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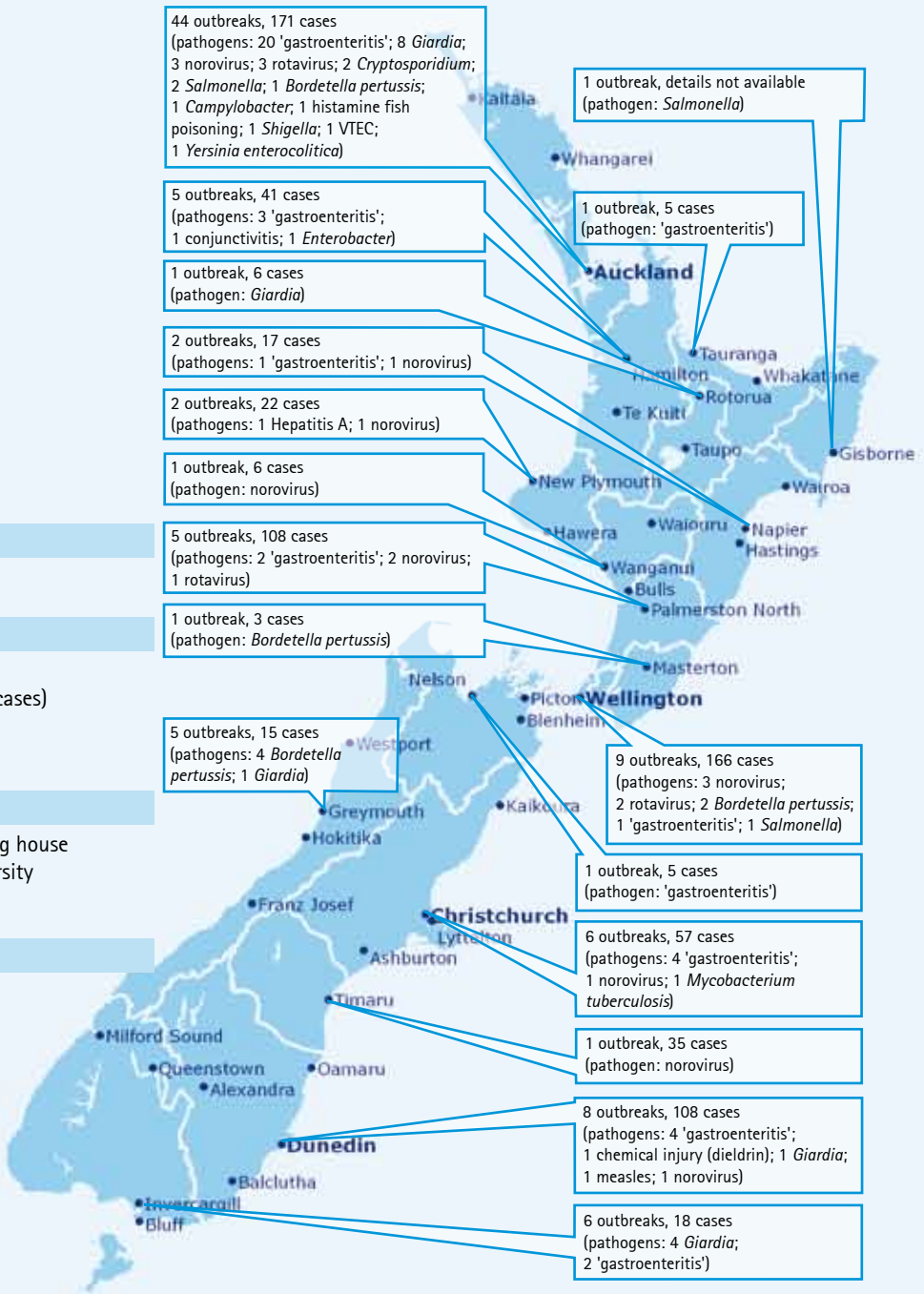
New Zealand Public Health Surveillance Report

June 2009: Covering January – March 2009

Contents & Highlights

This Quarter's Outbreaks

Notification and outbreak data in this issue are drawn from the January – March quarter of 2009. 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 8 April 2009.



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

1. Editorial

The emergence of Swine Influenza A(H1N1)

On 25 April 2009, the Director General of the World Health Organization (WHO) declared the outbreak of a disease caused by a novel H1N1 influenza virus in Mexico a public health emergency of international concern under the International Health Regulations 2005.[1,2] The virus was first identified by CDC in a specimen received from a patient who fell ill in San Diego County on 30 March 2009[3] but originated earlier in Mexico.[4] Currently designated Swine-Origin Influenza A(H1N1) (S-OIV) virus, it results from an "unusually mongrelised" recombination of at least four influenza A H1N1 influenza subtypes from North American and Eurasian swine, humans, and birds.[5] The initial clinical manifestations of S-OIV were typical of seasonal influenza, ranging from mild symptoms (fever, cough, sore throat, vomiting and diarrhoea) to severe illness (pneumonia, secondary complications and death). The incubation period may be as long as 1-7 days but is likely to be 1-4 days.[6] S-OIV is not currently known to be epizootic in pigs (although pigs could be infected by exposure to humans), but exhibits human-to-human transmission.

As of May 15, 2009, a total of 7520 cases[7] and 65 deaths[8] have been officially reported from 34 countries. All nine S-OIV cases confirmed in New Zealand to 15 May have been in or travelled from affected countries overseas[9,10]. The first three positive cases were among a group of Rangitoto College students and teachers returning to New Zealand from Mexico and initially tested positive for Influenza A on 26 April[11] and for S-OIV on 28 April.[12]

The current USA outbreak mostly affects children aged 18 years or younger.[13] Most adults have substantial immunity to seasonal influenza AH1 variants that have circulated in the human population between 1919 to the present. It is not yet known whether cross-reacting antibodies from previous seasonal AH1 infections provide protection against S-OIV. The reproduction rate (the number of new persons infected by each case also known as R_0) has been estimated to be in excess of 1.0 (1.16-3.1) using several modelling approaches and populations.[14] However, virulence and transmissibility could increase following adaptation to humans or reassortment with other human influenza viruses. Vaccine development and production is likely to take five-six months from the identification of the strain with subsequent limited supply.[15] However, anti-virals such as oseltamivir and zanamivir are currently effective.

The virus has caused sustained community level outbreaks in at least two countries in one WHO region and a pandemic is considered imminent – consistent with WHO Pandemic Alert Level 5.[16] The Ministry of Health is implementing containment measures including border control, quarantine measures, and prophylactic treatment. No secondary cases have yet been detected and containment measures appear to have been successful thus far.

ESR provides National Influenza Centre (NIC) laboratory confirmation and surveillance of S-OIV including anti-viral susceptibility testing. Increased capacity is available in the event of a pandemic or other emergency through collaboration with National Centre for Biosecurity and Infectious Disease - Wallaceville[17] partner MAF's Investigation and Diagnostic Centre. ESR has also been supporting the response to S-OIV through epidemiological and laboratory surveillance and analysis. Prompt clinical and laboratory diagnosis, and vigilant surveillance systems including Sentinel General Practice influenza surveillance and influenza-like illness surveillance will be of prime importance in the coming southern hemisphere winter season.

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

Dr Virginia Hope and Dr Sue Huang, ESR NCBID

2. Notifiable Disease Surveillance

The following is a summary of disease notifications for the January – March quarter of 2009 and cumulative notifications and rates calculated for a 12-month period (April 2008 – March 2009). 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 8 April 2009. As this information may be updated over time, these data should be regarded as provisional.

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

VACCINE PREVENTABLE DISEASE

Hepatitis B

- *Notifications:* 14 notifications in the quarter (2008, 13); 39 notifications over the last 12 months (2008, 64) giving a rate of 0.9 cases per 100,000 population (2008, 1.5); a statistically significant decrease
- *Comments:* cases were aged between 20 and 74 years, with no cases under the age of 16 years

Haemophilus influenzae type b

- *Notifications:* 1 notification in the quarter (2008, 5); 5 notifications over the last 12 months (2008, 14) giving a rate of 0.1 cases per 100,000 population (2008, 0.3); a statistically significant decrease
- *Comments:* the case was aged 62 years

Invasive Pneumococcal Disease

- *Notifications:* 125 notifications in the quarter
- *Comments:* cases were aged between 2 months and 92 years, with 7 cases under the age of 2 years
- *Note:* Invasive pneumococcal disease became notifiable on 17 October 2008, therefore comparisons between quarters and 12-month rates are not valid

Measles

- *Notifications:* 28 notifications in the quarter (2008, 0); 40 notifications in the last 12 months (2008, 19) giving a rate of 0.9 cases per 100,000 population (2008, 0.4); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (3 cases) and from the same quarter last year (0 cases); 12 cases were laboratory confirmed

Mumps

- *Notifications:* 9 notifications in the quarter (2008, 29); 56 notifications over the last 12 months (2008, 82) giving a rate of 1.3 cases per 100,000 population (2008, 1.9); a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly decrease from the same quarter last year (29 cases); 2 cases were laboratory confirmed

Pertussis

- *Notifications:* 338 notifications in the quarter (2008, 67); 688 notifications over the last 12 months (2008, 294) giving a rate of 16.1 cases per 100,000 population (2008, 7.0); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (181 cases) and from the same quarter last year (67 cases)

ENTERIC INFECTIONS

Campylobacteriosis

- *Notifications:* 1,895 notifications in the quarter (2008, 1,760); 6,828 notifications over the last 12 months (2008, 9,894) giving a rate of 159.9 cases per 100,000 population (2008, 234.0); a statistically significant decrease

- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (2,349 cases) and a statistically significant quarterly increase from the same quarter last year (1,760 cases)

Gastroenteritis

- *Notifications:* 134 notifications in the quarter (2008, 179); 645 notifications over the last 12 months (2008, 629) giving a rate of 15.1 cases per 100,000 population (2008, 14.9); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (252 cases) and from the same quarter last year (179 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:* 442 notifications in the quarter (2008, 492); 1,296 notifications over the last 12 months (2008, 1,392) giving a rate of 30.4 cases per 100,000 population (2008, 32.9); not a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (337 cases)

Shigellosis

- *Notifications:* 42 notifications in the quarter (2008, 25); 130 notifications over the last 12 months (2008, 128) giving a rate of 3.0 cases per 100,000 population (2008, 3.0); no change
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (23 cases) and from the same quarter last year (25 cases)

Typhoid Fever

- *Notifications:* 20 notifications in the quarter (2008, 9); 40 notifications over the last 12 months (2008, 34) giving a rate of 0.9 cases per 100,000 population (2008, 0.8); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (5 cases) and from the same quarter last year (9 cases)

VTEC Infections

- *Notifications:* 78 notifications in the quarter (2008, 49); 156 notifications over the last 12 months (2008, 118) giving a rate of 3.7 cases per 100,000 population (2008, 2.8); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (24 cases) and from the same quarter last year (49 cases)

ENVIRONMENTAL EXPOSURES & INFECTIONS

Chemical Poisoning

- *Notifications:* 1 notification in the quarter (2008, 0); 2 notifications over the last 12 months (2008, 11) giving a rate of 0.0 cases per 100,000 population (2008, 0.3); a statistically significant decrease

Cryptosporidiosis

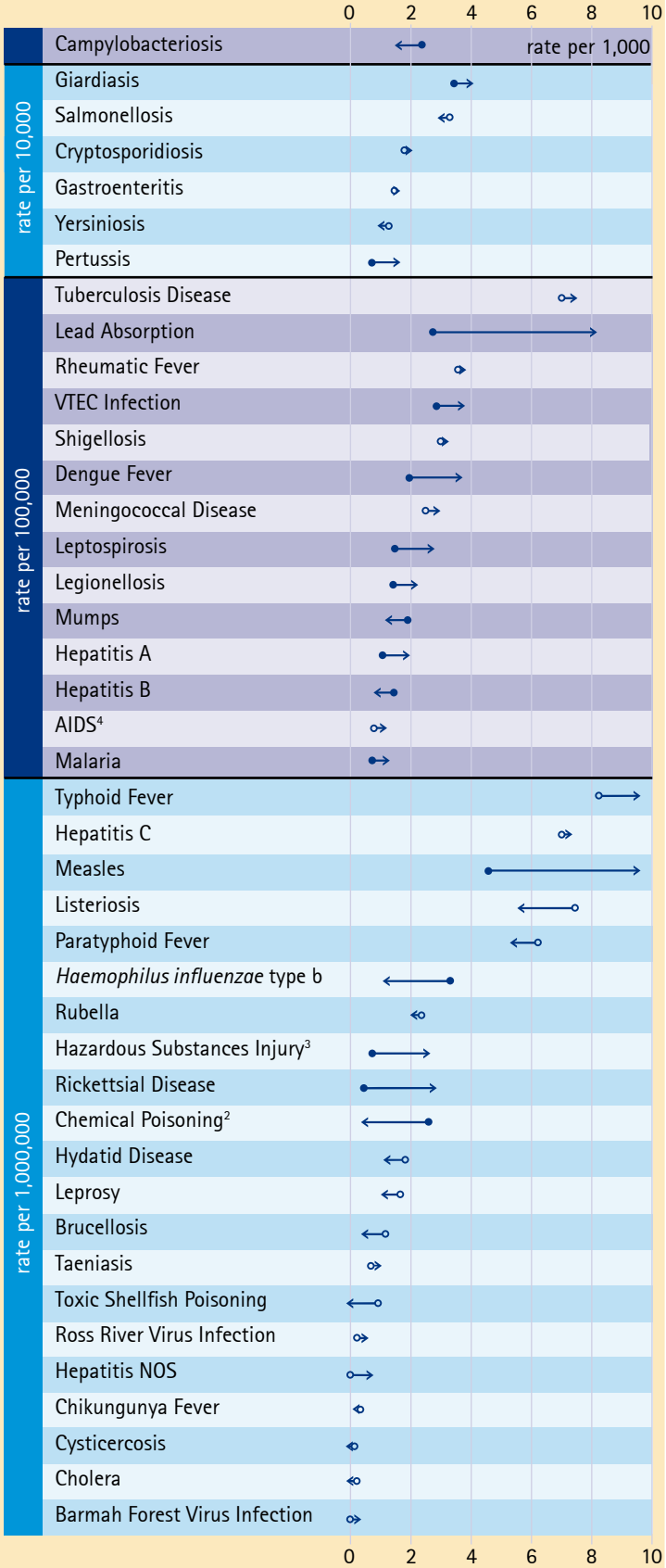
- *Notifications:* 132 notifications in the quarter (2008, 83); 813 notifications over the last 12 months (2008, 794) giving a rate of 19.0 cases per 100,000 population (2008, 18.8); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (314 cases) and a statistically significant increase from the same quarter last year (83 cases)

Giardiasis

- *Notifications:* 474 notifications in the quarter (2008, 417); 1,717 notifications over the last 12 months (2008, 1,415) giving a rate of 40.2 cases per 100,000 population (2008, 33.5); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (388 cases)

National Surveillance Data

12-Monthly Notification Rate Changes[1]



Notifications per 1,000 or 10,000 or 100,000 or 1,000,000 persons

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 April 2008 – March 2009 and compared to previous 12-month rates

[2] From the environment

[3] Hazardous Substance Injury became notifiable in EpiSurv as of 19 September 2007

[4] 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

Hazardous Substances Injury

- *Notifications:* 3 notifications in the quarter (2008, 0); 11 notifications over the last 12 months (2008, 3) giving a rate of 0.3 cases per 100,000 population (2008, 0.1); a statistically significant increase

Hepatitis A

- *Notifications:* 10 notifications in the quarter (2008, 18); 81 notifications over the last 12 months (2008, 43) giving a rate of 1.9 cases per 100,000 population (2008, 1.0); a statistically significant increase
- *Comments:* cases were aged between 7 and 56 years, with 4 cases under the age of 16 years

Lead Absorption

- *Notifications:* 81 notifications in the quarter (2008, 59); 339 notifications over the last 12 months (2008, 113) giving a rate of 7.9 cases per 100,000 population (2008, 2.7); a statistically significant increase
- *Comments:* cases were distributed by age as follows: 1 (1-4 years), 1 (5-14 years), 12 (15-24 years), 31 (25-44 years), 27 (45-64 years), and 9 (65 years and over); there were 69 male cases, 9 female cases, and 3 of unknown sex; 45 cases recorded an occupation that involved exposure to lead: foundry worker (8), painter (6), radiator repairer (3), automotive electrician (1), demolition contractor (1), gunsmith (1), labourer (1), lead worker (1), recycling pick up driver (1), scrap metal dealer (1), student (1), and 20 cases were not specified
- *Note:* since June 2007 the blood lead level for reporting has lowered from 0.72 to 0.48 µmol/l

Legionellosis

- *Notifications:* 32 notifications in the quarter (2008, 16); 89 notifications over the last 12 months (2008, 58) giving a rate of 2.1 cases per 100,000 population (2008, 1.4); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (16 cases)

Leptospirosis

- *Notifications:* 19 notifications in the quarter (2008, 22); 117 notifications over the last 12 months (2008, 61) giving a rate of 2.7 cases per 100,000 population (2008, 1.4); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (37 cases). There were 18 male cases, and 1 female case; 8 farmers, 5 meat process/freezing workers, and a shepherd, civil engineer, sharemilker, shearer, tourist and a white-water rafting guide (each 1 case)

Yersiniosis

- *Notifications:* 165 notifications in the quarter (2008, 181); 493 notifications over the last 12 months (2008, 547) giving a rate of 11.5 cases per 100,000 population (2008, 12.9), not a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (105 cases)

NEW, EXOTIC & IMPORTED INFECTIONS

Dengue Fever

- *Notifications:* 70 notifications in the quarter (2008, 31); 152 notifications over the last 12 months (2008, 83) giving a rate of 3.6 cases per 100,000 population (2008, 2.0); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (33 cases) and from the same quarter last year (31 cases); 62 cases were laboratory confirmed; 67 cases were overseas during the incubation period and the travel history of 3 cases was unknown. Places visited were Tonga (31), Fiji (15), Samoa (9), Vanuatu (5), Australia and Timor-Leste (1), Burma (1), Cook Islands (1), India (1), Laos (1), and Papua New Guinea (1)

Malaria

- *Notifications:* 18 notifications in the quarter (2008, 8); 50 notifications over the last 12 months (2008, 31) giving a rate of 1.2 cases per 100,000 population (2008, 0.7); a statistically significant increase
- *Comments:* 16 cases had malaria parasites in a blood film; all cases were overseas during the incubation period

Rickettsial Disease

- *Notifications:* 2 notifications in the quarter (2008, 0); 12 notifications over the last 12 months (2008, 2) giving a rate of 0.3 cases per 100,000 population (2008, 0.0); a statistically significant increase

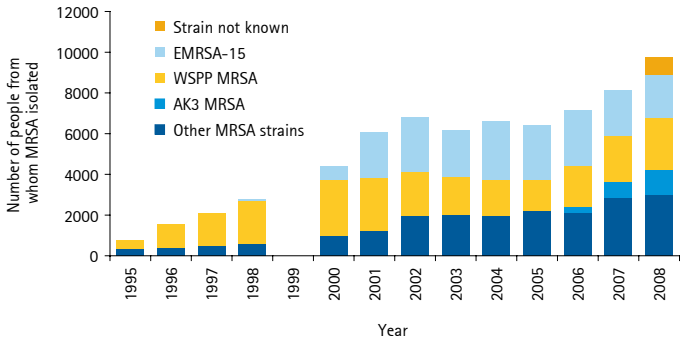
3. Other Surveillance Reports

Annual survey of MRSA, August 2008

ESR conducts annual one-month surveys of methicillin-resistant *Staphylococcus aureus* (MRSA) to provide information on the epidemiology of MRSA in New Zealand. Hospital and community microbiology laboratories were asked to refer all MRSA they isolated during August 2008 to ESR. During the month, MRSA were referred from 736 people (706 patients and 30 staff) (Figure 1). In addition, one hospital laboratory, which was unable to refer isolates for the survey, reported that they isolated MRSA from 73 people during the month. These 73 MRSA isolations have been included in the data presented in Figure 1, and when calculating the national and district health board (DHB) MRSA incidence rates. All other analyses included in this report are based only on the MRSA isolates referred to ESR for the survey.

The annualised incidence of MRSA in 2008 was estimated at 227.4 MRSA per 100,000 population – an 18.8% increase on the 2007 rate of 191.5.

Figure 1. MRSA isolations, 1995–2008[a]

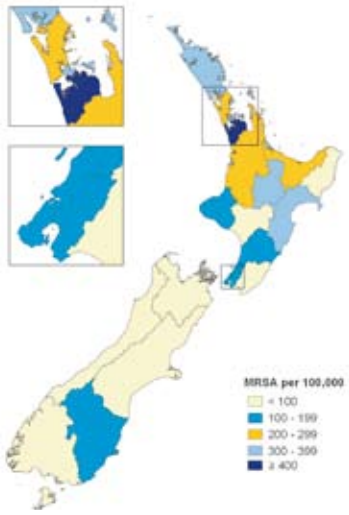


[a] Data for 1995 to 1998 are based on continuous surveillance of all MRSA isolations. Data for 2000 to 2008 are annualised and based on one-month surveys conducted in these years. No survey was undertaken in 1999. The category 'Strain not known' for 2008 represents the number of people identified with MRSA by the laboratory which could not refer isolates.

There continue to be marked geographic variations in the incidence of MRSA in New Zealand (Figure 2). Differences in screening policies may contribute to some of the differences in incidence between DHBs. Since 2003, there have been significant ($P<0.05$) increases in the incidence of MRSA in Northland, the combined Waitemata/Auckland/Counties Manukau, Waikato, Lakes, Bay of Plenty, Taranaki, Hawke's Bay, MidCentral, Capital and Coast, Canterbury, and Otago DHBs.

Seven MRSA strains were predominant in 2008 and represented 83.2% of all MRSA isolations. As has been the situation for the last eight years, the two most commonly identified MRSA strains were the WSPP MRSA strain, which accounted for 31.5% of isolates, and the EMRSA-15 strain which accounted for 25.8% of isolates (Figure 1). The prevalence of other strains was: AK3 MRSA strain,15.1%; WR/AK1 MRSA strain, 8.9%; USA300 MRSA strain, 5.0%; AKh4 MRSA strain, 2.1%; and the Queensland MRSA clone, 2.1%. For a description of these MRSA strains, including their typical antimicrobial susceptibility patterns, follow the link to the report below.

Figure 2. Annualised incidence of MRSA by district health board, 2008



MRSA was reported as causing infection in 82.5% of the 639 patients for whom this information was provided. Among the 706 patients with MRSA, 38% were categorised as hospital patients and 62% as community patients. Patients were classified as hospital patients if they were in a healthcare facility (including residential-care facility) when MRSA was isolated or had been in a healthcare facility in the three months before MRSA was isolated. The proportion of hospital patients is likely to be an underestimate due to it not including data from the hospital laboratory which could not refer isolates. The majority of EMRSA-15 and AKh4 MRSA (73% and 86%, respectively) were isolated from hospital patients or staff, whereas most WSPP MRSA, AK3 MRSA, WR/AK1 MRSA, USA300 MRSA and Queensland MRSA clone (78%, 81%, 77%, 64% and 86%, respectively) were isolated from people in the community.

For a more detailed report see www.surv.esr.cri.nz/PDF_surveillance/Antimicrobial/MRSA_2/aMRSA_2008.pdf.

Reported by Helen Heffernan, Communicable Disease Programme, ESR

4. Outbreak Surveillance

The following information is a summary of the outbreak trends for New Zealand, from data collected in the last quarter (January – March 2009). Comparisons are made to the previous quarter (October – December 2008), and to the same quarter in the previous year (January – March 2008). Note that the outbreak data in this section are notified to ESR by the Public Health Services.

General

- 99 outbreaks notified in this quarter (783 cases)
- 44 are 'final' reports (500 cases); 55 are 'interim' reports (283 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 14.8 cases per outbreak in the previous quarter (14.9 cases per outbreak in the same quarter of last year)
- 3 hospitalisations: *Salmonella* Typhi
- 1 death: norovirus

Pathogens

- 11 norovirus outbreaks (339 cases) during this quarter
- 10 'gastroenteritis' outbreaks (69 cases)
- 7 *Giardia* outbreaks (25 cases)
- 6 *Bordetella pertussis* outbreaks (19 cases)
- 4 rotavirus outbreaks (23 cases)
- 2 *Cryptosporidium* outbreaks (7 cases)
- 1 *Enterobacter spp.* outbreak (8 cases)
- 1 Hepatitis A outbreak (2 cases)
- 1 *Salmonella* Typhi outbreak (4 cases)
- 1 *Shigella spp.* outbreak (4 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.

- 35 person-to-person, from (non-sexual) contact with an infected person (including droplets): 10 norovirus (315 cases), 6 *Giardia* (23 cases), 6 *B. pertussis* (19 cases), 5 gastroenteritis (58 cases), 4 rotavirus (23 cases), 1 *Cryptosporidium* (4 cases), 1 *Enterobacter* (8 cases), 1 Hepatitis A (2 cases), and 1 *Shigella* (4 cases)
- 10 environmental, from contact with an environmental source (e.g. swimming): 3 *Giardia* (10 cases), 3 norovirus (122 cases), 2 *Cryptosporidium* (7 cases), and 2 gastroenteritis (15 cases)
- 5 waterborne, from consumption of contaminated drinking water: 4 *Giardia* (15 cases), and 1 *Cryptosporidium* (3 cases)
- 3 foodborne, from consumption of contaminated food or drink (excluding water): 3 gastroenteritis (6 cases)
- 1 zoonotic: *Giardia* (6 cases)
- 2 'other' mode of transmission: 1 gastroenteritis (29 cases), and 1 norovirus (24 cases)
- 5 'unknown' mode of transmission: 3 gastroenteritis (7 cases), 1 norovirus (24 cases), and 1 *S.* Typhi (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.

- 15 home: 5 *Giardia* (18 cases), 3 *B. pertussis* (9 cases), 2 norovirus (44 cases), 1 *Enterobacter* (8 cases), 1 *Cryptosporidium* (3 cases), 1 rotavirus (3 cases), 1 Hepatitis A (2 cases), and 1 gastroenteritis (2 cases)
- 8 rest home: 7 norovirus (269 cases), and 1 gastroenteritis (13 cases)
- 7 childcare: 3 rotavirus (20 cases), 1 norovirus (38 cases), 1 gastroenteritis (29 cases) 1 *Giardia* (5 cases), and 1 *B. pertussis* (2 cases)
- 5 café: 5 gastroenteritis (10 cases)
- 4 hospital (continuing care): 2 norovirus (59 cases), and 2 gastroenteritis (14 cases)
- 2 camp: 2 *Giardia* (6 cases)
- 2 community: 1 *B. pertussis* (6 cases), and 1 *S.* Typhi (4 cases)
- 1 'other' food outlet: norovirus (6 cases)
- 1 farm: *Giardia* (6 cases)
- 1 school: *B. pertussis* (3 cases)
- 1 swimming/spa pool: *Cryptosporidium* (3 cases)
- 1 takeaway: norovirus (2 cases)
- 2 'other setting': 1 *Enterobacter* (8 cases), and 1 *Giardia* (3 cases)
- 4 outbreaks with no setting selected: 1 *B. pertussis* (2 cases), 1 *Cryptosporidium* (4 cases), 1 gastroenteritis (3 cases), and 1 *Shigella spp.* (4 cases)

5. Outbreak Case Reports

An Outbreak of *Clostridium* gastroenteritis at a boarding house

On 29 October 2008, Auckland Regional Public Health Service (ARPHS) was notified by the manager of a boarding house that approximately 100 out of 180 of their secondary school students were unwell with abdominal pain and/or diarrhoea. Two assemblies were set up at the boarding house to undertake face to face interviews about food consumption, symptoms and timing of illness. Within 30 hours of the initial notification, 172 questionnaires were completed, a response rate of over 90%. Leftover food samples preceding the illness and faecal samples from symptomatic cases were collected for microbiological analyses. A Hazard Analysis Critical Control Point (HACCP) assessment of the caterer was undertaken on 30 October 2008.

The initial notification suggested a point source outbreak. A total of 120 out of 172 interviewees (70%) met the case definition of having a gastroenteritis illness and had consumed food at the boarding house on 27 and 28 October 2008. The narrow range of illness onset time among cases (standard deviation of seven hours) suggested the outbreak had a low secondary attack rate. The duration of illness was relatively short with more than two-thirds of all cases recovering within 24 hours of symptom onset. Based on multiple logistic regression analyses, consumption of the seafood dish at dinner time on 28 October was independently associated with illness with an odds ratio of 111. The median incubation period was 12 hours after the suspected implicated seafood dish was consumed.

Microbiological analyses and case investigation supported *Clostridium perfringens* and *Bacillus* species as possible causes of the outbreak. However, *C. perfringens* was the most likely primary microbiological aetiology given that it was found at very high levels at >2.5 x 10^6 colony forming units/gram (CFU/g) in the seafood dish and *C. perfringens* at 2.0 x 10^6 CFU/g and *C. perfringens* enterotoxin were also isolated in the faecal sample of an affected case. *Bacillus cereus* was also isolated from the seafood dish. However, it was detected at a level (1.9 x 10^3 CFU/g) much lower than the level (>1.0 x 10^5 CFU/g) usually associated with illness. Nevertheless, a co-infection with *Bacillus* sp. could not be excluded, as *Bacillus* sp. and enterotoxin were also isolated from the same faecal specimen from which *C. perfringens* had been detected.

Optimum growth of *C. perfringens* (vegetative cells) occurs at temperatures of 43-47°C, with a generation time of 7.1 minutes.[1] *Clostridium perfringens* is a ubiquitous organism in the environment and there are usually many opportunities for the spores of *C. perfringens* to contaminate food.[2,3] Heating foods to 70-80°C readily destroys vegetative cells but heat resistant spores can survive the initial cooking process. Moreover, the cooking process can germinate spores into vegetative cells and if the cooked food is left to cool slowly, then the resulting vegetative cells can rapidly multiply to high levels.[1,4] As time and temperature were not monitored during preparation of the dish (which involved multiple cooking and hold holding steps), maintenance of adequate internal temperature could not be guaranteed. Furthermore, the practice of leaving "saved" dinner at room temperature could have facilitated rapid growth of any vegetative cells present.

Clostridium perfringens related gastroenteritis outbreaks are often associated with institutions or catering establishments which produce large amounts of food.[5-7] Cooking foods in large quantities can lead to poor heat penetration resulting in pockets of food being sub-optimally heated.[8] The seafood mixture was cooked in a 40L pot which, according to the caterer, had been infrequently stirred (stirring prevents pockets of inadequately heated food as well as providing some aeration). *Clostridium perfringens* is an anaerobic bacteria. The liquor mass of food provided an ideal anaerobic environment that would have been enhanced by any dissolved oxygen being driven off by the heating process.[8]

Clostridium related gastroenteritis outbreaks are often under reported. It has been estimated only 1-5% of all cases are reported.[6] The prompt epidemiological and microbiological analyses provided confirmation of this outbreak's aetiology, which allowed appropriate measures to be implemented to prevent a recurrence. The response rate in this outbreak investigation was remarkably high (>90%) compared to a previous gastroenteritis outbreak investigation (27%) involving a boarding house in March 2008. The good working relationship with the boarding house manager and the undertaking of face to face interviews in two pre-organised assemblies were identified as factors contributing to the success of this outbreak investigation.

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

Reported by Wing Cheuk Chan, Public Health Medicine Registrar; Jenny Wong, Food Act Officer; Shikha David, Health Protection Officer; Corina Grey, Public Health Medicine Registrar; and Richard Hoskins, Medical Officer of Health; Auckland Regional Public Health Service

An outbreak of norovirus at a large New Zealand university

A doctor from the university medical centre notified the local Public Health Service (PHS) of the outbreak at 9:30am on 31 October 2008. An outbreak team was formed including the Health and Safety Manager and the Hostel Manager from the university, and three Health Protection Officers and the Medical Officer of Health from the PHS. The team maintained regular contact with the university medical centre. It was exam week at the university.

Infection control measures were introduced with the aim of swiftly containing the outbreak and reducing the risk of spread to the local community including:

(1) Signage on the doors of hostels limiting access.

(2) Regular communication to all campus users regarding outbreak management and educational messages around hand hygiene through the university's intranet/email.

(3) Discouraging students from affected hostels entering the common dining hall by delivering pre-packaged meals to the hostel common rooms.

(4) Regular delivery of bottled water, and electrolytes to cases.

(5) Disposable vomit bags for cases and biohazard bins outside each hostel.

(6) The campus medical centre ran a dedicated medical clinic twice a day for assessment of gastroenteritis symptoms in rooms near the hostels (to discourage attendance at the university medical centre or use of off campus medical services).

(7) Provision of hand sanitiser at entrances and exits around the University.

(8) Education of contract cleaners, hostel and catering staff.

(9) Increasing campus cleaning from once to twice daily.

(10) Review of sickness policy for catering staff with emphasis on the importance of not attending work for 48 hours after suffering from diarrhoea or vomiting.

(11) Audit of hand hygiene facilities and procedures for all catering staff.

(12) Identification of areas requiring terminal cleaning prior to the summer school commencing.

(13) Allowing students to apply for aegrotat/impaired performance rather than attend exams if symptomatic.

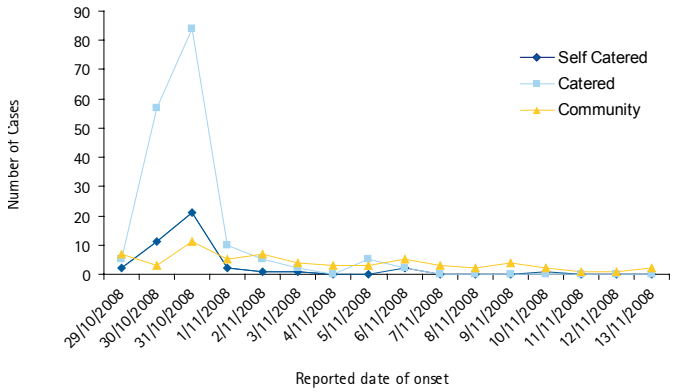
(14) Separate exam hall for those with recent symptoms who still wished to proceed with examination.

Case logs were maintained in each hostel which included the name of the hostel, and the onset and duration of illness for each case. Questionnaires were issued to hostel residents by their hall manager. Additional questionnaires and a case log were held at the health and safety office for students and staff who did not reside on campus. Case logs and questionnaires were forwarded to the PHS on a daily basis. The outbreak appeared to be centred in the catered hostels on campus.

A total of 288 cases were identified during the outbreak investigation, six of whom were laboratory confirmed with norovirus (NV) infection by ESR (including one food handler). The cases comprised of people from catered hostels (66%), self–catered hostels (13%), and those who work or study on campus but resided in the local community (21%). The epidemic curve

for each of these groups is shown in Figure 3. The attack rate for the total hostel population was 27%. However, the attack rate for those in catered hostels was 36% compared with 18% in the self-catered halls.

Figure 3. Disease onset between catered & self-catered hostels compared to reported cases who lived off campus



The likelihood of a point source outbreak associated with the food court was further supported by an inspection and interview undertaken with the commercial caterers for the campus who identified a number of symptomatic food handlers who had experienced onset of illness while at work and had to be sent home. Two of the food handlers had onsets of 29 October. A third symptomatic food handler at another campus site had a similar onset time on 29 October. This food handler had not eaten food from the main kitchens but was involved in unpacking food from there on the day prior to their illness.

Epidemiological findings support a point source outbreak that was common to a large number of people. The investigation findings support a hypothesis that the most likely source was food, or a common fomite or contaminated surface(s) within the large commercial food court on campus. Students living in catered hostels have three meals a day provided at the food court and the same premises is the main supplier of food to others at the university.

Concurrent with this outbreak the PHS investigated two other NV outbreaks in rest home settings. The NV typing results from all three outbreaks were indistinguishable from each other. This finding supports the theory that circulation of NV in the community is manifested in institutional outbreaks.

This outbreak serves as a reminder of the importance of good food hygiene (especially hand washing) and robust staff sickness policies, and the virulence of NV. Given the nature of the university environment, this outbreak is likely to have entered the local community. However, we believe the impact was significantly reduced by effective community education and the swift implementation of infection control procedures adapted from the national guidelines and tailored to this unique situation.

Reported by Tui Shadbolt, Coordinator Health Protection; Jill McKenzie, Medical Officer of Health; Brett Munro and Chris Bland, Health Protection Officers; MidCentral Public Health Service

Typhoid fever outbreak in Porirua

In December 2008, 80 members of a youth group from a Pacific Island church in Porirua visited Samoa and Tokelau. The entire group lived in Porirua and all were of the same Pacific ethnic origin. The church Minister warned them that there was an epidemic of typhoid fever in Samoa but the group decided to travel there with most of them travelling over on the same flight. The group stayed in the same church hall accommodation in Samoa and in villagers' homes in Tokelau.

On 29 December 2008 Regional Public Health (RPH) was notified that one youth (Case A), who had returned to New Zealand, was in hospital with confirmed typhoid fever. The onset of illness in this case indicated that they could have been infected in either Samoa or Tokelau. On 6 January 2009 RPH was notified of another confirmed case (Case B), but an adult helper, who had also travelled to Samoa with the group and stayed in the same hall. This case's incubation period suggested that she had become infected in Samoa, and not in Tokelau. Then on 27 January a third case (Case C) was notified who was also part of the youth trip but an adult helper. She could not be laboratory confirmed because by the time a diagnostic faecal specimen was taken she had already started a course of ciprofloxacin and so, as expected, *Salmonella* Typhi was not detected in her specimen. Her clinical picture fitted that of typhoid fever, and infection in Samoa.

A fourth case (Case D), also eventually linked to the youth trip, was notified on 4 February but as a case of Barmah Forest virus infection (BFVI) based on an initial equivocal serological result which later raised to IgM reactive status. However, this youth's only recent travel to Australia had been a trip to Sydney for four days in mid September 2008 and had never had symptoms of BFVI. She was hospitalised soon after returning to New Zealand from the youth group trip and her consultant considered she had been infected with typhoid fever in spite of the initial BFVI serology result. This was also the opinion of RPH, as her symptoms were typical of typhoid fever and included the relatively rare "rose rash" on the torso. The serology was eventually confirmed in Australia as BFVI IgG positive, but the IgG value suggested past infection with BFVI or another closely related alphavirus. IgM was negative.

Medical centres in Porirua were faxed alerts to raise awareness that patients might seek attention and be showing symptoms of typhoid fever. In addition, a letter providing details of symptoms of typhoid fever was sent to the Minister of the church and circulated to all members of the youth group who had travelled to Samoa and Tokelau. This additional case finding did not reveal any other cases.

Whilst the Cases A to D were being investigated, two other cases (E and F) of typhoid fever were notified for Porirua. Both were adult Pacific Islanders who had no connection with the church group, and were of different ethnicities to the group. Case E was infected in Samoa and Case F in Fiji or Tuvalu.

Isolates from Cases A, B, E, and F were typed by ESR as all being *S.* Typhi phage type E1a, a common type from the Pacific Islands (pers com. M Dufour, ESR). There were no obvious sources for any of these infections but the outbreak cases drank only bottled water and so a contaminated food(s) is most likely. In addition, there does not appear to have been personal contact between the case with the earliest onset date (B) and the other youth group trip associated cases (A, C, D).

None of the cases had been vaccinated against typhoid fever and they probably received no travel medicine advice from a medical practitioner. Slinko et al. note that "a common purpose for overseas travel by foreign-born Australian residents and their children is "visiting friends and relatives" (VFR), usually in the country of origin. It has been noted in other developed countries that VFR travellers account for a disproportionate amount of illness from malaria, hepatitis A and typhoid on return to the country of residence".[1] This puts a burden on public health services to investigate and control these introduced diseases. RPH Public Health Nurses also noted an increase in skin infections in Porirua this year and amongst VFR travellers.

The outbreak of typhoid fever and associated non-outbreak cases was a burden to RPH resources to follow up at a busy time of the year when many staff were on leave. I reiterate Slinko et al.'s point that vaccination and health education must be combined to ensure adequate protection for VFR and other travellers. Such primary health care needs to be both accessible and affordable for population groups living in more socio-economically deprived communities as is often the case for Polynesians living in Porirua.

References

1. Slinko V, Jarvinen K, Beard F, and McCall B 2008. Notifications of enteric diseases in returning travellers who visit friends and relatives overseas: A call to action. *Communicable Diseases Intelligence* 32(3): 333-4.

Reported by Quentin Ruscoe, Health Protection Officer, Regional Public Health, Hutt Valley DHB

6. Laboratory Surveillance

Editor's note. The format of the Pathogen Surveillance section is being altered to allow ESR's various infectious disease laboratories to provide an analysis and summary of their data, usually on an annual basis. We hope to provide the readership with more useful information from the laboratory-based surveillance of infectious disease being performed at ESR. The title of the section will change to Laboratory Surveillance.

Influenza in New Zealand in 2008

During the 2008 winter season, 3,945 consultations for influenza-like illness (ILI) were reported from a national sentinel network of 85 general practices. It is estimated that ILI resulting in a visit to a general practitioner affected over 47,697 New Zealanders (1.2% of total population) during the season, compared with an estimated 32,771 in 2007. Compared with the 1997-2007 period, the influenza activity in 2008 is described as moderate with two peaks, one in the middle of July and another in the middle of August. The ILI consultation rates varied greatly among Health Districts with the highest rates being reported from Northland and Eastern Bay of Plenty Health Districts. In 2008, a total of 1,054 influenza viruses were identified, higher than 744 in 2007 and 768 in 2006. Influenza A(H3N2) viruses predominated in June/July while influenza B predominated in August/September.

Influenza B

During the period 1990-2008, influenza B viruses predominated for five years; 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 1980's. 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/Malaysia/2504/2004). For reasons not wholly understood, the B/Victoria/2/87 lineage viruses remained geographically restricted to Asia until 2001. During 1990-2001, B/Yamagata lineage viruses circulated exclusively in New Zealand. For the first time in 2002, the B/Victoria lineage viruses spread to New Zealand and completely replaced B/Shanghai lineage virus. Since 2003, the two lineages have been co-circulating in New Zealand with the B/Victoria lineage predominating every three years in 2005 and 2008. The influenza B was associated with high disease burden in young children and the B/Victoria lineage viruses tended to be associated with more explosive school outbreaks than the B/Yamagata lineage viruses.

In 2008, the majority of influenza viruses (58.3%, 462/793 of typed and subtyped viruses) were characterised as influenza B and represented 59.8% (630/1,054) of the total viruses. Influenza B/Victoria lineage viruses (most representative strain-B/Malaysia/2506/2004-like) were the predominant lineage viruses representing 44.6% (354/793) of typed and subtyped viruses. Influenza B/Yamagata lineage viruses (most representative strain-B/Florida/4/2006-like) represented 13.6% (108/793) of typed and subtyped viruses.

Influenza A(H3N2)

Influenza A(H3N2) viruses have often been associated with more severe disease and with excess pneumonia and influenza mortality. During 1990-2008, 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 A(H3N2) epidemic. The highest hospitalisations (591) were recorded in 2003 due to a A(H3N2) predominant season.

In 2008, influenza A(H3N2) represented 41.0% (325/793) of the typed and subtyped viruses and 30.8% (325/1,054) of the total viruses; 215 viruses were antigenically subtyped as A/Brisbane/10/2007 (H3N2) - like viruses.

Influenza A(H1N1)

During the period 1990-2008, influenza A(H1N1) viruses predominated for three seasons in 1992 (85.7%), 2000 (36.0%), and 2001 (54.4%). Influenza A(H1N1) viruses circulated in significant proportion in 2007 (32.1%). In 2008, only six A(H1N1) viruses (0.8%) were detected, of which, only four were available for antiviral susceptibility testing and were all resistant to oseltamivir. The results of the fluorometric neuraminidase inhibition assay indicated that the four viruses had highly reduced sensitivity to oseltamivir with IC50 values in the range of 500-1700 nM, typical of the recently global emerging oseltamivir-resistant A(H1N1) viruses. Genetic analysis of the neuraminidase gene confirmed that the four viruses had the H274Y mutation (histidine-to-tyrosine at codon 274 in N2 nomenclature), conferring resistance to oseltamivir. These four viruses were isolated from a two month old male infant (1), a 15 year old female (1), and a 49 year old female (2). None of the patients or their close contacts had received Tamiflu prior to sample collection. The WHO National Influenza Centre has reported the findings to the WHO. The summary report can be found at www.who.int/csr/disease/influenza/NICsummaryreport_NZ_2008.pdf.

The global emergence and rapid spread of oseltamivir-resistant influenza A(H1N1) viruses carrying a neuraminidase gene with an H274Y amino acid substitution has been observed since January 2008. Previously, viruses carrying this mutation are presumed to exhibit attenuated pathogenicity[1], compromised transmission[2], and reduced lethality[3]. However, current widespread circulation of oseltamivir-resistant influenza A(H1N1) viruses associated with typical influenza illnesses and viral pneumonia suggest that these viruses retain significant transmissibility and pathogenicity.[4,5] Preliminary data indicate that oseltamivir-resistant influenza A(H1N1) viruses do not cause different or more severe symptoms compared to oseltamivir-sensitive influenza A(H1N1) viruses.[6,7] For the first time since 2008, these oseltamivir-resistant influenza A(H1N1) viruses have spread to New Zealand. These oseltamivir-resistant influenza A(H1N1) viruses pose challenges for the selection of antiviral medications for treatment and chemoprophylaxis of influenza. CDC has issued interim recommendations.[8] It has become increasingly important to establish and sustain a national antiviral monitoring program in New Zealand which would provide timely local surveillance information to assist clinicians in choosing appropriate antiviral agents for their patients. It also provides additional reasons for clinicians to test patients for influenza virus infection in order to select appropriate antiviral medications. So far, these oseltamivir-resistant influenza A(H1N1) viruses are antigenically similar to the influenza A/Brisbane/59/2007 (H1N1)-like strain represented in the New Zealand vaccine for 2009, and vaccination should continue to be considered the primary prevention strategy regardless of oseltamivir sensitivity.

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

Reported by Sue Huang, ESR NCBID

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

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