

## TRENDS

Reports of **arbovirus** infections (mainly **Ross River virus** infections from Greater Murray Area, Western NSW, and increasingly the Hunter, and a handful of **Barmah Forest** virus infections from the Mid North Coast [Table 3]) remain above the historical average (Figure 1). The huge increase in reported **hepatitis A** cases in February is a legacy of Wallis Lake oysters (see the *Bulletin*, Jan-Feb 1997). Reports of **pertussis**, while still above expectations for this time of year, declined in February after a steady increase in the previous 12 months (Figure 1).

## INFLUENZA

With winter approaching, the ugly spectre of influenza will raise its head soon. So start immunising people at risk for complications now.

Influenza is an acute respiratory infection characterised by fever, headache, myalgia, prostration, coryza, sore throat and cough. The virus is transmitted by respiratory droplets and may be shed up to 24 hours before onset of illness. Adults are infectious for 3-5 days after onset and children for up to 7 days<sup>1,2</sup>. The incubation period is usually 1-5 days. Most people recover after 2-7 days, but in people  $\geq 65$  years or with pre-existing medical conditions, serious complications including pneumonia and death may follow.

### Epidemics

In the past 400 years, local and widespread epidemics and worldwide pandemics have been reported. In 1918-19, 21 million flu deaths were reported worldwide. Community attack rates generally range between 10 per cent and 30 per cent during epidemics, even higher in confined environments such as nursing homes and boarding schools<sup>2</sup>.

### Viral types

The virus type is determined by the antigenic properties of the nucleoprotein. Subtypes of influenza A are classified according to surface glycoproteins. H refers to the haemagglutinin and N to the neuraminidase components. Frequent mutations of viral genes result in emergence of variants that are described by geographic site, the culture number and the year of isolation. Pandemics of type A occur irregularly and result from the emergence of completely new subtypes (antigenic shift), while annual epidemics of types A or B result from minor antigenic changes (antigenic drift). Type C has been associated with sporadic cases and minor localised outbreaks.

### Surveillance

Surveillance of influenza-like illness (ILI) in NSW begins in April each year, through GP sentinel network, school absentee rates and lab virology and serology reports<sup>3</sup>.

### Flu vaccine

Influenza vaccine is reviewed annually so that changes in the composition can be made to counter antigenic shifts and drifts in the circulating viruses. The Australian Influenza Vaccine Committee recommended the following antigens for the 1997 vaccine<sup>4</sup>:

- H<sub>1</sub>N<sub>1</sub> A/Texas/36/91 (H<sub>1</sub>N<sub>1</sub>)-like strain
- H<sub>3</sub>N<sub>2</sub> A/Wuhan/359/95 (H<sub>3</sub>N<sub>2</sub>)-like strain
- B/Beijing/184/93-like strain.

Immunisation confers about 70 per cent protection against infection for about one year<sup>5</sup>, and annual vaccination with

current vaccine is required to provide continuing protection. A single dose of vaccine is sufficient for most people. Little or no improvement in antibody responses occurs when a second dose is given to adults during the same season. However, two doses  $\geq 4$  weeks apart are recommended for people with impaired immune function.

The vaccine should be refrigerated at 2-8°C, and administered by deep subcutaneous or intramuscular injection. The vaccine is not recommended in pregnancy (because of the risk of febrile reactions), for people with anaphylactic hypersensitivity to eggs or people with acute febrile illnesses.

### Australia 1996

In NSW and other parts of Australia in 1996<sup>7</sup> an increase in influenza A (H<sub>3</sub>N<sub>2</sub>) was recorded, peaking in July. There was little influenza B activity reported. In NSW the consultation rate of influenza like illness among patients of sentinel GP practices peaked at 2.5 per cent, well below epidemic levels.

### Northern hemisphere, 1997

During the 1996-97 northern hemisphere season, many countries reported moderate to severe influenza epidemics. Activity peaked in Western Europe and North America in December 1996 or January 1997, and began in central and eastern Europe in mid-January<sup>6</sup>.

### Who should be vaccinated?

NHMRC has revised its recommendations for influenza vaccination to maximise protection of high-risk people. Vaccination is recommended for people who, because of age or underlying medical conditions, are at increased risk for complications of influenza. In addition, staff caring for people at risk (e.g. in a nursing home) should consider vaccination to minimise spread to these people.

### Target groups for influenza vaccinations<sup>8</sup>:

#### Routinely vaccinate:

- Individuals  $\geq 65$  years old
- Aboriginal and Torres Strait Islander adults  $\geq 50$  years old
- Adults with chronic debilitating diseases (especially chronic cardiac, pulmonary, renal and metabolic disorders)
- Children with cyanotic congenital heart disease
- Adults and children receiving immunosuppressive therapy
- Residents nursing homes and other chronic care facilities.

#### Consider vaccinating:

- Staff caring for immunocompromised patients
- Staff of nursing homes and other chronic care facilities.

Because antibody levels begin to decline within a few months of vaccination, at-risk people should be immunised in autumn just before the flu season begins. However, opportunities to vaccinate people having infrequent or irregular contacts with health care providers should be maximised by immunising high risk people when vaccine for the influenza season becomes available.

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## Infectious diseases

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Vaccination is generally not recommended for people outside these risk groups, because they are not at increased risk of serious complications.

(Adapted from: Southern Sydney Public Health Unit. Public Health Update 1995; 2:5.)

1. Mandell G, Bennett J, Dolan R (eds). Principles and Practice of Infectious Diseases. 4th ed. USA: Churchill Livingstone, 1995.
2. Benenson A (ed). Control of communicable diseases in man. 15th ed. USA: American Public Health Association, 1990.
3. NSW Health Dept. 1993 Infectious diseases notifications. *Public Health Bulletin* 1995; 6(S1):59.
4. Anonymous. Composition of Australian influenza vaccine for the 1997 winter. *Comm Dis Intell* 1996; 20:465-466.
5. NHMRC. The Australian immunisation procedure handbook. 6th ed. Canberra: Australian Government Publishing Service, 1997.
6. Recommended composition of influenza virus vaccines for use in the 1997-1998 season. *Weekly Epid Rep* 1997; 72:57-62.
7. Curran M, Moser K. National influenza surveillance 1996. *Comm Dis Intell* 1997; 21:101-105.

### US CASE OF HUMAN RABIES FROM A DOG BITE IN NEPAL

Earlier this year, the United States Centers for Disease Control and Prevention reported the case of a 32-year-old American woman who died on August 20, 1996 from an illness characterised by rapid neurologic deterioration. Rabies had been clinically suspected on August 14, and was confirmed on August 17. Here is a summary of the investigation that implicated a dog in Kathmandu, Nepal, as the probable source of exposure.

#### Case history

The patient initially sought care at a hospital emergency department (ED) in New Hampshire on August 12 for a two-day history of paraesthesias and pain radiating up her left arm from the site of a healed bite. She reported being bitten by a dog on her left hand on June 7 while in Kathmandu, but did not receive rabies post-exposure prophylaxis (PEP) for the bite. Physical examination was normal, and left cervical radiculopathy was diagnosed. Anti-inflammatory and analgesic drugs were prescribed and she was discharged.

On August 14 the patient returned to the ED with complaints of progressive difficulty breathing, throat spasms, nausea and vomiting, and reported severe pharyngeal spasms when she drank fluids or showered. Physical findings included an oral temperature of 36.3°C, pulse rate of 64 beats a minute, respiratory rate of 26 breaths a minute and blood pressure of 106/60mmHg. The patient was alert, oriented and in no acute distress. She had a normal sensory examination; however, painful spasms of the bulbar musculature of the lower face and throat were noted when she brought a cup to her mouth or when air was blown in her face. Routine laboratory evaluation, an electrocardiogram and radiographs of the chest and lateral neck were normal. On the basis of history and symptoms, clinical rabies was suspected and the patient was transferred to a hospital in Massachusetts for further

evaluation and treatment. On admission, a computerised tomography scan of her head was normal. Cerebrospinal fluid evaluation was normal except for a white blood cell count of 42 cells/L, with a differential of 12 per cent neutrophils, 56 per cent lymphocytes, and 32 per cent monocytes. The patient was initially treated with rabies immunoglobulin (RIG) and human diploid cell vaccine (HDCV) in the standard post-exposure regimen<sup>1</sup>.

Over the subsequent 12 hours the patient developed increasing agitation, anisocoria (unequal pupils), salivation and worsening facial and pharyngeal spasms. She received experimental treatment with high-dose intravenous and intrathecal RIG; however, her condition continued to deteriorate. On August 15, a full-thickness nuchal skin biopsy and saliva sample were obtained and sent to CDC for rabies diagnosis. Both tested positive for rabies virus on August 17 by a nested polymerase chain reaction (PCR) procedure. Patient serum collected on August 16 also was antibody positive, containing a virus-neutralising titre of 1:9 by the rapid fluorescent focus inhibition test. Nucleotide sequence analysis of the PCR product conducted at CDC on August 18 implicated a variant of rabies virus associated with dogs from the Indian subcontinent. On August 20 neurologic evaluation of the patient revealed no brainstem or cortical function, and life support was discontinued. Because rabies was suspected on admission, appropriate precautions were observed and no employee at the Massachusetts hospital required PEP.

The patient had been travelling for six months in New Zealand, Australia, Thailand and Nepal. She was bitten on the left hand while petting a stray dog on June 7 while in Kathmandu. The wound was immediately washed with peroxide and rubbing alcohol. The dog was observed for about 45 minutes and appeared normal, and no rabies testing was performed on the animal. The patient was reportedly unable to obtain PEP in Kathmandu or Bangkok, Thailand, and was advised to go to Sydney, Australia, for definitive medical care. On June 12, she was examined at a hospital in Sydney and was told that RIG and rabies vaccine were not immediately available and to return the following day for treatment. Because the patient had reportedly received conflicting information from other sources regarding her risk for rabies and the benefit of PEP after the delay between exposure and treatment, she elected not to return to the hospital for treatment.

The patient returned to the United States around June 30. While visiting relatives on August 3, salivary contact (i.e. kissing and sharing of utensils and drink glasses) was reported with five people. One other contact, a travelling companion, also reported salivary contact. All six people were administered PEP. The patient developed her first symptoms on August 10. An investigation was initiated to determine other close contacts to the patient on or after July 31. Other than the six contacts previously noted, a doctor in New Hampshire who initially examined the patient in the ED also received PEP.

#### Comment

Two cases of human rabies were reported in Australia in 1987 and 1990, linked to overseas exposures in India<sup>2</sup> and South East Asia<sup>3</sup>. In the United States, of 30 cases reported since 1980, 14 (47 per cent) have been associated with exposure to dogs; 12 of the 14 were presumed to have been acquired outside the United States.

Although the incubation period for rabies is usually 1-3 months, longer incubation periods have been reported<sup>3</sup>. Prevention of disease after exposure is effective only if PEP is administered before the onset of clinical disease. Although treatment should be initiated as soon as possible, the stage of the incubation period during which infection becomes intractable is unknown. Therefore, PEP is recommended for administration any time before the onset of symptoms, regardless of the time elapsed since exposure. RIG still may be administered for up to one week after the rabies vaccine series has been initiated. However, administration of RIG more than one week after initiation of the vaccine series is not recommended because antibodies to the virus already will have been induced by the vaccine.

#### The Australian connection

In this case, had the patient elected to receive PEP in Sydney, a delay of about five days would have occurred. There have been no reported failures of PEP in association with the correct implementation of the treatment regimen in the United States<sup>1</sup>, where the median interval between exposure and administration of PEP is about five days. *This report highlights the importance of taking a careful exposure history when evaluating people who may have come into contact with potentially rabid animals overseas.* Where such an exposure is identified, rapid administration of RIG and rabies vaccine is essential.

*To arrange PEP, or where there is any doubt about its need, clinicians should seek advice from their local Public Health Unit.* With the discovery of a new rabies-like lyssavirus in flying foxes and insectivorous bats in Australia and the identification of a human fatality associated with this virus in 1996<sup>5</sup>, use of PEP in NSW has increased dramatically in recent months.

#### Travellers at risk

The risk for rabies for international travellers is greatest in areas where canine rabies is still highly endemic, including many parts of Africa, Asia and Central and South America. Two countries where the patient in this report had extended

stays – Nepal and Thailand – are considered to be areas where dog rabies is highly endemic. Pre-exposure vaccination with HDCV or rabies vaccine adsorbed should be considered for people living in or visiting (for greater than 30 days) areas where rabies is endemic and appropriate PEP may not be readily obtained.

Pre-exposure vaccination does not eliminate the need for additional therapy after an exposure but does simplify the post-exposure regimen by eliminating the need for RIG and decreasing the number of required vaccine doses<sup>6</sup>. Because rabies virus may be present in the saliva of infected animals 3-4 days before onset of clinical symptoms<sup>7</sup>, people who are bitten or scratched by any animal should thoroughly wash all wounds with soap and water and immediately seek medical consultation to evaluate the need for PEP<sup>1,2</sup>. Where there is a delay between a high-risk exposure and presentation for treatment, PEP should be administered regardless of the delay.

(Adapted from: CDC. Human Rabies – New Hampshire. *MMWR* 1997; 46:7-12.)

1. CDC. Rabies prevention – United States, 1991: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR* 1991; 40(no. RR-3).
2. CDC. Human rabies – Florida, 1996. *MMWR* 1996; 45:719-20,727.
3. Smith JS, Fishbein DB, Rupprecht CE, Clark K. Unexplained rabies in three immigrants in the United States, a virologic investigation. *N Engl J Med* 1991; 324:205-11.
4. Helmick CG. The epidemiology of human rabies postexposure prophylaxis, 1980-1981. *JAMA* 1983; 250:1990-6.
5. Fraser GC, Hooper PT, Lunt RA et al. Encephalitis caused by a lyssavirus in fruit bats in Australia. *Emerging Infectious Diseases* 1996; 2:327-31.
6. CDC. Health information for international travel, 1994. Atlanta: US Department of Health and Human Services, Public Health Service, 1994: 125-8.
7. Vaughn JB Jr, Gerhardt P, Newell KW. Excretion of street rabies virus in the saliva of dogs. *JAMA* 1965; 193:363-8.
8. Faoagali JL, De Buse P, Strutton GM, Samaratinga H. A Case of Rabies. *Med J Aust* 1988; 149:702-707.
9. Grattan-Smith PJ, O'Regan WJ, Ellis PSJ, O'Flaherty SJ, McIntyre PB, Barnes CJ. A second Australian case, with a long incubation period. *Med J Aust* 1992; 156:651-654.

## Public Health Laboratory Network

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compliance with the Public Health Act 1991 and other legislation designed to prevent illness and protect the health of the population. It is appropriate for the agenda and priorities for the network of designated public health laboratories to be determined by the Chief Health Officer.

The agenda and priorities could then be incorporated in service agreements between the Department and Area Health Services responsible for managing designated laboratories.

To assist the Chief Health Officer with the development and maintenance of the PHLN it is proposed that a Public Health Laboratory Advisory Committee be formed, with membership including balanced input from the Department, the Area Health Services, laboratories (both public and private) and Public Health Units.

#### THE FUTURE

It will be important for the PHLN to be reviewed constantly, taking into account rapidly changing technologies, new threats to public health, and changing priorities. This will require a willingness to terminate services no longer required, as well as a preparedness to enhance existing services and develop new services.

**FIGURE 1**

**REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, 12 MONTHS TO JANUARY 1997  
BY MONTH OF ONSET (WITH HISTORICAL COMPARISON)**

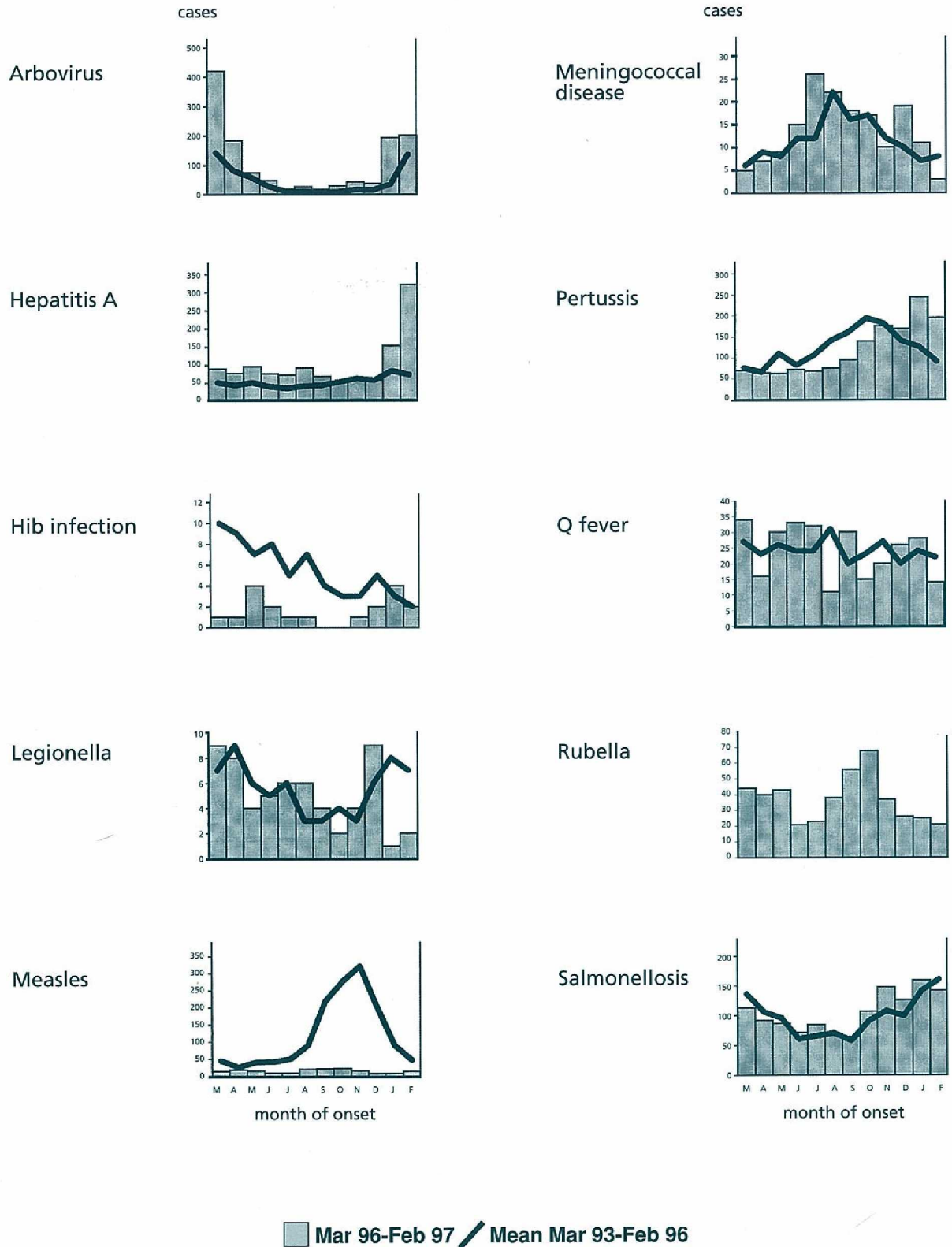


TABLE 3

## INFECTIOUS DISEASE NOTIFICATIONS FOR NSW RECEIVED IN MARCH 1997, BY AREA HEALTH SERVICES

Condition	Area Health Service																	Period	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	Total for Mar**	Total to date**
	<b>Blood-borne and sexually transmitted</b>																		
AIDS	1	1	1	1	-	-	1	2	4	-	-	1	-	-	-	-	-	12	115
HIV infection*	-	2	-	-	-	-	2	1	4	-	-	-	-	-	-	-	-	26	110
Hepatitis B - acute viral*	1	-	2	1	-	-	-	1	1	-	1	1	-	-	-	-	-	8	14
Hepatitis B - other*	58	33	46	6	84	4	5	8	57	2	1	6	1	1	-	1	3	251	958
Hepatitis C - acute viral*	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	1
Hepatitis C - other*	86	45	98	34	77	31	45	30	122	53	20	9	4	5	3	14	17	694	2,208
Hepatitis D - unspecified*	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	1	3
Hepatitis E	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0	4
Hepatitis, acute viral (NOS)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Gonorrhoea*	7	1	1	-	2	-	-	1	11	3	-	1	1	-	-	1	-	29	124
Syphilis	12	2	6	1	10	-	-	1	15	2	-	3	4	-	-	1	-	57	147
<b>Vector-borne</b>																			
Arboviral infection*	2	2	1	2	3	-	12	4	3	6	23	8	20	4	19	61	5	175	530
Malaria*	-	2	3	-	-	1	-	1	3	-	-	-	-	-	-	-	-	10	40
<b>Zoonoses</b>																			
Brucellosis*	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1	2
Leptospirosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5
Q fever*	-	-	-	-	-	-	1	-	-	1	5	2	2	-	-	1	1	13	62
<b>Respiratory/other</b>																			
Legionnaires' disease	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	1	11
Meningococcal (invasive) infection	1	-	-	2	1	-	1	-	-	-	-	-	-	-	-	-	-	5	20
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Mycobacterial tuberculosis	2	5	2	-	5	-	-	-	3	-	-	-	-	-	-	-	-	17	72
Mycobacteria other than TB	11	4	8	-	1	-	6	-	1	-	5	-	-	-	-	2	-	38	97
<b>Vaccine-preventable</b>																			
Adverse event after immunisation	-	-	2	-	-	-	1	-	-	-	-	-	-	-	-	1	-	4	11
<i>H. influenzae</i> B (invasive) infection	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1	7
Measles	1	2	1	-	1	4	2	1	2	-	-	-	-	-	-	-	1	15	36
Mumps*	-	-	1	-	-	-	-	-	-	-	-	-	-	7	-	-	-	8	14
Pertussis	12	29	23	14	28	4	29	8	24	-	7	9	9	2	2	3	3	206	645
Rubella*	-	1	-	-	3	-	3	-	3	-	-	2	1	-	-	1	-	14	51
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
<b>Faecal-oral</b>																			
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Foodborne illness (NOS)	-	-	-	-	-	1	-	-	7	-	-	-	-	-	-	-	-	8	33
Gastroenteritis (instit)	-	-	-	-	-	-	16	-	-	-	-	-	-	1	-	-	-	17	173
Hepatitis A	7	15	13	1	5	5	8	2	10	3	7	7	5	2	2	2	-	94	568
Listeriosis*	-	-	2	-	-	-	-	1	1	-	-	-	-	-	-	-	-	4	9
Salmonellosis (NOS)*	14	29	12	5	8	10	20	6	26	27	6	12	2	1	2	5	4	189	486
Typhoid and paratyphoid*	-	-	-	-	1	-	-	-	1	-	-	-	-	-	-	-	-	2	6

\* lab-confirmed cases only

\*\* includes cases with unknown postcode

*Abbreviations used in this Bulletin:*

CSA Central Sydney Health Area, SES South Eastern Sydney Health Area, SWS South Western Sydney Health Area, WSA Western Sydney Health Area, WEN Wentworth Health Area, NSA Northern Sydney Health Area, CCA Central Coast Health Area, ILL Illawarra Health Area, HUN Hunter Health Area, NRA Northern Rivers Health Area, MNC Mid North Coast Health Area, NEA New England Health Area, MAC Macquarie Health Area, MWA Mid West Health Area, FWA Far West Health Area, GMA Greater Murray Health Area, SA Southern Health Area, OTH Interstate/Overseas, U/K Unknown, NOS Not Otherwise Stated.

Please note that the data contained in this Bulletin are provisional and subject to change because of late reports or changes in case classification. Data are tabulated where possible by area of residence and by the disease onset date and not simply the date of notification or receipt of such notification.