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Front cover: Surveillance Section, Australian Government Department of Health and Ageing.
Images sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia.
Clockwise from top left: Administering oral polio vaccine in India on National Immunization Day, Chris Zahniser ; Australian Immunisation Handbooks; bakery items are common source of foodborne disease.

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Quarterly reports

Re-emerging poliomyelitis – is Australia's surveillance adequate?

David N Durrheim,[1] Peter Massey,[2] Heath Kelly[3]

Abstract

In the past two years there has been a resurgence of polio with 21 previously polio-free countries importing wild poliovirus. Wild poliovirus importations into polio-free areas will continue to occur until endemic transmission is interrupted globally. Australia's acute flaccid paralysis (AFP) surveillance falls well short of the target of more than 80 per cent of AFP cases having two adequate stool specimens taken at least 24 hours apart within 14 days of onset for poliovirus examination. As most AFP cases are hospitalised, AFP should be immediately notifiable by hospitals to public health units or state or territory public health authorities to ensure appropriate follow up, including stool specimens. *Commun Dis Intell* 2006;30:275–277.

Keywords: acute flaccid paralysis, surveillance, poliovirus

Poliomyelitis (polio) is a devastating infectious disease that has been controlled for many years but not yet eradicated. In 2003, within 15 years of the World Health Assembly adopting a resolution calling for the global eradication of polio, the number of polio-endemic countries had decreased from 125 to 6 (Afghanistan, Egypt, India, Niger, Nigeria and Pakistan).[1] In 2006 the World Health Organization reclassified Egypt and Niger as no longer endemic as all recent wild poliovirus isolations in these countries have been confirmed by genetic sequencing as importations. However, in the past two years there has been a resurgence of polio with 21 previously polio-free countries importing wild poliovirus type 1 and four countries (Indonesia, Somalia, Sudan, and Yemen) experiencing outbreaks of more than 100 polio cases.[2] In 2005, there were 1,856 confirmed polio cases compared to less than 500 in 2001.[3] The rapid spread of polio from northern Nigeria, where there was a breakdown of polio vaccination during 2003 and 2004, through west and central Africa to the Horn of Africa, the Arabian Peninsula, and Indonesia, sounds a timely warning to all countries that polio has not yet been eradicated and each country should prepare for the possibility of importation of wild poliovirus.[4] With polio on our doorstep, Australia's surveillance system must be strengthened to enable timely identification of possible cases and rapid control.

As polio has a variable incubation period, generally of 7–10 days but ranging from two days to a month, and approximately 99 per cent of infections are asymptomatic or present as non-specific febrile illnesses, travellers may appear well at their point of entry into a country. Clearly the risk for importation is greatest for countries adjacent to polio-endemic countries, however, migration poses a risk for reintroduction of wild poliovirus to all countries. The risk for local transmission after importation will depend primarily on two factors; vaccination coverage in the local community and living conditions, particularly the frequency of faecal contamination of drinking water supply.

Until recently, Australia's risk of importation of wild poliovirus was considered extremely low. Before the large outbreak of polio in Indonesia that followed importation into West Java in March 2005, the last case of wild polio in Australia's neighbourhood was in a young girl in Cambodia in March 1997.[5] Although many Australians, particularly young adventure tourists, travel to polio-endemic countries, most but not all have been vaccinated against polio. Also, Australia welcomes many visitors from polio-endemic countries or countries with recent polio re-introduction, and accepts refugees who are more likely to have been exposed to infected environments and may be inadequately vaccinated against polio. The increasing trend of placing refugees in rural areas, where there is a demand for unskilled and semi-

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skilled labour, juxtaposes with a greater likelihood of faecal contamination and inadequate treatment of domestic water supply. If these communities have inadequate immunisation coverage, then there is a risk of local transmission.

Given the potential for wild poliovirus importation it is necessary to strengthen Australia's polio surveillance. The occurrence of vaccine-derived poliovirus outbreaks in the Philippines (2001) and Indonesia (2005) provides further impetus for ensuring sensitive surveillance for imported cases in Australia.[6] Acute flaccid paralysis (AFP) surveillance is coordinated by the National Polio Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory where all testing of stools for poliovirus is conducted. The Australian Paediatric Surveillance Unit (APSU) has been funded to assist in surveillance of acute flaccid paralysis the classical clinical syndrome associated with polio disease, since March 1995. More than 1,000 paediatricians and child health specialists report monthly to the APSU on more than 10 disease presentations during the previous month. When there is a positive response a more detailed follow-up questionnaire is sent to the reporting doctor and for AFP cases, a 60 day outcome questionnaire is additionally sent for completion when a final diagnosis is not possible from first questionnaire and laboratory test results. This assists in determining the likelihood of polio where stool surveillance was inadequate as residual paralysis more commonly is associated with polio than other causes of AFP.

AFP surveillance for cases of acute onset of flaccid paralysis in one or more limbs or of bulbar paralysis in any child less than 15 years of age proved its value in meeting the World Health Organization (WHO) benchmark rate of detecting at least one non-polio AFP per 100,000 population less than 15 years of age per annum, required for proving the adequacy of polio surveillance.[7] The passive reporting system that existed in Australia prior to 1995 had failed to meet this target necessary for Australia to be included with the rest of the Western Pacific in being declared polio-free.[8] The monthly return rate of surveillance forms to APSU has consistently exceeded 90 per cent. Australia met the WHO detection target in 2000, 2001 and 2004.[9,10] Detection levels have been heterogeneous between states and territories, and although low reference rates may result in some variability due to chance, the fact that certain states have never met the target suggests that their public health surveillance for AFP is sub-optimal.[11,12]

An effective public health response to importation of wild polio virus may be constrained by the delays in reporting through the current surveillance system. Of considerable concern is performance in meeting the second of the WHO indicators for adequate AFP surveillance, namely that more than 80 per cent of AFP cases should have two adequate stool specimens taken at least 24 hours apart and within 14 days of onset for examination by an accredited Global Polio Network laboratory.[13] Between 2000 and 2005 the stool specimen examination rate has ranged nationally between 24 per cent and 40 per cent, and considerably lower if Queensland performance is excluded. The only timely way to rule out polio as the cause of AFP is adequate laboratory examination of stool specimens that are correctly submitted with due consideration of the need for refrigeration to maintain polio virus. Thus, the current low levels of stool submission and delays in reporting pose a public health threat.

It is necessary to supplement current surveillance with a complementary system that will provide rapid confirmation that AFP cases are not due to wild poliovirus importation. It is not surprising, given the dramatic clinical picture, that a high proportion of children with AFP are hospitalised. For example, 96 per cent (137/143) of notified AFP cases between March 1995 and December 1999 were hospitalised.[8] Therefore, hospital-based notification has great potential for rapidly detecting acute AFP presentations and permitting public health follow up to exclude polio. Queensland has already made notification of AFP the responsibility of hospitals and New South Wales is presently considering the same approach.

Wild poliovirus importations from polio-endemic countries into polio-free areas will continue to occur until endemic transmission is interrupted globally. Ensuring sensitive and timely polio surveillance can limit polio transmission subsequent to importation. This requires that all cases of AFP presenting to hospitals be immediately notified to the local public health unit or state or territory public health authority to ensure a rapid appropriate response, including the immediate dispatch of stool specimens to confirm the diagnosis. In states and territories where local public health capacity is limited, it may be appropriate to train hospital infection control nurses as the primary response agents to confirm the clinical case definition, collect and dispatch the necessary stool specimens observing the 'reverse cold chain', and implement appropriate infection control measures.[14]

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Burden and causes of foodborne disease in Australia: Annual report of the OzFoodNet network, 2005

The OzFoodNet Working Group

Abstract

In 2005, OzFoodNet sites recorded 25,779 notifications of seven potentially foodborne diseases, which was 12.5 per cent higher than the mean for the previous five years. Diseases with significant increases in 2005, when compared to historical reports include: Shiga toxin-producing *Escherichia coli,* shigellosis, haemolytic uraemic syndrome, salmonellosis and campylobacteriosis. The most significant increases were those due to *Salmonella* (13.1%) and *Campylobacter* (5.1%) because of the frequency of these infections. Reports of listeriosis were lower than previous years and there were only four materno-foetal infections compared to seven in 2004. Sites reported 624 outbreaks of gastroenteritis and foodborne disease in 2005. One hundred and two of these were foodborne and affected 1,926 persons, hospitalised 187 and caused four deaths. Among foodborne outbreaks, *Salmonella* Typhimurium was the most common pathogen and restaurants were the most common place where food implicated in outbreaks was prepared. Outbreaks associated with fish, poultry meat, and mixed meat dishes were common. There were several large outbreaks of salmonellosis, including one associated with dips at a Turkish restaurant, one with alfalfa sprouts, and two due to egg-based dishes. In addition, there were several multi-state investigations of *Salmonella* infection during 2005, including one large outbreak of *S.* Typhimurium 135 implicating poultry meat from retail supermarkets. Sites identified a source of infection for 39 per cent (41/104) of investigations into clusters of salmonellosis. Overall, 97.4 per cent of *Salmonella* notifications on state and territory surveillance databases recorded complete information about serotype and phage type. This report highlights the considerable burden of disease from food sources in Australia and the need to continue to improve food safety. *Commun Dis Intell* 2006;30:278–300.

Keywords: surveillance, foodborne disease, disease outbreak, Salmonella, Enteritidis, Campylobacter, Listeria, Shigella, *typhoid*

Introduction

Foodborne disease is a considerable burden on Australian society with 5.4 million cases annually, costing an estimated $1.2 billion dollars.[1] While the majority of cases of foodborne disease are mild and do not require medical attention, the sheer number of affected people taking time from work to recover or care for affected family members make up approximately 60 per cent of these costs. In addition, the costs to food businesses implicated in outbreaks of disease can be significant, although they are difficult to ascertain.[1]

There are over 200 different types of illness that may be transmitted by food, although only a handful are specifically notifiable to health departments.[2] Due to the mild nature of foodborne diseases, most cases do not appear in surveillance statistics collected by health departments. In Australia, for every notification of *Salmonella* and *Campylobacter* there are approximately 6.9 (95% credible interval 4.0–16.4) and 9.6 (95% credible interval 6.2–22.4) cases in the community respectively.[3] The proportion of cases that are notified varies considerably by disease, as the severity of various illnesses differ markedly.[2,3]

In 2005, the OzFoodNet Working Group was (in alphabetical order): Rosie Ashbolt (Tas), Jenny Barralet (Qld), Robert Bell (Qld), Kylie Begg (ACT), Phillipa Binns (NT), Dennis Bittisnich (DAFF), Andrew Black (ACT), Barry Combs (SA), Christine Carson (WA), Scott Crerar (FSANZ), Craig Dalton (HNE Health), Gerard Fitzsimmons (DoHA), Robyn Gibbs (WA), Joy Gregory (Vic), Gillian Hall (NCEPH), Geoff Hogg (MDU), Melissa Irwin (NSW), Martyn Kirk (DoHA), Karin Lalor (Vic), Deon Mahoney (FSANZ), Tony Merritt (HNE Health), Roseanne Muller (NT), Sally Munnoch (HNE Health), Jennie Musto (NSW), Lillian Mwanri (SA), Leonie Neville (NSW), Chris Oxenford (DoHA, NCEPH), Rhonda Owen (DoHA), Raj Patil (DAFF), Nevada Pingault (WA), Jane Raupach (SA), Mohinder Sarna (WA), Mark Salter (FSANZ), Cameron Sault (Tas), Craig Shadbolt (NSWFA), Russell Stafford (Qld), Nicola Stephens (Tas), Barbara Telfer (NSW) Hassan Vally (NCEPH, WA), Tory Worgan (HNE Health), Kefle Yohannes (DoHA)

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Health departments use surveillance of infectious diseases for observing trends, preventing further spread of infections, detecting outbreaks and monitoring the effects of interventions.[4] The source of infection is difficult to determine in sporadic cases of enteric diseases as they may be acquired from infected persons, animals, contaminated water or foods and other sources within the environment. In outbreaks of enteric diseases the modes of transmission are more likely to be determined. Where these outbreaks are foodborne they can be useful for developing policy to prevent further disease.[5]

In 2000, the Australian Government Department of Health and Ageing established the OzFoodNet network to enhance surveillance for foodborne disease.[6] This built upon an 18-month trial of active surveillance in the Newcastle region of New South Wales. OzFoodNet was modelled on the Centers for Disease Control and Prevention's FoodNet surveillance system. The OzFoodNet network consists of epidemiologists employed by each state and territory health department to conduct investigations and applied research into foodborne disease. The network involves many different collaborators, including the National Centre for Epidemiology and Population Health, and the Public Health Laboratory Network. OzFoodNet is a member of the Communicable Diseases Network Australia, which is Australia's peak body for communicable disease control.[7] The Australian Government Department of Health and Ageing funds OzFoodNet and convenes committees to manage the network, and a committee to review the scientific basis for various research projects.

This is the fifth annual report of OzFoodNet and covers data and activities for 2005.

Methods

Population under surveillance

In 2005, the coverage of the network included the entire Australian population, which was estimated to be 20,328,609 persons.[8] In 2005, the Hunter New England Area Health Service hosted an OzFoodNet site, which supplemented statewide foodborne disease surveillance across New South Wales.

Data sources

Rates of notified infections

All Australian states and territories require doctors and/or pathology laboratories to notify patients with infectious diseases that are important to public health. Western Australia is the only jurisdiction where laboratory notification is not mandatory under legislation, although most laboratories still notify the health department by agreement. OzFoodNet aggregated and analysed data on patients notified with the following diseases or conditions, a proportion of which may be acquired from food:

- *Campylobacter* infections;
- Non-typhoidal *Salmonella* infections;
- *Listeria* infections;
- Shiga toxin producing *Escherichia coli* infections and haemolytic uraemic syndrome;
- typhoid; and
- *Shigella* infections.

To compare notifications in 2005 to historical totals, we compared crude numbers and rates of notification to the mean of the previous five years. Where relevant, we used data from the National Notifiable Diseases Surveillance System (NNDSS) and OzFoodNet sites to analyse data for specific subtypes of infecting organisms.

The date that notifications were received by each jurisdiction was used for analysis. To calculate rates of notification, we used the estimated resident populations for each state or territory as at June 2005.[8] For cases of neonatal listeriosis infections we used birth data from the Australian Institute of Health and Welfare.[9]

Gastrointestinal and foodborne disease outbreaks

OzFoodNet collected information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2005. An outbreak of foodborne disease was defined as an increase in the number of reports of a particular infection or illness associated with a common food or meal. A cluster was defined as an increase in infections that were epidemiologically related in time, place or person where investigators were unable to implicate a vehicle or determine a mode of transmission. An example is a temporal or geographic increase in the number of cases of a certain type of *Salmonella* serovar or phage type. Another example is a community-wide increase of cryptosporidiosis that extends over some weeks or months. In this category, some outbreaks where the mode of transmission was indeterminate have been included.

OzFoodNet epidemiologists collate summary information about the setting where the outbreak occurred, where food was prepared, the month the outbreak occurred, the aetiological agent, the number of persons affected, the type of investigation conducted, the level of evidence obtained and the food vehicle responsible for the outbreak. To summarise the data, we categorised outbreaks by aetiological agents, food vehicles and settings where the implicated food was prepared. Data on outbreaks due to transmission from animals and cluster investigations were also

summarised. The number of outbreaks and documented causes may vary from summaries published by individual jurisdictions.

Surveillance evaluation

OzFoodNet compared the results of surveillance across different sites, including rates of reporting outbreaks, and investigation of clusters of *Salmonella*. To measure the quality of national surveillance data, OzFoodNet examined the completeness of information on state and territory databases in 2005. The proportions of *Salmonella* notifications with serotype and phage type information were compared with results for previous years.

Results

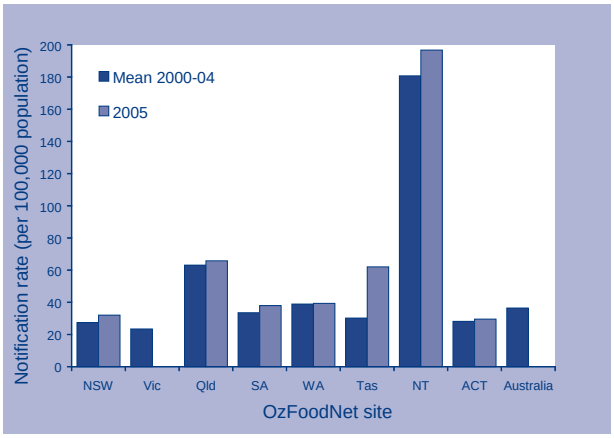
Rates of notified infections

In 2005, OzFoodNet sites reported 25,779 notifications of seven diseases that were potentially foodborne. This was a 12.5 per cent increase from the mean of 22,827 notifications for the previous five years. Reports for these seven diseases make up almost a quarter of notifications to the National Notifiable Diseases Surveillance System.[10] A summary of the number and rates of notifications by OzFoodNet sites is shown in Appendix 1.

Salmonella *infections*

In 2005, OzFoodNet sites reported 8,376 cases of *Salmonella* infection, which equated to 41.2 cases per 100,000 population and an increase of 13.1 per cent from the mean for the previous five years (Figure 1). The rates ranged from 28.3 cases per 100,000 population in Victoria to 196.8 cases per 100,000 population in the Northern Territory, which traditionally has the highest rates of all jurisdictions.

Figure 1. Notification rates of *Salmonella* infections, 2005, compared to the mean of the notification rate (2000–2004), by OzFoodNet site



Overall, notification rates of salmonellosis for 2005 were increased in all states and territories, particularly in Tasmania (105.3%), Victoria (20.8%) and New South Wales (17.0%) compared to historical means. The major increase in Tasmania was due to large outbreaks of *S.* Typhimurium 135 in November and December 2005.

The male to female ratio for salmonellosis was 1:1. The highest age-specific rate of *Salmonella* infection was 200.8 cases per 100,000 population in males aged 0–4 years. Notification rates were also elevated in the 5–9 year age group with a further peak in notification rates in the 20–29 year age group.

Rates of salmonellosis were highest in northern areas of Australia. The highest rate is consistently reported in the Kimberley region of Western Australia.[8] Western Australia reported that the Kimberley region had a rate of 262 per 100,000 population, which represents a 17 per cent decrease for the regional notification rate from the previous year. In Western Australia, rates of salmonellosis were higher in Indigenous people in all age groups, particularly in children aged 0–4 years. In the Northern Territory, Indigenous people had 1.8 times the rate of salmonellosis notifications compared to non-Indigenous people with the highest burden amongst the 0-4 year age group who had 1.4 times the rate of non-Indigenous children in the same age group.

During 2005, the most commonly reported *Salmonella* serotype was *S.* Typhimurium. There were 836 notifications of *Salmonella* Typhimurium 135 (including a subgroup locally designated 135a) to OzFoodNet sites making it the most common infection (Table 1). This compared to 578 notifications of this phage type in 2004. *Salmonella* Typhimurium 197 increased dramatically in 2005 with 536 notifications, which was a 102 per cent increase from 266 notifications in 2004. The highest specific rates for single subtypes reported by OzFoodNet sites were *S.* Typhimurium 135 and *S.* Mississippi in Tasmania, and *S.* Ball and *S.* Saintpaul in the Northern Territory with rates of 36.3, 12.2, 23.7, and 23.7 per 100,000 population, respectively. These subtype-specific rates were almost as high as the total rate of *Salmonella* notifications in some other jurisdictions.

Salmonella *Enteritidis*

S. Enteritidis is a serotype that can infect the internal contents of eggs through the oviducts of infected chickens, predominantly with *S.* Enteritidis phage type 4.[11,12] People may often become infected with this serotype after eating raw or undercooked eggs. This phage type has caused major problems in the northern hemisphere where it has become established in commercial egg laying flocks, although the incidence has declined in many countries.[11,12]

Table 1. Numbers, rates and proportions of the top 5 *Salmonella* infections, 2004 to 2005, by OzFoodNet site*

OzFoodNet site	*Salmonella* type (sero/phage type)	Top 5 infections					
		2005 n	Rate†	Proportion‡ (%)	2004 n	Rate	Ratio§
Australian Capital Territory	Typhimurium 170/108	14	4.3	14.6	31	9.6	0.5
	Typhimurium 135	13	4.0	13.5	5	1.5	2.6
	Typhimurium 9	10	3.1	10.4	6	1.9	1.7
	Stanley	5	1.5	5.2	2	0.6	2.5
	Hvittingfoss	4	1.2	4.2	0	0.0	–
	Typhimurium 44	4	1.2	4.2	0	0.0	–
New South Wales	Typhimurium 170/108	373	5.5	17.2	351	5.2	1.1
	Typhimurium 9	154	2.3	7.1	108	1.6	1.4
	Typhimurium 197	109	1.6	5.0	43	0.6	2.5
	Typhimurium 135	181	2.7	8.3	178	2.6	1.0
	Birkenhead	82	1.2	3.8	77	1.1	1.1
Northern Territory	Ball	48	23.7	12.0	50	25.0	1.0
	Saintpaul	48	23.7	12.0	48	24.0	1.0
	Litchfield	21	10.4	5.3	15	7.5	1.4
	Weltevreden	15	7.4	3.8	8	4.0	1.9
	Chester	12	5.9	3.0	12	6.0	1.0
	Kinondoni	10	4.9	2.5	6	3.0	1.7
Queensland	Saintpaul	276	7.0	10.6	225	5.8	1.2
	Virchow 8	190	4.8	7.3	247	6.4	0.8
	Typhimurium 197	145	3.7	5.6	145	3.7	1.0
	Typhimurium 135	137	3.5	5.3	185	4.8	0.7
	Aberdeen	135	3.4	5.2	118	3.0	1.1
	Hvittingfoss	135	3.4	5.2	110	2.8	1.2
South Australia	Typhimurium 9	57	3.7	9.7	46	3.0	1.2
	Infantis	48	3.1	8.2	17	1.1	2.8
	Typhimurium 64	47	3.0	8.0	4	0.3	11.8
	Typhimurium 135	47	3.0	8.0	44	2.9	1.1
	Typhimurium 170/108	33	2.1	5.6	70	4.6	0.5
Tasmania	Typhimurium 135	176	36.3	58.5	2	0.4	88.0
	Mississippi	59	12.2	19.6	63	13.1	0.9
	Typhimurium 9	10	2.1	3.3	4	0.8	2.5
	Typhimurium 170/108	7	1.4	2.3	3	0.6	2.3
	Typhimurium 44	5	1.0	1.7	0	0.0	–
Victoria	Typhimurium 197	279	5.6	19.6	59	1.2	4.7
	Typhimurium 135	191	3.8	13.4	137	2.8	1.4
	Typhimurium 9	118	2.3	8.3	145	2.9	0.8
	Typhimurium 170/108	63	1.3	4.4	88	1.8	0.7
	Typhimurium 44	50	1.0	3.5	7	0.1	7.1
Western Australia	Oranienburg	63	3.1	8.0	5	0.3	12.6
	Typhimurium 135	69	3.4	8.7	74	3.7	0.9
	Enteritidis 6A	35	1.7	4.4	21	1.1	1.7
	Saintpaul	32	1.6	4.0	46	2.3	0.7
	Muenchen	30	1.5	3.8	23	1.2	1.3

Table 1. Numbers, rates and proportions of the top 5 *Salmonella* infections, 2004 to 2005, by OzFoodNet site,* *continued*

OzFoodNet site	*Salmonella* type (sero/ phage type)	Top 5 infections					
		2005 n	Rate†	Proportion‡ (%)	2004 n	Rate	Ratio§
Australia	Typhimurium 135	836	4.1	10.0	578	2.9	1.4
	Typhimurium 197	536	2.6	6.4	266	1.3	2.0
	Typhimurium 170/108	535	2.6	6.4	647	3.2	0.8
	Saintpaul	434	2.1	5.2	395	2.0	1.1
	Typhimurium 9	428	2.1	5.1	360	1.8	1.2

* Where there were multiple fifth ranking *Salmonella* types all data have been shown, giving more than five categories for some sites.

† Rate per 100,000 population.

‡ Proportion of total Salmonella notified for this jurisdiction in 2005.

§ Ratio of the number of reported cases in 2005 compared to the number reported in 2004.

S. Typhimurium 135 includes a local variant phage type 135a, which is not a recognised international classification.

Australia is largely free of *S.* Enteritidis phage type 4 infections except in people returning from overseas. There are other phage types of *S.* Enteritidis that are endemic in Australia, although the sources of these local infections are poorly understood.

In 2005, OzFoodNet concluded data collection for a case control study of *S.* Enteritidis infections to determine the risk factors for infection. OzFoodNet epidemiologists enrolled cases of *S.* Enteritidis that were acquired in Australia between 2001 and 2005 to assess food-based and zoonotic risk factors for infection and compare them to population-based controls. The results of this study are still being collated for analysis.

During 2005, OzFoodNet sites recorded 387 cases of *S.* Enteritidis, of which 84 per cent (289/343) had travelled overseas (Table 2). Relevant travel histories were difficult to obtain, as people had often travelled to several countries before returning to Australia. Asian countries were commonly mentioned, and reflect that they are common travel destinations for Australians. In the Asian region, cases of *S.* Enteritidis infection reported travelling to Bali (37%), Singapore (9%), Indonesia (9%), and Thailand (9%). Travel history could not be determined for 11 per cent (44/387) of cases. The most common infecting phage types were 6a (76 cases), 1b (38), 1 (28) and 4 (21).

Overall, 14 per cent (54/387) of patients infected with *S.* Enteritidis acquired their infection in Australia. The median age of cases was 29 years (age range 0.3–96 years) and 35 per cent were male. Locally-acquired *S.* Enteritidis infections predominantly occurred in Queensland, where 76 per cent (41/54) of all locally-acquired infections were reported. Most locally-acquired infections in Queensland were due to phage type 26 (Table 3). Locally-acquired *S.* Enteritidis infections are strongly seasonal and infections decreased markedly in the winter of 2005 (Figure 2).

Table 2. Number of *Salmonella* Enteritidis infections, 2005, by travel history and state or territory

OzFoodNet site	History of travel overseas			Total
	Yes	No	Unknown	
Australian Capital Territory	8	1		9
New South Wales	67	6	20	93
Northern Territory			1	1
Queensland	20	41	19	80
South Australia	20	1	2	23
Tasmania	2	1		3
Victoria	71	3	2	76
Western Australia	101	1		102
Total	289	54	44	387

Figure 2. *Salmonella* Enteritidis infections acquired in Australia, 2003–05, by phage type and month of notification

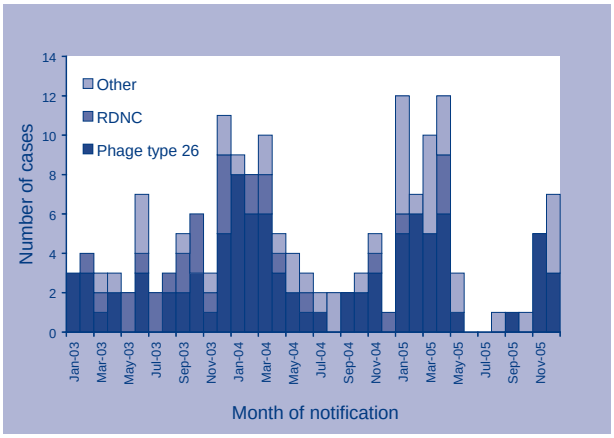


Table 3. Number of locally-acquired *Salmonella* Enteritidis infections, 2005, by phage type and state or territory

Phage type	State or territory							Total
	ACT	NSW	Qld	SA	Tas	Vic	WA	
1		1						1
4			1					1
7	1							1
13			1					1
26			29		1		1	31
14 var			1					1
1B		1						1
21B var				1				1
26 var						2		2
26 var/26						1		1
4B		1						1
6A		3						3
RDNC*			3					3
RDNC/12			1					1
Untypable			5					5
Total	1	6	41	1	1	3	1	54

* 'Reaction Does Not Conform' (RDNC) represents phage type patterns that are not yet assigned.

Salmonella *clustering*

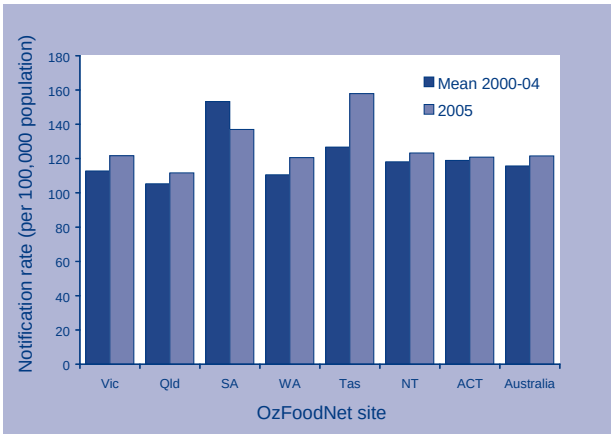
In total, state and territory health departments conducted 104 investigations into clusters and point source outbreaks of salmonellosis during 2005. A source of infection was identified for 39 per cent (41/104) of these investigations. Approximately 61 per cent (63/104) of these outbreaks were due to various phage types of *S.* Typhimurium.

Campylobacter *infections*

In 2005, OzFoodNet sites reported 16,479 cases of *Campylobacter* infection, equating to a rate of 121.6 cases per 100,000 population. This rate represented a five per cent increase over the mean for the previous five years (Figure 3). Tasmania, experienced the greatest increase, with the notification rate in 2005 being 27 per cent above the mean of the previous five years. The only state to experience a decrease in notification rate was South Australia (-11%). The highest and lowest rates of *Campylobacter* notification were in Tasmania (157.9 cases per 100,000 population) and in Queensland (111.7 cases per 100,000 population). Data for campylobacteriosis were not available for New South Wales.

Rates of *Campylobacter* infection were consistently high in all age groups in all jurisdictions. The highest rate of notifications was in males in the 0–4 year age group (268 cases per 100,000 population), with a secondary peak in the 20–29 year age group for both males and females. Fifty-five per cent of notified cases were male. There were 12 identified outbreaks of *Campylobacter* during 2005, nine of which were suspected to be foodborne.

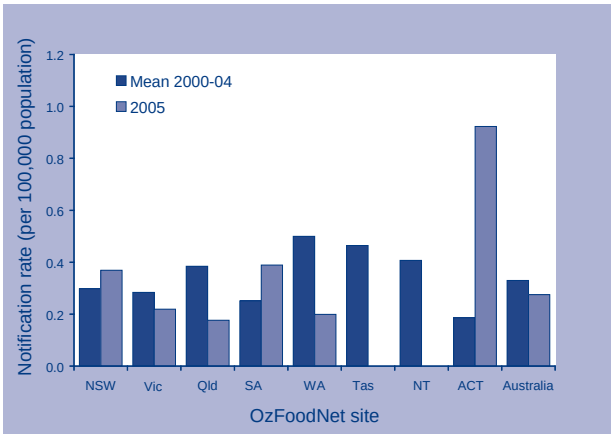
Figure 3. Notification rates of *Campylobacter* infections, Australia, 2005, compared to mean rates for 2000 to 2004, by OzFoodNet site excluding New South Wales



Listeria

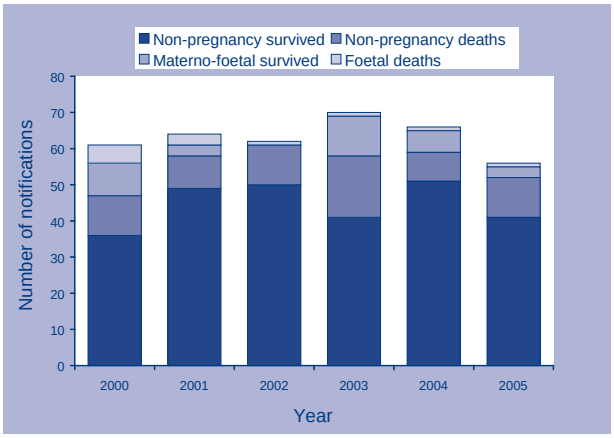
OzFoodNet sites reported 56 cases of listeriosis in 2005, which represents a notification rate of 0.3 cases per 100,000 population (Figure 4). This was a 17 per cent decrease in the notification rate compared to the five-year historical mean. South Australia investigated a common source outbreak of listeriosis associated with cold meats. The Australian Capital Territory investigated three cases during 2005, although no common source was identified.

Figure 4. Notification rates of *Listeria* infections, Australia, 2005, compared to mean rates for 2000–2004, by OzFoodNet site



Four materno-foetal infections were reported during 2005, giving a rate of 1.6 cases per 100,000 births. The rate of materno-foetal infections has been steadily declining in recent years. Victoria, Western Australia, New South Wales and Queensland each reported single cases in neonates during 2005. Twenty-five per cent (1/4) of infected neonates died during 2005 (Figure 5).

Figure 5. Notifications of *Listeria* showing non-pregnancy related infections and deaths, and materno-foetal infections and deaths, Australia, 2000 to 2005

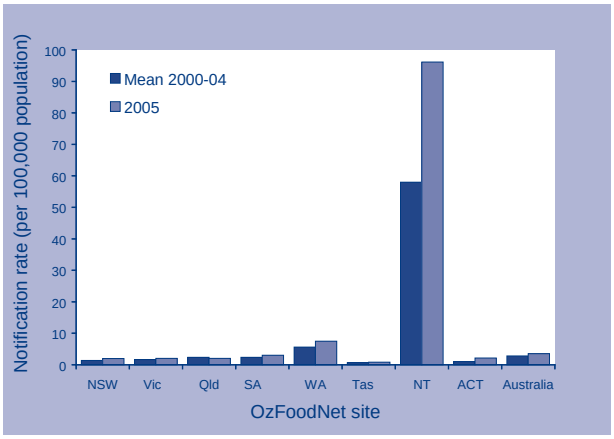


Ninety-three per cent (52/56) of infections during 2005 were reported in persons who were either elderly and/or immunocompromised. Among non-pregnancy related cases, the male to female ratio was approximately 1:1. The highest age specific rate was 1.6 cases per 100,000 population, reported in males in the 60–64 years age group and females over the age of 75 years. Twenty-seven per cent (11/52) of non-pregnancy associated cases died, which was similar to previous years. However, it is difficult to establish whether listeriosis is the cause of death as many cases have terminal illness due to immunocompromising conditions.

Shigella

OzFoodNet sites reported 721 cases of shigellosis during 2005, which equated to a notification rate of 3.5 cases per 100,000 population (Figure 6). This was a 26 per cent increase in the rate of notification compared with historical averages, after adjusting for the introduction of notifications from New South Wales in January 2001.

Figure 6. Notification rates of *Shigella* infections, Australia, 2005, compared to mean rates for 2000 to 2004, by OzFoodNet site*



* Shigellosis became notifiable in New South Wales from 2001 onwards.

The highest rate of notification was in the Northern Territory (96 cases per 100,000 population), which was almost 30 times higher than the overall Australian rate. Rates of shigellosis are considerably higher in Indigenous communities, which is reflected in the rates of states and territories with higher proportions of Indigenous peoples in the general population. In Western Australia, the rates of shigellosis were in excess of 300 cases per 100,000 population in Indigenous people aged 0–4 years and 75 years or older.

Overall, the notification rate for shigellosis was elevated in all jurisdictions, except for Queensland which had 7.2 per cent fewer notifications than the previous five years. The male to female ratio of shigellosis cases was approximately 1:1. The highest age specific notification rates were in males and females in the 0–4 year age group, with 19.1 and 16.6 cases per 100,000 population, respectively. There was one small outbreak of shigellosis of unknown mode of transmission in New South Wales in July 2005.

In 2005, *Shigella sonnei* biotypes a and g were the most common strains infecting people, with 167 and 136 notifications respectively. Mannitol negative *Shigella flexneri* 4a also increased in Central Australia during February and March 2005. These increases were particularly noted in South Australia and the Northern Territory. In Australia, the mode of transmission for the majority of shigellosis infections was through person-to-person transmission or were acquired overseas.

Typhoid

OzFoodNet sites reported 52 cases of typhoid infection during 2005, representing an overall notification rate of 0.3 cases per 100,000 population (Figure 7). The notification rate decreased 22 per cent when compared to the five year historical mean. The highest rates were reported in New South Wales and Western Australia with rates of 0.4 and 0.3 cases per 100,000 population respectively. Tasmania, the Northern Territory and the Australian Capital Territory did not report any cases.

Where travel status was known, sites reported that 96 per cent (45/47) of typhoid cases had recently travelled overseas (Table 4). Thirty per cent (14/47) of these cases had recently travelled from Indonesia or Bali where the predominant phage types were A (3 cases), D2 (2 cases) and E2 (2 cases). Twenty cases had travelled to the Indian subcontinent and the predominant phage type of *S. Typhi* was E1a (5 cases). The two non-travelling cases were either long-term carriers or infected by close contact with a known carrier. Travel status was unknown for five cases. Information on phage type was reported for 81 per cent (42/52) of isolates.

Figure 7. Notification rates of typhoid infections, Australia, 2005, compared to mean rates for 2000 to 2004, by OzFoodNet site

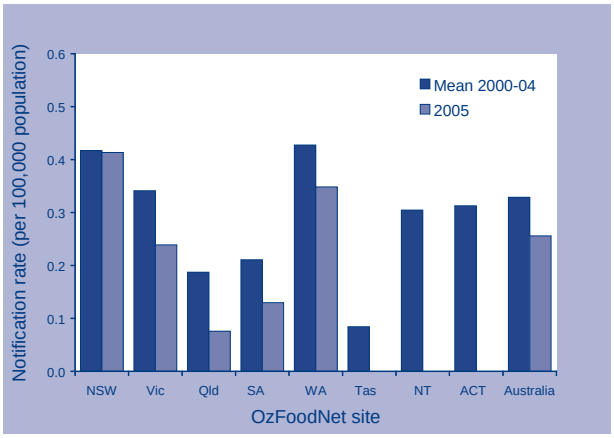


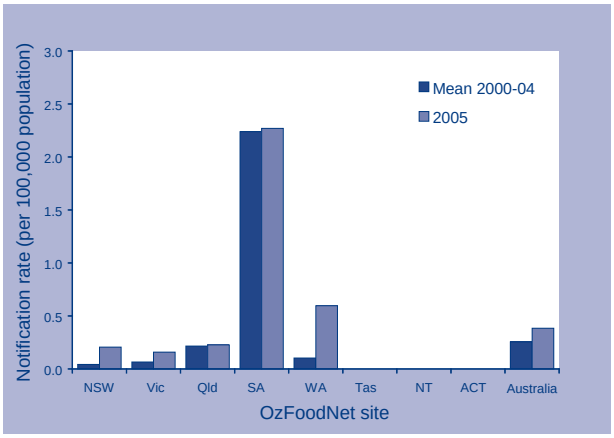
Table 4. Travel status for notified typhoid cases, Australia, 2005

Country	Number of cases	Predominant phage type (cases)
Africa	1	A (1)
Locally acquired	2	E1a (1), untypable (1)
Bali	1	Degraded (1)
Bangladesh	4	E1 (1), E1a (1), E7 (1), unknown (1)
Cambodia	1	E1A (1)
China	1	Unknown (1)
Guinea	1	A (1)
India	12	A (1), E1 (1), E1a (4), E9 (1), E2 (1), untypable (1), degraded (1), Unknown (2)
Indonesia	13	A (3), D2 (2), E2 (2), degraded (1), untypable (2), unknown (3)
Malaysia	1	D2 (1)
Nepal	1	Unknown (1)
Pakistan	3	M1 (2), unknown (1)
Samoa	3	E1a (1), E1 (1), E7 (1)
South America	1	A (1)
Sri Lanka	1	Degraded (1)
Tanzania	1	A (1)
Unknown	5	D2 (1), E1a (2), E2 (1), unknown (1)

Shiga toxin-producing Escherichia coli *infections*

OzFoodNet sites reported 78 cases of Shiga toxin-producing *E. coli* (STEC) infection during 2005, compared to 50 for 2004. These numbers do not include cases of haemolytic uraemic syndrome (HUS) where an STEC organism was isolated or detected in stool samples, as they are reported separately under the category of HUS. The notification rate of 0.4 cases per 100,000 population was a 50.8 per cent increase over the mean rate for previous years (Figure 8). The elevated number of cases reported in 2005 was the result of enhanced screening for STEC in bloody stools in some jurisdictions, such as Western Australia, Victoria, and the Hunter – New England area of New South Wales. Previously, only South Australia has had a program of testing stools containing blood for STEC, which accounts for the consistently high rate of notification in this State.

Figure 8. Notification rates of Shiga toxin-producing *Escherichia coli* infections, 2005, compared to mean rates for 2000–2004, by OzFoodNet site



South Australia (35 cases) reported the majority of cases and had the highest rate of notification of 2.3 cases per 100,000 population. All sites reporting cases had significant increases in the number of cases notified, except for Queensland and South Australia where the notification rates were similar to previous years. There were no cases reported from Tasmania, the Australian Capital Territory or the Northern Territory during 2005. The male to female ratio of cases was 0.8:1, contrasting with a male: female ratio of 0.5:1 in 2004. In 2005, the highest rate of reported infection was in females aged 5–9 and 45–49 years, with a rate of 0.8 cases per 100,000 population in both these age groups. The highest rate reported for males was 0.7 per 100,000 population in the 20–24 years age group.

E. coli serotype O157 was responsible for 39 per cent (15/38) of infections where serotype information was available in 2005, compared to 52 per cent in 2004. *E. coli* O111 was the second most common serotype and was responsible for 26 per cent (10/38) of reports compared to 15 per cent (5/33) in 2004 (Table 5). In 2005, twice as many notified cases of *E. coli* O157 were female compared to males.

Table 5. Number of notified cases of Shiga toxin-producing *Escherichia coli*, 2005, by state and serotype

Serotype	State					Total
	NSW	Qld	SA	Vic	WA	
O157	2	2	5	4	2	15
O111	1	1	7	0	1	10
O26	0	3	1	2	0	6
O113	0	0	3	0	0	3
O103	0	0	1	0	0	1
O77	0	0	0	1	0	1
O112	0	0	1	0	0	1
O166	0	1	0	0	0	1
Non-O157 non-O111	0	0	0	0	9	9
Unknown	11	2	17	1	0	31
Total	14	9	35	8	12	78

There were two clusters of cases investigated during 2005, both of which occurred in the community in South Australia. The mode of transmission and source were not identified for either cluster. In the first cluster, three serotype O111 cases with similar pulsed-field gel electrophoresis (PFGE) patterns attended the same church, but other links were not identified. One of these cases had HUS and another was a sibling of the HUS case. In a cluster of nine cases in November, there were a range of different serotypes including two O111 isolates with identical PFGE patterns.

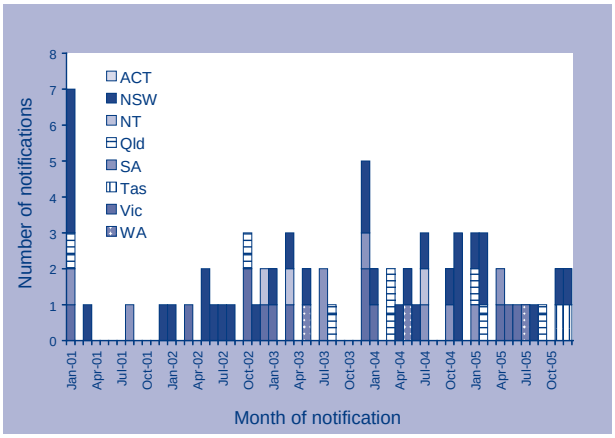
The serotype was not identified in 51 per cent (40/78) of cases as polymerase chain reaction (PCR) tests are commonly used for diagnosis. These PCR tests detect the presence of toxin producing genes, and serotype-specific PCR tests only detect serotypes O157, O111 and O113. Culture of *E. coli* is not routinely carried out. In South Australia, the Hunter and Western Australia only stools containing macroscopic blood were screened for Shiga toxins 1 and 2 genes, unless specifically requested by the treating doctor. 'H' typing information was available for only 34 per cent (16/47) of isolates that were serotyped in 2005. There were six infections due to

E. coli O157:H-, five due to *E. coli* O26:H11, two due to *E. coli* O157:H7, one each of serotypes O111:H-, O166:H15, and O77:H28.

Haemolytic uraemic syndrome

There were 17 cases of haemolytic uraemic syndrome reported during 2005, which was a rate of 0.1 case per 100,000 population. This compared to 16 cases of HUS in 2004. New South Wales reported six of these cases, Victoria and Queensland both reported three cases each, Queensland and Tasmania both reported two cases each, and Western Australia reported 1 case in 2005 (Figure 9).

Figure 9. Numbers of notified cases of haemolytic uraemic syndrome, Australia, 2001 to 2005, by month of notification and state or territory

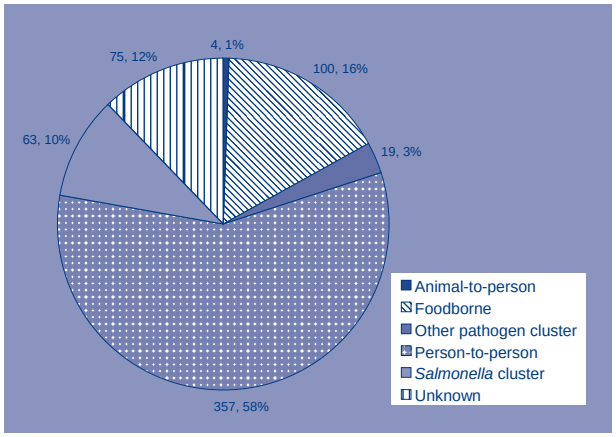


Sixty-five per cent of cases were male. The highest rates of notification were in males and females aged 0–4 years, with rates of 1.2 and 0.7 cases per 100,000 population respectively. Sites reported that STEC were detected in the faeces of 53 per cent (9/17) of cases. Three cases were infected with serotype O111, two cases were infected with O157; one was OR:H- and three cases were STEC positive by PCR. One notified case was due to a non-enteric pathogen—*Streptococcus pneumoniae*. There was some clustering of HUS cases in 2005, with Tasmania investigating two apparently linked cases of *E. coli* O157:H- 54(var) in November and December, although no source was identified.

Gastrointestinal and foodborne disease outbreaks

During 2005, OzFoodNet sites reported 624 outbreaks of gastrointestinal illness affecting 10,865 persons. The mode of transmission for 57 per cent (358/624) of outbreaks was suspected to be person-to-person transmission (Figure 10).

Figure 10. Foodborne and gastroenteritis outbreaks reported by OzFoodNet sites, Australia, 2005, by mode of transmission (*n*=624 outbreaks)



These person-to-person outbreaks were responsible for 66 per cent (7,222/10,865) of all persons affected by outbreaks and three deaths. Forty-six per cent (163/358) of the person-to-person outbreaks occurred in aged care facilities, while 23 per cent (84/358) and 12 per cent (42/358) of outbreaks occurred in child care and hospital settings, respectively. Thirty-seven per cent (134/358) of person-to-person outbreaks were caused by norovirus, while 51 per cent (183/358) were of unknown aetiology, many of which were suspected to be due to a viral pathogen.

Sites conducted investigations into 147 different clusters or point source outbreaks where the mode of transmission was not determined, including 63 clusters due to various strains of *Salmonella.* Four outbreaks were suspected to be due to animal-to-person infection, three of these were due to *Salmonella* and one was due to *Cryptosporidium*.

Foodborne disease outbreaks

In 2005, there were 102 foodborne disease outbreaks giving an overall rate of 5.0 outbreaks per million population. These outbreaks affected 1,975 persons, hospitalised 166 persons and caused four deaths. A summary description of all foodborne outbreaks is shown in Appendix 2.

Queensland reported the largest number of outbreaks (31%, 32/102 of all outbreaks reported) (Table 6). The reporting rates of foodborne outbreaks for different OzFoodNet sites ranged from 0.7 per million population in New South Wales to 15.4 per million population in the Australian Capital Territory. The majority of outbreaks occurred in summer and autumn (Figure 11).

Aetiological agents

The most common agent responsible for foodborne disease outbreaks was *Salmonella*, which caused 32 per cent (33/102) of outbreaks (Table 7). These outbreaks affected a total of 1,200 persons with a hospitalisation rate of 13 per cent (150/1,200). *S.* Typhimurium was responsible for 79 per cent (26/33) of foodborne *Salmonella* outbreaks. Four fatalities were reported from three separate outbreaks of *Salmonella,* two of which occurred in aged care homes and one other occurred in an institutional setting. The highest hospitalisation rate was for listeriosis although this was only one small outbreak.

Figure 11. Outbreaks of foodborne disease, Australia, 2001 to 2005, by selected aetiological agents

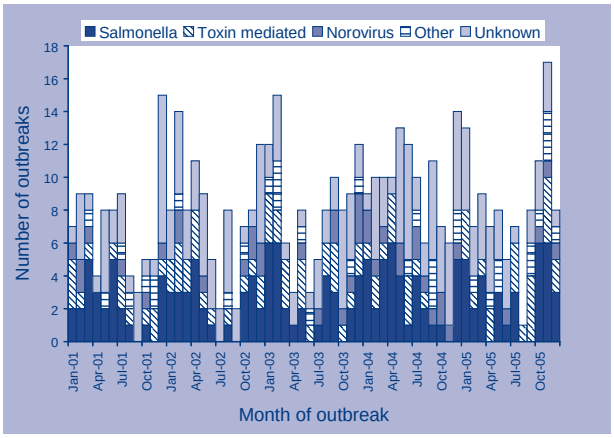


Table 6. Outbreaks of foodborne disease in Australia, 2005, by OzFoodNet site

State or territory	Number of outbreaks	Persons affected	Mean size (persons)	Hospitalised	Fatalities	Outbreaks per million population
Australian Capital Territory	5	51	10.2	4	0	15.4
New South Wales	19	246	12.9	24	1	0.7
Northern Territory	2	9	4.5	1	0	9.9
Queensland	32	292	9.1	69	3	8.1
South Australia	6	163	27.2	5	0	3.9
Tasmania	6	205	34.2	10	0	12.4
Victoria	27	808	29.9	40	0	5.4
Western Australia	5	198	39.6	13	0	2.5
Total	102	1,975	19.4	166	4	5.0

Table 7. Aetiological agents responsible for foodborne disease outbreaks, number of outbreaks and persons affected, Australia, 2005

Agent category	Number of outbreaks	Persons affected	Mean outbreak size (persons)	Hospitalised	Fatalities
Campylobacter sp.	9	93	10.3	2	0
Ciguatera	10	57	5.7	2	0
Clostridium perfringens	4	76	19.0	0	0
Histamine poisoning	5	12	2.4	0	0
Listeria monocytogenes	1	3	3.0	3	
Norovirus	4	91	22.8	2	0
Salmonella other	7	180	25.7	24	4
Salmonella Typhimurium	26	1,020	39.2	126	0
Staphylococcus aureus	2	4	2.0	0	0
Vibrio parahaemolyticus	1	2	2.0	0	0
Unknown	33	437	13.2	7	0
Total	102	1,975	19.4	166	4

Fifteen of the 21 outbreaks of illness due to toxins in 2005 were related to contaminated fish. Outbreaks of ciguatera and histamine poisoning, were small with a mean of 5.7 and 2.4 persons affected respectively. There were four outbreaks of *Clostridium perfringens* intoxication and two of *Staphylococcus aureus* intoxication. There were nine outbreaks of *Campylobacter* affecting 93 people, and one outbreak of vibriosis affecting two people. There were four outbreaks of norovirus affecting 91 people. Thirty-two per cent (33/102) of outbreaks were of unknown aetiology, which affected 437 persons including seven cases who were hospitalised.

Food vehicles

There was a wide variety of foods implicated in outbreaks of foodborne disease during 2005 (Table 8), although investigators could not identify a specific food vehicle in 30 per cent (31/102) of outbreaks. Contaminated fish was the most common food vehicle and was responsible for 16 per cent (16/102) of outbreaks. Ten were due to ciguatera fish poisoning and five due to small outbreaks of histamine poisoning. Queensland reported nine of the ciguatera outbreaks from locally-caught fish, with Victoria reporting one ciguatera outbreak caused by fish sourced from Fiji. Four out of five outbreaks of histamine poisoning were associated with the consumption of tuna, with the remaining outbreak associated with an unknown species of fish.

Table 8. Categories of food vehicles implicated in foodborne disease outbreaks, Australia, 2005

Agent category	Number of outbreaks	Persons affected	Hospitalised
Fish	16	80	2
Mixed meat dish	9	152	19
Poultry	9	76	4
Sauces and gravy	6	125	11
Mixed dish	4	38	4
Cakes	3	129	13
Dessert	3	56	34
Dips	3	475	26
Sandwiches	3	123	0
Seafood	3	57	22
Suspected eggs	3	28	2
Egg-based dishes	2	11	2
Salad dishes	2	162	12
Water	2	34	2
Pizza	1	9	0
Pork	1	25	1
Suspected water	1	22	0
Unknown	31	373	12
Total	102	1,975	166

Poultry and mixed meat dishes were responsible for nine outbreaks each. Sauces and gravies were implicated as the cause of six outbreaks, which included four outbreaks relating to eggs. Egg-based dishes caused two outbreaks, and a further three outbreaks were suspected as being due to eggs. In addition, there were two outbreaks due to desserts containing raw eggs; and two due to cakes and one due to sandwiches where cross contamination from eggs was suspected. In total, investigators identified 14 outbreaks of salmonellosis where eggs were suspected or proven to be the actual source of contamination of the implicated food.

There were two outbreaks associated with drinking water, one of which was associated with a municipal water supply. There were three outbreaks due to dips, including one very large outbreak associated with food served at a Turkish restaurant in Victoria. Outbreaks due to desserts had the highest hospitalisation rate, with 61 per cent (34/56) of people affected in three outbreaks being admitted to hospital.

Outbreak settings

The most common settings where food was prepared in outbreaks was at restaurants (33%), followed by the home (12%), events catered for by professional companies (11%) and aged care homes (8%) (Table 9). Foods that were contaminated in primary production environments, such as fish contaminated with ciguatoxin, were classified as 'primary produce' and were responsible for 12 per cent of outbreaks. Food prepared in bakeries and at takeaway stores were responsible for five outbreaks each, while food prepared at school camps was responsible for three outbreaks. The setting where people ate the food was similar to where it was prepared. There were 11 outbreaks in aged care homes, two of which were due to food prepared elsewhere and one was suspected to be due to contaminated tank water.

Investigative methods and levels of evidence

States and territories investigated 24 outbreaks using retrospective cohort studies and 10 outbreaks using case control studies, with one investigation using both methodologies. Forty-two per cent (10/24) of cohort studies were used for outbreaks of unknown aetiology, which is similar to previous years. Thirty-eight per cent (9/24) of investigations using cohort studies were for *Salmonella* outbreak investigations. Sixty-five outbreaks relied on descriptive information

Table 9. Settings where food implicated in disease outbreaks was prepared, Australia, 2005

Setting category	Number of outbreaks	Persons affected	Hospitalised
Restaurant	34	956	73
Private residence	20	180	40
Commercial caterer	11	218	10
Aged care	8	117	3
Takeaway	5	19	4
Bakery	5	141	13
Camp	3	32	0
Hospital	2	14	3
Institution	2	40	4
Other	2	36	4
Primary produce	2	7	0
Grocery store/ delicatessen	2	6	0
Not applicable	1	8	0
School	1	36	1
Child care facility	1	33	0
Unknown	3	132	11
Total	102	1,975	166

to attribute a foodborne cause or identify a food vehicle, while no individual patient data was collected in two outbreaks.

To attribute the cause of the outbreak to a specific food vehicle, investigators obtained analytical evidence from epidemiological studies of 19 outbreaks. Microbiological evidence of contaminated food was found in 12 outbreaks, with a further five outbreak investigations obtaining both microbiological and analytical evidence. Investigators obtained analytical and/or microbiological evidence for 39 per cent (13/33) of *Salmonella* outbreaks, which was similar to 33 per cent for 2004. Sixty-three per cent (66/102) of outbreaks relied on descriptive evidence to implicate a food or foodborne transmission. These were mainly smaller outbreaks or were in settings where patient interviews were difficult to collect such as aged care facilities.

Significant outbreaks

There were five outbreaks affecting 50 or more persons in 2005, which is similar to previous years. Four were due to *Salmonella* Typhimurium and one was due to *Salmonella* Oranienburg. Two of the outbreaks occurred at restaurants, two in the community and one was associated with a bakery. The largest outbreak was due to *S.* Typhimurium 197 in Victoria during January. This outbreak affected in excess of 448 people and was related to dips served at a Turkish restaurant.

Two large outbreaks of *S.* Typhimurium 135 occurred in Tasmania during October and December, and affected a total of 184 people. These outbreaks were associated with cakes prepared at a bakery and raw egg sauces from a restaurant. A common egg-farm supplied eggs to both of the implicated premises. Eggs from this farm were associated with two additional smaller outbreaks in Tasmania.

In November, the Western Australian Department of Health investigated an outbreak of *Salmonella* Oranienburg. The outbreak extended into the first four months of 2006, and affected at least 125 people. The Health Department conducted a case control study that implicated commercially produced alfalfa sprouts, which was later confirmed microbiologically. The other outbreak affecting more than 50 people occurred in South Australia and involved 81 people with 46 of them diagnosed with *S.* Typhimurium 64 after eating rolls with various fillings from a restaurant.

There were 20 outbreaks affecting between 20 and 50 persons. Six of these outbreaks occurred in association with food prepared at restaurants and five with food prepared by commercial caterers. A wide range of food vehicles were responsible for these outbreaks. Six outbreaks were due to *Salmonella*, of which serotype Typhimurium was responsible for five of these.

Cluster investigations

During 2005, states and territories conducted 82 investigations of clusters of enteric diseases that affected 1,076 people and hospitalising at least 65 people. Investigators were unable to determine the mode of transmission or source of infections for these clusters, which were due to organisms such as *Salmonella*, *Campylobacter*, Shiga toxin-producing *E. coli* and hepatitis A. These clusters do not include all investigations conducted at the state, territory or public health unit level, but the number is indicative of the effort to investigate enteric diseases in Australia. Seventy-seven per cent (63/82) of these investigations related to clusters of *Salmonella,* where the mean number of cases was 10.8 and the total number of persons affected was 683. *S.* Typhimurium was responsible for 49 per cent (31/63) of cluster investigations. Investigations of clusters of *S.* Typhimurium involved more cases with a mean of 13.5 persons than for non-Typhimurium strains with a mean of 8.3 persons. Of the remaining 32 investigations, 24 other different *Salmonella* serovars were involved.

During 2005, there were major increases in *Cryptosporidium* infections in eastern States of Australia. This was reflected in 53 per cent (10/19) of cluster investigations relating to *Cryptosporidium*. The mean size of *Cryptosporidium* cluster investigations was 33.1 persons, which was considerably larger than that for other pathogens. Five of the investigations of *Cryptosporidium* infection were related to contaminated swimming pool water, and the source was unknown for the remaining five outbreaks.

There were three investigations into clusters of campylobacteriosis, two each of *Giardia* and STEC infections, and one each of *Shigella* and hepatitis A infections. The true number of clusters investigated is difficult to ascertain, as public health units or local governments do not record all cluster investigations they conduct. States and territories may also have different definitions and triggers for investigating clusters.

In 2005, OzFoodNet investigated several multi-state clusters of *Salmonella*, including:

- cases of *S.* Typhimurium 135 in the Australian Capital Territory, and New South Wales associated with a yum cha meal in Sydney;
- *S.* Hvittingfoss infections in eastern States of Australia in June and July;
- *S.* Havana cases in New South Wales, Western Australia, South Australia and Victoria in November; and
- *S.* Typhimurium phage types 44 and 135 in all Australian states and territories, except the Northern Territory, in November and December.

OzFoodNet site epidemiologists and state and territory investigators conducted case control studies for two of these multi-state investigations. In June, the source of *S.* Hvittingfoss infections were investigated using a case control study, although no source was identified.[13] In the investigation of *S.* Typhimurium phage types 135 and 44, OzFoodNet initiated a case control study investigating the association between infection with these two phage types and consumption of chicken or eggs. Phage type 135 was significantly associated with consumption of chicken purchased from retail supermarkets. The findings of the case control study for *S.* Typhimurium 44 were equivocal, although 62 per cent (8/13) of point source outbreaks of this phage type occurring during this investigation were suspected to be associated with consumption of eggs.

Surveillance evaluation

Australian surveillance of infectious diseases notified under legislation to state and territory health departments is very effective. The high quality of the data is due to the quality of laboratory services, including reference testing, and awareness of the medical community about the need to notify. In the past 10–15 years, there have been progressive improvements in the capacity of health departments to detect and investigate foodborne diseases at state and territory and national levels. To improve surveillance, OzFoodNet regularly evaluates surveillance and compares data collected at different sites.

National information sharing

In 2005, all jurisdictions contributed to a fortnightly national report to identify clusters of foodborne illness that were occurring across state and territory boundaries. The cluster report was useful for identifying common events affecting different parts of Australia. The cluster report supplemented information sharing on a closed list server, teleconferences and at quarterly face-to-face meetings. In addition, all jurisdictions contributed data to the NNDSS for several diseases that were potentially transmitted by food. In 2005, OzFoodNet made greater use of NNDSS data on specific serotypes and phage types of *Salmonella*, which allowed the detection of clusters and outbreaks at the national level.

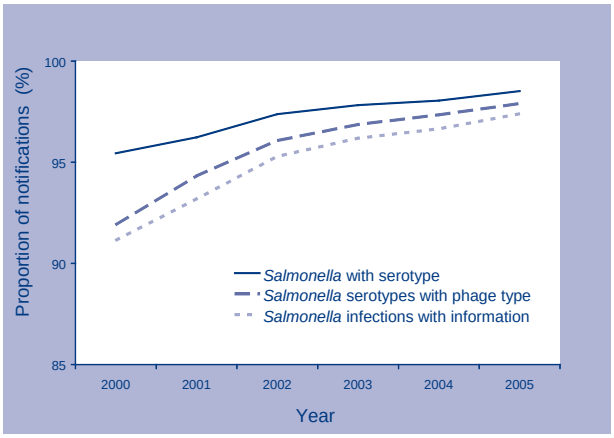
Outbreak reporting and investigation

During 2005, the Australian Capital Territory site reported the highest reporting rate of outbreaks of foodborne disease (15.4 outbreaks per million population), along with Tasmania (12.4 outbreaks per million population). Tasmania also reported the highest rate of foodborne salmonellosis outbreaks (8.2 outbreaks per 100,000 population). The rates of other sites reporting foodborne *Salmonella* outbreaks ranged between 0.5–4.9 outbreaks per million population. Queensland investigated the largest number of foodborne disease outbreaks (32 outbreaks; 8.1 per million population). States and territories conducted 36 analytical studies (cohort or case control studies) to investigate foodborne disease outbreaks, which was slightly less than that of the previous year.

Completeness of Salmonella *serotype and phage type reports*

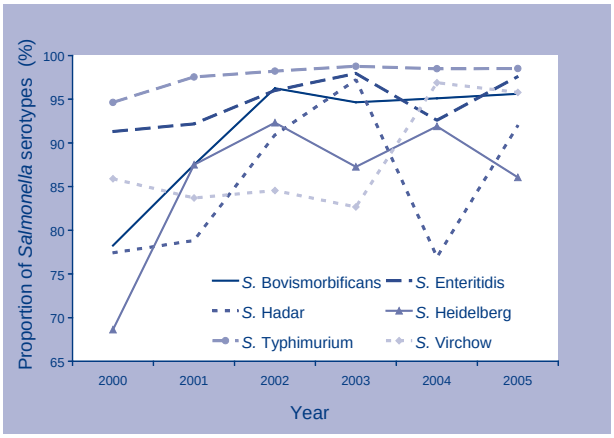
Overall, 97.4 per cent (8,153/8,371) of *Salmonella* notifications on state and territory surveillance databases in 2005 contained information about serotype and/or phage type (Figure 12). This was an increase of 0.7 per cent from 2004.

Figure 12. Proportion of *Salmonella* infections notified to state and territory health departments with serotype and phage type information available, Australia, 2000 to 2005



Phage type recording on the four most prevalent serotypes—Typhimurium, Bovismorbificans, Enteritidis and Virchow—were all greater than 95 per cent complete for phage type information on surveillance databases. Phage type recording was lowest for serotypes Heidelberg and Hadar, with 18.0 per cent (6/43) and 8.0 per cent (2/25) of reports on databases missing the phage type, respectively (Figure 13). Queensland had the highest proportion of complete *Salmonella* notification (99.8%), while six sites reported 95 per cent or higher.

Figure 13. Proportion of *Salmonella* infections for six serotypes notified to state and territory health departments with phage type information available, Australia, 2000 to 2005



Discussion

This report highlights the rates of diseases due to microbiologically contaminated food in Australia. In particular the increasing notification rates of *Salmonella* and *Campylobacter* are concerning.

For *Salmonella* in 2005, reports of several phage types of *S.* Typhimurium were increased and health departments conducted at least 63 investigations of *S.* Typhimurium illness clustered in time, place or person. The rate of campylobacteriosis was particularly high despite health departments conducting relatively few investigations. If we extrapolate using estimated rates of under-reporting, there may have been as many as 153,000 to 554,000 cases of *Campylobacter* occurring in the community during 2005.[2,3] It is likely that approximately 75 per cent of these *Campylobacter* infections would be foodborne in origin.[14]

The notification rates of *Campylobacter* and *Salmonella* in Australia are ten and three times higher respectively than for FoodNet sites in the United States of America (USA).[15,16] The reasons for this are unclear, but are currently being explored. The USA has observed declining incidence of campylobacteriosis in recent years.[16,17] In comparison to New Zealand, Australia has similar rates of salmonellosis and lower rates of campylobacteriosis.[18] New Zealand has seen progressively increasing rates of campylobacteriosis for several years.[19] The reasons for the elevated rates in New Zealand are unclear, but local risk factors for infection include consumption of under-cooked poultry and contact with animals. Australian case control studies of campylobacteriosis have also found that these are important risk factors for infection.[20]

The overall rate of typhoid infections decreased in 2005 and there were fewer locally-acquired typhoid infections. In contrast, the rate of travel-acquired *Salmonella* Enteritidis remained similar to previous years. The number of locally-acquired *S.* Enteritidis infections in 2005 was similar to previous years, and were predominantly reported from Queensland. It was concerning to see an outbreak of *S.* Enteritidis 26var in an aged care home in January 2005 in Victoria, although this was an isolated event. Human surveillance of *S.* Enteritidis infections is very important to monitor for the incursion of this serotype into egg-laying flocks of poultry.[20,21]

In previous years' reports we have noted the considerable variation of the rates of STEC notifications in different Australian states and territories.[23] During 2005, Western Australia, Victoria and the Hunter enhanced surveillance for STEC, which was reflected in increased rates in these regions. Internationally, *E. coli* O157:H7 is the predominant strain reported from surveillance data.[16] In Australia, *E. coli* O157 was also the most common, but the rates were much lower than those observed overseas and many other strains were also common in Australia. Jurisdictions investigated clustering of cases for both STEC and HUS, although they were unable to identify common sources of infection.

While notifications of enteric infections provide information on the burden of disease they are hard to interpret due to the difficulties in establishing the sources of transmission. Summaries of foodborne disease outbreaks provide a systematic way to assess information for the development of food safety policy.[5,24] Australian outbreak data for 2005 highlights several areas where continued vigilance or improvements in food safety are needed, including: fish-related outbreaks, alfalfa sprout production, and poultry and egg-associated salmonellosis.

Fish is the most common food vehicle for identified outbreaks in Australia, although they usually only affect small numbers of people.[25] The two most common intoxications associated with fish—ciguatera and histamine poisoning—are poorly recognised by clinicians and often not reported to health departments. Ciguatera outbreaks in Australia occur almost exclusively in Queensland where amateur fishermen catch fish on affected reefs. However during 2005, three outbreaks of ciguatera occurred where people purchased contaminated fish from retailers. The outbreaks of histamine poisoning in 2005 were almost all associated with tuna. Some of these investigations implicated tuna imported from Asia, although these were unable to be traced back to a common source (personal communication, C Shadbolt, New South Wales Food Authority, July 2006). It was encouraging to see that there were no outbreaks associated with escolar fish in 2005, which has previously caused outbreaks of oily diarrhoea or histamine poisoning.[26]

There were nine outbreaks related to consumption of poultry, making it the second most common food vehicle following fish. *Salmonella* was the aetiological agent in two of these outbreaks, *Campylobacter* in two, *Clostridium perfringens* in one and the aetiology was not determined for the remaining four outbreaks. In addition to these nine outbreaks, OzFoodNet coordinated investigations into a large multi-state cluster of *S.* Typhimurium 135 in November and December 2005. In this investigation microbiological and epidemiological evidence indicated that poultry from retail stores was the likely cause for the outbreak. Food Standards Australia New Zealand are preparing a primary production standard for poultry meat in cooperation with industry and other stakeholders, which will aim to reduce human illness associated with poultry meat.

During 2005, there were four outbreaks of *S.* Typhimurium 135 in Tasmania linked to the same egg farm. Eggs are a common cause of foodborne disease outbreaks, despite Australia not having *S.* Enteritidis endemic in layer flocks.[25] OzFoodNet found that eggs may be responsible for 14 per cent of all foodborne disease outbreaks in 2005, which is higher than previous years. The predominant cause of these outbreaks was *S.* Typhimurium, which has a lower potential for trans-ovarian transmission in layer flocks than *S.* Enteritidis.[27] Outbreaks in Australia may be occurring from surface contamination of eggs or through very low rates of trans-ovarian transmission.[25] Food Standards Australia New Zealand are in the process of establishing a committee to develop a national standard for the primary production of eggs.

The outbreak of *S.* Oranienburg associated with contaminated alfalfa sprouts in Western Australia was the first well-documented outbreak associated with sprouts in Australia. There have been many outbreaks of sprout-associated illness overseas, some of which have implicated seed originating from Australia.[28] These overseas outbreaks traced back to Australian seed have been due to a variety of pathogens, including: *E. coli* O157:NM; *S*. Kottbus; *S*. Bovismorbificans; and *S*. Saintpaul.[28–31] The National Enteric Pathogen Surveillance Scheme records 26 isolations of various serotypes of *Salmonella* from sprouts over the last 20 years (personal communication, Joan Powling, March 2006). The Western Australian outbreak highlighted several areas where alfalfa seed production may be vulnerable to contamination, including growing lucerne pasture and processes within sprouting facilities.[28] Following the outbreak, the Implementation Sub-Committee of the Food Regulation Standing Committee formed a working group to consider ways to improve food safety of these products.

Forty-four per cent of foodborne outbreaks occurred in association with foods prepared at restaurants and commercial caterers, which is similar to previous years. Aged care homes were also common settings for foodborne disease outbreaks and resulted in three of the four outbreak-associated deaths in 2005. Foodborne outbreaks constituted only 6 per cent (11/189) of all outbreaks in aged care homes, but the risk of residents dying was significantly higher for foodborne transmission when compared to other modes of transmission (relative risk 10.2, 95 per cent confidence interval 2.0–58.2). Outbreaks in aged care settings are very difficult to investigate due to the poor recall of food consumption by patients, meaning that a food vehicle was identified in only three outbreaks.

It is important to recognise some of the limitations of the data in this report. Surveillance data are inherently biased and require careful interpretation. These biases include the higher likelihood that certain population groups will be tested, and different testing regimes may be used in different states and territories, resulting in different rates of disease.[3] Some of the numbers of notifications are small, as are populations in some jurisdictions. This can make rates of notification unstable and meaningful interpretation difficult. Importantly, some of the most common enteric pathogens are not notifiable, partic-

ularly norovirus and enteropathogenic *E. coli.* There are some pathogens, such as *Campylobacter,* that are very common but are not often recognised as causing outbreaks. This means relying on outbreak data to set food safety policy will under-estimate the importance of certain pathogens and food vehicles as a cause of human illness and over-estimate others.[5] There can also be considerable variation in assigning causes to outbreaks depending on investigation methods, number of cases and circumstances of the outbreak.

Health agencies conducting surveillance for foodborne disease must constantly improve their practices and evaluate their efforts. This should involve stakeholders such as laboratories, clinicians, and other government departments. The number of analytical studies that health departments used to investigate outbreaks is evidence of robust inquiry into the causes of these diseases. During 2005, OzFoodNet coordinated or participated in the investigation of several multi-state outbreaks. For these multi-state investigations, outbreak investigation team members entered de-identified data into a web-based database—NetEpi—for hypothesis generation and case control studies.[13] This method of data collection was very rapid compared to other methods. Using the Internet to collect information in outbreak settings is a powerful tool for widely dispersed outbreaks and will become routine in the future.[32]

OzFoodNet has shown the benefits of regular communication about surveillance data for detecting national outbreaks. In May 2005, OzFoodNet and the NSW Health Department held an advanced outbreak investigation workshop to improve Australian epidemiologists' abilities to respond to foodborne disease outbreaks. This follows a consultation that OzFoodNet held in 2004, which identified that training and capacity building in disease investigation were important for national preparedness.

It is important that this report assist with the development of food safety policy for Australia. In previous years we have identified similar food vehicles and settings where food is prepared, which indicate that current controls may be inadequate. National surveillance of foodborne diseases is critical to provide data to evaluate these efforts. Ideally, these data would be compared in a timely fashion with data arising from surveillance of hazards in foods and pathogens in animals, as many foodborne diseases have a zoonotic origin.[33,34]

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Appendix 1. Number of cases and rates per 100,000 population of potentially foodborne diseases reported to OzFoodNet sites, Australia, 2005

Condition		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Campylobacter	cases	393	NN	250	4,427	2,113	766	6,108	2,422	16,479
	rate	120.9	NN	123.3	111.7	137.0	157.9	121.6	120.5	121.6
Salmonella	cases	96	2,174	399	2,607	586	301	1,422	791	8,376
	rate	29.5	32.1	196.8	65.8	38.0	62.0	28.3	39.4	41.2
Shiga toxin *Escherichia coli*	cases	0	14	0	9	35	0	8	12	78
	rate	0.0	0.2	0.0	0.2	2.3	0.0	0.2	0.6	0.4
Haemolytic uraemic syndrome	cases	0	6	0	3	2	2	3	1	17
	rate	0.0	0.1	0.0	0.1	0.1	0.4	0.1	0.0	0.1
Typhoid	cases	0	28	0	3	2	0	12	7	52
	rate	0.0	0.4	0.0	0.1	0.1	0.0	0.2	0.3	0.3
Shigella	cases	7	134	195	80	47	4	103	151	721
	rate	2.2	2.0	96.2	2.0	3.0	0.8	2.1	7.5	3.5
Listeria	cases	3	25	0	7	6	0	11	4	56
	rate	0.9	0.4	0.0	0.2	0.4	0.0	0.2	0.2	0.3

NN Not notifiable.

Appendix 2. Outbreak summary for OzFoodNet sites, Australia, 2005

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiological study†	Responsible vehicles
Australian Capital Territory	Jan	Commercial caterer	Unknown	7	0	A	C	Strawberries, smoked salmon & grapes
	Mar	Restaurant	*S.* Hessarek	5	2	AM	C	Hollandaise sauce
	Apr	Restaurant	*Campylobacter*	11	1	A	C	Chicken salad & chicken pasta
	Jun	Commercial caterer	Norovirus	25	1	A	CCS	Pork bruschetta & duck tart
	Jul	Restaurant	Unknown	3	0	A	CCS	Unknown
New South Wales	Jan	Private residence	*S.* Typhimurium 197	43	13	A	CCS	Lambs liver
	Mar	Commercial caterer	Unknown	13	0	A	CCS	Beef casserole
	Mar	Restaurant	Unknown	3	2	D	D	Chicken Caesar salad burger
	Apr	Restaurant	Unknown	5	0	D	D	Lamb & beef
	Apr	Restaurant	Unknown	2	0	D	D	Chicken
	May	Institution	Unknown	37	1	D	CCS/C	Self serve salad bar
	May	Restaurant	Unknown	2	0	D	C	Chicken
	Jul	Restaurant	*S.* Typhimurium 9	16		D	D	Suspected raw egg dishes
	Aug	Aged care	Unknown	12		D	N	Unknown
	Aug	Restaurant	Unknown	3	0	D	D	Suspected coleslaw
	Sep	Restaurant	Unknown	9	0	A	C	Ham pizza
	Oct	Child care facility	*S.* Typhimurium 197	33	0	D	C	Unknown
	Oct	Takeaway	*Campylobacter*	5	0	D	D	Unknown
	Oct	Institution	*S.* Birkenhead	3	3	D	D	Suspected pureed food
	Nov	Restaurant	*C. perfringens*	23	0	D	C	Suspected yellow rice
	Nov	Restaurant	*S.* Typhimurium 44	8	2	D	D	Caesar salad dressing
	Nov	Takeaway	*S.* Typhimurium 9	4	3	D	D	Chicken, rice, coleslaw, potatoes
	Nov	Private residence	Histamine poisoning	4	0	M	N	Tuna steak
	Unknown	Restaurant	Unknown	24	0	D	D	Pasta + pizza
Northern Territory	May	Private residence	Unknown	5	0	D	D	Vietnamese pork rolls
	Jul	Private residence	*S.* Typhimurium RDNC	4	1	D	D	Vietnamese rice paper rolls

Appendix 2. Outbreak summary for OzFoodNet sites, Australia, 2005, *continued*

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiological study†	Responsible vehicles
Queensland	Jan	Aged care	*C. perfringens*	36	0	M	D	Braised steak & gravy
	Jan	Not applicable	Mixed *Salmonella*	8	0	M	D	Rainwater
	Jan	Primary produce	Ciguatera	4	0	D	D	Mackerel
	Jan	Primary produce	Ciguatera	2	0	D	D	Black trevally
	Feb	Other	*S.* Typhimurium 12	10	2	D	D	Unknown
	Mar	Primary produce	Ciguatera	2	0	D	D	Yellowtail kingfish
	Apr	Primary produce	Ciguatera	17	2	D	D	Spanish mackerel
	Apr	Commercial caterer	Unknown	11	0	D	C	Unknown
	Apr	Grocery store/ delicatessen	*S. aureus*	2		M	D	Custard filled dumplings
	May	Bakery	*S.* Typhimurium 197	13	7	D	D	Egg based bakery products
	May	Private residence	*Campylobacter*	5	0	D	D	Unknown
	May	Takeaway	*S.* Typhimurium 170/108	2	1	D	D	Chicken meat
	Jul	Restaurant	*S.* Typhimurium 9	40	29	A	C	Bread and butter pudding
	Jul	Restaurant	*C. perfringens*	3	0	M	D	Beef rendang
	Jul	Restaurant	Histamine poisoning	2	0	D	D	Yellowfin tuna
	Sep	Primary produce	Ciguatera	5	0	D	D	Black kingfish
	Sep	Aged care	*Campylobacter*	3	0	D	D	Unknown
	Sep	Unknown	*Campylobacter*	2	0	D	D	Unknown
	Sep	Primary produce	Ciguatera	2	0	D	D	Spanish mackerel
	Sep	Primary produce	Ciguatera	2	0	D	D	Trevally
	Sep	Takeaway	*S. aureus*	2		M	D	Chips and gravy
	Oct	Other	*S.* Chester/Saintpaul	26	2	AM	CCS	Municipal water
	Oct	Commercial caterer	*S.* Potsdam	6	4	D	D	Unknown
	Nov	Restaurant	Unknown	18	0	A	CCS	Seafood mornay & rice
	Nov	Restaurant	*C. perfringens*	14	0	M	D	Chicken and lamb guvec
	Nov	Restaurant	Unknown	5	0	D	D	Unknown
	Nov	Private residence	*S.* Typhimurium 44	3	0	D	D	Egg and bacon roll
	Nov	Camp	*S.* Typhimurium 44	2	0	D	D	Unknown

Appendix 2. Outbreak summary for OzFoodNet sites, Australia, 2005, *continued*

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiological study†	Responsible vehicles
Queensland, *continued*	Dec	Private residence	*S.* Typhimurium 44	23	22	D	D	Prawn soup
	Dec	Primary produce	Ciguatera	10	0	D	D	Barracuda
	Dec	Primary produce	Ciguatera	8	0	D	D	Yellowtail kingfish
	Dec	Grocery store/ delicatessen	*Campylobacter*	4	0	D	D	Chicken kebabs
South Australia	Feb	Restaurant	*S.* Typhimurium 9	13	0	A	CCS	Unknown
	May	Restaurant	*S.* Typhimurium 170/108	9		A	CCS	Marinated chicken roll
	Jun	Restaurant	*S.* Typhimurium 64	81		A	C	Bread roll with fillings
	Nov	School	*Campylobacter*	36	1	D	C	Unknown
	Nov	Hospital	*Listeria*	3	3	M	D	Cold meats
	Dec	Restaurant	Norovirus	21	1	D	D	Dips
Tasmania	Feb	Restaurant	Histamine poisoning	2	0	D	D	Yellowfin tuna
	May	Private residence	*Vibrio*	2	0	D	D	Suspected seafood
	Oct	Bakery	S. Typhimurium 135‡	107	6	AM	C	Bakery products
	Oct	Restaurant	S. Typhimurium 135	11	2	D	D	Sauces/dressings containing raw egg
	Nov	Bakery	S. Typhimurium 135	6	0	D	D	Salad rolls/sandwiches
	Dec	Restaurant	S. Typhimurium 135	77	2	AM	C	Mayonnaise & tartare sauce
Victoria	Jan	Restaurant	S. Typhimurium 197	448	25	M	D	Dips
	Jan	Commercial caterer	Unknown	40	0	A	CCS	Veal rolls & red curry
	Jan	Aged care	Unknown	30	0	D	D	Unknown
	Jan	Commercial caterer	Unknown	29	0	A	C	Chicken vol-au-vents
	Jan	Private residence	Unknown	10	1	D	D	Unknown
	Jan	Aged care	*S.* Enteritidis 26var	7	2	D	D	Suspected eggs
	Jan	Private residence	*S.* Typhimurium 126 var 4	5	0	D	D	Suspected eggs
	Feb	Camp	*Campylobacter*	22	0	M	C	Suspected water
	Feb	Restaurant	Unknown	16	0	A	C	Seafood platter, baked fish & octopus
	Feb	Bakery	Unknown	6	0	D	D	Suspect pork rolls
	Mar	Private residence	*S.* Typhimurium 12	15	0	D	C	Unknown
	Mar	Commercial caterer	*S.* Typhimurium 9	14	5	D	C	Chocolate mousse

Appendix 2. Outbreak summary for OzFoodNet sites, 2005, *continued*

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiological study†	Responsible vehicles
Victoria, *continued*	Mar	Restaurant	*S.* Typhimurium 9	13	5	M	D	Hollandaise sauce
	Mar	Aged care	Unknown	11	0	D	D	Unknown
	Mar	Takeaway	Unknown	6	0	M	D	Hommus dip
	Jul	Restaurant	Histamine poisoning	2	0	A	D	Tuna
	Jun	Commercial caterer	Unknown	17	0	A	C	Gravy & pork
	Jun	Hospital	Unknown	11	0	D	D	Unknown
	Aug	Primary produce	Ciguatera	5	0	D	D	Fijian snapper
	Sep	Restaurant	Unknown	11	0	A	C	Suspected Spanish mackerel
	Oct	Commercial caterer	Norovirus	36	0	A	C	Sandwiches
	Oct	Aged care	Unknown	6	0	D	D	Unknown
	Nov	Aged care	Unknown	12	1	D	D	Unknown
	Nov	Bakery	Norovirus	9	0	D	D	Cakes
	Nov	Unknown	*Campylobacter*	5	0	D	D	Unknown
	Nov	Primary produce	Histamine poisoning	2	0	D	D	Fish
	Dec	Restaurant	*S.* Typhimurium 170/108	20	1	D	C	Suspected pork
Western Australia	Apr	Commercial caterer	Unknown	20	0	D	D	Unknown
	Jun	Private residence	Unknown	17	1	D	D	Unknown
	Oct	Restaurant	Unknown	21	1	D	C	Unknown
	Oct	Restaurant	Unknown	15	0	D	C	Unknown
	Nov	Primary produce	*S.* Oranienburg	125	11	AM	CCS	Alfalfa sprouts

* A=analytical epidemiological evidence; D=descriptive evidence: M=microbiological evidence.

† C=cohort study; CCS=case control study; D=descriptive study; N=individual patient data not collected.

‡ All four outbreaks of *S.* Typhimurium 135 in Tasmania were due to the local variant phage type 135a, which is not a recognised international classification

Communicable Diseases Network Australia annual report, 2005

Introduction

In Australia, although the surveillance and prevention of communicable diseases is largely the legislative responsibility of the states and territories, a nationally consistent approach to communicable disease management is obviously desirable. This report aims to highlight the communicable disease challenges that Australia faces, and the integral role the Communicable Diseases Network Australia (CDNA) plays in providing a cohesive national response to these threats.

The report describes the activities of the CDNA in 2005. Section two provides some background to the Network and section three outlines the significant changes to the National Notifiable Diseases Surveillance System (NNDSS) and the notable communicable disease activity for 2005. Section four gives examples of other important work that CDNA did in 2005, including its response to particular disease outbreaks and important policy questions, and also outlines some projects that are of strategic importance to CDNA, without CDNA being integrally involved in them. The achievements and challenges of CDNA's working groups and subcommittees in 2005 are highlighted in section five.

The varied and complex work of the Network is evident from this report, as is the increasing demand for CDNA's contributions to communicable disease policy. With the emergence of new infections, the threat of antimicrobial resistance and bio-terrorism, climate change and the re-emergence of infections previously thought to be well controlled, communicable disease has become one of the highest public health priorities both in Australia and overseas. Although vaccination has reduced the morbidity and mortality associated with many diseases, the epidemiology of these diseases and their vaccination coverage require enhanced monitoring, and intensified control efforts are required in the disease elimination phases. CDNA's involvement extends to the international sphere, for example, advising on refugee pre-departure assessments for malaria.

About the Communicable Diseases Network Australia

Introduction

In 1989, as part of a joint initiative of the National Health and Medical Research Council and the Australian Health Ministers' Advisory Council (AHMAC), the Communicable Diseases Control Network was established. This network is now known as the Communicable Diseases Australia Network.

CDNA reports to the AHMAC, formerly through the National Public Health Partnership (NPHP). On 30June 2006, the NPHP was restructured into two committees, the Australian Health Protection Committee (AHPC) and the Australian Health Development Committee. CDNA is now a subcommittee of the AHPC.

Objectives

The CDNA vision statement outlines the role of the network:

> 'The Communicable Diseases Network Australia will provide national public health leadership and co-ordination on communicable disease surveillance, prevention and control, and offer strategic advice to governments and other key bodies on public health actions to minimise the impact of communicable diseases in Australia and the region' (CDNA, 2005).

CDNA's key objectives are to:

- promote best practice prevention and management of communicable diseases;
- develop and coordinate national surveillance programs for communicable diseases;
- develop policy and to provide policy advice on the control of communicable diseases;
- support and strengthen training and capacity building in the communicable disease field;

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- provide a resource for the investigation and control of outbreaks of communicable disease; and
- liaise with and support other communicable disease control agencies and programs in the region.

Representation

The Network includes representatives from the Australian Government, state and territory governments, key organisations in the communicable diseases field, representatives from New Zealand and the Secretariat of the Pacific Community (as observer members) and other individuals with relevant experience.

Network meetings

CDNA conducts fortnightly teleconferences to share and evaluate the latest developments in communicable disease surveillance and holds additional teleconferences, as required, to obtain specialist assistance and coordinate actions when outbreaks or potential outbreaks occur.

Subcommittees and working groups

Increasingly, CDNA receives requests to provide comment on national policies or surveillance and control issues that have national implications, and issues public statements when appropriate.

To ensure this capability, CDNA utilises the skills and expertise of a wide network of people through the formation of subcommittees and working groups that produce policies, practice guidelines and other outputs. The achievements of these committees and working groups in 2005 are presented in section five of this report.

Highlights from the National Notifiable Diseases Surveillance System

In 2005, there were significant improvements in the NNDSS. Most states and territories began daily transmission of data to NNDSS through the Data Acquisition System (DAS), whilst the remainder transmitted data three times per week. DAS is an automated system which provides a quality check on all incoming data.

- In addition, the National Surveillance Committee (NSC), a subcommittee of CDNA, worked towards obtaining complete and consistent reporting of data through NNDSS. During 2005 the completeness and quality of data improved, particularly in the reporting of influenza type and meningococcal serogroups. The improvement in the timeliness and completeness of NNDSS data has enabled CDNA to review national data each fortnight at CDNA teleconferences since January 2005. (See also the report from the NSC on page 312).

In 2005, NNDSS reported on 61 diseases and conditions. Only three diseases were not notifiable in all jurisdictions—campylobacteriosis (New South Wales), incident hepatitis C (Queensland) and in South Australia influenza was not notifiable, although reports were made to NNDSS. Syphilis notifications were reported by all jurisdictions in two categories—less than two years duration and greater than two years or unknown duration. The bioterrorist agents, tularaemia and smallpox, were made notifiable in all jurisdictions and added to the NNDSS. A list of the diseases that are currently nationally notifiable can be found on the Australian Government Department of Health and Ageing's (DoHA) website at: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-surveil-nndss-casedefs-distype.htm

The major disease activities detected by NNDSS in 2005 were increases in *Chlamydia* and gonococcal infections, continuing a trend evident for some years. There were high rates of pertussis in New South Wales and an increase in notifications of cholera, and hepatitis E acquired overseas. Analysis of NNDSS data also demonstrated a decline in notifications of meningococcal C disease, following the introduction of a meningococcal C vaccination program in January 2003.

Enhanced (or additional) data collections in NNDSS for tuberculosis and invasive pneumococcal disease continued in 2005. Most states and territories were able to send enhanced data on these two diseases directly to NNDSS by the end of 2005. The reporting and analysis of these data were improved by greater data timeliness and consistency. During 2005, CDNA approved, in principle, the collection of enhanced data on three sexually transmitted infections: gonococcal infections, syphilis (of less than 2 years duration) and donovanosis. Enhanced surveillance data collection on meningococcal infections has also been proposed for 2006.

The DoHA website provides access to NNDSS data via a set of user defined queries which allow aggregated data to be viewed by disease, state and time period.

Selected challenges

The following section gives examples, listed in alphabetical order, of the issues that CDNA dealt with in 2005, outside of its subcommittees and working groups. Some of the topics are CDNA's core business (for example, responding to changes in the epidemiology of pertussis and developing policy for

the management of health care workers infected with bloodborne viruses). Others are mentioned because the topics themselves are of strategic importance to CDNA even though CDNA may only be involved on the periphery (for example, antimicrobial resistance and the Biosecurity Surveillance System).

Antimicrobial resistance

The Joint Expert Technical Advisory Committee on Antibiotic Resistance (JETACAR) was established by the then Minister for Health and Aged Care, the Hon. Dr Michael Wooldridge and the then Minister for Primary Industries and Energy, the Hon. John Anderson MP, in April 1998. The JETACAR report, *The use of antibiotics in food-producing animals: antibiotic-resistant bacteria in animals and humans*[1] (1999) was presented to the Ministers in September 1999. The JETACAR report itself proposed an antibiotic resistance management program encompassing human and animal use of antibiotics.

Progress has been made in addressing the recommendations of the JETACAR report. Major areas of concern continue to include: healthcare associated infections caused by bacteria such as 'golden staph' (usually methicillin resistant *Staphylococcus aureus),* vancomycin resistant enterococci and multi-resistant gram negative organisms such as *Acinetobacter* species; community acquired infections with resistant organisms; the use of antibiotics in animals, particularly in stock feeds; and the possibility of resistant bacteria being transmitted from animals to humans, and contributing to resistant infections in humans.

CDNA and the Public Health Laboratory Network (PHLN) have continued to provide advice and feedback on antimicrobial resistance activities to the DoHA. CDNA and PHLN are also represented on antimicrobial resistance related committees such as the Expert Advisory Group on Antimicrobial Resistance, and via these channels, maintain a watching brief on this important issue.

Avian and pandemic influenza

The increasing number overseas of human cases of highly pathogenic avian influenza (H5N1) since December 2004 and the threat of an influenza pandemic has prompted the Australian Government to ensure effective planning and response at a national level.

The CDNA has been providing specific policy advice to the Australian Government relating to planning and response processes, which includes management of human cases of avian influenza. CDNA has been working closely with the National Influenza Pandemic Action Committee (NIPAC) and the then Australian Health Disaster Management Policy Committee (AHDMPC) and has contributed to the development of the *Australian Management Plan for Pandemic Influenza* – June 2005.[2] Together, these groups provide comprehensive national leadership and international linkage for the coordination of planning and response to an influenza pandemic.

CDNA has specifically assisted in the following areas:

- epidemiological and disease control advice to the Australian Government Chief Medical Officer, NIPAC and the AHDMPC for the management and containment of avian influenza to prevent a pandemic;
- advice on methods, definitions and protocols for national surveillance of human cases of avian influenza and contacts of cases;
- advice on situational management according to global and Australian phasing, and assistance with the development of public health protocols and guidelines to address the situation; and
- liaison with other networks such as the Public Health Laboratory Network and infectious diseases sectors of Australia to ensure appropriate clinical guidelines are in place for timely investigation and management of suspected and confirmed cases.

The CDNA provides an operational resource for the investigation and control of suspected and confirmed cases of avian influenza and potential outbreaks of pandemic influenza in Australia.

In late 2005, the CDNA also participated in *Exercise Eleusis.* This national exercise simulated an outbreak of avian influenza and evaluated the industry and government's national capability to manage a zoonotic disease outbreak.

Biosecurity Surveillance System

In the 2004–05 Budget, the DoHA received funding to improve national communicable disease surveillance through the development and implementation of the following information technology systems, which together, make up the Biosecurity Surveillance System (BSS):

- a secure Outbreak Case Reporting System (OCRS);
- improvements to the NNDSS;
- development of a Sentinel GP Surveillance System; and
- a secure communication system.

In December 2005, the Australian Government provided additional funding for the development of the Syndromic Surveillance System (SSS). The SSS is intended to strengthen national surveillance and provide early warning of an influenza pandemic in Australia. The SSS will build on the infrastructure and protocols developed for the BSS.

Analysis and design of the surveillance systems commenced in 2005. The interim OCRS, NetEpi, was enhanced and trialled by OzFoodNet and the Jurisdictional Executive Group of CDNA, and subsequently implemented in July 2005. NetEpi continues to be used by OzFoodNet and is available for use by jurisdictions and the DoHA National Incident Room.

The Health Alert Network, the 'in-confidence' network allowing communication and collaboration amongst the health surveillance community, is being designed and built in-house and is due for implementation in 2006.

Jurisdictions and CDNA are represented on a number of BSS Special Interest Groups (SIG) such as the Laboratory eNotification and Data and Coding Standards SIG and the Cluster and Outbreak Detection SIG. Both of these SIGs conducted meetings in 2005. The Cluster and Outbreak Detection SIG met with disease surveillance algorithm researchers from the Centre of Epidemiology and Research (New South Wales Health), the Centre for Mathematical and Information Systems (Commonwealth Scientific and Industrial Research Organisation), and the Australian Biosecurity Cooperative Research Centre.

General information about the BSS is available on the DoHA website at: http://www.health.gov.au/internet/wcms/publishing.nsf/content/biosecurity%20surveillance%20system-1

Bloodborne virus infection in health care workers

On 22 September 2005, the *Guidelines for Managing the Issues of Blood-Borne Virus Infection In Health Care Workers*[3] was endorsed by CDNA.

With this endorsement, CDNA adopts the same rights-based, minimum compulsion approach to the problem of health care workers infected with HIV, hepatitis B or hepatitis C that has proved so successful in the containment of the general HIV epidemic since the mid-1980s. The document identifies and supports the equal rights for health workers to privacy as their infected patients.

The *Guidelines for Managing the Issues of Blood-Borne Virus Infection In Health Care Workers* contains the following recommendations:

- Where restriction of a health care worker's practice may be necessary, psychological, financial and other support must be provided to encourage self-presentation to a physician with the necessary knowledge and experience in the field. Also, physicians managing such health care workers should be able to seek the advice of a jurisdictional expert advisory panel. Rather than instating specific discriminatory regulations, including compulsory testing, responsible behaviour by both infected health care workers and their treating physicians, to ensure patient safety, can be enforced through the ordinary legal penalties for unprofessional behaviour that already exist in all the jurisdictions.
- With one exception, the restrictions that should be placed on the practice of a health care worker infected with a bloodborne virus, should depend on the real risk of transmission and should be tailored to each individual case. The relevant risks include the worker's level of viraemia, the nature of the practice (namely whether it involves exposure prone procedures and how invasive they are) and the worker's experience. The exception is HIV infection, which should, at present, be an absolute criterion for exclusion from performing exposure prone procedures, even though the likelihood of transmission from a health care worker with low or undetectable virus during an exposure prone procedure is most probably close to zero.
- It is strongly recommended that anyone entering into any undergraduate or postgraduate training, which involves exposure prone procedures, should be aware of their bloodborne virus status and seek professional advice if infected since it is recognised that training is a high risk time for transmission.
- It is essential to ensure the involvement of the relevant jurisdictional registration boards, in order to provide consistent management of infected health care workers.

Dengue

Outbreaks

Two outbreaks of dengue occurred in 2005, both of dengue type 4. The first occurred in the Torres Strait and involved 56 confirmed cases and the second in Townsville, resulting in 18 confirmed cases.

The Torres Strait outbreak, affecting Thursday, Darnley and Murray Islands, was controlled using the *Dengue Fever Management Plan 2005–2010*[4] (*DFMP*) developed the previous year. This required the rapid mobilisation of a large 'dengue intervention force' (comprising 20 health staff sourced from

Queensland Health's Tropical Public Health Unit Network (TPHUN) and Torres Strait) for a two week period. Important factors contributing to the success of this operation included:

- increased levels of awareness and cooperation of residents and other government agencies;
- the Queensland Health funded campaign to remove rubbish that may act as mosquito breeding sites on Thursday Island in 2004; and
- comprehensive repair of screening on rainwater tanks on Thursday Island in 2004.

The Dengue Fever Management Plan 2005–2010

The *DFMP* was revised and distributed by Queensland Health to guide and coordinate the management of dengue fever by local and state government in north Queensland.

The *DFMP* focuses on three central components of dengue management: disease surveillance; mosquito control and surveillance; and education.

There are three levels of dengue activity:

- ongoing prevention: where there is no current dengue activity in the zone;
- response to sporadic cases: where there is no current dengue activity in the zone, but the TPHUN is notified of an imported case of dengue or a possible locally-acquired case; and
- outbreak response: where one or more locally-acquired cases occurs concurrently in the zone.

The *DFMP* also outlines ongoing research into dengue transmission and control. The *DFMP* is available on the Queensland Health website at: http://www.health.qld.gov.au/dengue/managing_outbreaks/default.asp or the *Dengue in North Queensland* website at: http://www.health.qld.gov.au/dengue/default.asp

Dengue prevention campaign

In June 2005, Queensland Health's TPHUN developed a new dengue fever prevention campaign, after a survey revealed only one-third of Townsville and Cairns residents took steps to get rid of dengue mosquito breeding sites. The 'Stop mozzies breeding' awareness campaign features posters, post cards, brochures, bin stickers and fridge magnets.

Dengue and CDNA

The CDNA contributes to dengue control primarily through advice from the National Arbovirus and Malaria Advisory Committee (NAMAC). During 2005, NAMAC was involved in the *Aedes albopictus* delimiting survey and subsequent surveillance and control activities in the Torres Strait and the Tennant Creek *Aedes aegypti* eradication program. The possible spread or introduction of *Aedes aegypti* from its present distribution in Queensland is being closely monitored. Although the *Aedes albopictus* mosquito is not as good a vector as *Aedes aegypti*, the prevention of the introduction and establishment of *Aedes albopictus* remains a high priority because this mosquito has the potential to spread widely over Australia, including southern areas (see report from the NAMAC on page 310).

Gastrointestinal and foodborne diseases

Foodborne disease is an important part of the CDNA's work, as contaminated food often causes multi-state outbreaks and requires a coordinated response. OzFoodNet—Australia's system for enhanced foodborne disease surveillance—and Food Standards Australia New Zealand are both members of CDNA. In addition, all states and territories have responsibility for investigating and controlling foodborne and gastrointestinal diseases.

Each fortnight at their teleconference, the CDNA reviews notifications of potentially foodborne diseases to the NNDSS and reports of outbreaks in jurisdictions. This allows CDNA to monitor the status of foodborne diseases and detect multi-state outbreaks. OzFoodNet conducted several multi-state outbreak investigations during 2005, under the auspices of CDNA. These included outbreaks of *Salmonella* Hvittingfoss, *Salmonella* Havana, *Salmonella* Typhimurium 44, and *Salmonella* Typhimurium 135a.

CDNA considered papers on food safety and foodborne illness, including illness associated with chicken meat and eggs, and efforts to improve national outbreak coordination. At the CDNA 2005 Communicable Disease Control conference two sessions on foodborne and enteric diseases highlighted the work that states and territories conduct through CDNA.

The recent *Exercise Eleusis* on avian influenza involved CDNA and touched on many food-related issues. CDNA made use of the membership of Food Standards Australia New Zealand to prepare advice relating to consumption of egg and poultry products.

Investigation into an outbreak of desquamating rash among clients in treatment for opioid dependence

In late 2004, the NSW Health Department received several reports of a desquamating rash among clients of the methadone program. In response, NSW

Health, in collaboration with the CDNA, initiated a series of investigations to identify the likely cause, including active surveillance for cases, a survey of dosing points and a case control study.

Over 380 cases were identified across Australia, largely in New South Wales. Almost all cases were identified among clients prescribed one form of methadone. No abnormality or contaminant was identified on testing suspected batches of methadone. While the exact cause of the outbreak could not be determined, batches of methadone temporally associated with the outbreak were quarantined from use, and the outbreak subsided.

This investigation highlighted the importance of a coordinated approach to the investigation and response to national disease outbreaks.

Pertussis

Background

Pertussis was first notifiable in South Australia in 1909 and in most jurisdictions from the early 1930s. National compilation of pertussis data ceased in 1949 and did not recommence until 1979.[5] The current case definition allows for reporting of both laboratory-confirmed cases and clinical cases (with or without an epidemiologic link), although the majority of cases are laboratory-confirmed only.[6]

Diphtheria-tetanus-pertussis vaccine was introduced in 1953 and childhood immunisation programs have included pertussis vaccine since that time. Pertussis is a cyclic disease. Epidemics occur every 3–5 years, although rates of notifications in current peak periods correspond with the troughs of pre-immunisation days. In 2004, Western Australia experienced an outbreak of pertussis in which notification rates slightly exceeded 100 cases per 100,000 population. In 2005, the cyclic epidemic affected South Australia (95.1/100,000), New South Wales (86.6/100,000) and the Australian Capital Territory (96.3/100,000).[6]

Control strategies

In the early to mid-1990s, a National Pertussis Working Party was convened to develop strategies to control pertussis in Australia. The *Guidelines for the control of pertussis in Australia*[8] were developed and became an authoritative document on notification, investigation, case management and public health management of pertussis.

Whilst information contained in the Guidelines is no longer current, the practice of supporting vaccination programs, appropriate case management, chemoprophylaxis for defined contacts and other outbreak control measures remains a priority of CDNA. The objective of public health management of pertussis is to reduce outbreaks of disease and reduce morbidity and mortality, especially in infants who are at high risk of severe disease and adverse outcomes. Priorities for management of pertussis are also now contained in *The Australian Immunisation Handbook.*[9] Public health authorities in most jurisdictions have inadequate resources to investigate all notifications of pertussis, but follow regularly reviewed practices in an aim to identify high risk contacts and identify and manage potential outbreaks. CDNA is also a focal point for scrutinising responses to the cyclical epidemic, and to provide support to general and specific outbreak control measures, such as those implemented during 2005.

Vaccination

Since 1999, the funded childhood schedule has included an acellular pertussis vaccine that is considerably less reactogenic than its whole-cell predecessor. In January 2004, the 15-year-old diphtheria-tetanus (dT) booster was replaced by dTpa, which includes an acellular pertussis component. It is anticipated that increasing uptake of this booster will reduce transmission of pertussis by reducing disease in adolescents and young adults, who are recognised as a significant reservoir of infection. This vaccine is also recommended for adults who have contact with infants and young children – including parents, carers and child care workers. CDNA continuously advocates and promotes uptake of this vaccine among these groups.

Challenges

Aspects of pertussis control remain a challenge. Members of the CDNA have in recent years conducted research to improve the efficiency of pertussis investigation and follow-up. During outbreaks, information alerts to clinicians and settings such as child care have been shown to increase detection of disease during the infectious period, enhancing the window of opportunity to identify and manage vulnerable contacts.[10]

Highlights from the subcommittees and working groups

The following section describes the achievements and challenges of CDNA's working groups and subcommittees in 2005, in alphabetical order.

Case Definitions Working Group

Background

The Case Definitions Working Group was convened in 2001 to revise or develop standard surveillance case definitions for all nationally notifiable diseases for reporting to the DoHA. The Working Group comprises members representing all states and territories, the DoHA, the PHLN, OzFoodNet, the National Centre in HIV Epidemiology and Clinical Research (NCHECR), the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) and other communicable disease experts. Laboratory definitions previously developed by the PHLN formed the basis for the Surveillance Case Definitions, with clinical and epidemiological elements added, as appropriate.

Major activities

At the beginning of 2005 the Working Group was presented with 17 case definitions for which review had been requested. The definitions for review were:

- *Chlamydia;*
- cholera;
- Creutzfeldt-Jakob disease – classical (cCJD);
- Creutzfeldt-Jakob disease – variant (vCJD);
- hepatitis E;
- human immunodeficiency virus (HIV) – newly acquired;
- HIV – unspecified;
- influenza;
- Japanese encephalitis virus infection;
- Kunjin virus infection;
- meningococcal – invasive disease;
- pertussis;
- severe acute respiratory syndrome (SARS);
- syphilis – infectious (primary, secondary, and early latent), less than 2 years duration;
- syphilis – more than 2 years duration;
- syphilis – congenital; and
- tularaemia.

The Working Group met three times, by teleconference, through 2005 and has submitted to CDNA final recommendations on all but three definitions from the review list. The recommended changes are unlikely to have a significant impact on the number of cases notified.

Case definitions still under review include cCJD, vCJD and SARS.

Communicable Disease Control Conference Organising Committee

The 2005 Communicable Diseases Control Conference, convened by the CDNA and the PHLN was held at the Convention Centre, Sydney on 2–3 May 2005. Because effective disease control and prevention demands close collaboration among experts in many different fields, and the conference aimed to bring these people together, the theme was *Piecing Together the Jigsaw.*

Dr John Watson from the United Kingdom Health Protection Agency and Dr David Butler from the Canadian Public Health Agency were among the keynote speakers. Along with presentations (both oral and poster) from those who submitted abstracts, panel discussions were held on 'Avian influenza', 'Current and future challenges and opportunities in communicable diseases control in Australia' and 'Public health issues from the Asian tsunami disaster'.

The conference was a great success with the highlights being the oral presentations and keynote speakers.

Improving Indigenous Identification in Communicable Diseases Reporting Project Working Group

Background

In late 2004, the DoHA received a report titled *Improving Indigenous Identification in Communicable Diseases Reporting.*[11] The CDNA was requested to provide input to the DoHA response and consequently established the Improving Indigenous Identification in Communicable Diseases Reporting Project (IIICDRP) Working Group to prepare a written report.

Improving the quality of Indigenous identification in communicable disease reporting will make a contribution to better health for Aboriginal and Torres Strait Islander peoples. Benefits identified arise from improved data collection leading to enhanced quality data and a clearer picture and understanding of communicable diseases in Aboriginal and Torres Strait Islander populations. This enables appropriate actions to address the identified issues and allows the measurement of change over time. One of the major contextual challenges to these identified benefits, especially for Indigenous people with communicable diseases, is diagnosis and data capture at the outset. *Improving Indigenous Identification in Communicable Diseases Reporting* aims to provide some insight into how Indigenous identification can

be improved in communicable disease reporting and recommends a number of short, medium and longer term strategies.

Standardising the process of collecting and reporting Indigenous identification for all communicable diseases in all jurisdictions is the highest order recommendation made in the report.

Major activities

The IIICDRP Working Group will focus primarily on those recommendations which will be implemented by jurisdictions. The aim of the working group is to categorise the recommendations into those that:

- are able to be implemented immediately without the need for further funding;
- could be implemented with further funding; and
- are not feasible for implementation by jurisdictional health authorities.

The Working Group expects to have a written report for the CDNA's endorsement finalised in 2006. This report will provide an analysis of the recommendations in the original discussion paper and focuses on key recommendations which are likely to lead to maximum improvements in the Indigenous notification system.

Infection Control Guidelines Working Group

Background

The Infection Control Guidelines Working Group was established to review components of the *Infection Control Guidelines for the Prevention of Transmission of Infectious Diseases in the Health Care Setting*[12] (ICGs) relating to CJD (Chapter 31 and Appendix 9) and the section on autoclaving of asthma spacers (Section 17.6.2 and 17.6.3). The Working Group is comprised of national experts from the fields of clinical infectious diseases, health care associated infection prevention and control units, infection management services, with representation from the Australian National CJD Registry and CJD Incident Panel, state and territory governments and the DoHA.

Major activities

The Working Group conducted two face-to-face meetings in 2005, which achieved the following outcomes.

1. An approach to the review of the CJD components of the ICGs was established, including;
   - major revision of the CJD chapter to exclude non-essential information;
   - consolidation of the CJD components into a single chapter;
   - inclusion of evidence for recommendations made in the guidelines;
   - development of risk levels arising from possible exposure to CJD;
   - inclusion of risk assessment tools and action items for practitioners to use in the health care settings; and
   - recommendations on the infectivity of the tissues from the anterior eye.
2. Following consultation with the Therapeutic Goods Administration, advice was provided to the National Asthma Council regarding reprocessing of single patient use spacers.

The following activities are planned for the 2006:

- link the revised cCJD infection control document to separate infection control guidelines for vCJD;
- seek input from dentists and maxillofacial surgeons with regards to procedures considered high risk in their profession and infection control;
- consider management practices for surgical instruments used during and after the diagnostic and therapeutic procedures;
- agree on a clearance process for the updated chapter on cCJD; and
- develop timetable for the revision of the remaining ICG chapters.

Inter-Governmental Committee on HIV/AIDS, Hepatitis C and Related Diseases

Background

The Inter-Governmental Committee on HIV/AIDS, Hepatitis C and Related Diseases (IGCAHRD) is the key advisory body to the NPHP, through the CDNA, on policy and program issues and activities related to the response to HIV/AIDS, hepatitis C and sexually transmissible infections (STIs). The committee comprises representatives from all states and territories, the DoHA, and community-based organisations which represent people affected by HIV, hepatitis C and STIs. Three subcommittees, which aim to improve data standardisation nationally and develop methods to improve national surveillance for HIV, viral hepatitis and STIs, also report to IGCAHRD.

Major activities

During 2005, the IGCAHRD was involved in the development and endorsement of the *National HIV/AIDS, STI and Hepatitis C Strategies 2005–2008*[13–15] and the *National Aboriginal and Torres Straits Islander Sexual Health and Blood-Borne Virus Strategy 2005–2008*.[16] All four of these strategies were officially launched by the Minister for Health and Ageing in 2005. Subsequent to the strategy launches, the IGCAHRD played a key role in the development and endorsement of Implementation Plans for each of the strategies.

During 2005, representatives of the IGCAHRD were also involved in the following activities:

- participation in the planning and implementation of World AIDS Day 2005;
- the review of the national anti-retroviral guidelines for treatment of HIV;
- co-chairing the review of the national HIV Testing Policy;
- the process the National Pathology Accreditation Advisory Council is undertaking at the request of IGCAHRD to develop accreditation standards for laboratories which perform HIV and hepatitis C virus testing;
- the development of evidence-based guidelines for hepatitis C treatment, care, support education and prevention in correctional settings;
- the revision of national projections for hepatitis C;
- the Hepatitis C Surveillance Strategy Review Subcommittee;
- the reference group for the economic evaluation of hepatitis C in Australia;
- the review of the work plans, terms of reference and governance arrangements for the viral hepatitis, HIV and STI surveillance subcommittees of IGCAHRD;
- the development of a framework for mapping of HIV, hepatitis C and STI-related prevention and education activities;
- the Ministerial Advisory Committee on AIDS, Sexual Health and Hepatitis C (MACASHH) Research Round Table;
- observer at MACASHH and its three subcommittees; and
- the National *Chlamydia* Screening Pilot Program reference group.

In addition:

- The STI Surveillance Subcommittee has contributed toward the establishment of a minimum national dataset for enhanced (or additional) surveillance for STIs and conducted a national review of laboratory testing data collected for *Chlamydia.*
- The National Viral Hepatitis Surveillance Committee has been likewise preparing a draft dataset for newly acquired hepatitis B and has also prepared an information sheet, which has been used to promote enhanced surveillance of newly acquired hepatitis C among GPs; and
- the National HIV Surveillance Committee is working toward the establishment of surveillance of HIV subtypes among cases of newly diagnosed HIV infection, is carrying out an assessment of the completeness of AIDS notification through linkage to the National Death Index and is reviewing the content of national HIV/AIDS notification forms.

During 2005 the IGCAHRD developed the *Infrastructure Benchmarks for the Design, Implementation and Evaluation of HIV/AIDS, STI and Hepatitis C Health Promotion Programs*[17] and submitted the document to CDNA for endorsement prior to release.

IGCAHRD members continue to be involved in the on-going analysis of HIV, STI and hepatitis C notification and social research data; and the program response to the increases in HIV, gonorrhoea, *Chlamydia* and syphilis. The IGCAHRD will continue to work with all key stakeholders and DoHA to progress activities identified in the *National Strategy Implementation Plans 2005–2008.*[18]

Inter-pandemic Influenza Working Group

Background

The Inter-pandemic Influenza Working Group was formed in 2004 to develop guidelines for the management of influenza outbreaks in residential care facilities (RCFs). The Working Group consists of public health representatives from all Australian states and territories, with support from the DoHA.

RCFs are considered to be high-risk environments for influenza due to communal living arrangements and the continual close proximity of residents. Nursing homes and hostels catering for the elderly are especially high-risk environments due to the older age of residents and high prevalence of chronic medical conditions.[19,20]

Major activities

In 2005, the Working Group developed the *Guidelines for the Prevention and Control of Influenza Outbreaks in Residential Care Facilities in Australia.*[21]

The purpose of the Guidelines is to provide national best practice guidelines for staff of public health units for preventing, defining and managing outbreaks of influenza in RCFs in Australia during inter-pandemic periods.

The main strategies emphasised in the Guidelines to prevent and manage outbreaks are vaccination prior to the influenza season and during an outbreak, the use of antiviral therapy for treatment and prophylaxis, infection control measures including restriction of movement between affected and unaffected areas and minimising contact between affected and unaffected persons during an outbreak, and maintaining good surveillance in RCFS so that appropriate interventions can be promptly instituted.

The Guidelines will be distributed to all public health units in Australia in 2006 and copies will be available to residential care facilities on request. They are also available on the DoHA website at: http://www.health.gov.au/internet/wcms/publishing.nsf/content/cda-pubs-other-flu_guidel.htm

Meningococcal Disease Working Party

Background

The terms of reference for the Meningococcal Disease Working Party are to consider current evidence in the epidemiology and management of meningococcal disease and submit recommendations to CDNA as appropriate, which may include the following activities:

- revisions to the *Guidelines for the early clinical and public health management of meningococcal disease in Australia;*[22]
- consideration of issues relating to the implementation of the National Meningococcal C Vaccination Program; and
- provision of advice on the management of outbreaks of meningococcal disease to health authorities.

The Working Party is comprised of national experts from the fields of clinical infectious diseases, microbiology, surveillance, and public health; and representation from public health units, state governments, the DoHA and the NCIRS. Ms Maureen Watson, representing the National Immunisation Committee, provides the important link with the routine vaccination program. The Working Party has also benefited from the insights provided by Dr Diana Martin as the New Zealand representative, on the issues faced in the New Zealand situation of hyperendemic group B disease, and the specific vaccination program being rolled out in response.

Major activities

In 2005, the Working Party continued its review of the current guidelines, which commenced in 2004. The group has met regularly by teleconference, and has almost completed draft changes to the Guidelines, mostly in relation to the use of the conjugate meningococcal C vaccine for contacts of cases, and for cases. Further consideration of new available evidence for the definition of contacts requiring clearance antibiotics has also been undertaken, along with updates of the case definition to take account of new technologies and new routine protocols in laboratories, and of the national surveillance dataset. It is expected that a draft will be ready for consideration by CDNA in 2006.

The Working Party has noted, with pleasure, the excellent coverage attained for children 12 months of age with the meningococcal C conjugate vaccine, and the reasonable coverage achieved in children aged 2–6 years from the 'catch up' program, based on the Australian Childhood Immunisation Register. Parallel with these achievements, the overall incidence of meningococcal disease in Australia has fallen from 3.5 cases per 100,000 population in 2001 to 2.0 cases per 100,000 population in 2004. During this period, the incidence of serogroup C isolates has decreased by 45 per cent.

National Arbovirus and Malaria Advisory Committee

Background

The National Arbovirus and Malaria Advisory Committee, reporting through the CDNA, makes recommendations on arbovirus and malaria surveillance, strategic arbovirus and malaria disease management and vector control. The Committee provides expert technical advice on malaria and arboviruses to assist in the detection, management and control of real or potential outbreaks of arboviral disease. The NAMAC includes individuals with expertise in surveillance, vector virology control, quarantine and clinical care, representing agencies with a substantial interest in this area.

Major activities

The NAMAC has been developing national flavivirus outbreak management guidelines (dengue virus, Japanese encephalitis virus, West Nile virus, Murray

Valley encephalitis virus). The *Interim National Guidelines for the Prevention, Management and Control of Murray Valley Encephalitis Virus*[23] was completed by NAMAC and endorsed by CDNA in 2005. Work on the guidelines for dengue, Japanese encephalitis and West Nile is progressing.

During 2005, the International Organisation for Migration (IOM) and the Department of Immigration and Multicultural Affairs sought NAMAC clarification on the pre-departure malaria treatment requirements for refugees. The new requirements were clarified and CDNA endorsed the *Recommendations for refugee pre-departure assessment/treatment for malaria* prepared by the IOM.

The NAMAC is also assisting the DoHA and Australian Quarantine Inspection Service in the development of a memorandum of understanding (MOU). The MOU will detail Commonwealth and state and territory co-operative arrangements in relation to vector control and surveillance at Australian borders. The NAMAC provided advice on vector control and surveillance according to the *Quarantine Act 1908*.

A delimiting survey of *Aedes albopictus* (dengue mosquito vector) in the Torres Strait and adjoining northern Cape York Peninsula was carried out in collaboration with NAMAC in May 2005. To address the *Aedes albopictus* mosquito incursion in north Queensland that was detected in the survey and the associated human health implications, a NAMAC working group, which included members of the Island Coordinating Council, met on June 2005. Subsequent recommendations which included '*that intensive control and surveillance in the Torres Strait begin immediately to make use of the dry season and be continued for 3 years'* were subsequently endorsed by the full NAMAC on 16 June 2005 and then CDNA. Australian Government funding assistance is being provided to Queensland Health to conduct a mosquito elimination program in the Torres Strait

The Tennant Creek *Aedes aegypti* Eradication Project continues to progress. This is a joint Northern Territory Government Department of Health and Community Services and DoHA project.

Mosquito incursions are a recurring problem in northern Australia. The DoHA held a meeting in December 2005 with the Australian Government Department of Agriculture, Fisheries and Forestry (DAFF) and northern Australian jurisdictions to discuss issues concerning mosquito and disease control in northern Australia and strategies to address these emerging problems. It is anticipated that this will lead to longer term planning to respond to mosquito incursions.

National Enteric Pathogen Surveillance Scheme Steering Committee

Background

Membership of the National Enteric Pathogen Surveillance Scheme (NEPSS) Steering Committee includes representatives from all state and territory health departments, the DoHA, and DAFF, OzFoodNet and Food Standards Australia New Zealand.

The primary task of the NEPSS Steering Committee is to ensure that the agreed key performance indicators in the contract with the University of Melbourne Microbiological Diagnostic Unit (MDU) Public Health Laboratory are met. This process is performed electronically, and if issues arise a teleconference is convened.

The agreed key performance indicators for 2004–05 related to the:

- quality, amount and timeliness of data collected and inputted into the database by the MDU;
- ease of stakeholders and other approved persons in accessing the data;
- quality of the analyses of the data undertaken by the MDU;
- MDU's compliance with the format for reports and data request reports agreed with the Steering Committee; and
- time and frequency of the reports and data request reports.

Major activities

For the 2004–05 contract period, the NEPSS Steering Committee acknowledged that the agreed key performance indicators were met.

In addition, the DoHA funded Dr Diane Lightfoot of the MDU to attend the Enter-net Workshop and International Collaboration on Enteric Disease meetings in Madrid, Spain, from 1–4 June 2005.

The CDNA Joint Executive Group endorsed the project proposal *External review of the National Enteric Pathogens Surveillance Scheme* in July 2005. The NPHP agreed to fund the project proposal for $30,000 in November 2005. The review will occur in 2006.

National Immunisation Committee

Background

The National Immunisation Committee (NIC) was first established in 1993 and is the peak body responsible for overseeing the development, implementation and delivery of the National Immunisation Program (NIP).

Membership of the NIC during 2005 comprised of representatives from the Australian Government, the states and territories, the Australian Divisions of General Practice, the Royal Australian College of General Practitioners and the Australian Local Government Association. At the end of 2005 it was agreed to expand the membership of the NIC and invite additional representatives from the Rural Doctors Association of Australia, the Consumers Health Forum and the Australian Medical Association.

Major activities

During 2005 the NIC oversaw the implementation and delivery of both the National Childhood Pneumococcal Vaccination Program and the National Pneumococcal Vaccination Program for Older Australians which commenced on 1 January 2005. Take-up for the National Childhood Pneumococcal Vaccination Program has been greater than anticipated with 76.6 per cent of children eligible for the catch-up component of the program up-to-date for pneumococcal vaccination as at 31 December 2005 and approximately 97 per cent of babies born in January 2005 having had their first scheduled dose of Prevenar, according to data from 31 December 2005.

Vaccine supply shortages, including influenza, were also managed by the Committee during 2005 to ensure continuity of supply to immunisation providers and doctors.

The NIC was also involved in the rollout of the National Varicella (Chickenpox) Vaccination Program, the replacement of oral polio vaccine with inactivated polio vaccine which commenced on 1 November 2005, and the introduction of hepatitis A vaccine for Indigenous children under 5 years of age living in Queensland, the Northern Territory, Western Australia and South Australia.

The *National Vaccine Storage Guidelines – Strive for 5*[24] was finalised and released by the NIC. The document is aimed at Australian vaccination service providers and gives them a concise, practical and user-friendly guide to vaccine storage as well as outlining the basic principles for safe vaccine management. *Understanding Childhood Immunisation*[25] was also revised and released. *Understanding Childhood Immunisation* is given to all new parents at the birth of their baby, and contains easy to understand information on vaccines funded under the NIP, the diseases that are protected against, and side effects caused by the vaccines and what to do about them. Both of these documents were sent to all general practices during 2006 and are available from the Immunise Australian Program website at: http://immunise.health.gov.au/

During 2005, the NIC convened a workshop to review vaccine safety and the reporting of adverse events following immunisation. The *National Vaccine Safety Workshop* made several recommendations which will be pursued by NIC in 2006.

National Surveillance Committee

Background

The role of the National Surveillance Committee is to:

- develop policy and processes relating to national reporting of notifiable diseases;
- work toward national consistency in reporting of notifiable diseases;
- identify and address deficiencies in current surveillance processes; and
- advise and respond to the CDNA, including the subcommittees, on issues relating to strategic planning and processes for the national surveillance of communicable disease.

The membership of the NSC consists of epidemiologists and data managers from each state and territory and the Australian Government and representatives from OzFoodNet, the NCHECR and the NCIRS.

Major activities

Over the course of four meetings in 2005 the Committee agreed upon a nationally consistent process for dealing with cross-border issues in relation to disease notifications. The implementation of enhanced (or additional) STI and invasive meningococcal disease surveillance also commenced. Furthermore, NNDSS core data revisions have been completed and a new version of the data specifications is ready for endorsement by CDNA.

National Tuberculosis Advisory Committee

Background

The terms of reference for the National Tuberculosis Advisory Committee (NTAC) in 2005 were to provide strategic and expert advice to the CDNA on a coordinated national and international approach to

tuberculosis (TB) control and to develop and review nationally agreed strategic and implementation plans for the control of TB in Australia.

The current membership of NTAC includes jurisdictional representation from those responsible for the TB programs in their respective jurisdictions. This representation includes nurse managers with TB expertise, public health physicians, clinicians practising in TB clinics, thoracic physicians, infectious diseases physicians, a microbiologist and the DoHA secretariat.

Major activities

In 2005, the NTAC continued to push the TB agenda forward by endorsing a number of national guidelines as part of their key strategies under the *National Strategic Plan for TB Control in Australia Beyond 2000.*[26]

To date, NTAC has endorsed the following guidelines:

- *The BCG vaccine: information and recommendations for use in Australia;*[27]
- *Guidelines for Australian Mycobacteriology Reference Laboratories;*[28]
- *Procedures for Health Assessments of Unauthorised Fisherpersons Apprehended of the North Coast of Australia;*[29] and
- *National Guidelines for Overseas Travel for Patients with Pulmonary Tuberculosis.*[30]

In 2006, NTAC is working to revise its strategic plan and agenda for action for the next three years. The new draft strategic plan outlines the minimum requirements and resources for all jurisdictional TB services. Under the proposed plan, NTAC have identified the following issues for 2006–2009:

- maintaining political commitment to eliminating TB in Australia;
- maintaining current high level of diagnostic and management services for TB;
- ensuring the free availability of drugs for first and second line treatment;
- improving the management of latent TB;
- ensuring adequate pre-migration screening, in particular, of health workers entering Australia from countries with a high risk of TB; and
- encouraging research to improve diagnostic tools.

The role of the NTAC committee has become increasingly important over recent years as issues relating to the prevention and control of TB have emerged, including the recruitment of healthcare providers from high incidence TB and multidrug-resistant TB areas. Also, there has been considerable concern over the reduced availability of human tuberculin purified protein derivative for tuberculin skin testing and TB drugs for the treatment of cases. NTAC continues to play an important role in ensuring that effective treatment is made available.

Norovirus Guidelines Working Group

The CDNA Norovirus Working Group was established in 2004 and to date has shared the guidelines that have been developed in each state and territory for the management of infectious gastrointestinal illness or viral gastroenteritis. The Working Group has recommended a project officer be contracted to develop national norovirus guidelines. A part-time project officer has been appointed (located in South Australia) to develop the first draft of these guidelines for discussion by the Norovirus Working Group in 2006.

Pneumococcal Working Party

Background

The Pneumococcal Working Party is a joint initiative of the CDNA and the Australian Technical Advisory Group on Immunisation formed in 2000, along with a number of subgroups. In 2005 the active subgroup was the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG).

The EIPDSWG's role is to continue and improve national enhanced (or additional) surveillance of pneumococcal disease, including the review of what should be included in the dataset and to provide reports on the status of pneumococcal disease in Australia and guidelines for control.

The Working Group's membership consists of representatives from the CDNA, the Surveillance Policy and Systems Section of the DoHA and each state and territory. A representative from the Immunisation Section of DoHA attends as required and the PHLN provides laboratory surveillance when needed.

Major activities

One of the EIPDSWG's major achievements in 2005 was that enhanced surveillance is now available nationally for all children aged less than five years. There have also been ongoing improvements in the amount and type of data collection including agreement to collect additional risk factor information on child care attendance and repeat episodes of IPD. In addition, more than 90 per cent of all isolates are serotyped in most jurisdictions.

During 2005, the EIPDSWG prepared the 2004 annual report titled *Invasive pneumococcal disease in Australia, 2004,*[31] for publication in *Communicable Diseases Intelligence* (*CDI*) and submitted an abstract for the *5th International Symposium on Pneumococci and Pneumococcal Diseases* in Alice Springs to be held in 2006.

Concerns raised and identified through the EIPDSWG have contributed to the development of a proposal by NCIRS to examine the efficacy of the 23-valent vaccine in Australia, particularly in Indigenous adults and those with underlying conditions. Data collected through the EIPDSWG will be used in the analysis.

One of the challenges faced by the EIPDSWG is that although the Australian Government has negotiated individual contracts with the jurisdictions to assist in the collection of enhanced invasive pneumococcal disease surveillance data, the funds are inadequate for the amount of resources required to collect comprehensive data in all jurisdictions. The importance of enhanced data has been highlighted in a recent NCIRS draft position paper on conjugate vaccine failures in Australia and the need for serotype and antibiotic resistance pattern information is well accepted.

Public Health Laboratory Network

Background

The PHLN is a collaborative group of laboratory representatives from all jurisdictions in Australia. The aim of the PHLN is to provide strategic advice and share expertise at the national level in order to enhance the national capacity for the laboratory-based detection and surveillance of agents and vectors of communicable diseases in Australia. This is achieved by the sharing of knowledge and expertise within the PHLN; consultation with other laboratories, organisations and individuals with specialised expertise; and communication with other public and private laboratories in the jurisdictions.

The PHLN was established in 1996 as part of the implementation of the *National Communicable Diseases Surveillance Strategy* to complement the CDNA. PHLN holds monthly teleconferences and has at least one face-to-face meeting per year.

Major activities

In 2005, the PHLN met monthly by teleconference to discuss ongoing issues surrounding laboratory diagnostics. Some key areas of discussion included laboratory biosafety, laboratory capacity (particularly in relation to dealing with a pandemic) and laboratory biosecurity. The PHLN worked to develop standard protocols and guidelines such as the *Laboratory precautions for samples collected from patients with suspected viral haemorrhagic fevers.*[32]

In March 2005, the PHLN hosted a *Neisseria gonorrhoeæ* Workshop to:

- review the current status of gonococcal nucleic acid detection test in Australia;
- evaluate the accuracy of the existing tests;
- advise on further assessment of tests; and
- develop guidelines for monitoring of antibiotic susceptibility.

A paper titled *Guidelines for the use and interpretation of nucleic acid detection tests for* Neisseria gonorrhoeæ *in Australia: a position paper on behalf of the Public Health Laboratory Network*[33] was published in *CDI*.

During 2005, the DoHA engaged the services of Dr Janice Lanser to focus on completing and revising the Laboratory Case Definitions (LCDs). This work continues to progress.

The DoHA provided a range of equipment and test kits to allow the rapid detection (within 30 minutes) of biological agents and toxins, including anthrax, ricin and botulinum toxin. This equipment was distributed to state and territory PHLN laboratories in June 2005 and have already proved valuable for the Australian Capital Territory public health laboratory which used the kits to rapidly discount the presence of anthrax in 'white powders' received by foreign embassies in early to mid-June 2005.

PHLN held their annual face-to-face meeting on 5–6 September 2005. The first day consisted of workshops on 'white powders'/suspicious substances plus a session on eNotification. The second day consisted of discussion on issues ranging through counter terrorism, laboratory capacity, classification of physical containment facilities, and molecular diagnosis of gonococcal infection.

A bioterrorism workshop on laboratory capacity was held on 6 December 2005, which outlined a plan for public health pathology services and their capacity to handle health emergencies. In addition, laboratory capacity planning for pandemic influenza, SARS and biosecurity continued in 2005.

The major challenge for PHLN in 2006 is to work with the Australian Government and relevant committees such as CDNA to establish the Network as

a subcommittee under the new AHPC. In accordance with AHPC's governance structure, PHLN has developed terms of references and a work plan which will maintain the collaborative network of pathology and microbiology laboratory leaders and continue to provide strategic advice to enhance the national capacity for the laboratory-based detection and surveillance of agents and vectors of communicable diseases in Australia.

Through the agreed work plan and strategies, PHLN will continue to:

- communicate and consult widely with government, microbiologists and other public health professionals in public and private sector laboratories and to identify any need for additional essential resources;
- ensure early warning of communicable disease outbreaks through laboratory data sharing via laboratory reporting systems and regular teleconferences; and
- provide a first point of contact for all jurisdictional and national issues relating to laboratory diagnosis or surveillance of communicable diseases by identifying and utilising additional specialised expertise as needed.

Trachoma Steering Committee

Background

The CDNA Trachoma Steering Committee was established in September 2003 to develop guidelines to improve consistency in trachoma screening and treatment programs.

Major activities

During 2005, the Committee continued to prepare the guidelines. Comments were sought from CDNA members and key interest groups and all submissions received during the consultation were taken into consideration in finalising the guidelines. The CDNA endorsed the *Guidelines for the Public Health Management of Trachoma in Australia,*[34] in September 2005.

The Guidelines establish a minimal best practice approach for trachoma screening, diagnosis and treatment. They recommend that state and territory population health units collect trachoma data in accordance with a minimal national trachoma dataset and report these to a national trachoma database. Monitoring of antibiotic resistance to treatment (azithromycin) is also recommended.

In support of the trachoma management guidelines, the Minister for Health, the Hon. Tony Abbott MHR, announced in December 2005, a government commitment of $920,000 over the next three years to try to reduce the incidence of trachoma.

A proportion of this funding will be allocated to establish a surveillance unit to monitor trachoma prevalence and control measures in regions where trachoma control activities are currently undertaken. Remaining funding will be allocated towards essential training for health care workers to identify, treat and report incidences of trachoma.

The CDNA will have continued involvement in the management of trachoma through membership of advisory groups that will oversee implementation of the Guidelines.

Future directions

With reporting lines having changed in 2006, CDNA will work closely with the new AHPC and related committees to ensure that suitable governance arrangements are in place for the Network to provide national public health leadership and co-ordination on communicable disease surveillance, prevention and control, and offer strategic advice to governments and other key bodies on public health actions.

As well as the ongoing surveillance of communicable diseases, identified priorities for 2006–07 include:

- finalising the revision of the *Guidelines for the early clinical and public health management of meningococcal disease in Australia;*
- developing national norovirus, pertussis, seasonal influenza and measles guidelines;
- progressing harmonisation of public health legislation across Australian jurisdictions;
- further contributing to the development of the BSS; and
- continuing to contribute to pandemic influenza planning, including participating in *Exercise Cumpston* in October 2006.

Experience with SARS and avian influenza has highlighted that international collaboration is essential for the prevention and control of communicable diseases. Projects with international linkages that CDNA has planned for 2006 include the development of pre-departure health screening for refugees from South Asia and the Middle East and protocols for people being deployed to disaster areas.

Due to issues such as the emergence of new infections and bio-terrorism, and the intensified efforts required for the monitoring and elimination of vaccine preventable diseases, CDNA's workload continues to grow. One of its greatest challenges for the future will be ensuring workforce capacity for the consideration and progression of crucial communicable disease policies to enhance Australia's communicable disease capability.

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Abbreviations

AHDMPC	Australian Health Disaster Management Policy Committee
AHMAC	Australian Health Ministers' Advisory Council
AHPC	Australian Health Protection Committee
AIDS	acquired immunodeficiency syndrome
AMRLN	Australian Mycobacterium Reference Laboratory Network
BSS	Biosecurity Surveillance System
cCJD	classical Creutzfeldt-Jakob disease
CDI	*Communicable Diseases Intelligence*
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
DAFF	Department of Agriculture, Fisheries and Forestry
DAS	Data Acquisition System
DFMP	*Dengue Fever Management Plan 2005–2010*
DoHA	Department of Health and Ageing
dT	diphtheria-tetanus vaccine for use in adults
dTpa	adult/adolescent formulation diphtheria-tetanus-acellular pertussis vaccine
EIPDSWG	Enhanced Invasive Pneumococcal Disease Surveillance Working Group
HIV	Human immunodeficiency virus

ICGs	*Infection Control Guidelines for the Prevention of Transmission of Infectious Diseases in the Health Care Settings*
IGCAHRD	Inter-Governmental Committee on HIV/AIDS, Hepatitis C and Related Diseases
IIICDRP	Improving Indigenous Identification in Communicable Disease Reporting Project
IOM	International Organisation for Migration
JETACAR	Joint Expert Technical Advisory Group on Antibiotic Resistance
MACASHH	Ministerial Advisory Committee on AIDS, Sexual Health and Hepatitis C
MDU	Microbiological Diagnostic Unit
MOU	memorandum of understanding
NAMAC	National Arbovirus and Malaria Advisory Committee
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NCIRS	National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
NEPSS	National Enteric Pathogens Surveillance Scheme
NIC	National Immunisation Committee
NIP	National Immunisation Program
NIPAC	National Influenza Pandemic Action Committee
NNDSS	National Notifiable Diseases Surveillance System
NPHP	National Public Health Partnership
NSC	National Surveillance Committee
NTAC	National Tuberculosis Advisory Committee
OCRS	Outbreak Case Reporting System
PHLN	Public Health Laboratory Network
RCFs	residential care facilities
SARS	Severe Acute Respiratory Syndrome
SIG	Special Interest Groups
SSS	Syndromic Surveillance System
STIs	sexually transmitted infections
TB	tuberculosis
TPHUN	Tropical Public Health Unit Network
vCJD	variant Creutzfeldt-Jakob disease

Annual report: surveillance of adverse events following immunisation in Australia, 2005

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Abstract

This report summarises Australian passive surveillance data for adverse events following immunisation (AEFI) reported to the Adverse Drug Reactions Advisory Committee for 2005, and describes reporting trends over the six year period 2000 to 2005. There were 839 AEFI records for vaccines received in 2005. This is an annual AEFI reporting rate of 4.1 per 100,000 population, the lowest since 2000 and a 22 per cent decrease compared with 2004 (1,081 records; 5.4 AEFI records per 100,000 population). The decrease was not consistent across age groups. Reporting of AEFI increased for children aged <1 year in 2005 (60.7 versus 50.3 per 100,000 population) and decreased for the 7 to <20 year age group (0.9 versus 8.9 per 100,000 population). Dose-based AEFI reporting rates in 2005 were 11.0 per 100,000 doses of scheduled vaccines for children aged <7 years and 2.0 per 100,000 doses of influenza vaccine for adults aged ≥18 years. The majority of records described non-serious events while 9 per cent (n=72) described AEFIs defined as serious. There was one report of death in an older person following influenza vaccine and one of non-polio acute flaccid paralysis in an infant, both temporally associated with immunisation. The most frequently reported individual AEFI was injection site reaction in children following a fifth dose of diphtheria-tetanus-acellular pertussis vaccine (79 reports per 100,000 doses). The increase in the population-based AEFI reporting rate for children aged <1 year in 2005 coincided with the introduction of national immunisation programs for conjugate pneumococcal vaccine in January 2005 and inactivated poliovirus vaccine in November 2005. The fall in reporting rates for older children and adolescents follows the completion of the national meningococcal C catch-up program in early 2005. The consistently low reporting rate of serious AEFIs demonstrates the high level of safety of vaccines in Australia. *Commun Dis Intell* 2006;30:319–333.

Keywords: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine safety

Introduction

This report summarises national passive surveillance data for adverse events following immunisation (AEFI) reported to the Adverse Drug Reactions Advisory Committee (ADRAC) by 31 March 2006. The report focuses on AEFI reported for vaccines administered during 2005 and trends in AEFI reporting for the six year period 2000 to 2005.

The aim of passive post-licensure AEFI surveillance is to monitor vaccine and immunisation program safety and to detect population-specific, rare, late-onset or unexpected adverse events that may not be detected in pre-licensure vaccine trials.[1–3] An 'adverse event following immunisation' is defined as any serious or unexpected adverse event that occurs *after* a vaccination has been given which may be related to the vaccine itself or to its handling or administration.[1] An AEFI can be *coincidentally* associated with the *timing* of immunisation without necessarily being caused by the vaccine or the immunisation process.

In Australia, AEFIs are notified to ADRAC (an expert committee of the Therapeutic Goods Administration) by state and territory health departments, health care professionals, vaccine manufacturers and members of the public.[4] All reports received by ADRAC are evaluated using internationally consistent criteria[5] and are reviewed at regular meetings. Passive AEFI surveillance data have been collated in the ADRAC database since 2000 and are used to monitor trends, detect signals and generate hypotheses. Reports summarising national AEFI surveillance data have been published regularly since 2003.[6–10]

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There were several important changes to the Australian National Immunisation Program Schedule (NIPS) in 2005[11] that impact on the AEFI surveillance data presented in this report:

(i) A national childhood pneumococcal conjugate vaccine program commenced on 1 January 2005. Since then, infants routinely receive the vaccine at 2, 4 and 6 months of age while children born from 1 January to 31 December 2004 were to receive a catch-up schedule with the number of doses dependent on the child's age.

(ii) A national adult pneumococcal polysaccharide vaccine program commenced on 1 January 2005 for those aged 65 years and over. This was in addition to the funded program for Indigenous adults aged 50 years and over.

(iii) On 1 November 2005, inactivated poliovirus vaccine (IPV) replaced oral poliovirus vaccine (OPV) for all age groups. All states and territories introduced multi-component vaccines to deliver IPV in combination with other antigens to children at 2, 4 and 6 months and 4 years of age. All these IPV-containing combination vaccines include diphtheria-tetanus-acellular pertussis (DTPa) antigens (i.e. quadrivalent vaccines) and some also include hepatitis B and/or *Haemophilus influenza* type b (Hib) antigens (i.e. pentavalent and hexavalent vaccines). The specific combination vaccines administered at 2, 4, and 6 months of age vary between states and territories but all provide DTPa-IPV vaccine at 4 years of age.

(iv) A national varicella program commenced on 1 November 2005 with doses due at 18 months of age or at 12–13 years of age.

(v) Diphtheria-tetanus-acellular pertussis (adult formulation) (dTpa) vaccine was recommended in place of the adult formulation of diphtheria-tetanus (dT) vaccine for young adolescents in routine school-based immunisation programs.

Changes to the NIPS in 2003 also impact on the interpretation of trend data. On 1 January 2003, the meningococcal C conjugate immunisation program commenced when the vaccine was introduced into the schedule at 12 months of age, with a catch-up program for all those born between 1984 and 2001.[11] Also, in September 2003, the dose of DTPa at 18 months of age was removed from the schedule. Since then, DTPa has been given at 2, 4 and 6 months, and 4 years of age.[4]

Methods

Adverse events following immunisation data

De-identified information was released to the National Centre for Immunisation Research and Surveillance for all drug and vaccine adverse event notifications received by ADRAC between 1 January 2000 and 31 March 2006. Readers are referred to previous AEFI surveillance reports for a description of the AEFI surveillance system and methods used to evaluate AEFI reports received by ADRAC.[6,7]

ADRAC database records* were eligible for inclusion in the analysis if:

- a vaccine was recorded as 'suspected' of involvement in the reported adverse event *and* either
  - (a) the vaccination occurred between 1 January 2000 and 31 December 2005 *or*
  - (b) for records where the vaccination date was not recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2005.

Study definitions of adverse events following immunisation outcomes and reactions

AEFIs were defined as 'serious' or 'non-serious' based on information recorded in the ADRAC database and criteria similar to those used by the World Health Organization[5] and the US Vaccine Adverse Events Reporting System.[12] In this report, an AEFI is defined as 'serious' if the record indicated that the person had recovered with sequelae, been admitted to hospital, experienced a life-threatening event, or died.

Typically, each AEFI record listed several symptoms, signs and diagnoses that had been re-coded from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).[13] To simplify data analysis, we grouped MedDRA® coding terms to create a set of reaction categories. Firstly, reaction categories were created that were analogous to the AEFIs listed and defined in the *Australian Immunisation Handbook* (8th edition).[4] Additional categories were created for MedDRA® coding terms that were listed in more than one per cent of AEFI records (e.g. headache, irritability, cough). Reaction terms listed in less than one per cent of records were grouped into broader

*. The term 'AEFI record' is used throughout this report because a single AEFI notification to ADRAC can generate more than one record in the database. For example if a notification describes an injection site reaction plus symptoms and signs of a systemic adverse event (e.g. fever or generalised allergic reaction), two records will appear in the database: one record containing information relevant to the injection site reaction and one record for the systemic adverse event.

categories based on the organ system where the reaction was manifested (e.g. gastrointestinal, neurological).

Data analysis

All data analyses were performed using the SAS version 9 computer program.[14] The distribution of AEFI records was analysed by age, gender and jurisdiction. Average annual population-based reporting rates were calculated for each state and territory and by age group using population estimates obtained from the Australian Bureau of Statistics.

The frequency and age distribution of AEFI outcomes, reaction categories and vaccines listed as 'suspected' of involvement in the reported adverse event were assessed. For each vaccine, the age distribution of vaccinees notified with AEFIs was calculated as well as the proportion of AEFI records where (i) the vaccine was the only suspected vaccine or drug, (ii) the AEFI record was assigned a 'certain' or 'probable' causality rating, and (iii) the AEFI was defined as 'serious'. Because many AEFI records listed more than one suspected vaccine and several reaction terms to describe an adverse event, column totals in the relevant tables exceed the number of AEFI records analysed.

Dose-based AEFI reporting rates were estimated for influenza vaccine for adults aged 18 years and over and for eight childhood vaccines funded through the National Immunisation Program (i.e. DTPa, DTPa-HepB, Hib, Hib-HepB, polio, MMR, MenCCV, 7vPCV) for children aged <7 years. Dose-based AEFI reporting rates for vaccines received in 2005 were compared to 2004 reporting rates and to the average annual reporting rate for the four years 2001 to 2004. Dose-based AEFI reporting rates were not determined for other vaccines and age groups due to the lack of reliable denominator data for the number of vaccine doses distributed or administered.

Denominator data to estimate influenza AEFI reporting rates in 2005 were obtained from the 2004 national influenza coverage survey[15] for the 18–39 years, 40–64 years and ≥65 years age groups as a survey was not conducted in 2005. The number of administered doses of each of the eight childhood vaccines was calculated from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99 per cent of children aged <7 years.[16] Vaccine doses administered between 1 January and 31 December 2005 were estimated for the age groups <1 year, 1 to <2 years, and 2 to <7 years (i.e. the age at vaccination). Reporting rates were not calculated for vaccines introduced into NIPS in November 2005 (i.e. IPV combination vaccines and varicella vaccine) due to inaccurate numerator and denominator data in the very early stages of these programs.

Notes on interpretation

Caution is required when interpreting the AEFI data presented in this report. Due to reporting delays and late onset of some AEFIs, the data are considered preliminary, particularly for the fourth quarter of 2005. Data published in previous reports for 2000–2004[6–10] differ to that presented in this report for the same period because the data are updated to include AEFIs notified to ADRAC for vaccines administered before 2005.

The information collated in the ADRAC database is intended primarily for signal detection and hypothesis generation. While reporting rates of AEFIs can be estimated using appropriate denominators such as the number of vaccine doses administered, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected AEFIs, and the variable quality and completeness of information provided in individual notifications.[6–10,17]

It is also important to note that this report is based on vaccine and reaction term information collated in a database and not on comprehensive clinical notes. Individual database records list symptoms, signs and diagnoses that were used to define a set of reaction categories based on the case definitions provided in the 8th edition of the *Australian Immunisation Handbook*.[4] These reaction categories are similar, but not identical, to case the definitions of adverse events.

The reported symptoms, signs and diagnoses in each AEFI record in the ADRAC database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines. The causality ratings of 'certain', 'probable' and 'possible' assigned to individual AEFI records describe the likelihood that a suspected vaccine was or vaccines were associated with the reported reaction at the level of the individual. Factors that are considered in assigning causality ratings include the timing (minutes, hours, etc) and the spatial correlation (for injection site reactions) of symptoms and signs in relation to vaccination, and whether one or more vaccines were administered.[6] Because children in particular receive several different vaccines at the same time, all administered vaccines are often listed as 'suspected' of involvement of a systemic adverse event as it is usually not possible to attribute the AEFI to a single vaccine.

Results

Summary of data

There were a total of 839 AEFI records in the ADRAC database where the date of vaccination or onset of an adverse event, if vaccination date was not reported,

occurred between 1 January and 31 December 2005. This is a decrease of 22 per cent compared with 2004 when there were 1,081 AEFI records. In 2005, approximately four per cent of AEFI notifications resulted in more than one AEFI record in the database (usually of an injection site reaction and a systemic reaction). This was the same as in 2004 and lower than previous years when approximately 10 per cent of notifications resulted in more than one AEFI record.[6,7,9]

Seventy-two (9%) of the 839 AEFI records for 2005 were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event or death). A total of 401 (48%) AEFI records were assigned causality ratings of 'certain' (n=272, 44%) or 'probable' (n=29, 3%).

Adverse events following immunisation reporting trends

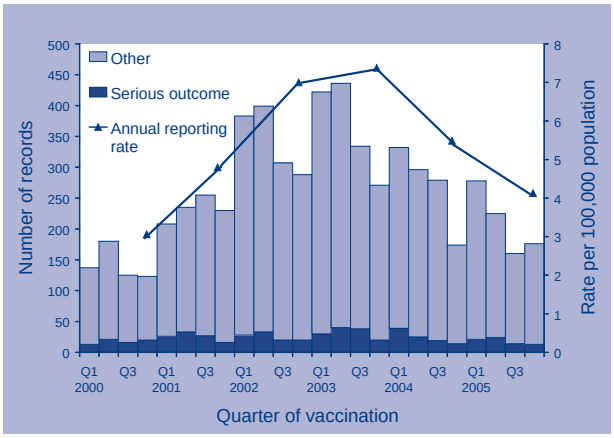
The AEFI reporting rate for 2005 was 4.1 per 100,000 population and was the lowest since 2000 (Figure 1). The trends in AEFI notifications shown in Figure 1 are reflected in the trends in vaccines frequently suspected of involvement in reported AEFIs (Figure 2), and in the types of reactions frequently reported (Figure 3). The decline in the number of AEFI records for vaccines administered in 2005 compared with earlier years follows reductions in the number of AEFIs notified that involved DTPa vaccine or MenCCV (Figure 2).

A seasonal pattern of AEFI reporting, seen in previous years, was apparent in 2005 with the highest number of AEFI notifications for vaccinations administered in the first half of the year (Figure 1). The seasonal peak corresponds to the months when more vaccinations are administered in Australia, particularly among 5-year-old children receiving DTPa and measles-mumps-rubella (MMR) vaccines prior to commencing school in February and older Australians receiving influenza and pneumococcal polysaccharide (23vPPV) vaccines during the autumn months (March to June) (Figure 2). There was also a peak in the first quarter of 2005 following the introduction of the 7-valent pneumococcal conjugate vaccine (7vPCV) into the childhood schedule.

Age and gender distribution

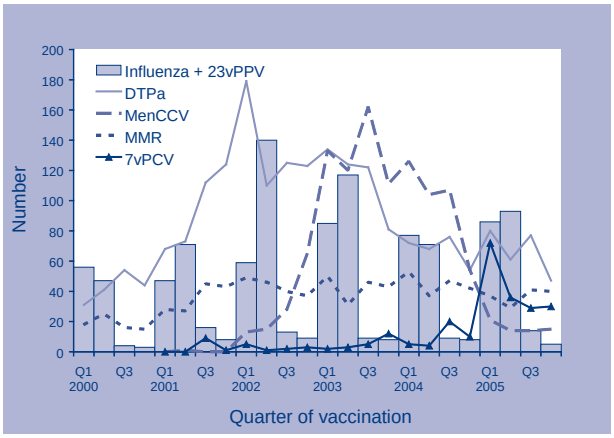
Sixty-three per cent (n=530) of AEFI records for 2005 were for children aged <7 years, compared with 45 per cent in 2004. The AEFI reporting rate in 2005 was highest among children aged <1 year (60.7 per 100,000 population), the age group that receives the greatest number of vaccinations. While the overall population-based reporting rate declined in 2005 compared with 2004 (4.1 versus 5.4 per 100,000), the trend varied by age group (Figure 4). There was

Figure 1. Adverse events following immunisation, ADRAC database, 2000 to 2005, by quarter of vaccination



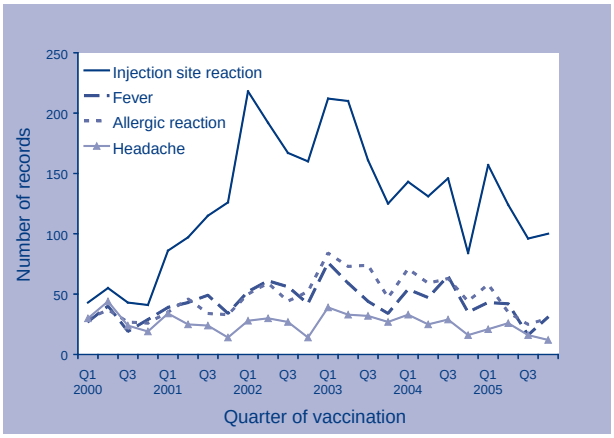
For reports where the date of vaccination was not recorded, the date of onset was used as a proxy for vaccination date.

Figure 2. Frequently suspected vaccines, adverse events following immunisation, ADRAC database, 2000 to 2005, by quarter of vaccination



See appendix for abbreviations of vaccine names.

Figure 3. Selected frequently reported adverse events following immunisation, by quarter of vaccination, ADRAC database, 2000 to 2005



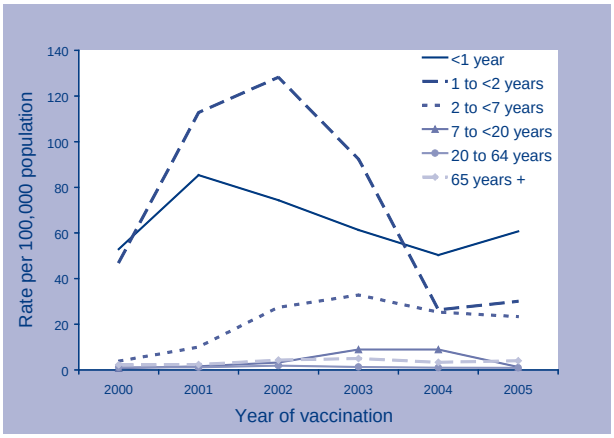
an increase among children aged <1 year (60.7 versus 50.3 per 100,000 population in 2005 and 2004 respectively), children aged 1 to <2 years (30.1 versus 26.3 per 100,000 population) and adults aged ≥65 years (4.0 versus 3.3 per 100,000 population). The reporting rate decreased in other age groups with the largest decrease in the 7 to <20 year age group (0.9 versus 8.9 per 100,000 population) (Figure 4).

The overall male to female ratio was 1:1.2, similar to previous years. The gender ratio varied by age group with slightly lower AEFI reporting rates for females aged <7 years (male:female 1:0.8) and higher reporting rates for females aged ≥7 years (male:female 1:3.1).

Geographical distribution

As noted in previous reports,[6,7,9] the AEFI reporting rate varied between states and territories for vaccines received during 2005 (Table 1). The Australian Capital Territory and the Northern Territory had the highest reporting rates (16.3 and 13.8 per 100,000 population, respectively) while New South Wales, Queensland and Western Australia had the lowest rates (2.7, 2.8 and 2.8 per 100,000 population, respectively). Reporting rates were higher in 2005 compared with those published for 2004[9] for South Australia (11.7 versus 8.3 per 100,000 population), Tasmania (4.3 versus 1.2 per 100,000 population) and Victoria (3.9 versus 2.5 per 100,000 population) and lower in the other states and territories. The increase in reporting rates for South Australia, Tasmania and Victoria appears to be partly related to changes in AEFI surveillance and reporting practices in 2005 compared with 2004 (see Discussion).

Figure 4. Reporting rates of adverse events following immunisation per 100,000 population, ADRAC database, 2000 to 2005, by age group and year of vaccination



Adverse events following immunisation outcomes

Fifty-nine per cent of reported AEFIs in 2005 were defined as 'non-serious' while nine per cent were defined as 'serious' (Table 2)—the same percentage as occurred in 2003 and 2004. One death was recorded as temporally related to influenza vaccine and/or another medication. Fewer 'serious' AEFIs

Table 1. Adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2005, by jurisdiction

Jurisdiction	AEFI records		Annual reporting rate per 100,000 population*			
	n	%	Overall	'Certain' or 'probable' causality rating†	'Serious' outcome‡	Aged <7 years
Australian Capital Territory	53	6	16.3	6.2	0.92	123.4
New South Wales	185	22	2.7	1.1	0.31	16.4
Northern Territory	28	3	13.8	6.4	0	87.2
Queensland	110	13	2.8	1.2	0.25	18.3
South Australia	180	21	11.7	6.4	0.71	102.3
Tasmania	21	3	4.3	3.3	0.62	18.6
Victoria	194	23	3.9	2.2	0.18	31.3
Western Australia	56	7	2.8	1.1	0.40	19.2
Other§	12	1	na	na	na	na
Total	839	100	4.1	2.0	0.32	29.7

* Average annual rates per 100,000 population calculated using mid-2005 population estimates (Australian Bureau of Statistics).

† See previous report[6] for criteria used to assign causality ratings.

‡ AEFI records defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening or death – see Table 2).

§ Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. All AEFI records in this category were notified by pharmaceutical companies.

were assigned 'certain' or 'probable' causality ratings compared with 'non-serious' AEFIs (21% versus 51%) (Table 2). Vaccines listed in records where the outcome was defined as 'serious' are shown in Table 3.

Vaccines and adverse events following immunisation

Twenty-eight vaccines were recorded as 'suspected' of involvement in the adverse events described in the 839 AEFI records for vaccines received in 2005 (Table 3). The most frequently suspected individual vaccine was DTPa with 257 (31%) records (Table 3). Vaccines containing diphtheria, tetanus and acellular pertussis antigens (including combination vaccines and dTpa) were suspected in 381 (45%) records. The second most frequently reported vaccine was 7vPCV with 171 (20%) records. The percentage of records where only one vaccine was suspected of involvement in the adverse event differed by vaccine, as did the percentage assigned causality ratings of 'certain' or 'probable', and defined as 'serious' (Table 3).

AEFI reporting trends differed by vaccine (Figure 2). Reports related to the MMR vaccine remained relatively stable. The number of reports where DTPa vaccine was suspected of involvement in the reported AEFI declined further in 2005 following a peak in the first quarter of 2002, and particularly after the dose due at 18 months of age was removed from the schedule in September 2003. Records listing MenCCV as a suspected vaccine decreased and stabilised in 2005 following a peak in 2003, which coincided with the commencement of the routine (at 12 months of age) and catch-up (aged 1–19 years) MenCCV programs. Records listing 7vPCV as a suspected vaccine peaked at 72 in the first quarter of 2005, following the commencement of the universal infant program on 1 January 2005, then stabilised to approximately 30 records per quarter (Figure 2).

Adverse events following immunisation reactions

The distribution and frequency of reactions listed in AEFI records for 2005 are shown in Tables 4 and 5. In Table 4, only the reaction categories analogous to those listed in the *Australian Immunisation Handbook*[4] are shown. In Table 5, other reaction categories are listed in descending order of frequency.

The most frequently reported adverse events were injection site reaction (57% of 839 AEFI records) followed by allergic reaction (18%), fever (16%) and rash (9%) (Table 4). Injection site reactions were the most commonly reported adverse event following receipt of 23vPPV (84%; 99/118), DTPa (79%; 202/257), MMR (59%; 87/147) and influenza (45%; 42/94) vaccines, administered alone or in combination with other vaccines.

More severe AEFIs included reports of anaphylactic reaction (n=7), severe allergic reaction involving the respiratory and/or circulatory system (n=13), hypotonic-hyporesponsive episode (HHE, n=11), thrombocytopenia (n=3), encephalitis (n=1) and convulsion (n=14), acute flaccid paralysis (AFP; n=1), Guillain-Barré syndrome (GBS; n=1) and sudden death (n=1). The death occurred in a 75-year-old

Table 2. Outcomes of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2005

Outcome	AEFI records		'Certain' or 'probable' causality rating†		Age group‡ <7 years		Age group‡ ≥7 years	
	n	%*	n	%§	n	%§	n	%§
Non-serious	496	59	254	51	333	67	155	31
Not recovered at time of report	201	24	90	45	116	58	81	40
Not known (missing data)	70	8	42	60	46	52	38	43
Serious:	72	9	15	21	35	49	34	47
recovered with sequelae	(1)		(0)		(0)		(1)	
hospital admission	(60)		(20)		(31)		(26)	
life-threatening event	(10)		(0)		(4)		(6)	
death	(1)		(0)		(0)		(1)	
Total	839	100	401	48	530	63	291	35

* Percentages relate to the total number of AEFI records (n=839).

† Causality ratings were assigned to AEFI records using criteria described previously.[6]

‡ AEFI records where both age and date of birth were not recorded are not shown.

§ Percentages relate to the number of AEFI records with the specific outcome e.g. of 496 AEFI records with a 'non-serious' outcome, 51 per cent had causality ratings of 'certain' or 'probable' and 67 per cent were for children aged less than 7 years.

Table 3. Vaccine types listed as 'suspected' in records of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2005

Suspected vaccine type*	AEFI records	One suspected vaccine or drug only†		'Certain' or 'probable' causality rating‡		'Serious' outcome§		Age group‖ <7 years		≥7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶	n	%¶
DTPa	257	146	57	143	56	11	4	254	99	0	–
7vPCV**	171	62	36	31	18	19	11	169	99	1	1
MMR	147	30	20	28	19	10	7	142	97	4	3
23vPPV	118	97	82	83	70	10	8	6	5	108	92
Influenza	94	71	76	33	35	17	18	1	1	92	98
Polio	92	3	3	2	2	12	13	87	95	4	4
Hib	74	4	5	3	4	6	8	72	97	1	1
MenCCV	64	18	28	12	19	7	11	53	83	11	17
DTPa-hepatitis B	57	3	5	3	5	1	2	57	100	0	–
DTPa-IPV††	47	26	55	25	53	4	9	45	96	0	–
Hib-hepatitis B	47	2	4	2	4	11	23	46	98	1	2
dTpa	37	28	76	19	51	1	3	0	–	33	89
Hepatitis B	29	18	62	9	31	3	10	7	24	22	76
Varicella††	20	13	65	3	15	1	5	15	75	4	20
dT	14	12	86	8	57	0	–	0	–	14	100
Hepatitis A	12	9	75	4	33	2	17	3	25	9	75
DTPa-IPV-hepB-hib††	8	2	25	0	–	2	25	8	100	0	–
Hepatitis A-typhoid	6	6	100	2	33	1	17	0	–	6	100
Japanese encephalitis	6	3	50	1	17	1	17	1	17	5	83
Hepatitis A + B	5	3	60	0	–	3	60	0	0	5	100
BCG	3	1	33	1	33	1	33	1	33	2	67
DTPa-IPV-hepB††	3	0	–	0	–	0	–	3	100	0	–
Men4PV	3	1	33	0	–	1	33	0	–	3	100
Rabies	3	3	100	1	33	0	–	0	–	2	67
Tetanus	3	2	67	1	33	1	33	0	–	3	100
Typhoid	2	1	50	0	–	1	50	0	–	2	100
Yellow fever	2	2	100	0	–	0	–	0	–	1	50
Q fever	1	1	100	0	–	1	100	0	–	1	100
Total‡‡	839	567	68	401	48	72	9	530	63	291	35

* See appendix for abbreviations of vaccine names.

† AEFI records where only one vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[6]

§ 'Serious' outcomes are defined in the Methods section (see Table 2 also).

‖ AEFI records not shown if both age and date of birth were not reported.

¶ Percentages are calculated for the number of AEFI records where the specific vaccine was suspected of involvement in the AEFI, e.g. DTPa was listed as 'suspected' in 257 AEFI records; this was the only suspected vaccine in 57 per cent of the 257 AEFI records, 55 per cent had 'certain' or 'probable' causality ratings, 4 per cent were defined as 'serious' and 99 per cent were for children aged less then 7 years.

** Pneumococcal conjugate vaccine added to the National Immunisation Program Schedule on 1 January 2005.

†† Varicella vaccine and combination vaccines containing inactivated poliovirus were added to the National Immunisation Program Schedule on 1 November 2005.

‡‡ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than one vaccine.

Table 4. Reaction categories of interest* mentioned in records of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2005

Reaction category*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						<7 years		≥7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Injection site reaction	477	312	65	359	75	312	65	157	33
Allergic reaction¶	148	42	28	38	26	97	66	48	32
severe allergic reaction¶	13	0	–	1	8	3	23	10	77
Fever	132	4	3	39	30	89	67	42	32
Rash	75	21	28	18	24	60	80	14	19
Abnormal crying	30	5	17	4	13	29	97	1	3
Arthralgia	14	2	14	5	36	2	14	11	79
Convulsions	14	8	57	3	21	10	71	3	21
HHE**	11	5	45	1	9	11	100	0	–
Lymphadenopathy/itis††	8	1	13	3	38	3	38	5	63
Anaphylactic reaction	7	2	29	1	14	2	29	5	71
Parotitis	3	1	33	0	–	3	100	0	–
Thrombocytopenia	3	1	33	0	–	3	100	0	–
Abscess	1	1	100	1	100	0	–	1	100
Acute flaccid paralysis	1	0	–	0	–	1	100	0	–
Arthritis	1	1	100	0	–	0	0	1	100
Death	1	0	–	0	–	0	–	1	100
Encephalitis	1	0	–	0	–	0	–	1	100
Guillain-Barré syndrome	1	0	–	0	–	0	–	1	100
Osteomyelitis	1	0	–	0	–	1	–	0	–
Brachial neuritis	0	–	–	–	–	–	–	–	–
Encephalopathy	0	–	–	–	–	–	–	–	–
Meningitis	0	–	–	–	–	–	–	–	–
Orchitis	0	–	–	–	–	–	–	–	–
Osteitis	0	–	–	–	–	–	–	–	–
Sepsis	0	–		–	–	–	–	–	–
SSPE‡‡	0	–	–	–	–	–	–	–	–
Toxic shock syndrome	0		–	–	–	–	–	–	–
Total§§	839	448	53	401	48	530	63	291	35

* Reaction categories were created for the AEFIs of interest listed and defined in the *Australian Immunisation Handbook*, (8th edition, p 22–3 and 271–5)[4] as described in the Methods section.

† AEFI records where only one reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[6]

§ Not shown if neither age nor date of birth were recorded.

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 477 AEFI records listing injection site reaction, 65 per cent listed only one type of reaction while 75 per cent had a causality rating of 'certain' or 'probable' and 65 per cent were for children aged less than 7 years.

¶ Allergic reaction includes skin and/or gastrointestinal (e.g. diarrhoea, vomiting) symptoms and signs.[4] The category 'severe allergic reaction' includes allergic reaction with involvement of the circulatory and/or respiratory system but not recorded in the ADRAC database as 'anaphylactic reaction'.[4]

†† Includes lymphadenitis following BCG vaccination and the more general term of 'lymphadenopathy'.

** Hypotonic-hyporesponsive episode.

‡‡ Subacute sclerosing panencephalitis.

§§ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than one reaction term.

Table 5. 'Other'* reaction terms listed in records of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2005

Reaction term*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						<7 years		≥7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Malaise	48	0	0	20	42	21	44	26	54
Oedema	38	5	13	22	58	19	50	17	45
Pain	37	0	–	19	51	9	24	27	73
Nausea	28	0	–	12	43	7	25	20	71
Respiratory rate/rhythm change	28	2	7	3	11	14	50	14	50
Irritability	27	0	–	6	22	27	100	0	–
Headache	24	10	42	12	50	5	21	18	75
Pallor	22	0	–	5	23	15	68	6	27
Myalgia	19	1	5	4	21	1	5	18	95
Syncope	19	5	26	9	47	1	5	17	89
Increased sweating	17	0	–	7	41	3	18	12	71
Dizziness	16	0	–	8	50	0	0	14	88
Heart rate/rhythm change	15	1	7	1	7	9	60	6	40
Anorexia	13	0	–	5	38	8	62	5	38
Reduced sensation	13	2	15	3	23	0	0	13	100
Cough	10	0	–	2	20	4	40	5	50
Other									
General non-specific	31	2	6	11	35	12	39	19	61
Neurological	19	2	11	6	32	8	42	11	58
Psychological	18	2	11	7	39	11	61	7	39
Cardiovascular	17	1	6	3	18	8	47	9	53
Gastrointestinal	14	2	14	2	14	5	36	8	57
Haematological	14	0	–	3	21	7	50	6	43
Eye or ear	13	1	8	4	31	6	46	7	54
Respiratory	13	3	23	2	15	4	31	9	69
Skin	13	3	23	2	15	8	62	5	38
Musculoskeletal	11	2	18	3	27	2	18	9	82
Metabolic/endocrine	10	2	20	1	10	6	60	1	10
Infection	7	0	–	2	29	8	114	2	29
Renal/urogenital	5	1	20	2	40	1	20	4	80

* Reaction terms not listed in the *Australian Immunisation Handbook*[4] but included in AEFI records in the ADRAC database. The top part of the table shows reaction terms included in one per cent or more of AEFI records; the bottom part of the table shows reaction terms grouped by organ system that were included in less than one per cent of AEFI records.

† AEFI records where only one reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[6]

§ Not shown if neither age nor date of birth were recorded.

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 477 AEFI records listing injection site reaction, 65 per cent listed only one type of reaction while 75 per cent had a causality rating of 'certain' or 'probable' and 65 per cent were for children aged less than 7 years.

person who became unwell two days after receiving an influenza vaccine and died eight days later. Both influenza vaccine and a prescription medication were coded as 'possibly' related to the person's death. The single case of GBS was in a 57-year-old person following receipt of influenza vaccine. The case of AFP (transverse myelitis) occurred in a 6-month-old child following receipt of oral polio vaccine and was reported by the Australian AFP Surveillance Program.[18] The Polio Expert Committee classified the case as 'non-polio AFP'.

Five of the seven AEFI reports of anaphylactic reaction were for adults—three following influenza vaccine and two following receipt of a combined hepatitis A and B vaccine. Of the 14 reports of convulsion, 10 were in children aged <7 years following routinely scheduled combinations of vaccines. The most commonly suspected vaccines were MMR (n=4) and polio (n=4). All 11 reports of HHE listed 7vPCV as suspected of involvement, usually in combination with other routine childhood vaccines.[9,10,19] DTPa-containing vaccines were listed as suspected of involvement in the HHE for nine of the 11 children.

Reactions mentioned in fewer than one per cent of AEFI records in 2005 are shown in the lower portion of Table 5, grouped by organ system categories. The most commonly reported category was coded as 'general non-specific' reactions, which included reaction terms such as 'feeling hot', 'feeling cold' and 'discomfort'.

The trends in the most frequently reported types of reactions changed over time (Figure 3). Overall, there were fewer reports of injection site reaction in 2005 compared with previous years. Reports of allergic reaction, fever and rash were less variable over time and reports of headache were lower in 2005 compared with 2004 and 2003, consistent with the decrease in reporting of adverse events following MenCCV as the adolescent catch-up program was concluded.

Although there were fewer reports of injection site reaction in 2005, the percentage of reports for 23vPPV that listed injection site reaction as an AEFI has increased over time. This is particularly evident for adults aged ≥65 years where the percentage of reports for 23vPPV that listed injection site reaction, increased from 50 per cent of reports in 2001 to 87 per cent in 2005 (Figure 5).

Dose-based adverse events following immunisation reporting rates

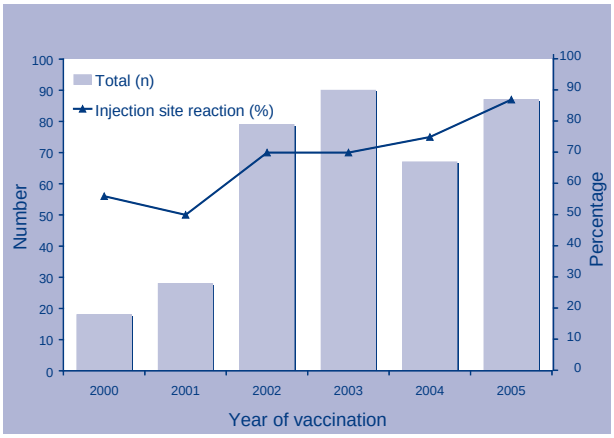
Influenza vaccine and adults aged ≥18 years

In 2005, influenza vaccine was suspected of involvement in 91 AEFI records for people aged ≥18 years. The dose-based AEFI reporting rates (using 2004 coverage data), by age group, are shown in Table 6. As seen previously,[6,7,9] the AEFI reporting rate in 2005 was lower among influenza vaccinees aged ≥65 years than for younger vaccinees (Table 6). The most frequently reported adverse events were injection site reaction, fever and allergic reaction (0.9, 0.3 and 0.3 per 100,000 doses, respectively). The estimated reporting rate of injection site reactions in 2005 was approximately 50 per cent higher than seen than in 2004 for all age groups. Again, the highest reporting rate of injection site reactions was among younger vaccinees aged 18–39 years (1.5 per 100,000 doses) compared with the 40–64 year and ≥65 year age groups (1.1 and 0.5 per 100,000 doses, respectively). The single report of GBS following influenza vaccination in a person aged 40–64 years (Table 4) corresponds to a reporting rate of 0.06 per 100,000 doses for persons aged 40–64 years and 0.02 per 100,000 doses for persons aged ≥40 years, compared with 0.03 per 100,000 doses in 2004.[9]

Scheduled vaccines for children aged <7 years

Dose-based AEFI reporting rates for eight NIPS vaccines administered during 2005 to children aged <7 years are shown in Table 7 (by vaccine) and Table 8 (by age group). The reporting rate for 7vPCV, which was added to the NIPS in January 2005, was 14.7 per 100,000 doses recorded on the ACIR. Dose-based AEFI reporting rates for most vaccines were

Figure 5. Trends in reporting of all adverse events and injection site reactions following 23vPPV for adults aged ≥65 years, ADRAC database, 2000 to 2005, by year of vaccination



similar to, or lower than, 2004 estimates although there was an increase in the AEFI reporting rates for DTPa and Hib-HepB (Table 7).

The reporting rate across all vaccines for children aged <7 years declined slightly in 2005 (11.0 versus 13.0 per 100,000 doses) (Table 7), and varied by age group (Table 8). The rate increased among children aged <1 year (5.9 versus 5.5 per 100,000 doses), was stable for children aged 1 to <2 years and decreased slightly for children aged 2 to <7 years (Table 8). This age group had the highest dose-based reporting rate among children aged <7 years (30.1 per 100,000 doses). The main contributor to this was injection site

Table 6. Reporting rate of adverse events following immunisation (AEFI) per 100,000 doses of influenza vaccine,* 18 years and over, ADRAC database, 2005

AEFI category†	Age group	AEFI records‡ (n)	Vaccine doses* (n)	Rate per 100,000 doses§		Ratio of 2005 to 4-yr mean‖
				2005	2004	
Overall	≥18 years	91	4,447,500	2.0	1.8	–¶
	18–39 years	19	732,700	2.6	2.7	–¶
	40–64 years	48	1,653,300	2.9	2.2	1.0
	≥65 years	24	2,061,500	1.2	1.1	0.9
Serious	≥18 years	16	4,447,500	0.36	0.27	–¶
	18–39 years	1	732,700	0.14	0.0	–¶
	40–64 years	9	1,653,300	0.54	0.54	1.8
	≥65 years	6	2,061,500	0.29	0.24	1.0

* Number of administered doses of influenza vaccine estimated from the 2004 national influenza survey.[15]

† AEFI category includes all records, and those defined as 'serious' where influenza vaccine was suspected of involvement in the reported adverse event. The definition of a 'serious' outcome is shown in the Methods section.

‡ Number of AEFI records in which influenza vaccine was 'suspected' and the vaccination was administered in 2005.

§ The estimated reporting rate of adverse events per 100,000 administered doses of influenza vaccine.

‖ Ratio of the reporting rate per 100,000 doses for 2005 and the average (mean) reporting rate per 100,000 doses for the previous 4 years (2001–2004).

¶ Influenza immunisation rates for the 18–39 year age group were not estimated before 2004, therefore the 4-year average AEFI reporting rates and rate ratios for this age group have not been estimated.

Table 7. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* by vaccine, children aged less than 7 years, ADRAC database, 1 January to 31 December 2005

Suspected vaccine type†	AEFI records (n)	Vaccine doses* (n)	Rate per 100,000 doses‡		Ratio of 2005 to 4-yr mean§
			2005	2004	
DTPa	254	474,852	53.5	47.9	0.9
DTPa-HepB	57	388,029	14.7	15.4	0.7
Hib	72	408,237	17.6	20.4	0.6
Hib-HepB	46	283,650	16.2	9.1	1.5
Polio	87	856,211	10.2	10.3	0.8
7vPCV	169	1,156,487	14.7	–	–
MenCCV	53	304,969	17.4	30.8	–
MMR	142	505,333	28.1	33.6	0.9

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 January and 31 December 2005.

† AEFI records where the vaccine was one of those listed as 'suspected' of involvement in the reported adverse event. See appendix for abbreviations of vaccine names.

‡ The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the ACIR.

§ Ratio of the AEFI reporting rate per 100,000 doses for 2005 and the average (mean) reporting rate per 100,000 doses for the previous four years (2001–2004). The reporting rate ratio was not estimated for vaccines funded by the National Immunisation Program for less then five years.

reactions following DTPa vaccine (reporting rate of 79.0 per 100,000 doses). The reporting rate of injection site reactions following DTPa vaccine in this age group has been stable at 76–80 per 100,000 doses for the four years 2003–2005.

For all age groups, the dose-based reporting rates of AEFI defined as 'serious' were lower in 2005 compared with 2004 and the average rate for the four years 2001–2004 (Table 8). The reporting rate for HHE following 7vPCV was 1.35 per 100,000 doses for children aged <1 year. This is similar to the combined reporting rates of HHE following DTPa or DTPa-HepB vaccine (1.33 per 100,000 doses) and to rates estimated previously for DTPa containing vaccines (1.23 per 100,000 doses).[9]

Discussion

The data show an overall decrease in AEFI reports in 2005 compared with the three previous years (2002–2004), although this was not consistent across age groups, vaccines or states and territories. A number of factors may explain the observed AEFI reporting trends including several significant changes to the funded NIPS in the past few years and known differences in AEFI surveillance and reporting practices between states and territories and over time. Importantly, the proportion of reports coded as 'serious' remained stable at nine per cent, while the dose-based reporting rate of serious AEFIs for children aged <7 years decreased from 1.0 to 0.7 per 100,000 doses (Table 8).

The largest increase in AEFI reports in 2005 occurred among children aged <1 year and coincided with the introduction of the universally funded 7vPCV program for children in this age group from 1 January 2005. As frequently observed following the introduction of new vaccines or the expansion of an immunisation program,[3,7,9,20] AEFI reports where 7vPCV was suspected of involvement peaked in the first quarter of 2005 then stabilised in the next three quarters (Figure 3). Observed increases in the dose-based reporting rates of DTPa and Hib-HepB vaccines in 2005, compared with 2004 (Table 7), may relate to increased reporting of AEFI following 7vPCV as the vaccines are given to children at the same time points in the immunisation schedule.

Table 8. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* ADRAC database, 1 January to 31 December 2005, by age group, for children aged less than 7 years

AEFI category†	Age group	AEFI records‡ (n)	Vaccine doses* (n)	Rate per 100,000 doses§		
				2005	2004	Ratio of 2005 to 4–yr mean‖
All records	Total	482	4,374,768	11.0	13.0	0.6
	<1 year	150	2,535,194	5.9	5.5	0.8
	1 to <2 years	65	951,887	6.8	6.8	0.2
	2 to <7 years	267	887,687	30.1	33.8	1.0
'Serious' outcome†	Total	30	4,374,768	0.7	1.0	0.6
	<1 year	15	2,535,194	0.6	0.9	0.6
	1 to <2 years	9	951,887	0.9	1.0	0.5
	2 to <7 years	6	887,687	0.7	1.2	0.8
'Certain' or 'probable' causality rating†	Total	206	4,374,768	4.7	5.3	0.6
	<1 year	20	2,535,194	0.8	0.8	0.5
	1 to <2 years	16	951,887	1.7	1.0	0.1
	2 to <7 years	170	887,687	19.2	18.2	1.1

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 January and 31 December 2005.

† AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those defined as 'serious' where at least one of the seven vaccines shown in the table was suspected of involvement in the reported adverse event. Causality ratings were assigned using the criteria described previously.[6] The definition of a 'serious' outcome is described in the Methods section.

‡ Number of AEFI records in which the vaccine was coded as 'suspected' and the vaccination was administered between 1 January and 31 December 2005.

§ The estimated rate of adverse events records per 100,000 vaccine doses recorded on the ACIR.

‖ Ratio of the reporting rate per 100,000 doses for 2005 and the average (mean) reporting rate per 100,000 doses for the previous four years (2001–2004). The reporting rate ratio was not estimated for vaccines included in the National Immunisation Program for less then five years.

The overall dose-based reporting rate for 7vPCV was 14.7 per 100,000 doses, lower than for most of the vaccines given to children <7 years where dose-based reporting rates can be estimated (Table 7). The types of reactions following 7vPCV were similar to those reported in the USA for 7vPCV including mild allergic reaction, rash, fever and fussiness.[10,19] Reports of HHE following administration of 7vPCV have occurred in the USA and Australia, although a causal relationship has not been established between the vaccine and HHE.[19]

There was a significant decrease in the number of AEFI reports in 2005 for children aged 7 to <20 years following the completion of the school-based MenCCV catch-up program at the end of 2004. The majority of AEFI reports mentioning MenCCV vaccine were for children aged 1 to <2 years who received the vaccine as part of the routine childhood schedule at approximately 12 months of age. Reporting of adverse events following MenCCV appear to have stabilised with an average of 13 reports received per quarter in 2005, down from a peak of 96 reports in first quarter of 2003 when the program commenced (Figure 2).

Children aged 2 to <7 years continue to have the highest dose-based AEFI reporting rates of all age groups with injection site reactions following a 5th dose of acellular pertussis-containing vaccines being the largest contributor. Injection site reactions and extensive limb swelling are a known and relatively frequent adverse event associated with 4th and 5th doses of acellular pertussis-containing vaccines.[21,22] Studies show that children recover without sequelae.[21,22] The reporting rate has stabilised at 76–80 reports of injection site reaction per 100,000 doses of DTPa vaccine over the four years to 2005. This trend may be influenced in the future by the removal of the 4th dose of DTPa (due at 18 months of age) from the schedule in September 2003[4] and replacement of DTPa with DTPa-IPV for the dose due at 4 years of age in November 2005.[11]

The AEFI reporting rate for adults ≥65 years of age increased slightly between 2004 and 2005 from 3.3 to 4.0 per 100,000 population, while the dose-based AEFI reporting rate for influenza vaccine remained stable at 1.2 per 100,000 (using 2004 denominator data). Most of the increase in AEFI reporting for this age group between 2004 and 2005 appears to be related to an increase in the number of reports of injection site reaction following 23vPPV (Figure 5). Published data suggest that the incidence of injection site reactions following a second dose of 23vPPV is higher than for the first dose,[23] although one study from the US Vaccine Safety Datalink project found there was relatively little difference in the rate of medical consultation for injection site reaction following a first versus second dose of 23vPPV.[24] Dose number was recorded for only 45 per cent of AEFI records in the ADRAC database for injection site reaction following 23vPPV among those aged ≥65 years. However, of these, approximately two-thirds indicated that the reaction followed a second dose of 23vPPV.

States and territories differ markedly in AEFI surveillance practices and reporting practices. Previously, clear patterns were evident where differences in population-based AEFI reporting rates generally corresponded to the type of AEFI reporting requirements in each state and territory.[7,9] Specifically, Victoria and Tasmania, which both request that general practitioners and other reporters notify AEFI directly to ADRAC, had lower reporting rates than other states and territories. However, in 2005, population-based AEFI reporting rates increased for both Victoria and Tasmania while the overall reporting rate and that for most states and territories decreased (Table 1). The change appears to be related to increased reporting of AEFIs by nurse immunisers in Victoria and Tasmania and coincides with changes to the nurse immuniser accreditation program in Victoria in 2004 to emphasise AEFI reporting (H Pitcher, personal communication), and an increase in the number of nurse immunisers in Tasmania in 2005 (A Misrachi, personal communication). The higher reporting rate for South Australia in 2005 compared with the published rate for 2004[9] is related to increased timeliness of reporting to ADRAC by the cut-off date for inclusion of data in the annual report (31 March of each year).

Conclusions

The data presented in this report indicate that the majority of AEFIs that occur in Australia and are reported to ADRAC are mild, transient and expected vaccine side-effects such as injection site reaction, fever and minor allergic reaction. There was one report of death in an older person following influenza vaccine and another medication and one of non-polio acute flaccid paralysis in an infant. Both were temporally associated with immunisation, and causation was assessed as possible. Serious AEFIs remained stable at nine per cent of all reports to ADRAC and the overall rate of serious AEFI per 100,000 vaccine doses declined among children aged <7 years.

The benefits of immunisation in preventing disease significantly outweigh the risks of immunisation-related adverse events for the Australian population. Immunisation coverage and disease notification data continue to show high immunisation coverage levels[16,25] and low rates of vaccine preventable diseases with significant reductions on the incidence, morbidity and mortality of diseases such as Hib, invasive pneumococcal disease, meningococcal C disease and measles.[25–29]

This is the sixth regular report analysing AEFIs in Australia detected by the national passive surveillance system.[6–10] The data reported here demonstrate that the system is able to detect both known rarer adverse events and expected changes in AEFI reporting trends following changes to the NIPS. The next planned report, analysing AEFI data for children aged <7 years to 30 June 2006, will provide further information on AEFIs reported for new vaccines introduced into the schedule for children aged <7 years from November 2005, including IPV combination vaccines and varicella vaccine.

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Appendix

Abbreviations of vaccine types

23vPPV: 23–valent pneumococcal polysaccharide vaccine

7vPCV: 7–valent pneumococcal conjugate vaccine

BCG: Bacille Calmette-Guèrin (i.e. tuberculosis)

dT: diphtheria-tetanus – adolescent and adult formulation

DTPa: diphtheria-tetanus-pertussis (acellular) – paediatric formulation

dTpa: diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation

DTPa-hepB: combined diphtheria-tetanus-pertussis (acellular) and hepatitis B

DTPa-IPV: combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus (quadrivalent)

DTPa-IPV-hepB: combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus and hepatitis B (pentavalent)

DTPa-IPV-hepB-hib: combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus, hepatitis B and *Haemophilus influenzae* type b vaccine (hexavalent)

HepB: hepatitis B

Hib: *Haemophilus influenzae* type b

Hib-hepB: combined *Haemophilus influenzae* type b and hepatitis B

Men4PV: meningococcal polysaccharide tetravalent vaccine

MenCCV: meningococcal C conjugate vaccine

MMR: measles-mumps-rubella

polio: poliovirus (oral and inactivated vaccine)

Annual report of the Australian National Poliovirus Reference Laboratory 2005

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Abstract

In May 1988 the World Health Assembly adopted a resolution for the global eradication of poliomyelitis. Since then two target dates for eradication (2000 and 2003) have passed and the struggle to eradicate the poliovirus continues. Australia's commitment to the worldwide campaign began in December 1994 with the designation of the National Poliovirus Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory and the initiation of acute flaccid paralysis (AFP) surveillance in March 1995. During 2005 the National Poliovirus Reference Laboratory did not isolate any wild or vaccine derived polioviruses from the 42 samples collected from eighteen cases of acute flaccid paralysis in Australian residents. Three Sabin–like polioviruses were isolated from three cases of acute flaccid paralysis but all were considered incidental isolations by the Polio Expert Committee and not implicated in the disease of the patients. After exceeding the World Health Organization target of one case of AFP per 100,000 children aged less than 15 years in 2004, Australia's non-polio AFP rate in 2005 fell to 0.75 cases per 100,000 children. The high number of wild poliovirus importations reported globally in 2005 into previously polio free countries, highlights the need for a sensitive AFP surveillance system within Australia and for specimens from AFP cases to be forwarded to the National Poliovirus Reference Laboratory. *Commun Dis Intell* 2006;30:334–340.

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus

Introduction

Acute flaccid paralysis (AFP) is the main clinical manifestation of poliomyelitis and occurs in approximately one per cent of poliovirus infections. Surveillance for AFP cases in children along with high polio immunisation coverage has been the hallmark of the World Health Organization (WHO) Global Polio Eradication Program since its inception. AFP surveillance in Australia is conducted by the National Polio Reference Laboratory (NPRL) located at the Victorian Infectious Diseases Reference Laboratory (VIDRL) in conjunction with the Australian Paediatric Surveillance Unit (APSU). All faecal specimens collected from cases of AFP in Australia, are forwarded to the NPRL for testing for poliovirus and other enteroviruses. All cases of AFP are reviewed by the Australian Polio Expert Committee (PEC). The NPRL is also the national laboratory for the Pacific Island countries and Brunei Darussalam, and is a regional reference laboratory for the WHO Western Pacific Region.

Since September 1966 the Australian Standard Immunisation Schedule has included the Sabin oral poliovirus vaccine (OPV) as the vaccine of choice for immunisation against poliovirus infection.[1] OPV is a trivalent vaccine comprising all three poliovirus serotypes. After administration of the vaccine, the viruses multiply in the gut of the recipient and can be excreted for up to six weeks from immunocompetent individuals.[2] Longer excretion times for immunocompromised recipients have been documented.[3]

In November 2005 the Australian National Immunisation Program introduced inactivated poliovirus vaccine (IPV) as the recommended vaccine.[4] The introduction of IPV into the schedule will eliminate the risk of vaccine associated poliomyelitis (VAPP) which occurs in one in 2.5 million administered doses of OPV.[5] Vaccination with IPV will also eliminate the isolation of incidental polioviruses in Australian virology laboratories as vaccine viruses will no longer be excreted by poliovirus vaccine recipients.

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The activities of the Australian National Poliovirus Reference Laboratory in 2005 are summarised in this annual report which also includes a comparison of AFP surveillance in Australia against the major targets nominated by WHO.

Methods

The approach adopted by Australia for AFP surveillance is as follows:

- paediatricians reviewing a patient aged less than 15 years and presenting with AFP or a clinician reviewing a patient of any age suspected of poliomyelitis are requested to notify the national AFP surveillance co-ordinator at VIDRL. Notification of the case is also included on the paediatrician's monthly report card to the APSU;
- two faecal specimens should be collected 24 to 48 hours apart and within 14 days of onset of paralysis;
- the faecal specimens should be referred for testing to the WHO accredited NPRL located at VIDRL;
- clinicians are requested to complete a clinical questionnaire upon notification of the case;
- the PEC convened by the Australian Government Department of Health and Ageing reviews the clinical and laboratory data for all notified cases of AFP; irrespective of whether they are an eligible or ineligible case;
  - the PEC, case definition for AFP is: 'An eligible case is, any child under 15 years of age with acute flaccid paralysis (including Guillain-Barré syndrome) or any person of any age with paralytic illness if polio is suspected.
  - An ineligible case is an AFP case outside the case definition: patients aged greater than 15 years, an overseas resident, or a case notified in error by a clinician.
- the PEC classifies cases of AFP as poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated poliomyelitis; non-polio AFP; or non-AFP;
- a follow-up questionnaire is sent to notifying clinicians if the PEC requires more information regarding the AFP case before a final classification can be made;
- Australian AFP data is forwarded to WHO on a quarterly basis for inclusion in the global AFP surveillance data published in the *Weekly Epidemiological Report*, (available from http://www.who.int/wer/en/);
- at the end of each calendar year a small number of eligible cases are not classified by the PEC as clinical and laboratory data are not available from the notifying clinician.

On receipt at the NPRL, faecal specimens are extracted in a 10 per cent v/v chloroform solution in Modified Essential Medium with Earles salts and inoculated onto a series of continuous cell lines. The main WHO cell line utilised for the isolation of poliovirus is L20B,—a mouse epithelial cell line with cell surface expression of the human poliovirus receptor, CD155.[6,7] The NPRL employs three other cell lines for the isolation of poliovirus and other enteroviruses. They are RD (human rhabdomyosarcoma) also recommended by the WHO, HEp2 Cincinnati (human epidermoid carcinoma) and HEL (human embryonic lung). Other laboratories within Australia refer enteroviruses of unknown serotype to the NPRL for further characterisation. All polioviruses, whether isolated from AFP cases or other sources, undergo a process known as intratypic differentiation (ITD). ITD distinguishes between wild and vaccine strains of poliovirus. ITD involves a genetic based method, [polymerase chain reaction (PCR)] and an antigenic based method, [enzyme–linked immunosorbent assay (ELISA)]. These methods have been described in detail in previous annual reports.[8,9]

Polioviruses isolated from Australian AFP cases and those with discordant ITD results are sequenced routinely by the NPRL. Two regions of the poliovirus genome are sequenced. The VP1 capsid gene where greater than one per cent changes compared to the parental OPV strain, indicates the presence of a vaccine-derived poliovirus as defined by the WHO.[10] A portion of the 3D gene is also sequenced to provide information on whether the virus has undergone a recombination event with another poliovirus or enterovirus during replication.

The NPRL is accredited annually as a national and regional polio reference laboratory, through proficiency testing and an on-site visit by the WHO.

Results

AFP surveillance

In 2005, no Australian AFP cases were due to wild poliovirus, VDPV or VAPP. There were a total of 59 notifications, 36 eligible cases, 15 duplicate notifications and eight ineligible cases.

Polio Expert Committee reviews

Clinical and laboratory information was available for review by the PEC for 37 of the 44 eligible and ineligible AFP notifications. This included 30 of

the 36 eligible cases, and seven ineligible cases. Six eligible cases and one ineligible case, remain unclassified by the PEC due to lack of clinical and laboratory data.

The WHO target for AFP surveillance in a non-endemic country is one case of AFP per 100,000 children aged less than 15 years.[11] Australia's non-polio AFP notification rate in 2005 for Australian residents was 0.75 cases per 100,000 children.

Notification rates in States and Territories

Three of the unclassified cases were from Western Australia with the others located in New South Wales and Queensland. The differences in the rates of notification of AFP cases between states and territories continued in 2005 as reported previously,[8] with only New South Wales and Tasmania reaching the expected target. AFP data for Australian states and territories is presented in Table 1.

Faecal collection

Adequate faecal collection at 19 per cent was well below the expected WHO target of 80 per cent of notified cases[11] (Table 2). Adequate faecal collection is defined by WHO as two faecal specimens collected 24 hours apart and within 14 days of onset of paralysis. Four of the seven (57%) AFP cases with adequate faecal collection were from New South Wales with the remaining three cases from Queensland. Six of the seven cases with adequate faecal collection were first notified to the NPRL, but 38 (64%) of all notifications were first notified to the APSU.

Table 1. Notified acute flaccid paralysis cases, aged less than 15 years, 2005, by Australian state or territory of residence

State or territory	Estimated population aged <15 years*	Expected number of AFP cases per year	Number of eligible notifications	Number of eligible cases classified by the PEC	Notification rate per 100,000 population aged <15 years for eligible cases	Notification rate per 100,000 population aged <15 years for cases classified by the PEC
ACT	62,448	0.5	0	0	0.00	0.00
NSW	1,319,450	13	18	17	1.40	1.30
NT	50,521	0.5	0	0	0.00	0.00
Qld	807,065	8	9	7	1.10	0.88
SA	283,610	3	0	0	0.00	0.00
Tas	96,516	1	1	1	1.00	1.00
Vic	958,596	10	4	4	0.40	0.40
WA	390,274	4	4	1	1.00	0.25
Australia	3,978,221	40	36	30	0.90	0.65

* Australian Bureau of Statistics, estimated population, preliminary – 30 June 2005. ABS publication 3201.0, June 2005.

AFP Acute flaccid paralysis.

PEC Polio Expert Committee.

Table 2. AFP surveillance compared with WHO indicator targets for children less than 15 years, Australia, 2005

WHO indicator target for AFP cases of children less than 15 years	Australia's surveillance for AFP cases with onset in 2005	Australia's AFP surveillance rates for 2005
Non-polio AFP case rate of 1.0 per 100,000 children (40 cases for Australia in 2005).	36 eligible AFP cases notified.	AFP notification rate: 0.9 cases per 100,000 children.
	30 eligible AFP cases classified by the PEC as non-polio AFP.*	Non-polio AFP notification rate: 0.75 cases per 100,000 children.
More than 80% of notified AFP cases with 2 adequate stool specimens collected at least 24 hours apart within 14 days of onset of paralysis.	7 eligible AFP cases with 2 or more adequate specimens per case.	Referral of adequate specimens from AFP cases: 19% (7/36) of the eligible cases.

* Six cases require clinical information from the referring doctor before final classification by the PEC.

Polio Expert Committee classification of acute flaccid paralysis cases

Gullian-Barré Syndrome continued to be the most common diagnosis of non-polio AFP cases classified by the PEC (30% of cases) in 2005, followed by transverse myelitis (14%) and infant botulism (8%). Poliovirus type 3 (PV3), Sabin-like, was isolated from one case of transverse myelitis and one case of infant botulism. Poliovirus type 2 (PV2), Sabin-like, was isolated from one case of transverse myelitis in a patient aged greater than 15 years. All isolations were considered incidental by the PEC.

Laboratory testing of specimens

AFP cases

The NPRL received a total of 42 samples from 18 AFP cases within Australia in 2005. This included 32 faecal specimens from 13 cases of AFP and one faecal extract from one case of AFP in children aged less than 15 years. A further seven faecal specimens and two swabs were collected from four AFP cases aged greater than 15 years. Results of testing are presented in Table 3.

Isolations of poliovirus

In June 2005, PV3 was isolated from three faecal specimens from a six-month-old child from Queensland. The specimens were collected 69, 70 and 71 days post-vaccination with OPV. The virus was characterised as Sabin-like by the WHO approved methods of ITD. Three further specimens, collected 106, 113 and 162 days post-vaccination, did not yield any poliovirus. The VP1 gene was sequenced for all three poliovirus isolates. The nucleotide homology for the VP1 gene to the parental Sabin strain was greater than 99 per cent for all three poliovirus isolates confirming their classification as Sabin-like. No evidence of a recombination event was detected in the 3D gene.

The PEC classified the case as non-polio AFP, diagnosed as transverse myelitis with the isolation of a Sabin-like PV3 that may have a possible association.

In August 2005, PV3 was isolated from two faecal specimens collected from a four-month-old infant in Queensland. The onset of symptoms occurred seven days post-vaccination with OPV. Faecal specimens were collected 14 and 15 days post-vaccination. The viruses were characterised as Sabin-like by the WHO approved methods of ITD. The VP1 gene was sequenced and the nucleotide homology for the VP1 gene to the parental Sabin strain was greater than 99 per cent, confirming the classification as Sabin-like.

Faecal specimens were tested with mice, and a type B/E toxin producing *Clostridium botulinum* was detected. Based on this evidence the PEC classified the case as non-polio AFP, diagnosed as infant botulism.

A patient aged greater than 15 years presented with AFP 10 days post-vaccination with OPV in September 2005. Vaccine had been administered prior to travel to Indonesia, where the onset of symptoms occurred. PV2 was isolated from a faecal specimen collected 23 days post-vaccination upon return to Australia. The virus was characterised as Sabin-like by the WHO approved methods of ITD. The VP1 gene was sequenced and the nucleotide homology for the VP1 gene to the parental Sabin strain was greater than

Table 3. Test results of specimens and isolates referred to the Australian National Poliovirus Reference Laboratory, Australia, 2005

Result	Isolations from AFP cases*	Isolations from non-AFP referred samples	Total
Poliovirus Sabin-like type 1	0	6	6
Poliovirus Sabin-like type 2	2†	4	6
Poliovirus Sabin-like type 3	5	1	6
Adenovirus	2	0	2
Rhinovirus	0	1	1
NPEV‡	0	10	10
No virus isolated	33	3	36
Total	42	25	67

* Includes eligible and ineligible cases.

† Isolated from an ineligible case.

‡ NPEV: non-polio enterovirus. Molecular sequence results of NPEV from non-AFP sources identified coxsackievirus B2 (3 isolates), echovirus 11 (1 isolate), echovirus 18 (2 isolates), echovirus 25 (1 isolate) and echovirus 30 (3 isolates).

AFP Acute flaccid paralysis.

99 per cent, confirming the classification as Sabin-like. The 3D gene had 100 per cent homology to the parental Sabin strain and therefore no evidence of a recombination event. PV2 was also isolated from a rectal swab received by the NPRL in October 2005. The PEC classified the case as non-polio AFP, diagnosed as transverse myelitis with the isolation of an incidental Sabin-like PV2.

Adenovirus was isolated and confirmed by PCR from two faecal specimens from one case of AFP from New South Wales. No serotyping was performed on this isolate. The increase in adenovirus isolations from AFP cases observed during 2004[8] did not continue in 2005.

No enterovirus was isolated after 14 days in culture, from the remaining 33 faecal specimens, faecal extract and swab received from all AFP cases.

Polio serology

Polio serology testing is available through the NPRL for patients with a suspected case of acute poliomyelitis.

Polio serology was performed on paired sera from a nine-year-old child from South Australia with onset of paralysis in September 2004. The titres determined for poliovirus type 1, 2 and 3 in the acute and convalescent sera, were consistent with evidence of past infection or immunisation with poliovirus type 1, 2 and 3 but there was no evidence of seroconversion to any of the three poliovirus serotypes. The PEC classified the case as non-polio AFP diagnosed as anterior horn cell disease (motor neuropathy) causing monoplegia. No faecal specimens were available for this case.

Samples from sources other than AFP cases

Eleven polioviruses were identified from 23 samples referred from sources other than AFP cases during 2005 (Table 3).

A laboratory in South Australia referred 20 untyped enteroviruses to the NPRL for further identification. Nine polioviruses were isolated from the referred isolates and eight tested as Sabin-like with the WHO approved methods of ITD. One further poliovirus type 1 was Sabin-like by PCR but did not react in the ELISA test. The virus was sequenced and confirmed as Sabin-like with a 99.7 per cent nucleotide homology to the parental Sabin strain in the VP1 gene, and no evidence of recombination was detected in the 3D region with 100 per cent homology to the parental Sabin strain. This result was confirmed by the Global Specialised Laboratory in Japan according to WHO protocol for polioviruses with discordant ITD results. This virus referred to the NPRL by the laboratory in South Australia was isolated from a three-month-old infant from the Northern Territory. The infant had no clinical evidence of AFP.

Sequencing of 10 of the other referred isolates from South Australia identified coxsackievirus B2, and echovirus 11, 18, 25 and 30. One further isolate from a nasopharyngeal aspirate was confirmed as a rhinovirus, and two isolates failed to passage, which may have been due to loss of virus titre.

A bowel specimen was referred from a four-month-old infant who had died of sudden infant death. PV3 was isolated from the bowel specimen and classified as Sabin-like by WHO approved methods of ITD.

A faecal specimen from a three-month-old infant from New South Wales with asthma but thought to have an enteroviral co-infection was referred to the NPRL. A PV2 was isolated from the specimen and subsequently tested as Sabin-like by WHO approved methods of ITD. No further investigation of this case was undertaken.

A faecal specimen from a six-month-old infant with monoclonal proliferation was referred for enteroviral studies. No enterovirus was isolated from this specimen.

A summary of enteroviruses tested at the NPRL between 1995 and 2005 is presented in Table 4.

Regional reference laboratory activities

As a WHO regional reference laboratory, the NPRL received a total of 252 specimens and isolates during January to December 2005, from national poliovirus laboratories and hospitals in the Western Pacific Region. This included six specimens from three AFP cases from the Pacific Islands, four specimens from two cases of AFP from Brunei Darussalam, 36 specimens and isolates from the Philippines and 61 specimens and isolates from Malaysia. A further 145 specimens and isolates from Papua New Guinea, were referred for retesting as part of an ongoing laboratory quality assurance program.

Laboratory accreditation

The NPRL retained its full accreditation status for 2005 as a national laboratory for Australia, the Pacific Island countries and Brunei Darussalam and as a regional reference laboratory for the Western Pacific Region.

Discussion

In 2005, there were 0.75 cases of AFP per 100,000 children aged less than 15 years, detected in Australia. This is less than the WHO standard target

Table 4. Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, 1995 to 2005

Year	Poliovirus		Non-polio enterovirus	Non-enterovirus detected or no virus detected	Total samples tested
	Sabin-like	Non-Sabin-like*			
1995	190		200	13	403
1996	224		198	9	431
1997	124		76	0	200
1998	52		15	4	71
1999	60	1	9	9	79
2000	45		44	47	136
2001	46	5	33	75	159
2002†	36		21	49	106
2003	9		15	47	71
2004	6		26	61	93
2005	18		10	39	67

* Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory.

† Two poliovirus isolates had discordant results by ITD. Sequencing confirmed the isolates as Sabin-like, with <1.0 per cent variation from the parental Sabin strain.

for AFP surveillance in a polio non-endemic country, of one case of AFP per 100,000 children. Since the establishment of AFP surveillance in Australia in 1995, the WHO rate has only been reached or exceeded in 2000, 2001 and 2004.[8,9]

Adequate faecal sampling in 2005 was achieved in only 19 per cent of eligible AFP cases—well below the WHO target of 80 per cent and the lowest level recorded since the introduction of AFP surveillance in Australia.[8,9] An increased awareness of the need to collect faecal specimens is required amongst notifying paediatricians and may increase Australia's rate of collection of adequate faecal specimens to enable Australia to meet the WHO requirement of at least 80 per cent of notified AFP cases with two adequate faecal specimens.[11] During the next year the PEC will also be implementing changes to facilitate the completion of the clinical questionnaire and the collection of faecal specimens.

With the removal of OPV, and the introduction of IPV into the Australian National Immunisation Program, laboratories will see a gradual decrease in the number of incidental poliovirus isolations to zero. Consequently the isolation of a poliovirus will represent a potentially imported virus as will untyped enteroviruses. Between 1 January and 31 December 2005, 40 uncharacterised polioviruses, and 185 untyped enteroviruses were reported to LabVISE.[12] Australian virology laboratories are therefore encouraged to forward any untyped enteroviruses and uncharacterised polioviruses to the NPRL for further characterisation. Thus from 2006, the isolation of any poliovirus by an Australian virology laboratory needs to be fully investigated, as demonstrated by two reports from the United States of America during 2005. In March 2005 an imported case of VAPP occurred in an unvaccinated adult on return from a student exchange program in Cost Rica.[13] In August 2005 a VDPV was isolated from an unvaccinated, immunocompromised seven-month-child presenting without AFP.[14] In both cases the detection of the poliovirus was due to thorough laboratory investigation and serotyping of isolated viruses.

Globally, the number of wild poliovirus confirmed cases reported, increased from 1,266 in 2004 to 1,962 in 2005 with the number of polio cases in non-endemic countries increasing from 256 in 2004 to 1,034 in 2005. A majority of cases were due to wild poliovirus importations originating mainly from India and Nigeria.[15] Sixteen countries reported importations of wild poliovirus while the number of polio endemic countries remained at six. Egypt and Niger interrupted transmission of indigenous poliovirus during 2005.[16] After 10 years of 'polio free' status, Indonesia detected 303 cases of poliomyelitis during 2005. Genetic analysis of the poliovirus isolated from the index case linked the outbreak to polioviruses circulating in Sudan, which originated from Nigeria.[17]

With an increase in the number of countries reporting importations, it is imperative Australia maintains a sensitive AFP surveillance system able to detect an imported case of poliomyelitis.[18] As we move closer

to global eradication, the classification of all AFP case notifications by the PEC will become crucial to maintaining Australia's polio free status.

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Communicable and vaccine-preventable conditions under surveillance by the APSU: 2005 update

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Background

The Australian Paediatric Surveillance Unit (APSU) conducts national active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions. Studies of communicable and vaccine-preventable diseases are supported by the Australian Government Department of Health and Ageing through its communicable diseases program. This report is a summary of surveillance results for communicable and/or vaccine preventable diseases studied through the APSU in 2005.

In 2005, eight communicable or vaccine preventable conditions were studied:

- acute flaccid paralysis (AFP);*
- congenital cytomegalovirus infection;
- congenital rubella infection;
- perinatal exposure to HIV and HIV infection;
- neonatal herpes simplex virus infection;
- hepatitis C virus infection;
- non-tuberculous *Mycobacterium* infection; and
- neonatal group B *Streptococcus* infection.†

Methods

APSU study protocols are developed with collaborating investigators and/or institutions and the objectives and chief investigators for each study are listed in Table 2. The methodology used to conduct surveillance is described in detail elsewhere.[1,2]

The APSU aims to provide epidemiological information that is representative of the Australian population and maximal case ascertainment is a high priority. Despite a representative mailing list (93% of all paediatricians in active clinical practice in Australia participate in monthly surveillance) and high monthly response rates, complete case ascertainment is unlikely. This is particularly relevant in remote communities where children have limited access to paediatricians. However, for most conditions studied by the APSU no other national data are available to estimate completeness of ascertainment. APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximize cases identified. Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate for these conditions in the relevant Australian populations.

Results

In 2005, 1,148 clinicians participated in the monthly surveillance of 14 conditions, (including the 8 listed above), with an overall monthly response rate of 93 per cent. Questionnaire return rate is greater than 80 per cent for most studies. Table 1 shows the number of confirmed cases reported in 2005 and for the whole study period and the reported rate per 100,000 population.[3]

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policy makers and the community. The APSU is often the only source of national data that includes clinical and/or laboratory details and data on both in-patients and out-patients. The key findings for studies undertaken in 2005 are summarised in Table 2.

Further information on the above studies may be obtained by contacting the APSU: website www.apsu.org.au Telephone 02 9845 3005; email: apsu@chw.edu.au, or the Principal Investigator for each study.

* Although the aim of this surveillance is to identify AFP due to poliomyelitis or associated with polio vaccination, there are many non-infectious causes of AFP.

† The study of neonatal group B *Streptococcus* infection commenced in July 2005.

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Table 1. Confirmed cases identified for 2005 and for the total study period

Condition	Date study commenced	Questionnaire response for total study period (%)	Number of confirmed cases		Reported rate for total study period (per 10^5 per annum)
			2005	Total study period	
Acute flaccid paralysis	March 1995	90	30*	368*	0.86‡
Congenital cytomegalovirus	Jan 1999	66	9	57	3.25§
Congenital rubella (with defects)	May 1993	96	NIL	50	0.10‡
Perinatal exposure to HIV;	May 1993	90	13	266	8.08§
HIV infection			5	45	1.99§,‖
Neonatal herpes simplex virus infection	Jan 1997	95	9	78	3.45§
Hepatitis C virus infection	Jan 2003	80	7	31	0.26‡
Non-tuberculous mycobacteria	July 2004	80	24†	44†	0.47
Neonatal B group *Streptococcus* infection	July 2005	56	30	30	¶

* All reported cases that have been classified by the Polio Expert Committee were 'non-polio AFP' according to WHO criteria.

† Includes confirmed and probable cases.

‡ Based on population of children aged ≤-15 years as estimated by the Australian Bureau of Statistics.[3]

§ Based on number of births as estimated by the Australian Bureau of Statistics.[3]

‖ All HIV infections except for one (source of infection unknown) resulted from perinatally acquired HIV.

¶ The study of neonatal groups B *Streptococcus* infection commenced in July 2005. Due to the short surveillance period of only 6 months, a rate is not reported.

Table 2. Results summary

Condition and principal investigator	Objectives	Key findings
Acute flaccid paralysis (AFP) Prof Heath Kelly, Victorian Infectious Diseases Reference Laboratory	To determine the notification rate of AFP in children aged <15 years. To determine whether AFP is caused by poliovirus infection and if so, whether it is a wild, vaccine, or vaccine-derived strain of poliovirus. To determine other causes and the clinical picture of AFP in Australia.	Decreased notification rates resulted in failure to reach the WHO AFP surveillance target of 1 case per 10^5 aged <15 years per annum. The primary diagnoses for AFP remain Guillain-Barré syndrome and transverse myelitis.[4] There were 3 cases of AFP due to infant botulism in 2005. Only 19 per cent of cases had adequate faecal specimen collection—well below the 80 per cent WHO target. An outbreak of 303 cases of wild poliovirus was recorded in Indonesia. Sixteen countries reported importations of wild poliovirus. Continued surveillance is required to detect imported cases and keep Australia polio free.[5]
Congenital cytomegalovirus (cCMV) infection Prof William Rawlinson, Virology Division, Department of Microbiology, Prince of Wales Hospital, Sydney	The study aims to determine: • the incidence of congenital and suspected congenital CMV infection; • the presenting features and clinical spectrum of disease due to congenital CMV; • the genotypes of CMV which cause congenital disease; • current therapy for congenital CMV infection; and • the epidemiology of congenital CMV prior to trials of vaccines and antivirals.	cCMV continues to be the most common infectious cause of malformations in Australia. cCMV infection was not associated with maternal illness in approximately one third of cases, and should be considered regardless of maternal history. cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture; use of PCR for urinary screening for CMV may increase diagnostic yield.[6] Universal neonatal hearing screening programs may also help identify new cases.
Congenital rubella (with defects) A/Prof Cheryl Jones, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney	To document the incidence of congenital rubella infection. To determine the vaccination status of mothers of infected infants. To monitor the effectiveness of the current vaccination program.	There were no cases of congenital rubella reported in 2005. As the risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs, such women should have serological testing for rubella after arrival in Australia, and vaccination when appropriate. Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk of congenital rubella to the fetus.
Perinatal exposure to HIV and HIV infection Ann McDonald, National Centre in HIV Epidemiology and Clinical Research	To identify new cases of perinatal exposure to HIV, paediatric HIV infection, and AIDS. To describe the pattern of perinatal exposure to HIV in Australia. To monitor the perinatal HIV infection transmission rate and use of interventions for reducing the risk of mother-to-child transmission. To describe the natural history of paediatric HIV infection.	In 2005, 13 cases of perinatal exposure were reported. The main source of infection for the mother was through heterosexual contact with a high risk partner.[7] Six reported cases of HIV infection in children were newly diagnosed in 2005, including 5 cases of perinatally-acquired HIV infection and 1 case of HIV infection acquired in a high HIV prevalent country in sub-Saharan Africa. The 5 cases of perinatal HIV infection were all born in Australia. These cases were reported through national surveillance for newly diagnosed HIV infection. Although the mother's HIV infection was diagnosed prenatally in 2 cases, interventions such as elective caesarean, avoidance of breast feeding and anti-viral therapies were not used. Antenatal diagnosis of the mother's HIV infection and use of interventions is required to minimise the risk of mother-to-child HIV transmission.

Table 2. Results summary, *continued*

Condition and principal investigator	Objectives	Key findings
Neonatal herpes simplex virus infection (HSV) A/Prof Cheryl Jones, Herpes Virus Research Unit, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney	To determine the incidence of neonatal HSV infection in Australia, its mortality and morbidity. To determine its mode of presentation e.g. localised, disseminated or complicated by encephalitis or pneumonitis and mode of transmission. To determine whether there is delay between presentation, diagnosis and initiation of treatment.	Over half of neonatal HSV infections in Australia are caused by HSV type 1, in contrast to the USA where HSV type 2 predominates. Typical herpetic lesions of the skin, eye or mouth were not evident in half of infants identified with neonatal HSV infection, which makes early diagnosis difficult. Disseminated HSV infection in the newborn may be associated with the early onset of pneumonitis in infants (in whom the chest X-ray may be normal). This is highly lethal unless antiretroviral therapy is initiated.
Hepatitis C virus infection (HCV) A/Prof Cheryl Jones, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney	To determine the reported incidence of newly diagnosed HCV infection in Australian children. To describe the clinical presentation, investigation and management of newly diagnosed HCV infection in Australian children. To document the presence of known risk factors for HCV infection in an Australian paediatric population. To determine the prevalence of co-infection with hepatitis B virus (HBV) and/or human immunodeficiency virus in Australian children with newly diagnosed HCV infection.	Perinatal transmission is the main source of HCV infection in Australian children. In the APSU study infants at risk were born to mothers who used IV drugs, had invasive procedures overseas or had tattoos.[8] Most HCV-infected children are clinically asymptomatic with mildly elevated liver function test at diagnosis. However, HCV induced chronic liver disease and liver failure have been reported among children. Given that 1–2 per cent of Australian women of child-bearing age are infected with HCV, the reported rates of infection are lower than predicted. This may be due to the lack of a consistent approach to identifying children with HCV infection.[9]
Non-tuberculous *Mycobacterium* infection (NTMI) Dr Pamela Palasanthiran, Paediatric Infectious Diseases Specialist, Department of Immunology and Infectious Diseases, Sydney Children's Hospital Randwick, NSW	To estimate the incidence of newly diagnosed NTM infection in children seen by child health specialists in Australia. To describe the epidemiology and spectrum of disease and document known risk factors. To describe diagnostic investigations used in Australia; frequency of use of skin testing and the clinical utility of the test, including differential skin testing. To describe the management of NTM in Australia and the response to treatment.	This infection most often presents as lymphadenitis predominantly in immunocompetent children. *Mycobacterium avium intracellulare* and *Mycobacterium fortuitum* are the most common organisms isolated in Australian children. Surgery is the most commonly offered therapy and in NTMI lymphadenitis complete excision is associated with a lower risk of relapse. There is marked heterogeneity in the antimicrobials and course prescribed. Despite therapy, relapse occurs in about 20 per cent of cases.[10]
Neonatal and infant *Streptococcus agalactiae* (group B streptococcus – GBS) sepsis Prof Lyn Gilbert, Centre for Infectious Diseases and Microbiology, Institute for Clinical Pathology and Medical research, Westmead Hospital, Westmead NSW	To determine: • the current incidence of early and late onset neonatal GBS infection; • the incidence of maternal and infant risk factors; • the proportion of early onset GBS infection in infants of women who have been given intrapartum antibiotic prophylaxis; • short-term mortality and morbidity of early and late onset GBS infection; and • the distribution of GBS genotypes between isolates.	Preliminary results only, as the surveillance period is only 6 months. Over half of the reported cases have been early onset at less than 8 days of age. The number of notifications received so far is consistent with other available data.

A comparison of data sources for the surveillance of seasonal and pandemic influenza in Victoria

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Abstract

Understanding the characteristics of available influenza or influenza-like illness (ILI) surveillance systems is important for seasonal influenza surveillance and pandemic preparedness. We compared five influenza or ILI data sources in Victoria: notifications of laboratory-confirmed influenza to the Victorian Department of Human Services; hospital emergency presentations and hospital admissions; sentinel general practitioner surveillance; and medical locum service surveillance. Seasonal trends for influenza and ILI activity were similar for all data sources. Community ILI surveillance, operating as sentinel GP, locum service or hospital emergency department surveillance, in conjunction with notification of laboratory-confirmed influenza, would provide adequate inter-pandemic surveillance for influenza in Victoria and, by extension, in any Australian jurisdiction. Other surveillance systems would be needed for early pandemic case or cluster detection, while pandemic monitoring would be better achieved by a more automated system. *Commun Dis Intell* 2006;30:345–349.

Keywords: disease surveillance, influenza, pandemic

Introduction

Each year, influenza is responsible for significant mortality and morbidity in Australia.[1] Community surveillance monitors seasonal activity due to influenza or influenza-like illness (ILI) and may facilitate influenza pandemic preparedness, although different surveillance systems may be needed in inter-pandemic or pandemic periods.

The Victorian general practitioner (GP) sentinel surveillance scheme is an established surveillance scheme for monitoring ILI.[2,3] Thresholds related to seasonal ILI activity allow a quick assessment of the level of circulating influenza and an indication of when community ILI activity may coincide with increases in hospital presentations.[4,5] However, coordination is resource intensive for both participating sentinel practitioners and the surveillance team.[2] During 2003 and 2004 a pilot study using a medical out of hours locum service, the Melbourne Metropolitan Locum Service (MMLS), concluded that trends of ILI seasonal activity from GP sentinel surveillance and from the locum service were comparable.[6] Other data sources for influenza and/or ILI currently available in Victoria include hospital emergency department presentations, in-patient discharges and laboratory-confirmed notifications. Automated syndromic surveillance is being developed.

This study compares the utility of all available influenza or ILI data sources to support surveillance during epidemic and pandemic periods in Victoria.

Methods

Data sources

Five influenza or ILI data sources were compared: GP sentinel surveillance, the MMLS, the Victorian Emergency Minimum Dataset (VEMD), the Victorian Admitted Episodes Dataset (VAED) and notifications of laboratory-confirmed influenza to the Department of Human Services, Victoria (DHS).

Laboratory supported general practitioner sentinel surveillance, operational in Victoria during the influenza season (May to September) since 1998, records the number of patients fulfilling the ILI case definition of cough, fever and fatigue, and the total

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number of patients seen each week. Respiratory specimens taken from a proportion of cases permit diagnosis of laboratory-confirmed influenza or other respiratory viruses.[2,7]

MMLS was established in 1980 as a deputising GP service, with locum doctors attending patients in their homes within an approximately 35-kilometre radius of the Melbourne metropolitan area. Demographic and clinical information from patients seen by doctors from the MMLS are routinely entered into a database within 24 hours of a consultation. A final diagnosis free text including either the term 'flu' or 'influenza' is extracted from this database as an alternative form of ILI surveillance.[6]

The VAED, maintained by the Victorian DHS, collates hospital discharge data compiled by individual private and public hospitals in Victoria.[8] This dataset contains demographic and clinical information on each episode of patient care, with the clinical information coded in the format of the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification (ICD-10-AM).[9,10] Similarly the VEMD collates information from presentations to Victorian hospital emergency departments. Persons admitted to hospital via the emergency department will be recorded in both the VEMD and the VAED.[11]

DHS collates notifications of diagnoses of laboratory-confirmed influenza, as required under the *Health (Infectious Diseases) Regulations 2001.*[12] These data include laboratory-confirmed diagnoses recorded from GP sentinel surveillance and the VAED as well as diagnoses confirmed by the 11 Victorian laboratories that conduct influenza confirmatory testing.

Data extraction and comparisons

GP sentinel surveillance, MMLS and notification data were extracted for the period January 2001 to December 2005 by either week of consultation or date of notification as applicable. VAED and VEMD data were available from January 2001 to July 2004 and were extracted by date of admission or date of emergency department presentation respectively. Data extraction from VAED and VEMD were restricted to ICD-10-AM codes for laboratory-confirmed influenza (J10) and influenza-like illness (J11).

Seasonal influenza activity was compared by number and age group of cases for each data source from January 2001 to July 2004. Comparisons were extended to December 2005 for GP sentinel surveillance, MMLS and laboratory-confirmed notification data. Age groups analysed were less than 15 years (school age), 15–44 years (young adult), 45–64 years, (adult) and 65 years and older (older adult).

A summary comparison of the relative strengths and weaknesses of each of the five surveillance systems was made, based on the criteria of:

- ease of access of data (to the surveillance coordinator at VIDRL);
- timeliness of data;
- potential for year-round surveillance; and
- facility for laboratory testing of respiratory specimens from patients with ILI.

Results

Seasonal activity

A clear seasonal trend for ILI or laboratory-confirmed influenza over the winter months was apparent from each data source (Figure 1). The marked seasonal peak of ILI activity during August 2001 was evident from VEMD, VAED, MMLS and GP sentinel surveillance. Notification data indicated this seasonal peak a month later. The seasonal peak in 2002 was detected by GP sentinel surveillance and VEMD in June and a month later by the other data sources. For 2003, the seasonal peak was observed in August by all data sources. Only GP sentinel surveillance and VEMD indicated any increase in activity during the low influenza season of 2004 whereas in 2005, the three available data sources all detected the seasonal peak in August.

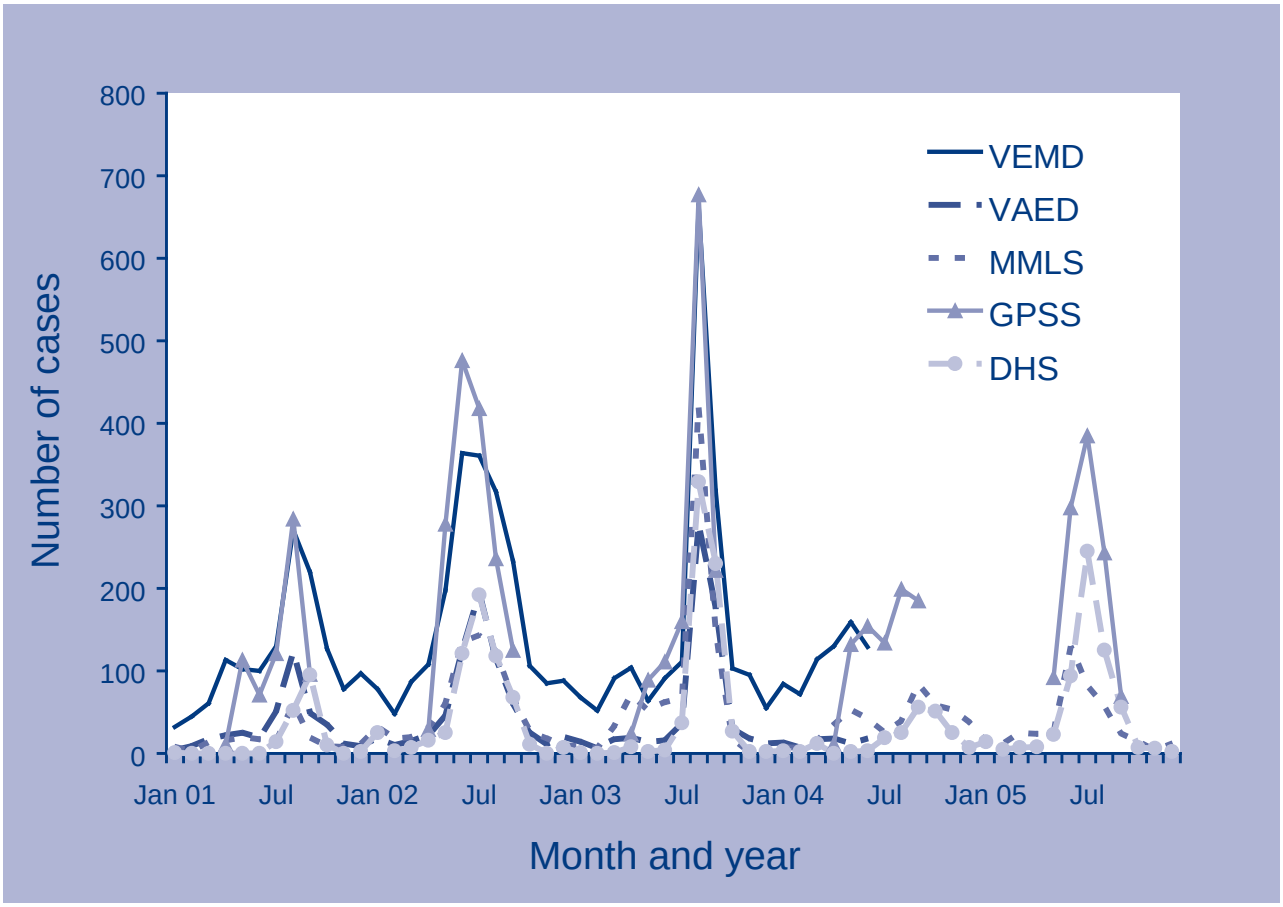
Age

Comparisons between the five data sources classified according to age group are presented in Figure 2. Patients in the 15–44 year age group comprised the highest proportion of notifications in GP sentinel surveillance and the VEMD. Emergency departments notified a higher proportion of children (≤14 years) than other data sources. Children also formed a relatively high proportion of laboratory-confirmed notifications. The elderly comprised a higher proportion of locum service notifications or hospital admissions.

Strengths and weaknesses

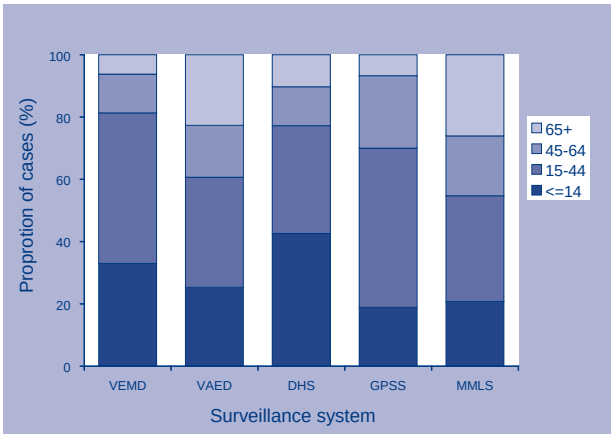
The strengths and weaknesses of the five surveillance systems are presented in the Table. MMLS surveillance was timely, easily accessed and available all year but could not confirm influenza by laboratory testing. Laboratory confirmed data, while available from each of the other data sources, were only available in a timely manner from GP sentinel surveillance and DHS notifications. GP sentinel surveillance was relatively labour intensive. As currently

Figure 1. Number of influenza-like illness or confirmed influenza cases, Victoria, 2001 to 2005, by data source



DHS	Department of Human Services (notifications)	GPSS	General Practitioner Sentinel Surveillance
MMLS	Melbourne Medical Locum Service	VAED	Victorian Admitted Episodes Data
VEMD	Victorian Emergency Department Data		

Figure 2. Proportion of ILI or influenza cases, Victoria, 2001 to 2004, by age group and data source



DHS Department of Human Services (notifications)

GPSS	General Practitioner Sentinel Surveillance
MMLS	Melbourne Medical Locum Service
VAED	Victorian Admitted Episodes Data
VEMD	Victorian Emergency Department Data

available on a routine basis, the delay in availability of hospital data (VAED and VEMD) renders these systems unsuitable for timely surveillance.

Discussion

Data from Victorian hospitals, sentinel GP surveillance, a medical locum service and influenza notifications to DHS are all useful indicators of influenza activity. Despite similar overall trends in activity, community-based ILI data sources demonstrated increased activity prior to notifications of laboratory-confirmed influenza in three of the five influenza seasons studied, and at the same time for the other two seasons. This is likely to reflect the time taken to process a specimen, confirm the presence of influenza and forward the notification to DHS.

While this retrospective analysis permits observation of similarities in the data sources, not all data sources are available in a time period that would support rapid decision-making. MMLS and DHS notification data are available daily. GP sentinel

Table. Comparison influenza and influenza like illness data source features, Victoria

System	Laboratory-confirmed influenza	Influenza-like illness	Data available by period	Ease of data access by surveillance coordinator at VIDRL	Potential for year round surveillance
MMLS	No	Yes	Daily	Can be downloaded from MMLS website at any time	Available all year round
GPSS	Partial	Yes	Weekly	Surveillance tally sheets faxed to VIDRL each week	Available during influenza season but could be expanded to non-influenza season
DHS	Yes	No	Daily	Available on DHS website and updated daily	Available all year round
VAED	Partial	Yes	Monthly*	Accessible for DHS, otherwise available only with >12 month lead-time	Available all year round but lacking timeliness
VEMD	Partial	Yes	Twice monthly*	Accessible for DHS, otherwise available only with >12 month lead-time	Available all year round but lacking timeliness

* Data availability for approved surveillance purposes only.
DHS Department of Human Services (laboratory-confirmed notifications)
GPSS General Practitioner Sentinel Surveillance
MMLS Melbourne Medical Locum Service
VAED Victorian Admitted Episodes Data
VEMD Victorian Emergency Department Data
VIDRL Victorian Infectious Diseases Reference Laboratory

surveillance data are available weekly. Hospital data, either from admissions or emergency presentations, although collected at individual institutions in real time, have an approximate 18-month delay to collation and availability for dissemination to agencies other than DHS. However, monthly VAED and fortnightly VEMD preliminary data can be made available for approved surveillance purposes. New South Wales has developed a system for the timely use of hospital data for ILI surveillance.[13]

Although MMLS data are the most accessible, they do not provide the opportunity for laboratory sampling. This can lead to reduced specificity, as several other respiratory viruses may present as an influenza-like illness in the absence of laboratory confirmation.[7,14,15]

There are other potential influenza surveillance data sources not considered as part of this study. These include mortality data and workplace absenteeism. Several studies have highlighted the difficulty of interpreting mortality data for influenza activity, as only the primary cause of death may be recorded without the attributing complication by influenza.[16] Likewise workplace absenteeism surveillance is difficult to interpret in light of the non-specific nature of absenteeism.[17,18]

Community ILI surveillance, operating as sentinel GP,[2] locum service[6] or hospital emergency department[13] surveillance, in conjunction with notification of laboratory-confirmed influenza,[12] would provide adequate inter-pandemic surveillance for influenza in Victoria and, by extension, in any Australian jurisdiction. Using more than one surveillance system improves the age range of patients captured by surveillance and allows validation of surveillance findings.

Community surveillance is, however, unlikely to detect the first case or cluster of cases in a pandemic, given the very low proportion (as few as 1%–2%) of all consultations that need to be monitored to describe seasonal influenza activity. This small proportion is unlikely to be sufficiently sensitive to detect an early case or cluster of a new viral sub-type.[19] Hospital-based surveillance is likely to capture more severe illness but will only be useful in detecting early cases or a cluster if it is timely.[13] Early case detection may rely on targeted border surveillance or the investigation of unusual disease clusters.[20] Other options will need to be considered. Pandemic monitoring will best be achieved by automated surveillance systems, such as those provided by the locum service in Victoria and hospital emergency department in New South Wales, that will be able to operate when there is the potential for high workforce absenteeism. Strengthening these surveillance systems in the inter-pandemic period should assist pandemic preparedness.

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Circulation and antigenic drift in human influenza B viruses in SE Asia and Oceania since 2000

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Abstract

During annual influenza epidemics, influenza B viruses frequently co-circulate with influenza A viruses and in some years, such as 2005, large outbreaks have occurred while in other years, the virus virtually disappears. Since 1987 there have been two lineages of influenza B viruses co-circulating in various countries and causing disease in humans. The proportions of these two lineages vary from year to year and country to country. For example, in 2005, the B/Victoria/2/87 lineage was predominant in New Zealand while in Australia the B/Yamagata/16/88 lineage was more common. Antigenic and genetic analysis has revealed gradual movement in the both lineages. Careful monitoring of the two virus lineages is important, as they are antigenically distinct. This is an important consideration for influenza vaccine formulation decisions, as only one influenza B component is traditionally included in the annual trivalent influenza vaccine. *Commun Dis Intell* 2006;30:350–357.

Keywords: Influenza B, serology, vaccines, vaccination, phylogenetic, evolution

Introduction

Influenza type B makes up an important component of the overall disease burden of influenza in humans. The proportion of influenza type A and influenza type B viruses circulating varies each year and in each country, as does the lineage of influenza B strains. In Australia in 2005, 26.6 per cent of the laboratory confirmed influenza cases that were typed were influenza type B,[1] while in New Zealand 87.2 per cent of influenza viruses typed were type B[2] and influenza B infection was associated with three deaths in adolescents.[3] During the early 1980s, a new lineage of B influenza emerged in humans that was antigenically and genetically distinct from the existing lineage of influenza B. Since then this lineage and the existing influenza B lineage have co-circulated and caused seasonal outbreaks in Australia and the Asia-Pacific region. The two lineages are represented by the reference strains B/Victoria/2/87 and B/Yamagata/16/88. These two lineages are antigenically quite distinct as antisera raised in ferrets to one lineage have no cross-reactive neutralising antibody against the second lineage.[4] They also form distinctly divergent genetic groups based on their haemagglutinin genes where there are some 27 amino acid differences.[5]

During the 1980s B/Victoria-like viruses were the predominant B lineage throughout the region, while from 1991 to 2000 the B/Yamagata lineage predominated in many countries with the B/Victoria lineage confined mainly to East Asia.[6] From 2000 onward, the B/Victoria lineage was again seen in increasing proportions outside Asia and was the predominant B lineage in the region in 2002 and in many countries in 2005. Each year the Australian influenza vaccine formulation is updated to incorporate new variants based on strains currently circulating or anticipated to circulate in the region. Currently only one type B strain, representing one of the two lineages, can be incorporated into the vaccine. As the two lineages have no cross reactivity, in years where both strains are circulating, the decision as to which lineage is selected can be difficult to determine. In this article we describe the distribution of the B/Victoria and B/Yamagata lineages in Australia and the Asia-Pacific region from 2000 to 2005 and compare the antigenic and genetic drift of these two lineages over this period.

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Methods

Viruses and antigenic analysis

Influenza B viruses were received from World Health Organization (WHO) national influenza centres; WHO influenza collaborating centres; Environmental Science and Research, Wellington, New Zealand; and other regional laboratories and hospitals from Australia, New Zealand, and the Asia-Pacific region. Viruses were received as isolates passaged in cell culture or as original clinical samples in which influenza B antigen had been detected by immunofluorescence or were positive for influenza B by (RT-PCR). Once received at the centre, the isolates were cultured in MDCK cells and monitored for growth by cytopathic effects and the presence of haemagglutination activity using turkey red blood cells (RBCs) as previously described.[7] Positive samples were typed using the haemagglutination inhibition assay (HAI) against a panel of known standard reference viruses and their homologous ferret antiserum.[7] Ferret antisera were pre-treated with Receptor destroying enzyme (RDE) (Denka Seikan, Japan), to remove non-specific inhibitors prior to use.

Human serology

CSL Limited provided sera from vaccinated adults (aged 18–60 years) and the elderly (61–85 years) undergoing vaccination field trials in 2004 and 2005. The vaccine used in these trials (Fluvax™, CSL Limited, Australia) contained influenza strains representing the currently circulating strain as recommended by the Australian Influenza Vaccine Committee (AIVC) and the Therapeutic Goods Administration (TGA). Influenza A(H3N2), A(H1N1) and type B, at a concentration of 15 ug/ml haemagglutinin were included in the vaccine. The vaccines given in 2004 and 2005 differed in their type B component, with the 2004 vaccine containing B/Brisbane/32/2002 (B/Victoria lineage), and the 2005 vaccine containing B/Jiangsu/10/2003 (B/Yamagata lineage). Blood samples were taken prior to vaccination and four weeks later. Pre- and post-vaccination sera were RDE treated and antibody levels tested by haemagglutination inhibition assay using turkey RBCs as the indicator cells against the vaccine strains and selected strains from the current 2004 and 2005 influenza seasons. For the 2004 samples, sera were assayed against egg grown B/Brisbane/32/2002 while for the 2005 samples, sera were assayed against egg grown B/Jiangsu/10/2003, the strains contained in the respective vaccines. The panels of sera were pre-selected from the subjects who showed a significant rise in post-vaccination titre compared to the pre-vaccination titre. Geometric titres and the number of subjects with HAI titres ≥ 40 were determined for each group. Prior to use in HAIs, B viruses were 'split' using an ether treatment method as previously described.[8] Briefly, viruses were mixed with an equal volume of Diethyl Ether (Merck) and vigorously stirred without frothing for four hours by magnetic stirrer. After mixing, the two layers were allowed to separate and the lower layer containing the split virus was removed. Residual ether was removed from the virus layer by slowly bubbling through gaseous nitrogen.

Sequencing RNA extraction, RT-PCR and sequencing were performed as previously published.[9]

Sequences were assembled using the Lasergene Seqman package IV (DNAStar V5.3) and phylogenetic relationships determined with PHYLIP V 3.5.7,[10] using the neighbour-joining method on Australian National Genomic Information Service and dendrograms were drawn using Treeview.[11]

Results

Haemagglutination inhibition assays

Table 1 shows the HAI assay of B viruses from the region representative of B/Victoria and B/Yamagata lineages from 2004 to 2005. Ferret sera were raised against reference strains representing the B/Victoria lineage (B/Brisbane/32/2002) and B/Yamagata (B/Shanghai/361/2002) and tested by HAI against isolates received at the WHO Collaborating Centre. The B/Victoria and B/Yamagata lineages were serologically distinct, for example the ferret sera raised to B/Brisbane/32/2002, a B/Victoria lineage virus, gave good HAI titres to B/Victoria-like strains but none against viruses from the B/Yamagata lineage. The converse was also true for ferret sera raised to B/Shanghai/361/2002, a B/Yamagata lineage virus, which reacted with B/Yamagata-like viruses but showed no cross reactivity to strains of the B/Victoria lineage. Two viruses associated with the deaths in children/adolescents in New Zealand in 2005, B/Wellington/21/2005 and B/Waikato/28/2003 are also shown in Table 1 and both were of the B/Victoria lineage and reacted similarly to other B/Victoria-like viruses tested.

Circulation of influenza type B lineages in the Asia-Pacific region

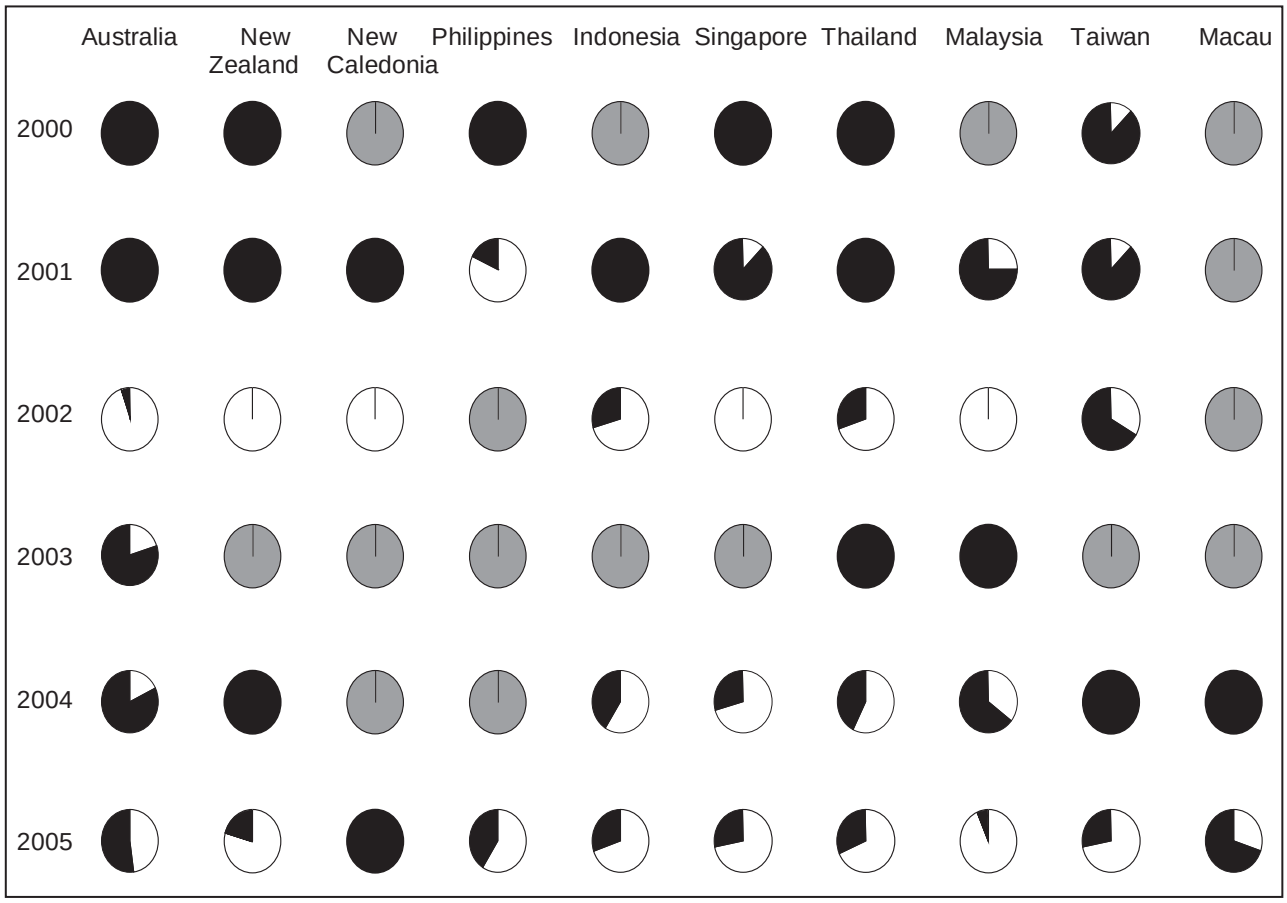
The distribution of the two type B lineages in the Asia-Pacific region were based on samples received at the centre from 2000 to 2005 and typed by HAI analysis as shown in Figure 1. Countries where less than five B viruses were detected in that year were not included.

In 2000, the B/Yamagata lineage viruses predominated throughout the Asia-Pacific region with five of the six countries studied having B/Yamagata-like viruses exclusively. In 2001 although the B/Yamagata lineage once again predominated, the prevalence

Table 1. Haemagglutination inhibition assay of B/Victoria and B/Yamagata-like viruses

	Ferret antiserum to		
	B/Brisbane/32 (B/Victoria lineage)	B/Shanghai/361 (B/Yamagata lineage)	Lineage
Reference antigens			
B/Brisbane/32/2002	320	<20	B/Victoria
B/Malaysia/2506/2004	320	<20	B/Victoria
B/Shanghai/361/2002	<20	640	B/Yamagata
B/Jiangsu/10/2003	<20	1,280	B/Yamagata
Test antigens			
B/Singapore/18/2004	160	<20	B/Victoria
B/Waikato/222/2005	80	<20	B/Victoria
B/Perth/112/2005	160	<20	B/Victoria
B/Malaysia/737/2005	160	<20	B/Victoria
B/Wellington/21/2005	160	<20	B/Victoria
B/Waikato/28/2005	160	<20	B/Victoria
B/Macau/131/2004	<20	640	B/Yamagata
B/Taiwan/142/2005	<20	640	B/Yamagata
B/Christchurch/103/2005	<20	640	B/Yamagata
B/Thailand/299/2005	<20	320	B/Yamagata
B/Victoria/501/2005	<20	640	B/Yamagata
B/Philippines/561/2005	<20	320	B/Yamagata

Figure 1. Circulation of influenza type B viruses in the Asia-Pacific region, 2000 to 2005



Black = the proportion of viruses typed as B/Yamagata-lineage.

White = the proportion of viruses typed as B/Victoria-lineage.

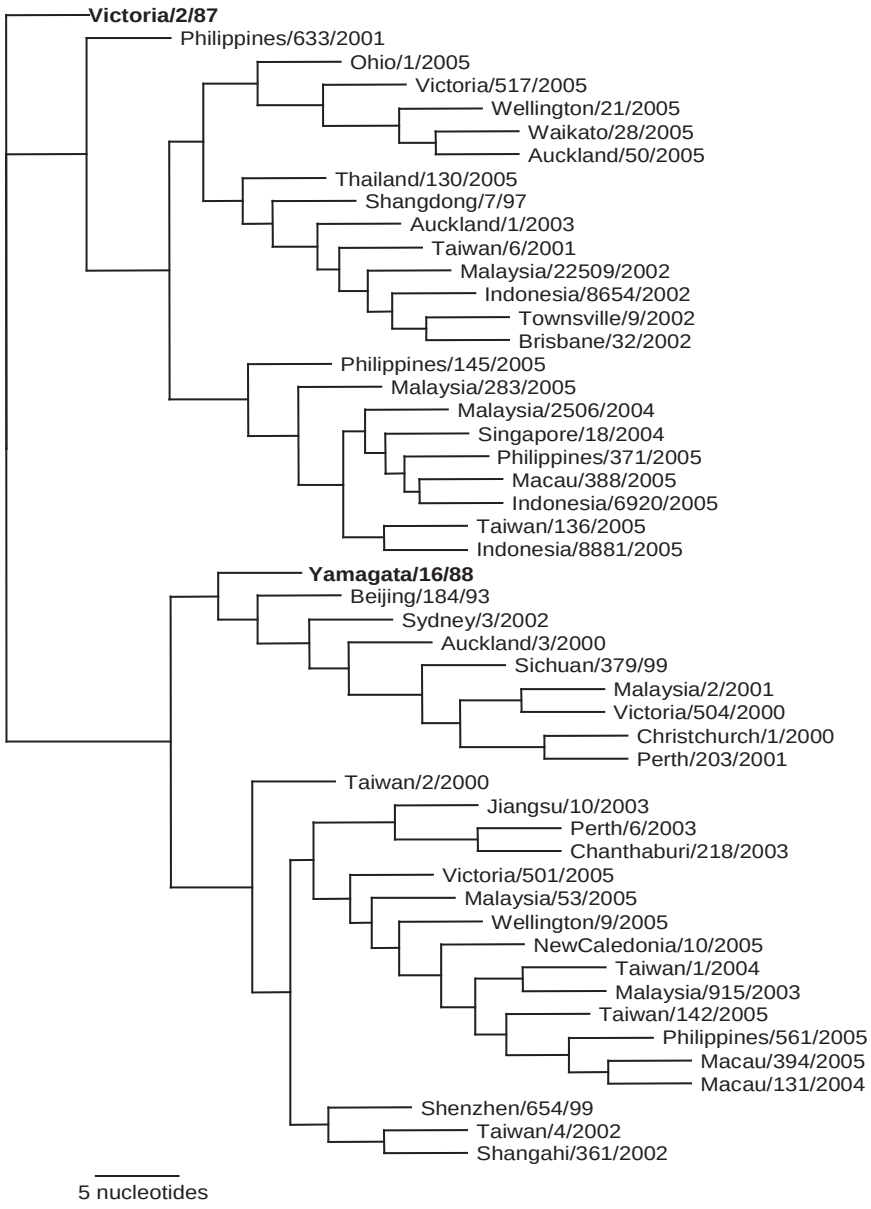
Grey circles indicate insufficient samples (<5) to determine proportions.

of the B/Victoria lineage in the region was beginning to increase. Four of nine countries had some B/Victoria- like virus activity and in the Philippines 82 per cent of isolates tested were from this lineage. In 2002 the B/Victoria lineage predominated almost exclusively in the region, yet Taiwan still had a greater proportion of B/Yamagata-like isolates (67%). The year 2003 was predominantly an A(H3) year and influenza B was virtually absent from the region with the exception of low levels of activity in Australia, Taiwan and Malaysia which mainly were of B/Yamagata lineage. In 2004 country to country variation was at its greatest with New Zealand, Taiwan and Macau almost exclusively B/Yamagata, while Indonesia, Singapore and Thailand had predominantly B/Victoria-like viruses, with mixed lineages in Australia and Malaysia. In 2005, type B activity was widespread throughout the region. Influenza type B activity in New Zealand was at epidemic levels and was dominated by the B/Victoria lineage (79%), while Australia had a predominantly A(H3) season,[1,2] and the B viruses were almost evenly divided between the two lineages. The B/Victoria lineages predominated in the Asia-Pacific region in 2005, with the exception of New Caledonia, which had viruses exclusively from the B/Yamagata lineage.

Phylogenetic analysis

The phylogenetic tree (Figure 2) shows the two divergent Influenza type B lineages based on nucleotide differences in the HA1 region of the haemagglutinin gene. The recent vaccine strains for the two lineages were B/Brisbane/32/2002 (B/Victoria) in 2003 and

Figure 2. Phylogenetic tree showing the two distinct lineages of B influenza viruses and representative isolates from the region during 2000 to 2005



Reference viruses shown in bold

2004 and B/Jiangsu/10/2003 (B/Yamagata) in 2005. Both lineages showed continued evolution with time, however the B/Victoria lineage showed little drift in the early years (2000–03) with viruses appearing similar to B/Shangdong/7/97 but more extensive drift has been seen recently (2004–05). Australian B/Victoria-like viruses isolated in 2005 phylogenetically grouped mainly around the B/Malaysia/2506/2004 clade (data not shown) or the B/Ohio/1/2005 clade (Figure 2). The B/Yamagata viruses in contrast, have shown slow but continued drift over the same period.

Cross protective efficacy in vaccinated subjects to the B/Victoria and B/Yamagata lineages

Tables 2a and 2b show the antibody response in vaccinated adult (18–64 years) and elderly (65–80 years) subjects against the vaccine strain received and representative viruses from strains representing the B/Victoria and B/Yamagata lineages. The 2004 vaccine contained a B/Victoria lineage virus (B/Brisbane/32/2002, Table 2a), while the 2005 vaccine contained a B/Yamagata lineage virus (B/Jiangsu/10/2003, Table 2b). Adults vaccinated against one B lineage type had reduced post-vaccination geometric mean titres and had a lower percentage of titres, ≥40 to viruses from the alternative lineage. This indicated that adults or the elderly vaccinated with influenza vaccine against one type B lineage would have reduced protection against infection with the alternative lineage if it were circulating. However there was a moderate rise in antibody geometric mean titre (GMT) levels against viruses representing the alternative lineage with both the 2004 and 2005 vaccines in both adults and the elderly sera examined, albeit some fivefold lower GMTs than viruses from the matched lineage.

Table 2a. Antibody response from vaccinees with the 2004 Australian influenza vaccine containing B/Brisbane/32/2002 (B/Victoria lineage)

Population	n	Antigen	GMT		% HAI titre ≥40	
			Pre	Post	Pre	Post
Adults	24	B/Brisbane/32/2002*	18.9	174.4	25	96
		B/Wulumuqi/26/04*	19.4	190.2	25	96
		B/Victoria/501/2005†	23.1	106.8	46	96
		B/Jiangsu/10/2003†	14.1	50.4	46	79
Elderly	24	B/Brisbane/32/2002*	14.6	195.8	17	96
		B/Wulumuqi/26/04*	14.1	179.5	8	96
		B/Victoria/501/2005†	15.4	51.9	21	67
		B/Jiangsu/10/2003†	10.0	41.2	54	88

* B/Victoria/2/87 lineage.

† B/Yamagata/16/88 lineage.

Table 2b. Antibody response from vaccinees with the 2005 Australian influenza vaccine containing B/Jiangsu/10/2003 (B/Yamagata lineage)

Population	n	Antigen	GMT		% HAI titre ≥40	
			Pre	Post	Pre	Post
Adults	24	B/Jiangsu/10/2003†	18.3	109.9	33	88
		B/Florida/7/2004†	35.6	155.4	50	96
		B/Malaysia/2506/2004*	23.1	36.7	42	58
		B/Brisbane/32/2002*	26.7	54.9	54	79
Elderly	24	B/Jiangsu/10/2003†	15.4	123.3	21	92
		B/Florida/7/2004†	20.0	119.8	33	83
		B/Brisbane/3/2005†	13.3	65.3	29	79
		B/Malaysia/2506/2004*	15.0	28.3	21	50
		B/Brisbane/32/2002*	18.3	37.7	29	54

* B/Victoria/2/87 lineage.

† B/Yamagata/16/88 lineage.

Australian influenza vaccine composition and circulating B viruses

Table 3 shows the comparison of the vaccine strains recommended by the Australian Influenza Vaccine Committee (AIVC) with the predominant circulating B-lineage. The AIVC is the committee in Australia, which advises the TGA on the appropriate strains which should be included into the Australian influenza vaccine each year. This decision is made in October each year some 9–10 months prior to the next influenza season. The vaccine component was well matched with the circulating strain in two of the three years in which there was clearly a predominant lineage (2000–2002). In the following three years mixed lineages were seen in Australia, and while the B/Yamagata lineage viruses were in the majority in these years the vaccine contained a B/Yamagata lineage virus in only one of these years (2005). The decision to include a B/Victoria-lineage virus in the 2003 vaccine was due to the predominance of B/Victoria-like viruses in Australia and elsewhere in 2002. The same lineage was selected for the 2004 vaccine as the B/Victoria-like viruses still predominated worldwide in 2002–03 and Australia only had a handful of B viruses in 2003 that were from both lineages.

Discussion

A significant amount of the impact of influenza is due to the influenza B viruses.[12] While influenza B infections are usually associated with a lower mortality than influenza A infections, occasional deaths can occur. Influenza B infections are often in children who are generally unvaccinated, as was the case in New Zealand in 2005 where two children and one adolescent died following influenza B infection.[3] Two of these cases developed *Staphylococcus aureus* pneumonia and septicemia and in the other case the subject was on aspirin for another condition and developed Reye's Syndrome.[3] Childhood deaths from influenza B infections are rare but do occur,[13] however, they are far more common following influenza A outbreaks as was evidenced in the 2003–04 influenza season in the United States of America where 153 deaths were reported in children under 18 years of age.[14] Influenza B outbreaks can also occur in schools,[15,16] on cruise ships[17] and in nursing homes,[18,19] causing significant morbidity. This makes the matching of the B vaccine strain to the circulating strain an important part of minimising the effects of the virus.

Influenza B viruses, unlike influenza A viruses, have multiple evolutionary lineages which can co-exist for considerable periods of time.[20] This has occurred since the early 1980s when a new lineage (B/Yamanashi/16/88-like) appeared to evolve from B/USSR/100/83-like viruses[4] and from then on has co-circulated with the existing virus lineage (B/Victoria/2/87-like).[4,5] During this time, the patterns of circulation have changed periodically and over the last six years both lineages have predominated in particular countries in particular years, until recently when both lineages have co-circulated in the same countries at the same time. Interestingly, sera from naive ferrets that are generated by infections with a single virus (and have not been exposed to other human influenza viruses), show little or no cross-reactivity between the two B lineages. In contrast, sera from vaccinated humans (adults and elderly) do show some cross-boosting when vaccinated with virus from one lineage against the other lineage *in vitro*, although this cross-boosting is at a much lower level than the boosting obtained with viruses from the same lineage. Presumably this is due to a combination of prior exposure or vaccine priming but may also be in part due to differences in the type of immune responses generated with the killed viral vaccines used in humans as opposed to the live virus given to ferrets.

Phylogenetically both lineages have shown modest antigenic drift over the last six years. In the last 2–3 years the B/Victoria lineage viruses have shown more drift than seen previously, resulting in a change of vaccine recommendation for 2006 to

Table 3. The annual vaccine recommendations by the Australian Influenza Vaccine Committee (AIVC) and the predominant B virus lineage that circulated in Australia during that year

Year	AIVC recommended B strain	B vaccine lineage	Circulating B lineage (Australia)
2000	B/Yamanashi/166/98	Yamagata†	Yamagata
2001	B/Sichuan/379/99	Yamagata	Yamagata
2002	B/Sichuan/379/99	Yamagata	Victoria
2003	B/Shangdong/7/97 or B/Brisbane/32/2002	Victoria*	Mixed
2004	B/Shangdong/7/97 or B/Brisbane/32/2002	Victoria	Mixed
2005	B/Jiangsu/10/2003	Yamagata	Mixed

* B/Victoria/2/87 lineage.

† B/Yamagata/16/88 lineage.

B/Malaysia/2506/2004 from B/Brisbane/32/2002 (or B/Shangdong/7/97), as recommended for the 2004 Australian influenza B vaccine component. In the last six years there have been three changes in the Yamagata lineage derived vaccines with the most recent change being made in the 2005 Australian vaccine where B/Jiangsu/10/2003 was used.

It is unknown why B/Victoria lineage viruses that were limited to East Asia in 2000 and for most of the previous decade, have re-emerged but a similar phenomenon was seen with the A(H1N1) strain A/Bayern/262/95. These strains circulated worldwide in 1995–1998, while the A/New Caledonia/20/99-like strains were limited to Asia during this period. Subsequently the A/Bayern-like viruses were completely replaced by the A/New Caledonia-like viruses, which are still circulating.[6] Interestingly, since 2001 the B/Victoria viruses have also undergone reassortment with B/Yamagata viruses and now practically all B viruses contain a B/Victoria-lineage haemagglutinin and a B/Yamagata-lineage neuraminidase.[6,9] This reassortment has occurred previously with B viruses[21] and may represent a further evolutionary strategy that influenza B viruses[22] have to evade the immune system and prolong co-circulation of dual lineages.

The continued co-circulation of two influenza B lineages makes selection of the best matched influenza B virus for the annual influenza vaccine difficult, especially as this decision has to be made some 9–10 months before the peak of the upcoming influenza season. This lag is required to allow manufacturers to produce sufficient vaccine and for regulators to produce reference reagents and to licence the vaccines. In recent years only the Japanese manufacturers have included two B viruses in their influenza vaccine making it a quadrivalent vaccine (with an A(H1N1) and an A(H3N2) virus). WHO, the manufacturer and regulators in other countries have not embraced this approach due to its impact on production capacity, cost, and the lack of time to produce reagents. Indeed, the Japanese manufacturers now also only produce a trivalent influenza vaccine with a single B component. It is worth noting that in many sera from post-vaccinated adults and the elderly, modest but useful levels of antibody were produced against viruses from the alternative B lineage not present in the vaccine. This partial cross-reactivity reduces the need for an additional B virus lineage to be added to the vaccine currently. However, if the two lineages continue to drift apart (and co-circulate), ultimately the only way of ensuring optimal vaccine coverage against viruses of both lineages may be to include both lineages in the influenza vaccine. Alternatively, other types of vaccines such as the live attenuated influenza vaccine (Flumist®, MedImmune Vaccines Inc., USA) may offer some advantage in terms of breadth of protection against co-circulating lineages over the conventional killed influenza vaccines.[23]

Acknowledgments

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Nosocomial and community transmission of measles virus genotype D8 imported by a returning traveller from Nepal

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Abstract

Measles is uncommon in Australia due to effective national vaccination strategies. In mid-2003, a cluster of nine cases of measles occurred in western Sydney. The index case was a 29-year-old traveller recently returned from Nepal. The case presented to hospital and transmitted the disease to two others in the Emergency Department. Further cases resulted from both community and nosocomial transmission. The median age of cases was 24 years, with three cases in children aged under four years. Only one person had a documented history of measles vaccination, a child who had received one dose of vaccine overseas. One case was a 2-month-old infant whose mother was immune and two cases were hospital staff members. Molecular analysis of measles virus isolates from four cases revealed the same D8 genotype, a strain previously identified in Nepal. Staff vaccination strategies implemented as a result of the outbreak were poorly patronised despite nosocomial transmission. As diseases such as measles become rare it is important to thoroughly investigate any outbreaks, and to maintain a high index of suspicion of measles, particularly in travellers presenting with a rash having returned from measles-endemic areas. Genetic analysis is important in tracing the origins of an outbreak, and to confirm relatedness between cases. The highly infectious nature of measles virus also underscores the need for appropriate infection control in minimising risk of nosocomial transmission. Such policies are of increasing importance with the emergence of novel viruses or the threat of pandemic influenza. *Commun Dis Intell* 2006;30:358–365.

Keywords: measles, nosocomial, genotype D8, transmission

Introduction

Measles is a highly infectious and serious disease responsible for significant morbidity and mortality in undeveloped countries.[1] In Australia, locally acquired measles is now uncommon, largely due to effective measles vaccination initiatives. Outbreaks are most often due to overseas acquisition of the virus with subsequent infection of susceptible individuals in Australia[2,3] or spread through conscientious objectors to vaccination.[4]

The reported incidence of measles has declined since the introduction of surveillance in 1991, to a record low level of 0.07 cases per 100,000 population in 2005.[5] While possible cases presenting with a measles-like rash are sometimes reported to the local public health unit (PHU) by general practitioners and hospital physicians, the majority of reported cases do not fit the case criteria for measles as defined by the NSW Health Department.[6] Moreover, confirmatory diagnostic testing is not ordered for most patients who present with a rash, confounding accurate determination of the incidence of vaccine preventable diseases such as measles and rubella in Australia.

Following the National Measles Control Campaign in 1998, immunity to measles amongst children aged 6–11 years in Australia increased from 84 per cent to 94 per cent.[7] However, there remains a high-risk population of young adults in Australia, born between 1975 and 1981, who may have not contracted measles during childhood due to its declining incidence,

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but who also may not have been vaccinated due to lower vaccine coverage at the time.[8] This population of young adults is therefore at risk of measles, as demonstrated in four outbreaks in Victoria, occurring between 1999[9] and 2003.[3] Such outbreaks amongst a susceptible population reinforce the need for continued surveillance and follow-up of cases of measles, rapid testing, and the maintenance of a high index of suspicion for measles amongst travellers presenting with a rash. In three of the outbreaks, the index case acquired measles overseas and imported the disease into Australia. The index case for the remaining outbreak was not identified.[3]

Outbreaks of measles can be characterised by genetic analysis of virus isolates. Sequence analysis of the haemagglutinin (H) gene and of the hypervariable region of the nucleoprotein (N) gene can help in identifying geographic sources of the disease, particularly in countries with few locally acquired cases and where effective immunisation strategies are in place. Studies in Canada, the United States of America and Australia have shown that the measles virus genotypes found in the outbreaks described in the reports resulted from importation of the virus rather than local acquisition.[10,11,12] Genetic relatedness between cases and reference to known strains is an important part of an outbreak investigation, and can assist in informing eradication and control strategies.

This paper describes a cluster of nine cases of measles in western Sydney that presented in mid-2003. The genetic analysis of the measles virus obtained from specimens, the role of transmission through the hospital Emergency Department, and the importance of vaccination are discussed.

Methods

Case definition

The case definition for a confirmed case of measles was clinically defined measles-like illness with laboratory confirmation by one of the following: detectable measles virus-specific IgM or IgG seroconversion in serum, detection of measles virus antigen by immunofluorescence or measles virus RNA by polymerase chain reaction (PCR) from respiratory swabs or serum, or isolation of measles virus from blood, swabs or aspirates. Symptoms of clinically-defined illness included rash, cough, coryza and temperature over 38°C.[6]

Outbreak investigation and contact tracing

PHU officers investigating the outbreak followed response protocols outlined in the NSW Health Notifiable Diseases Manual.[6] Local general practitioners, infectious diseases physicians and emergency departments were alerted and provided with information about isolation and laboratory testing of suspected cases. PHU staff undertook contact tracing of patients following presentations to the Emergency Department (3 cases) and a general practice (2 presentations by one case). If the period of time from exposure to follow-up was short enough for vaccination or immunoglobulin prophylaxis to be effective, adults aged between 18 and 32 years were advised to receive measles, mumps and rubella (MMR) vaccination, and children aged under 12 months were offered immunoglobulin. These age groups were targeted as being susceptible to measles as the adults may have not contracted measles during childhood or not have been vaccinated, and the infants were too young to have received the first dose of MMR vaccine. Older children who had not received two doses of MMR vaccine were also advised to be vaccinated.

Hospital Infection Control staff contacted staff members at the hospital who were exposed to an infectious case. Those aged 32 years or younger were advised to receive MMR vaccination.

Laboratory diagnosis

Nasopharyngeal specimens, or nose and throat swabs, were collected from patients presenting with clinically-defined measles-like illness. For measles antigen detection, acetone-fixed smears of swabs were stained with measles-specific monoclonal antibodies (Chemicon International, Temecula, USA) and fluorescein isothiocyanate-conjugated anti-mouse immunoglobulin in an indirect immunofluorescence assay.

Vero cell monolayers in tube cultures were inoculated with 0.2 ml suspension of respiratory tract samples, incubated at 37°C and observed daily for cytopathic effects (CPE) characteristic of measles virus. Cultures showing CPE were confirmed by staining with measles-specific monoclonal antibodies described above.

A diagnostic measles PCR was performed using nucleoprotein (N) region primers in a nested format (outer sense 5'TACCCTCTGCTCTGGAGCTATGCC3', outer antisense 5'CTCGCACCTAGTCTAGAAG3' and inner sense 5'TATCACTGCCGAGGATGCAAG3', inner antisense 5'TGTCTGAGCCTTGTTCTTCCG3'). Total RNA was extracted from 200 µl serum using the Roche High Pure RNA kit (Roche Diagnostics, Germany). First round amplification was performed using Hot Start Amplitaq gold DNA taq polymerase and buffer (Applied Biosystems, Branchburg, New Jersey, USA) supplemented with 2.0 mM $MgCl_2$, 0.2 mM of each dNTP, 0.2 µM of both primers and cycling profiles of denaturation at 95°C (1 minute), annealing at 53°C (40 seconds), and extension at

72°C (1 minute) for 30 cycles. For the nested PCR, 1 µl of the outer product was amplified under similar conditions, except with an annealing temperature of 61°C. PCR amplicons of 379 nucleotides were visualised by electrophoresis on 1.5 per cent agarose gel, followed by sequencing for confirmation and comparison to other outbreak and reference measles sequences.[12,13]

Testing for measles-specific IgM and IgG serum antibodies was performed using the Enzygnost (Dade Behring, Germany) enzyme immunoassay according to the manufacturer's instructions.

Measles virus genotyping

For measles genotyping, total RNA was extracted using the Roche High Pure RNA kit (Roche Diagnostics, Germany) from either the swab samples, or from 200 µl of serum or Vero cells infected with measles virus harvested when CPE involved at least 30 per cent of the cell monolayer. RNA from serum and swabs was eluted in 50 µl and from infected Vero cells in 100 µl of elution buffer. A 456-nucleotide (nt) sequence coding for the carboxy-terminal of the nucleoprotein (N) gene and the full length (1,854 nt) of the haemagglutinin (H) gene were amplified in a single round PCR from four isolates. Amplicons were sequenced in both directions using sequencing primers described elsewhere.[14]

Nucleotide sequences of the N and H genes were aligned using the Clustal W (1.7) program and phylogenetic trees were generated by the Phylip program (Phylogeny Inference Package version 3.5) using the DNA distance matrix program (version 3.57) followed by neighbour-joining tree. Treeview (version 1.5) was used to draw the unrooted trees. Access to these programs was through www.angis.org.au, the website of the Australian National Genomic Information Service (ANGIS). The reference measles strains and Genbank Accession numbers used in the phylogenetic analyses are listed in the Table.[12,13]

Table. Measles genotypes used for the genetic characterisation of the outbreak isolates

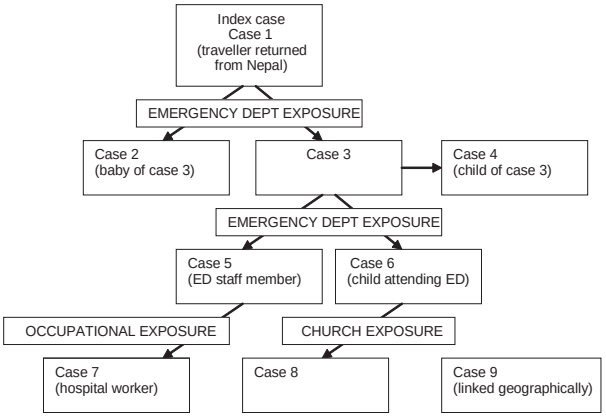
Reference strain	Genotype	Haemagglutinin gene, Genbank Accession no.	Nucleoprotein gene, Genbank Accession no.
Edmonston-wt.USA/54	A	U03669	U01987
Yaounde.CAE/12.83	B1	AF079552	U01998
Libreville.GAB/84	B2	AF079551	U01994
Ibadan.NIE/97/1	B3	AJ239133	AJ232203
New York.USA/94	B3	L46752	L46753
Tokyo.JPN/84/K	C1	AY047365	AY043459
Erlangen.DEU/90	C2	Z80808	X84872
Maryland.USA/77	C2	M91898	M89921
Bristol.UNK/74 (MVP)	D1	Z80805	D01005
Johannesburg.SOA/88/1	D2	AF085198	U64582
Illinois.USA/89/1	D3	M81895	U01977
Montreal.CAN/89	D4	AF079554	U01976
Palau.BLA/93	D5	L46757	L46758
Bangkok.THA/93/1	D5	AF009575	AF079555
New Jersey.USA/94/1	D6	L46749	L46750
Victoria.AUS/16.85	D7	AF247202	AF243450
Illinois.USA/50.99	D7	AY043461	AY037020
Manchester.UNK/30.94	D8	U29285	AF280803
Janakpur.NEP/2.99/1	D8	AJ250061	AJ250069
Victoria.AUS/12.99	D9	AY127853	AF481485
Kampala.UGA/3.01	D10	AY923214	AY923203
Goettingen.DEU/71	E	Z80797	X84879
Madrid.SPA/94 SSPE	F	Z80830	X84865
Berkeley.USA/83	G1	AF079553	U01974
Amsterdam.NET/49.97	G2	AF171231	AF171232
Gresik.INO/17.02	G3	AY184218	AY184217
Hunan.CHN/93/7	H1	AF045201	AF045212
Beijing.CHN/94/1	H2	AF045203	AF045217

Results

Outbreak details

Details of the outbreak are shown in Figure 1. The index case, Case 1, a 29-year-old male, presented to the Emergency Department of a large teaching hospital in June 2003 with a three-day history of fever, cough and feeling unwell. He presented with a morbilliform rash, fever, cough, coryza and temperature over 38°C. He was asked to return the next day whereupon his condition had worsened and he was admitted. The case had returned from Nepal via a stop-over in Bangkok seven days before his first presentation. A travel history was taken on admission and a number of conditions considered. The case claimed to have had measles previously and to have been vaccinated against measles in the past. He had not been vaccinated with MMR immediately prior to the trip to Nepal. Measles was considered as a possible diagnosis upon his first presentation and he was isolated. Upon his second presentation, however, no specific infection control procedures were implemented. The local PHU was not notified and a public holiday long weekend provided an additional delay before measles was confirmed.

Figure 1. Transmission of measles between cases



Upon confirmation of measles, Emergency Department contacts of Case 1 were notified of the risk of measles. It was too late for immunoglobulin prophylaxis. Two contacts from the one family (Cases 2 and 3, aged 2 months and 27 years respectively) developed measles with the rash appearing between 8 and 16 days post-contact. Case 3 was the father of Case 2. Subsequently, measles was transmitted to a 15-month-old sibling of Case 2 (Case 4) who was not vaccinated against measles. The 17-year-old mother, whose immune status was indicated by measles-specific IgG in a serum sample, did not contract measles.

Case 3 presented to the Emergency Department and, despite being promptly isolated, transmitted the infection to an Emergency Department staff member (Case 5, aged 30 years) and a 3½-year-old child (Case 6) who was being treated in the Emergency Department. Both developed a rash 11 days after contact. Another staff member subsequently developed measles (Case 7, aged 38 years), the likely source being Case 5.

The 3½-year-old child (Case 6) visited a church whilst infectious and transmitted measles to an unvaccinated adult (Case 8, aged 24 years) who developed a rash 14 days after contact. An additional case (Case 9, aged 21 years), living in the same geographical area as the other cases, was notified to the local PHU prompting investigation of possible exposures. If Case 9 was linked to any of the other cases, the timing of symptoms indicated that infection could only have been through contact with Case 8. However the nature of any such contact could not be determined. It is possible that other cases occurred in the area but were not diagnosed as measles or were not notified.

The median age of cases was 24 years (range 2 months to 38 years), with three cases in children aged under 4 years.

Clinical and laboratory details

All cases except the infant (Case 2) presented with rash, fever, and cough. The infant presented with rash, fever and coryza. Koplik spots were detected in four patients. Six patients also presented with conjunctivitis. Abnormal liver function tests were noted in all adult cases tested (n=4), an uncommon manifestation of measles.[15] All patients had at least one laboratory test confirming measles virus infection. All cases were confirmed cases according to NSW Health criteria.[6] Four of the six adult cases and one of the three child cases required hospitalisation for between two and five days.

The incubation period of the infant's illness (Case 2) was short, with the rash occurring eight days post-contact. The period between contact and rash for the 3½-year-old child (Case 6) was 11 days. The other child (Case 4) contracted measles from Case 3 or Case 2 and the exact date of transmission was unknown. The adults for whom precise details of contact were known developed a rash between 11 and 16 days post-exposure.

Vaccination status

Only one person (Case 6) had documented evidence of measles vaccination, having received a dose of vaccine overseas. The other two children in the outbreak were unvaccinated either because they were

too young (Case 2) or parental choice (Case 4). Of the six adult cases, three thought they had received one dose of vaccine (Cases 1, 3, 9), two thought they had measles in childhood (Cases 1, 8) and one thought that measles serology had been previously tested and that immunity was adequate (Case 5). No details of the vaccination or disease history were obtained for Case 7.

Outbreak investigation

A total of 496 people were identified as possible contacts at either the Emergency Department or general practitioner (GP) surgery. Of these, 184 contacts from the Emergency Department and 72 contacts from the GP surgery were in the 'at risk' age group of 18 to 32 years, or were infants aged under 12 months. Telephone contact was made with 61 per cent of the Emergency Department contacts and with 52 per cent of the GP surgery contacts. A letter was sent to the remainder. All contacts were provided with advice about the risk of measles and disease symptoms, and were recommended vaccination if necessary. There was difficulty in tracing all contacts presenting to both the Emergency Department and the general practice as some patients had not provided any or accurate contact details on presentation. PHU officers attempted to visit two families who could not be contacted by telephone, to advise parents about the need for immunoglobulin prophylaxis for exposed infants. One family was contacted in this way, while the other family had moved and could not be located.

PHU follow-up also involved providing information to contacts who had attended church with an infectious case. The church community of more than 500 members was provided with information in Arabic about the risk of measles, disease symptoms, and vaccination. A beauty salon and child-care centre were also contacted and provided with the same advice.

The hospital infection control team undertook contact tracing of 34 staff members who had possible exposure to measles and were in the risk age group of between 18 and 32 years. Seventeen of these staff members (50%) received MMR vaccination as a result. The remainder had either a past history of measles or measles serology indicating immunity. A staff health program recommending MMR vaccination was implemented at the hospital, targeting staff born after 1970. Information and education about measles and MMR vaccination were distributed by letter to susceptible staff, and to all staff through hospital networks including a staff newsletter and electronically. MMR vaccination was also offered at different locations throughout the hospital. Only 17 per cent of staff born after 1970 (134 out of 788 people identified from staff records) took the opportunity to receive MMR vaccination with 15 per cent of staff declining and 62 per cent not responding to the program. Only six per cent of those targeted indicated they had previously been vaccinated. A second staff health program, aimed at staff who did not respond to the first program and at new staff, identified 461 staff. Of these, three per cent had been previously vaccinated (14 people) and 14 per cent (63 people) received MMR. Ten per cent declined vaccination and 74 per cent did not respond to the program.

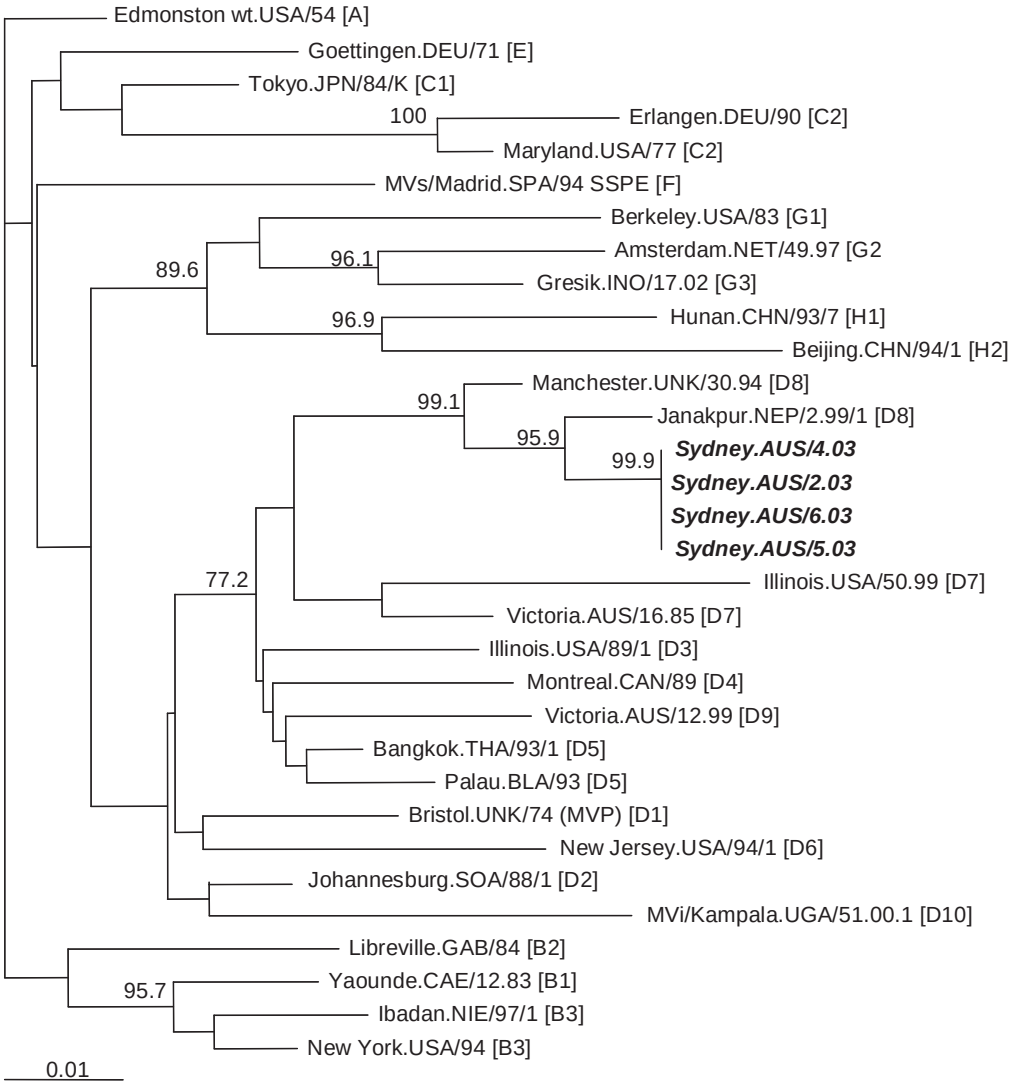
Genetic analysis of measles viruses

Measles virus was isolated from five patients (Cases 2, 3, 5, 7 and 8) including two cases from the same family. Sequencing of the N genes of four isolates (only one isolate from the family was sequenced) showed that they were identical, and were more closely related to a Nepalese measles genotype D8 (Janakpur NEP/2.99/1, Table) isolate[16] than to other sequences, (Figure 2).[12,13] Similar results were obtained with phylogenetic analysis of the H gene sequences (data not shown). The D8 genotype has occasionally been detected in Australia previously (the last time in early 2001), although the country of origin was not always available.[17] The D8 genotype was confirmed on RNA from three isolates sent to the Measles Reference Laboratory at VIDRL (Doris Chibo, personal communication). To confirm the specificity of the diagnostic PCR compared to measles virus isolation and antigen detection from respiratory tract specimens, a 355 nt sequence of the amplicon generated from serum of the index case showed 100 per cent similarity to the N gene sequence of one of the outbreak isolates, Sydney.AUS/4.03 (data not shown). The Genbank Accession numbers of the four sequences from this outbreak are DQ852617, DQ852618, DQ852619 and DQ852620.

Discussion

This paper describes a cluster of nine laboratory-confirmed cases of measles, eight of which were linked through contact. The index case had returned from Nepal three days prior to onset of fever and seven days prior to onset of rash. As the incubation time for measles is about 10 days, varying from 7 to 18 days to onset of fever and 14 days to onset of rash,[6] the most likely scenario is that the disease was acquired in Nepal. Phylogenetic analyses of the N and H regions of the measles viruses isolated from four cases indicated that sequences were most closely related to each other and the Janakpur. NEP/2.99/1 strain, a genotype D8 isolate of Nepalese origin,[16] further suggesting the acquisition of measles in Nepal and transmission to contacts in Australia. As outbreaks of measles become rarer in countries where vaccination programs have been effective, it is important to thoroughly investigate any incursions of disease. Genetic analysis of measles

Figure 2. Phylogenetic relationship based on the carboxy terminal end of the N gene (456nt) of the four Sydney isolates to recent isolates, Janakpur.NEP/2.99/1,[16] MVi/Kampala.UGA/51.00.1,[13] and other reference strains quoted by the WHO.[12]



Significant bootstrap values are indicated at the nodes (1,000 replicates, in %) of the unrooted tree. The isolates from this cluster are shown in bold. The scale indicates 1 per cent nt difference and [ ] denotes the genotypes of the reference strains.

isolates can aid in identifying the geographic and personal source of the outbreak, confirm relatedness of cases within outbreaks, and identify routes of transmission.[13,18]

An interesting aspect of this cluster was the occurrence of measles in a 2-month-old baby whose 17-year-old mother was immune. In most cases, maternal antibodies from measles infection or vaccination will protect newborns. Antibody levels wane after 6 to 9 months and measles vaccination is offered at 9 to 12 months in most countries. It has been suggested that infants of women who have received measles vaccine may experience earlier loss of maternal antibody than infants whose mothers were immune due to natural infection.[19] This may result in insufficient protection for these children prior to their scheduled vaccination. The mother of the infant in this case thought she had contracted measles when aged five years and living overseas. She also recalled missing a high school vaccination. It is therefore likely that her immunity was due to natural disease and, accordingly, her infant would be expected to have passive immunity. The fact that the child developed measles indicates that a diagnosis of measles in a very young child presenting with measles-like symptoms, although unexpected, should not be discounted. An interesting feature of the infant's illness was a short incubation period with the rash appearing only eight days after exposure. Incubation periods for measles are typically shorter in children.[20]

The index case did not have a documented history of measles vaccination and was in an age group at higher risk. The lack of documented vaccination history amongst all but one case reinforces the importance of vaccination as a protective measure against the disease. Furthermore, patient recall of vaccination or disease cannot be relied upon as sufficient evidence. In this outbreak the index case claimed to have been vaccinated and to have had clinical measles. These claims may have reduced the index of suspicion of measles in the Emergency Department despite the classical measles symptoms and recent travel history. The claims are also a likely reason for lack of MMR vaccination prior to travel to a measles-endemic area. The National Health and Medical Research Council recommends MMR vaccination for travellers born during or since 1966 who have not received two doses of MMR vaccine.[21] The importance of documented history of vaccination rather than patient recall should be emphasised to potential travellers seeking advice about travel vaccination.

One child in this cluster (Case 4) could have received MMR vaccination shortly after exposure, an action that may have prevented the disease. However, despite advice from the PHU, the child was not vaccinated and subsequently developed measles. Transmission within the Emergency Department played a significant role in this outbreak with contact at that site being responsible for five cases. The highly infectious nature of measles was demonstrated by the development of measles in a staff member who did not come into contact with the patient, but who entered the room fifteen minutes after it had been vacated. Guidelines from NSW Health indicate that susceptible persons should not have entered the room for two hours after the infectious case had left.[6] On the other hand, a properly protected workforce would obviate the need for such measures to protect staff and would allow hospital resources to be used efficiently. Healthcare workers should be aware of their vaccination status and ensure their vaccinations are up-to-date. The issues of nosocomial transmission of measles and the need for staff vaccination are not new.[22,23] However their importance is not always reflected in a proactive approach by healthcare workers to ensure they are properly protected. At this hospital, some staff members in the age group at higher risk were reluctant to be vaccinated with MMR, even after two colleagues had contracted measles at work. Programs to promote MMR vaccination to staff during and after the outbreak were poorly patronised by clinical staff, with a better response from non-clinical staff. Studies of healthcare workers have attributed poor uptake of influenza vaccine to factors including a lack of awareness of its importance and concern about side effects,[24] and a perception that the health care worker was 'healthy' and did not need the vaccine.[26] If the same or similar reasons are responsible for poor uptake of MMR vaccine amongst hospital staff, a strong promotional campaign backed by institutional strategies is needed to address the problem.

Clearly, crowded hospital environments such as Emergency Departments are risky places in terms of transmission of airborne diseases and this outbreak highlights the need for appropriate infection control procedures in the event of suspected measles. Such infection control policies and procedures are of increasing importance in the effective management of disease outbreaks, and to respond to the emergence of novel viral diseases such as severe acute respiratory syndrome, and avian influenza.

The delay in diagnosis and confirmation of the index case was problematic as it resulted in delayed contact tracing and follow-up. With low rates of measles notifications in Australia, the improbability of measles as a diagnosis may result in delays in laboratory testing and diagnosis, quarantine of the case, and contact tracing, vaccination or immunoglobulin treatment. Until measles is eradicated, vigilance is required amongst physicians treating patients who present with a rash, or who have recently returned from overseas. Early action in notifying public health authorities and infection control staff, and the timely provision of immunoglobulin or MMR vaccination to those at risk are crucial steps in minimising the risk of secondary cases.

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A profile of HIV testing in Victoria, 1984 to 2004

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Abstract

HIV testing is an important public health strategy and collection of HIV testing data is a component of overall HIV surveillance activities. This paper describes changes in HIV testing patterns in relation to HIV diagnoses in Victoria between 1984 and 2004. HIV testing and diagnosis data were extracted from surveillance databases maintained at the Burnet Institute. The annual number of HIV tests performed in Victoria increased from 2,879 in 1984, to 193,927 in 2004. Between 1991 and 2004, the male HIV testing rate per 100,000 population increased from 2,754 to 3,710 and the female rate from 2,395 to 4,453. The proportion of HIV tests conducted by private laboratories increased from less than 1 per cent in 1991 to 75 per cent in 2004. The number of HIV diagnoses increased from 140 in 1999 to 233 in 2002 and then fell to 217 in 2004. The HIV diagnosis rate per 100,000 tests increased from 98.9 in 1999 to 137.7 in 2000 then decreased to 111.9 in 2004. The overall rate of HIV diagnosis per 100,000 tests was 291.6 for males and 25.9 for females. Increased testing among males is a good outcome considering the majority of HIV diagnoses in Victoria are among men who have sex with men (MSM). Increased testing among females probably relates to increased antenatal screening. The inability to collect sexual orientation and reason for test data limited interpretations. To provide a better understanding of the impact of testing on the HIV epidemiology, especially among MSM, linked HIV sentinel surveillance has been implemented in Victoria. *Commun Dis Intell* 2006;30:366–372.

Keywords: HIV test, HIV epidemiology, laboratories, diagnosis rate, HIV surveillance

Introduction

In Victoria, HIV passive surveillance involves case reporting of all new HIV diagnoses to the Burnet Institute. The Burnet Institute manages HIV surveillance on behalf of the Department of Human Services (DHS) and in collaboration with the Victorian Infectious Diseases Reference Laboratory (VIDRL). HIV passive surveillance has shown that the number of new HIV diagnoses in Victoria increased by 60 per cent between 1999 and 2003 (from 140 to 225 annual diagnoses) and approximately 70 per cent of diagnoses were among men who have sex with men (MSM).[1]

HIV testing data is an important element of HIV surveillance as it provides a denominator to help interpret passive surveillance data. Passive surveillance is simple, however data generated from the system can be difficult to interpret as trends over time may also be influenced by access to testing and other factors that may affect people's willingness to be tested for HIV. Testing data can be useful in evaluating the impact of interventions, policy, and HIV programs aimed at increasing awareness of HIV and promoting testing. For example, after the Grim Reaper campaign in 1987, data from South Australian testing laboratories suggested that there had been little change in the testing practices of those at risk, whereas there was a large increase in testing by those at low HIV risk.[2]

HIV testing first became available in Victoria in late 1984 and was originally performed by the Red Cross Blood Bank (RCBB) and four laboratories; the Microbiological Diagnostic Unit (MDU), the State Reference Laboratory at Fairfield Hospital (now VIDRL), the Royal Melbourne Hospital and the Alfred Hospital.[3] Selected private laboratories were authorised to conduct HIV tests in 1991, and as of 2004 there were 31 laboratories (including 28 diagnostic, plus the Victorian Institute of Forensic Medicine (VIFM), CSL, and RCBB authorised by the DHS to perform HIV serology diagnostic testing in Victoria. Testing laboratories all utilise an enzyme linked immunoassay (ELISA) method for their HIV

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testing. Sera from positive tests are forwarded to VIDRL for repeat ELISA testing and confirmation by the Western Blot method.

This paper describes the changes in HIV testing numbers, rates and demographics of those tested in comparison to HIV passive surveillance results in Victoria between 1984 and 2004.

Methods

Since 1985, clinicians have been advised by DHS to use a designated HIV request form when requesting an HIV antibody test to accompany the specimen to the laboratory.[4] This form was designed to capture demographic and epidemiological information on the person being tested including date of birth, sex, postcode, specimen date, reason for test, personal category (MSM, sex worker, drug user, or none of these), and HIV testing history. The form also allows the clinician to record a name code for confidentiality reasons rather than a full name as for other standard laboratory request forms.

The HIV request form has also been used for HIV testing budget allocation. From 1994 until December 2005, Victoria had in place a 'user pays' system for HIV testing, where laboratories could forward specimens collected from individuals who belong to one of several specified risk groups (sex worker, MSM, injecting drug user, homeless youth, or person reporting sexual contact with one of the former) to VIDRL or MDU for free testing.[5,6] VIDRL and MDU were subsidised by the Government for this testing. Any other individuals not identified as 'high risk' paid a fee of approximately $20 for HIV testing. This system was established to encourage testing among high risks groups and reduce the amount of funding provided for testing among low risk groups.[6] However, difficulties in collecting the payment meant most private laboratories stopped collecting this fee from patents in the mid-1990s and for efficiency reasons chose to do the HIV testing in-house at no charge to the patient (in any or no risk group). All positive screening tests were still referred to VIDRL (state HIV reference laboratory) for confirmation using Western Blot. As of November 2005, HIV testing came onto the Pathology Services Table, allowing a Medicare rebate to be received by the patient, and forwarded on to the laboratories (except for life insurance patients).

Over time, the number of requests received by private laboratories for HIV testing has increased markedly and the utilisation of the DHS HIV request form by clinicians has decreased. The DHS HIV request form is now mainly used by a select number of clinics who utilise VIDRL directly for HIV/sexually transmitted infections testing and see a high case load of the high risk groups for HIV infection, i.e. MSM.

As HIV testing was not a Medicare rebatable item in Australia prior to 2006, testing data were not available from the Health Insurance Commission, as with other sexually transmissible infections like *Chlamydia*. Therefore, since 1991 in Victoria, the Victorian DHS Infectious Diseases Regulations specify that all laboratories performing HIV antibody testing are required to report the total number of HIV tests each quarter to the Burnet Institute (on behalf of DHS). Other information collected on the HIV request form and reported to the Burnet Institute by laboratories includes sex, date of birth, patient postcode, personal category, and reason for test. The name code of the patient is not forwarded.

Data sources

HIV testing data between 1991 and 2004 were extracted from the HIV testing database held at the Burnet Institute. Although all variables were recorded on the request form from 1985, electronic data were unavailable prior to 1996, so data from this period were extrapolated from DHS annual infectious disease surveillance reports[7] and entered into the HIV testing surveillance database. The following time periods were used for analysis: 1984 to 2004 for total test numbers and diagnoses; 1991 to 2004 for sex and laboratory type data—1991 being when this information was first reported, and 1996 to 2004 for data on age.

HIV passive surveillance data between 1984 and 2004 were extracted from the HIV surveillance database also held at the Burnet Institute.

Total HIV testing numbers were obtained annually from the National Reference Laboratory (NRL)—NRL collect these data for national laboratory quality assurance purposes. These data allowed us to assess the completeness of HIV testing data collected by the Burnet Institute.

The Australian Bureau of Statistics census population data for Victoria were used for analysis (1991–1995 from the 1991 census;[8] 1996–2000 from the 1996 census; and 2001–2004 from the 2001 census).

Statistical analysis

Tests performed for non-diagnostic purposes (the RCBB, CSL and VIFM) were excluded from the analysis.

HIV testing data totals from the NRL were compared with HIV testing data collected by the Burnet Institute; an overall and annual percentage difference was calculated.

HIV testing rates per 100,000 population were calculated by multiplying the number of new HIV tests by 100,000 and dividing by the population. HIV

diagnosis rates per 100,000 tests were calculated by multiplying the number of new HIV diagnoses (taken from the HIV surveillance database) by 100,000 and dividing by the total number of HIV tests for the time period. Rates were also calculated by sex.

Statistical analysis was descriptive and performed using Microsoft Excel and Microsoft Access.

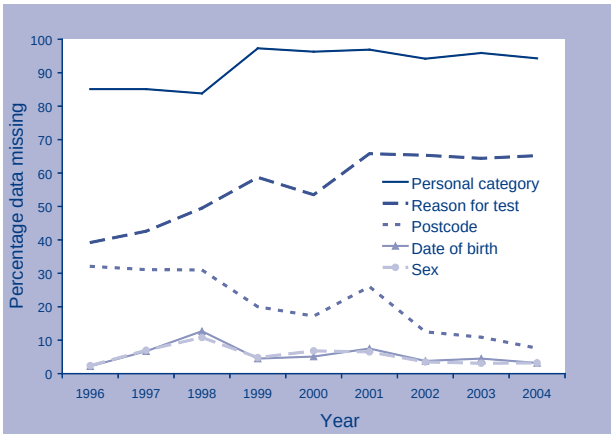
Results

Data quality

The overall number of tests received by the Burnet Institute was similar to the number received by NRL with 0.3 per cent more tests received by Burnet Institute than NRL (annual difference range –4.1% to 6.2%).

Figure 1 shows the proportion of missing data by variable. The proportion of data missing for postcode decreased over time from 32 per cent in 1996 to only eight per cent in 2004. The proportion incomplete for 'reason for test' increased from 39 per cent in 1996 to 68 per cent in 2004; with a larger proportion being incomplete among private laboratories (55% in 1996 and 75% in 2004) compared to public laboratories (38% in 1996 and 42% in 2004). Personal category was consistently greater than 80 per cent incomplete (Figure 1).

Figure 1. Percentage of data missing for each variable, 1996 to 2004, by year

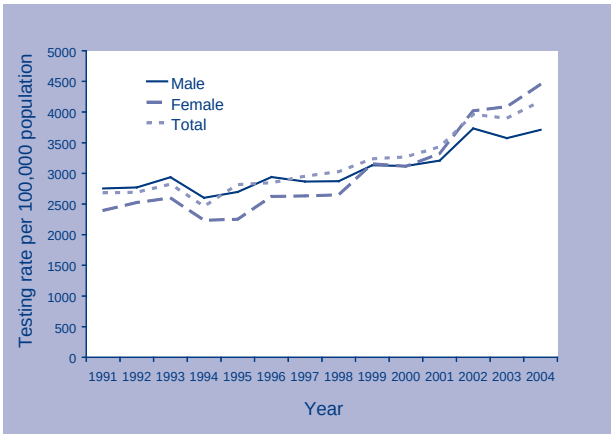


Total tests and testing rates per 100,000 population

Between 1984 and 2004, Victorian laboratories reported 291,301 HIV antibody tests to the Burnet Institute. During this time the number of tests performed per year increased from 2,879 in 1984 to 113,923 in 1991, and to 193,927 in 2004 (Table).

The rate of HIV tests in Victoria per 100,000 population increased from 2,684.2 in 1991 to 4,175.0 in 2004 (Figure 2).

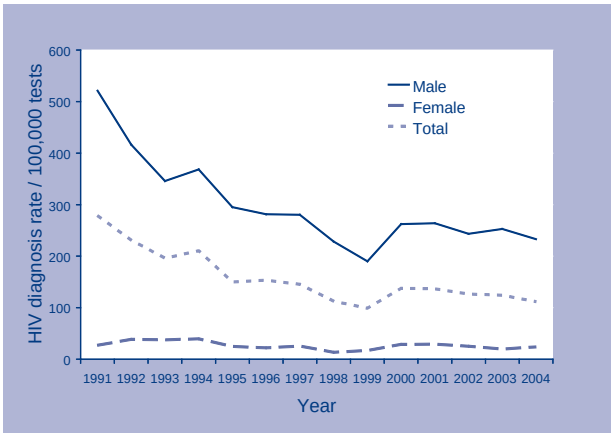
Figure 2. Rate of HIV tests per 100,000 population, Victoria, 1991 to 2004, by sex



HIV diagnoses rate per 100,000 tests

Between 1984 and 2004 there was a total of 5,291 new HIV diagnoses in Victoria. The number of HIV diagnoses decreased from 317 in 1991 to 140 in 1999 then increased by 66 per cent to 233 in 2002 and then fell by 7 per cent to 217 in 2004. In 1991 the diagnoses rate per 100,000 tests was 278.3 per 100,000 tests and similar to the pattern of diagnoses the rate decreased over the next decade to a low of 98.9 diagnoses per 100,000 tests in 1999, increased markedly to 137.7 per 100,000 in 2002, but declined slightly to 111.9 per 100,000 in 2004 (Figure 3).

Figure 3. Rate of HIV diagnoses per 100,000 tests, Victoria, 1991 to 2004, by sex



Sex

Since 1991, of those individuals where sex was known, 49.6 per cent (n=933,883) were male and 50.4 per cent (n=948,697) were female (Table).

Table. Number of HIV tests conducted and number of new HIV diagnoses, Victoria, 1984 to 2004, by sex

Year	Total HIV tests*	HIV tests: males*	HIV tests: females*	Number of new HIV diagnoses†	HIV diagnoses: males†	HIV diagnoses: females†
1984	2,879	§	§	181	174	4
1985	19,906	§	§	526	507	6
1986	25,130	§	§	349	333	7
1987	51,746	§	§	339	328	8
1988	61,264	§	§	289	269	19
1989	72,700	§	§	329	309	18
1990	96,258	§	§	305	287	16
1991	113,923	57,748	51,429	317	301	14
1992	114,294	58,119	54,240	264	242	21
1993	119,831	61,580	55,808	235	213	21
1994	104,574	54,553	47,974	220	201	19
1995	119,692	56,564	48,350	179	167	12
1996	124,547	63,245	58,314	191	178	13
1997	129,180	61,672	58,516	188	173	15
1998	132,438	61,747	58,960	149	141	8
1999	141,498	67,343	70,104	140	128	12
2000	143,061	67,092	69,237	197	176	20
2001	159,347	73,092	78,564	218	193	23
2002	183,981	85,088	95,138	233	207	24
2003	181,125	81,477	96,712	225	206	19
2004	193,927	84,563	105,351	217	197	25
Total	2,291,301‡	933,883§	948,697§	5,291‡	2,723	246

* From HIV testing surveillance.

† From HIV passive surveillance.

‡ Includes transgender individuals and those where sex is unknown.

§ Sex not available prior to 1991.

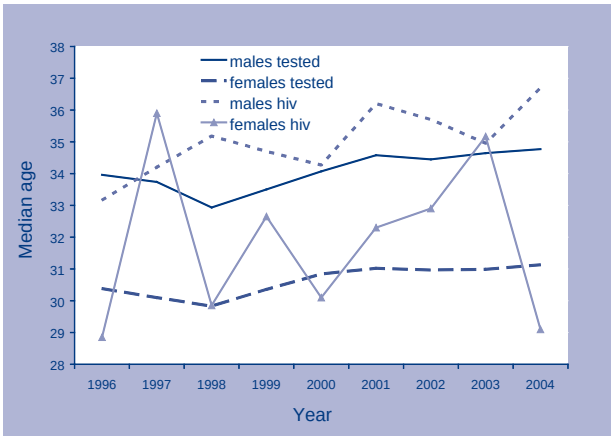
The rate of HIV testing per 100,000 population increased between 1991 and 2004 in both males (from 2,754.3 to 3,710.4) and females (from 2,394.7 to 4,452.9). Between 1991 and 1998 the rate of tests per 100,000 population was higher in males than in females, however since 1999 the rate was higher among females in Victoria (Figure 2).

Between 1991 and 2004, 2,723 males and 246 females were diagnosed with HIV in Victoria (Table). The overall HIV diagnosis rate per 100,000 tests in males was 291.6. The rate decreased from 521.2 in 1991 to 190.1 in 1999 and increased to 243.3 in 2002. The overall female HIV diagnoses rate per 100,000 tests was 25.9, remaining reasonably steady over time.

Age

The overall median age of males tested for HIV was 34.1 years compared to 30.8 years for females. The annual median age of males tested for HIV was consistently higher than for females and the annual median age of both sexes increased over time (Figure 4).

Figure 4. Median age of people tested for HIV and diagnosed with HIV, Victoria, 1996 to 2004, by sex

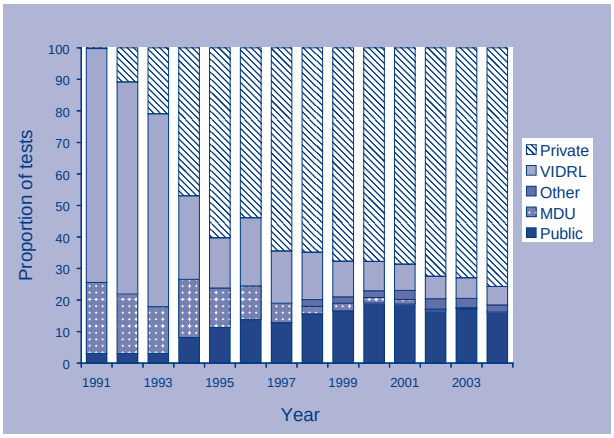


The overall median age of males diagnosed with HIV was 35.2 years, compared to 31.5 for females. Although the median age of females was highly variable due to small numbers the annual median age of males was consistently higher than for females, except for 1997 (Figure 4).

Testing by laboratory type

In the 1980s all HIV serological tests were performed by four main public laboratories. Since 1991 the proportion of testing done by private laboratories has increased substantially, with private laboratories conducting 76 per cent of all HIV tests in 2004 in Victoria (Figure 5).

Figure 5. Proportion of tests performed by different categories of laboratories Victoria, 1991 to 2004



VIDRL – State Reference Laboratory.

PUBLIC – Public Pathology Laboratories and Public Hospital Laboratories.

PRIVATE – Private Pathology Laboratories.

OTHER includes military and IVF laboratories.

MDU – Microbiological Diagnostic Unit.

Discussion

This analysis has shown that the number of HIV tests performed in Victoria has increased markedly since the implementation of HIV testing in 1984. The male to female ratio of tests performed has remained approximately equal overall, although in recent years the proportion of tests among females has increased. Similar to trends observed in HIV surveillance, the HIV diagnosis rate calculated using testing data as a denominator decreased over time but showed a marked increase in 2000, an increase which has not returned to the lower rate of 98.9 per 100,000 tests observed in 1999. The majority of HIV tests are now performed by private laboratories.

HIV testing data also allows for assessment of the extent of HIV testing in a population. Testing is an important public health strategy; it provides clinicians with an opportunity to offer information and education to patients to reduce their risk.[9] Positive HIV results can also be followed up with partner notification and contact tracing. Early detection can also allow individuals to take action to prevent further transmission and ensures timely introduction of treatment, which is known to relate to better clinical outcomes.[10] Antenatal HIV screening is important as the diagnosis allows for interventions to be implemented during pregnancy, labour and post-partum which reduce the risk of mother-to-child transmission.[11]

This analysis has shown a marked increase in HIV testing among females which most likely relates to increased incorporation of HIV into standard antenatal screening in Australia. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists recommends universal HIV testing at the time of the first antenatal visit.[11] The estimated proportion of pregnant women screened for HIV in Australia has increased from around 20 per cent in 1991–92,[12] to 33 per cent in 1999.[13]

The total number of HIV tests performed annually among males has also increased substantially over the past two decades, which is a good public health outcome considering the major risk group for HIV in Victoria is MSM.[1] The testing guidelines for HIV/STIs for MSM released by the Royal Australasian College of Physicians, recommend annual HIV testing.[14] However, it is possible that a large proportion of testing among males was due to testing among low risk groups as part of other screening (i.e. insurance screening) rather than among the high risk group MSM. Unfortunately, the high proportion of missing data on risk group (personal category) means that testing patterns and diagnosis rates cannot be determined specifically for MSM from HIV testing surveillance.

HIV testing data also allows for the assessment of the impact of any changes in testing on the current Victorian HIV epidemiology as passive HIV surveillance trends over time may also be influenced by testing behaviour or testing campaigns that encourage testing. Although the diagnosis rates trends were similar to passive surveillance diagnoses trends, suggesting that the increases in diagnoses observed between 1999 and 2002 were unlikely to have been influenced by marked increases in HIV testing, without information specifically about MSM the HIV data currently collected is probably not sensitive enough to assess this accurately.

This analysis was limited by the quality of the data reported. Although data completion for date of birth, sex and total test numbers were high, many testing

laboratories sent incomplete data on other variables, often because these data are not recorded on the test request form or because the laboratory does not have the capacity to extract the information efficiently. The standard pathology request forms used for requesting tests from private laboratories do not ask for information on risk group or reason for test, this hinders data collection given that the majority of tests are now conducted by private laboratories. Furthermore, at the time of analysis, private laboratories were not provided with funding to conduct HIV serology tests and therefore had little motivation to collect the additional data. The shift in HIV testing from public to private laboratories means that data quality has become poorer and data completeness has decreased over time. The lack of complete information, especially on risk category and reason for test, reduces the utility of HIV testing surveillance for interpreting trends based on denominator data. One option to improve completeness of the epidemiological information could be to encourage clinicians to use the HIV test request form and to fund laboratories for the data extraction. However this option probably isn't sustainable.

The incorporation of sentinel surveillance data within subgroups at high risk, i.e. MSM, to compliment total test numbers may be a better solution for future analysis of HIV testing patterns and diagnosis rates within specific groups in the community.[15] With funding from DHS, the Burnet Institute in collaboration with DHS, VIDRL and the Melbourne Sexual Health Centre implemented a linked HIV sentinel surveillance system in early 2006 which will be conducted over three years. This system involves collection of demographic data, HIV testing history, and sexual behaviour information by clinicians using a questionnaire from all clients undergoing HIV testing at selected clinics with a high case load of MSM. This information will be linked to the HIV test result. The system will enable us to determine the total HIV tests conducted among MSM seen at the clinics and the proportion testing positive. These data will be used to gain a more comprehensive picture of at risk people being tested and the impact of testing on HIV epidemiology.

The data presented in this paper have shown that HIV testing has increased substantially in the past two decades in Victoria, in both males and females with more testing occurring among female in recent years. Collection of HIV data is an important element of overall HIV surveillance, however in this system poor data quality has limited the usefulness of the system. New strategies have been implemented to overcome some of these limitations.

Acknowledgements

HIV passive surveillance and HIV testing surveillance are managed on behalf of, and with the funding of, the Victorian Department of Human Services. HIV testing surveillance would not be possible without the cooperation and support of contributing laboratories. We gratefully acknowledge the partner notification officers from the DHS; Tom Carter, Beth Hatch, Jane Tomnay and Alan Breschkin from VIDRL for their important work in HIV passive surveillance. We would also like to thank Marina Karakaltsas from The National Serology Reference Laboratory for her assistance with this analysis.

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Erratum

In the last issue Communicable Diseases Surveillance Highlights (*Commun Dis Intell* 2006;30:253), it was stated that "…..the National meningococcal C Vaccination Program which commenced in 2003 and completed vaccination of all under 19-year-olds by the end of 2004."

It has been brought to our attention that the Program has not been completed in some Australian states and territories and that there are no accurate data on the proportion of under 19-year-olds who were vaccinated, although data are available on the vaccination coverage in under 7-year-olds through the Australian Childhood Immunisation Register.

We apologise for this misleading statement.

Campylobacter outbreak due to chicken consumption at an Australian Capital Territory restaurant

Andrew P Black,[1] Martyn D Kirk,[2] Geoff Millard[3]

Abstract

***Campylobacter* is the most common cause of bacterial gastroenteritis in Australia, with 15,008 notifications in 2004. This represents only a small fraction of the total cases of *Campylobacter*. Despite this, outbreaks are rarely reported. This report describes the investigation of an outbreak of campylobacteriosis following a restaurant meal in the Australian Capital Territory. The outbreak was identified by a general practitioner who notified the Health Protection Service, ACT Health. A retrospective cohort investigation of the 27 work colleagues who attended lunch at the restaurant was conducted. Eleven cases were identified with two culture positive for *Campylobacter*. An association between eating several dishes containing chicken was identified. This outbreak highlights the important identified risk for *Campylobacter* infection from commercially prepared chicken. It also demonstrates the important role of clinicians in notifying disease outbreaks. *Commun Dis Intell* 2006;30:373–377.**

Keywords: Campylobacter*, outbreak, gastroenteritis, foodborne disease*

Introduction

Campylobacter is the most common bacterial cause of gastrointestinal infection in Australia, with 15,008 notifications to health authorities in Australia in 2004.[1] However, the number of notified cases represents only a small percentage of the total cases of C*ampylobacter* and it has been estimated that the true burden is approximately 277,000 cases annually.[2] Outbreaks of campylobacteriosis are infrequent and the majority of infections appear to be sporadic. The reasons for this include the microbiological characteristics of the organism, the lack of public health follow-up of cases and the incomplete strain characterisation in microbiology laboratories.[3] Evidence from the *Campylobacter* Sentinel Surveillance scheme in the United Kingdom[4] suggests that *Campylobacter* outbreaks may be more common than previously suspected. Recent outbreaks have resulted from contamination of drinking water, raw milk, and cross contamination from high risk foods including chicken, salad and dairy products.[5–12]

A recent case-control study of *Campylobacter* infections in Australia identified that eating and preparing chicken was responsible for approximately 30 per cent of *Campylobacter* cases.[13] Raw chicken is commonly contaminated with *Campylobacter*. A retail survey in the Australian Capital Territory in 2000 found 20.6 per cent of raw chicken samples were positive for *Campylobacter*.[14] However, retail chicken surveys in other countries have identified much higher levels of *Campylobacter* ranging from 32 per cent to 83 per cent of samples.[15–18] Despite such high levels of contamination with *Campylobacter*, chicken has not been identified as a major cause of the infrequent *Campylobacter* outbreaks.

This report describes the epidemiological, microbiological and environmental investigation of a *Campylobacter* outbreak following a meal at a restaurant in the Australian Capital Territory in 2005.

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Methods

In April 2005 the Health Protection Service of ACT Health was notified of a number of cases of gastrointestinal illness by a general practitioner whose patient had tested positive for *Campylobacter*. Investigations revealed that the patient's illness followed a lunch with work colleagues at a local restaurant approximately three days earlier.

Hypothesis generating interviews were conducted with two cases: the index case, and another work colleague who was hospitalised with *Campylobacter enteritis*. A retrospective cohort study was undertaken with interviews conducted by telephone. The cohort was defined as the people who attended the workplace lunch at the restaurant. A questionnaire was used to obtain information about the onset and nature of any gastroenteritis illness, exposure to foods at the lunch banquet and contact with other people ill with gastroenteritis either prior to, or after the individual's illness. A case was defined as a person who attended the restaurant lunch on 8 April who had diarrhoea between 9 April and 18 April. Questionnaires were completed with each person who attended the lunch. Data were entered and analysed with SPSS Version 11.

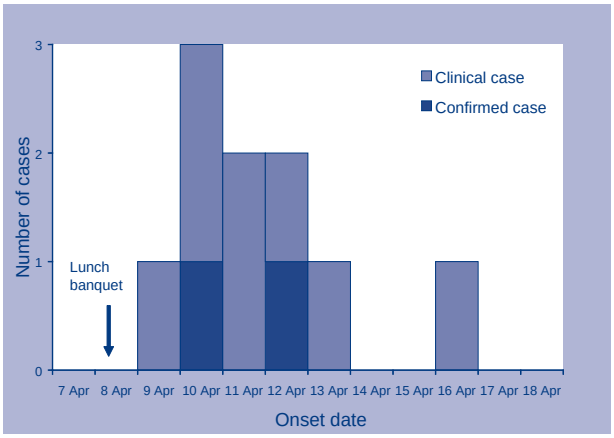
To investigate the environmental cause of the outbreak, ACT Health Protection Service staff visited the restaurant to audit food safety and collect samples for analysis.

Results

Cohort study

Using the questionnaire, public health officers interviewed all 27 people identified in the cohort. The median age of respondents was 33.5 years (range 19–45 years) and 20 (74%) were female (Figure).

Figure. Cases of gastroenteritis among group attending restaurant lunch on 8 April, by date of onset



There were 11 cases identified with a median age of 35 years (range 21–45 years) and six (55%) were female. Other symptoms apart from diarrhoea were nausea in 10 cases (91%), abdominal pain in eight cases (72%), vomiting in six cases (54%), and fever in six cases (54%). Duration of illness was between three and seven days for nine of the cases. One case was hospitalised.

Twenty of the cohort had eaten a banquet and the other seven people had ordered separate dishes from the á la carte menu. Ten of the cases (91%) had eaten the banquet compared to 62 per cent of people who were not ill. The risk ratio for eating the banquet was 3.5 (95% confidence interval (CI) 0.5–22.6). The attack rate for those who ate the banquet was 50 per cent (Table). The banquet included a selection of pizza and pasta dishes and warm chicken salad. The one case that ordered from the menu had a mega meat pizza.

Foods from the banquet with the highest risk ratios (RR) were: warm chicken salad RR 3.5 (95% CI 0.5–22.6) with an attack rate of 50 per cent and chicken mushroom (pollo funghi) pasta, RR 3.4 (95% CI 1.5–7.8) with an attack rate of 86 per cent. People eating chicken in any dish had a Relative Risk of 3.5 (95% CI 0.5–22.6) for developing gastroenteritis. It was not possible to perform stratified analysis of these chicken dishes as all cases that ate chicken mushroom pasta also consumed the warm chicken salad. None of the respondents mentioned that the chicken or other meat dishes were under-cooked.

There were no additional *Campylobacter* cases linked to the same restaurant through a search of the ACT Notifiable Disease database or routine investigation of *Campylobacter* questionnaires sent to all cases in the Australian Capital Territory between March–May 2005.

Microbiological investigation

Three faecal specimens were obtained, two were positive for *Campylobacter*. No speciation was performed by either of the two pathology laboratories receiving these samples. All three samples were negative for other pathogens including norovirus and rotavirus.

Environmental investigation

An environmental audit of the restaurant revealed no major deficiencies in food safety although the pizza bar ingredients were being stored at 6–8°C, and there was no soap in the kitchen hand basin.

Fresh samples of the warm chicken salad, chicken mushroom pasta, tandoori chicken pizza, original pizza and four toppings pizza were obtained from

Table. Attack rates and relative risk for foods eaten at the restaurant lunch on Australian Capital Territory, 8 April 2005

Food	Ate			Did not eat			
	Ill	Total	Attack rate (%)	Ill	Total	Attack rate (%)	RR (95% CI)
Pizza	9	19	47	2	8	25	1.9 (0.5–6.9)
Original	5	7	71	6	20	30	2.4 (1.1–5.4)
Super special	3	8	38	8	19	42	0.9 (0.3–2.5)
Marinara	3	5	60	8	22	36	1.7 (0.7–4.1)
Quattro gusti	5	7	71	6	20	30	2.4 (1.05–5.4)
Vegetarian	3	6	50	8	21	38	1.3 (0.5–3.5)
American	4	6	67	7	21	33	2.0 (0.9– 4.6)
Mushroom	5	7	71	6	20	30	2.4 (1.1–5.4)
Cappriciosa	2	3	67	9	24	38	1.8 (0.7–4.6)
Napoletana	2	3	67	9	24	38	1.8 (0.7–4.6)
Aussie	2	3	67	9	24	38	1.8 (0.7–4.6)
Tropical	2	3	67	9	24	38	1.8 (0.7–4.6)
Margherita	2	3	67	9	24	38	1.8 (0.7–4.6)
Mexicana	4	5	80	7	22	32	2.5 (1.2–5.3)
Calabrese	3	3	100	8	24	33	3.0 (1.7–5.3)
Pasta	9	19	47	2	8	25	1.9 (0.5–6.9)
Napoletana	5	8	63	6	19	32	2.0 (0.8–4.6)
Arrabiata	3	4	75	8	23	35	2.2 (1.0–4.8)
Bolognese	3	5	60	8	22	36	1.7 (0.7–4.1)
Carbonara	5	9	56	6	18	33	1.7 (0.7–4.0)
Primavera	1	3	33	10	24	42	0.8 (0.2–4.2)
Alla matriciana	3	3	100	8	24	33	3.0 (1.7–5.3)
Calabrese	1	2	50	10	25	40	1.3 (0.3–5.4)
Ortolana	1	1	100	10	26	38	2.6 (1.6–4.2)
*Pollo funghi	6	7	86	5	20	25	3.4 (1.5–7.8)
Pesto	3	8	38	8	19	42	0.9 (0.3–2.5)
Marinara	4	7	57	7	20	35	1.6 (0.7–3.9)
*Zefferelli	3	5	60	8	22	36	1.7 (0.7–4.1)
Salmon	1	1	100	10	26	38	2.6 (1.6–4.2)
*Warm chicken salad	10	20	50	1	7	14	3.5 (0.5–22.6)
Drinks							
Water	10	22	45	1	5	20	2.3 (0.4–13.9)
Wine	7	13	54	4	14	29	1.9 (0.7–5.0)
Leftover food taken home	2	4	50	9	23	39	1.2 (0.4–3.4)
Ate chicken in any dish	10	20	50	1	7	14	3.5 (0.5–22.6)

* Indicates contains chicken.

the restaurant during inspection. These samples were negative for pathogens. However, the samples were not tested for *Campylobacter* due to lack of accreditation in the ACT Government Analytical Laboratory at that time.

Discussion

Campylobacteriosis has been the most common notifiable infectious enteric disease in the Australian Capital Territory since 1991, with 383 notifications in 2004. These cases appear to be sporadic. This is the first *Campylobacter* outbreak detected in the Australian Capital Territory. This outbreak highlights important identified risks for *Campylobacter* infection, particularly chicken prepared in a commercial catering setting.[3] The vehicle for this outbreak was likely to have been either the warm chicken salad or the chicken mushroom pasta. Risks associated with undercooked chicken have been highlighted in other studies.[9,13] This outbreak highlights the importance of ensuring that chicken is thoroughly cooked and taking measures to prevent cross-contamination of ready to eat foods with raw chicken.

Recent work in the United Kingdom has highlighted the importance of strain characterisation to improve identification of *Campylobacter* outbreaks and understanding the different epidemiology of different species.[3,19] This outbreak highlights the lack of microbiological investigation as routine laboratory practice in the Australian Capital Territory and New South Wales is limited to isolating *Campylobacter* spp. and no further typing is performed. The ACT Government Analytical Laboratory has since undergone NATA accreditation of its methods for detecting *Campylobacter* in food and this should enable more complete microbiological investigation in future outbreaks.

This outbreak was notified by a doctor and may otherwise have been missed as there were only two cases notified to Communicable Disease Control, ACT Health. The Australian Capital Territory has recently updated the Notifiable Disease Code of Practice and now requires dual notification by doctors and hospitals as well as laboratories. This report highlights the additional important role of clinicians in notifying disease outbreaks.[20]

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National Vaccine Safety Workshop: summary and draft recommendations

Glenda Lawrence, on behalf of the National Immunisation Committee

Introduction

A National Vaccine Safety Workshop was held at the University of Sydney on 17 November 2005. The workshop was sponsored by the National Centre for Immunisation Research and Surveillance (NCIRS), the Australian Government Department of Health and Ageing (DoHA) and the National Immunisation Committee (NIC). It was attended by 40 invited representatives of federal, state and territory health departments, the Australian Technical Advisory Group on Immunisation, the Therapeutic Goods Administration (TGA), the Adverse Drug Reactions Advisory Committee (ADRAC), the Australian Medical Association, the Royal Australian College of General Practitioners, the Australian Divisions of General Practice (ADGP), clinical immunisation specialists, consumers and industry. The aims of the workshop were to review current post-licensure vaccine safety practices in Australia and to work towards developing a national vaccine safety strategy.

The first part of the workshop consisted of a series of presentations outlining international and current Australian practices. This formed the basis for the second part of the workshop where participants divided into three working groups to discuss issues and formulate draft recommendations in the areas of (i) surveillance; (ii) clinical management and research; and (iii) communication. Final workshop recommendations were reached by consensus.

Presentations

International overview

Mike Gold (Women's and Children's Hospital, Adelaide) outlined initiatives by the World Health Organization in developing a set of indicators for national regulatory authorities to assess vaccine safety practices, including the ability to detect and investigate adverse events following immunisation (AEFI) and ensure transparency and accountability. In the United States of America, the role of vaccine safety was recently separated from the immunisation program to avoid perceived conflicts of interest. Internationally, the focus of vaccine safety programs is moving away from the purely population health focus of AEFI surveillance to one where both the individual and the population are considered. This new paradigm includes surveillance, clinical management and communication.

Vaccine safety in Australia

National overview

Ian Boyd (TGA), David Isaacs (ADRAC) and Glenda Lawrence (NCIRS) described the current national passive AEFI surveillance system from different perspectives, and identified the major strengths and weaknesses of the system. Strengths included the centralised notification, review, analysis and regular publication of summary data. Weaknesses included the significant differences in surveillance practices between the states and territories, and the conflicting priorities of timely reporting at the national level versus complete reporting after case investigations are concluded. The complexity of analysis and interpretation of AEFI surveillance data was highlighted, as well as the need for better communication of available information to providers and consumers.

Paul Roche (DoHA) highlighted parallels between disease surveillance systems and AEFI surveillance at the local, jurisdictional and national levels. Mechanisms implemented in Australia's communicable diseases surveillance processes to improve consistency between states and territories, timeliness of reporting to the national system and case management at the local level could serve as a model for AEFI surveillance.

State and territory perspectives

Each state and territory representative spoke briefly about AEFI surveillance practices, clinical management and communication processes in their jurisdiction. Surveillance practices differ considerably as do the level of resources available for surveillance and clinical management of AEFI. All jurisdictional representatives indicated that systems were in place for individuals to consult clinical specialists regarding AEFI. Many indicated the need to improve

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education and communication with providers and consumers, and between jurisdictions. All indicated a willingness to address issues of communication and consistency in AEFI surveillance practices at a national level.

Special initiatives

Data linkage

Sarah Dugdale (South Australian Vaccine Safety Data Linkage Project) summarised a pilot data linkage project that is being conducted in South Australia. Like the United States of America and United Kingdom AEFI data linkage programs, the pilot South Australian project aims to link clinical and immunisation records to detect both known and unknown AEFIs, test hypotheses and investigate signals identified through passive AEFI surveillance. Surveys of consumers and providers found a high level of acceptance of data linkage for this specific purpose. Assessment of the feasibility of routine data linkage is in progress.

Special immunisation clinics

Nick Wood (NCIRS) gave an overview of the roles and practices of clinics located in major hospitals in Sydney, Canberra, Melbourne, Adelaide and Perth that specialise in the management of children who may have experienced an AEFI. Staff from each clinic collaborate via regular national teleconferences to discuss specific clinical management issues and have recently conducted a clinical trial on the re-immunisation of children who have had a large local reaction to a diphtheria-tetanus-acellular pertussis vaccine. Future plans include the development of clinical protocols for the management of AEFI and harmonisation of clinic databases to allow a summary report to be produced annually.

Working groups

At the start of the afternoon session, Mark Ferson (NSW Health), summarised the themes and issues that arose from the presentations and discussion of current international and Australian post-marketing vaccine safety practices. Participants then divided into the three working groups (according to individual interest). Discussion in each working group was lead by a person with relevant expertise in the specific area on which the working group focussed.

The Surveillance Working Group was facilitated by Paul Roche (DoHA). The group discussed the objectives of AEFI surveillance, the events that should be under surveillance, ways to improve the passive surveillance process and options regarding active surveillance.

The Clinical Management and Research Working Group was facilitated by Mike Gold (Women's and Children's Hospital, Adelaide). The group discussion focussed on mechanisms to improve access to specialist advice on the clinical management of children and adults with AEFI, particularly advice about re-immunisation. They also discussed research objectives.

The Communication Working Group was facilitated by Julie Leask (NCIRS). The group identified the major communication players and their information needs. Discussion also focussed on the need for clear communication between all stakeholders about vaccine safety and how best to canvass consumer input.

Workshop recommendations

Final workshop recommendations were reached by consensus and are summarised below.

Surveillance

1. Implement a simple national system for passive AEFI surveillance that retains ADRAC at its core.
2. Clarify the objectives of AEFI surveillance at the local, jurisdictional and national levels.
3. Conduct surveillance for vaccine failures through disease surveillance processes rather than AEFI surveillance processes.
4. Review the AEFI surveillance case definitions for inclusion in the next (9th) edition of the *Australian Immunisation Handbook*.
5. Improve the timeliness and completeness of data submission to ADRAC.
6. Amend the current ADRAC (blue) notification form to collect data relevant to AEFI.
7. Improve feedback between ADRAC and providers and consumers with aggregate reports, or at the individual level where possible.
8. Ensure that the passive surveillance system is functioning appropriately before considering ongoing active surveillance at a national level while recognising that there is the occasional need to conduct active surveillance to investigate specific issues.

Clinical management and research

9. Ensure that providers and consumers have access to expert opinion on the clinical management of AEFI.
10. Standardise and collate data for the individual special AEFI clinics, and report summary data annually using Brighton Collaboration case definitions.

11. Develop uniform national guidelines on the clinical management of AEFI. This process could be assisted by the production of an annual report for all AEFI special clinics.

12. Review the resource requirements to implement recommendations 9 to 11.

Communication

13. Produce AEFI report summaries in an easily digestible format to circulate to Divisions of General Practice, public health units, state and territory health departments, consumers who report AEFI, and other relevant groups. AEFI data should be reported within the broader context of program evaluation and disease prevention.

14. Produce and distribute brochures and online information for providers and consumers about AEFI reporting procedures and the availability of special AEFI clinics.

15. Convene a meeting to assess ways to obtain input from consumers on vaccine safety.

16. Develop mechanisms to enhance communication between states and territories regarding vaccine safety issues.

Conclusion

In March 2006, the recommendations arising from the workshop were considered at a meeting of the National Immunisation Committee. The committee convened the AEFI Working Party to review, prioritise and progress all the recommendations. Members of the AEFI Working Party include representatives of NCIRS (Chair), DoHA, jurisdictions, ADRAC, TGA and ADGP. Linkage has also been established between the AEFI Working Party and the specialist AEFI clinical group to progress recommendations related to the clinical management of AEFIs. The AEFI Working Party meets regularly by teleconference and reports progress to the NIC.

Acknowledgements

The National Vaccine Safety Workshop was sponsored by the National Centre for Immunisation Research and Surveillance, the Australian Government Department of Health and Ageing and the National Immunisation Committee.

The Steering Committee, responsible for planning the National Vaccine Safety Workshop, included Jennie Roe (DoHA), Letitia Toms (DoHA), Sue Campbell-Lloyd (NSW Health), Christine Selvey (Northern Territory Health Department), Mike Gold (Women's and Children's Hospital, Adelaide), Maureen Watson (Department of Human Services, South Australia), Peter McIntyre (NCIRS) and Glenda Lawrence (NCIRS).

The sponsors thank all presenters and participants for their time and valuable contributions to the workshop and to the report. We particularly thank: Mark Ferson (NSW Health) for undertaking the vital role of workshop facilitator; Paul Roche, Mike Gold and Julie Leask for facilitating the small group discussions; and NCIRS staff Kristine Macartney, Nick Wood, Rob Menzies, Helen Quinn and Julie Leask for drafting sections of the report and Jan Michniewicz for organising the workshop.

OzFoodNet quarterly report, 1 April to 30 June 2006

The OzFoodNet Working Group

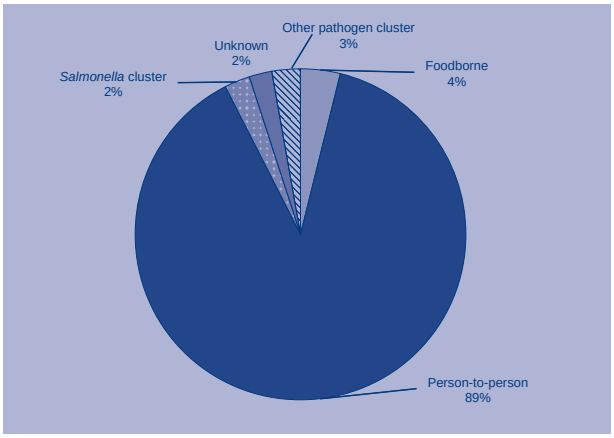
Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigation of outbreaks of gastrointestinal illness and clusters of disease potentially related to food occurring in Australia between 1 April and 30 June 2006.

Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change, as the results of outbreak investigations can take months to finalise.

During the second quarter of 2006, OzFoodNet sites reported 578 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under-represent the true burden of these infections. In total, these outbreaks affected 14,688 people of which there were 306 hospitalised and 10 deaths. The majority (89%, n=514) of outbreaks resulted from infections suspected to be spread from person-to-person (Figure). There was considerable activity during the second quarter of 2006 as jurisdictions reported an increase in the number of outbreaks of enteric illness involving institutions. Of the 514 outbreaks in institutions, 350 (68%) were in aged care facilities, 93 (18%) outbreaks were in hospitals, 47 (9%) were in child care facilities and 24 (5%) were in various other settings. Norovirus was identified as a cause of illness in 202 (58%) of the outbreaks in aged care facilities and was suspected in many more.

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 April to 30 June 2006



Foodborne disease outbreaks

There were 22 foodborne disease outbreaks during the second quarter of 2006 where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 233 people and resulted in 26 people being admitted to hospital. This compares with 27 outbreaks for the second quarter of 2005 and 26 outbreaks in the first quarter of 2006.

Salmonella was responsible for eight outbreaks during the quarter, with *Salmonella* Typhimurium being the most common serotype. *S.* Typhimurium 9, *S.* Typhimurium 170/108, and *S.* Typhimurium 135a were each responsible for one outbreak. Other *Salmonella* serotypes causing outbreaks were Anatum, Bovismorbificans 11, Oranienburg, Oslo, and Singapore. Single outbreaks were caused by norovirus and *Clostridium perfringens*, with an additional two outbreaks suspected to have been caused by *Clostridium perfringens*. One outbreak was suspected to have been caused by *Staphylococcus aureus* intoxication. The remaining nine outbreaks were caused by unknown aetiological agents.

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All data are reported using the date the report was received by the health agency.

Eight outbreaks reported in the quarter were associated with food prepared by restaurants, four by commercial caterers, three in private residences and three by a commercial food manufacturer. Single foodborne disease outbreaks were associated with an aged care facility, a nationally franchised fast food restaurant, an item of primary produce, and a takeaway food premises.

To investigate these outbreaks, sites conducted three cohort studies and one case control study. Descriptive data were collected for 17 outbreaks and individual patient data were not available for one outbreak. Investigators obtained microbiological evidence linking a food vehicle to illness in three outbreaks, analytical epidemiological evidence in two outbreaks and a combination of microbiological and analytical evidence for one outbreak. For the remaining 16 outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

Queensland reported six outbreaks of foodborne illness during the quarter. *S.* Typhimurium 135a was responsible for illness in 11 people that had attended a privately catered function. The food was prepared by family members, some of whom reported gastrointestinal symptoms during the week prior to the function. An outbreak of suspected *Staphylococcal aureus* intoxication involving three members of one family was probably caused by inadequate refrigeration of stored sushi by the takeaway food shop.

New South Wales reported eight foodborne disease outbreaks during the second quarter of 2006. At least 70 people attending a community function were ill with gastroenteritis consistent with *Clostridium perfringens* intoxication. *Clostridium perfringens* was identified in samples of leftover chicken curry. An environmental inspection of the caterer's premises revealed temperature abuse and inadequate facilities for the preparation of large quantities of food. An investigation of gastroenteritis associated with a restaurant catered party identified 15 cases of illness. Three clinical specimens were positive for norovirus, however both the epidemiological and environmental investigation did not identify a causative food item, exposure, or person who was infectious with gastroenteritis at the time of the function. A food vehicle and organism were not confirmed in the remaining six outbreaks

Four outbreaks of foodborne disease were reported by Victoria during the quarter including an outbreak of *Salmonella* Oranienburg implicating a brand of alfalfa sprouts. The investigation was triggered by a recall of alfalfa sprouts by the manufacturer in May as a result of the quality assurance program isolating *S.* Oranienburg. Fifteen cases of *S.* Oranienburg notified to the Department of Human Services in Victoria were investigated and it was confirmed that seven cases had eaten the recalled brand of sprouts. *S.* Oranienburg was detected in alfalfa seeds collected from the sprout manufacturer and also found in leftover sprouts from two case's homes. This follows a similar investigation of a large *S.* Oranienburg outbreak caused by contaminated alfalfa sprouts in Western Australia from November 2005 to February 2006.[1]

An investigation into an increase in cases of *S.* Bovismorbificans commenced in March. Thirteen cases (12 notified in Victoria and 1 case in South Australia) reported eating 'smallgoods' meat in the seven days before their onset of illness. Of these, 10 cases reported eating capocollo – a cured pork product and six cases identified the same brand. Local councils sampled a wide range of products and *Salmonella* Bovismorbificans 11 was detected in this implicated brand of capocollo. As a result, the manufacturer conducted a voluntary recall of this product in May. The two remaining Victorian outbreaks of foodborne illness involved cases who were ill with gastroenteritis consistent with *Clostridium perfringens* intoxication.

Two foodborne disease outbreaks were investigated in South Australia during the quarter. One involved an investigation of *S.* Typhimurium 9 where six cases had dined at the same hotel. Eating sweet potato and fetta cheese salad, a new menu item in the hotel, was significantly associated with illness. There were 19 cases of *S.* Typhimurium 170/108 with an illness onset around late May 2006. A case control study was conducted and showed a statistically significant association between the consumption of ravioli and illness; OR 44 (95% CI 2.7-1348). Ravioli samples from a consumer and retail outlet tested positive for *S.* Typhimurium 170/108. Molecular typing was conducted and the ravioli samples had a similar pattern when compared to the isolates of cases that had eaten ravioli. These findings led to a product recall of the commercially produced ravioli by the manufacturer.

The Northern Territory investigated two cases of *S.* Oslo notified in the same week. Both cases had eaten sticky rice balls with shredded chicken from a market vendor. An environmental inspection found that the product was held at ambient temperature until sold at the market.

Western Australia investigated six cases of gastroenteritis caused by *S.* Anatum with onset of illness over a two week period from late May 2006. All cases lived in or near a south west regional town and these were the first cases of *S.* Anatum in this region since 1999. Five of the six cases had eaten at the same nationally franchised fast food restaurant in the week prior to the onset of their illness. Samples

of food were collected from the nationally franchised fast food restaurant during an environmental investigation but were negative for *Salmonella* species.

OzFoodNet sites reviewed national data during the quarter following an international recall of Cadbury chocolate implicated in British cases of *S.* Montevideo. OzFoodNet investigated Australian cases of *S*. Montevideo and none were associated with chocolate consumption.

Tasmania and the Australian Capital Territory did not report a foodborne outbreak in the second quarter of 2006.

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Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* April to June, 2006

State	Month of outbreak	Setting prepared	Infection/illness	Number affected	Evidence	Responsible vehicle
NSW	April	Restaurant	Norovirus	15	D	Unknown
	May	Commercial caterer	*Clostridium perfringens*	70	M	Chicken curry
		Commercial caterer	Unknown	7	D	Suspect noodles or garnish
	June	Restaurant	Unknown	4	D	Suspect Nile perch filets
		Restaurant	Unknown	6	D	Suspect oysters
		Private residence	Unknown	21	A	Suspect birthday cake
		Restaurant	Unknown	8	D	Unknown
		Commercial caterer	Unknown	3	D	Suspect potato salad
NT	May	Private residence	*Salmonella* Oslo	2	D	Suspect sticky rice balls with chicken
Qld	April	Takeaway	*Suspected Staphylococcus aureus*	3	D	Sushi roll
		Restaurant	*Salmonella* Singapore	2	D	Chow mein
		Private residence	*Salmonella* Typhimurium 135a	11	D	Unknown
		Commercial caterer	Unknown	6	D	Unknown
	May	Restaurant	Unknown	2	D	Chicken teriyaki sushi roll (nori roll)
	June	Restaurant	Unknown	3	D	Unknown
SA	May	Commercial manufactured food	*Salmonella* Typhimurium 108	23	AM	Ravioli
	June	Restaurant	*Salmonella* Typhimurium 9	6	A	Sweet potato and fetta cheese salad
Vic	May	Primary produce	*Salmonella* Oranienburg	15	M	Alfalfa
		Commercial manufactured food	*Salmonella* Bovismorbificans 11	13	M	Capocollo
		Commercial caterer	Suspected *Clostridium perfringens*	10	D	Unknown
	June	Aged care facility	Suspected *Clostridium perfringens*	5	D	Unknown
WA	June	National franchised fast food restaurant	*Salmonella* Anatum	6	D	Sandwiches/rolls

* No foodborne outbreaks were reported in the Australian Capital Territory or Tasmania during the quarter.

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

A Analytical epidemiological association between illness and one or more foods.

M Microbiological confirmation of agent in the suspect vehicle and cases.

Communicable diseases surveillance

Highlights for 2nd quarter, 2006

Communicable disease surveillance highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. These national data collections are complemented by intelligence provided by state and territory communicable disease epidemiologists and/or data managers. This additional information has enabled the reporting of more informative highlights each quarter.

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from state or territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

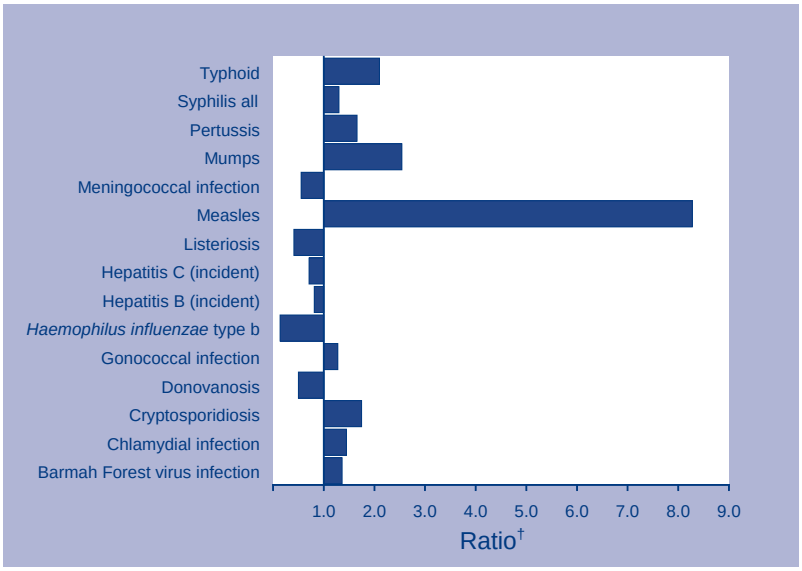
Figure 1 shows the changes in selected disease notifications with an onset in the second quarter of 2006, compared with the five year mean for the same period. The following diseases were above the five year mean: cryptosporidiosis, chlamydial infection, gonococcal infection, measles, mumps, pertussis, Barmah Forest virus infection, and typhoid. Diseases for which the number of notifications was below the five year mean for the same period included hepatitis B, hepatitis C, listeriosis, and meningococcal infection.

Gastrointestinal illnesses

Cryptosporidiosis

There were 919 notifications of cryptosporidiosis during the quarter which is 1.7 times the five year mean for the same period. All jurisdictions reported cases but the majority were from Victoria (360), Queensland (203) and New South Wales (182). This continued a trend reported in the first quarter. Four hundred and forty-seven (72%) notifications had information on the infecting species and all were identified as *Cryptosporidium parvum* infection.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 April to 31 June 2006 with historical data*



* Selected diseases are chosen each quarter according to current activity. Five year averages and the ratios of notifications in the reporting period in the five year mean should be interpreted with caution. Changes in surveillance practice, diagnostic techniques and reporting, may contribute to increases or decreases in the total notifications received over a five year period. Ratios are to be taken as a crude measure of current disease activity and may reflect changes in reporting rather than changes in disease activity.

† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

After observing a marked increase in the number of cryptosporidiosis notifications, the Communicable Disease Control Unit in Victoria attempted to gather risk factor information (using telephone interview or postal questionnaire) for all cases notified between 1 January and 31 May 2006. In the second quarter, a total of 14 swimming pools were associated with two or more confirmed cases of cryptosporidiosis. An additional outbreak was linked to a special school. Control measures for implicated pools included hyperchlorination and advice to facility managers about preventing contamination and control measures. Person-to-person spread was the suspected mode of transmission in the school and infection control advice was provided to the manager by environmental health officers, (Joy Gregory and James Fielding, personal communication).

Typhoid

There were 21 notifications of typhoid during the quarter which was 2.1 times the five year mean for the same period. Notifications were mainly from New South Wales (4), Victoria (5) and four each from Western Australia and Queensland. The imported status in the notifications showed 16 were imported from overseas, four were locally-acquired and one was unknown.

Sexually transmissible infections

Chlamydial infections

There were 11,192 notifications of chlamydial infection in the quarter which was 1.4 times the five year mean. The highest rates of notification continued to be in the 20–24 year age group, for both females (1,477 cases per 100,000 population) and males (846 cases per 100,000 population).

Gonococcal infections

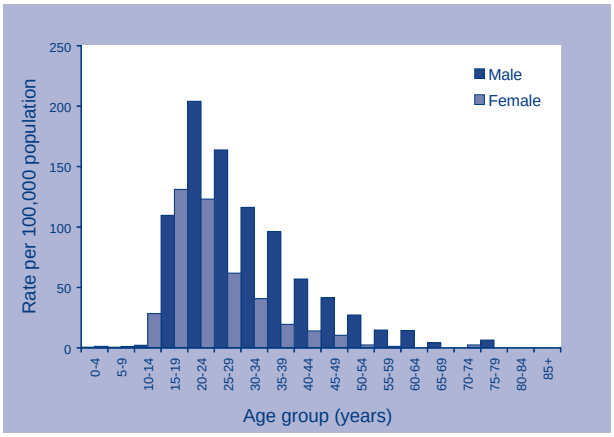
There were 2,294 notifications of gonococcal infection in the quarter which was 1.3 times the five year mean. There was a higher incidence in men, compared to women (2:1). The highest rates of notification were report in the 20–24 year age group for males (204 cases per 100,000 population) and the 15–19 year age group for females (131 cases per 100,000 population) (Figure 2).

Vaccine preventable diseases

Measles

There were 96 cases of measles reported in the quarter, which is 8.3 times the five year mean for the same period. Cases were reported from New South Wales (46 cases), Western Australia (25 cases), Tasmania (11), South Australia (8), Victoria (4) and one each in

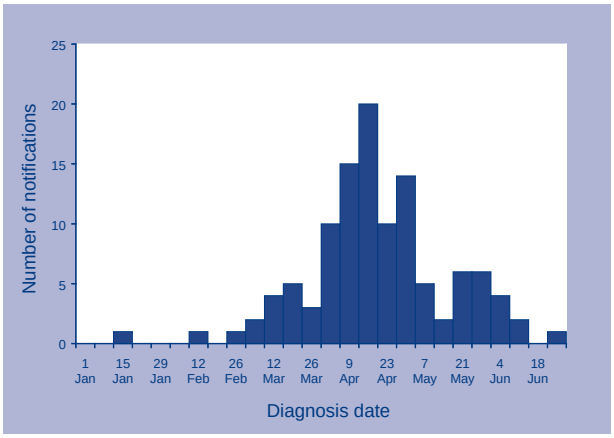
Figure 2. Notification rates of gonococcal infections, Australia, 1 April to 30 June 2006, by age group and sex



Queensland and the Australian Capital Territory. Of the 96 cases, 40 were male and 56 female; 23 were aged less than 5 years and the remainder were aged between 5 and 30 years. All of the children with vaccination status recorded were unvaccinated.

One national outbreak occurred during this quarter, with cases notified from all States and Territories except the Northern Territory. The outbreak began at Easter 2006 and subsided in June 2006 (Figure 3). Two smaller outbreaks occurred at the end of the 1st quarter.

Figure 3. Measles notifications, Australia, 1 January to 30 June 2006



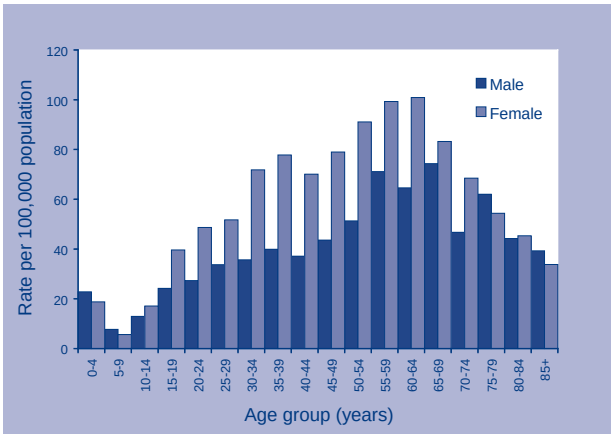
Mumps

There were 84 notifications of mumps in the quarter, which was 2.4 times the five year mean for the same period. There were 46 male and 42 female cases with an age range from 3 to 74 years.

Pertussis

There were 2,482 pertussis notifications were received in the quarter which was 1.7 times the five year mean for the same period. The majority of notifications were reported by New South Wales (1,143) and Queensland (486). Infants less than one year accounted for 1.4 per cent (35 cases) of the 2,482 notifications. The highest rate of infection in females occurred in the 60–64 year age group, (100 cases per 100,000 population). The highest rate in males was 74 cases per 100,000 population in the 65–69 year age group (Figure 4).

Figure 4. Notification rates of pertussis, Australia, 1 April to 30 June 2006, by age group and sex



Vectorborne diseases

Barmah Forest virus infection

There were 635 cases of Barmah Forest virus (BFV) infection in the quarter which was 1.4 times the five year mean for the same period. The majority of cases were from Queensland (288 cases) and New South Wales (218). Nationally, the infection rate was 13.8 cases per 100,000 population, but it was higher in the Northern Territory at 67.1 cases per 100,000 population (34 cases) and Queensland with 29.1 cases per 100,000 population.

Other bacterial infections

Meningococcal infection

There were 69 notifications of meningococcal infection in the quarter which was 0.6 times the five-year mean. Of the 69 cases, 47 (68%) were serogroup B, 5 (7%) were serogroup C, 2 were serogroup Y, 1 was serogroup A, and the serogroups of the remaining 14 cases was unknown. There were three deaths reported in the quarter, one each in patients with serogroup B, C and not typed.

Of the serotype C cases, one was aged less than one year and the remainder were aged 17 to 31 years. No cases were vaccinated.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 32,727 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 April and 30 June 2006 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

There were 3,519 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2006 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Smallpox	All jurisdictions
Tularemia	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (all)	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)*	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)†	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis virus	All jurisdictions
Kunjin virus	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses unspecified	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Laboratory confirmed influenza is not notifiable in South Australia but reports are forwarded to NNDSS.

† Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004.

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2006, by date of onset*

Disease	State or territory								Total 2nd quarter 2006†	Total 1st quarter 2006	Total 2nd quarter 2005	Last 5 years mean 2nd quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	0	14	2	13	1	1	27	16	74	67	59	90.8	141	178.2	0.8
Hepatitis B (unspecified)	30	835	70	257	59	14	377	76	1,718	1,652	1,611	1,643.6	3,370	3,262.0	1.0
Hepatitis C (incident)	5	12	1	0	9	0	30	29	86	120	99	121.4	206	252.0	0.7
Hepatitis C (unspecified)	52	1,392	57	756	99	67	622	196	3,241	3,884	3,102	3,633.2	7,125	7,513.4	0.9
Hepatitis D	0	6	0	0	0	0	0	0	6	9	4	6.6	15	11.4	0.9
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	1	0.3	0	1.0	0.0
Campylobacteriosis§	85	NN	63	919	473	119	1,242	383	3,284	3,636	3,530	3,417.8	6,920	7,466.2	1.0
Cryptosporidiosis‖	36	182	12	203	66	8	370	42	919	1,510	828	525.6	2,429	1,514.2	1.7
Haemolytic uraemic syndrome	0	0	0	0	0	0	0	0	0	5	4	2.4	5	5.6	0.0
Hepatitis A	0	25	7	3	3	0	8	12	58	105	87	97.8	163	209.6	0.6
Hepatitis E	0	1	0	1	1	0	1	0	4	7	7	4.6	11	12.2	0.9
Listeriosis	0	5	0	0	0	0	1	1	7	25	14	17.0	32	34.2	0.4
Salmonellosis (NEC)	25	406	108	667	141	47	302	168	1,864	3,099	1,957	1,842.8	4,963	4,537.8	1.0
Shigellosis	1	16	33	23	7	1	20	31	132	188	180	140.0	320	314.0	0.9
SLTEC, VTEC¶	0	3	0	4	8	0	0	2	17	17	32	15.0	34	31.2	1.1
Typhoid	0	7	1	4	0	0	5	4	21	23	10	10.0	44	37.6	2.1
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	1	1.0	0	2.0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2006, by date of onset,* *continued*

Disease	State or territory								Total 2nd quarter 2006†	Total 1st quarter 2006	Total 2nd quarter 2005	Last 5 years mean 2nd quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection**	199	2,835	515	2,811	805	215	2,459	1,353	11,192	11,698	10,909	7,730.6	22,890	15,368.0	1.4
Donovanosis	0	0	1	1	0	0	0	0	2	0	2	4.0	2	9.6	0.5
Gonococcal infection	7	398	512	387	191	10	338	451	2,294	2,346	2,059	1,794.8	4,640	3,571.2	1.3
Syphilis (all)	1	233	90	91	15	6	153	21	610	610	590	538.4	1,227	621.6	1.3
Syphilis < two years duration	0	21	42	38	1	0	62	8	172	158	161	154.0	330	286.5	1.1
Syphilis >two years or unspecified duration	1	212	48	53	14	6	91	13	438	452	429	400.0	897	745.5	1.1
Syphilis - congenital	0	2	3	0	0	0	0	0	5	3	6	4.8	8	8.0	1.0
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Haemophilus influenzae type b	0	1	0	0	0	0	0	0	1	2	1	7.2	3	12.4	0.1
Influenza (laboratory confirmed)‖	8	57	5	151	8	2	111	27	369	179	796	378.0	548	530.8	1.0
Measles	1	46	0	1	8	11	4	25	96	16	2	11.6	112	35.0	8.3
Mumps	0	50	1	22	4	0	6	5	88	42	74	34.6	130	64.2	2.5
Pertussis	77	1,143	21	486	484	5	220	46	2,482	2,418	2,556	1,496.8	4,900	3,000.0	1.7
Pneumococcal disease (invasive)‖	7	166	10	62	26	6	74	38	389	208	459	543.4	597	830.2	0.7
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	11	0	4	0	0	2	0	17	7	14	28.6	24	60.8	0.6
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.6	0.0
Tetanus	0	0	0	0	0	0	0	0	0	1	1	0.6	1	2.4	0.0
Vectorborne diseases															
Barmah Forest virus infection	1	218	34	288	45	0	10	39	635	731	438	466.0	1,366	789.2	1.4
Dengue	2	10	3	35	3	0	1	4	58	60	43	92.2	118	247.4	0.6
Flavivirus infection (NEC)	0	0	0	9	0	0	0	0	9	21	8	16.2	30	37.8	0.6
Japanese encephalitis virus‖	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.4	0.0
Kunjin virus‖	0	0	0	3	0	0	0	1	4	1	0	1.4	5	6.4	2.9
Malaria	4	29	18	91	6	10	28	22	208	215	168	152.2	423	361.8	1.4
Murray Valley encephalitis virus‖	0	0	0	0	0	0	0	1	1	0	0	0.0	1	2.0	0.0
Ross River virus infection	0	294	28	574	28	5	14	154	1,097	3,345	556	1,200.2	4,442	2,469.2	0.9

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2006, by date of onset,* *continued*

Disease	State or territory								Total 2nd quarter 2006†	Total 1st quarter 2006	Total 2nd quarter 2005	Last 5 years mean 2nd quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‖	0	0	0	0	0	0	0	0	0	1	0	0.0	1	0.0	0.0
Australian bat lyssavirus‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	0	0	4	0	0	1	0	5	18	4	6.0	23	14.4	0.8
Leptospirosis	0	5	0	52	1	0	0	0	58	49	36	48.0	107	111.0	1.2
Lyssavirus unspecified‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	1	19	0	1	0	0	14	1	36	44	53	49.2	80	89.8	0.7
Q fever	1	33	2	25	6	0	3	0	70	97	116	148.4	167	307.8	0.5
Other bacterial infections															
Legionellosis	0	24	2	8	13	1	13	11	72	98	77	89.2	170	166.2	0.8
Leprosy	0	0	0	0	0	0	0	1	1	1	1	1.2	2	4.6	0.8
Meningococcal infection††	0	18	2	16	6	2	21	4	69	74	74	124.6	143	227.6	0.6
Tuberculosis	3	98	6	34	15	4	76	34	270	293	268	239.0	563	478.6	1.1
Total	553	8,594	1,637	8,764	2,874	553	6,553	3,198	32,726	38,116	30,839	26,757.5	70,842	55,233.0	1.2

* Date of onset = the true onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified were analysed by the date of notification.

† Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‖ Notifiable from January 2001 only. Ratio and mean calculations are based on the last five years.

¶ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

** Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory which excludes ocular specimens, and Western Australia which excludes ocular and perinatal infections.

†† Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

Note Ratios for Syphilis < 2 years; syphilis >2 years or unspecified duration based on 2 years data

NN Not notifiable.

NEC Not elsewhere classified.

Table 3. Notification rates of diseases, 1 April to 30 June 2006, by state or territory. (Rate per 100,000 population)

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	0.0	0.8	3.9	1.3	0.3	0.8	2.2	3.2	1.5
Hepatitis B (unspecified)	36.9	49.3	138.1	25.9	15.3	11.5	30.0	15.1	33.8
Hepatitis C (incident)	6.2	0.7	2.0	0.0	2.3	0.0	2.4	5.8	1.7
Hepatitis C (unspecified)	64.0	82.2	112.4	76.3	25.7	55.2	49.5	39.0	63.8
Hepatitis D	0.0	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	104.6	NN	124.3	92.7	122.7	98.1	98.9	76.2	96.9
Cryptosporidiosis	44.3	10.7	23.7	20.5	17.1	6.6	29.5	8.4	18.1
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.0	1.5	13.8	0.3	0.8	0.0	0.6	2.4	1.1
Hepatitis E	0.0	0.1	0.0	0.1	0.3	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.3	0.0	0.0	0.0	0.0	0.1	0.2	0.1
Salmonellosis (NEC)	30.8	24.0	213.0	67.3	36.6	38.7	24.1	33.4	36.7
Shigellosis	1.2	0.9	65.1	2.3	1.8	0.8	1.6	6.2	2.6
SLTEC, VTEC‡	0.0	0.2	0.0	0.4	2.1	0.0	0.0	0.4	0.3
Typhoid	0.0	0.4	2.0	0.4	0.0	0.0	0.4	0.8	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible infections									
Chlamydial infection§	244.8	167.4	1,015.8	283.7	208.8	177.2	195.8	269.2	220.3
Donovanosis	0.0	0.0	2.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	8.6	23.5	1,009.9	39.1	49.5	8.2	26.9	89.7	45.1
Syphilis (all)	1.2	13.8	177.5	9.2	3.9	4.9	12.2	4.2	12.0
Syphilis <2 years duration	0.0	1.2	82.8	3.8	0.3	0.0	4.9	1.6	3.4
Syphilis >2 years or unspecified duration	1.2	12.5	94.7	5.3	3.6	4.9	7.2	2.6	8.6
Syphilis - congenital	0.0	0.1	5.9	0.0	0.0	0.0	0.0	0.0	0.1

Table 3. Notification rates of diseases, 1 April to 30 June 2006, by state or territory. (Rate per 100,000 population), *continued*

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Influenza (laboratory confirmed)	9.8	3.4	9.9	15.2	2.1	1.6	8.8	5.4	7.3
Measles	1.2	2.7	0.0	0.1	2.1	9.1	0.3	5.0	1.9
Mumps	0.0	3.0	2.0	2.2	1.0	0.0	0.5	1.0	1.7
Pertussis	94.7	67.5	41.4	49.0	125.5	4.1	17.5	9.2	48.8
Pneumococcal disease (invasive)	8.6	9.8	19.7	6.3	6.7	4.9	5.9	7.6	7.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.6	0.0	0.4	0.0	0.0	0.2	0.0	0.3
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Barmah Forest virus infection	1.2	12.9	67.1	29.1	11.7	0.0	0.8	7.8	12.5
Dengue	2.5	0.6	5.9	3.5	0.8	0.0	0.1	0.8	1.1
Flavivirus infection (NEC)	0.0	0.0	0.0	0.9	0.0	0.0	0.0	0.0	0.2
Japanese encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.2	0.1
Malaria	4.9	1.7	35.5	9.2	1.6	8.2	2.2	4.4	4.1
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Ross River virus infection	0.0	17.4	55.2	57.9	7.3	4.1	1.1	30.6	21.6
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.3	0.0	5.2	0.3	0.0	0.0	0.0	1.1
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	1.2	1.1	0.0	0.1	0.0	0.0	1.1	0.2	0.7
Q fever	1.2	1.9	3.9	2.5	1.6	0.0	0.2	0.0	1.4
Other bacterial infections									
Legionellosis	0.0	1.4	3.9	0.8	3.4	0.8	1.0	2.2	1.4
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Meningococcal infection‖	0.0	1.1	3.9	1.6	1.6	1.6	1.7	0.8	1.4
Tuberculosis	3.7	5.8	11.8	3.4	3.9	3.3	6.1	6.8	5.3

* Rates are subject to retrospective revision.

† Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‡ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory which excludes ocular specimens, and Western Australia which excludes ocular and perinatal infections.

|| Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable.

NEC Not elsewhere classified.

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2006, and total reports for the year†

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	23	0	0	9	6	3	0	41	1	52	3
Mumps virus	1	0	0	4	0	0	6	0	11	12	23	18
Rubella virus	0	0	0	1	0	0	3	0	6	3	8	7
Hepatitis viruses												
Hepatitis A virus	0	0	1	1	2	0	1	0	5	11	16	17
Hepatitis D virus	0	0	0	1	1	0	0	0	2	4	4	6
Hepatitis E virus	0	0	0	0	0	0	1	0	1	3	4	9
Arboviruses												
Ross River virus	0	9	4	149	15	0	2	9	188	74	976	282
Barmah Forest virus	0	5	0	61	7	0	4	0	77	76	221	130
Flavivirus (unspecified)	0	0	0	9	0	0	0	0	9	7	39	20
Adenovirus not typed/ pending	0	36	0	8	9	0	1	0	65	172	218	285
Herpesviruses												
Herpes virus type 6	0	0	0	0	0	0	1	0	1		2	1
Cytomegalovirus	0	60	0	31	51	6	12	0	160	240	485	411
Varicella-zoster virus	1	32	1	165	41	0	6	0	246	366	598	730
Epstein-Barr virus	0	4	22	133	33	5	6	68	271	412	814	981
Other DNA viruses												
Parvovirus	0	0	0	16	6	0	9	0	31	24	82	79
Picornavirus family												
Coxsackievirus A9	0	3	0	0	0	0	0	0	3	1	5	2
Coxsackievirus A16	0	2	0	0	0	0	0	0	2	3	2	3
Echovirus type 11	0	1	0	0	0	0	0	0	1	2	1	3
Echovirus type 18	0	1	0	0	0	0	0	0	1	3	1	10
Echovirus type 22	0	1	0	0	0	0	0	0	2	1	4	1
Echovirus type 30	0	4	0	0	0	0	0	0	4	10	15	19
Rhinovirus (all types)	0	9	0	0	1	0	0	0	11	89	28	171
Enterovirus not typed/ pending	2	16	0	4	0	0	0	0	22	41	76	65
Ortho/paramyoviruses												
Influenza A virus	0	0	0	3	6	0	2	0	11	96	40	120
Influenza B virus	0	2	0	1	5	0	6	0	14	49	20	82
Parainfluenza virus type 1	0	12	0	0	6	0	2	0	22	19	43	29
Parainfluenza virus type 2	0	3	0	1	0	0	0	0	5	27	6	33
Parainfluenza virus type 3	0	4	0	0	0	0	0	0	4	53	16	99
Respiratory syncytial virus	0	110	0	62	3	2	8	0	232	720	333	832
Other RNA viruses												
Rotavirus	0	22	0	0	7	5	1	0	44	170	107	239
Norwalk agent	0	0	0	0	0	0	273	0	273	80	463	95

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2005, and total reports for the year,† *continued*

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	5	212	1	483	209	13	8	0	938	1,348	2,415	2,539
Chlamydia psittaci	0	3	0	0	0	0	13	0	16	16	26	30
Chlamydia species	0	0	0	0	0	0	1	0	1		1	
Mycoplasma pneumoniae	0	10	3	128	22	4	66	18	251	244	596	502
Coxiella burnetii (Q fever)	0	0	2	9	5	0	4	0	20	52	68	87
Rickettsia tsutsugamushi	0	0	0	0	3	0	0	0	3	8	20	19
Rickettsia - spotted fever group	0	0	0	0	5	0	0	0	5	48	62	97
Streptococcus group A	0	1	0	104	0	0	22	0	127	138	264	242
Yersinia enterocolitica	0	1	0	0	0	0	0	0	1	2	4	6
Brucella species	0	0	0	1	0	0	0	0	1	1	3	3
Bordetella pertussis	0	12	0	55	65	0	27	0	162	365	591	751
Legionella pneumophila	0	3	0	0	1	0	3	0	7	7	15	14
Legionella longbeachae	1	0	0	0	1	0	0	0	2	7	10	19
Cryptococcus species	0	0	0	3	0	0	0	0	3	15	13	25
Leptospira species	0	0	0	4	0	0	0	0	4	13	11	16
Treponema pallidum	0	30	0	129	25	0	1	0	198	329	473	581
Toxoplasma gondii	0	4	0	3	2	1	5	0	15	10	31	20
Total	10	635	34	1,569	540	42	497	95	3,519	5,372	9,305	9,733

* State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

† Data presented are for reports with reports dates in the current period.

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 April to 30 June 2006*

State or territory	Laboratory	April 2006	May 2006	June 2006	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	126	126	108	360
	New Children's Hospital, Westmead	64	60	–	124
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	25	8	–	33
	South West Area Pathology Service, Liverpool	49	60	50	165
Queensland	Queensland Medical Laboratory, West End	359	739	540	1,638
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	540	–	–	540
Tasmania	Northern Tasmanian Pathology Service, Launceston	11	15	6	32
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	7	13	–	20
	Royal Children's Hospital, Melbourne	13	25	21	59
	Victorian Infectious Diseases Reference Laboratory, Fairfield	161	180	83	424
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	33	91	–	124
Total		1,388	1,323	808	3,519

* The complete list of laboratories reporting for the 12 months, January to December 2005, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health setting and to detect trends in consultation rates.

There are currently about 40 general practitioners participating in the network from all states and territories. Seventy-five per cent of these are in metropolitan areas and the remainder are rural based. Between 3,000 and 4,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published.

In 2006, six conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. Definitions of these conditions were published in Commun Dis Intell *2006;30:158.*

Data from 1 January to 30 June 2006 compared with 2005 are shown as the rate per 1,000 consultations in Figures 5 and 6.

Figure 5. Consultation rates for influenza-like illness, ASPREN, 1 January to 30 June 2006, by week of report

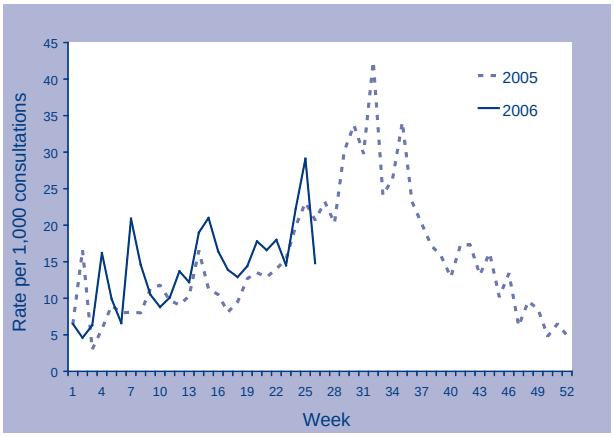
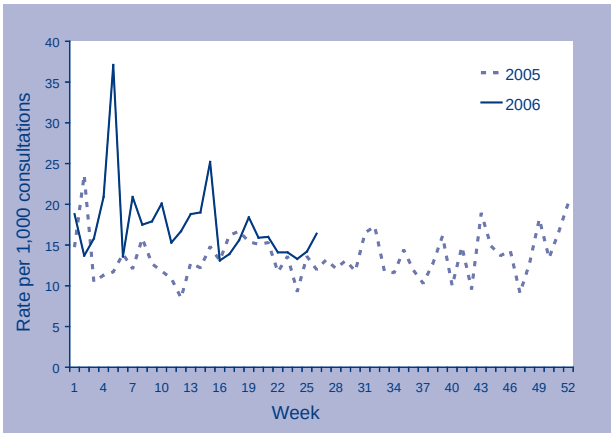


Figure 6. Consultation rates for gastroenteritis, ASPREN, 1 January to 30 June 2006, by week of report



Childhood immunisation coverage

Tables 6, 7 and 8 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 January and 31 March 2005, at 24 months of age for the cohort born between 1 January and 31 March 2004, and at 6 years of age for the cohort born between 1 January and 31 March 2000 according to the Australian Standard Vaccination Schedule.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2006;30:157 and for a full description of the methodology used by the Register see* Commun Dis Intell *1998;22:36-37.*

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1435, Email: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally by 0.5 percentage points to 90.7 per cent (Table 6), whilst coverage for all individual vaccines due at 12 months of age also increased by 0.4–0.5 percentage points. The only significant movements in coverage for individual vaccines by jurisdiction was in Tasmania, where coverage for all four vaccines due at 12 months increased by 2.5–2.8 percentage points.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia also increased marginally from the last quarter by 0.3 percentage points to 92.4 per cent (Table 7). There were no significant changes in coverage in any jurisdiction for 'fully immunised' coverage or for coverage for individual vaccines. It is notable that the estimate for 'fully immunised' at 24 months of age has been higher than the 12 months coverage estimate since the 18 month DTPa booster was no longer required from September 2003.

It is also notable that, for the two vaccines where no further doses are due between 6 months and 24 months of age (DTP and polio), coverage at the national level was 95.2 per cent and 95.2 per cent respectively at 24 months versus 92.2 and 92.1 per cent at 12 months. This suggests that delayed notification or delayed vaccination is making an important contribution to the coverage estimates at 12 months of age and that the 'fully immunised' estimate in particular is likely to be a minimum estimate.

Table 8 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at 6 years of age for Australia and by state or territory. Surprisingly, 'fully immunised' coverage for Australia decreased 1.1 percentage points and is the lowest it's been since early 2003. Coverage decreased in almost all jurisdictions except in the Northern Territory where it increased by 2.6 percentage points. Victoria and Western Australia experienced the most significant decreases, 2 and 1.8 percentage points respectively. It appears that the driver of this decrease is a drop in coverage for polio vaccine, which mirrored the decrease in 'fully immunised' coverage. A change in the immunisation schedule occurred in November 2005, with oral polio vaccine replaced with the injectable inactivated poliovirus vaccine, together with DTPa. It is possible that this change may have been associated with problems in completing encounter forms.

Figure 7 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 6 years, although the rate of increase has slowed over the past two years for all age groups. The Figure shows that there have now been 11 consecutive quarters where 'fully immunised' coverage at 24 months of age has been greater than 'fully immunised' coverage at 12 months of age, following the removal of the requirement for 18 month DTPa vaccine. However, both measures have been above 90 per cent for this 30-month period and show levels of high coverage for the vaccines included maintained over a significant period of time. Currently, coverage for the more recent vaccines, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months, are not included in the 12 or 24 months coverage data respectively.

Figure 7. Trends in vaccination coverage, Australia, 1997 to 2005, by age cohorts

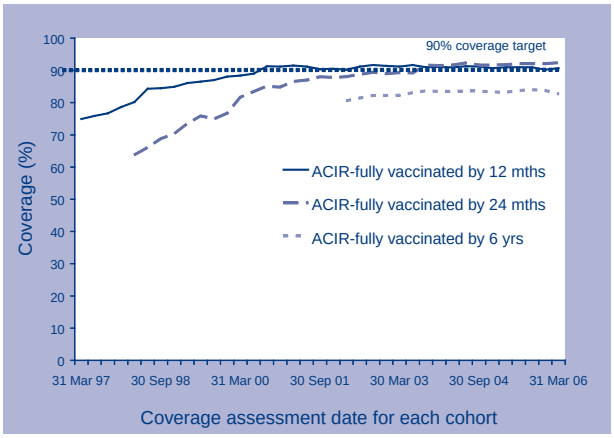


Table 6. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2005; assessment date 30 June 2006

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,058	22,163	863	13,601	4,474	1,376	15,462	6,612	65,609
Diphtheria, tetanus, pertussis (%)	91.4	91.8	91.4	92.1	92.2	95.6	93.4	90.6	92.2
Poliomyelitis (%)	91.4	91.7	91.1	92.0	91.9	95.4	93.3	90.5	92.1
Haemophilus influenzae type b (%)	93.7	93.5	94.9	94.2	94.8	96.1	94.9	93.7	94.2
Hepatitis B (%)	93.8	94.8	95.5	94.6	95.3	96.0	94.8	93.5	94.7
Fully immunised (%)	90.7	90.1	90.6	90.8	91.0	93.8	91.8	89.1	90.7
Change in fully immunised since last quarter (%)	-1.4	+0.1	-0.9	+0.5	+0.4	+2.6	+1.5	-0.2	+0.5

Table 7. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2004; assessment date 30 June 2006*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,053	21,756	900	13,417	4,513	1,367	15,813	6,600	65,419
Diphtheria, tetanus, pertussis (%)	96.8	95.0	97.3	94.9	94.7	96.3	96.0	94.4	95.2
Poliomyelitis (%)	96.8	94.9	97.3	94.9	94.8	96.4	95.9	94.4	95.2
Haemophilus influenzae type b (%)	95.2	93.3	95.0	93.9	93.5	95.3	94.6	92.7	93.8
Measles, mumps, rubella (%)	95.3	93.4	95.7	93.8	94.1	95.0	95.0	93.1	94.0
Hepatitis B(%)	97.3	95.7	97.8	95.5	95.5	97.0	96.4	95.2	95.8
Fully immunised (%)	94.2	91.7	94.4	92.2	92.2	93.6	93.5	91.3	92.4
Change in fully immunised since last quarter (%)	+2.1	+0.1	+0.1	+0.4	+1.4	-0.8	+0.3	+1.2	+0.3

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2005;29:329.

Table 8. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2000; assessment date 30 June 2006

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,014	22,676	879	13,812	4,682	1,561	16,007	6,800	67,431
Diphtheria, tetanus, pertussis (%)	85.9	85.5	86.3	84.4	83.5	84.2	87.5	79.9	85.0
Poliomyelitis (%)	85.1	84.1	86.2	83.2	83.1	83.8	85.9	78.7	83.8
Measles, mumps, rubella (%)	84.8	85.2	86.2	84.5	83.6	84.1	87.6	79.9	85.0
Fully immunised (%)[1]	83.2	83.0	84.6	81.8	82.4	82.6	85.1	77.3	82.7
Change in fully immunised since last quarter (%)	-3.8	-1.0	+2.6	-0.0	-0.2	-1.0	-2.0	-1.7	-1.1

Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick NSW 2031 for the Australian Gonococcal Surveillance Programme.

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2006;30:157.*

Reporting period 1 January to 31 March 2006

The AGSP laboratories received a total of 1,110 isolates in this quarter of which 1,089 underwent susceptibility testing. This is slightly more than the 985 reported in the first quarter of 2005. A total of 1,001 isolates were received for the same period in 2004 and 1,051 in 2003. About 31 per cent of this total was from New South Wales, 26 per cent from Victoria, 13 per cent from each of Queensland and the Northern Territory, 9 per cent from Western Australia and 5 per cent from South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter 366 (33.6%) of all isolates examined were penicillin resistant by one or more mechanisms. Ninety (8.3%) were penicillinase producing (PPNG) and 276 (25.3%) resistant by chromosomal mechanisms, (CMRNG). The proportion of all strains resistant to the penicillins by any mechanism ranged from 3.4 per cent in the Northern Territory to 51 per cent in New South Wales.

Figure 8 shows the proportions of gonococci fully sensitive (MIC ≤0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥1 mg/L) or else penicillinase producing aggregated for Australia and by state or territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

The highest number of PPNG was found in Victoria where the 32 PPNG were 10.9 per cent of all isolates. Thirteen PPNG representing 13.5 per cent of all isolates were found in Western Australia, 18 (12.5%) in Queensland and 20 (5.8%) in New South Wales. Five PPNG were found in the Northern Territory. South Australia was the only jurisdiction with no PPNG. More isolates were resistant to the penicillins by separate chromosomal mechanisms and CMRNG notably increased in both New South Wales (156 isolates, 45.5% of all gonococci tested, double the 2005 number and proportion) and Victoria (86 isolates, 29.4%, twice the number in 2005). Increases in CMRNG were also noted in Queensland over the equivalent period in 2005 (to 13 from 5 and 10.4% from 2.8% of isolates) and Western Australia (10, 10.4%) and eight (14%) in South Australia. CMRNG were reported from Tasmania and the Australian Capital Territory, but not the Northern Territory.

Figure 8. Categorisation of gonococci isolated in Australia, 1 January to 31 March 2006, by penicillin susceptibility and region

FS Fully sensitive to penicillin, MIC ≤0.03 mg/L.

LS Less sensitive to penicillin, MIC 0.06–0.5 mg/L.

RR Relatively resistant to penicillin, MIC ≥1 mg/L.

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

Ceftriaxone

Seven isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected; five in New South Wales and two in Queensland. Fifteen strains of this type were found in this period in 2005.

Spectinomycin

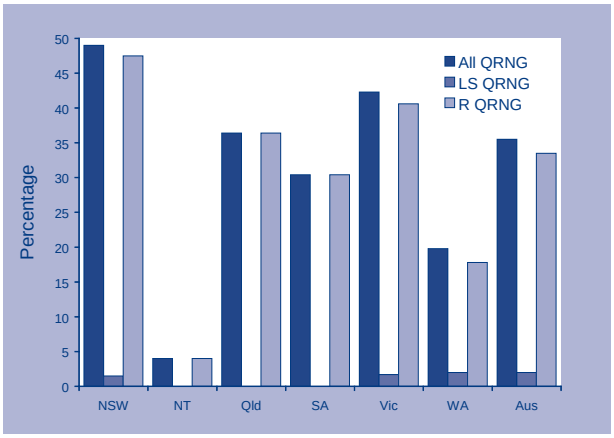
All isolates susceptible to this injectable agent.

Quinolone antibiotics

The total number (387) and proportion (35.5%) of quinolone resistant *N. gonorrhoeae* (QRNG) were both substantially higher than the corresponding figures in the first quarter of 2005 (283 QRNG, 29.7%), 2004 (188 QRNG, 20.5%) and 2003 (108 isolates, 11.5%). The majority of QRNG (375 of 387, 97%) exhibited higher-level resistance. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥1 mg/L) groups.

QRNG were again widely distributed and were detected in all jurisdictions (Figure 9). The highest number and proportion of QRNG was found in New South Wales where 168 QRNG represented 49 per cent of isolates. In Victoria there were 124 QRNG (42.3% of isolates), in Queensland 52 (36.4%), in South Australia 17 (30.4%) and in Western Australia 19 (19.6%). Six QRNG were detected in the Northern Territory and two each in Tasmania and in the Australian Capital Territory. These numbers represent increases, sometimes considerable, in all states and territories, except for Victoria where numbers decreased.

Figure 9. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2006 , by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

Nationally the number (115) and proportion (10.6%) of high level tetracycline resistant *Neisseria gonorrhoeae* (TRNG) detected decreased when compared with 2005 data (145 QRNG, 15.5%) but approximated the 2004 (107, 11.7%) figures. TRNG were found in all states and territories.

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Meningococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Meningococcal Surveillance Programme.

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence. For more information see Commun Dis Intell *2006;30:157.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2006, are included in this issue of Communicable Diseases Intelligence *(Table 9).*

Table 9. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2006, by jurisdiction and serogroup

Jurisdiction	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	06					1	1							1	1
	05			1	2	1	2							2	4
	04			0	4	2	4							2	8
New South Wales	06			13	22	1	2	1	1	0	1	0	3	15	29
	05			17	33	2	9	2	3	3	3	0	1	24	49
	04			22	37	5	9	1	2	2	2	5	11	37	61
Northern Territory	06			1	2									1	2
	05			2	3	2	2							4	5
	04			1	6	1	1			1	1			3	8
Queensland	06	2	2	10	25	3	4							15	31
	05			12	21	2	6							14	27
	04	1	1	11	23	5	12	1	1	1	1	6	8	19	40
South Australia	06			3	6			1	1					4	7
	05			4	4	0	2							4	6
	04			5	9									5	9
Tasmania	06			2	3	0	1							2	4
	05			2	2									2	2
	04			0	2					1	1	1	3	2	6
Victoria	06			19	29	0	2	0	1	0	2			19	34
	05	1	1	8	15	2	3			0	2	0	1	11	22
	04			18	28	9	9	1	3			1	2	25	42
Western Australia	06			4	9									4	9
	05			4	9			1	2					5	11
	04			8	12	1	3							9	14
Total	06	2	2	52	96	5	10	2	3	0	3	0	3	61	117
	05	1	1	50	89	9	24	3	5	3	5	0	2	66	126
	04	1	1	65	121	19	37	4	6	5	5	8	18	102	188

Q2 = 2nd quarter.

YTD = Year to 30 June 2006.

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2006, as reported to 30 June 2006, are included in this issue of Communicable Diseases Intelligence *(Tables 10 and 11).*

Table 10. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 January to 31 March 2006, by sex and state or territory of diagnosis

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2005	This period 2004	YTD 2005	YTD 2004
HIV diagnoses	Female	2	17	0	3	3	0	4	7	36	26	36	26
	Male	2	94	3	24	13	1	64	6	207	204	207	204
	Not reported	0	1	0	0	0	0	0	0	1	0	1	0
	Total*	4	112	3	27	16	1	68	13	244	230	244	230
AIDS diagnoses	Female	0	1	0	0	0	0	0	1	2	8	2	8
	Male	0	19	1	0	1	0	13	0	34	43	34	43
	Total*	0	20	1	0	1	0	13	1	36	51	36	51
AIDS deaths	Female	0	1	0	2	0	0	1	0	4	2	4	2
	Male	0	4	0	2	1	0	2	0	9	14	9	14
	Total*	0	5	0	4	1	0	3	0	13	16	13	16

* Totals include people whose sex was reported as transgender.

Table 11. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2005, and reported by 30 June 2006, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	32	834	18	247	94	8	344	189	1,766
	Male	257	13,194	128	2,613	896	96	5,058	1,167	23,409
	Not reported	0	231	0	0	0	0	22	0	253
	Total*	289	14,288	146	2,869	991	104	5,444	1,363	25,494
AIDS diagnoses	Female	10	245	3	68	31	4	105	37	503
	Male	93	5,324	42	1,010	394	50	1,939	419	9,271
	Total*	103	5,586	45	1,080	426	54	2,054	458	9,806
AIDS deaths	Female	7	135	1	41	20	2	60	24	290
	Male	73	3,560	26	654	274	32	1,387	292	6,298
	Total*	80	3,705	27	697	294	34	1,455	317	6,609

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella*. NEPSS receives reports of* Salmonella *isolates that have been serotyped and phage typed by the six* Salmonella *laboratories in Australia.* Salmonella *isolates are submitted to these laboratories for typing by primary diagnostic laboratories throughout Australia.*

A case is defined as the isolation of a Salmonella *from an Australian resident, either acquired locally or as a result of overseas travel, including isolates detected during immigrant and refugee screening. Second and subsequent identical isolates from an individual within six months are excluded, as are isolates from overseas visitors to Australia. The date of the case is the date the primary diagnostic laboratory isolated* Salmonella *from the clinical sample.*

Quarterly reports include historical quarterly mean counts. These should be interpreted cautiously as they may be affected by outbreaks and by surveillance artefacts such as newly recognised and incompletely typed Salmonella*.*

NEPSS may be contacted at the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; by telephone: +61 3 8344 5701, facsimile: +61 3 8344 7833 or email joanp@unimelb.edu.au

Scientists, diagnostic and reference laboratories contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 April to 30 June 2006 are included in Tables 12 and 13. Data include cases reported and entered by 20 July 2006. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2006;30:159–160.*

Second quarter 2006

There were 1,663 reports to NEPSS of human *Salmonella* infection in the second quarter of 2006, 43 per cent less than in first quarter of 2006. This decline after the summer peak is typical of seasonal trends in the incidence of salmonellosis in Australia. The second quarter count was nine per cent less than the comparable second quarter of 2005 and close to the 10-year historical mean for this period.

During the second quarter of 2006, the 25 most common *Salmonella* types in Australia accounted for 1,057 cases, 64 per cent of all reported human *Salmonella* infections. Twenty-two of the 25 most common *Salmonella* infections in the second quarter of 2006 were also among the 25 most commonly reported in preceding quarter.

The recent occurrence of particular *Salmonella* serovars and phage types reflects the established distribution and incidence of various common endemic strains, and the abatement of various local and widespread outbreaks of the last Australian summer.

The most common *Salmonella* was *S.* Typhimurium phage type 135. This historically common phage type caused widespread outbreaks in late 2005 and early 2006. *S.* Saintpaul was typically common in Queensland with an increase in cases reported from Western Australia and Victoria. A moderate increase in cases of *S.* Birkenhead, concentrated in southern Queensland and northern New South Wales, contributed to the prominence of this serovar. *S.* Typhimurium phage type 170 remains common, albeit somewhat less so than in 2004 and 2005.

S. Waycross, *S.* Weltevreden and *S.* Javiana were reported more frequently than expected (all mostly in Queensland), as were *S.* Typhimurium 44 and *S.* Oranienburg (both in Victoria).

Acknowledgement: We thank scientists, contributing laboratories, state and territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 April to 30 June 2006, as reported to 20 July 2006

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	20	366	48	618	60	49	335	167	1,663
Total contributing *Salmonella* types	17	106	25	111	36	14	90	68	211

Table 13. Top 25 *Salmonella* types identified in Australia, 1 April to 30 June 2006, by state or territory

National rank	*Salmonella* type	State or territory								Total 2nd quarter 2006	Last 10 years mean 2nd quarter	Year to date 2006	Year to date 2005
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 135	1	42	0	46	2	19	29	10	149	133	412	240
2	*S.* Saintpaul	1	3	2	49	2	0	14	24	95	90	261	265
3	*S.* Birkenhead	0	28	0	52	0	0	0	1	81	59	192	130
4	*S.* Typhimurium PT 170	2	41	0	7	0	2	24	2	78	65	244	352
5	*S.* Virchow PT 8	0	4	1	62	0	1	2	1	71	56	182	164
6	*S.* Typhimurium PT 9	1	19	0	8	2	3	23	1	57	118	215	274
7	*S.* Waycross	0	9	1	38	0	0	0	0	48	30	108	78
8	*S.* Muenchen	0	9	4	19	3	0	3	7	45	35	100	102
9	*S.* Aberdeen	0	3	0	40	0	1	0	0	44	33	111	111
10	*S.* Infantis	0	13	2	2	10	0	9	4	40	32	108	94
11	*S.* Typhimurium PT 44	0	3	0	2	3	0	28	2	38	12	115	6
12	*S.* Hvittingfoss	1	3	1	17	0	0	6	4	32	31	97	135
13	*S.* Typhimurium PT 12	0	15	0	2	1	0	4	8	30	20	70	77
14	*S.* Chester	0	5	2	9	3	1	2	7	29	41	92	119
15	*S.* Oranienburg	1	2	1	3	1	1	16	4	29	18	112	26
16	*S.* Weltevreden	0	3	3	20	0	0	1	0	27	10	57	29
17	*S.* Anatum	0	1	0	10	1	0	2	10	24	24	75	37
18	*S.* Typhimurium PT RDNC	0	8	1	7	1	2	4	1	24	19	63	57
19	*S.* Typhimurium PT 197	0	4	0	8	0	0	7	1	20	18	61	455
20	*S.* Typhimurium untypable	1	4	0	3	0	0	4	6	18	17	47	38
21	*S.* Stanley	0	10	0	2	0	0	4	1	17	11	42	36
22	*S.* Virchow PT 25 var 1	0	2	0	15	0	0	0	0	17	1.6	44	22
23	*S.* Javiana	0	5	0	11	0	0	0	0	16	7	25	22
24	*S.* Mississippi	0	0	0	0	0	9	4	1	14	18	69	49
25	*S.* Bovismorbificans PT 11	0	0	0	0	0	0	14	0	14	0.2	21	2

Overseas briefs

For the period 1 April to 30 June 2006

World Health Organization Disease Outbreak News

This material has been summarised from information provided by the World Health Organization (http://www.who.int).

Avian influenza – epidemiology of human H5N1 cases reported to WHO

30 June 2006

This week's issue of the *Weekly Epidemiological Record,*[1] published online by the World Health Organization (WHO), sets out results from the first analysis of epidemiological data on all 205 laboratory-confirmed H5N1 cases officially reported to WHO by onset date from December 2003 to 30 April 2006.

- Data used in the analysis were collected for surveillance purposes. Quality, reliability and format were not consistent across data from different countries. Despite this limitation, several conclusions could be reached.
- The number of new countries reporting human cases increased from four to nine after October 2005, following the geographical extension of outbreaks among avian populations.
- Half of the cases occurred in people under the age of 20 years; 90 per cent of cases occurred in people under the age of 40 years.
- The overall case-fatality rate was 56 per cent. Case fatality was high in all age groups but was highest in persons aged 10 to 39 years.
- The case-fatality profile by age group differs from that seen in seasonal influenza, where mortality is highest in the elderly.
- The overall case fatality rate was highest in 2004 (73%), followed by 63 per cent to date in 2006, and 43 per cent in 2005.
- Assessment of mortality rates and the time intervals between symptom onset and hospitalisation and between symptom onset and death suggests that the illness pattern has not changed substantially during the three years.
- Cases have occurred all year round. However, the incidence of human cases peaked, in each of the three years in which cases have occurred, during the period roughly corresponding to winter and spring in the Northern Hemisphere. If this pattern continues, an upsurge in cases could be anticipated starting in late 2006 or early 2007.

A more standardised collection of epidemiological data by countries and timely sharing of these data are needed to improve monitoring of the situation, risk assessment, and the management of H5N1 patients.

Reference

World Health Organization. Epidemiology of WHO-confirmed human cases of avian A(H5N1) infection. *Wkly Epidemiol Rec* 2006;81:249–260.

Avian influenza – situation in Indonesia – update 20

20 June 2006

The Ministry of Health in Indonesia has confirmed the country's 51st case of human infection with the H5N1 avian influenza virus.

The case, which was fatal, occurred in a 13-year-old boy from South Jakarta. He developed symptoms on 9 June one week after helping his grandfather slaughter diseased chickens at the family home. The boy was hospitalised on 13 June and died on 14 June. The grandfather remains healthy. Contact tracing and monitoring are under way to ensure no further cases arise from this exposure setting.

Of the 51 cases confirmed to date in Indonesia, 39 have been fatal.

Expert consultation

WHO, FAO, and the Indonesian Ministries of health and agriculture jointly convened an expert consultation in Jakarta from 21 to 23 June. The consultation was held, at the request of the government's national commission on avian influenza and pandemic influenza, to assess the avian influenza situation in poultry and humans.

The consultation, was to be attended by more than 40 national and international experts, and would review measures for addressing the widespread presence of the virus in poultry and offer advice on strategies for reducing the number of human cases. The experts were to also examine epidemiological and virological data collected during a month-long investigation of a cluster of cases among family members in the Kubu Simbelang village of North Sumatra.

More than three weeks (two times the maximum incubation period) have passed since the last case in the cluster died on 22 May. Daily house-to-house monitoring for influenza-like illness was conducted throughout the village and in health care facilities where patients were treated, and no further cases were detected. While these findings indicate no significant changes in the epidemiology of the disease, results from investigation of the cluster will be reviewed as they may yield lessons useful in the investigation and interpretation of other large clusters where human-to-human transmission is suspected.

Several viruses have been isolated from the seven confirmed cases in the cluster and these have been fully sequenced at WHO reference laboratories in Hong Kong and the USA. Experts from these laboratories will be presenting their findings for review during the consultation.

Cholera

Sudan – update

21 June 2006

Between 21 April and 18 June 2006, the Federal Ministry of Health (FMoH) has reported a total of 2,007 cases, including 77 deaths (case fatality rate, CFR=3.8%), of acute watery diarrhoea in 9 of the 15 states in northern Sudan. Of these cases 35.3 per cent (CFR=4.9%) occurred in Khartoum state, while 26 per cent occurred in North Kordofan state. The overall CFR during this period was 3.8 per cent.

The National Public Health Laboratory of the FMoH confirmed the isolation of *Vibrio cholerae* 01 Inaba in 70 of the 139 stool samples (50%) collected so far from these states.

The FMoH has formed a task force, including UNICEF and WHO, to coordinate the overall response to the epidemic. WHO is also supplying diarrhoeal disease kits as well as laboratory supplies for the collection of samples and enteric disease bacteriology kits for establishing field laboratories to increase laboratory surveillance capacities in several affected states.

Between 28 January and 14 June 2006, a total of 16,187 cases, including 476 deaths (CFR=3%), of acute watery diarrhoea has been reported in 8 out of 10 states in southern Sudan (see previous report). *Vibrio cholerae* 01 Inaba has been laboratory confirmed in several stool samples by African Medical and Research Foundation (AMREF) laboratories in Nairobi.

A task force under the Ministry of Health of the Government of Southern Sudan (MOH/GoSS), including the FMoH, WHO, other UN and non-governmental partners has been established to coordinate the overall public health response. Several control measures are being implemented to contain the outbreak: strengthening the surveillance and reporting system, standardising case management and promoting health education and hygiene, with the chlorination of public water supplies.

Cholera

Angola – update

21 June 2006

As of 19 June 2006, Angola has reported a total of 46,758 cases including 1,893 deaths with an overall (case fatality rate, CFR 4.0%). Fourteen of the 18 provinces were affected; of all cases, 49 per cent occurred in Luanda and 17 per cent in Benguela provinces. The CFR, broken down by province, ranges between 1 and 30 per cent.

Although current trends show a decline in most provinces, a daily incidence of around 125 cases was still being reported.

A plan of action for cholera has been drawn up and agreed upon by all partners at country level, for short, medium and long-term response to the outbreak.

WHO was sending Interagency Diarrhoeal Disease Kits to the most affected provinces and continues to support the Ministry of Health in its surveillance, water and sanitation, social mobilisation and logistics activities.

Plague in the Democratic Republic of the Congo

14 June 2006

As of 13 June 2006, WHO has received reports of 100 cases of suspected pneumonic plague, including 19 deaths in Ituri District, Oriental Province. Suspected cases of bubonic plague have also been reported but the total number is not known at this time. Preliminary results from rapid diagnostic tests in the area confirm pneumonic plague. Additional laboratory analysis, including tests by culture, is ongoing on 18 samples.

Ituri is known to be the most active focus of human plague worldwide, reporting around 1,000 cases a year. The first cases in this outbreak occurred in a rural area, in the Zone de Santé of Linga, in mid-May.

A team from Médecins sans Frontières (Switzerland), WHO and the Ministry of Health has been in the area to assess the situation and provide support to the local health authorities. Isolation wards have been established to treat patients; close contacts are being traced and receiving chemoprophylaxis. However, control measures have been difficult to implement because of security concerns in the area.

Polio – world update

Source: June 2006 Monthly Situation Report, Global Polio Eradication Initiative [edited]

Data as at 21 June 2006

Nigeria

In 2006, 467 cases of polio have been reported to June, compared to 168 cases for the same period in 2005. Five states – Bauchi, Jigawa, Kaduna, Kano and Katsina – account for 86 per cent of the national caseload.

India

Fifty-three cases of polio have been reported in 2006 (compared with 18 for the same period in 2005). Two of the cases are from Madhya Pradesh (first case since 8 November 2003) and Jharkhand (first case since 9 October 2005). However, since the beginning of the year, polio transmission is increasingly restricted compared with the previous year, to key districts of western Uttar Pradesh and Bihar. Moradabad District in Uttar Pradesh accounts for 22 of the 53 cases nationwide this year.

Pakistan and Afghanistan

In Pakistan, six cases of polio have been reported this year, compared with 10 for the same period last year. In Afghanistan, 13 cases have been reported this year, compared with three for the same period last year. Two of the cases are in the provinces of Uruzgan and Zabul, previously unaffected by polio.

Namibia

Four cases of an outbreak of acute flaccid paralysis in Namibia were virologically confirmed to be wild poliovirus type-1. Genetic sequencing has determined that the virus is of Indian origin and was imported from Angola, which reported 10 cases in 2005 (most recent case November 2005). An international and regional rapid response team is assisting the government and a response activity using mOPV1 started on 21 June, the first of three nationwide rounds. The majority of the more than 100 suspected cases are adults, and 15 have died. Namibia began routine immunisation for polio in 1990; the cause of the largely adult outbreak is yet to be determined. The paralysis-to-infection rate of poliovirus is higher among adults than in children, as is the fatality rate.

Bangladesh

An additional two cases of polio were reported in Bangladesh (onset of paralysis on 23 January and 14 April, i.e. prior to the NIDs), bringing the total since the initial importation of polio to three. The new cases are in the centre of the country and on the western border with India.

Myanmar

A polio case originally reported as wild poliovirus has been genetically found to be a vaccine-derived poliovirus. No further cases have been reported, despite strengthened disease surveillance.

Somalia and Ethiopia

In Somalia, 25 cases of polio have been reported in 2006. Polio appears to be on the decline in Mogadishu, formerly the epicentre of the outbreak. The risk of further spread across the Horn of Africa remains high. In Ethiopia, three cases have been reported this year, in Somali and Amhara regions.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org).

Botulism from home-canned bamboo shoots, Thailand

Source: MMWR *2006; 14 April; 55:389–392 [edited]*

On 14 March 2006, an annual religious rite was observed in Nawaimai village, Pakaluang sub-district, Baan Luang district, Nan province. Villagers from Pakaluang and neighbouring sub-districts joined the event. That day, several persons who attended the festival visited local health-care providers with symptoms of gastroenteritis. Personnel from the Ministry of Public Health Field Epidemiology Training Program (FETP) were notified of a possible foodborne outbreak on 15 March 2006. Illnesses progressed to include bulbar muscle paralysis, with respiratory depression requiring ventilatory support in three patients, at which time a botulism outbreak was suspected. A quick door-to-door survey conducted by village volunteers identified 354 villagers who had attended the event, of whom 200 (56%) ate food served at the event.

Of the 163 persons with illness, 141 (86.5%) were admitted to area hospitals. All 141 hospitalised patients and 10 patients treated as outpatients were systematically queried about their symptoms.

The majority of those patients experienced abdominal pain (116; 76.8%), dry mouth (76; 50.3%), and nausea (76; 50.3%); some had dysphagia (52; 37.7%), vomiting (53; 35.1%), diplopia (26; 17.2%), ptosis (16; 10.6%), and weakness of extremities (14; 9.3%). Forty-two (29.8%) of the hospitalised patients required mechanical ventilation.

Home-canned bamboo shoots were the only item eaten by 100 per cent of affected persons, although bamboo shoots were routinely consumed with the chilli and shrimp paste. The bamboo shoots had been produced locally by a women's group in the village. The shoots had been processed in 20 litre cans with approximately 13 kg of shoots per can. A total of 53 cans were produced during September 2005; 46 cans were sold during September 2005 to February 2006, primarily in the district where they were made. As of 10 April 2006, a total of 25 patients remained hospitalised, and 9 (36%) were still on respirators. No patients had died.

Measles in Europe

Source: Eurosurveillance weekly release, 15 June 2006. [Edited]

An outbreak of measles in children and young people has been occurring since the beginning of January 2006 in the German state of Nordrhein-Westfalen, and consequently, several governmental agencies in countries throughout Europe, and the WHO Pan American Health Organization, are advising travellers to Germany, especially football fans and people travelling to this state, to ensure that they have had measles vaccination before their trip. Three of the 12 cities where matches are being played are located in Nordrhein-Westfalen (Köln/Cologne, Dortmund and Gelsenkirchen), although sporadic measles cases only have been reported in these cities.

The latest cumulative total of notified measles cases in the Nordrhein-Westfalen outbreak now stands at 1,452 (an incidence in the state of 8/100 000 inhabitants). Most cases have been reported in children and young people. Although this number is still increasing, the weekly reported case numbers are falling, from a peak of 151 cases in April (week 17, 2006) to fewer than 50 per week currently. This outbreak has so far resulted in four cases of measles encephalitis and one case of measles meningitis.

There are also ongoing outbreaks of measles in a number of other European countries, the largest being in the Ukraine, where it is affecting mainly young adults, and the case total exceeded 20,000 at the end of February 2006. The Ukrainian national football team is one of the 32 World Cup qualifying teams and Ukrainian fans have travelled to Germany to attend matches. The Ukrainian team's first match took place on 14 June in Leipzig, eastern Germany.

Mumps

Source: MMWR Dispatch 18 May 2006/55 (Dispatch);1–5 [edited]

The Centers for Disease Control and Prevention and state and local health departments continue to investigate an outbreak of mumps that began in Iowa in December 2005 and involved at least 10 additional states as of 2 May 2006.

During the period 1 January to 2 May 2006, 11 states reported 2,597 cases of mumps. Eight states (Illinois, Iowa, Kansas, Missouri, Nebraska, Pennsylvania, South Dakota, and Wisconsin) reported mumps outbreaks with ongoing local transmission or clusters of cases; three states (Colorado, Minnesota, and Mississippi) reported cases associated with travel from an outbreak state. The majority of mumps cases [1,487 (57%)] were reported from Iowa; states with the next highest case totals were Kansas (371), Illinois (224), Nebraska (201), and Wisconsin (176). Of the 2,597 cases reported overall, 1,275 (49%) were classified as confirmed, 915 (35%) as probable, and 287 (11%) as suspect. The classification for 120 (5%) cases was unknown. Twelve mumps viral isolates from six states were characterised; all were mumps genotype G.

As of 10 May, a total of 11 persons potentially infected with mumps who travelled by aircraft during 26 March to 25 April, had been identified on 33 commercial flights operated by eight different airlines. Notifications had either been initiated or completed for persons potentially exposed on all identified flights. As of 12 May, of about 575 persons potentially exposed on the flights, 132 had received follow-up greater than 25 days after their potential exposure. Two cases of mumps were identified, possibly associated with transmission during air travel. Both cases occurred among Iowa residents, one of whom was a travelling companion of a person known to have mumps.

Chikungunya in the Indian Ocean – genetic analysis

Source: Public Library of Science Medicine, Editors' Statement, 23 May 2006 [edited]

Since late 2004, a large outbreak of chikungunya fever has been taking place in the Indian Ocean. For example, on the island of Reunion, approximately one third of the total population of 770,000 were reportedly infected by April 2006. Sylvain Brisse and 22 colleagues report the first molecular analysis of the chikungunya viruses involved in the outbreak. The complete genome sequence of viral isolates from six patients and partial sequences of isolates from 121 patients at different stages and locations of the outbreak reveal unique and evolving genetic features.

The authors report the nearly complete genome sequence of six selected viral isolates (isolated from five sera and one cerebrospinal fluid), along with partial sequences of glycoprotein E1 from a total of 127 patients from Reunion, Seychelles, Mauritius, Madagascar, and Mayotte islands. Results indicate that the outbreak was initiated by a strain related to East-African isolates, from which viral variants have evolved following a traceable micro-evolution history. Unique molecular features of the outbreak isolates were identified. The authors conclude that unique molecular features of the analysed Indian Ocean isolates of chikungunya virus demonstrate their high evolutionary potential and suggest possible clues for understanding the atypical magnitude and virulence of this outbreak.

Reference

Genome microevolution of chikungunya viruses causing the Indian Ocean outbreak. *PLoS Medicine* 2006;3 No. 7.

Crimean-Congo haemorrhagic fever in Russia

Source: Ami-Tass News Agency, 14 June 2006 [edited]

As of 8 June 2006, 50 cases of Crimean-Congo haemorrhagic fever (CCHF) have been registered in the Southern Federal District of Russia, including four fatal cases. The first cases of CCHF were registered in the middle of April 2006 in Stavropol region, and in the beginning of May in the Republic of Kalmykia and the Rostov region.

In 2006 there has been a marked expansion in the distribution of CCHF cases: new cases have been detected where no cases have been observed in recent years. Consequently, late recognition of the disease and late referral for medical attention have resulted in severe manifestation of the disease.

Most cases occurred during care of agricultural animals in private facilities. It is especially important to undertake preventive treatment of animals against tick infestation. However, due to insufficient allocation of finances for these purposes, fewer than 50 per cent of animals had been protected against ticks by the end of May 2006.