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New Zealand Public Health Surveillance Report

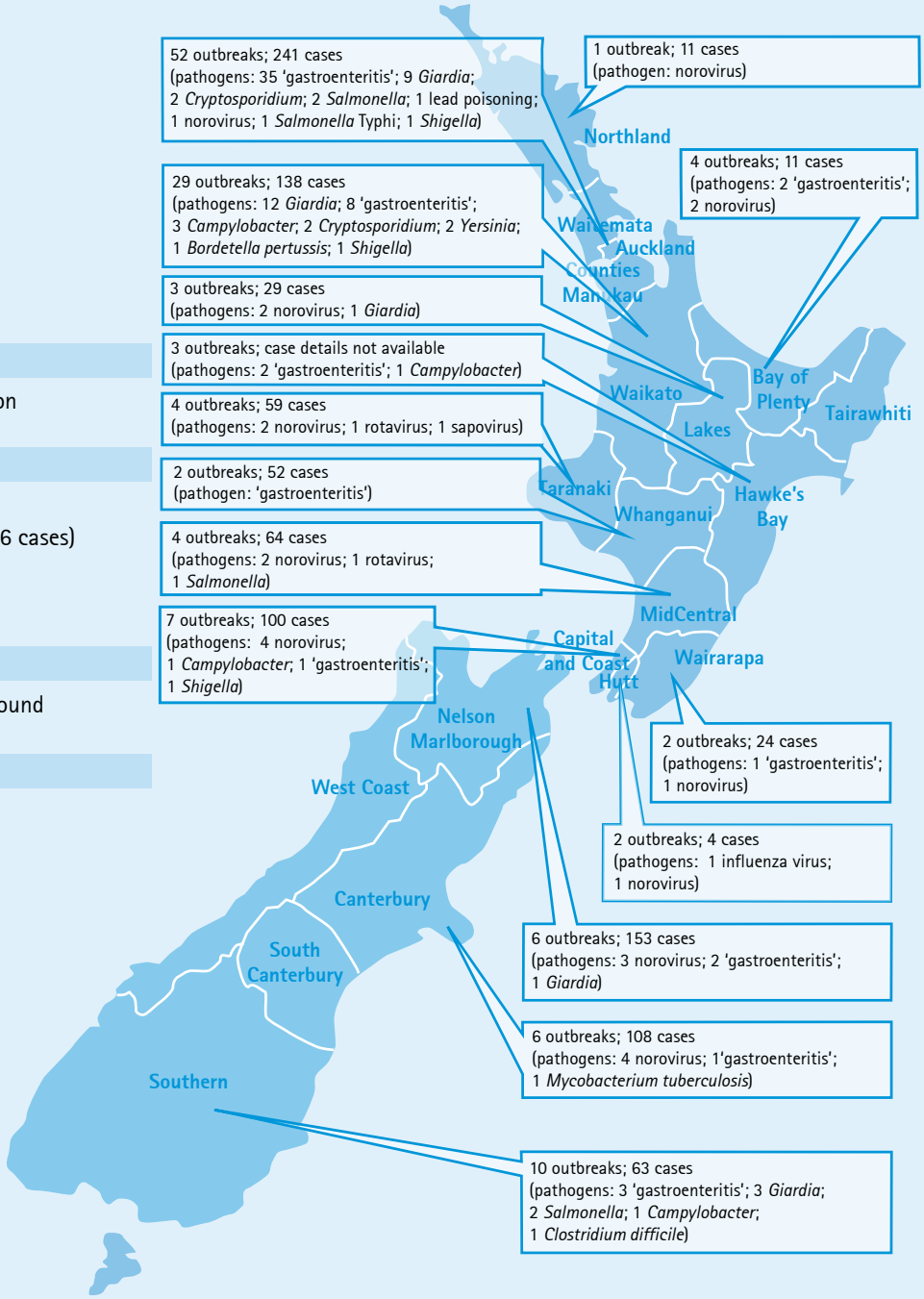
September 2012: Covering April to June 2012

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 2012. The outbreak map on this page consists of all outbreak information, final and interim. The total number of outbreaks and cases by region and outbreak pathogen are reported, as notified up to 4 July 2012. Outbreaks reporting exposures in more than one geographic location are assigned to the District Health Board with the most cases.



1. Editorials

Changes in the *Bordetella pertussis* population: Are they vaccine driven?

The causative organism of pertussis (whooping cough), the bacterium *Bordetella pertussis*, is endemic in many developed countries and, despite high rates of vaccination, increasing numbers of people infected with pertussis are regularly observed. New Zealand is currently in the throes of an epidemic with a significant increase in notifications from September 2011.[1] It has been suggested that the increase in infections in developed countries could be due to waning protective immunity in adolescents and adults, or changes in the population structure of *B. pertussis*.[2]

Typing of *B. pertussis* is particularly challenging as the organism is genetically homogeneous with little diversity, having only recently emerged as a human pathogen (0.3 to 2.5 million years ago).[3] A number of molecular methods have been applied including multilocus variable number of tandem repeat analysis (MLVA), which looks at differences in the number of short DNA repeats at several loci,[4–6] single nucleotide polymorphism analysis (SNP) looking at single point mutations throughout the genome,[7] and sequence typing looking at variations in the sequence of surface proteins and virulence factors. All these methods have identified variations in the *B. pertussis* genome and have been used to describe changes in the *B. pertussis* population structure.

A number of studies have characterised isolates from different time periods relating to the introduction of the whole cell vaccine (WCV) or acellular vaccine (ACV). Before the application of molecular methods to *B. pertussis*, changes in the fimbrial serotypes were identified following the introduction of the WCV. A shift from serotype 2 to serotype 3 and 2,3 was seen in the bacterial population.[8] This serotype shift suggested that the population was adapting to the introduction of the vaccine. Recent MLVA analysis of United Kingdom (UK) isolates showed that before the introduction of the WCV in 1957, the *B. pertussis* population was genetically diverse and that following the introduction of the WCV the diversity was reduced.[5] Interestingly, during the 1970s when vaccination rates in the UK dropped significantly, there was a subsequent increase in diversity and also in serotype 2 isolates. As the rates of vaccination increased, diversity and serotype 2 numbers fell once more. Changes in the MLVA types over time were noted and one particular pattern, MLVA-27, emerged around 1983 and came to dominate after 2002. This particular MLVA type has been identified worldwide and is the dominant MLVA type found in a number of countries.[6,7] Characterisation of Australian *B. pertussis* isolates using SNP confirmed the shift in the bacterial population following the introduction of the WCV and suggested that further changes were occurring following the introduction of the ACV.[6] These observations suggest that the *B. pertussis* population is changing and that the change may be driven by the vaccines.

The re-emergence of pertussis in spite of high rates of vaccination has prompted studies of changes in surface proteins and virulence factors, especially those used in the ACV. The ACV contains individual antigen components generally derived from a single strain of *B. pertussis*. It can be either a three- or five-component vaccine. The three-component vaccine contains pertussis toxin (Ptx), pertactin (Prn), a surface adhesion protein, and filamentous haemaglutinin. The five-component vaccine also contains fimbriae (Fim2 and Fim3). Analysis of the *ptxA* gene identified eight variants with the ACV component being *ptxA*2. Thirteen variants have been described for the *prn* gene with *prn*1 as the component of the ACV. Analysis of UK isolates identified a shift from a mix of *ptxA*1 and *ptxA*2 (pre-WCV and during WCV use) to *ptxA*1 such that all isolates post-ACV are now *ptxA*1 (the UK WCV contained *ptxA*1 and *ptxA*2).[5] Similarly, there has been a shift in *prn* alleles with *prn*2 currently dominating, although *prn*1 and *prn*3 are present at low levels. A similar picture has emerged in Australia with the shifts being associated with the introduction of the ACV.[6,7]

In addition to the variants described above, a mutation in the promotor for the toxin gene, known as *ptxP*3, has been identified.[9] This mutation results in significantly higher levels of toxin production, and increased numbers of isolates containing this variant have been identified in a number of countries.[10–12] The emergence and dominance of the *ptxA*1-, *prn*2- and *ptxP*3-containing strains across the globe has been used as evidence of vaccine-driven adaptation. Isolates from the current New Zealand epidemic also contain the *prn*2 and the *ptxP*3 alleles, suggesting the situation in New Zealand mirrors that seen elsewhere in the world. Further characterisation of the New Zealand isolates is underway.

Unravelling the role that changes in the *B. pertussis* population play in the emergence of pertussis in vaccinated populations is difficult. Infection among recently vaccinated children is rare indicating that the vaccine is still highly effective.[13] A number of factors influence the progress of the disease such as vaccination rates, vaccine schedules, the efficacy of the vaccine and the duration of vaccine protection, as well as changes to the bacterial population. It has been suggested that strains producing increased levels of toxin gain an advantage by prolonging their residence in vaccinated populations providing them with a better opportunity to transmit. Understanding the complex interplay of pathogen, host and vaccine requires continued monitoring of the isolates associated with this disease.

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

Reported by Dr Phil Carter, Health Programme, ESR.

2. Notifiable Disease Surveillance

The following is a summary of disease notifications for the April to June quarter of 2012 and cumulative notifications and rates calculated for a 12-month period (July 2011 to June 2012). For comparative purposes notification numbers and rates are presented in brackets for the same period 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 4 July 2012. 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

Invasive Pneumococcal Disease

- *Notifications:* 113 notifications in the quarter (2011, 132); 528 notifications over the last 12 months (2011, 523), giving a rate of 12.0 cases per 100,000 population (2011, 12.0), not a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (71 cases). Cases were aged between 7 days and 100 years, with 7 cases under the age of 2 years.

Measles

- *Notifications:* 15 notifications in the quarter (2011, 71); 563 notifications over the last 12 months (2011, 117), giving a rate of 12.8 cases per 100,000 population (2011, 2.7), a statistically significant increase.

- *Comments:* there has been a statistically significant quarterly decrease from previous quarter (57 cases) and from the same quarter last year (71 cases). 11 cases were laboratory confirmed.

Mumps

- *Notifications:* 7 notifications in the quarter (2011, 18); 35 notifications over the last 12 months (2011, 47), giving a rate of 0.8 cases per 100,000 population (2011, 1.1), not a statistically significant decrease.

- *Comments:* there has been a statistically significant quarterly decrease from the same quarter last year (18 cases).

Pertussis

- *Notifications:* 1404 notifications in the quarter (2011, 180); 4280 notifications over the last 12 months (2011, 764), giving a rate of 97.2 cases per 100,000 population (2011, 17.5), a statistically significant increase.

- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (1244 cases) and from the same quarter last year (180 cases).

ENTERIC INFECTIONS

Campylobacteriosis

- *Notifications:* 1315 notifications in the quarter (2011, 1315); 7429 notifications over the last 12 months (2011, 6611), giving a rate of 168.6 cases per 100,000 population (2011, 151.4), a statistically significant increase.

- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (2220 cases).

Salmonellosis

- *Notifications:* 227 notifications in the quarter (2011, 266); 991 notifications over the last 12 months (2011, 1220), giving a rate of 22.5 cases per 100,000 population (2011, 27.9), a statistically significant decrease.

- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (343 cases).

VTEC Infections

- *Notifications:* 36 notifications in the quarter (2011, 46); 118 notifications over the last 12 months (2011, 175), giving a rate of 2.7 cases per 100,000 population (2011, 4.0), a statistically significant decrease.

Yersiniosis

- *Notifications:* 127 notifications in the quarter (2011, 85); 549 notifications over the last 12 months (2011, 430), giving a rate of 12.5 cases per 100,000 population (2011, 9.8), a statistically significant increase.

- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (85 cases).

INFECTIOUS RESPIRATORY DISEASES

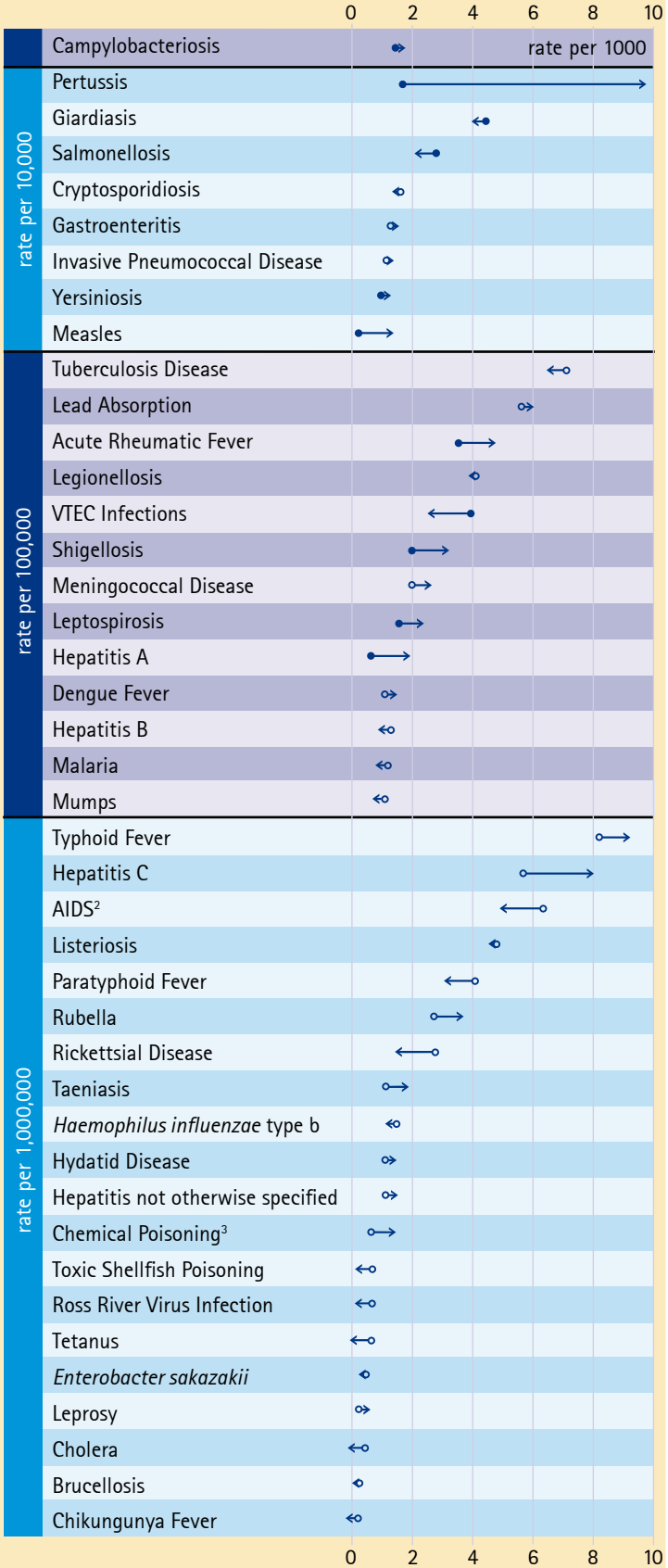
Acute Rheumatic Fever

- *Notifications:* 76 notifications in the quarter (2011, 39); 206 notifications over the last 12 months (2011, 156), giving a rate of 4.7 cases per 100,000 population (2011, 3.6), a statistically significant increase.

- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (42 cases) and from the same quarter last year (39 cases). Cases were distributed by age as follows: 54 (5–14 years), 18 (15–24 years), and 4 (25–44 years). 73 cases were an initial attack of acute rheumatic fever and 3 cases were recurrent attacks.

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 2011 to June 2012 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.

ENVIRONMENTAL EXPOSURES & INFECTIONS

Cryptosporidiosis

- *Notifications:* 131 notifications in the quarter (2011, 78); 688 notifications over the last 12 months (2011, 695), giving a rate of 15.6 cases per 100,000 population (2011, 15.9), not a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (78 cases).

Giardiasis

- *Notifications:* 444 notifications in the quarter (2011, 465); 1808 notifications over the last 12 months (2011, 1976), giving a rate of 41.0 cases per 100,000 population (2011, 45.2), a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (511 cases).

Lead Absorption

- *Notifications:* 91 notifications in the quarter (2011, 57); 265 notifications over the last 12 months (2011, 246), giving a rate of 6.0 cases per 100,000 population (2011, 5.6), not a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (57 cases). Cases were distributed by age as follows: 1 (1–4 years), 1 (5–14 years), 11 (15–24 years), 30 (25–44 years), 36 (45–64 years), 12 (65 years and over). There were 88 male and 3 female cases. 50 cases were recorded as having an occupation that involved exposure to lead. Most commonly recorded occupations were painter/decorator (8 cases), metal worker (3 cases), and welder (2 cases). 30 cases did not have an occupation specified.

Legionellosis

- *Notifications:* 38 notifications in the quarter (2011, 21); 177 notifications over the last 12 months (2011, 181), giving a rate of 4.0 cases per 100,000 population (2011, 4.1), not a statistically significant decrease.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (21 cases). 12 notifications from this quarter remain under investigation, a proportion of these will fail to meet the case definition and be classified as 'not a case'.

Leptospirosis

- *Notifications:* 45 notifications in the quarter (2011, 20); 101 notifications over the last 12 months (2011, 69), giving a rate of 2.3 cases per 100,000 population (2011, 1.6), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (20 cases) and from the same quarter last year (20 cases). There were 38 male and 7 female cases. 32 cases were recorded as having an occupation identified as high risk for exposure. Most commonly recorded occupations were farmer/farmer worker (17 cases) and meat process worker (8 cases). One case did not have an occupation specified.

NEW, EXOTIC & IMPORTED INFECTIONS

Dengue Fever

- *Notifications:* 18 notifications in the quarter (2011, 7); 57 notifications over the last 12 months (2011, 47), giving a rate of 1.3 cases per 100,000 population (2011, 1.1), not a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (7 cases). 17 cases were laboratory confirmed. 17 cases were overseas during the incubation period of the disease. Places visited or resided in were Fiji (6 cases), Thailand (4 cases), Indonesia, Kiribati (2 cases each), Cambodia, Papua New Guinea, Philippines (1 case each).

Hepatitis A

- *Notifications:* 10 notifications in the quarter (2011, 3); 79 notifications over the last 12 months (2011, 28), giving a rate of 1.8 cases per 100,000 population (2011, 0.6), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly decrease from the previous quarter (54 cases). Cases were aged between 6 and 64 years, with 3 cases under the age of 16 years. Overseas travel information was recorded for 9 (90.0%) cases. Of these, 4 (44.4%) cases had not travelled overseas during the incubation period of the disease.

Shigellosis

- *Notifications:* 37 notifications in the quarter (2011, 15); 133 notifications over the last 12 months (2011, 89), giving a rate of 3.0 cases per 100,000 population (2011, 2.0), a statistically significant increase.
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (15 cases). Overseas travel or prior travel information was recorded for 19 (51.4%) cases. Of these, 6 (31.6%) cases had not travelled overseas during the incubation period and had no prior history of travel that could account for their infection.

3. Other Surveillance Reports

Pertussis in New Zealand: morbidity and immunisation

Introduction

Pertussis, or whooping cough, is a vaccine-preventable infectious disease caused by the bacterium *Bordetella pertussis*. New Zealand continues to experience high level of pertussis notifications that have been increasing since mid-2010, despite increasing vaccine uptake. While notifications may underestimate the true incidence of the disease they are still useful for looking at trends and differences between population groups. Pertussis notification data show a four- to five-year cycle with large epidemics occurring in 2000 and 2004, and a much smaller epidemic peak in 2009.

On-time vaccination for infants is recommended at 6 weeks, 3 months and 5 months in addition to children receiving boosters at 4 years and 11 years.[1] Surveillance data show that the incidence rates of pertussis cases and hospitalisations vary with vaccination status, and also across age groups, ethnicity and District Health Boards.[2]

The incidence and severity (hospitalisations) of pertussis, based on notifications (EpiSurv) and vaccination records using national immunisation registration (NIR) data, are examined in this paper with a focus on ethnic differentials.

Morbidity

Pertussis morbidity for the period 1 January to 20 July 2012 is shown in Table 1. Overall the European ethnic group had the highest notification rate followed by Māori and Pacific Peoples, while the Pacific Peoples and Māori ethnic groups had considerably higher rates for more severe illness that require hospitalisation. Over the past four years Māori has been overtaken by the Pacific Peoples ethnic group in this regard. Possible reasons for increased severe illness in these ethnic groups include a lack of or reduced access to primary care, preexisting conditions, and cultural factors such as large families or households that may increase person to person exposures, but also low immunisation coverage.

Table 1. Pertussis notifications, hospitalisations and rates by ethnicity, 1 January to 20 July 2012

Ethnicity	Cumulative cases[1], all age groups				< 1 year age group		
	All cases	ASR	Hospitalisations	ASR	Cases	Rate	Hospitalisation rate
Māori	366	55.3	44	5.1	53	377.7	235.2
Pacific Peoples	105	40.3	28	9.0	25	488.2	371.0
Other	99	25.7	5	1.3	6	114.2	38.1
European	2090	78.3	61	2.6	105	353.5	101.0
Unknown	176		4		7		
Total	2836	70.4	142	3.5	196	346.1	148.3

[1] Pertussis notifications, hospitalisations and associated rates (per 100,000 population)
ASR, age standardised rate (cases per 100,000 population)

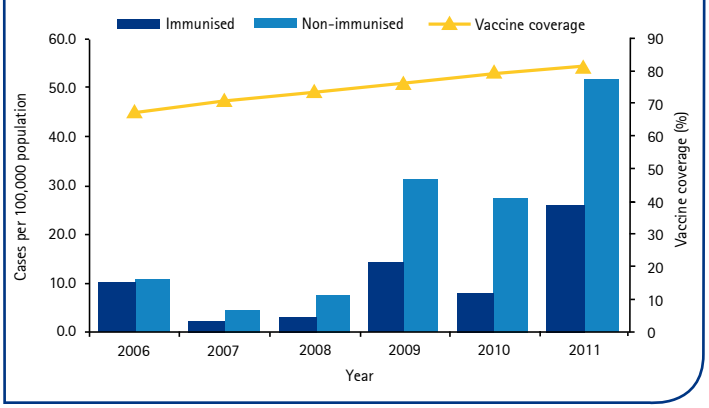
Pertussis rates were significantly higher in the less than one year age group, with highest rates in the Pacific Peoples, followed by Māori and European ethnic groups. This age group also had higher rates of hospitalisations with the highest rate in the Pacific Peoples, followed by Māori, European, and Other ethnic groups (Table 1).

Immunisation and morbidity

Based on NIR data (at milestone ages up to 5 years), vaccine uptake has been increasing since 2006 (Figure 1). While uptake has been increasing in all ethnic groups, disparities remain with rates in the Māori and Pacific Peoples ethnic groups being below the European and Asian ethnic groups (see supplementary figure at www.surv.esr.cri.nz/surveillance/NZPHSR.php). Since 2008, the Asian ethnic group has consistently shown the highest uptake rates. Overall however, immunisation by age 5 years is still too low at 81.8% (as of 2011) to protect children, especially from severe illness.

The incidence rate of pertussis in the past four years, among the non-immunised was higher compared with those who had been immunised with at least one dose of vaccine (Figure 1). The difference in the incidence rate between the two groups has been increasing with higher vaccine coverage over this period. The increase in pertussis cases since mid-2010 may be associated with low vaccine coverage or waning immunity in the adolescents. Waning immunity eventually increases the cohort of susceptible adolescents and adults who can transmit pertussis to vulnerable non-immunised or incompletely immunised infants.[3] Also, changes in the population structure of *B. pertussis*, which may affect the current pertussis vaccine effectiveness, has been associated with increased disease burden.[4]

Figure 1. Vaccine coverage[1] and pertussis notification rate by immunisation status[2], 2006 to 2011



[1] Vaccine coverage was estimated from NIR data
[2] Immunisation status of pertussis notifications was based on EpiSurv notification data (Immunisation status is recorded in EpiSurv by health professionals using a range of sources to update the information including the NIR, parental recall or Well Child book records).

Notification data show that the proportion of hospitalisations has been consistently higher among non-immunised cases compared with cases immunised with at least one dose of vaccine against pertussis. Since 1999, the proportion of hospitalisations have been twice as high among non-immunised cases compared with the immunised (see supplementary figure at www.surv.esr.cri.nz/surveillance/NZPHSR.php). This suggests that immunisation is protective against more severe disease.

The majority of hospitalisations occurred among infants who are the most vulnerable group of the population. Severe illness in infants may be associated with the fact that very little protection against pertussis is passed to the newborn via the placenta or breast milk or maternal antibodies confer a short-lived protection.[5] Van Rie's study[5] suggests that babies born to immunised mothers had higher levels of specific antibodies to *B. pertussis* compared with babies born to non-immunised mothers. However, the proportion of infants born with protective concentrations of maternal antibodies is difficult to ascertain and currently not known. Therefore, based on the existing evidence, the best protection for infants is to reduce the burden of disease in the community especially among those coming into contact with newborn infants and to have these infants immunised following the current Schedule as soon as possible.

Summary

- Pacific Peoples and Māori have considerably higher rates of more severe pertussis illness that require hospitalisation.
- Children under the age of one year had significantly higher pertussis morbidity, especially among the Pacific Peoples and Māori ethnic groups.
- The non-immunised population had greater incidence of pertussis compared with the immunised groups, and thus is at the highest risk for the illness.
- Immunisation seems to have prevented severe illness among pertussis cases.
- Vaccine uptake has increased since 2006, although coverage is still low.

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

Reported by Ange Bissielo and Don Bandaranayake, 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 2012). Comparisons are made to the previous quarter (January to March 2012), and to the same quarter in the previous year (April to June 2011). Note that the outbreak data in this section are notified to ESR by the Public Health Services.

General

- 133 outbreaks notified in this quarter (1008 cases).
- 87 are 'final' reports (862 cases); 46 are 'interim' reports (146 cases) that have yet to be finalised and closed.

All data that follow relate to final reports only.

- 9.9 cases on average per outbreak, compared with 9.6 cases per outbreak in the previous quarter (14.2 cases per outbreak in the same quarter of last year).
- 33 hospitalisations: 'gastroenteritis' (22 cases), norovirus (9 cases), *Giardia* and *Salmonella* (1 case each).
- One death (norovirus).

Pathogens

- 28 'gastroenteritis' outbreaks (298 cases).
- 20 norovirus outbreaks (378 cases).
- 17 *Giardia* outbreaks (67 cases).
- 6 *Campylobacter* outbreaks (26 cases).

- 4 *Cryptosporidium* outbreaks (14 cases).
- 4 *Salmonella* outbreaks (11 cases).
- 2 rotavirus outbreaks (32 cases).
- 1 *Bordetella pertussis* outbreak (4 cases).
- 1 *Clostridium difficile* outbreak (6 cases).
- 1 lead poisoning outbreak (10 cases).
- 1 sapovirus outbreak (11 cases).
- 1 *Shigella* outbreak (2 cases).
- 1 *Yersinia* outbreak (3 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.

- 66 person-to-person, from (non-sexual) contact with an infected person (including droplets): 19 norovirus (367 cases), 17 'gastroenteritis' (264 cases), 16 *Giardia* (64 cases), 4 *Cryptosporidium* (14 cases), 3 *Campylobacter* (14 cases), 2 rotavirus (32 cases), 1 *B. pertussis* (4 cases), 1 *C. difficile* (6 cases), 1 *Salmonella* (5 cases), 1 sapovirus (11 cases), and 1 *Yersinia* (3 cases).
- 17 foodborne, from consumption of contaminated food or drink (excluding water): 11 'gastroenteritis' (40 cases), 3 *Campylobacter* (15 cases), 2 *Giardia* (5 cases), and 1 norovirus (11 cases).
- 4 waterborne, from consumption of contaminated drinking water: 2 *Giardia* (6 cases), 1 *Campylobacter* (2 cases), and 1 *Cryptosporidium* (3 cases).
- 14 environmental, from contact with an environmental source (e.g., swimming): 4 *Giardia* (11 cases), 3 *Cryptosporidium* (11 cases), 3 norovirus (40 cases), 2 *Campylobacter* (9 cases), 1 'gastroenteritis' (21 cases), and 1 lead poisoning (10 cases).
- 8 zoonotic, from contact with infected animal: 3 *Campylobacter* (8 cases), 3 *Giardia* (9 cases), 1 *Cryptosporidium* (3 cases), and 1 *Salmonella* (2 cases).
- 3 'other' modes: 2 *Giardia* (4 cases) and 1 norovirus (11 cases).
- 6 mode of transmission unknown: 3 'gastroenteritis' (9 cases), 2 *Salmonella* (4 cases), and 1 *Shigella* (2 cases).

Circumstances of Exposure

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

- 27 home: 13 *Giardia* (52 cases), 4 *Cryptosporidium* (14 cases), 3 *Campylobacter* (11 cases), 3 *Salmonella* (9 cases), 2 'gastroenteritis' (9 cases), 1 *B. pertussis* (4 cases), and 1 *Yersinia* (3 cases).
- 15 long term care facility: 10 norovirus (230 cases) and 5 'gastroenteritis' (89 cases).
- 13 childcare centre: 5 'gastroenteritis' (76 cases), 4 norovirus (59 cases), 2 rotavirus (32 cases), 1 *Campylobacter* (3 cases), and 1 sapovirus (11 cases).
- 12 restaurant/café/bakery: 8 'gastroenteritis' (30 cases), 2 *Campylobacter* (12 cases), and 2 norovirus (15 cases).
- 4 hospital (acute care): 1 *C. difficile* (6 cases), 1 'gastroenteritis' (15 cases), 1 norovirus (22 cases), and 1 *Salmonella* (5 cases).
- 2 camp: norovirus (26 cases).
- 2 school: 1 'gastroenteritis' (22 cases) and 1 norovirus (26 cases).
- 1 farm: *Giardia* (3 cases).
- 1 fast food restaurant: 'gastroenteritis' (4 cases).
- 1 hotel/motel: 'gastroenteritis' (12 cases).
- 1 other institution: 'gastroenteritis' (35 cases).
- 1 supermarket/delicatessen: 'gastroenteritis' (2 cases).
- 1 swimming/spa pool: *Giardia* (2 cases).
- 1 takeaways: 'gastroenteritis' (2 cases).
- 1 workplace: lead poisoning (10 cases).
- 5 'other setting': 4 *Giardia* (15 cases) and 1 *Campylobacter* (3 cases).
- 7 outbreaks had two exposure settings recorded.
- 6 outbreaks had no exposure settings recorded.

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

- 11 restaurant/café/bakery: 8 'gastroenteritis' (30 cases), 2 *Campylobacter* (12 cases), and 1 norovirus (11 cases).
- 3 home: 1 *Campylobacter* (3 cases), 1 'gastroenteritis' (4 cases), and 1 *Giardia* (2 cases).
- 1 farm: *Giardia* (2 cases).
- 3 outbreaks had no preparation settings recorded.
- 1 outbreak had two preparation settings recorded.

5. Outbreak Case Reports

A campylobacteriosis outbreak at a rural camping ground

Introduction

Hawke's Bay Public Health Unit investigated an outbreak of gastroenteritis in a school group, comprising children and their adult helpers, that stayed at a private rural camping ground from 21 to 23 March 2012. We report on the outbreak investigation.

Investigation

The campers had taken all their own food and prepared all their own meals. A parent, who is a food scientist, commented that she observed no deficiencies in food storage or handling.

The camp's water comes from a local river. Water is pumped to a storage tank then gravity fed to a treatment plant where it is filtered and treated with ultraviolet (UV) radiation. Heavy rainfall had occurred and river levels had risen, peaking the night before the camp began. The ultra-violet treatment unit was not operating correctly and the filters were inadequate.

Eight drinking-water samples taken on three different days after the camp (26 to 30 March) all contained *Escherichia coli* and raised total coliform counts. *Campylobacter* was cultured from one drinking-water sample.

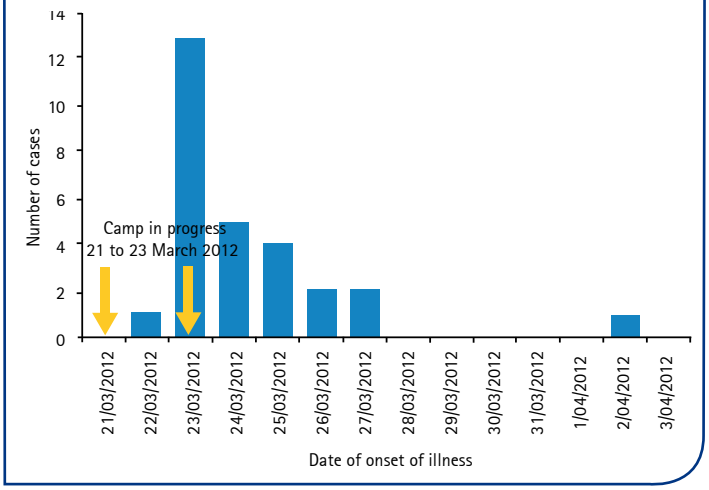
Seven stool samples from cases grew *Campylobacter*. ESR analysed four of the faecal *Campylobacter* isolates and the single drinking-water *Campylobacter* isolate using pulsed-field gel electrophoresis. Each isolate had a different pattern. All of the pulsotypes were on the

Pulsenet database and were predominantly from bovine/ovine sources and, infrequently, chicken sources. ESR commented: 'A herd of cows will contain multiple genotypes of *Campylobacter*. Water contaminated with bovine faeces from a herd would be expected to contain many genotypes of *Campylobacter*. Common source food outbreaks tend to have the same genotype of *Campylobacter*.'

All camping group members responded to a retrospective cohort questionnaire survey of risk factors. Of the 38 members, 28 (73.6%) met the case definition (see www.surv.esr.cri.nz/surveillance/NZPHSR.php) for a confirmed or clinical Campylobacteriosis case – this included two secondary cases in household members who did not attend the camp.

The epidemic curve of onset dates of illness shows the typical pattern of a point-source outbreak (Figure 2). The median duration of symptoms for the 19 cases who had recovered by the time of interview was 3 days (range: less than 1 day to 11 days).

Figure 2. Epidemic curve for the camping ground outbreak of campylobacteriosis



Analysis of the questionnaire data showed that drinking-water was the only significant risk factor. All of the people who reported not drinking the camp's water remained well, whereas 89.3% of those who reported drinking the camp's water became ill (risk ratio could not be calculated).

Eight additional gastrointestinal illness cases were found in three other groups that visited the camping ground from January 2012 to the end of March 2012; one was a confirmed *Campylobacter* case.

Discussion

Campylobacter caused this outbreak and the camp's drinking-water was the source. The retrospective cohort questionnaire study showed that drinking-water was the only significant risk factor for illness. The camp's drinking-water contained coliforms and *Campylobacter*. When the outbreak happened, the camp's treatment plant was ineffective at treating the river water to the requirements of the drinking-water standards for New Zealand. This was due to the system not being designed to cope with the river in flood, inadequate filtration and the non-operational UV system.

A potable water supply is being established and campers are bringing their own drinking-water in the interim.

The contamination of river water (and therefore drinking-water) by animal effluent during heavy rainfall was highlighted in a recent Ministry of Agriculture and Forestry Technical Paper[1] which reported that 'The environmental models, aided by further strain typing of isolates from river water during flood flows (funded by DairyNZ), confirm that *Campylobacter* concentrations during low flow periods are usually low and are dominated by wild bird species. However during flood periods concentrations become much higher, are dominated by ruminant species, and this is the result of local runoff from farmed areas.'

Two other gastroenteritis outbreaks have been investigated from camping grounds in the Hawke's Bay region. An outbreak investigation of 40 *Campylobacter* cases following a school camp in December 2005, identified recreational exposure to river water as the likely infection source, but the drinking-water was also contaminated. Investigation of another *Campylobacter* outbreak at a youth camp in 1992 found that the untreated water supply could have been the outbreak source.[2]

These outbreaks highlight the potential for local authority enhancement of camp drinking-water safety through camping ground registration processes.

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

Reported by Maree Rohleder, Health Protection Officer and Lester Calder, Medical Officer of Health, Hawke's Bay Public Health Unit.

6. Laboratory Surveillance

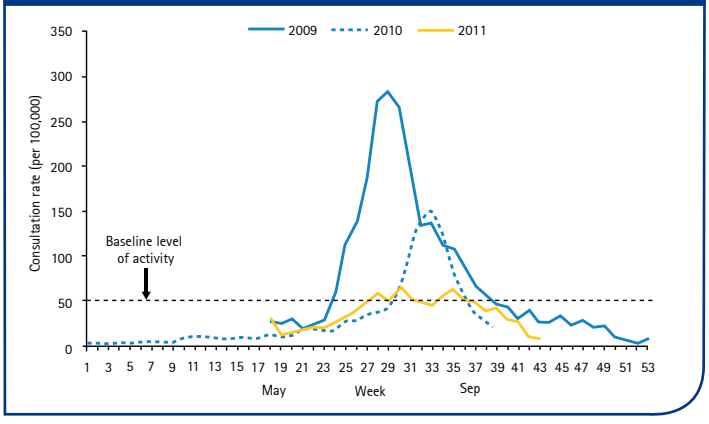
Influenza surveillance, 2011

In 2011, 88 sentinel practices were recruited from all 20 District Health Boards (DHBs) under ESR's sentinel General Practice-based surveillance. Some sentinel practices did not report every week. On average, 81 practices, with a total patient roll of 385,108 people, participated in influenza sentinel surveillance each week from May to October 2011 (extended by one month to cover the Rugby World Cup event held in New Zealand). During the surveillance period, 3596 consultations for influenza-like illness (ILI) were reported. It is estimated that ILI resulting in a visit to a general practitioner affected over 41,133 people in New Zealand (0.9% of total population) in 2011.

The average weekly consultation rate from May to September 2011 was 40.4 per 100,000 patient population, this was lower than the rate in 2010 (50.9) and 2009 (109.2). This is the fourth lowest rate since 1997 with the lowest recorded in 2000 (32.5 per 00,000). The highest consultation rate was recorded in 1997 (163.7 per 100,000), followed by 1999 (112.3).

Overall, influenza activity in 2011 was low. The influenza consultation rate remained at or below the baseline level (50.0 per 100,000 patient population) from weeks 18 to 27, and it then increased to a peak in week 30 (25 to 31 July), with a consultation rate of 66.1 per 100,000 patient population (Figure 3). The 2011 peak was lower than the peaks in 2010 (151.6 per 100,000) and 2009 (284.0). Since 1997, the highest peaks were in 2009 (284.0 per 100,000) and 1997 (244.2). The lowest peak was recorded in 2000 (41.7 per 100,000).

Figure 3. Weekly sentinel surveillance consultation rates for influenza-like illness, 2009 to 2011



Consultation rates varied among DHBs, with the highest rates in Waitemata (65.4 per 100,000 patient population), Whanganui (56.8) and South Canterbury (56.4) DHBs.

Ministry of Health hospitalisation data for 2011 recorded 532 hospitalisations with the primary reason for admission being influenza. This was lower than in 2010 (998 hospitalisations) and 2009 (1517).

In terms of the number of hospitalisations by week discharged, 88.2% (469) occurred from June to October. The highest number of hospitalisations (134) occurred in September. Hospitalisations peaked in week 33. Numbers of sentinel and non-sentinel influenza viruses detected peaked in weeks 33 and 36, and ILI consultation numbers peaked in week 30.

A total of 1268 influenza viruses were identified in 2011, lower than in 2010 (2012 viruses) and 2009 (4900). Of the 1268 viruses identified, 336 came from sentinel practice surveillance from May to October, compared with 349 in 2010 (January to September) and 624 in 2009 (May to December). There were 932 non-sentinel viruses identified in 2011 compared with 1663 in 2010 and 4276 in 2009.

In 2011, influenza B was the predominant strain, 46.7% (592/1268) among all viruses and 50.3% among all typed and subtyped viruses. Among all lineage-determined influenza B viruses (280), the B/Victoria lineage viruses (B/Brisbane/60/2008-like strain) represented the highest proportion (98.6%, 276/280). Seasonal A(H3N2) represented 36.8% (466/1268) of all viruses and 39.6% (466/1176) of all typed and subtyped viruses. There was a small proportion of influenza A(H1N1) pdm09 viruses among all typed and subtyped viruses (9.3%, 118/1268).

Noticeable changes have occurred in terms of predominant patterns in the numbers and percentages of typed and subtyped influenza viruses from 1990 to 2011. These are described next.

Influenza A(H1N1) viruses

In 2011, influenza A(H1N1) viruses represented 9.3% of all viruses. All of these were the pandemic strain, A(H1N1)pdm09. The antigenic data from New Zealand isolates indicate that most of the A(H1N1)pdm09 viruses currently circulating are closely related to the vaccine strain A/California/7/2009 (H1N1). The seasonal influenza A(H1N1) strain has not been detected since 2010 in New Zealand.

Influenza A(H3N2) viruses

In 2011, 39.6% of the typed and subtyped viruses were influenza A(H3N2). They were antigenically closely related to the 2011 vaccine strain A/Perth/16/2009 (H3N2)-like strain.

From 1990 to 2011, 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 due to influenza (94 in 1996) in New Zealand was recorded during an influenza A(H3N2) epidemic. The highest number of influenza hospitalisations (552) recorded by the Ministry of Health was in 2003 and was due to a season predominated by influenza A(H3N2) viruses.

Influenza B viruses

In 2011, there were 592 influenza B viruses detected, of which 280 were antigenically typed: 276 as B/Victoria lineage (B/Brisbane/60/2008-like) and four as B/Yamagata lineage (B/Florida/4/2006-like).

From 1990 to 2011, influenza B viruses predominated for six seasons in 1991 (92.3%), 1995 (68.8%), 1997 (53.5%), 2005 (87.0%), 2008 (58.3%) and 2011 (46.7%). 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.

Since the B/Victoria lineage viruses were introduced into New Zealand in 2002, this strain and B/Yamagata lineage viruses have been co-circulating in New Zealand. B/Victoria lineage viruses have predominated over the B/Yamagata lineage viruses every 3 years (in 2002, 2005, 2008 and 2011). The influenza B viruses have 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.

Characterisation of the influenza viruses isolated during the 2011 winter indicated that there was no requirement to change any of the three components of the current vaccine. Accordingly, the 2012 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 an influenza A(H1N1)pdm09 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 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

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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

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