the same quarter last year (261 cases)

Infectious Respiratory Diseases

Acute Rheumatic Fever

- *Notifications*: 17 notifications in the quarter (2006, 39); 78 notifications over the last 12 months (2006, 105) giving a rate of 1.9 cases per 100,000 population (2006, 2.6); a statistically significant decrease

• *Comments*: there has been a statistically significant quarterly decrease from the same quarter last year (39 cases); notifications were distributed by age as follows, 1 (1-4 years); 6 (5-9 years); 8 (10-14 years) and 2 (15-19 years); all notifications were initial attacks of rheumatic fever

Enteric Infections

Campylobacteriosis

• *Notifications*: 2,427 notifications in the quarter (2006, 3,600); 14,989 notifications over the last 12 months (2006, 16,169) giving a rate of 362.1 cases per 100,000 population (2006, 394.5); a statistically significant decrease

• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (4,646 cases) and from the same quarter last year (3,600 cases)

Gastroenteritis

• *Notifications*: 120 notifications in the quarter (2006, 242); 663 notifications over the last 12 months (2006, 790) giving a rate of 16.0 cases per 100,000 population (2006, 19.3); a statistically significant decrease

• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (147 cases) and from the same quarter last year (242 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*: 322 notifications in the quarter (2006, 322); 1,259 notifications over the last 12 months (2006, 1,451) giving a rate of 30.4 cases per 100,000 population (2006, 35.4); a statistically significant decrease

• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (374 cases)

Shigellosis

• *Notifications*: 41 notifications in the quarter (2006, 16); 112 notifications over the last 12 months (2006, 176) giving a rate of 2.7 cases per 100,000 population (2006, 4.3); a statistically significant decrease

• *Comments*: there has been a statistically significant quarterly increase from the same quarter last year (16 cases)

Typhoid

• *Notifications*: 11 notifications in the quarter (2006, 5); 63 notifications over the last 12 months (2006, 22) giving a rate of 1.5 cases per 100,000 population (2006, 0.5); a statistically significant increase

• *Comments*: there has been a statistically significant quarterly decrease from the previous quarter (23 cases)

Environmental Exposures and Infections

Chemical Poisoning

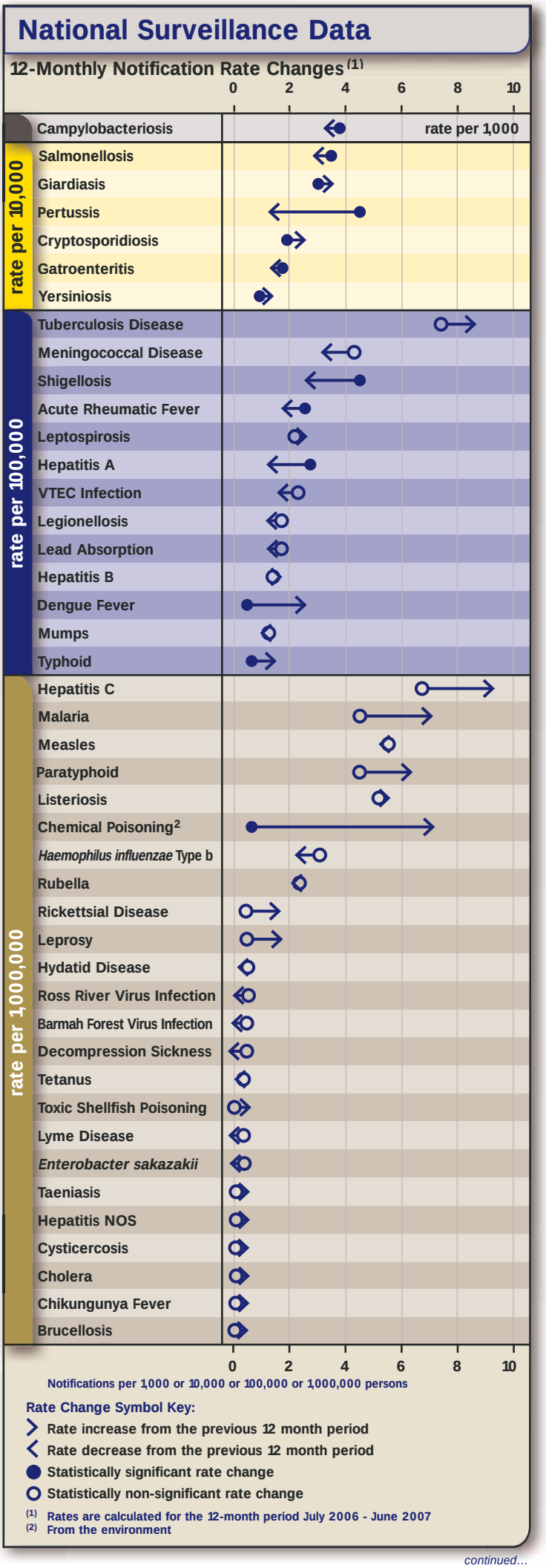
• *Notifications*: 3 notifications in the quarter (2006, 2); 30 notifications over the last 12 months (2006, 4) giving a rate of 0.7 cases per 100,000 population (2005, 0.1); a statistically significant increase

Cryptosporidiosis

• *Notifications*: 215 notifications in the quarter (2006, 86); 985 notifications over the last 12 months (2006, 816) giving a rate of 23.8 cases per 100,000 population (2006, 19.9); a statistically significant increase

• *Comments*: there has been a statistically significant increase from the same quarter last year (86 cases)

National Surveillance Data



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Giardiasis

- *Notifications*: 366 notifications in the quarter (2006, 319); 1,352 notifications over the last 12 months (2006, 1,233) giving a rate of 32.7 cases per 100,000 population (2006, 30.1); a statistically significant increase

Hepatitis A

- *Notifications*: 8 notifications in the quarter (2006, 26); 59 notifications over the last 12 months (2006, 120) giving a rate of 1.4 cases per 100,000 population (2006, 2.9); a statistically significant decrease
- *Comments*: there has been a statistically significant quarterly decrease from the same quarter last year (26 cases); notified cases were aged between 14 and 85 years, with 1 case under the age of 16 years

Yersiniosis

- *Notifications*: 114 notifications in the quarter (2006, 107); 523 notifications over the last 12 months (2006, 438) giving a rate of 12.6 cases per 100,000 population (2006, 10.7); a statistically significant increase

New, Exotic and Imported Infections

Dengue Fever

- *Notifications*: 32 notifications in the quarter (2006, 5); 105 notifications over the last 12 months (2006, 16) giving a rate of 2.5 cases per 100,000 population (2006, 0.4); a statistically significant increase
- *Comments*: there has been a statistically significant decrease from the previous quarter (63 cases) and a statistically significant increase from the same quarter last year (5 cases); 28 notifications were laboratory confirmed, 1 case is listed as probable and 3 cases are under investigation; 31 cases were overseas during the incubation period and the travel history of 1 case is unknown. Places visited were Malaysia (1), Indonesia (2), East Timor (1), Rarotonga (2), Cook Islands (17), Samoa (4), Fiji (1), Tahiti (2) and French Polynesia (1)

3. Other Surveillance Reports

Antimicrobial susceptibility among invasive isolates, 2006

Streptococcus pneumoniae, *Neisseria meningitidis* and *Haemophilus influenzae* isolated from normally sterile sites are routinely referred to ESR for the national laboratory-based surveillance of invasive disease due to these organisms. The antimicrobial susceptibility of all viable invasive isolates of these three organisms referred in 2006 was tested. More detailed information is available on www.surv.esr.cri.nz/antimicrobial/antimicrobial_resistance.php

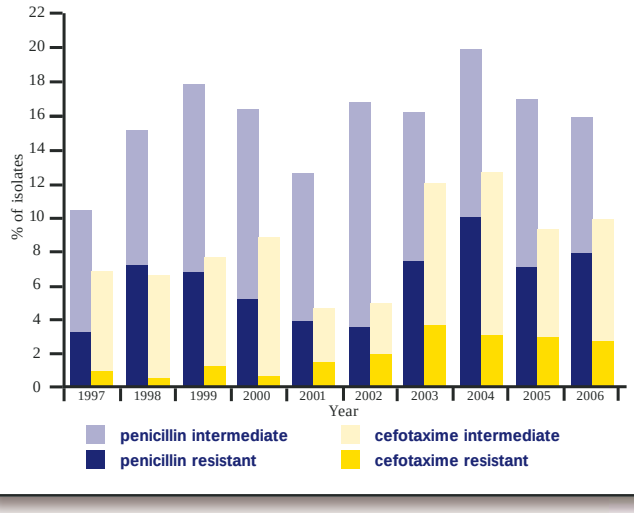
Streptococcus pneumoniae

The antimicrobial susceptibility of 522 invasive *S. pneumoniae* isolates was tested in 2006. Eight percent were penicillin resistant (MIC ≥2 mg/L) and another 8% had intermediate penicillin resistance (MIC 0.12-1 mg/L). While penicillin resistance has been quite variable over the last 10 years, the prevalence has remained between 7-10% since 2003 which is similar to the prevalence in the late 1990s (Figure 1).

Applying the Clinical and Laboratory Standards Institute (CLSI) meningitis interpretive standards, 3% of the invasive pneumococci in 2006 were cefotaxime resistant (MIC ≥2 mg/L) and 7% had intermediate cefotaxime resistance (MIC 1 mg/L). Applying the non-meningitis interpretive standards, 1% were cefotaxime resistant (MIC ≥4 mg/L) and 2% had intermediate cefotaxime resistance (MIC 2 mg/L). There has been a trend of increasing resistance to third-generation cephalosporins in recent years, although during the last four years there have been no further increases (Figure 1). All isolates were susceptible to vancomycin and moxifloxacin.

In 2006, capsular antigen types 19F, 14, 9V, 6B and 23F accounted for all of the penicillin-resistant invasive pneumococci, and serotypes 19F, 14 and 9V accounted for all the cefotaxime-resistant isolates. These serotypes are included in the 7-valent pneumococcal conjugate vaccine which will be added to the national immunisation schedule in 2008.

Figure 1. Penicillin and cefotaxime* resistance among invasive *S. pneumoniae*, 1997-2006



*cefotaxime susceptibility based on the CLSI meningitis interpretive standards

Neisseria meningitidis

The antimicrobial susceptibility of 85 meningococcal isolates from cases of invasive disease in 2006 was tested. There was no resistance to penicillin, ceftriaxone, rifampicin or ciprofloxacin. Twelve percent of isolates had reduced penicillin susceptibility, with MICs of 0.12-0.5 mg/L. Isolates with reduced penicillin susceptibility have been increasing over the last 10 years. However, meningococcal infections due to such isolates are still treatable with penicillin.

Haemophilus influenzae

The antimicrobial susceptibility of 52 invasive *H. influenzae* isolates was tested in 2006. Eight of the 52 isolates were serotype b. Twelve percent of isolates produced ß-lactamase and another 21% were ampicillin resistant but ß-lactamase negative. There was no resistance to cefotaxime or rifampicin.

Antimicrobial susceptibility among *Salmonella*, 2006

Each year a representative sample of non-typhoidal *Salmonella*, chosen from isolates routinely referred to ESR for serotyping, is tested for antimicrobial susceptibility. In addition, all isolates of *S.* Typhi, *S.* Paratyphi A and *S.* Paratyphi B are tested. More detailed information is available on www.surv.esr.cri.nz/PDF_surveillance/Antimicrobial/SAL_2006.pdf

Antimicrobial resistance among *Salmonella* remains relatively uncommon in New Zealand. Among the 574 non-typhoidal *Salmonella* tested in 2006, 93% were fully susceptible to all 12 antimicrobials tested. Two percent of isolates were ampicillin resistant and 1% were co-trimoxazole resistant. All isolates were susceptible to third-generation cephalosporins, ciprofloxacin and co-amoxiclav.

Although none of the 39 *S.* Typhi or 14 *S.* Paratyphi isolates tested were resistant to ciprofloxacin, 36% were resistant to nalidixic acid. Fluoroquinolone (ciprofloxacin)-susceptible strains of *Salmonella* that are resistant to the older-generation quinolone, nalidixic acid, may be associated with clinical failure or delayed response when fluoroquinolones are used to treat extra-intestinal salmonella infections. All typhoidal *Salmonella* were susceptible to third-generation cephalosporins.

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 (April – June 2007). Comparisons are made to the previous quarter (January – March 2007), and to the same quarter in the previous year (April – June 2006). Note that the outbreak data in this section are notified to ESR by the Public Health Services.

General

- 96 outbreaks notified in this quarter (929 cases)
- 76 are 'final' reports (866 cases); 20 are 'interim' reports (63 cases) that have yet to be finalised and closed

All following data pertain to final reports only.

- 11.4 cases on average per outbreak, compared with 10.5 cases per outbreak in the previous quarter (14.4 cases per outbreak in the same quarter of last year)
- 24 hospitalisations: norovirus (13 cases), *Salmonella* (5 cases), *Shigella* (4 cases), *Campylobacter* (1 case), and gastroenteritis (1 case)
- 1 death (norovirus)

Pathogens

- 23 norovirus outbreaks (638 cases) during this quarter
- 18 'gastroenteritis' outbreaks (98 cases)
- 13 *Cryptosporidium* spp. outbreaks (40 cases)
- 6 *Campylobacter* outbreaks (16 cases)
- 4 *Salmonella* outbreaks (10 cases)
- 4 *Shigella* outbreaks (19 cases)
- 3 *Giardia* outbreaks (29 cases)
- 2 *Clostridium perfringens* outbreaks (9 cases)
- 1 Ciguatera fish poisoning (2 cases)
- 1 Hepatitis B outbreak (3 cases)
- 1 VTEC/STEC 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.

- 36 person-to-person, from (non-sexual) contact with an infected person (including droplets): 15 norovirus (584 cases), 9 *Cryptosporidium* spp. (31 cases), 6 gastroenteritis (70 cases), 3 *Giardia* (29 cases), and 3 *Shigella* (15 cases)
- 16 foodborne, from consumption of contaminated food or drink (excluding water): 5 *Campylobacter* (14 cases), 4 gastroenteritis (15 cases), 2 *C. perfringens* (9 cases), 2 norovirus (124 cases), 2 *Salmonella* (6 cases), and 1 VTEC/STEC (2 cases)
- 13 environmental, from contact with an environmental source (e.g. swimming): 8 norovirus (272 cases), 3 *Cryptosporidium* spp. (8 cases), 1 VTEC/STEC (2 cases), and 1 *Giardia* (3 cases)
- 2 sexual, from sexual contact with an infected person: 1 *Giardia* (3 cases), and 1 Hepatitis B (3 cases)
- 2 waterborne, from consumption of contaminated drinking water: 1 *Campylobacter* (3 cases), and 1 *Cryptosporidium* spp. (2 cases)
- 1 other mode of transmission: 1 norovirus (via fomites) (29 cases)
- 23 mode of transmission unknown: 9 gastroenteritis (19 cases), 7 norovirus (18 cases), 2 *Cryptosporidium* spp. (4 cases), 2 *Salmonella* (4 cases), 1 *Campylobacter* (2 cases), 1 Ciguatera fish poisoning (2 cases), and 1 *Shigella* (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.

- 21 home: 7 *Cryptosporidium* spp. (16 cases), 3 gastroenteritis (6 cases), 3 *Giardia* (29 cases), 3 *Shigella* (15 cases), 2 *Campylobacter* (7 cases), 1 VTEC/STEC (2 cases), 1 norovirus (3 cases), and 1 *Salmonella* (4 cases)
- 11 rest home: 10 norovirus (359 cases) and 1 gastroenteritis (3 cases)
- 9 café: 4 gastroenteritis (15 cases), 2 *Campylobacter* (5 cases), 2 *C. perfringens* (9 cases), and 1 norovirus (36 cases)
- 5 hospital (continuing care): 4 norovirus (152 cases) and 1 gastroenteritis (3 cases)
- 3 takeaway: 1 *Campylobacter* (2 cases), 1 gastroenteritis (2 cases), and 1 *Salmonella* (2 cases)
- 2 caterers: 2 norovirus (124 cases)
- 2 childcare: 1 *Cryptosporidium* spp. (13 cases), and 1 *Giardia* (24 cases)
- 2 swimming/spa pool: 2 *Cryptosporidium* spp. (5 cases)
- 1 hospital (acute care): norovirus (10 cases)
- 1 prison: gastroenteritis (55 cases)
- 1 camp: norovirus (60 cases)
- 1 workplace: *Giardia* (3 cases)
- 1 supermarket: *Campylobacter* (4 cases)
- 4 'other setting': 2 *Salmonella* (4 cases), 1 *Cryptosporidium* spp. (2 cases), and 1 *Shigella* (4 cases)
- 19 outbreaks with no setting selected: 8 gastroenteritis (17 cases), 6 norovirus (15 cases), 2 *Cryptosporidium* spp. (4 cases), 1 *Campylobacter* (2 cases), 1 Ciguatera fish poisoning (2 cases), and 1 Hepatitis B (3 cases)

5. Outbreak Case Reports

Typhoid fever outbreak in Porirua, January 2007

Three cases of confirmed *Salmonella* Typhi are described from Porirua that were notified to Regional Public Health (RPH) between 31 January and 12 February 2007.

Case 1, a female student, presented to an accident and medical (A&M) clinic on 30 January with a 10 day history of fever, sore throat and headache, followed by diarrhoea and vomiting. She was quite unwell and dehydrated and so was hospitalised until 5 February. Her sister, Case 2, developed similar but milder symptoms one day earlier on 19 January but completely recovered without medical consultation/treatment.

Case 3 was a male in his mid-50s with no connections to the two sisters. His onset was 17 January with initial symptoms of headaches, myalgia, fever, nausea and vomiting. He was seen by a GP and at an A&M clinic on several occasions. No diagnosis was made but he was treated with various antibiotics. Following two seizures he was admitted to Intensive Care at Wellington Hospital. His typhoid fever was later confirmed by faecal specimen culture. Case history could initially only be obtained from his family.

All three cases were infected by the same type – *Salmonella* Typhi DT E1A. None of the cases had travelled overseas or were believed initially to have been in contact with anyone who had travelled overseas in their exposure periods. Some food premises were looked into for the two sisters, particularly a bakery where high-risk foods had been purchased. However, staff at the bakery had not travelled overseas, none had been recently recruited, and all had been well.

The connection between the three cases, and the likely risk factor, was only made when Case 3 indicated that he had been visited by a New Zealand-based Samoan representative-level sportsperson who had just returned from an overseas sports tour via Samoa. His name was then recalled as a household contact of Cases 1 and 2 - he is a cousin to the sisters.

The sportsperson stated he had had some diarrhoea starting on 7 January, following his return from a 2.5 week stay in Samoa on 5 January. There was a typhoid fever epidemic in Samoa at the time of his stay, but he was not aware of having contact with anyone with typhoid fever-like symptoms. He provided a faecal specimen that tested negative for *S*. Typhi.

He had brought food back from Samoa that had been purchased from Apia main market. This was:

(1) Palusami, umu-cooked packs of taro in coconut milk covered in taro leaves, and wrapped in aluminium foil. This was believed to have been brought back un-refrigerated.

(2) Banana chips in small, unlabelled plastic bags.

The sportsperson had given some of this food to the families of Cases 1 and 2, and Case 3 around 5 January. Case 3 had eaten the Palusami but Cases 1 and 2 only ate the banana chips. It is likely that other persons in both families ate one or both food items, but this could not be ascertained accurately. Two leftover packages of banana chips were analysed by ESR but no *S*. Typhi was detected.

These imported food items were the most likely source of infection. It is likely that food items were sporadically contaminated. Also there may have been cross-contamination of food items when eaten in the two households.

No secondary cases occurred. There was also a case of *S*. Typhi DT E1A in MidCentral DHB at the same time, but no connection could be established with the Porirua outbreak.

There was concern from Auckland Regional Public Health Service (where an outbreak unconnected to the Porirua one occurred), as well as RPH, about the legal importation into New Zealand of traditional foods from Samoa, and the risk of their being contaminated with pathogens such as *S*. Typhi.

Discussion between RPH and the New Zealand Food Safety Authority (NZFSA) resulted in the design of an educational brochure "Taking food between New Zealand and the Pacific Islands". This outlines food safety tips, largely to ensure food is chilled in transit. There is also information on the need to declare umu packs on arrival in New Zealand under biosecurity laws. The brochure has been translated into Samoan and Tongan. It will be distributed shortly via public health units, airports, and airlines and will be placed on the NZFSA website. There is also information on bringing umu packs into New Zealand in NZFSA's publication "Umu Pasifika, Food Safety for Pacific Peoples".[1]

1 www.nzfsa.govt.nz/consumers/food-safety-topics/umu-pasifika/scinzfsaumubook.pdf

Reported by Quentin Ruscoe, Health Protection Officer, Disease Investigation, Regional Public Health

Viral gastroenteritis outbreak in a school

The Northland Medical Officer of Health was notified on 30 May 2007 of a possible outbreak when a large number of pupils at a small country school called in sick with a gastrointestinal illness. Thirteen out of the 54 pupils and a teacher at the school had developed vomiting and diarrhoea on the 29 May. It was decided to investigate because the date of onset suggested a common source outbreak.

The local health protection officer (HPO) and school public health nurse (PHN) obtained the names of sick children and carried out interviews. The parents of children were asked if their children could supply faecal specimens for microbiological analysis. The HPO visited the school and inspected the water supply and the wastewater disposal system. It was noted that the school swimming pool had been closed for 2-3 months. Children brought their own lunches to school and there had not been any common meals prior to the outbreak.

Ultimately, the parents of 17 sick children were interviewed. The median age of the children was eight years. The most common symptoms of illness were stomach cramps (100%), vomiting (94%), and diarrhoea (59%). The median duration of illness was 24 hours with a range of 10 to 96 hours. Two of the children consulted by a doctor were clinically diagnosed with norovirus infection.

One of the children had developed vomiting and diarrhoea at home on the evening of 27 May 2007. She had come to school on the morning of the 28 May and according to her teacher looked unwell and smelt of vomit. Eight children in her class, four children from other classrooms and the teacher of the index case also became unwell with vomiting and diarrhoea during the day and evening of the 29 May. A further seven children from the school were unwell over the next five days. Ten household contacts also became unwell during this time period.

The water supply to the school was roof-collected water with UV treatment and filtration by a 1-micron filter. The treatment equipment was serviced yearly and the most recent water test, carried out on 23 March 2007, was negative for *Escherichia coli* and total coliforms. The wastewater disposal system was a collection tank which was pumped to a septic tank with a disposal field. At the time of inspection the septic tank and pump had no signs of spills or leaks, the vent pipes were well contained and the disposal field was located well outside the school boundary. It was noted that some of the hand washing basins in classrooms were not supplied with soap and towels. There were no hot water connections to the basins in the children's toilets.

The symptoms, duration and severity of illness were consistent with gastroenteritis caused by norovirus. Confirmed outbreaks of norovirus were known to have occurred in two rest homes and a hospital in the Northland area during May. The distribution of the cases suggest that the index case brought the illness to the school on the 28 May and the resulting cases occurred through person-to-person spread or contact with contaminated surfaces at the school and in the homes of cases. The infectiousness of norovirus is well described and hospitals with sophisticated infection control procedures can have difficulty in controlling outbreaks.[1]

The PHN and HPO supplied the school with pamphlets about the disease and provided advice about exclusion of sick children and staff, how staff could protect themselves from the disease and recommendations about cleaning and disinfection of contaminated areas. Advice on how to prevent further spread included:

(1) Sick children and staff should be immediately sent home and not return until at least 48 hours after their last diarrhoea or vomit.

(2) Hands should be washed for 20 seconds in soap and water and dried thoroughly with a paper towel for 20 seconds.

(3) Staff cleaning up vomit or diarrhoea should have access to appropriate personal protective equipment.

(4) Toilet areas and surfaces touched by hands should be cleaned by a 0.1% bleach solution.

(5) Bottles of hand sanitiser should be strategically placed around the school in classrooms and in the staff room.

In addition the school was advised to increase its drinking water testing frequency to monthly and to update the contact details for the caregivers of its students.

1 Lynn S, Toop J, Hanger C and Miller N. 2004. Norovirus outbreaks in a hospital setting: the role of infection control. NZMJ 117(1189). URL: www.nzma.org.nz/journal/117-1189/771/

Reported by Natasha Thompson, Health Protection Officer, Phillippa van der Pol, Public Health Nurse, Dr Jonathan Jarman, Medical Officer of Health, Northland District Health Board

Cryptosporidiosis outbreak and subsequent presumed chlorine poisoning at an aquatic centre

On 24 November 2006, the Auckland Regional Public Health Service (ARPHS) identified an outbreak of cryptosporidiosis associated with an aquatic centre after two confirmed cryptosporidiosis cases reported recent exposure to the pools. A total of 23 cases of cryptosporidiosis were eventually associated with the centre with onset dates between September and December 2006, of which 10 (43%) were probable cases and 13 (57%) were confirmed on stool testing.

Two visits to the centre were undertaken by ARPHS staff, and pool management processes were reviewed. ARPHS recommended improvements to pool management, including improved faecal incident response procedures. As ongoing cases were being reported, it was agreed with the pool operators that pool decontamination be undertaken using chlorine dioxide. Initial decontamination was carried out on 8 December. Only one case of cryptosporidiosis was subsequently notified to ARPHS that could have been due to exposure to pools at the centre following decontamination.

On 9 and 10 December, several people, mainly children, developed symptoms of respiratory and ocular irritation following exposure to pools at the same centre, including several members of a water polo team using the pools on 10 December. The initial presumption was that these people may have been suffering from chlorine poisoning related to the recent pool disinfection. By the time ARPHS became aware of this incident on 11 December, the pools had been closed.

A probable case was defined as any person who swam at the centre during the period 9-10 December 2006 and subsequently experienced symptoms consistent with chlorine poisoning (ocular, nasal and respiratory irritation). Case finding was undertaken through calls to hospitals in the region. In total, 23 probable cases were identified with exposure to pools at the centre during this period and a clinically compatible illness. No cases were confirmed, since no environmental samples were available. The age range for cases was 7-20 years, and 11 cases (48%) were hospitalised, with no deaths. Shortness of breath was the most common symptom, occurring in 83% of cases, followed by cough (65%), wheeze (65%) and nausea/vomiting (35%).

ARPHS staff reviewed pool testing and treatment records for the period preceding the poisoning incident. All results were within the guideline values in the Pool Water Quality standards, NZS 5826:2000. Air quality sampling was undertaken for chlorine gas by an independent contractor on 11 and 12 December, before the pools reopened, and all results were below the reference exposure limit of 1ppm.

The cause of this poisoning incident remains uncertain; however, the symptoms experienced by cases were consistent with poisoning by a chlorine-related agent, with ocular, nasal and respiratory irritation. It is possible that an overdose of chlorine dioxide occurred during pool disinfection.

This incident illustrates both the potential for cryptosporidiosis outbreaks associated with swimming pools, and the potential risks associated with swimming pool decontamination. A previous swimming pool-associated cryptosporidiosis outbreak in the Hutt Valley region in 1998 resulted in 122 cases.[1]

Chlorine dioxide is a useful option for swimming pool *Cryptosporidium* disinfection, since *Cryptosporidium* is much more resistant to chlorine than chlorine dioxide.[2] Swimming pool-associated chlorine poisoning incidents have been reported in the past, including the centre itself[3] and a Wellington pool,[4] though we could find none related to chlorine dioxide.

While the Pool Water Quality standards, NZS 5826:2000, mention chlorine dioxide as an agent that may be used to inactivate *Cryptosporidium*,[2] they do not provide an explicit protocol for its use. Given the hazards associated with chlorine dioxide disinfection, such a protocol would be of benefit.

NZS 5826:2000 provides little information on air quality, though this is closely related to water quality. It has been said that 'the air above indoor swimming pools... needs to be assessed and managed as carefully as the water'.[5] Evidence is emerging that the chronic respiratory effects of chloramines in indoor swimming pools may be important.[5,6] Further guidance may be needed for the appropriate management of air quality for indoor swimming pools, especially regarding ventilation requirements.

1 Baker M, Russell N, Roseveare C, et al. 1998. Outbreak of cryptosporidiosis linked to Hutt Valley swimming pool. New Zealand Public Health Report 5(6): 41-5.

2 Standards New Zealand 2000. NZS 5826:2000 Pool Water Quality. Standards New Zealand, Wellington.

3 Waitakere City Council 2002. Media releases November 2002. [cited 11 Apr 2007]. Available from: www.waitakere.govt.nz/WhaHap/nm/mr/2002/nov02.asp

4 Ruscoe Q. 2007. Mass chlorine poisonings at a swimming pool. New Zealand Public Health Surveillance Report 5(1): 5.

5 Thickett KM, McCoach JS, Gerber JM, et al. 2002. Occupational asthma caused by chloramines in indoor swimming-pool air. *Eur Respir J.* 19(5): 827-832.

6 Bernard A, Carbonnelle S, Dumont X, Nickmilder M. 2007. Infant swimming practice, pulmonary epithelium integrity, and the risk of allergic and respiratory diseases later in childhood. *Pediatrics* 119(6): 1095-1103.

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6. Pathogen Surveillance

Unless otherwise reported, pathogen surveillance covers the April – June 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

- 329 human and 213 non-human isolates were confirmed (2006, 317 and 289 respectively)

VTEC/STEC (ERL)

- 20 isolates of O157 were laboratory confirmed (2006, 23)
- 3 human cases of O126:HNM, a recognised enteropathogenic strain (EPEC), were identified
- 2 were household contacts aged 11 months (male) and 14 months (female), third case unrelated
- EPEC are historically associated with outbreaks of infantile diarrhoea. They possess the *eae* gene and a virulence plasmid but do not produce the verocytotoxin

Other (ERL)

- *Vibrio cholera* 01 biotype El Tor subtype Inaba
- male, 76 years, Auckland, recent travel to India

Norovirus (Norovirus Reference Laboratory)

- 37 confirmed norovirus outbreaks of which 20 occurred in rest homes and hospital settings
- 14 outbreaks occurred in April, 12 in May and 11 in June

continued...

- 7 outbreaks were associated with catered food settings, including takeaway bars and restaurants, and 2 outbreaks occurred in school-related situations
- a large outbreak occurred at an international sports tournament in Christchurch and was associated with food-borne transmission following poor food-handling
- all outbreaks except one were caused by Genogroup II strains. One outbreak in a catered setting was caused by a Genogroup I norovirus
- 27/37 outbreak strains have been genotyped. The predominant genotype continues to be GII/4, accounting for 15/27 outbreaks, including 10 outbreaks in healthcare institutions. Other genotypes identified were GII/2, GII/6, GII/12 and GII/8. Six recombinant GII/3 – GII/4 norovirus strains were also identified

Legionellosis and Environmental Legionella

- 20 legionellosis cases were laboratory identified
- 1 death associated with legionellosis was reported
- 18 lab-proven cases have been notified, with a further 7 notified cases not being laboratory-proven
- all 20 lab-proven cases were sporadic community acquired pneumonia cases
- 14 fitted the confirmed case definition and 6 fitted the probable case definition
- the 14 confirmed cases demonstrated either antibody titres >512 on two or more occasions (1 case), or at least a four-fold rise in antibody titre by the legionella IFAT (7 cases), or a rising titre to at least 1024 (1 case), or were culture-positive (5 cases)
- the 6 probable cases demonstrated either stable antibody titres of 512 (2 cases), or a single antibody titre of >512 (4 cases)
- *L. pneumophila* serogroup 1 was identified as the causative agent in 7 cases
- *L. pneumophila* serogroup 12 was identified as the causative agent in 1 case
- *L. pneumophila* serogroup 13 was identified as the causative agent in 1 case
- *L. pneumophila* serogroup 1 & 12 serological cross-reaction showed in 2 cases
- *L. longbeachae* was identified in a further 5 cases
- *L. sainthelensi* was identified as the causative agent in 2 cases
- *L. gormanii* was identified as the causative agent in 1 case
- *L. jordanis* was identified as the causative agent in 1 case
- Legionellae isolated from industrial water systems including cooling towers included *L. pneumophila* serogroups 1, 5 & 8, *L. anisa* and *L. sainthelensi*
- Legionellae isolated from composts and soils included *L. bozemanii*, and *L. longbeachae* serogroups 1 and 2
- the high number of cases may be attributable to the seasonally warmer April - May period

Respiratory Viruses

Influenza Virus

- 93 influenza viruses were reported from sentinel and laboratory-based surveillance (2006, 175)
- 88 were identified as influenza A, 38 as A/New Caledonia/20/99 (H1N1) – like strains, 1 as A(H1N1) not-antigenically-subtyped, 3 as A/Wisconsin/67/2005 (H3N2) –like strains, 2 as A(H3N2) not-antigenically-subtyped, and 44 as A not-subtyped
- 5 were identified as influenza B, 1 as B/Shanghai/361/2002 – like strain, and 4 as B not-antigenically-typed

Respiratory Syncytial Virus, Rhinovirus & Parainfluenza Virus

- 38 cases of respiratory syncytial virus were reported (2006, 92)
- 6 rhinoviruses were reported (2006, 7)
- 11 parainfluenza viruses were reported (2006, 25). Among them, 9 were further typed as parainfluenza type 2, 2 as type 3

Adenoviruses and Enteroviruses

Adenoviruses

- 168 adenoviruses were reported (2006, 64)
- Adenovirus type 8 and type 3 were the predominant serotypes. The outbreak of adenovirus type 8 has continued since the first quarter (January to March 2007). For more details of this outbreak, please see the website: www.surv.esr.cri.nz/PDF_surveillance/NZPHSR/2007/NZPHSR2007June.pdf
- 146 adenoviruses were serotyped as adenovirus type 1 (1), type 2 (4), type 3 (26), type 4 (4), type 7 (1), type 8 (92), type 14 (1), type 15/29 (1), type 19 (1), type 37 (10), and untypable (5)

Enteroviruses

- 40 enteroviruses were reported (2006, 33)
- 19 enteroviruses were serotyped as Coxsackie B4 (2), Echovirus 6 (1), Echovirus 30 (6), and Enterovirus type 71 (10)

Special Bacteriology

Listeria monocytogenes

- 8 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)
- 2 cases were perinatal, both resulted in intrauterine death
- 6 cases were in adults who were elderly and/or had underlying illness

Corynebacterium diphtheriae

- 3 isolates of *Corynebacterium diphtheriae* were received for toxigenicity testing, typing and surveillance purposes
- 1 var. *mitis* isolate was from blood of 27 year old male from Hamilton; 1 var. *gravis* isolate was from blood of 57 year old female from Auckland; 1 var. *mitis* isolate was from cutaneous source, patient aged 29 years from Auckland
- the 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.

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