Volume 9 Issue 3 ISSN 1176-2888 (Print) ISSN 1178-8313 (Online)

New Zealand Public Health Surveillance Report

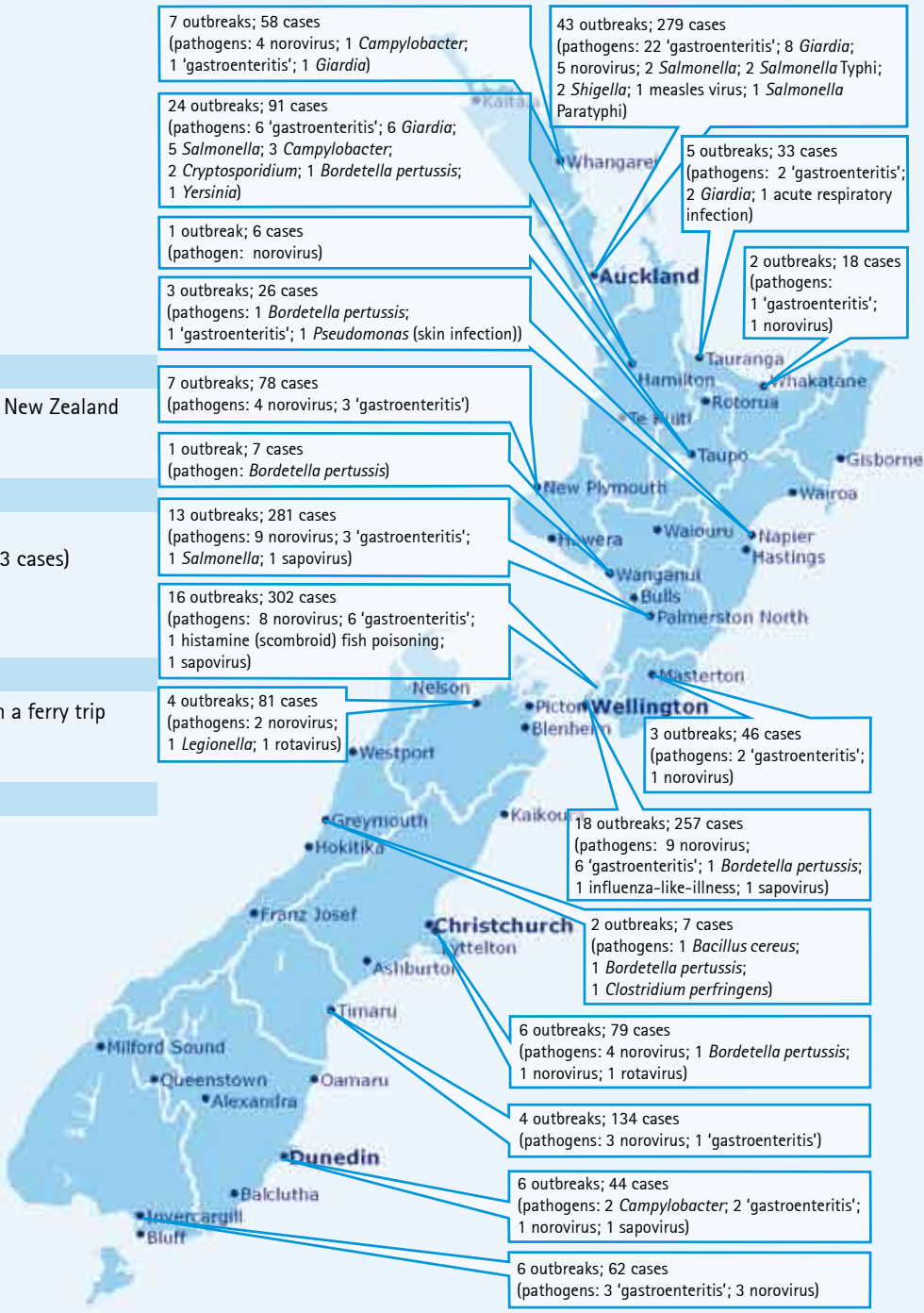
September 2011: Covering April to June 2011

Contents & Highlights

The latest reports from Sexually Transmitted Infections Surveillance, Antimicrobial Resistance, Virology and Enteric Reference Laboratories are available at www.surv.esr.cri.nz

This Quarter's Outbreaks

Notification and outbreak data in this issue are drawn from the April to June quarter of 2011. 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 6 July 2011. Three outbreaks involved more than one pathogen therefore individual pathogen outbreak numbers may not sum to totals.



1. Editorial

Outbreak of *E. coli* O104:H4 in Germany, May 2011

In 1982, O157 Shiga toxin-producing *Escherichia coli* (STEC) was recognised as a cause of illness. It has subsequently emerged as an important foodborne pathogen, causing outbreaks world wide and serious sequelae such as haemolytic-uremic syndrome (HUS). Since 1982, more than 100 other O serogroups of STEC (non-O157 STEC) have been associated with sporadic and epidemic human diarrheal diseases. Increasingly non-O157 STEC are reported as causative agents of human illness and represent a significant burden overseas.[1] The Centers for Disease Control and Prevention estimates that non-O157 STEC cause 36,700 illnesses, 1100 hospitalisations and 30 deaths in the United States each year.[2] The true incidence and burden of illness caused by these *E. coli* serotypes however remain unknown. In 2000, only 68% of clinical laboratories in the United States tested routinely for infections due to O157 STEC, with fewer laboratories testing for non-O157 STEC infections.2 A recent example of importance of non-O157 STEC as a human pathogen was illustrated in the recent outbreak of *E. coli* O104:H4 in Germany. On 23 May 2011, an Early Warning Response System notice was issued by the Robert Koch Institute in Germany due to an increase in the number of clinical cases of HUS and bloody diarrhoea caused by STEC. An unusual feature of this outbreak was that the majority of HUS cases were adults, and two-thirds were females. As of 7 July 2011 in the European Union, the number of non-HUS STEC cases was 3016 (16 deaths) and the number of HUS cases was 752 (28 deaths). On 10 June 2011, epidemiological and food chain evidence suggested that bean and seed sprouts were the outbreak source in Germany. On 24 June 2011, France reported a cluster of eight patients (near Bordeaux) with bloody diarrhoea, seven of whom had developed HUS. In three of the patients, infection with *E. coli* O104:H4 had been confirmed. A joint assessment by the European Food Safety Authority and the European Centre for Disease Control (ECDC) declared that fenugreek seeds imported from Egypt seemed to be the link between the German and the French outbreak.

The outbreak strain

The outbreak strain, *E. coli* O104:H4, was characterised and shown to carry disease-causing genes (*aggR* and *stx2*) from two types of pathogenic *E. coli*, enteroaggregative *E. coli* (EAggEC) and enterohemorrhagic *E. coli* (EHEC).

EHEC are a subset of STEC considered to be pathogenic to humans. They produce Shiga toxins (Stx1 and/or Stx2) and attaching and effacing lesions (*eae*). The most significant serotype is O157:H7. The symptoms range from mild diarrhoea to severe bloody diarrhoea with complications such as HUS mainly in children under 5 years of age. The main reservoir for EHEC is ruminant animals.

EAggEC, first described in 1987, do not secrete the heat-stable or heat-labile toxins of enterotoxigenic *E. coli* (ETEC) and produce a characteristic aggregative pattern of adherence to Hep2 cells in culture. This is due to the presence of an aggregative adherence fimbria, the expression of which is regulated by *aggR*. EAggEC are associated with acute or persistent diarrhoea, especially in developing countries, and they are a significant cause of travellers' diarrhoea. Their pathogenicity is poorly understood.

Taken together, these data indicate that the outbreak strain is a typical EAggEC strain that has acquired *stx2*. This *E. coli* strain may persist among human populations as EAggEC are common in all populations of the world and have no animal reservoir.

Is this a new strain?

E. coli O104:H4 was first identified in Germany in 2001 where it caused two HUS cases. It was subsequently isolated in France in 2004, Korea in 2005, The Republic of Georgia in 2009, and in Finland in 2010.

Genomic comparison of the two strains from Germany (2001 and the current outbreak strain) indicates that the current outbreak strain has acquired *stx2* and multiple antibiotic resistance genes, making this strain highly pathogenic.

Microbiological investigation in New Zealand

In New Zealand laboratory testing currently undertaken is unlikely to identify non-O157 STEC infections.[3] Here, we (a) give examples of recommended methods for the identification of non-O157 STEC infections (including *E. coli* O104:H4) in clinical samples[4] and (b) look at the capability that Enteric Reference Laboratory (ERL) at ESR has for the detection, isolation and characterisation of non-O157 STEC including *E. coli* O104:H4.

a) Examples of recommended methods to detect non-O157 STEC in clinical samples are:

- Use of a differential and selective medium such as CT-SMAC/SMAC
- Shiga toxin immunoassays of enrichment broth cultures for the detection of Stx1 and Stx2 (e.g., ImmunoCard STAT® EHEC).

b) ERL capabilities for routine surveillance of STEC are:

- Multiplex PCR for the detection of s*tx1, stx2, eae, hlyA*
- Use of a differential and selective media: CT-SMAC and EHEC O104 PCR for the rapid confirmation of the O104 serotype
- K9 antisera (slide agglutination). *E. coli* K9 is identical to the *E. coli* O104 antigen and will cross react with *E. coli* O104
- ESBL agar for the detection of ESBL-producing *E. coli* (including *E. coli* O104:H4), if mixed samples are received
- Further characterisation of pure isolates by conventional serotyping, biochemical identification and Verocell assay (toxin production).

Since 1997 the ERL has identified sporadic cases of non-O157 STEC. The ERL remains in contact with the Institut Pasteur in Paris and the ECDC as new protocols for the rapid identification of *E. coli* O104:H4 are made available.

Public health implications and advice on the surveillance and reporting of *E. coli* infections and recommended precautions *(adapted from a World Health Organization communication[5])*

The World Health Organization (WHO) has advised member states to strengthen surveillance of severe diarrhoeal disease, particularly bloody diarrhoea, and to share information rapidly at all levels. At the international level, it was recommended this should be done through the International Health Regulations' reporting procedures. It was also advised that the investigation should also make full use of specialised reference laboratories and collaborating centres.

Until investigations are completed, the WHO has advised consumers in the WHO European Region that bean sprouts or sprouted seeds – whether commercially or home grown – should only be eaten when they have been thoroughly cooked. *E. coli* bacteria are killed at 70°C. In addition, people should wash their hands after handling seeds intended for planting or sprouting.

Further, people should always follow normal food-hygiene measures, including washing fruit and vegetables thoroughly in clean running water, washing their hands after using the toilet and before and after handling food, keeping raw and cooked food separate and using different equipment when handling raw and cooked food, and cooking food thoroughly. People experiencing bloody diarrhoea are advised to seek medical attention urgently.

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

Reported by Muriel Dufour, NCBID Microbiology and Don Bandaranayake, Health Intelligence Team, ESR.

2. Notifiable Disease Surveillance

The following is a summary of disease notifications for the April to June quarter of 2011 and cumulative notifications and rates calculated for a 12-month period (July 2010 to June 2011). 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 RG and Altman DG 2000. Proportions and their differences. In: Statistics with Confidence. BMJ Books, Bristol]. Data contained within this report are based on information recorded in EpiSurv by public health service staff up to 6 July 2011. As this information may be updated over time, these data should be regarded as provisional.

National surveillance data tables are available at www.surv.esr.cri.nz

VACCINE PREVENTABLE DISEASE

Hepatitis B

- *Notifications:* 25 notifications in the quarter (2010, 12); 63 notifications over the last 12 months (2010, 57) giving a rate of 1.4 cases per 100,000 population (2010, 1.3); not a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (12 cases) and from the same quarter last year (12 cases). Cases were aged between 17 and 55 years.

Invasive Pneumococcal Disease

- *Notifications:* 135 notifications in the quarter (2010, 148); 526 notifications over the last 12 months (2010, 635), giving a rate of 12.0 cases per 100,000 population (2010, 14.7), a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (76 cases). Cases were aged between 4 months and 95 years, with 5 cases under the age of 2 years.

Measles

- *Notifications:* 71 notifications in the quarter (2010, 8); 117 notifications over the last 12 months (2010, 245), giving a rate of 2.7 cases per 100,000 population (2010, 5.7), a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (35 cases) and from the same quarter last year (8 cases). 44 cases were laboratory confirmed.

Mumps

- *Notifications:* 22 notifications in the quarter (2010, 10); 51 notifications over the last 12 months (2010, 61), giving a rate of 1.2 cases per 100,000 population (2010, 1.4), not a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (10 cases). 5 cases were laboratory confirmed.

Pertussis

- *Notifications:* 188 notifications in the quarter (2010, 181); 773 notifications over the last 12 months (2010, 1217), giving a rate of 17.7 cases per 100,000 population (2010, 28.2), a statistically significant decrease.

Rubella

- *Notifications:* 9 notifications in the quarter (2010, 1); 15 notifications over the last 12 months (2010, 1), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (1 case). 3 cases were laboratory confirmed.

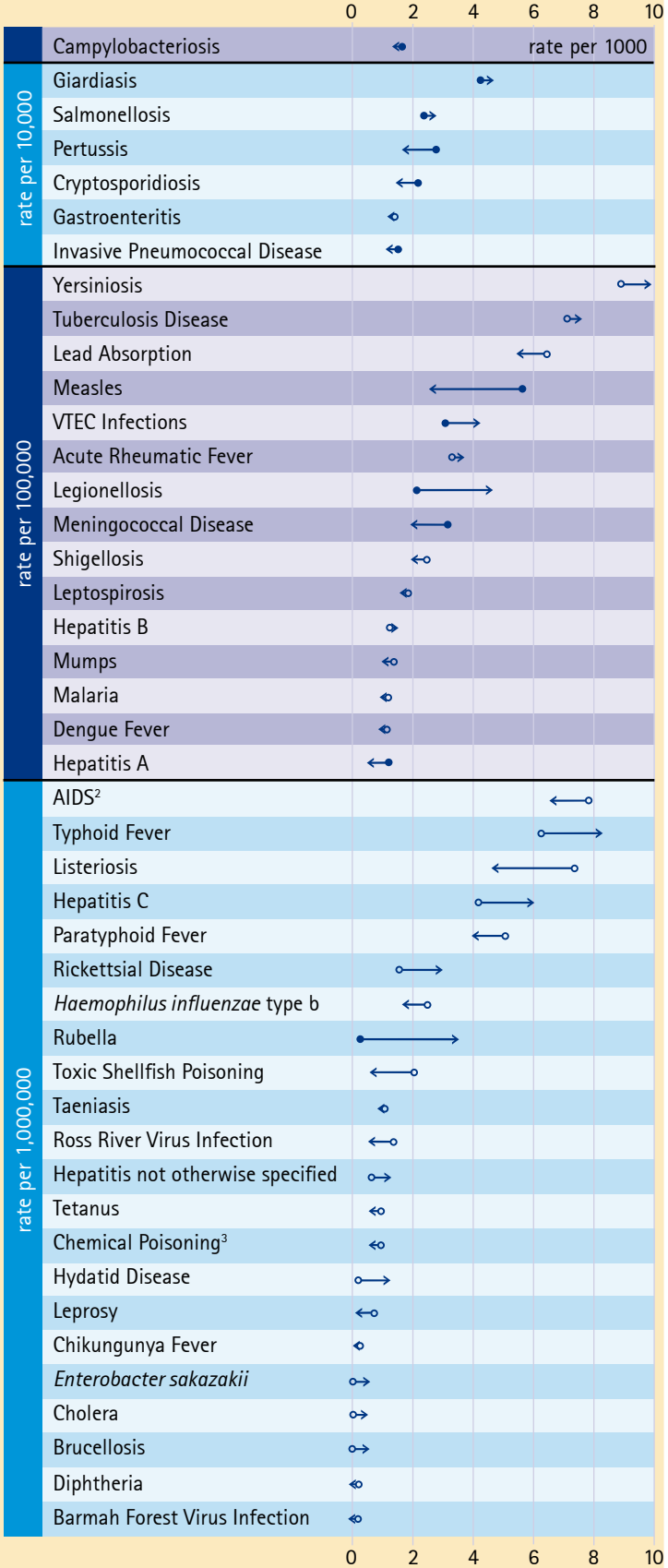
INFECTIOUS RESPIRATORY DISEASES

Meningococcal Disease

- *Notifications:* 25 notifications in the quarter (2010, 28); 90 notifications over the last 12 months (2010, 137), giving a rate of 2.1 cases per 100,000 population (2010, 3.2), a statistically significant decrease.
- *Comments:* cases were distributed by age as follows: 4 (<1 year), 5 (1–4 years), 1 (5–14 years), and 15 (15 years and over); 9 cases were the epidemic strain.

National Surveillance Data

12-Monthly Notification Rate Changes[1]



Notifications per 1000 or 10,000 or 100,000 or 1,000,000 population

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 July 2010 to June 2011 and compared to previous 12-month rates.

[2] Data provided by the AIDS Epidemiology Group, University of Otago. Note: changes in the 12-month notification rate should be interpreted with caution as this often reflects late notifications.

[3] From the environment.

ENTERIC INFECTIONS

Campylobacteriosis

- *Notifications:* 1316 notifications in the quarter (2010, 1412); 6612 notifications over the last 12 months (2010, 7571), giving a rate of 151.4 cases per 100,000 population (2010, 175.4), a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (1481 cases).

Gastroenteritis

- *Notifications:* 150 notifications in the quarter (2010, 103); 603 notifications over the last 12 months (2010, 639), giving a rate of 13.8 cases per 100,000 population (2010, 14.8), not a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (103 cases).
- *Note:* this is not a notifiable disease per se except in persons with a suspected common source or with a high risk occupation. 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:* 271 notifications in the quarter (2010, 224); 1225 notifications over the last 12 months (2010, 1020), giving a rate of 28.0 cases per 100,000 population (2010, 23.6), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (369 cases) and a statistically significant quarterly increase from the same quarter last year (224 cases).

VTEC Infections

- *Notifications:* 53 notifications in the quarter (2010, 34); 183 notifications over the last 12 months (2010, 130), giving a rate of 4.2 cases per 100,000 population (2010, 3.0), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (34 cases).

Yersiniosis

- *Notifications:* 86 notifications in the quarter (2010, 78); 431 notifications over the last 12 months (2010, 384) giving a rate of 9.9 per 100,000 population (2010, 8.9), not a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (129 cases).

ENVIRONMENTAL EXPOSURES & INFECTIONS

Cryptosporidiosis

- *Notifications:* 79 notifications in the quarter (2010, 187); 696 notifications over the last 12 months (2010, 979), giving a rate of 15.9 cases per 100,000 population (2010, 22.7), a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly decrease from the same quarter last year (187 cases).

Giardiasis

- *Notifications:* 464 notifications in the quarter (2010, 535); 1975 notifications over the last 12 months (2010, 1847), giving a rate of 45.2 cases per 100,000 population (2010, 42.8), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (616 cases) and from the same quarter last year (535 cases).

Legionellosis

- *Notifications:* 38 notifications in the quarter (2010, 32); 199 notifications over the last 12 months (2010, 94), giving a rate of 4.6 cases per 100,000 population (2010, 2.2), a statistically significant increase.

NEW, EXOTIC & IMPORTED INFECTIONS

Hepatitis A

- *Notifications:* 3 notifications in the quarter (2010, 16); 28 notifications over the last 12 months (2010, 54), giving a rate of 0.6 cases per 100,000 population (2010, 1.3); a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly decrease from the same quarter last year (16 cases). Cases were aged between 8 and 87 years, with a single case under the age of 16 years. Overseas travel information was recorded for 2 cases. Of these, one case had not travelled overseas during the incubation period.

Malaria

- *Notifications:* 17 notifications in the quarter (2010, 7); 48 notifications over the last 12 months (2010, 50), giving a rate of 1.1 cases per 100,000 population (2010, 1.2), not a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (7 cases). All cases had malaria parasites in a blood film; 14 cases were overseas during the incubation period and 3 cases had prior history of overseas travel that could account for their infection. Places visited or resided in were India (10 cases), China, Cote d'Ivoire, Mozambique, Pakistan, South Africa, and the Solomon Islands (one case each).

Shigellosis

- *Notifications:* 15 notifications in the quarter (2010, 38); 90 notifications over the last 12 months (2010, 108), giving a rate of 2.1 cases per 100,000 population (2010, 2.5), not a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (33 cases) and from the same quarter last year (38 cases). Overseas travel information was recorded for 6 cases. Of these, 2 cases had not travelled overseas during the incubation period and had no prior history of travel that could account for their infection.

BLOOD- AND TISSUE-BORNE INFECTIONS

Hepatitis C

- *Notifications:* 9 notifications in the quarter (2010, 2); 26 over the last 12 months (2010, 18) giving a rate of 0.6 cases per 100,000 population (2010, 0.4); not a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (2 cases). Cases were aged between 3 months and 64 years, with a single case under the age of 16 years.

3. Other Surveillance Reports

Antimicrobial resistance among bacteria from selected New Zealand food-producing animals

Few data are available on antimicrobial resistance among bacteria associated with livestock in New Zealand. Therefore, over a 12-month period commencing October 2009, ESR was commissioned to undertake a survey of resistance among bacteria isolated from freshly dressed carcasses of very young (bobby) calves, pigs and broiler poultry in New Zealand abattoirs and processing plants. These animal groups were chosen as there is a greater use of antimicrobials in rearing these animals compared with other food-producing animals. The bacteria included were *Campylobacter* and *Salmonella*, representative of pathogenic bacteria, and *Escherichia coli* and Enterococci (*Enterococcus faecalis* and *Enterococcus faecium*), representative of commensal bacteria.

The bacterial isolates were sourced from samples routinely collected from freshly dressed carcasses as part of the National Microbiological Database programme – a monitoring programme that ensures common microbiological standards for foods. Antimicrobial susceptibility testing was performed by microbroth dilution (MBD) using the methods of the Clinical and Laboratory Standards Institute and commercially prepared MBD plates.[1,2] The antimicrobials tested included those commonly used in animal husbandry and those that are important for treating human infection. The aim was to test the antimicrobial susceptibility of 300 isolates of each of the four bacterial groups from each of the three animal groups. While the carcass sampling targets were met, the target of 300 isolates was not achieved for all bacteria from all animal groups. During

the 12-month survey period, only 56 and 11 *Campylobacter* were isolated from calves and pigs, respectively, and only 19, 6 and 3 *Salmonella* were isolated from calves, pigs and poultry, respectively.

Resistance was uncommon among *Campylobacter*. The majority (94.5%) of the *Campylobacter* isolates were *Campylobacter jejuni*. Among isolates of this species, 91.8% and 95.9% from calves and poultry, respectively, were susceptible to all antimicrobials tested. The only resistances identified among the *C. jejuni* isolates were ciprofloxacin resistance (2.7%) in poultry isolates, and streptomycin resistance in calf (8.2%) and poultry (1.0%) isolates. No *C. jejuni* were isolated from pigs.

Only 28 *Salmonella* isolates, in total, were included in this survey. Two isolates from calves were resistant to streptomycin, and two isolates from pigs were sulphonamide resistant and one had additional resistance to trimethoprim.

Among the *E. coli* isolates, 55.6% from poultry, 48.0% from calves and 35.0% from pigs were fully susceptible to all antimicrobials tested. There was no resistance to cefotaxime, ciprofloxacin or gentamicin among *E. coli* from any of the animal groups. None of the isolates produced extended-spectrum or AmpC β-lactamase. Rates of resistance to ampicillin, chloramphenicol, neomycin, spectinomycin, streptomycin, sulphonamides and tetracycline were relatively high among *E. coli* from some animals.

Excluding bacitracin and tylosin (as there are no reference breakpoints for the minimum inhibitory concentrations (MICs) of these antibiotics), and also excluding quinupristin/dalfopristin for *E. faecalis* (as this species is intrinsically resistant to quinupristin/dalfopristin), among the *E. faecalis* isolates, 53.1% from pigs and 42.2% from calves were susceptible to all the other antimicrobials tested compared with 17.9% of isolates from poultry. Among the *E. faecium* isolates, 31.6% from pigs were fully susceptible compared with 20.3% of isolates from poultry and 5.4% from calves. There was no resistance to ampicillin or vancomycin among *E. faecalis* or *E. faecium* isolates from any of the animal groups.

While the bacitracin MICs were not interpreted, the MICs for poultry enterococcal isolates were high, with 95.0% of *E. faecalis* and 98.7% of *E. faecium* isolates having bacitracin MICs ≥512 mg/L. Bacitracin is routinely used in the rearing of broiler poultry to control clostridial enteritis.

The prevalence of resistance among bacteria from the food-producing animals included in this survey was usually less than that reported for human isolates of the same bacterial species isolated in New Zealand in 2009, especially for the antibiotics of most importance in human medicine. Comparison of the results from this survey with the limited data available from earlier New Zealand studies of animal isolates does not suggest a trend of increasing resistance among bacteria from animals in New Zealand.

Comparison of the results of this survey with 2009 data from the Danish DANMAP surveillance system, which uses similar methodology to that used in this survey but does not include an animal category of very young calves, showed that, with the exception of sulphonamide resistance in *E. coli* from poultry, resistance was either lower in pigs and poultry in New Zealand or not significantly different for the antibiotics that were commonly tested in both this survey and the DANMAP system.[3]

This is the first such survey undertaken in New Zealand and should provide baseline information to monitor any changes in resistance among bacteria from food-producing animals as well as provide current information to guide policy decisions on the use of antimicrobials in animal husbandry in this country. The survey was funded by the New Zealand Food Safety Authority.

A more detailed report on the survey is available at http://www.foodsafety.govt.nz/elibrary/industry/antimicrobial-resistance-in-bacteria.pdf

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

Reported by Helen Heffernan, Health 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 to June 2011). Comparisons are made to the previous quarter (January to March 2011), and to the same quarter in the previous year (April to June 2010). Note that the outbreak data in this section are notified to ESR by the Public Health Services.

General

- 171 outbreaks notified in this quarter (1889 cases).
- 110 are 'final' reports (1666 cases); 61 are 'interim' reports (223 cases) that have yet to be finalised and closed.

All data that follow relate to final reports only.

- 15.1 cases on average per outbreak, compared with 11.1 cases per outbreak in the previous quarter (8.5 cases per outbreak in the same quarter of last year).
- Nine hospitalisations: norovirus (3 cases), *Pseudomonas* (skin infections) (2 cases), 'gastroenteritis', *Giardia*, histamine (scombroid) fish poisoning and *Shigella* (1 case each).
- No deaths.
- Three outbreaks involved more than one pathogen therefore individual pathogen outbreak numbers may not sum to totals.

Pathogens

- 48 norovirus outbreaks (1132 cases).
- 25 'gastroenteritis' outbreaks (325 cases).
- 14 *Giardia* outbreaks (45 cases).
- 8 *Salmonella* outbreaks (28 cases).
- 3 *Campylobacter* outbreaks (9 cases).
- 4 sapovirus outbreaks (69 cases).
- 2 *Bordetella pertussis* outbreaks (9 cases).
- 2 rotavirus outbreaks (54 cases).
- 2 *Shigella* outbreaks (6 cases).
- 1 acute respiratory infection outbreak (10 cases).
- 1 *Bacillus cereus* outbreak (2 cases).
- 1 *Clostridium perfringens* outbreak (2 cases).
- 1 histamine (scombroid) fish poisoning outbreak (9 cases).
- 1 *Pseudomonas* (skin infection) outbreak (11 cases).
- 1 *Salmonella* outbreak (9 cases).

Modes of Transmission

Note that reporting allows for multiple modes of transmission to be selected. In some instances no modes of transmission are selected for outbreaks notified to ESR.

- 91 person-to-person, from (non-sexual) contact with an infected person (including droplets): 46 norovirus (1105 cases), 19 'gastroenteritis' (287 cases), 11 *Giardia* (36 cases), 6 *Salmonella* (23 cases), 4 sapovirus (69 cases), 2 *B. pertussis* (9 cases), 2 *Campylobacter* (6 cases), 2 rotavirus (54 cases), and 1 acute respiratory infection (10 cases).
- 20 environmental, from contact with an environmental source (e.g., swimming): 13 norovirus (303 cases), 3 'gastroenteritis' (52 cases), 2 *Giardia* (5 cases), 2 rotavirus (54 cases), 1 *Salmonella* (9 cases), and 1 sapovirus (9 cases).
- 18 foodborne, from consumption of contaminated food or drink (excluding water): 5 *Salmonella* (13 cases), 4 'gastroenteritis' (16 cases), 3 norovirus (15 cases), 2 *Campylobacter* (7 cases), 1 *B. cereus* (2 cases), 1 *C. perfringens* (2 cases), 1 *Giardia* (3 cases), 1 histamine (scombroid) fish poisoning (9 cases), and 1 *Shigella* (3 cases).
- 6 waterborne, from consumption of contaminated drinking water: 2 *Giardia* (6 cases), 2 *Salmonella* (6 cases), 1 *Campylobacter* (4 cases), and 1 *Shigella* (3 cases).
- 3 zoonotic, from contact with infected animal: 2 *Giardia* (4 cases) and 1 *Salmonella* (3 cases).
- 2 'other' mode: 1 *Giardia* (6 cases) and 1 *Pseudomonas* (skin infection) (11 cases).
- 4 mode of transmission unknown: 2 'gastroenteritis' (18 cases), 1 norovirus (25 cases), and 1 *Shigella* (3 cases).

Circumstances of Exposure

Common 'settings' where the exposures occurred are identified below.

- 39 long term care facility: 29 norovirus (793 cases), 7 'gastroenteritis' (118 cases), 1 acute respiratory infection (10 cases), 1 *B. pertussis* (5 cases), and 1 sapovirus (19 cases).
- 24 home: 12 *Giardia* (40 cases), 5 *Salmonella* (13 cases), 3 'gastroenteritis' (16 cases), 2 *Campylobacter* (6 cases), and 2 norovirus (13 cases).
- 17 childcare centre: 10 'gastroenteritis' (150 cases), 2 norovirus (51 cases), 3 sapovirus (53 cases), 1 *Giardia* (2 cases), 1 rotavirus (17 cases), and 1 *Salmonella* (9 cases).
- 11 hospital (acute care): 10 norovirus (199 cases), 1 'gastroenteritis' (10 cases), and 1 rotavirus (37 cases).
- 3 restaurant/café/bakery: 1 *Campylobacter* (3 cases), 1 'gastroenteritis' (6 cases), and 1 norovirus (6 cases).
- 2 takeaways: 1 histamine (scombroid) fish poisoning (9 cases) and 1 norovirus (2 cases).
- 1 cruise ship, airline, tour bus, train: norovirus (30 cases).
- 1 workplace: norovirus (3 cases).
- 4 'other setting': 1 'gastroenteritis' (4 cases), 1 norovirus (8 cases), 1 *Pseudomonas* (skin infection) (11 cases), and 1 *Salmonella* (2 cases).
- 1 supermarket/delicatessen: 1 *C. perfringens* (2 cases) and 1 *B. cereus* (2 cases).
- 1 outbreak had two exposure settings recorded.
- 8 outbreaks had no exposure settings recorded.

Common 'settings' where the preparations occurred in foodborne outbreaks are identified below.

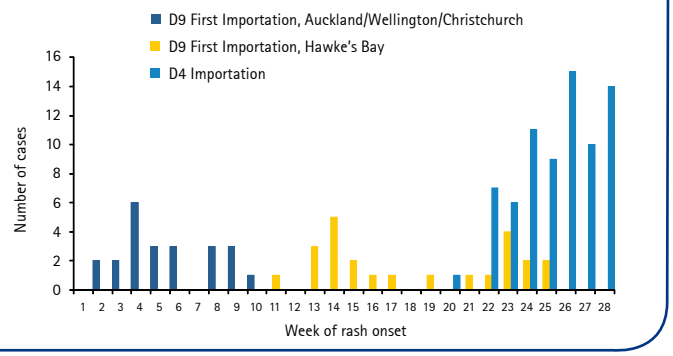
- 6 home: 2 *Salmonella* (5 cases), 1 *B. cereus* (2 cases), 1 *Campylobacter* (4 cases), 1 *C. perfringens* (2 cases), 1 gastroenteritis (2 cases) and 1 norovirus (7 cases).
- 2 takeaways: 1 'gastroenteritis' (4 cases) and 1 histamine (scombroid) fish poisoning (9 cases).
- 1 caterers: 'gastroenteritis' (4 cases).
- 1 restaurant/café: *Campylobacter* (3 cases).
- 1 supermarket/delicatessen: 'gastroenteritis' (2 cases).
- 1 outbreak had two preparation settings recorded.

5. Outbreak Case Reports

Measles outbreaks in Auckland, January to June 2011

Measles is a highly communicable disease that spreads readily in communities with inadequate immunisation rates. Between January and June 2011, Auckland experienced seven separate importations of measles from overseas involving several distinct genotypic strains. Two of these events led to large outbreaks involving nearly 100 secondary cases (Figure 1). These two large outbreaks are described.

Figure 1. Measles notifications from Auckland importations, January to June 2011



The first outbreak was linked to an index group of 17 people who returned to New Zealand from Singapore and the Philippines, via Australia, on 11 January 2011.[1] Three of these people were infectious on the flights and one became infectious later, according to rash onset dates. The first notification to Auckland Regional Public Health Service was on 21 January 2011. Diagnosis was based on clinical presentation and serology. Five passengers on the Brisbane to Auckland flight subsequently developed measles with rash onsets 13 to 15 days post-exposure, as did one additional case with no epidemiological linkage to the index group. Five of this group, including the "sporadic" case, had identical genotypic sequences (genotype D9) which they shared with three Australian cases who had travelled on the Singapore to Brisbane flight with the index group. This genetic sequence is novel on the Centers for Disease Control database. During the next 5 weeks, a further 11 cases were notified in Auckland with genetically identical sequences to the earlier cases. Of the 20 Auckland cases, six were aged less than 15 months (not eligible for the measles, mumps and rubella (MMR) vaccine), and none of the 14 persons eligible for the MMR vaccine had been immunised. Of these 20 cases, 12 were Māori, four were Pacific Islanders, two were European and the remaining cases were of unknown ethnicity. This outbreak led to secondary cases outside the Auckland region in Hawke's Bay (24 cases), Wellington (2 cases) and Canterbury (1 case).

The last of the D9 Auckland cases was epidemiologically linked to the first case in a Hawke's Bay outbreak which started on 17 March 2011. This outbreak has continued with 24 cases confirmed to date (6 cases have been genotyped and are all genotype D9). Of the 24 cases confirmed so far, 18 cases were Māori and six were of European ethnicity, and three of these cases were aged less than 15 months (not eligible for MMR vaccination). Of the 21 cases eligible for immunisation, one child had received only one MMR dose, despite being eligible for two doses, one adult was of unknown immunisation status and the remaining 19 cases were unimmunised, mostly by choice.

The second large outbreak in Auckland began with notification on 28 May 2011 of a case of measles in a school child. Active case finding in the school community identified a further six cases with similar onset dates and all were closely linked to one room and one social group. Contact histories led to the conclusion that exposure had probably occurred on 19 May 2011 in the school. The likely index case was a classmate with probable exposure in the United Kingdom in mid May 2011. Six of the first seven notified cases had identical sequences of genotype D4, a genotype that is circulating in European countries, including the United Kingdom. The school's immunisation register was rapidly updated and indicated 70 pupils were not fully immunised (13% of the roll), all of whom were excluded from school. A further 17 cases were notified in the following 2 weeks, 14 of them with direct epidemiological links to the initial cluster at the school, with either household, school or community transmission. The other three cases could not be linked to the initial cluster. At the time of writing, there were 48 cases with rash onsets before 30 June 2011 in this outbreak. Of these, only one case was immunised age appropriately (aged 3 years, one MMR dose in 2008) and none were aged less than 15 months (not eligible for MMR). Forty-seven were European and one case was of Māori ethnicity.

During these outbreaks hundreds of non-immune students (and a few teachers) have been excluded from schools and early childhood education centres in both Auckland and Hawke's Bay.

Outbreaks of measles in New Zealand can be reduced and eventually stopped by increasing immunisation coverage and intensification of public health efforts, including post-exposure prophylaxis, isolation, quarantine and communication to the public and health professionals. The recent outbreaks described here have predominantly affected unimmunised people. Had the index cases been immune the outbreaks might have been completely avoided. Efforts should be maintained to improve New Zealand's on-time immunisation coverage, and to ensure people are immunised appropriately for measles before international travel.

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

Reported by Richard Hoskins, Medical Officer of Health, Auckland Regional Public Health Service and Lester Calder, Medical Officer of Health, Hawke's Bay District Health Board.

A school camp gastroenteritis outbreak associated with a ferry trip

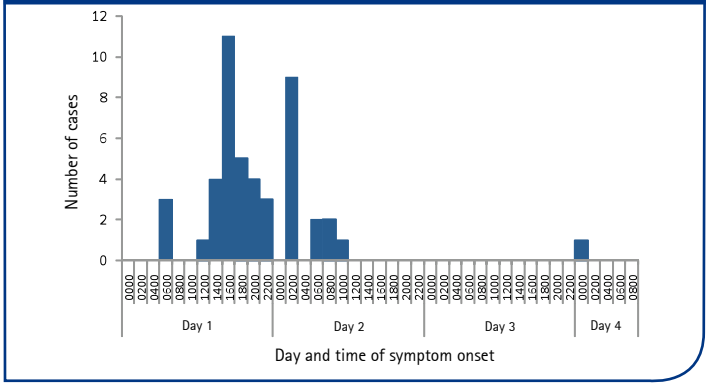
On Wednesday 30 March 2011 the Public Health Service Nelson was notified of an outbreak of gastroenteritis amongst a school group from Wellington staying at a local adventure camp. The group of 53 students (two classes) and six adults (teachers and parent helpers) had arrived at the camp the previous afternoon after travelling from Wellington on a Cook Strait ferry and charter bus. The first case suffered symptoms at approximately 6 am on 30 March 2011. By 5 pm that day 20 children had been unwell with vomiting and/or diarrhoea (Figure 2).

The camp is located in a rural setting on the outskirts of Nelson and consists of cabin-style accommodation, a commercial standard kitchen (run by camp kitchen staff) and large dining hall. It offers various outdoor activities and includes a swimming pool, waterslide and pond. The camp has its own treated drinking water supply which is monitored on a regular basis.

Two Health Protection Officers and a Medical Officer of Health visited the camp at 7 pm on 30 March 2011. By this time, the number of unwell people had risen to a total of 27 students and two adults (one teacher, one parent). Sick students and adults had been physically separated from those well as soon as they showed symptoms. No camp staff reported being ill. Details were collected on the onset of illness and symptoms, activities undertaken and foods eaten by the group. Advice was provided on preventing spread, cleaning, disinfection and isolation, including provision of personal protective supplies such as disposable gloves, gowns and alcohol gel. Faecal pottles were supplied to take specimens for the purpose of identifying the implicated organism.

Initial findings were that there had been no meetings or activities involving the whole group prior to meeting at the ferry, that they had not remained together on the ferry, had not shared a meal until arrival at the camp and that there were cases in both students and parents. Given this history, the working hypothesis was that this appeared to be a common source outbreak possibly from exposure to food or an activity at the camp. Norovirus was thought an unlikely causal agent, as the usual incubation period would suggest a common source on the ferry trip, and the ferry trip had been excluded based on the information provided.

Figure 2. Epidemic curve for the school camp norovirus outbreak associated with a ferry trip



The decision was made to conduct a cohort study to attempt to identify the source of the illness. An outbreak questionnaire was developed and carried out with all students and adults at the camp on 31 March 2011. The questionnaire covered food items and all activities offered at the camp from time of arrival until mid-morning on the day of onset for the first cases. The cook was questioned further on food sources and food handling practices. A faecal specimen was requested from the cook although he reported neither he nor his family had been ill. Drinking water and pond samples were collected for analysis. In total four faecal specimens were submitted to the laboratory. The three faecal specimens taken from the students and parents were positive for norovirus. The sample from the cook was negative. The drinking water was potable and the pond water samples showed nothing of concern. Forty-five cases were identified as part of the cohort study with one further case identified 2 days later in the camp manager. Risk ratios from the cohort study did not suggest any particular risk exposure.

Late in the investigation information was received regarding an outbreak of norovirus among staff on the same Cook Strait ferry that the school group had travelled on. Genotyping of specimens collected from one camp case and one Cook Strait ferry staff case showed both were GII.4 2008 variant. In 2011, up to 30 June, 78.7 % (63/80) of norovirus outbreaks in New Zealand were identified as caused by the GII.4 genotype, and of these 30.2% (19/63) were due to the 2008 variant. ESR advised there was high correlation between the two samples.

Follow up continued with the camp to ensure accommodation and facilities were sufficiently cleaned and disinfected prior to the next group's arrival. The bus company that transported the school group back to Picton was also given advice on cleaning the bus to prevent further spread of the disease, and information on norovirus was provided to the school to pass on to families of the group. Except for the subsequent notification of illness of the camp manager, no other reports of illness at the camp were received.

Our investigation concluded it was unlikely that the outbreak originated from a camp source. The cohort study was useful to rule out a food or activity source before the stool sample results were available as it informed our public health response. This outbreak highlighted the value in alerting all public health units if there is an outbreak associated with a national transport carrier. It also reminded us of the usefulness of having "ready to go" information sheets on cleaning guidelines for gastroenteritis outbreaks for tourist accommodations and bus companies.

Reported by Annamarie Clough, Health Protection Officer and Jill Sherwood, Medical Officer of Health. Nelson Marlborough Public Health Service.

6. Laboratory Surveillance

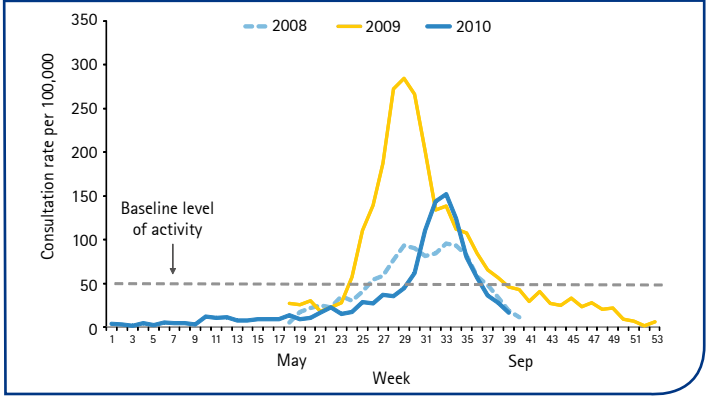
Influenza surveillance in 2010

Following the emergence of influenza A(H1N1) 09 in 2009, influenza surveillance and reporting continued beyond the normal autumn, winter and spring reporting period and carried on throughout the summer of 2009/10. Therefore, national influenza surveillance in 2010 was undertaken between January and September using a sentinel network of 91 general practices/practitioners. On average, 81 practices participated each week with a patient roll of 355,222 covering about 8.1% of the New Zealand population. It is estimated that influenza-like illness (ILI) resulting in a visit to a general practitioner affected 50,561 New Zealanders (1.2% of total population). During the surveillance period, 4112 consultations for ILI were reported and resulted in the cumulative incidence rate of 1035.6 per 100,000 patient population.

The average weekly consultation rate from May to September 2010 was 49.3 per 100,000 patient population, which is approximately half that of the 2009 rate (106.1 per 100,000). Prior to 2009, the highest rates were in 1997 (163.7 per 100,000 patient population) and 1999 (112.3 per 100,000). The lowest rate was recorded in 2000 (32.5 per 100,000).

Overall, influenza activity in 2010 was moderate (Figure 3). Influenza consultation activity remained below 50 per 100 000 from weeks 1 to 29, and then increased to a peak in week 33 (16 to 22 August), with a consultation rate of 151.6 per 100,000 patient population. The 2010 peak was lower than the peak in 2009 (284.0 per 100,000 patient population), but higher than the peaks in 2008 and 2007 (95.2 and 69.5 per 100,000 patient population, respectively). Since 1997, the highest peaks were in 2009 (284.0 per 100,000 patient population) and 1997 (244.2 per 100,000). The lowest peak was recorded in 2000 (41.7 per 100,000).

Figure 3. Weekly sentinel surveillance consultation rates for ILI, 2008 to 2010



Laboratory Surveillance continued

Consultation rates varied among District Health Boards (DHBs). ILI consultation rates above the national average were reported by Waikato DHB (87.6 per 100,000 patient population), followed by Hawke's Bay (82.6 per 100,000), Lakes (75.4 per 100,000), Hutt Valley (68.7 per 100,000), South Canterbury (68.7 per 100,000), Bay of Plenty (67.3 per 100,000), Capital & Coast (59.7 per 100,000), Canterbury (53.9 per 100,000), and Northland (51.3 per 100,000) DHBs. The geographical distribution of influenza activity also varied compared with the previous year. In particular, some regions (mainly small urban and rural areas) that had relatively low ILI activity in 2009 experienced higher levels of ILI activity in 2010.

In 2010, there were 998 hospitalisations with a primary diagnosis of influenza. This was lower than in 2009 (1517), but higher than in 2008 and 2007 (365 and 316 hospitalisations, respectively). Of these, 95.4% (952) occurred from June to October. The highest number of hospitalisations (517) occurred in August. Influenza hospitalisations peaked in weeks 31 and 32, while the sentinel and non-sentinel influenza virus detection and ILI consultations peaked in week 33.

A total of 2012 influenza viruses were identified in 2010, lower than in 2009 (4900 viruses) and higher than in 2008 (1054 viruses). Of the 2012 viruses identified, 349 came from sentinel practice surveillance from January to September. There were 1663 non-sentinel viruses identified in 2010 compared with 4276 in 2009 and 588 in 2008.

As in 2009, the pandemic A(H1N1) 09 strain was the predominant strain in 2010. No seasonal A(H1N1) virus was detected. Only a small number of seasonal A(H3N2) (12) and influenza B viruses (10) were detected in 2010. There are noticeable changes in terms of predominant patterns.

Influenza A(H1N1) viruses

In 2010, pandemic influenza A(H1N1) 2009 viruses predominated at 98.8% of the subtyped isolates. The antigenic data from New Zealand isolates indicate that most of the current circulating pandemic A(H1N1) 09 viruses are closely related to the vaccine strain A/California/7/2009 (H1N1). Although sequence analysis of the viruses from Australia, New Zealand and Singapore during 2010 indicated that there was increasing genetic drift (with two major subclades both with E374K and N125D amino acid changes from previously circulating viruses), the epidemiological, virological and serological data do not suggest a need to change the vaccine strain yet.

It seems that pandemic influenza A(H1N1) 2009 viruses have replaced seasonal influenza A(H1N1) viruses in 2010. In previous years, the seasonal influenza A(H1N1) strain predominated in three seasons (1992, 2000 and 2001) with associated relatively low hospitalisations (193 in 1992, 222 in 2000, and 343 in 2001).

Influenza A(H3N2) viruses

Influenza A(H3N2) viruses have often been associated with more severe disease and with excess pneumonia and influenza mortality. Since 1990, influenza A(H3N2) viruses predominated for 11 seasons in 1990 (83.2%), 1993 (65.7%), 1994 (98.7%), 1996 (99.1%), 1998 (51.7%), 1999 (73.7%), 2002 (68.0%), 2003 (99.6%), 2004 (91.3%), 2006 (86.3%), and 2007 (45.0%).

The highest number of deaths (94) in 1996 in New Zealand was recorded during an influenza A(H3N2) epidemic. The highest number of hospitalisations (552) was recorded in 2003 due to a season predominated by influenza A(H3N2) viruses. In 2010, only 0.7% of the subtyped viruses were influenza A(H3N2). They were antigenically closely related to the 2010 vaccine strain A/Perth/16/2009 (H3N2)-like strain.

Influenza B viruses

Since 1990, influenza B viruses predominated for 5 years in 1991 (92.3%), 1995 (68.8%), 1997 (53.5%), 2005 (87.0%), and 2008 (58.3%). Two antigenically distinct lineages of influenza B have co-circulated in many countries since the late 1980s. The B/Yamagata/16/88 lineage (most recent representative strain – B/Florida/4/2006) circulated worldwide, whereas the B/Victoria/2/87 lineage viruses only circulated in Asia and subsequently underwent independent evolution as an antigenically distinct lineage (most recent representative strain – B/Brisbane/60/2008). For reasons not wholly understood, the B/Victoria/2/87 lineage viruses remained geographically restricted to Asia until 2001.

From 1990 to 2001, B/Yamagata lineage viruses circulated exclusively in New Zealand. In 2002, the B/Victoria lineage viruses spread to New Zealand for the first time and completely replaced the B/Yamagata lineage viruses.

Since 2003, these two virus lineages have been co-circulating in New Zealand, with the B/Victoria lineage predominating in 2005 and 2008. The influenza B viruses had been associated with high disease burden in young children and the B/Victoria lineage viruses have been associated with more explosive school outbreaks than the B/Yamagata lineage viruses in New Zealand.

In 2010, there were only 10 influenza B viruses isolated. Most of the influenza B viruses were antigenically closely related to the B/Brisbane/60/2008-like strain.

Conclusion

Characterisation of the influenza viruses isolated during the 2010 winter indicated no requirement to change any of the three components of the current vaccine. Accordingly, the 2011 southern hemisphere winter influenza vaccine has the following composition:

A(H1N1)	an A/California/7/2009(H1N1)-like strain
A(H3N2)	an A/Perth/16/2009(H3N2)-like strain
B	a B/Brisbane/60/2008-like strain

Note: A/California/7/2009 (H1N1)-like strain is a pandemic A(H1N1) 09 strain.

Influenza immunisation is recommended for those at increased risk of complications from influenza due to either age or medical conditions. Influenza vaccination has been free for people aged 65 years and over since 1997. Since 1999, it has been extended to younger people with chronic illnesses who are at risk of developing complications from influenza.

A more detailed report is available at
http://www.surv.esr.cri.nz/virology/influenza_annual_report.php

Reported by Sue Huang and Liza Lopez, Health Programme, ESR.

Mycology

Tables detailing the biannual summary of opportunistic mycoses and aerobic actinomycetes in New Zealand are available at www.surv.esr.cri.nz/surveillance/NZPHSR.php

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

Reprinting: Articles in the New Zealand Public Health Surveillance Report may be reprinted provided proper acknowledgement is made to the author and to the New Zealand Public Health Surveillance Report as source.

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

Please send contributions and feedback 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

The content of this publication does not necessarily reflect the views and policies of ESR or the Ministry of Health.

ESR
Specialist Science Solutions
manaaki tangata taiao hoki
protecting people and their environment through science