Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 31 Number 4 Quarterly report December 2007

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ISBN 0725-3141

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2007;31:158–162.

Communicable Diseases Intelligence contributes to the work of the Communicable Diseases Network Australia (http://www.health.gov.au/cdna)

Website

http://www.health.gov.au/cda

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This journal is indexed by *Index Medicus*, Medline and the Australasian Medical Index

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Printed by Union Offset, Canberra

Annual reports

Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2006

The OzFoodNet Working Group

Abstract

In 2006, OzFoodNet sites reported 24,598 notifications of seven diseases or conditions that are commonly transmitted by food, representing an increase of 2.5% over the mean of the previous 5 years. The most frequently notified aetiological agents were *Campylobacter* (15,492 notifications) and *Salmonella* (8,331 notifications). *Salmonella* notifications increased in 2006 by 5.2% when compared to historical reports. The most common *Salmonella* serotype notified in Australia during 2006 was *Salmonella* Typhimurium, as in previous years. *S.* Saintpaul was the second most common serotype notified during 2006 as a result of a large multistate outbreak associated with rockmelons. During 2006, OzFoodNet sites reported 1,544 outbreaks of gastrointestinal illness including those transmitted by contaminated food. In total, these outbreaks affected 34,916 people and resulted in 769 people being admitted to hospital and 27 deaths. Food was suspected or confirmed as the primary cause for 115 of these outbreaks and affected 1,522 persons, hospitalised 146 persons but did not result in any deaths. *S.* Typhimurium was the most common aetiological agent in foodborne outbreaks and restaurants were the most common setting for foods implicated in foodborne outbreaks. Sixteen outbreaks were related to eggs during 2006 compared to five outbreaks in 2005. Eighty-one per cent (13/16) of these egg-associated outbreaks were due to various phage types of *S.* Typhimurium. Fresh fruits and vegetables, categorised as fresh produce were responsible for four outbreaks, all due to salmonellosis. Public health laboratories provided complete serotype and phage type information on more than 97% of all *Salmonella* notifications during 2006. Completeness of reporting for *Salmonella* was equivalent to 2005 and was essential to identifying and investigating outbreaks. This report demonstrates OzFoodNet's ability to detect and investigate the burden and causes of foodborne disease in Australia. OzFoodNet efforts assist agencies to develop food safety policy and prevent foodborne illness. *Commun Dis Intell* 2007;31:345–365.

Keywords: foodborne disease, surveillance, disease outbreak

Introduction

Foodborne illnesses are a substantial burden in Australia, with an estimated 5.4 million cases occurring annually, costing an estimated $1.2 billion dollars per year.[1] Most foodborne illnesses are mild and do not require medical attention, with the majority of cost associated with the large number of affected people taking time from work to recover or care for affected family members. There are numerous enteric pathogens commonly transmitted through food that may cause illness; only a handful of these illnesses are specifically notifiable to health departments.[2] Most foodborne illnesses are underreported in surveillance statistics collected by health departments.[3] The proportion of cases that are notified varies considerably by disease, as the severity of various illnesses differ markedly.[4,5]

Health departments use surveillance of infectious diseases for monitoring trends in illness, detecting outbreaks, and monitoring the effects of interventions.[6] The source of infection can be difficult to determine in sporadic cases of enteric diseases, that is, cases not associated with an outbreak, as they may be acquired through a variety of transmission routes including contaminated water or foods, other infected persons, animals, or other sources within the environment. In outbreaks of enteric infections, the mode of transmission is more likely to be determined, allowing development of policy to prevent further disease.[7]

In 2000, the Australian Government Department of Health and Ageing established OzFoodNet to provide national intelligence on foodborne disease.[8] OzFoodNet was modelled on the Centers for Disease Control and Prevention's FoodNet surveillance system.[9] The OzFoodNet network consists of epidemiologists employed by each state and territory health department to conduct investigations and applied research of foodborne disease. The network involves many different collaborators in addition to OzFoodNet site staff, including the National Centre for Epidemiology and Population Health and the Public Health Laboratory Network.

OzFoodNet has a member on the Communicable Diseases Network Australia, which is Australia's peak body for communicable disease control.[10] The Australian Government Department of Health and Ageing funds OzFoodNet and convenes committees to manage the network and review the scientific basis for various research projects.

This is the sixth annual report of OzFoodNet and covers data and activities for 2006.

Methods

Population under surveillance

In 2006, the coverage of the network included the entire Australian population, which was estimated to be 20,605,488 persons.[11] All states and territories in Australia (New South Wales, Victoria, Queensland, South Australia, Western Australia, Tasmania, the Northern Territory, and the Australian Capital Territory) participated in OzFoodNet in 2006. In addition, surveillance in New South Wales was supplemented by an additional OzFoodNet site hosted by the Hunter New England Area Health Service.

Data sources

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. OzFoodNet aggregated and analysed data on the following seven diseases or conditions, a proportion of which may be acquired from food:

- non-typhoidal *Salmonella* infections;
- *Campylobacter* infections (except in New South Wales);
- *Listeria* infections;
- *Shigella* infections
- typhoid; and
- Shiga toxin-producing *Escherichia coli* (STEC) infections and haemolytic uraemic syndrome (HUS).

To compare notifications in 2006 to historical totals, crude numbers and rates of notification were compared with either the mean of the previous 5 years or with data from the previous year. Specific sub-types of infecting organisms were analysed using data from the National Notifiable Diseases Surveillance System (NNDSS) and OzFoodNet sites. This report used a NNDSS dataset provided in June 2007 and was analysed by the date a notification was received by a jurisdiction. Numbers and rates may vary from those in the NNDSS 2006 annual report, which used a later NNDSS dataset and was analysed by date of diagnosis. The estimated resident populations for each state or territory as at June 2006[11] was used to calculate rates of notification. Birth data from the Australian Institute of Health and Welfare, National Perinatal Statistics Unit was used to calculate the incidence of neonatal listeriosis.[12]

Gastrointestinal and foodborne disease outbreaks

OzFoodNet collected information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2006. An outbreak of foodborne disease was defined as two or more people with 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.

OzFoodNet epidemiologists collated 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, outbreaks were categorised by aetiological agents, food vehicles and settings where the implicated food was prepared. Data on outbreaks due to transmission from water or from investigation of a cluster were also summarised. The number of outbreaks and documented causes reported here may vary from summaries previously published by individual jurisdictions as these can take time to finalise.

Results

Rates of notified infections

In 2006, OzFoodNet sites reported 24,598 notifications of seven diseases or conditions that are commonly transmitted by food. This represents a 2.5% increase from the mean of 24,020 notifications for the previous 5 years. Reports of these seven diseases make up almost a fifth of the notifications to the NNDSS.[2] A summary of the number and rate of notification of these is shown in Table 1.

Salmonella infections

In 2006, OzFoodNet sites reported 8,331 cases of *Salmonella* infection, a crude rate of 40.4 cases per 100,000 population. The 2006 rate was a 5.2% increase over the mean of the previous 5 years (Table 1). Notification rates ranged from 27.6 cases per 100,000 population in Victoria to 193 cases per 100,000 population in the Northern Territory,

Table 1. Number of notified cases, rate and 5-year mean rate per 100,000 population of potentially foodborne diseases, Australia, 2001 to 2006, by disease and state or territory

Disease		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Salmonella	Notified cases, 2006	125	2,081	399	2,749	574	193	1,406	804	8,331
	Rate, 2006	38.0	30.5	193.0	67.8	36.9	39.5	27.6	39.2	40.4
	Mean rate, 2001–2005	26.9	29.9	187.2	65.7	35.3	37.5	25.2	37.3	38.4
Campylobacter	Notified cases, 2006	414	*	272	4,006	2491	598	5,710	2001	15,492
	Rate, 2006	125.9	*	131.6	98.8	160.2	122.3	112.1	97.6	112.4
	Mean rate, 2001–2005	122.0	*	123.0	106.5	155.8	136.9	116.0	113.9	118.5
Listeria	Notified cases, 2006	1	24	0	3	5	0	13	13	59
	Rate, 2006	0.30	0.35	0.00	0.07	0.32	0.00	0.26	0.63	0.29
	Mean rate, 2001–2005	0.37	0.33	0.10	0.34	0.24	0.29	0.28	0.44	0.32
Typhoid	Notified cases, 2006	0	32	3	6	2	1	18	12	74
	Rate, 2006	0.00	0.47	1.45	0.15	0.13	0.20	0.35	0.59	0.36
	Mean rate, 2001–2005	0.19	0.43	0.20	0.21	0.20	0.08	0.34	0.42	0.33
Shigella	Notified cases, 2006	2	74	123	98	38	4	77	136	552
	Rate, 2006	0.61	1.08	59.5	2.42	2.44	0.82	1.51	6.63	2.68
	Mean rate, 2001–2005	1.12	1.53	65.3	2.11	2.57	0.75	1.56	5.91	2.77
Shiga-like toxin-producing *E. coli*	Notified cases, 2006	0	13	2	15	37	0	3	3	73
	Rate, 2006	0.0	0.2	1.0	0.4	2.4	0.0	0.1	0.1	0.4
	Mean rate, 2001–2005	0.00	0.08	0.00	0.23	2.23	0.08	0.10	0.23	0.29
Haemolytic uraemic syndrome	Notified cases, 2006	0	15	0	0	1	0	1	0	17
	Rate, 2006	0.00	0.22	0.00	0.00	0.06	0.00	0.02	0.00	0.08
	Mean rate, 2001–2005	0.00	0.10	0.30	0.03	0.12	0.08	0.05	0.03	0.07

* *Campylobacter* is not a notifiable disease in New South Wales.

which usually has the highest rate of salmonellosis. The male to female ratio for salmonellosis was 1:1. The highest age-specific rate of *Salmonella* infection was 203 cases per 100,000 population in males aged from 0–4 years. Notifications were also elevated for both males and females in the 5–9 year age group and in the 20–29 year age group.

In 2006, the most commonly reported *Salmonella* serotype was *S.* Typhimurium. The most commonly notified phage type was *S.* Typhimurium 135 (including *S.* Typhimurium 135a), with 751 notifications in 2006 (Table 2). *S.* Saintpaul was the second most common serotype notified in Australia and featured in the top five for all jurisdictions except South Australia. During 2006, there was a large multi-state outbreak of *Salmonella* Saintpaul associated with rockmelons, which contributed to the widespread notification of this serotype.

The highest specific rates for a single serotype were for *Salmonella* Mississippi (13.1 cases per 100,000 population) in Tasmania and *S.* Saintpaul (16 cases per 100,000 population) and *S.* Ball (15 cases per 100,000 population) in the Northern Territory.

Salmonella Enteritidis

Salmonella Enteritidis is an internationally important serotype of *Salmonella* that has caused widespread and prolonged outbreaks in the United States of America (USA) and Europe.[13,14] This serotype can infect the internal contents of eggs through the oviducts of infected chickens[15,16] but has not been associated with Australian egg layer flocks. The majority of cases in Australia are associated with overseas travel. OzFoodNet monitors the incidence of *S.* Enteritidis to detect outbreaks of locally-acquired cases.

During 2006, OzFoodNet sites reported 305 cases of *S.* Enteritidis (Table 3). Of those cases where travel status was reported, 85% (198/233) had travelled overseas and cases often reported visiting several countries. Asian countries were the most frequently reported travel destination, perhaps reflecting that these countries are common travel destinations for Australians. In the Asian region, cases of *S.* Enteritidis infection were reported after travelling to Indonesia (36, 15%), Thailand (30, 13%), and Singapore (24, 10%). The most com-

Table 2. Numbers, rates and proportions of the top 5 *Salmonella* infections, Australia, 2005 to 2006, by OzFoodNet site*

OzFoodNet site	*Salmonella* sero/phage type	2006 n	2006 rate†	Proportion‡ (%)	2005 n	2005 rate	2006/2005 ratio§
Australian Capital Territory	Saintpaul	14	4.3	11	3	0.9	4.7
	Typhimurium 135	12	3.6	10	13	4.0	0.9
	Typhimurium 170/108	11	3.3	9	14	4.3	0.8
	Typhimurium 9	7	2.1	6	10	3.1	0.7
	Typhimurium 44	7	2.1	6	4	1.2	1.8
New South Wales	Typhimurium 170/108	223	3.3	11	375	5.5	0.6
	Typhimurium 135	210	3.1	10	180	2.7	1.2
	Birkenhead	105	1.5	5	82	1.2	1.3
	Saintpaul	103	1.5	5	42	0.6	2.5
	Typhimurium 9	77	1.1	4	155	2.3	0.5
Northern Territory	Saintpaul	33	16.0	8	49	24.1	0.7
	Ball	31	15.0	8	48	23.6	0.6
	Typhimurium 135	21	10.2	5	1	0.5	21.0
	Chester	17	8.2	4	12	5.9	1.4
	Muenchen	16	7.7	4	9	4.4	1.8
	Infantis	16	7.7	4	8	3.9	2.0
Queensland	Saintpaul	267	6.6	10	274	6.9	1.0
	Virchow 8	215	5.3	8	190	4.8	1.1
	Typhimurium 135	177	4.4	6	136	3.4	1.3
	Birkenhead	154	3.8	6	128	3.2	1.2
	Aberdeen	136	3.4	5	136	3.4	1.0
South Australia	Typhimurium 135	79	5.1	14	47	3.0	1.7
	Typhimurium 170/108	62	4.0	11	36	2.3	1.7
	Typhimurium 9	58	3.7	10	57	3.7	1.0
	Infantis	37	2.4	6	48	3.1	0.8
	Anatum	22	1.4	4	6	0.4	3.7
Tasmania	Mississippi	64	13.1	33	59	12.1	1.1
	Typhimurium 135	40	8.2	21	176	36.2	0.2
	Typhimurium 170/108	15	3.1	8	7	1.4	2.1
	Typhimurium 9	15	3.1	8	10	2.1	1.5
	Saintpaul	6	1.2	3	2	0.4	3.0
Victoria	Typhimurium 135	158	3.1	11	191	3.8	0.8
	Typhimurium 9	125	2.5	9	118	2.3	1.1
	Typhimurium 44	115	2.3	8	50	1.0	2.3
	Typhimurium 170/108	100	2.0	7	63	1.3	1.6
	Saintpaul	76	1.5	5	22	0.4	3.5
Western Australia	Oranienburg	82	4.0	10	62	3.1	1.3
	Saintpaul	60	2.9	7	31	1.5	1.9
	Typhimurium 135	54	2.6	7	42	2.1	1.3
	Typhimurium 12	33	1.6	4	28	1.4	1.2
	Muenchen	31	1.5	4	30	1.5	1.0
Australia	Typhimurium 135	751	3.6	9	813	4.0	0.9
	Saintpaul	572	2.8	7	436	2.1	1.3
	Typhimurium 170/108	474	2.3	6	550	2.7	0.9
	Typhimurium 9	358	1.7	4	421	2.1	0.9
	Virchow 8	273	1.3	3	248	1.2	1.1

* 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 2006.

§ Ratio of the number of reported cases in 2006 compared to the number reported in 2005.

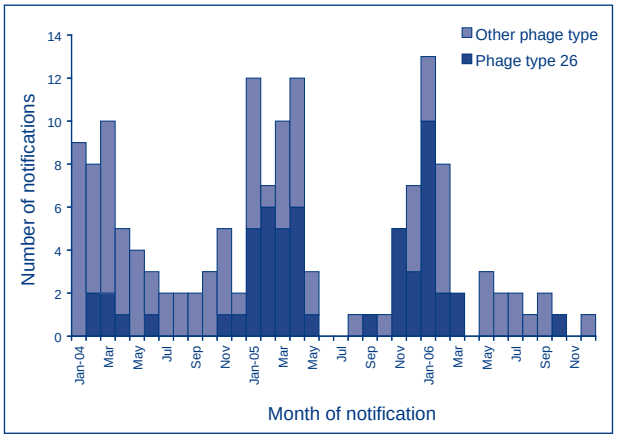
mon infecting phage types among cases who had travelled overseas were 6a (51 cases), 1 (38), 26 (37) and 4 (29). A travel history could not be determined for 24% (72/305) of cases in 2006, which was an increase from 11% (44/387) in 2005. The better reporting of travel history in 2005 may have been due to the completion of enhanced data collection in late 2005, for an OzFoodNet national study of locally-acquired *S.* Enteritidis.

Overall, 15% (35/233) of patients infected with *S.* Enteritidis acquired their infection in Australia. These 35 locally-acquired cases compares with an average of 53 cases per year for the previous 3 years. The median age of locally-acquired cases was 34 years (range 0–91 years) and 55% were male. Just over half of all locally-acquired *S.* Enteritidis cases during 2006 occurred in Queensland (57%, 20/35 cases); most of these cases were due to phage type 26 (65%, 13/20 cases). Locally-acquired *S.* Enteritidis cases continued to be highly seasonal, occurring primarily in the summer (Figure 1).

Table 3. Number of *Salmonella* Enteritidis infections, Australia, 2006, by travel history, and state or territory

OzFoodNet site	History of travel overseas			Total
	Yes	No	Unknown	
Australian Capital Territory	6	2	0	8
New South Wales	43	4	22	69
Northern Territory	6	1	3	10
Queensland	22	20	38	80
South Australia	11	2	1	14
Tasmania	4	0	0	4
Victoria	47	2	5	54
Western Australia	59	4	3	66
Total	198	35	72	305

Figure 1. *Salmonella* Enteritidis infections acquired in Australia, 2004 to 2006, by phage type and month of notification



Campylobacter infections

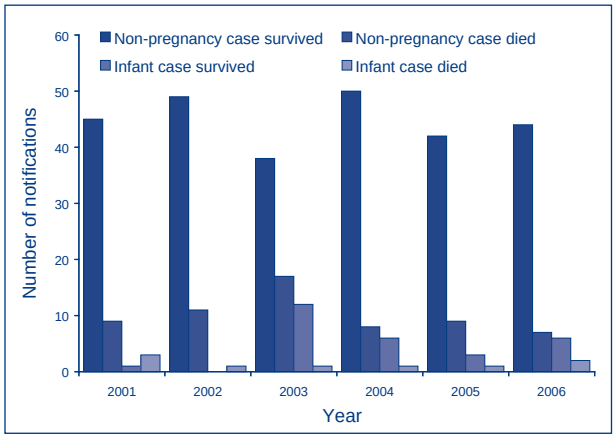
In 2006, OzFoodNet sites (excluding New South Wales) reported 15,492 cases of *Campylobacter* infection; a rate of 112.4 cases per 100,000 population. This rate was equivalent to the mean for the previous 5 years of 118.5 cases per 100,000 population (Table 1). Victoria, Queensland, Western Australia, and Tasmania all reported slight decreases in their rate of notification for 2006 compared to the mean for the previous 5 years. The lowest and highest rates of *Campylobacter* notification were in Western Australia (98 cases per 100,000 population) and in South Australia (160 cases per 100,000 population) respectively. The highest age-specific rate of notifications was in males in the 0–4 year age group (248 cases per 100,000 population) with a secondary peak in the 20–29 year age group for both males and females. Fifty-four per cent of notified cases were male.

Listeria infections

OzFoodNet sites reported 59 cases of listeriosis in 2006; a rate of 0.3 cases per 100,000 population (Table 1). The 2006 notification rate was equivalent to the 5-year historical mean (0.32 cases per 100,000 population).

Eighty-six per cent (51/59) of *Listeria* infections during 2006 were reported in non-pregnant persons, who were either elderly and/or immunocompromised. Among these non-pregnancy related cases, the male to female ratio was approximately 1:1, and 94% (48/51 were reported in persons aged 50 years or greater. Fourteen per cent (7/51) of non-pregnancy associated cases died, which was similar to previous years (Figure 2).

Figure 2. Notifications of *Listeria* showing non-pregnancy related infections and deaths, and materno-foetal infections and deaths, Australia, 2001 to 2006



Eight materno-foetal infections were reported during 2006, giving a rate of 3.1 cases per 100,000 births. New South Wales reported four cases, Western Australia reported two cases, and the Australian Capital Territory and Queensland each reported single cases during 2006. Twenty-five per cent (2/8) of infected neonates died during 2006, which was a consistent outcome reported in previous years.

Shigella infections

OzFoodNet sites reported 552 cases of shigellosis during 2006, a rate of 2.7 cases per 100,000 population (Table 1). This rate was equivalent to the mean for the previous 5 years of 2.8 cases per 100,000 population. As in previous years, the highest rate of notification was in the Northern Territory (60 cases per 100,000 population). In recent years, notification rates for shigellosis have decreased in all jurisdictions except Queensland and Western Australia. The male to female ratio of shigellosis cases was approximately 1:1.1. The highest age-specific notification rates were in the 0–4 years age group for both males (12.5 cases per population) and females (9.2 cases per 100,000 population). Mannitol negative *Shigella flexneri* 4a was the most common type reported in 2006 (Table 4). The most common *Shigella sonnei* biotypes, A and G, decreased during 2006 compared with 2005. It is estimated that approximately 10% of *Shigella* cases in Australia are due to foodborne transmission: other predominant modes of transmission of *Shigella* are overseas travel and through person-to-person transmission.[17] OzFoodNet sites did not identify any food-related outbreaks of *Shigella* during 2006.

Typhoid infections

OzFoodNet sites reported 74 cases of typhoid infection during 2006; a rate of 0.4 cases per 100,000 population (Table 1). This rate was equivalent to the mean for the previous 5 years of 0.3 cases per 100,000 population. The highest rate of typhoid was reported by the Northern Territory (1.5 cases per 100,000 population). The Australian Capital Territory reported no cases of typhoid during 2006.

Travel overseas, which is a significant risk factor for typhoid infection, was reported in 93% (68/73) of typhoid cases (Table 5). A single case (untypable) reported no overseas travel prior to their illness. Over a third of cases reporting travel overseas (27/68 cases) had travelled to India. The predominant typhoid phage types causing illness in travellers returning from India was E1 (14 cases) and E9 (5 cases).

Shiga toxin-producing Escherichia coli *infections*

OzFoodNet sites reported 73 cases of Shiga toxin producing *E. coli* (STEC) infection during 2006; a rate of 0.4 cases per 100,000 population (Table 1). The mean for the previous 5 years was 0.3 cases per 100,000 population. These numbers do not include cases of haemolytic uraemic syndrome (HUS) where an STEC organism was isolated or detected in stool samples as these are notified separately.

South Australia reported the majority of STEC cases and had the highest rate of notification at 2.4 cases per 100,000 population. South Australia continued a screening program for STEC in stools with visible blood. This accounts for the consistently high rate in South Australia compared with other jurisdictions. Other jurisdictions have also, at times, enhanced their screening programs resulting in increased notifications of STEC. The Australian Capital Territory and Tasmania reported no cases of STEC in 2006. The male to female ratio of cases was 0.8:1, similar to the ratio in 2005. The highest reported rate was for females in the 0–4 years age group (1.0 case per 100,000 population).

During 2006, *E. coli* serotype O157 was responsible for 58% (21/36) of infections where serotype information was available, compared to 39% in 2005 (Table 6). *E. coli* serotype O111 and O26 were the second most common serotype each with five cases reported. A serotype was not identified in 51% (37/73) of cases.

Table 4. Numbers, rates and proportions of the top 5 *Shigella* infections, Australia, 2005 to 2006

	2006 n	2006 Rate*	Proportion† %	2005 n	2005 Rate*	2006/2005 Ratio
Shigella flexneri 4a Mannitol negative	93	0.5	18	77	0.4	1.2
Shigella flexneri 4	82	0.4	16	46	0.2	1.8
Shigella sonnei biotype A	77	0.4	15	169	0.8	0.5
Shigella sonnei biotype G	73	0.4	14	136	0.7	0.5
Shigella flexneri 2a	53	0.3	10	78	0.4	0.7

* Rate per 100,000 population.

† Proportion of total *Shigella* notified for 2006.

Table 5. Travel status for notified typhoid cases acquired overseas, Australia, 2006

Country/region	Number of cases	Predominant phage type (# cases)
Asia	1	Degraded (1)
Bali	1	Unknown (1)
Bangladesh	8	D6 (1), E9 (3), degraded (1), untypable (1), unknown (2)
China	1	25 (1)
Ghana	1	A (1)
India	24	A1 (1), E1 (14), E9 (3), K1 (1), 51 (1), untypable (3), unknown (1)
India/other	3	E9 (2), O variant (1)
Indonesia	12	D2 (1), E2 (2), degraded (2), untypable (5), unknown (2)
Kenya	2	E1 (2)
Lebanon	1	D1 (1)
Nepal	1	E9 (1)
Pakistan	6	E1 (2), E9 (1), 38 (1), untypable (1), unknown (1)
Papua New Guinea	1	D2 (1)
Philippines	1	A (1)
Samoa	1	E variant (1)
Thailand	3	E9 (2) M1 (1)
Vietnam	1	Unknown (1)
Unknown	5	A(2), degraded (2), untypable (1)

Haemolytic uraemic syndrome

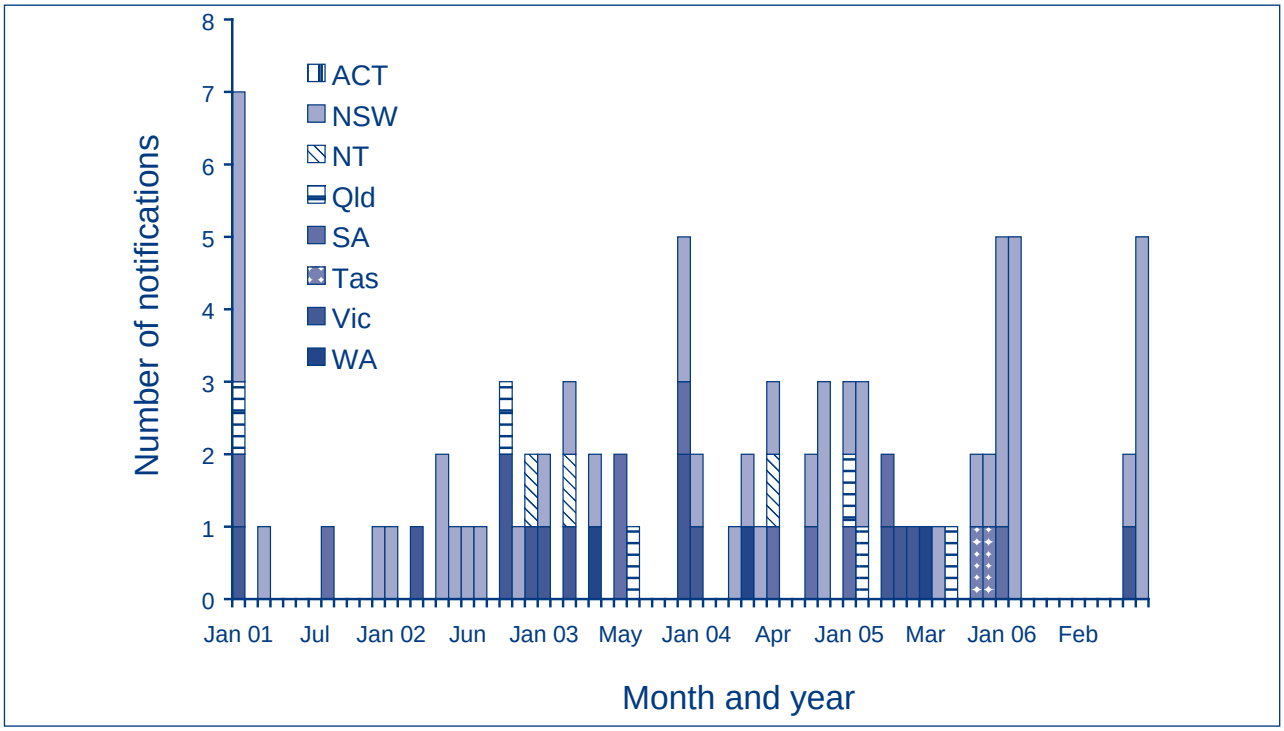
OzFoodNet sites reported 17 cases of haemolytic uraemic syndrome reported during 2006; a rate of 0.08 cases per 100,000 population (Table 1). This was the same number and a comparable rate to that reported in 2005 (Figure 3). New South Wales notified 15 cases, and Victoria and South Australia each reported a single case. Nine of the cases (53%) were female. The median age of HUS cases was 9 years and the age range was 1.2 to 81.4 years. The highest rate of notification was in males aged 0–4 years with a rate of 0.5 cases per 100,000 population. Sites reported that STEC was detected in the faeces of five HUS cases but a serotype was reported for one case from New South Wales (STEC O55).

The 15 HUS cases reported from New South Wales were part of two identified clusters from January/February and November/December 2006 (Figure 3). Enhanced surveillance and active case finding in renal units and children's hospitals may account for the observed increase in HUS cases in NSW. These cases were investigated initially by New South Wales public health units and then re interviewed by OzFoodNet staff to determine whether there were any links between cases. No common links or risk factors for infection were identified during these investigations.[18] OzFoodNet sites did not identify any cases of HUS between March and October 2006.

Table 6. Number of notified cases of Shiga toxin-producing *Escherichia coli,* Australia, 2006, by serotype, and state or territory

Serotype	State						Total
	NSW	NT	Qld	SA	WA	Vic	
O157	2	0	3	14	1	1	21
O111	1	0	3	1	0	0	5
O26	2	0	1	2	0	0	5
O113	0	0	1	0	0	0	1
O55	1	0	0	0	0	0	1
O153	0	0	0	0	1	0	1
Mixed infection	0	0	0	0	0	2	2
Unknown	7	2	7	20	1	0	37
Total	13	2	15	37	3	3	73

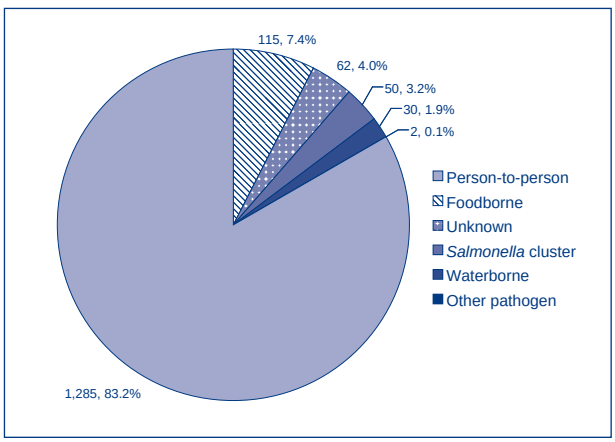
Figure 3. Numbers of notified cases of haemolytic uraemic syndrome, Australia, 2001 to 2006, by state or territory



Gastrointestinal and foodborne disease outbreaks

During 2006, OzFoodNet sites reported 1,544 outbreaks of gastrointestinal illness. These outbreaks affected 34,916 people and resulted in 769 people being admitted to hospital and 27 deaths. Person-to-person transmission was the mode of transmission for 83% (1,285/1,544) of outbreaks (Figure 4) and accounted for 92% (32,155/34,916) of all persons affected by outbreaks including 27 deaths.

Figure 4. Foodborne and gastroenteritis outbreaks ($n = 1544$) reported by OzFoodNet sites, Australia, 2006, by mode of transmission



Sixty per cent (777/1,285) of outbreaks associated with person-to-person transmission occurred in aged care facilities, while 20% (259/1,285) and 13% (167/1,285) occurred in hospital and child care settings, respectively. Fifty per cent (636/1,285) of person-to-person outbreaks were caused by norovirus, while 29% (370/1,285) were of unknown aetiology and 10% (132/1,285) were suspected to be due to a viral pathogen. *Cryptosporidium* and rotavirus were each responsible for 3% of person-to-person outbreaks (36/1,285 and 35/1,285, respectively).

There were 16 outbreaks of mixed infections. These outbreaks were due to norovirus in addition to other viral pathogens such as rotavirus, adenovirus, astrovirus, and non-viral pathogens such as *Campylobacter*, *Clostridium difficile*, and *Giardia*.

In 2006, OzFoodNet sites also investigated 30 outbreaks of recreational waterborne illness. These outbreaks affected 169 people, with no hospitalisations. All of these outbreaks occurred in Victoria and all were associated with swimming pools contaminated by *Cryptosporidium*.

Foodborne disease outbreaks

In 2006, there were 115 foodborne disease outbreaks giving an overall rate of 5.6 outbreaks per million population. These outbreaks affected 1,522 persons, hospitalised 146 persons but did not result in any deaths (Appendix 1).

New South Wales reported the largest number of outbreaks (38%, 44/115) (Table 7). The reporting rates of foodborne outbreaks for different OzFoodNet sites ranged from two outbreaks per million population in Tasmania to 14.5 outbreaks per million population in Northern Territory. The majority of outbreaks occurred in summer and autumn (Figure 5).

Aetiological agents

The most common agent responsible for foodborne disease outbreaks was *Salmonella*, which caused 36% (41/115) of outbreaks (Table 8). *S.* Typhimurium was responsible for 61% (25/41) of foodborne *Salmonella* outbreaks.

Figure 5. Outbreaks of foodborne disease, Australia, 2006, by selected aetiological agents and month of notification

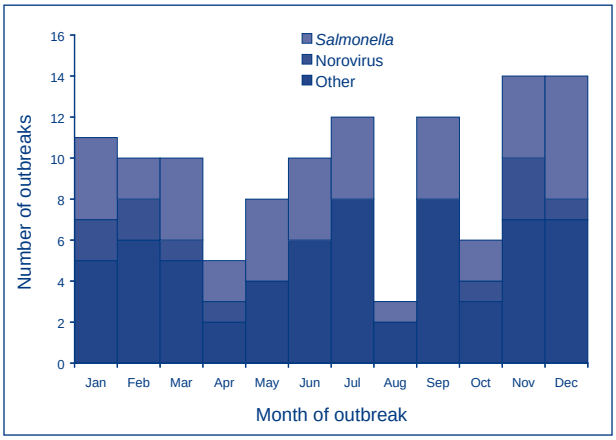


Table 7. Outbreaks of foodborne disease in Australia, 2006, by OzFoodNet site

State or territory	Number of outbreaks	People affected	Mean size (persons)	Hospitalised	Outbreaks per million population
Australian Capital Territory	3	27	9	1	9.1
New South Wales	44	496	11	65	6.4
Northern Territory	3	26	9	5	14.5
Queensland	28	403	14	23	6.9
South Australia	7	65	9	8	4.5
Tasmania	1	9	9	2	2.0
Victoria	21	293	14	18	4.1
Western Australia	5	92	18	4	2.4
Multi-state	3	111	37	20	n/a
Total	115	1,522	13	146	5.6

Table 8. Aetiological agents responsible for foodborne disease outbreaks, number of outbreaks and persons affected, Australia, 2006

Agent category	Number of outbreaks	People affected	Mean size (people)	Hospitalised	Hospitalisation rate
Bacillus cereus	1	14	14	0	0.0
Campylobacter spp.	4	67	17	4	6.0
Ciguatera	7	30	4	8	26.7
Clostridium perfringens	6	199	33	0	0.0
Hepatitis A	1	10	10	1	10.0
Histamine	4	12	3	7	58.3
Norovirus	11	369	34	4	1.1
Salmonella Typhimurium	25	258	10	76	29.5
Salmonella other	16	209	13	31	14.8
Staphylococcus aureus	1	3	3	0	0.0
Sodium nitrite	1	6	6	6	100.0
Vibrio cholerae	1	3	3	2	66.7
Unknown	37	342	9	7	2.0
Total	115	1,522	13	146	9.6

Eleven of the 19 outbreaks of illness due to toxins in 2006 were related to contaminated fish. Outbreaks of ciguatera fish poisoning (7 outbreaks) and histamine poisoning (4 outbreaks) were small with a mean of four and three persons affected, respectively. Other toxin related outbreaks included six outbreaks of *Clostridium perfringens* intoxication, and one outbreak each of *Staphylococcus aureus* and *Bacillus cereus* intoxication.

Aetiological agents responsible for foodborne outbreaks also included 11 outbreaks due to foodborne norovirus (369 people), four outbreaks due to *Campylobacter* species (67 people), one outbreak of hepatitis A (10 people) and one outbreak of *Vibrio cholerae* (3 people).

Thirty-two per cent (37/115) of outbreaks were of unknown aetiology. These outbreaks affected 342 people, including seven people who were hospitalised.

The highest hospitalisation rate was seen in one outbreak of methaemoglobinaemia due to sodium nitrite, where all six notified cases were hospitalised; however, this outbreak was identified via the hospital cases. High hospitalisation rates were also seen in one outbreak of *Vibrio cholerae*, where 67% (2/3 people) of people affected were hospitalised, and in four different outbreaks of histamine, where in total 58% (7/12 people) of people affected were hospitalised.

Food vehicles

There was a wide variety of foods implicated in outbreaks of foodborne disease during 2006 (Table 9), although investigators could not identify a specific food vehicle in 40% (46/115) of outbreaks.

In 2006, eggs and egg-containing dishes were the most common food vehicle and were responsible for 14% (16/115) of foodborne outbreaks. These 16 outbreaks affected 191 people and hospitalised 64 people and all were due to salmonellosis. Outbreaks where investigators epidemiologically or microbiologically implicated eggs eaten alone, that is, not in a dish with other ingredients, or where there was a high degree of suspicion that eggs eaten alone were the responsible vehicle, were included in the egg category. An egg-containing dish was defined as a dish where eggs were one of the main ingredients but not the only ingredient or where cross-contamination from eggs was the cause of the outbreak. Food items included in this category included desserts commonly made with raw eggs, such as gateau (cake) or chocolate mousse, as well as other foods made with raw eggs such as milkshakes and raw pikelet dough. Other foods included were items suspected to be cross-contaminated with eggs in their preparation such as hamburgers and bakery products. Contaminated fish was the second most common food vehicle and was responsible for 11% (13/115) of foodborne outbreaks. Seven outbreaks were due to ciguatera fish poisoning and four outbreaks were due to histamine poisoning. Queensland reported five of the seven ciguatera outbreaks, while Victoria and the Northern Territory reported one ciguatera outbreak each. Two of the four histamine outbreaks were associated with the consumption of tuna, while the other two histamine outbreaks were associated with eating yellowtail kingfish. An outbreak of Vibrio cholerae was caused by consumption of contaminated whitebait imported from Indonesia.[19] Another outbreak of unknown aetiology was associated with the consumption of Nile perch fillets. An outbreak of unknown aetiology was associated with oysters; this outbreak was classified as seafood rather than fish.

There were eight outbreaks associated with mixed dishes; this category includes dishes made up of multiple ingredients as well as buffet meals where a wide variety of foods and dishes were served. These dishes contained a variety of ingredients, including vegetables, meats, and spreads/dressings, which made it difficult to assign the cause to one food category. Consumption of poultry was responsible for six outbreaks and meat other than poultry for four outbreaks.

Table 9. Categories of food vehicles implicated in foodborne disease outbreaks, Australia, 2006

Vehicle category	Number of outbreaks	People affected	Hospitalised
Fish	13	49	17
Egg-containing dish	11	125	50
Mixed dish	8	67	4
Poultry	6	97	3
Eggs	5	66	14
Meat, not poultry	4	94	0
Fresh produce	4	122	19
Sushi	4	17	1
Salad dish	3	38	0
Sandwiches	3	28	1
Cake	2	31	0
Processed meat	2	20	4
Dips	1	2	0
Sodium nitrite	1	6	6
Seafood	1	6	0
Water	1	46	0
Unknown	46	708	27
Total	115	1,522	146

Fresh fruits and vegetables, categorised as fresh produce in Table 9, were responsible for four outbreaks, all due to salmonellosis. Single outbreaks were associated with rockmelon, paw paw, alfalfa sprouts, and in one outbreak bean sprouts were suspected to be the cause.

Other food vehicles implicated in outbreaks included sushi (4 outbreaks), salad dishes (3 outbreaks), sandwiches (3 outbreaks), cake (2 outbreaks), and processed meat (2 outbreaks; one due to salami and one due to capocollo). Single outbreaks were due to dips and drinking water. There was one outbreak of methaemoglobinaemia due to sodium nitrite (sold commercially as 'nutre powder') used in the preparation of food.

Outbreak settings

The most common settings where food was prepared in outbreaks was restaurants (41%, 47/115), and private residences (13%, 15/115). Foods prepared at a takeaway or by commercial caterers were each responsible for 10 outbreaks (Table 10). Foods that were contaminated in primary production environments ('primary produce'), such as fish contaminated with ciguatera toxin and fresh fruits and vegetables contaminated with *Salmonella*, accounted for another 10 outbreaks. Food prepared in aged care facilities and by commercial manufacturers was responsible for five and four outbreaks respectively, while food prepared at bakeries and camps was responsible for two outbreaks each. There was one outbreak each due to food prepared in a child care centre, an institution other than an aged care home or hospital, and a national franchised fast food restaurant. There was one outbreak due to food prepared in the community; this was an outbreak where the suspected food vehicle, eggs, was prepared separately by individual households and resulted in a community wide increase of cases of *S.* Typhimurium 44.

Investigative methods and levels of evidence

States and territories investigated 31 outbreaks using retrospective cohort studies and seven outbreaks using case control studies. Forty-two per cent (13/31) of cohort studies were used for outbreaks of unknown aetiology, which was a similar proportion to previous years. In 69 outbreaks, descriptive information was used to attribute a foodborne cause or identify a food vehicle. No individual patient data was collected in the remaining eight outbreaks.

To attribute the cause of the outbreak to a specific food vehicle, investigators obtained analytical evidence from epidemiological studies in nine outbreaks. Microbiological evidence of contaminated food was found in 14 outbreaks, with a further seven outbreak investigations obtaining both microbiological and analytical evidence. Investigators obtained analytical and/or microbiological evidence for 41% (17/41) of *Salmonella* outbreaks, which was similar to the proportion in 2005 (39%). Seventy-three per cent (85/115) of outbreaks relied on descriptive evidence to implicate a food or foodborne transmission.

Table 10. Food preparation settings implicated in disease outbreaks, Australia, 2006

Setting category	Number of outbreaks	People affected	Hospitalised
Restaurant	47	442	26
Private residence	15	100	22
Takeaway	10	110	9
Commercial caterer	10	202	3
Primary produce	10	141	26
Aged care facility	5	46	4
Commercially manufactured	4	25	4
Bakery	2	25	1
Camp	2	112	2
Child care centre	1	4	0
Community	1	43	9
Institution	1	47	32
National franchised fast food	1	24	0
Other	5	196	8
Unknown	1	5	0
Total	115	1,522	146

Significant outbreaks

There were nine outbreaks affecting 40 or more persons in 2006. Three of these outbreaks were due to norovirus, two were due to *Clostridium perfringens*, two were due to *S.* Typhimurium and there was one each due to *S.* Saintpaul and *Campylobacter jejuni*. In total, these significant outbreaks affected 594 people, with an average of 66 people per outbreak (range 41–122 people) and 56 people were hospitalised.

Multi-state outbreaks

In 2006, OzFoodNet conducted three multi-state investigations into foodborne disease outbreaks. In May, there was an outbreak of *S.* Bovismorbificans 11 in Victoria and South Australia due to capocollo (processed meat) manufactured in Victoria. There were 13 cases from Victoria and two cases from South Australia. This outbreak prompted a consumer level recall of nationally distributed capocollo due to microbial contamination with *S.* Bovismorbificans 11.

In October 2006, New South Wales identified an increase in cases of *S.* Saintpaul and began interviewing cases. OzFoodNet coordinated a multi-state investigation team for this outbreak when other eastern Australian states also reported increases in cases of *S.* Saintpaul. The investigation team conducted a case control study that implicated rockmelons as the source of infection. Identifying the sources of implicated rockmelons was very difficult and various serotypes of *Salmonella* were isolated from rockmelons, in packing environments and on farms.[20]

In November 2006, Western Australia and Queensland investigated a multi-state outbreak of *S.* Litchfield associated with paw paw (papaya) grown in Western Australia. Paw paw was implicated as the responsible food vehicle in a case control study conducted by Western Australia. Food sampling demonstrated that paw paw sold in retail outlets in Western Australia (Perth) were contaminated with *S.* Litchfield, however, the source of the paw paw contamination on specific farms was not found.

Cluster investigations

During 2006, states and territories conducted investigations into 114 clusters where the mode of transmission was unknown. This included 50 clusters of *Salmonella*, two clusters of other pathogens, and 62 clusters of unknown aetiology. These clusters affected 1,070 people and hospitalised at least 40 people.

Forty-three per cent (50/114) of all cluster investigations were related to *Salmonella*, where the mean number of cases was 7.2 per cluster and the total number of persons affected was 360, with at least 15 people hospitalised. *S.* Typhimurium was responsible for 34% (17/50) of *Salmonella* cluster investigations. Clusters of *S.* Typhimurium and non-Typhimurium strains involved similar numbers of people, with a mean of 7.8 persons and 6.9 persons per cluster, respectively. Of the remaining 33 clusters, 26 different *Salmonella* serotypes other than Typhimurium were involved.

There were two investigations into clusters of other pathogens; these were both mixed infections of norovirus and *Clostridium difficile*, one in the community and one in an institution other than an aged care facility or a hospital.

There were 62 investigations into clusters of unknown aetiology where the mode of transmission was unknown. These 62 clusters affected 671 persons (an average of 10.8 cases per cluster) and hospitalised at least 25 persons. Thirty-two per cent (20/62) of these cluster investigations were in aged care facilities, and 11% (7/62) were in the community. Commercial caterers, restaurants, and child care centres each accounted for 8% (5/62) of these clusters.

Completeness of *Salmonella* serotyping and phage typing

Overall, 97.4% of *Salmonella* notifications on state and territory notification databases contained information about serotype and/or phage type (Figure 6). This was similar to the 2005 proportion of 97.5%. On the six serotypes where phage typing was typically performed—Bovismorbificans, Enteritidis, Hadar, Heidelberg, Typhimurium and Virchow—were all greater than 95% complete (Table 11). The Australian Capital Territory reported complete serotype and phage type information for all *Salmonella* notifications during 2006.

Figure 6. Proportion of *Salmonella* infections notified to state and territory health departments with serotype and phage type information available, Australia, 2001 to 2006

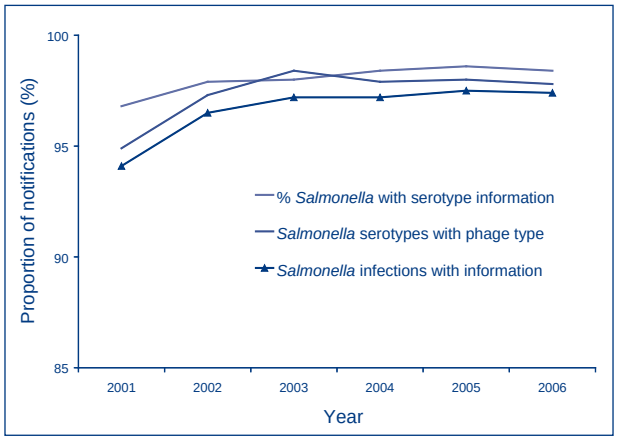


Table 11. Proportion of *Salmonella* infections notified to state and territory health departments with phage type information available for six serotypes, Australia, 2001 to 2006

	2001	2002	2003	2004	2005	2006
S. Bovismorbificans	87.5	96.3	94.6	94.1	94.7	95.7
S. Enteritidis	91.2	95.3	97.1	94.5	96.9	96.4
S. Hadar	77.4	90.9	97.1	89.7	92.0	100.0
S. Heidelberg	88.6	92.6	92.7	94.6	88.4	95.0
S. Typhimurium	96.9	97.9	98.8	98.8	98.7	98.1
S. Virchow	92.5	97.6	98.3	97.1	96.6	98.4

Discussion

This report summarises the rates of gastrointestinal diseases commonly transmitted by foods in Australia. Notification rates have remained stable in recent years, however the incidence was high compared to other countries.[21] The occurrence of campylobacteriosis has been consistently high in Australia and New Zealand compared with other developed countries for over a decade. This is despite identification of the main risk factors for infection.[21,22,23] Campylobacteriosis is responsible for a large burden of illness, but public health agencies are unable to recognise outbreaks due to the lack of a robust typing scheme.[24] In 2006, OzFoodNet sites identified four outbreaks of campylobacteriosis affecting 67 people compared with 15,492 notifications of this illness. There is a need to establish targets for the reduction of the incidence of campylobacteriosis in Australia as have been set in other countries, to assist governments and industry to make changes that will prevent illness.[25]

In 2006, there was an increase in the number of outbreaks relating to eggs and fresh produce. Sixteen outbreaks were associated with eggs or egg-based dishes, compared with five outbreaks in 2005. Eighty-one per cent (13/16) of these outbreaks were due to various phage types of *S.* Typhimurium. Investigations into outbreaks associated with eggs are challenging, as traceback of eggs to their source and to identify the origin of contamination can be difficult. OzFoodNet epidemiologists worked closely with primary production departments and food safety agencies in these outbreaks to ensure a complete traceback of eggs where possible. Food Standards Australia New Zealand has established a committee to develop a national primary production and processing standard for eggs, which, in the long term should reduce the number of outbreaks associated with eggs and egg products.[26]

There were four outbreaks associated with fresh produce in 2006, compared to one outbreak due to fresh produce in 2005. These four outbreaks, including two multi-state outbreaks, highlight the role of fresh fruits and vegetables in causing foodborne disease outbreaks. Although the implicated produce was traced back to farms in two of the outbreaks, the exact source of contamination was difficult to identify. Investigations of the farms producing rockmelons and paw paws revealed multiple *Salmonella* serotypes from a wide range of environmental samples; in particular, the water used to wash the produce during processing was contaminated with multiple *Salmonella* serotypes. This finding highlights a critical point of contamination. Since 2001, there have been 25 outbreaks associated with fresh produce in Australia.[27] The recent increase in produce-related outbreaks in Australia has also been seen in other developed countries. In particular, the USA reported several large outbreaks associated with the consumption of spinach,[28] lettuce,[29] and tomatoes[30] in 2006. There is a need for appropriate health messages for the public consuming potentially contaminated fresh produce as well as appropriate interventions to prevent contamination at the farm level.

In 2006, there were two outbreaks related to imported foods. One outbreak was associated with whitebait from Indonesia. The second outbreak was associated with sodium nitrite ('nutre powder') from China; sold in Chinese grocery stores as a flavour enhancer.[19] From 2001 to 2006, there have been 13 outbreaks associated with imported foods in Australia.[31] These two imported food outbreaks highlight the need to maintain communication with countries that provide Australia with food or items used in food. The outbreak of methaemoglobinaemia due to sodium nitrite was the first of its kind in Australia. Unintentional consumption of sodium nitrite has been the cause of outbreaks in other countries.[32,33]

In 2006, there were four outbreaks associated with sushi. These outbreaks are assumed to be due to inadequate refrigeration/storage.[34] While there are few published reports of gastrointestinal outbreaks associated with sushi, there are many potential avenues for contamination including improper storage of cooked rice and the use of high risk ingredients such as raw-egg mayonnaise.[35]

In 2006, more than 97% of *Salmonella* notifications contained complete information about serotype and/or phage type. The ability to type strains of *Salmonella* was essential for identifying and investigating outbreaks. The principal methods of differentiating *Salmonella* strains are serotyping and phage typing. Serotyping in Australia is conducted by public health reference laboratories in Queensland, New South Wales, Victoria, South Australia, and Western Australia. Tasmania, the Australian Capital Territory, and the Northern Territory forward their *Salmonella* isolates to South Australia or Victoria for serotyping and/or phage typing. Phage typing is conducted by the Microbiological Diagnostic Unit, Public Health Laboratory at the University of Melbourne in Victoria and the Institute of Medical and Veterinary Sciences in South Australia. During 2006, some jurisdictions used other methods to assist in locally differentiating *Salmonella* including multiple-locus variable-number tandem-repeats analysis and pulsed-field gel electrophoresis. Changes to *Salmonella* typing schemes need to be monitored to ensure that they enhance the ability to identify outbreaks and trends in the incidence of infection.

In 2006, all jurisdictions contributed to a fortnightly national report, which identified clusters of foodborne illness occurring across state and territory boundaries. This report was useful for identifying common events affecting different parts of Australia. This supplemented information sharing on a closed list server, teleconferences and at quarterly face-to-face meetings. In addition, OzFoodNet made greater use of web-based databases during the management of outbreaks, in particular the multi-state outbreaks, which greatly improved the timeliness and quality of these investigations.

In 2006, OzFoodNet sites reported 1,544 outbreaks, which was the largest number reported since surveillance began in 2000. The majority of these outbreaks were due to person-to-person transmission of highly infectious norovirus. While some settings, such as aged care homes and hospitals, show up more frequently in these investigations, outbreaks are easier to recognise, and therefore, report, where people live in close quarters. Better strategies are required to control norovirus.[36]

OzFoodNet reported a rate of 5.6 foodborne disease outbreaks per million population in 2006. This compares with rates of outbreak reporting in other developed countries. New Zealand reported a rate of 35 foodborne outbreaks per million population for 2006.[37] Published data on foodborne outbreak rates is available from 2004 for Germany (15 outbreaks per million population)[38] and from 2005 for the USA (estimated rate, 3.3 outbreaks per million population).[39,40] Direct comparisons of these rates are difficult due to the many differences in the surveillance of and reporting of outbreaks in each of these countries.

It is important to recognise some of the limitations of the data used in this report. Limitations of NNDSS surveillance data include differences in the likelihood that certain population groups will have laboratory tests and different testing regimes. This may explain part of the difference in the rates of laboratory-confirmed disease between jurisdictions and over time. Small numbers of notifications also mean that caution is required in the interpretation of differences between jurisdictions and over time. Importantly, some of the most common enteric pathogens are not notifiable, particularly norovirus, *Clostridium perfringens* and enteropathogenic *E. coli*. These organisms may be notified as the cause of outbreaks, but not as individual cases of disease. A limitation of the outbreak data provided by OzFoodNet sites for this report is the potential for variation in categorising features of outbreaks depending on investigator interpretation and circumstances. States and territories are working towards harmonising surveillance and outbreak data to address some of these issues.

Foodborne disease surveillance provides information to assist in the assessment of food safety policies and campaigns. A national program of surveillance for foodborne diseases and outbreak investigation has many benefits including identifying foods that cause human illness. Ongoing efforts to strengthen the quality of these data will ensure continued use by agencies to develop food safety policy and prevent foodborne illness.

Acknowledgements

We would like to thank the many epidemiologists, project officers, interviewers and research assistants at each of the OzFoodNet sites who contributed to this report. We also acknowledge the work of various public health professionals and laboratory staff around Australia who interviewed patients, tested specimens, typed isolates and investigated outbreaks. We would particularly like to thank jurisdictional laboratories, the Microbiological Diagnostic Unit Public Health Laboratory, the Institute of Medical and Veterinary Science, and the National Enteric Pathogen Surveillance Scheme for their help with foodborne disease surveillance in 2006. The quality of their work was the foundation of this report. OzFoodNet is an initiative of the Australian Government.

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Appendix

Foodborne outbreak summary for OzFoodNet sites, Australia, 2006

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Evidence	Epidemiological study	Responsible vehicle
Australian Capital Territory	February	Bakery	Unknown	10	0	D	D	Cake
	November	Primary produce	*Salmonella* Typhimurium 44	4	1	D	D	Free range eggs
	December	Primary produce	*Salmonella* Typhimurium 170	13	0	M	D	Free range eggs
New South Wales	January	Camp	*Salmonella* Typhimurium 170	3	1	D	D	Suspect chicken/beef hamburger cross-contaminated with eggs
	January	Restaurant	Unknown	13	0	D	D	Buffet meal
	January	Restaurant	Unknown	19	1	D	N	Unknown
	February	Restaurant	Scombroid	2	1	D	N	Tuna steaks
	February	Takeaway	Unknown	3	0	D	D	Chicken schnitzel in gravy
	March	Restaurant	*Salmonella* Typhimurium 170 var	2	2	D	D	Suspect pork dish or fried ice cream
	March	Restaurant	Unknown	2	0	D	D	Unknown
	March	Takeaway	*Salmonella* Montevideo	3	2	M	D	Plain hamburger cross-contaminated with eggs
	March	Restaurant	Unknown	22	0	D	C	Unknown
	April	Restaurant	Norovirus	15	0	D	C	Unknown
	May	Restaurant	Unknown	7	2	D	C	Unknown
	May	Commercial caterer	*Clostridium perfringens*	70	0	M	D	Chicken curry
	June	Commercially manufactured	*Salmonella* Typhimurium 170	2	0	D	D	Unknown
	June	Other	Unknown	3	0	D	D	Suspect potato salad
	June	Private residence	Unknown	4	0	D	D	Suspect Nile perch
	June	Private residence	Unknown	6	0	D	C	Suspect oysters
	June	Restaurant	Unknown	8	0	D	D	Unknown
	June	Private residence	Unknown	21	0	A	C	Cake
	July	Takeaway	*Salmonella* Typhimurium 135A	2	1	D	D	Suspected eggs
	July	Restaurant	Unknown	2	0	D	D	Unknown
	July	Child care centre	*Salmonella* Potsdam	4	0	D	D	Suspect pikelets made from whole eggs
	July	Takeaway	*Salmonella* Typhimurium 170	4	2	D	D	Suspect beef/chicken burgers with eggs
	July	Takeaway	*Salmonella* Typhimurium 170	4	3	M	D	Eggs

Foodborne outbreak summary for OzFoodNet sites, Australia, 2006, *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Evidence	Epidemiological study	Responsible vehicle
New South Wales, *continued*	July	Restaurant	Unknown	4	1	D	N	Buffet meal
	July	Restaurant	Unknown	5	0	D	D	Unknown
	July	Commercial caterer	Unknown	5	0	D	N	Unknown
	September	Commercially manufactured	*Salmonella* Typhimurium 170	2	0	D	D	Suspect dips
	September	Restaurant	Unknown	2	0	D	C	Chicken pizza
	September	Takeaway	Unknown	4	0	D	D	Unknown
	September	Restaurant	Unknown	5	0	D	D	Unknown
	September	Private residence	Sodium nitrite	6	6	M	N	Nutre powder
	September	Restaurant	Unknown	7	0	D	N	Pasta or pizza
	October	Restaurant	Scombroid	6	6	D	D	Yellowtail kingfish fillets
	October	Commercially manufactured	*Salmonella* Typhimurium 170	6	0	D	D	tuna and salmon sushi rolls
	November	Aged care facility	*Campylobacter jejuni*	3	3	AM	C	Undercooked chicken
	November	Private residence	*Vibrio cholerae*	3	2	D	D	Whitebait
	November	Restaurant	Unknown	7	0	D	N	Unknown
	November	Commercial caterer	*Bacillus cereus*	14	0	AM	C	Cooked chicken
	November	Restaurant	Unknown	15	0	D	D	Sandwiches
	November	Institution	*Salmonella* Typhimurium 170	47	32	AM	CCS	White chocolate mousse
	December	Restaurant	Unknown	5	0	A	CCS	Unknown
	December	Restaurant	Unknown	24	0	D	D	Banquet meal
	December	Commercial caterer	Unknown	25	0	D	C	Unknown
	December	Takeaway	*Clostridium perfringens*	80	0	AM	CCS	Roast pork
Northern Territory	January	Restaurant	Hepatitis A	10	1	D	D	Unknown
	May	Private residence	*Salmonella* Oslo	2	0	D	D	Suspected sticky rice balls with chicken
	September	Primary produce	Ciguatera fish poisoning	14	4	D	D	Slate sweetlips fish

Foodborne outbreak summary for OzFoodNet sites, Australia, 2006, *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Evidence	Epidemiological study	Responsible vehicle
Queensland	January	National franchised fast food	Unknown	24	0	D	C	Unknown
	February	Primary produce	Ciguatera fish poisoning	2	0	D	D	Cod
	February	Private residence	Scombroid	2	0	D	D	Blue fin tuna steaks
	February	Camp	Norovirus	66	2	D	D	Unknown
	March	Primary produce	Ciguatera fish poisoning	2	0	D	D	Trevally fish
	March	Primary produce	Ciguatera fish poisoning	4	4	D	D	Spanish mackerel
	March	Restaurant	Unknown	8	0	D	C	Unknown
	March	Restaurant	Norovirus	15	1	D	C	Unknown
	April	Restaurant	*Salmonella* Singapore	2	1	D	D	Chow mein
	April	Takeaway	*Staphylococcus aureus*	3	0	D	D	Sushi roll
	April	Commercial caterer	Unknown	6	3	D	D	Unknown
	April	Private residence	*Salmonella* Typhimurium 135a	11	5	D	D	Unknown
	May	Restaurant	Unknown	2	0	D	N	Chicken teriyaki sushi roll (nori roll)
	June	Restaurant	*Salmonella* Zanzibar	3	1	D	D	Unknown
	July	Primary produce	Ciguatera fish poisoning	2	0	D	D	Spanish mackerel
	July	Takeaway	Unknown	4	0	D	D	Suspected beef/lamb kebab
	July	Restaurant	Unknown	6	0	D	C	Unknown
	July	Restaurant	*Clostridium perfringens*	13	0	M	C	Chicken & lamb guvec
	August	Restaurant	*Salmonella* Typhimurium 135	6	1	D	D	Suspected chicken teriyaki sushi rolls
	September	Restaurant	*Clostridium perfringens*	6	0	D	D	Lamb korma
	September	Private residence	*Salmonella* Typhimurium 8	7	1	D	D	Unknown
	October	Primary produce	Ciguatera fish poisoning	4	0	D	D	Black kingfish
	November	Camp	*Campylobacter jejuni*	46	0	A	C	On-site water tank
	December	Restaurant	*Salmonella* Bareilly	4	0	D	D	Unknown
	December	Restaurant	*Salmonella* Typhimurium 197	7	3	D	D	Unknown
	December	Restaurant	Unknown	9	0	D	C	Unknown
	December	Restaurant	*Salmonella* Typhimurium 197	17	0	D	C	Unknown
	December	Other	Norovirus	122	1	A	C	Unknown

Foodborne outbreak summary for OzFoodNet sites, Australia, 2006, *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Evidence	Epidemiological study	Responsible vehicle
South Australia	January	Private residence	*Salmonella* Typhimurium 108	7	0	M	D	Homemade ice cream and ice cream topping
	February	Private residence	*Salmonella* Typhimurium 135	4	0	M	D	Silverside
	February	Restaurant	*Salmonella* Anatum	5	0	D	D	Beef burger with bacon and egg
	May	Other	*Salmonella* Typhimurium 108	23	7	AM	CCS	Ravioli
	June	Restaurant	*Salmonella* Typhimurium 9	6	0	A	C	Sweet potato and feta cheese salad
	December	Commercial caterer	*Campylobacter*	5	0	A	C	Chicken dish
	December	Bakery	*Salmonella* Typhimurium 9	15	1	AM	CCS	Egg through a bakery product
Tasmania	January	Private residence	*Salmonella* Typhimurium 44 and U302	9	2	D	C	Unknown
Victoria	January	Private residence	*Salmonella* Typhimurium 44	4	4	M	D	Milkshake containing raw egg
	January	Aged care facility	Unknown	5	0	D	D	Unknown
	January	Restaurant	Norovirus	9	0	D	D	Unknown
	January	Restaurant	Norovirus	15	0	D	C	Unknown
	February	Restaurant	Scombroid	2	0	M	D	Kingfish
	February	Commercial caterer	Norovirus	41	0	D	C	Unknown
	March	Unknown	*Salmonella* London	5	0	M	D	Salami (non commercial)
	March	Restaurant	*Salmonella* Saintpaul	11	1	M	D	Suspected bean shoots
	May	Commercial caterer	Suspected *Clostridium perfringens*	10	0	D	D	Unknown
	May	Primary produce	*Salmonella* Oranienburg	15	2	M	D	Alfalfa
	June	Aged care facility	Unknown	5	0	D	D	Unknown
	August	Commercial caterer	Unknown	7	0	D	C	Sandwiches
	August	Aged care facility	*Campylobacter*	13	1	D	D	Unknown
	September	Commercial caterer	Unknown	19	0	D	C	Unknown
	October	Restaurant	Norovirus	15	0	D	C	Unknown
	November	Primary produce	Ciguatera fish poisoning	2	0	D	D	Coral perch or coral trout
	November	Private residence	*Salmonella* Typhimurium 44	10	1	A	D	Hazelnut gateau cake made with raw egg mousse filling

Foodborne outbreak summary for OzFoodNet sites, Australia, 2006, *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Evidence	Epidemiological study	Responsible vehicle
Victoria, *continued*	November	Restaurant	Norovirus	13	0	D	C	Unknown
	November	Restaurant	Norovirus	29	0	D	C	Unknown
	December	Aged care facility	*Clostridium perfringens*	20	0	D	D	Unknown
	December	Community	*Salmonella* Typhimurium 44	43	9	D	D	Suspected eggs
Western Australia	June	Restaurant	*Salmonella* Anatum	6	1	D	D	Takeaway sandwiches and rolls
	September	Restaurant	*Salmonella* Kiambu	3	1	D	D	Unknown
	September	Restaurant	*Salmonella* Kiambu	35	2	D	C	Unknown
	October	Other	Unknown	19	0	D	C	Unknown
	November	Other	Norovirus	29	0	A	C	Salad
Multi-state	May	Commercially manufactured	*Salmonella* Bovismorbificans 11	15	4	M	D	Capocollo
	October	Primary produce	*Salmonella* Saintpaul	79	12	A	CCS	Rockmelon
	November	Primary produce	*Salmonella* Litchfield	17	4	AM	CCS	Paw paw

Surveillance report for active trachoma, 2006

National Trachoma Surveillance and Reporting Unit

Betty Tellis, Jill E Keeffe, Hugh R Taylor

Abstract

The National Trachoma Surveillance and Reporting Unit (NTSRU) was established in November 2006 to improve the quality and consistency of data collection and reporting of active trachoma in Australia. Active trachoma data collected in 2006, prior to the commencement of the NTSRU, were reported by the Northern Territory, South Australia and Western Australia. In most regions, Aboriginal children aged 5–9 years were screened for signs of active trachoma, following the World Health Organization simplified trachoma grading system. In the Northern Territory the Healthy School Aged Kids program conducted school-based screening for active trachoma in 74 schools in five regions (n=2,253). In South Australia Aboriginal school children presented for active trachoma screening when an eye team visited five Aboriginal Community Controlled Health Services (n=275). In Western Australia population health units in collaboration with staff from population health care services, conducted school based screening for active trachoma in 53 schools in four regions (n=1,719). Regional active trachoma prevalence for 2006 varied between the states and the Northern Territory with reported prevalences ranging from: Northern Territory = 2.5%–30%, South Australia = 0%–25%, and Western Australia = 18%–53%. Few data were reported on facial cleanliness or other aspects of the SAFE strategy, and no data were reported for trichiasis. *Commun Dis Intell* 2007;31:366–374.

Keywords: active trachoma, Australia, Northern Territory, South Australia, Western Australia, trachoma surveillance, SAFE strategy.

Introduction

Trachoma is the most common cause of infectious blindness with Australia the only developed country to still have blinding endemic trachoma.[1,2]

Thirty years ago the National Trachoma Eye Health Program found hyperendemic prevalence (>20%) of active trachoma in Aboriginal children.[3] Recent surveys that spanned 1989–1996 reported a similar story.[4,5] A review of the National Aboriginal and Torres Strait Islander Eye Health Program in northern and western Australia in 2003, found prevalence of active trachoma similar to those of 30 years ago.[6]

The Communicable Diseases Network Australia (CDNA) published the *Guidelines for the Public Health Management of Trachoma in Australia* to standardise methods for data collection and reporting of active trachoma prevalence and management.[2] This follows the principals of the World Health Organization (WHO) SAFE strategy for trachoma control that includes Surgery for trichiasis, Antibiotics for active trachoma, screening for Facial cleanliness and Environmental improvement.[7]

The National Trachoma Surveillance and Reporting Unit (NTSRU) was established in November 2006 with funding from the Australian Government to improve the overall quality and consistency of data collection and reporting on trachoma in Australia.

The NTSRU is responsible for:

- collecting trachoma data from the Northern Territory, South Australia and Western Australia;
- providing high quality national information on trachoma prevalence based on data received from the states and the Northern Territory;
- monitoring and reporting on antibiotic resistance to azithromycin where trachoma control activities are currently being undertaken;
- establishing a database that is to be consistent with the CDNA trachoma guidelines that is to be secure and confidential; and
- developing data collection forms that are culturally appropriate using language consistent with the policy in the CDNA trachoma guidelines and agreed to by the Trachoma Reference Group.

The NTSRU is advised by the Trachoma Reference Group and informed by the CDNA guidelines and existing surveillance units already in operation throughout Australia.

The purpose of this paper is to present data from the first surveillance report compiled by the NTSRU.

Methods

Case definition

Active trachoma refers to the presence of trachomatous inflammation-follicular (TF) and/or trachomatous inflammation-intense (TI), using the World Health Organization simplified trachoma grading classification system.[8] Later stages of trachoma are trachomatous scarring (TS), trachomatous trichiasis (TT) and corneal opacity (CO) (Appendix 1). Signs of trachoma are not mutually exclusive and should be graded independently. People are classified by their worst eye.

Hyperendemic prevalence usually refers to a prevalence of active trachoma of 20% or more in children aged 1–9 years.

Screening and data collection

Trachoma data for 2006 were reported by the Northern Territory, South Australia and Western Australia prior to the uniform adoption of the CDNA guidelines and the establishment of the NTSRU.[2]

Data for 2006 on screening of Aboriginal children for trachoma in schools or communities reported some or all of the following:

- date screening was conducted;
- trachoma grading classification used;
- number of schools or communities that conducted screening within the region of the state or territory;
- number of children that were examined in the school or community;
- age ranges of children examined less than 5 years, 5–9 years and 10–15 years;
- prevalence of active trachoma in children;
- number of children examined for clean faces;
- cases of trachomatous scarring; and
- information about treatment with azithromycin for affected children and their household and community contacts.

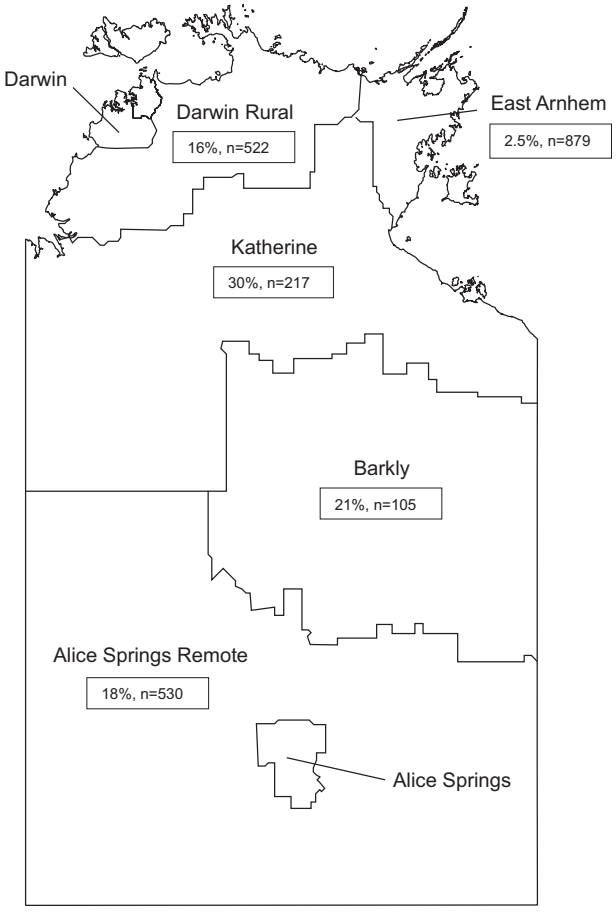
Data on TT and trichiasis surgery were not reported for 2006, however these will be reported in future surveillance reports.

Each state and territory determined the communities to be targeted for trachoma screening. School or community names were replaced with individual codes so that data from individual communities cannot be identified in this report.

Northern Territory

Trachoma screening was conducted from March to December 2006 by the Healthy School Aged Kids program in the Top End and Central Australia. Aboriginal Community Controlled Health Organisations (ACCHOs) also conducted screening. Population health workers screened Aboriginal children in all health regions (Map 1).

Map 1. Prevalence of active trachoma in Aboriginal children, Northern Territory, 2006, by region

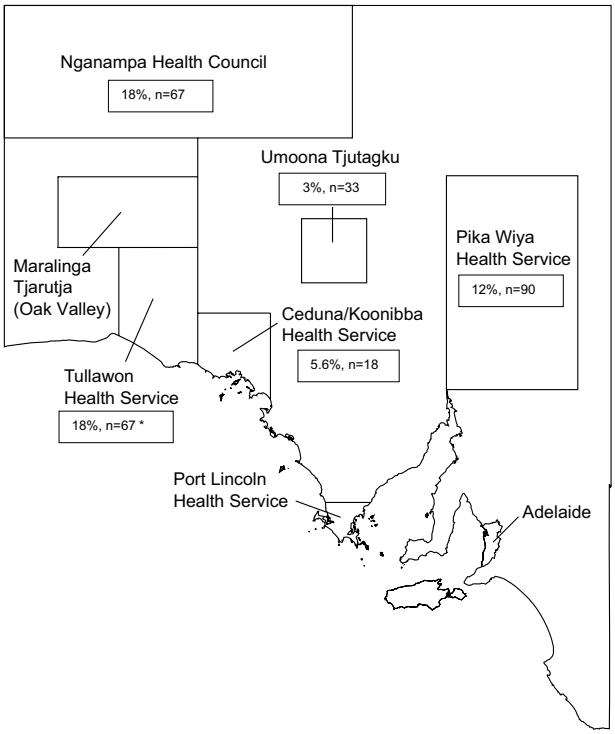


South Australia

Five areas serviced by Aboriginal Community Controlled Health Services (ACCHS) were visited by the screening team: Nganampa, Tullawon, Ceduna/Koonibba, Umoona Tjutagku and Pika Wiya (Map 2). The community of the Maralinga Tjarutja (Oak Valley) ACCHS was reported with the Tullawon ACCHS data. Screening for active trachoma was conducted twice throughout the year, from March to July and from August to December 2006 by all of these ACCHS, except the Ceduna/

Koonibba community that was visited once and the Umoona Tjutagku community that was visited three times. The screening team visited from 1–4 communities on each visit, and in many cases the combined data of groups of communities were reported. The data were reported by the Eye Health and Chronic Disease Specialist Support Program. Some children were seen at school and others were brought to the clinics by family members, Aboriginal health workers and other clinic staff when the ophthalmologists, optometrists and the screening coordinator visited the communities.

Map 2. Prevalence of active trachoma in Aboriginal children, South Australia, 2006, by Aboriginal Community Controlled Health Services

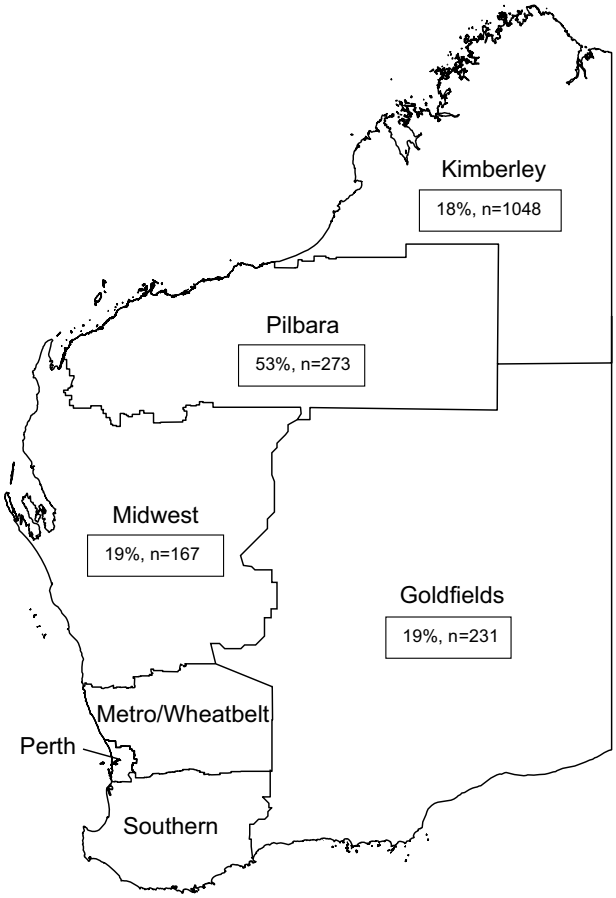


* The prevalence for Tullawon ACCHS includes data from Maralinga Tjarutja (Oak Valley) Aboriginal Community Controlled Health Services.

Western Australia

Trachoma screening was conducted from March to November 2006 by population health units working in collaboration with staff from primary health care services in four population health regions where trachoma is endemic, i.e. the Kimberley, Pilbara, Midwest and the Goldfields (Map 3). Only the Kimberley reported data on facial cleanliness, and only the Kimberley and Midwest regions reported data on treatment with antibiotics; details are reported in the results.

Map 3. Prevalence of active trachoma in Aboriginal children, Western Australia, 2006, by region



Data analysis

The state and territory maps used to present prevalence by region and ACCHS were created in Adobe illustrator version 10. Information from state and territory sources was used to define boundaries for regions and ACCHS.[9–11]

The proportion of children screened from within regions of the Northern Territory, South Australia and Western Australia was calculated using the Australian Bureau of Statistics (ABS) 2001 Census data.[12] The number of Aboriginal children reported by the ABS as being enrolled in pre– and primary schools was used as the denominator. In South Australia, the ABS 2001 Census data were reported for two out of three regions: Ceduna and Port Augusta. Children from the Ceduna/Koonibba and Tullawon ACCHS were reported within the Ceduna region and children from the Nganampa, Umoona Tjutagku and Pika Wiya ACCHS were reported within the Port Augusta region.

Regional prevalence figures were computed by aggregating community data of the number of children affected compared with the number of children

screened for active trachoma (Maps 1–3). In South Australia the prevalence was based on data from all occasions that the communities were visited. The proportion of communities with prevalences of 0%, 1% to <5%, 5% to <10%, 10% to <20%, 20% to <50% or ≥50% were reported in tables to illustrate communities with endemic and hyperendemic trachoma.

Results

The ABS 2001 Census data provide a means of comparison for the number of children examined within regions and ACCHS.[12] The number of endemic and hyperendemic communities within each region or ACCHS are reported as well as the number of communities that reported zero prevalence of active trachoma.

Northern Territory

A total of 2,253 Aboriginal children aged 1–9 years were screened in 73 schools or communities. Trachoma was graded using the WHO grading classification.

Prevalence by region varied from 2.5% to 30% (Map 1). The proportion of children examined in regions in the Northern Territory also varied: Alice Springs Remote = 42%, Barkly = 17%, Darwin Rural = 33%, East Arnhem = 74% and Katherine = 20% (Table 1). In the Northern Territory, 30 schools/communities reported zero prevalence of active trachoma, six reported prevalences between 10% and 19% and 22 reported prevalences ≥20% (Table 2). Five children in the Northern

Table 1. Number of Aboriginal children screened and the prevalence of active trachoma, Northern Territory, 2006, by region

	Alice Springs Remote	Barkly	Darwin Rural	East Arnhem	Katherine
Aboriginal population of children 0–14 years*	2,720	1,187	3,228	2,802	2,835
ABS school enrolment data†	1,273	616	1,573	1,190	1,065
Children targeted for screening	NR	NR	NR	NR	NR
Examined for active trachoma‡	530	105	522	879	217
Active trachoma prevalence§ (%)	18	21	16	2.5	30

* Data from the Australian Indigenous Geographical Classification Maps and Census Profiles, 2001.

† Australian Bureau of Statistics data of Aboriginal children enrolled in Government, Catholic and other non-government pre– and primary schools.

‡ Children aged 1–9 years were examined for active trachoma in Northern Territory schools/communities.

§ The number of children examined for active trachoma was used as the denominator to calculate the prevalence of active trachoma.

NR Not reported.

Table 2. Prevalence of active trachoma in Aboriginal children aged 1–9 years for communities, Northern Territory, 2006, by region

Community prevalence of active trachoma (%)	Number and proportion of communities with active trachoma*									
	Alice Springs Remote		Barkly		Darwin Rural		East Arnhem		Katherine	
	n	%	n	%	n	%	n	%	n	%
0	11	41	3	50.0	7	44	4	33.3	5	46
1 to <5	2	7	1	16.7	0	0	4	33.3	0	0
5 to <10	1	4	0	0.0	2	12	4	33.3	0	0
10 to <20	4	15	0	0.0	1	6	0	0.0	1	9
20 to <50	7	26	1	16.7	3	19	0	0.0	1	9
≥50	2	7	1	16.7	3	19	0	0.0	4	36
Total	27	100	6	100	16	100	12	100	11	100

* Of the 73 schools/communities that reported data, 30 of these communities had less than five children screened.

Territory were reported as having TS. Results of screening provided no information on facial cleanliness or TT.

Treatment

No information about antibiotic treatment was reported for 2006.

South Australia

A total of 275 Aboriginal children were examined in 17 schools/communities that were funded by the Eye Health and Chronic Disease Specialist Support Program. Data were reported for children aged 1–9 years, however it was acknowledged that the ages of the children could not be verified. The classification system used to grade trachoma was not specified.

Prevalence by ACCHOs varied from 3% to 18% (Map 2). The proportion of children examined in schools/communities in South Australia varied between the ACCHS and the screenings; in the first screening Ceduna = 12%, Port Augusta = 7% and in the second screening Ceduna = 10% and Port Augusta = 7.4% (Table 3). In the first series of screening in South Australia no schools/communities reported zero prevalence of active trachoma, three reported prevalences between 10% and 19% and three reported prevalences ≥20% (Table 4). In the second round of screening in South Australia three schools/communities reported zero prevalence of active trachoma, one reported prevalence of 12.8% and two reported prevalences ≥20%. In their third round of screening, the Umoona Tjutagku ACCHS reported zero prevalence of active trachoma for the children that were examined. Results of screening provided no information on facial cleanliness or TT.

Table 3. Number of Aboriginal children screened and the prevalence of active trachoma, South Australia, 2006, by regions serviced by an Aboriginal Community Controlled Health Service

	Ceduna/Koonibba		Port Augusta	
	Screening 1	Screening 2	Screening 1	Screening 2
Aboriginal population of children 0–14 years*	775	775	2,310	2,310
ABS school enrolment data†	380	380	1,186	1,186
Children targeted for screening	NR	NR	NR	NR
Examined for active trachoma‡	46	39	84	88
Active trachoma prevalence§ (%)	17	31	6	10

* Data from the Australian Indigenous Geographical Classification Maps and Census Profiles, 2001.

† Australian Bureau of Statistics data of Aboriginal children enrolled in Government, Catholic and non-government pre– and primary schools.

‡ The ages of the children screened could not be verified.

§ The number of children examined for active trachoma was used as the denominator to calculate the prevalence of active trachoma.

NR Not reported.

Table 4. Prevalence of active trachoma in Aboriginal children, South Australia, 2006, by communities serviced by an Aboriginal Community Controlled Health Service

Community prevalence of active trachoma (%)	Number of communities*				
	Ceduna/ Koonibba	Umoona Tjutagku	Tullawon†	Nganampa	Pika Wiya
0	0	0	0	0	0
1 to <5	0	0	0	0	0
5 to <10	1	0	0	0	1
10 to <20	0	1	0	2	0
20 to <50	0	0	1	1	1
≥50	0	0	0	0	0

* Data were provided for groups of communities, and in one of these groups only four children were screened.

† Includes data from the Maralinga Tjarutja (Oak Valley) boriginal Community Controlled Health Service.

Treatment

All children found to have active trachoma were referred to the clinics to be treated with antibiotics, except in the March screening of Ceduna/Kooniba where this information was not known.

Western Australia

A total of 1,719 Aboriginal children were screened from 53 schools/communities reported by Western Australia (Map 3). Data from the Pilbara region graded active trachoma as the presence of one or more follicles under the upper eyelid, and the Goldfields region did not specify the grading system that was used; others used the WHO grading classification.

Regional prevalence varied from 18% to 53% (Map 3). The proportion of children examined in regions in Western Australia varied: Kimberley = 62%, Pilbara = 33%, Midwest = 14% and the Goldfields = 21% (Table 5). Five schools/communities reported zero prevalence of active trachoma, six reported prevalences between 10% and 20% and 31 reported prevalences ≥20% (Table 6).

The Kimberley was the only region that provided data on facial cleanliness; of the 1,272 children examined for clean faces 939 were aged 1–9 years and 88% of them had clean faces. Reports from screening provided no information regarding TT.

Table 5. Number of Aboriginal children screened and the prevalence of active trachoma, Western Australia, 2006, by region

	Kimberley	Pilbara‡	Midwest	Goldfields
Aboriginal population of children 0–14 years*	5,101	1,702	2,335	2,284
ABS school enrolment data†	2,466	837	1,195	1,099
Children targeted for screening	2,624	NR	NR	NR
Examined for active trachoma§	1,521	273	167	231
Active trachoma prevalence‖ (%)	16	51	19	19

* Data from the Australian Indigenous Geographical Classification Maps and Census Profiles, 2001.

† Australian Bureau of Statistics data of Aboriginal children enrolled in Government, Catholic and non-government pre– and primary schools.

‡ Grading of TF ≥ 1 follicle under the upper eyelid.

§ Data for children aged 1–14 years were reported for the Kimberley and Pilbara regions, children aged 1–9 years were reported for the Midwest; and the Goldfields did not specify the ages of the school children screened.

‖ The number of children examined for active trachoma was used as the denominator to calculate the prevalence of active trachoma.

NR Not reported.

Table 6. Prevalence of active trachoma in Aboriginal children aged 1–9 years for communities, Western Australia, 2006, by region

Community prevalence of active trachoma (%)	Number and proportion of communities with active trachoma*							
	Kimberley		Pilbara†		Midwest		Goldfields	
	n	%	n	%	n	%	n	%
0	1	3	1	10	1	17	2	33
1 to <5	3	10	0	0	0	0	0	0
5 to <10	5	16	1	10	1	17	1	17
10 to <20	5	16	0	0	1	17	0	0
20 to <50	12	39	2	20	2	32	3	50
≥50	5	16	6	60	1	17	0	0
Total	31	100	10	100	6	100	6	100

* Of the 53 communities that reported data one, had fewer than five children screened (4 children), however the Kimberley did not provide information on the number of children screened within each community.

† Grading of TF = ≥ 1 follicle under the upper eyelid.

Treatment

The Kimberley Population Health Unit treated all children who showed clinical signs of active trachoma, with antibiotics at the time of screening provided consent had been granted. In some schools where the prevalence in the 1–9 year age group was greater than 10%, children aged 10–14+ years were treated with antibiotics regardless of their infection status. Household contacts of affected children were followed up in the community and given treatment with azithromycin. Where local knowledge was available to the Health District 90% of household contacts were treated with antibiotics. Of the 22 schools with active trachoma prevalence above 10%, 20 required community-wide treatment; in the remaining two communities cases were clustered.

The Midwest Population Health Unit treated affected children as soon as possible after the completion of screening using the WHO criteria.

Discussion

This report confirms previous reports that trachoma continues to be endemic in the Northern Territory, South Australia and Western Australia.[4,6] Most regions and ACCHS reported endemic trachoma for the communities that were screened in 2006, while hyperendemic trachoma was reported for 57 of the 133 schools or communities. The different grading criterion used by the Pilbara region may have led to an overestimation of active trachoma prevalence. Similarly, the small number of children examined in many communities may have resulted in imprecise estimates of the extent of active trachoma in other areas.

The proportion of children screened in each region showed that in most cases less than half of the children enrolled in pre– and primary schools were examined.[12] No specific information is available about the screening of children aged less than 5 years and school aged children who were not at school. As active trachoma is highest in young children, it would be advisable if children under five years were examined and accurately represented in the prevalence.[13] Limited information regarding the target population to be screened makes it difficult to accurately assess the screening coverage rates for children in trachoma endemic areas.

Some regions or ACCHS, such as East Arnhem (NT) and Ceduna/Koonibba (SA), reported <10% prevalence of active trachoma for all communities that were screened. Similarly, there were communities in each state and territory that reported zero prevalence of active trachoma. Repeated screening of communities or regions for some years is required before they can be designated as 'trachoma free'. The CDNA guidelines state that annual screening of endemic communities is required until active trachoma is less than 5% for five consecutive years.[2] The states and territory have not reported historical data for those communities that were no longer targeted for screening because trachoma is thought to be no longer present.

There were almost no data reported on facial cleanliness. The lack of facial cleanliness has been found to be a risk factor for reinfection and this is a key component of the SAFE strategy.[14,15] If children are not being examined for clean faces at the same time as they are screened for active trachoma, it is difficult to assess the success of health promotion campaigns that aim to break the cycle of reinfection.[7] Appropriate programs to promote awareness of the disease and implement environmental improvements need to be negotiated with individual high risk communities.[16]

Few data were reported on treatment of children with active trachoma and their household and community contacts. Where this information was provided, the timing of antibiotic administration after screening was not always specified. In some cases guidelines have been implemented differently in different regions.[5] For example, in some areas azithromycin was reported to have been given to affected children and sometimes to family members. It seems possible that the incomplete implementation of the SAFE strategy, and restricted antibiotic coverage, may explain the relatively small change in active trachoma observed over time in some communities, compared with the successful control of trachoma reported from other countries.[17,18]

Reporting data on later stages of trachoma is also important as this gives an indication of the history and progression of the disease in endemic communities. No data were reported on the presence of trichiasis or the performance of trichiasis surgery. The Surgery component of the SAFE strategy for treatment of the end stage of trachoma is important as without any intervention and follow-up, trichiasis will go on to cause irreversible blindness.[19] Without this information we have an incomplete picture of the full cycle of the disease and as a consequence are unable to adequately address the changes that need to be made to trachoma control programs.

The 2006 data reported were collected prior to the uniform adoption of the CDNA guidelines and the establishment of the NTSRU. The CDNA guidelines call for reporting of screening and trachoma control activities in children and adults. According to the CDNA guidelines, trachoma should be reported in children aged <5, 5–9, and 10–15 years and the WHO simplified grading should be used.[2]

The collection of data regarding trichiasis and trichiasis surgery will indicate the extent of the end stage of this disease and the implications for blindness in Aboriginal adults. Compliance with the CDNA guidelines on all aspects of the SAFE strategy, and specifically the treatment of affected children and household and community contacts, is critical to eliminate trachoma.

This report is confined to reported data on the trachoma screening of children with almost no information on facial cleanliness and treatment. The adoption of standardised methodology and coverage of communities will provide better data on the prevalence of active trachoma so that Australia is able to contribute compatible information for the global trachoma reports.[2] In 2007, reporting of data should specify the areas and communities that have trachoma and those where the absence of trachoma has been established. Data on all components of the SAFE strategy as well as monitoring of antibiotic resistance in remote Aboriginal communities will also be provided in future surveillance reports.

Acknowledgements

Funding

The National Trachoma Surveillance and Reporting Unit is supported by funding from the Australian Government Department of Health and Ageing.

Trachoma Reference Group

The National Trachoma Surveillance and Reporting Unit is advised by the Trachoma Reference Group, members of which include representatives from the following organisations:

Office for Aboriginal Torres Strait Islander Health, Australian Government Department of Health and Ageing

Surveillance Policy and Systems Section, Office of Health Protection, Australian Government Department of Health and Ageing

Centre for Disease Control, NT Department of Health and Community Services

Communicable Disease Control Directorate, Department of Health, Western Australia

Kimberley Population Health Unit, Department of Health, Western Australia

National Aboriginal Community Controlled Health Organisation

Country Health South Australia, Eye Health and Chronic Disease Specialist Support Program, Aboriginal Health Council of South Australia

Co-opted members as required

Public Health Laboratory Network

Data collection

The organisations that assisted in the collection and/or reporting of the data were:

Northern Territory

Centre for Disease Control, Northern Territory Department of Health and Community Services, Northern Territory

Healthy School Aged Kids Program: Top End

Healthy School Aged Kids Program: Central Australia

South Australia

Eye Health and Chronic Disease Specialist Support Program, Aboriginal Health Council of South Australia

Country Health South Australia

Ngananampa Health Council

Maralinga Tjarutja (Oak Valley) Health Service

Tullawon Health Service

Ceduna/Koonibba Health Service

Umoona Tjutagku Health Service

Pika Wiya Health Service

Western Australia

Communicable Diseases Control Directorate, Department of Health, Western Australia

Population Health Units and Aboriginal Community Controlled Health Services staff in the Goldfields, Kimberley, Midwest and Pilbara regions

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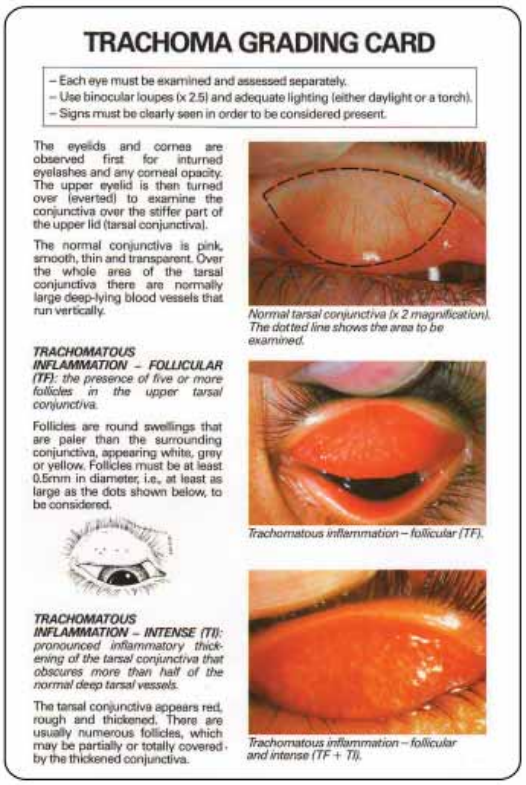
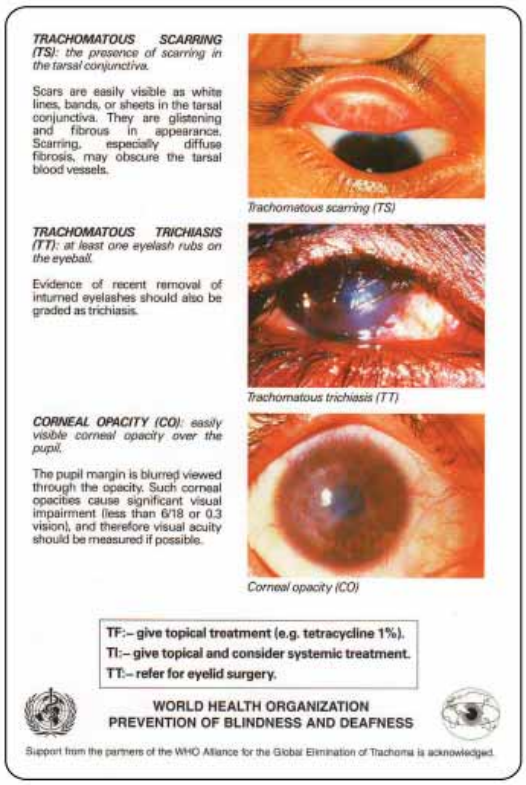
Appendix

World Health Organization simplified grading classification system





Australian Rotavirus Surveillance Program: Annual report, 2006–07

Carl D Kirkwood, David Cannan, Nada Bogdanovic-Sakran, Ruth F Bishop, Graeme L Barnes and the National Rotavirus Surveillance Group

Abstract

The National Rotavirus Reference Centre, together with collaborating laboratories Australia-wide, conducts a laboratory based rotavirus surveillance program. This report describes the serotypes of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2006 to 30 June 2007. One thousand and two faecal samples from across Australia were examined using a combined approach of monoclonal antibody immunoassays, reverse transcription-polymerase chain reaction and polyacrylamide gel analysis. Serotype G1 was the dominant serotype nationally, representing 36.7% of all strains, followed by serotype G9 (31.1%), and serotype G3 (23.3%). Serotype G2 represented less than 5% of strains, while no serotype G4 strains were identified. All G1, G3 and G9 strains assayed for P genotype contained the P[8] genotype, bar one G1 strain, which possessed a P[6]. Uncommon rotavirus genotypes, G8 (n=1) and G12 (n=2) were identified in children with acute gastroenteritis during this study period. *Commun Dis Intell* 2007;31:375–379.

Keywords: annual reports, rotavirus, disease surveillance, epidemiology

Introduction

Rotaviruses are the single most important cause of dehydration, hospitalisation and death due to severe gastroenteritis in young children worldwide. An estimated 600,000 children die annually of severe diarrhoea, however few of these deaths occur in developed countries.[1] Rotavirus induced disease accounts for up to 50% of childhood hospitalisations for diarrhoea in Australia. This represents 10,000 children hospitalised each year,[2] costing an estimated $26 million in direct costs.

In an effort to decrease the huge social and economic burden of rotavirus disease in Australia, two new rotavirus vaccines (Rotarix® [GlaxoSmithKline] and Rotateq® [Merck]) have been licensed and included in the National Immunisation Program free of charge to all young infants from 1 July 2007. Both vaccines were demonstrated to be safe and highly effective in prevention of severe diarrhoea and hospitalisation due to rotavirus infections during large-scale phase III clinical and efficacy trials, each involving over 60,000 children worldwide.[3,4]

Since 1999 the Australian Rotavirus Surveillance Program, has reported the changing annual pattern of dominant serotypes together with the multiple types identified in the Australian population each year. Results highlight the diversity of rotavirus strains capable of causing disease in children. Of particular importance was the emergence in 1999 and dominance in 2002 of serotype G9 strains nationally,[5] as well as the recent re-emergence of serotypes G3 and G4 as major causes of acute gastroenteritis in Australian children.[5,6]

Surveillance of rotavirus serotypes will provide important data to inform rotavirus vaccine programs. The impact of these two widely used vaccines on the natural pattern of circulating rotavirus strains is unknown and difficult to predict, given the different components of each vaccine. Continuing serotype surveillance should identify the effects that each vaccine program has on circulating strains, in particular, whether changes occur in serotype incidence and whether increased proportions of rare or uncommon types result.

The surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children in Australia continues to be undertaken by the National Rotavirus Reference Centre in Melbourne, together with collaborating laboratories across Australia. In this report we describe the surveillance results for the period 1 July 2006 to 30 June 2007, and identify the geographic distribution of the predominant rotavirus serotypes causing disease in Australian children.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories were collected, stored frozen and forwarded to Melbourne, together with relevant age and sex of the patient. Specimens were then serotyped using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the five major group A human rotavirus serotypes (G1, G2, G3, G4 and G9).[7] Strains that could not be assigned a G serotype were genotyped by using a hemi-nested multiplex reverse transcription/polymerase chain reaction (RT-PCR), using G specific oligonucleotide primers.[8] P genotypes were determined by using a hemi-nested multiplex RT-PCR assay.[9]

Polyacrylamide gel electrophoresis (PAGE) was used to classify rotavirus strains genetically into electropherotypes, and to examine the extent of sharing of the same electropherotype between collaborating centres.

Results

Number of isolates

A total of 1,002 specimens were received for analysis from Melbourne and the collaborating centres in Western Australia, the Northern Territory, and New South Wales (Table). Eight hundred and twenty-seven specimens were confirmed as rotavirus positive using our in-house EIA assay. Specimens containing insufficient specimen for testing (n=27), or specimens that were not confirmed to be positive for rotavirus (n=148) were not analysed further.

Age distribution

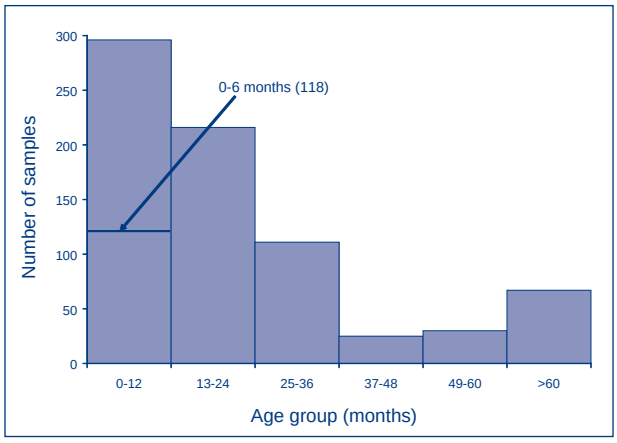
The overall age distribution of children with acute rotavirus gastroenteritis is depicted in the Figure. In the reporting period, 15.8% of cases were from infants aged 0–6 months, 23.9% of cases were from infants aged 7–12 months, 30% were from patients aged 13–24 months, and 14.9% were from patients aged 25–36 months. Overall, 84.6% of samples were from children aged 3 years or less, and 92% were from children aged 5 years or less.

During the study period, slightly more specimens from male than female children (n=446 vs. 309) were analysed.

Serotype distribution

The rotavirus serotypes identified in Australia from 1 July 2006 to 30 June 2007 are shown in the Table. Serotype G1 was the most common, representing 36.7% of all specimens, and was identified in all centres. It was the dominant strain in Sydney and Perth, and was the second most common type in Melbourne. Serotype G9 was the second most common serotype nationally, and represented 31.1% of specimens. It was identified in six of the eight collaborating centres but was the dominant type only in the Northern Territory, where it was responsible for a large outbreak of acute gastroenteritis between March and May 2007. Strains belonging to serotype G3 were the third most common type identified Australia-wide during this study period, and represented 23.3% of specimens. It was found in seven of the nine centres, and was dominant in Melbourne.

Cases of rotavirus, Australia, 1 July 2006 to 30 June 2007, by age group



Rotavirus G serotypes in Australia, 1 July 2006 to 30 June 2007

Centre	Total	Serotype													
		G1		G2		G3		G4		G9		mix		NR	
		%	n	%	n	%	n	%	n	%	n	%	n	%	n
Melbourne	180	29.4	53	13.9	25	39.4	71	0.0		14.4	26	0.0		2.8	5*
Sydney (POW)	32	75.0	24	0.0		0.0		0.0		21.9	7	0.0		3.1	1*
Sydney (Westmead)	25	32.0	8	28.0	7	8.0	2	0.0		24	6	0.0		8.0	2
Alice Springs	105	2.8	3	0.0		10.5	11	0.0		85.7	90	0.0		1.0	1
Darwin	141	2.8	4	0.7	1	16.3	23	0.0		75.2	106	0.7	1	4.3	6*
Western Diagnostic, NT	65	80.0	52	0.0		16.9	11	0.0		0.0		0.0		3.1	2
Perth	62	69.4	43	0.0		24.2	15	0.0		0.0		1.6	1	4.8	3
PathWest WA	217	53.9	117	2.3	5	27.7	60	0.0		10.1	22	0.5	1	5.5	12
Total	827	36.7	304	4.6	38	23.3	193	0.0	0	31.1	257	0.4	3	3.9*	32

An additional 175 specimens were omitted from analysis due to insufficient sample or because the specimen was not confirmed to be rotavirus positive.

* Two samples were identified as genotype G12 (Melbourne and Sydney) and one sample as genotype G8 (Darwin).

Serotype G2 strains were identified in four centres during the study, and represented less than 5% of the total strains identified. No serotype G4 strains were identified in any centre. Two genotype G12 strains were identified during the study, one in Sydney and one in Melbourne, while a single G8 strain was identified in Darwin.

P genotype was determined for 181 of the rotavirus positive samples. Sixty-four of the 65 G1 strains analysed were genotyped as P[8], and one sample was typed as P[6]. All of the G3 and G9 strains analysed were genotyped as P[8] (n= 41 and 70, respectively), while the five G2 strains analysed were all associated with P[4].

Less than 0.5% of the rotavirus samples contained multiple serotypes, and in 3.9% of the samples a serotype could not be identified. The latter could be samples with virus numbers below the detection limits of our assays, or could have contained inhibitors in extracted RNA that prevent the function of the enzymes used in RT and/or PCR steps. It is unlikely that these represent unusual serotypes not identified using standard methods, since none of the non-typeable isolates exhibited unusual PAGE patterns. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

Discussion

In 2006–2007 the Australian Rotavirus Surveillance Program showed that serotype G1 continued to remain the dominant serotype nationally, comprising 36.7% of all strains characterised. It was identified in all centres, and continues to be the dominant type on both sides of the country, in particular in Sydney and Perth. This survey continues to highlight the importance of serotype G1 as a major cause of disease in Australian children. Similarly, serotype G1 continues to be reported as the dominant type in epidemiological studies conducted throughout the world.[10,11]

Similar to previous reports, multiple serotypes continue to circulate within the Australian population, causing significant disease in Australian children during the study period. This serotype diversity is illustrated by G1, G3 and G9 strains, that were each identified in at least six locations and were each the dominant serotype in at least one site during 2006–07. In all bar one instance, each strain was associated with the P[8] VP4 protein. Thus G1P[8], G3P[8] and G9P[8] combinations were the predominant strains identified in children during the current surveillance period.

The 2006–07 reporting period was also characterised by another large outbreak of acute gastroenteritis in the Northern Territory during March to May 2007. Similar to the large outbreak in 2001, a serotype G9P[8] strain was identified as the causative type. The importance of serotype G9 Australia-wide has been reflected with the continued increase in predominance, as well as the nationwide distribution of G9 strains during the past 2 years.[12,13]

In the Northern Territory, Rotarix® (GlaxoSmith-Kline) a live attenuated human G1P[8] virus rotavirus vaccine, had been introduced by the government into the immunisation schedule at 2 and 4 months of age for children born after 1 August 2006. This occurred 11 months prior to the introduction of rotavirus vaccines into the National Immunisation Program. This early vaccine adoption has provided an opportunity to determine how effective the vaccine is in an outbreak setting. Although Rotarix does not contain a G9 VP7 protein, protection against G9 serotypes has been previously demonstrated in phase III clinical trials, probably mediated via the VP4 P[8] protein.[4] Preliminary studies of children admitted to hospital during the rotavirus outbreak in Alice Springs suggest that children who were vaccinated were protected against severe disease (Dr Julie Graham, unpublished observations). However, continued assessment of rotavirus vaccine efficacy is required to fully understand the impact rotavirus vaccine has on rotavirus disease.

The prevalence of serotype G3 has slightly increased during the current survey, being present in seven of eight sites, but more significantly it was the predominant type identified in Melbourne. This emergence of G3 in Melbourne completes the eastward spread of G3 across Australia. During the past 4 years, G3 has slowly increased in prevalence across Australia, initially being dominant in Western Australia in 2003–04, then Western Australia and Northern Territory (including both Alice Springs and Darwin) in 2004–05, then Alice Springs in 2005–06.[6,12,13] While G3 has been identified in small numbers in eastern states since 2003–04, this year represents the first instance when it has predominated. The movement of serotype G3 across Australia is similar to that seen previously for serotype G9 in the early 2000s.[14,15]

Uncommon rotavirus types continue to be of worldwide interest because of the possible impact they may have on rotavirus vaccine programs. This year, two uncommon types have been identified in Australian children. Strains exhibiting a genotype G12 VP7 protein were identified in Melbourne and Sydney extending the previous identification of G12 in 2005–06 in Sydney, to a second location. Thus identification of G12 strains in Australia continues the worldwide identification of this emerging serotype.[16,17] The second uncommon type identified during this survey was a single genotype

G8 strain seen in Darwin. This strain represents the first report of a G8 strain in Australia since 1996.[18] Thus these reports of uncommon strains continue to highlight their existence in Australia.

The rotavirus serotyping results from this survey, together with those of previous years, highlight the unpredictable nature of changes in the prevalence of rotavirus strains across Australia and the potential for new and emerging strains to spread throughout the continent. In addition, the identification of genotype G8 and G12 further illustrate the diversity of strains capable of causing severe disease in Australian children. The introduction of the two rotavirus vaccines into the National Immunisation Program occurred after the conclusion of this year's surveillance. Understanding the fluctuations in rotavirus serotypes, using multi-centre national surveillance, will provide valuable insight into vaccine efficacy over the next 3–5 years.

Acknowledgements

The Rotavirus Surveillance Program is supported by grants from the Australian Government Department of Health and Ageing, GlaxoSmithKline and CSL.

Dr Kirkwood is supported by an RD Wright Fellowship, National Health and Medical Research Centre.

Rotavirus positive specimens were collected from numerous centres throughout Australia. The significant time and effort involved in the collection, storage, packaging, compiling data and forwarding of specimens was much appreciated.

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Supplementary report: surveillance of adverse events following immunisation among children aged less than 7 years in Australia, 1 January to 30 June 2007

Glenda L Lawrence, Padmasiri E Aratchige, Richard Hill

Introduction

This report summarises national passive surveillance data collected by the Therapeutic Goods Administration (TGA) at 30 September 2007 for adverse events following immunisation (AEFI) reported for children aged less than 7 years who received vaccines between 1 January and 30 June 2007. The report includes all vaccines administered to children in this age group, with a focus on the vaccines included in the funded National Immunisation Program (NIP) schedule.[1]

The most recent change to the NIP schedule occurred on 1 November 2005 with the addition of a single dose of varicella vaccine at 18 months of age, and the replacement of oral poliovirus vaccines with combination vaccines containing inactivated poliovirus (IPV) for doses due at 2, 4 and 6 months of age and 4 years of age. All children receive IPV in combination with diphtheria-tetanus-acellular pertussis (DTPa) antigens (i.e. DTPa-IPV).[1,2] For doses due at 2, 4 and 6 months, some states and territories use combination vaccines that include hepatitis B (HepB) virus antigens (i.e. DTPa-IPV-Hepb; pentavalent) or both HepB and *Haemophilus influenzae* type b (Hib) antigens (i.e. DTPa-IPV-Hepb-Hib; hexavalent). Rotavirus vaccines were added to the NIP schedule on 1 July 2007: after this reporting period. However, there has been a funded rotavirus immunisation program for infants in the Northern Territory from August 2006[3,4] (using Rotarix), and two rotavirus vaccines have been available on the private market to children elsewhere in Australia.[1,4]

Average annual population-based AEFI reporting rates were calculated using mid-2005 population estimates. Reporting rates per 100,000 doses were calculated for vaccines on the NIP schedule using denominator data from the Australian Childhood Immunisation Register (ACIR). Rates were not calculated for the birth dose of HepB due to inaccurate reporting of doses to the ACIR.

All AEFI reports received by the TGA are reviewed by the Adverse Drug Reactions Advisory Committee (ADRAC), an independent expert advisory committee to the TGA. The data reported here are provisional only. It is important to note that an AEFI is defined as a medical event that is temporally associated with immunisation but not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system[5] methods used to analyse the data and information regarding limitations and interpretation of the data.[5–7] Often, several vaccines and reaction codes are listed in an AEFI record so the number of both vaccines and reaction codes will exceed the total number of AEFI records. For the purpose of this report, an AEFI is defined as 'serious' if there is a code of life-threatening severity or an outcome code indicating recovery with sequelae, admission to hospital, prolongation of hospitalisation or death.

Results

All vaccines

There were a total of 176 AEFI records (annualised rate of 19.7 per 100,000 population) for children aged <7 years for vaccines administered in the first 6 months of 2007. This was a 34% decrease on the 266 records (29.8 per 100,000 population) for the corresponding six-month period in 2006. Thirty-eight per cent (n=66) of records were for children aged <1 year, 11% (n=19) for children aged 1 to <2 years and 52% (n=91) for children aged 2 to <7 years. This is similar to previous years,[6,8] except that there were fewer reports for children aged 1 to < 2 years in 2007. The male to female ratio was 1.2 to 1, the same as the previous year.[6]

Ten per cent (n=17) of the 176 records listed outcomes defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening event or death). This was less than reported for the same period in 2006 (13.5%). For the first 6 months of 2007, there were no reports of death and all 17 children with 'serious' AEFIs were admitted to hospital. Serious and other significant AEFIs reported included anaphylaxis (1), severe allergic reactions involving the cardiovascular and/or respiratory systems (2) and seizure (3). There were 14 reports of hypotonic-hyporesponsive episode (HHE).

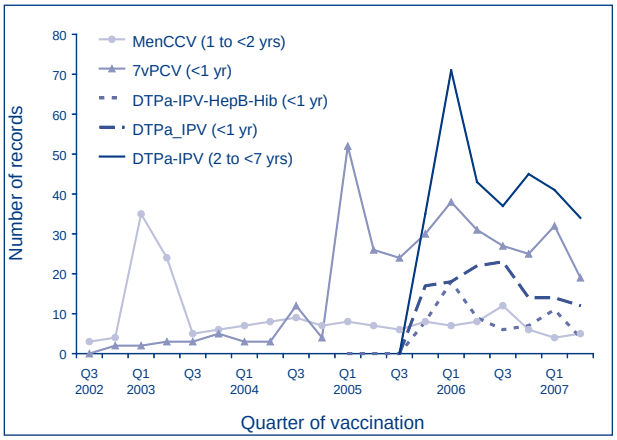
Of the 176 records, 13 listed one or more vaccines not included on the NIP schedule for children aged 2 months to <7 years as suspected of involvement in the reported AEFI. These were hepatitis B (n=2), influenza (n=3), and rotavirus (n=8) vaccines. There were a total of 14 reports for rotavirus vaccine including eight where it was the only suspected vaccine and six where NIP schedule vaccines were also listed as suspected of involvement in the reported AEFI. The most frequently reported signs listed in these 14 AEFI records were diarrhoea or vomiting (n=10) and fever (n=4).

National Immunisation Program schedule vaccines

One or more of the vaccines on the current NIP schedule for children aged 2 months or older were recorded as suspected of involvement in the reported adverse event for 163 of the 176 records analysed (Table). This is an AEFI reporting rate of 9.2 per 100,000 doses recorded on the ACIR with 1.0 per 100,000 doses defined as 'serious' AEFIs.

AEFI reporting rates per 100,000 vaccine doses were lower than for the same period in 2006 for all vaccines, age groups and reaction categories (Table). The largest reductions were for children aged 1 to <2 years and 2 to <7 years and for DTPa-containing vaccines, meningococcal C conjugate vaccine (MenCCV) and measles-mumps-rubella (MMR) vaccine. The reporting rate for AEFIs defined as 'serious' also decreased from 1.6 per 100,000 doses in 2006 to 1.0 per 100,000 doses in 2007. These changes appear to relate to a stabilisation of reporting to a baseline level after an initial increase following the introduction of multivalent IPV-containing vaccines in November 2005 (Figure), a reduction in the number of reports for the 1 to <2 year age group and a reduction in injection site reactions (ISR) following acellular pertussis containing vaccines at 4–5 years of age.

Reports of adverse events following immunisation, TGA database, 1 July 2002 to 30 June 2007, for vaccines recently introduced into the National Immunisation Program*



* Meningococcal C conjugate vaccine (MenCCV) was introduced into the NIP on 1 January 2003, 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005, and DTPa-IPV and DTPa-IPV-HepB-Hib vaccines in November 2005.

Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, Therapeutic Goods Administration database, January to June 2007

	AEFI records‡ (n)	Vaccine doses* (n)	Reporting rate per 100,000 doses§		
			Jan–June 2007	Jan–June 2006	Jan–June 2005
Vaccine†					
DTPa-containing vaccines	121	547,712	22.1	34.7	33.7
DTPa-IPV	101	345,564	29.3	44.2	–
Pentavalent (DTPa-IPV-HepB)	4	9,551	41.9	41.0	–
Hexavalent (DTPa-IPV-HepB-Hib)	16	192,597	8.3	16.8	–
Haemophilus influenzae type b	9	55,957	16.1	19.7	18.4
Haemophilus influenzae type b-hepatitis B	33	214,144	15.4	23.1	17.1
Measles-mumps-rubella	35	276,988	12.6	20.9	23.5
Meningococcal C conjugate	10	145,070	6.9	16.5	18.7
Pneumococcal conjugate	52	419,727	12.4	16.5	16.3
Varicella	14	131,065	10.7	13.3	–
Age group					
<1 year	56	990,723	5.7	8.6	6.2
1 to <2 years	19	488,695	3.4	7.5	7.5
2 to <7 years	88	311,245	28.3	38.7	25.4
AEFI category†					
Total	163	1,790,663	9.2	14.0	10.5
'Certain' or 'probable' causality rating	67	1,790,663	3.7	6.0	4.7
'Serious' outcome	17	1,790,663	1.0	1.6	0.6

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2007.

† Records where at least one of the nine vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[5] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.

‡ Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2007. More than one vaccine may be coded as 'suspected' if several were administered at the same time.

§ The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the Australian Childhood Immunisation Register.

AEFI reporting rates for the different DTPa-IPV combination vaccines varied by age group and vaccine type (Table). The type of DTPa-IPV vaccine (i.e. quadrivalent, pentavalent, hexavalent) delivered to children aged <1 year varies by jurisdiction. The pentavalent vaccine is only used in the Northern Territory where children have also received rotavirus vaccine since August 2006.

The reporting rate for quadrivalent DTPa-IPV vaccine includes reports for children aged <1 year who were scheduled to receive the vaccine at 2, 4, and 6 months of age (reporting rate of 12.7 per 100,000 doses) and reports for children aged 2 to <7 years (reporting rate of 55 per 100,000 doses). This is the lowest reporting rate for acellular pertussis-containing vaccines for children in the 2 to <7 year age group since 2002. Previously, this had consistently been over 90 per 100,000, due mainly to a high level of reporting of ISR.[6,8] In the first 6 months of 2007, the rate of ISR for DTPa-IPV vaccine declined to 48 per 100,000 doses, compared with 71 per 100,000 doses for the same period in 2006, and an average of 78 per 100,000 doses of DTPa vaccine for 2002–2005.

Discussion

There was a large reduction in AEFI reports to the TGA for vaccines administered to children aged <7 years in the first 6 months of 2007 compared with the corresponding period in 2006. The most plausible explanation for the reduction relates to changes in reporting practices for all vaccines and age groups, plus a large reduction in reports for ISR following DTPa-containing vaccines

among children aged 2 to <7 years. There may also have been some delayed reporting of AEFI for immunisations administered between January and June 2007, although in the analysis, we have included AEFI reports received by the TGA up to 30 September 2007.

The passive AEFI surveillance system is sufficiently sensitive to be able to detect changes in reporting practices that are known to occur following the introduction of new vaccines. In Australia, it is evident that there are initial high levels of reporting each time a new vaccine is introduced into the NIP schedule, followed by a reduction and stabilisation of reporting over time (Figure, Table). This appears to have occurred in the January to June period of 2007 compared with the first 6 months of 2006, for the vaccines introduced into the NIP schedule in November 2005. Immunisation providers are more likely to report suspected less serious AEFIs for vaccines with which they are not familiar.

Of particular interest is the reduction in the ISR reporting rate for acellular pertussis-containing vaccines among children aged 2 to <7 years.[9] This may reflect a birth cohort effect related to the removal from the NIP schedule in September 2003 of the dose due at 18 months of age.[1,2] A large majority of children receiving a school entry dose of DTPa-IPV in the first 6 months of 2007 would have received three doses of acellular pertussis-containing vaccines due at 2, 4, and 6 months of age, and a fourth dose at 4–5 years. The rate of ISR has fallen by 38% from 78 per 100,000 doses to 48 per 100,000 in the first 6 months of 2007.[6,7] This suggests that the removal of the dose due at 18 months of age has had a significant impact on ISR reporting rates for acellular-pertussis containing vaccines in this age group.

Conclusion

This report further demonstrates that changes to the NIP schedule are reflected in the national passive AEFI surveillance data.[6,8,10] The majority of AEFIs reported to the TGA were mild transient events and indicate the high safety level of the vaccines included in the NIP schedule. Close monitoring of passive AEFI surveillance data for vaccines administered to children continues through the TGA, in consultation with ADRAC and state and territory health departments.

Acknowledgements

We thank Mike Gold (ADRAC) and Peter McIntyre (NCIRS) for their contribution to this report. The National Centre for Immunisation Research and Surveillance is supported by the Australian Department of Health and Ageing, the New South Wales Health Department and the Children's Hospital at Westmead.

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Articles

The epidemiology of pertussis in the Australian Capital Territory, 1999 to 2005—epidemics of testing, disease or false positives?

Clare E Wylks, Ben Ewald, Charles Guest

Abstract

The increase in pertussis notifications since the 1990s in many countries, including Australia, has been attributed to improved diagnosis. This study aimed to describe the epidemiology of pertussis in the Australian Capital Territory from 1999 to 2005, determine whether the apparent changes could be accounted for by greater recognition and testing, and explore the impact of false positive serology results associated with faulty test kits. The Australian Capital Territory resident notification, laboratory and separation data from 1999 to 2005 were examined and the proportions of positive tests across time periods and age groups compared. Notification rates increased in the years 2000, 2003 and 2005. There was a shift in the age distribution of cases, from children and teenagers in 2000, to teenagers in 2003 and adults in 2005. Testing activity and notification activity were closely related. Comparing the epidemic periods to the preceding inter-epidemic periods, the proportion of positive tests was maintained or increased for all age groups combined and for adults and children (e.g. statistically significant increase from 7.8% to 14.0% in the 2005 epidemic in adults). During each epidemic the proportion of positive tests was statistically significantly higher in the age group with the highest notification activity. Despite similar testing rates in adults in 2003 and 2005, greater disease activity was reported in 2005. Although the numbers were small, polymerase chain reaction and culture positive test results increased in 2003 but not in 2005. The proportion of positive polymerase chain reaction results increased in 2003, providing strong evidence that the apparent epidemic of 2003 was due to a true increase in underlying disease activity. Because of the uncertainty surrounding the timing of the false positive serology results, the study provides weaker support for a true epidemic of pertussis in 2005. *Commun Dis Intell* 2007;31:383–391.

Keywords: whooping cough, *Bordetella pertussis,* epidemiology

Introduction

Since the 1990s, an increase in pertussis notifications has been reported for many countries, including Argentina, Australia, Canada, Italy, Japan, the Netherlands, Switzerland and the United States of America.[1,2] The increase has been noted particularly in adolescents and adults.[3,4,5]

Proposed reasons for increased incidence of infection include waning natural and vaccine induced immunity[6] and changes in the organism leading to a mismatch between the vaccine and circulating strains.[7] A New South Wales study concluded that the observed increase in pertussis notifications from 1988 to 2002 in adults reflected a true increase in disease.[8] Others have argued that the apparent increase may be due to increased recognition of disease that has been previously undetected.[9,10,11] This may occur through increased testing, particularly in the older age groups; the use of more sensitive tests;[12] or changes in reporting practices.

Disease due to waning immunity following natural infection in adults has certainly been documented at an individual level since the early 1900s[13] and studies of *Bordetella pertussis* infections in adolescents and adults during non-outbreak times suggest that the disease is common and endemic in this population.[14,15]

Many studies have examined trends in notification data, but few have also examined trends in the number and types of tests ordered. A study from British Columbia in Canada examined hospital separations, notifications and laboratory data during successive outbreaks during the 1990s and 2000, and demonstrated an increased incidence in 2000 in pre-teens and teens.[5]

This study examines Australian Capital Territory resident notification, laboratory and separation data from July 1999 to December 2005. There were no major changes in the notification case definition during this period. The use of serology for the diagnosis of pertussis was standard practice before the study period. Polymerase chain reaction

(PCR) became available in 2000 in the Australian Capital Territory and became widely used from 2003 onwards. Serology is thought to be more sensitive than PCR or culture[16] and studies have shown that the sensitivity of PCR compared to serology is around 60%.[11]

Pertussis is a diagnosis that is not easily made on clinical grounds alone, particularly in older children and adults. The typical whoop of whooping cough is generally not as frequent in adults,[9] in whom symptoms are indistinguishable from a viral upper respiratory tract infection in the early catarrhal phase, and a post viral cough in the later phase is common. The characteristic symptom in adults is a persistent cough, and testing for pertussis is often part of the investigation for a chronic cough. There is the potential for greater awareness of the disease among clinicians to cause an increase in the number of pertussis tests ordered.

This study aimed to describe the epidemiology of pertussis in the Australian Capital Territory from 1999 to 2005 and to determine whether the apparent changes could be accounted for by greater recognition and testing.

All three laboratories servicing the Australian Capital Territory used manufacturer A's *Bordetella pertussis* IgA enzyme-linked immunosorbent assay (ELISA) kit. In September 2006, the manufacturer, in consultation with the Therapeutic Goods Administration, issued a 'recall for product correction' for three batches of the kit which had been used in the Australian Capital Territory from mid-December 2005, because the cut-off determination point was set too low resulting in false positive results. We also aimed to explore the impact of this problem on the data.

Methods

Data sources

Notification data

Australian Capital Territory resident pertussis notifications from July 1999 to December 2005 were obtained from the Australian Capital Territory Health Protection Service. Data on cases that were notified, but excluded from the official notification data because they did not meet the surveillance case definition were also obtained. The surveillance case definitions used during the study period are specified in Table 1.

Testing data

Pertussis laboratory data on Australian Capital Territory residents was obtained for the same time period from the three major laboratories that service the region. There was a fourth laboratory that performed testing at the smaller of the two public hospitals from November 1999 to November 2002 but data from this laboratory was not available. As this hospital does not admit paediatric patients and adult pertussis rarely requires hospital admission, the pertussis testing data during this period from this laboratory is expected to only comprise a small proportion of the total tests. Recently, an additional New South Wales private laboratory has begun to service the Australian Capital Territory. A review of the 2005 notifications indicated that only one patient was tested at this laboratory, in December.

Laboratory Z provided the test details grouped by patient encounter (for example, if multiple tests were ordered on the same day for a patient, these were grouped). Laboratory Y was unable to provide culture data. The data provided by laboratory Z showed that during the study period, 1.8% (46 out of 2,506 patient encounters) of patients were tested by culture and no other method. Laboratory Z was not able to provide the results of some of its PCR tests in 2002 and 2003 – the missing results comprised 4.0% of the total PCR data in 2002 and 6.4% in 2003.

Serology was recorded as positive if IgA was detected. Laboratory X used only IgA antibodies to whole cell pertussis antigen, as did laboratory Y from September 2005 onwards. Laboratory Z and laboratory Y (prior to September 2005) used IgA and IgG to whole cell pertussis antigen. All three laboratories used manufacturer A's *B. pertussis* IgA ELISA kit.

Laboratory X sends samples to a reference laboratory for PCR testing – the method used is an in-house conventional PCR with end point fluorescence detection. Laboratory Y sends samples to another inter-state laboratory that uses an in-house real-time PCR assay performed on the Roche LightCycler instrument. Laboratory Z uses the Roche LightCycler real-time PCR.

Separation data

Australian Capital Territory resident hospital separation data for which the principal or other diagnosis was pertussis was obtained from the ACT Health Information Management Section from July 1999 to December 2005.

Analysis

Age-specific yearly and monthly notification rates were calculated using Australian Bureau of Statistics (ABS) mid-year population estimates.

Table 1. Surveillance case definitions

Time period	Probable case	Confirmed case
1997–2003[17]	A cough illness lasting 14 days or more with one or more of the following: paroxysms of coughing; inspiratory whoop or post-tussive vomiting, without other apparent case. OR A cough illness lasting 14 days or more in a patient with *B. pertussis*-specific IgA detected in serum.	*Laboratory confirmed* Isolation of *B. pertussis* from a clinical specimen. OR Positive PCR assay for *B. pertussis* undertaken in a laboratory with established expertise in the area. *Epidemiologically confirmed* A cough illness lasting 14 days or more in a patient who is epidemiologically linked to a laboratory confirmed case.
2003 onwards[18]	A probable case requires clinical evidence only: - a cough illness lasting two or more weeks; AND - paroxysms of coughing OR inspiratory whoop OR post-tussive vomiting.	A confirmed case requires: - laboratory definitive evidence; OR - laboratory suggestive evidence AND clinical evidence; OR - clinical evidence AND epidemiological evidence.* *Laboratory definitive evidence:* - isolation of *B. pertussis*; OR - detection of *B. pertussis* by nucleic acid testing. *Laboratory suggestive evidence:* - seroconversion or a significant increase in antibody level to *B. pertussis*; OR - single high IgA titre to whole cells; OR - detection of *B. pertussis* antigen by immunofluorescence assay (IFA). *Clinical evidence:* - a coughing illness lasting two or more weeks; OR - paroxysms of coughing OR inspiratory whoop OR post-tussive vomiting.

* The criteria for an epidemiologically confirmed case changed slightly to allow for a person with a cough of any duration who was epidemiologically linked to a confirmed case to be classified as a confirmed case.

An epidemic period was defined as one in which the monthly notification rate was greater than or equal to five per 100,000 for three consecutive months.

Age-specific testing rates were calculated using ABS mid-year population estimates as the denominator and an adjusted number of tests performed in each age group for the numerator (a rate based on the estimated number of people tested rather than on the total number of tests performed.) Based on the data provided by laboratory Z, the percentage of patients who received more than one test was calculated for each year of the study period. The adjusted testing rate was then calculated by reducing the total number of tests by these percentages. Less than 1% of patients received three tests in 2003, 2004 and 2005, so this was ignored. For these testing rates, the PCR tests for which the results were unavailable were still included in the number of tests.

The proportion of positive tests was calculated over time and across age groups. For these calculations, the total unadjusted number of tests was used but the PCR data for which results were unavailable were excluded. The number of positive results was used as the numerator rather than the number of notifications. Data from December 2005 were excluded due to the likelihood of false positive results.

As pertussis is a cyclical disease with epidemics occurring every 2–5 years,[10] the likelihood of detecting trends over time is influenced by the stage of the cycle. Therefore, the proportions of tests that were positive during the epidemic periods as defined above were compared to the proportions that were positive in the preceding inter-epidemic periods, and this was repeated for children and adults.

The median age of pertussis hospital separations each year was calculated.

Analysis was performed using Excel and STATA 8. Ninety-five per cent confidence intervals for the proportion of positive tests positive were calculated.

Ethics approval

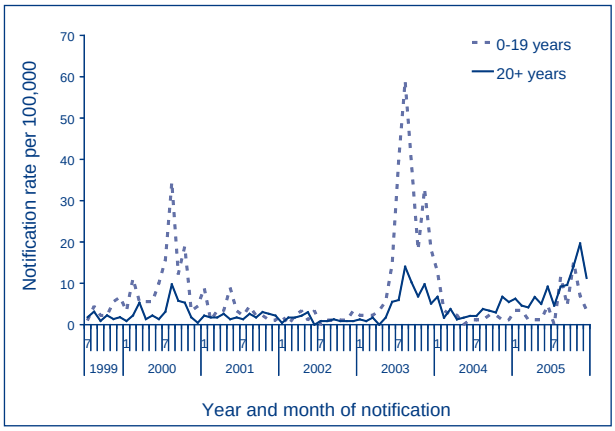
Ethics approval was obtained from the University of Newcastle Health Human Research Ethics Committee and the Australian Capital Territory Health Human Research Ethics Committee in 2005.

Results

Notifications

From July 1999 to December 2005 there were 1,180 pertussis notifications in the Australian Capital Territory. Females comprised 55.7% of the notifications. There were epidemics in 2000, 2003 and 2005. Figure 1 indicates that the peak monthly notification rate for those aged 20 years or greater was higher in 2005 than in 2000 or 2003. The annual notification rate in 2003 in this age group was 62.9 per 100,000 population and this increased to 104.6 per 100,000 population in 2005.

Figure 1. Monthly pertussis notification rate, Australian Capital Territory, July 1999 to December 2005, by age group

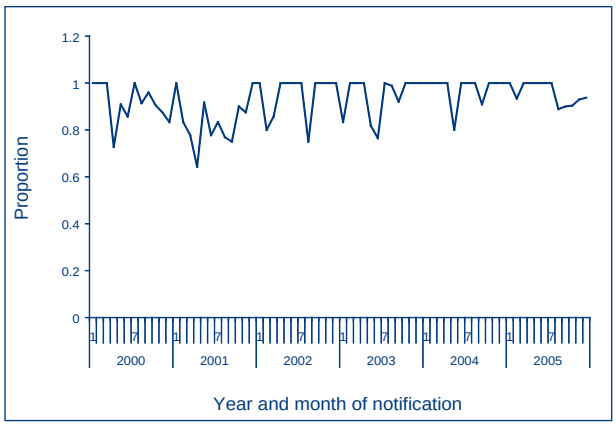


Over these time periods there were changes in the age distribution of cases. During 2000, the highest notification rates were in those aged 10–14 years (294.9 per 100,000) and 5–9 years (108.1 per 100,000). In 2003, the highest notification rates were in those aged 10–14 years (510.9 per 100,000) and 15–19 years (298.9 per 100,000). Those aged 40-49 years had a notification rate of 129.2 per 100,000 population. In 2005 the highest notification rate was in those aged 50–59 years (124.0 per 100,000) and those aged 60 years or greater (123.2 per 100,000). The proportion of cases in those aged greater than or equal to 20 years increased from 41.5% in 2003 to 83.6% in 2005.

Epidemics occurred in July–October 2000, June 2003–January 2004, and August–December 2005. Those aged 0–19 years experienced epidemics in February–October 2000, May 2003–January 2004, and August–November 2005 and those aged 20 years or greater in August–October 2000, June 2003–January 2004, and November 2004–December 2005 (although in those aged 20 years or greater the notification rate dropped to 4 per 100,000 in March 2005).

During the study period, there were very few cases notified that did not meet the surveillance case definition. Figure 2 demonstrates that there was no obvious trend in the proportion that did meet the case definition over time.

Figure 2. Proportion of all notified pertussis cases that met the surveillance case definition, Australian Capital Territory, 2000 to 2005

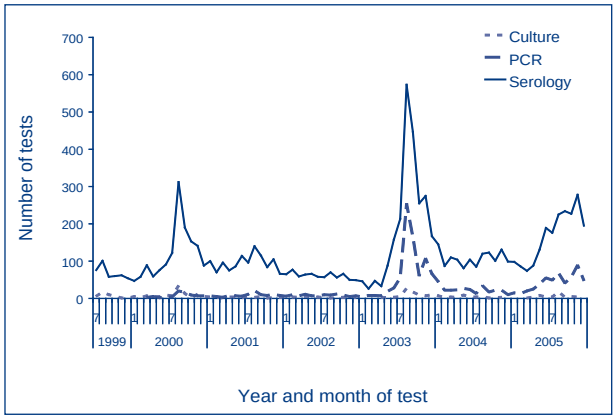


Testing data

Testing rates

During the study period, 11,600 pertussis tests were ordered on Australian Capital Territory residents (not including the missing culture data but including the PCR tests for which results were not available). Figure 3 shows that the number of tests increased during 2000, 2003 and 2005. Most of the total testing was by done by serological methods, with PCR becoming more common from 2003 onwards.

Figure 3. Pertussis tests performed, Australian Capital Territory, July 1999 to December 2005, by method



Based on examination of the data provided by laboratory Z, 1.6% of patients were tested using more than one method in 1999 and this increased over time to 9.7% in 2003 and 9.3% and 13.1% in 2004 and 2005 respectively. The combination was usually PCR and serology. However, the number of patients with at least one positive test result when tested via multiple methods was small. For example, in 2003 only 1.9% of all testing encounters involved a positive test result in a patient who was tested by multiple methods, and this only increased slightly to 2.0% in 2004 and 2.1% in 2005. Of the 196 patients who were tested via more than one method, there were only three instances (one each in 2003, 2004 and 2005) where more than one result was positive.

Figure 4 demonstrates that, excluding the serology data, there was an increase in positive tests in 2000 and 2003, but not in 2005.

Figure 4. Pertussis positive tests, PCR and culture only, Australian Capital Territory, July 1999 to December 2005

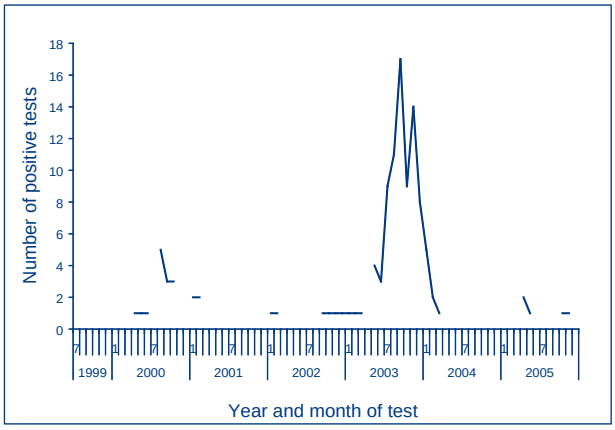


Figure 5 compares the notification and adjusted testing rates. Testing and notification activity were very closely related. Testing activity didn't appear to significantly precede or lag behind the notifications, but was sustained slightly for a period (for example, in 2001 and 2004) after the end of the epidemic.

Adjusted testing rates in adults and children are shown in Figure 6. During the 2003 epidemic the testing rate was higher in all age groups compared to preceding and following years. The testing rate was highest in those aged 10–14 years (192.2 per 10,000) and 15–19 years (157.8 per 10,000). In 2005, the adjusted testing rate in adults (72.4 per 10,000) was similar to the rate in 2003 (72.3 per 10,000). The highest rate in 2005 was in those aged less than one year (142.0 per 10,000), followed by those aged 40–49 years (81.0 per 10,000) and those aged 50–59 years (75.3 per 10,000). The adjusted

Figure 5. Pertussis monthly notification and adjusted testing rates, Australian Capital Territory, July 1999 to December 2005

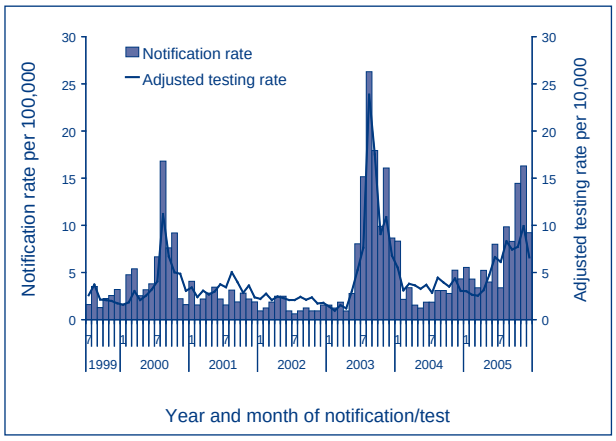
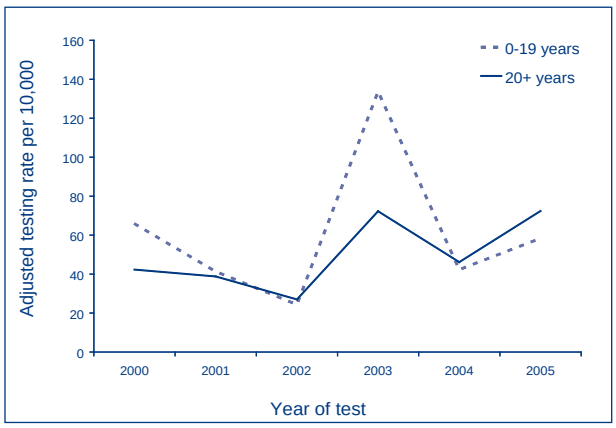


Figure 6. Adjusted yearly pertussis testing rates, Australian Capital Territory, 2000 to 2005, by age group



age specific testing rates were higher in 2003 than in 2005 for those aged 30–39 years, 40–49 years and 50–59 years. In 2005, adjusted age specific testing rates did not increase noticeably in the younger age groups except for those aged less than one year.

Proportion of positive test results

Tables 2 and 3 show the proportion of positive tests during epidemic and non-epidemic periods for those aged 0–19 years and 20 years or greater. Comparing the epidemic periods to the preceding inter-epidemic periods, the proportion stayed the same or increased overall (data not shown) and for both children and adults. The increase in the proportion of positive tests was statistically significant overall for the 2003 epidemic. In children, the increase was statistically significant in the 2003 epidemic, and in adults, was statistically significant in the 2005 epidemic. During each epidemic, the proportion of positive tests was statistically significantly higher in the affected age group, for example, 19.8% in children

Table 2. Proportion of pertussis tests positive in the Australian Capital Territory during epidemic and non-epidemic periods, age 0–19 years

Period	Proportion of tests positive	Percentage of tests positive	95% confidence interval
Jul 99–Jan 00	22/152	14.47	9.30, 21.09
Feb 00–Oct 00*	99/501	19.76	16.36, 23.52
Nov 00–Apr 03	70/751	9.32	7.34, 11.63
May 03–Jan 04*	210/1,293	16.24	14.27, 18.37
Feb 04–Jul 05	28/585	4.79	3.20, 6.84
Aug 05–Nov 05*	15/293	5.12	2.89, 8.30

* Epidemic period.

Table 3. Proportion of pertussis tests positive in the Australian Capital Territory during epidemic and non-epidemic periods, age 20+ years

Period	Proportion of tests positive	Percentage of tests positive	95% confidence interval
Jul 99–Jul 00	63/689	9.14	7.10, 11.55
Aug 00–Oct 00*	47/452	10.40	7.74, 13.59
Nov 00–May 03	134/1,914	7.00	5.90, 8.24
Jun 03–Jan 04*	157/1,789	8.78	7.51, 10.18
Feb 04–Oct 04	68/868	7.83	6.13, 9.83
Nov 04–Nov 05*	278/1,993	13.95	12.46, 15.55

* Epidemic period.

in 2000 versus 10.4% in adults, 16.2% in children in 2003 versus 8.8% in adults and 14.0% in adults in 2005 compared to 5.1% in children. Comparing the 2003 and 2005 epidemics in adults, the proportion of positive tests was statistically significantly higher in 2005 (14.0% versus 8.8%).

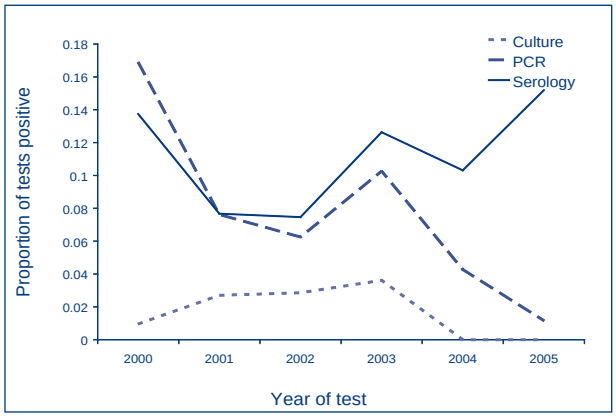
Figure 7 shows the proportion of positive test results by method for each year from 2000 to 2005. The proportion of positive test results via PCR was high in 2000 and 2003, and fell to its lowest level in 2005. The proportion of positive serology results was high in 2000 and 2003, and reached its highest level in 2005. The proportion of positive culture results was fairly steady but fell to its lowest level in 2005.

Children comprised 63.6% of the PCR tests in 1999 and this decreased to 39.0%–52.8% between 2000 and 2005. Overall in this population, serology was more likely to yield a positive test result than PCR or culture.

Hospital separation data

Table 4 summarises the hospital admissions (based on hospital discharge data) for which the primary or secondary diagnosis was pertussis during the study period. During the entire study period, 26% of admissions were in those aged 20 years or greater, and in 2005, this age group accounted for 50% of the admissions.

Figure 7. Proportion of pertussis tests positive, Australian Capital Territory, by test and year, 2000 to 2005



Discussion

Interpretation is complicated by false positive serological results. The product recall in September 2006 was for three batches of manufacturer A's pertussis IgA serology kit, which had been used in the Australian Capital Territory since mid-December 2005. If this was the earliest time at which the faulty kits were used in the Australian Capital Territory, this study does provide evidence that the increases in notifications during the study period were asso-

Table 4. Pertussis hospitalisations in the Australian Capital Territory (ACT residents) from July 1999 to December 2005

Year	Admissions: age less than 1 year	Admissions: age greater than or equal to 1 year (age of cases in years)	Median age of admission	Total
1999	4	1, (73)	0	5
2000	5	6, (2, 6, 10, 38, 46, 75)	2	11
2001	3	3, (29, 30, 64)	15	6
2002	2	1, (58)	0	3
2003	4	4, (1, 12, 17, 28)	1	8
2004	7	0	0	7
2005	2	4, (4, 21, 30, 57)	13	6

ciated with a true increase in underlying disease incidence and not merely the result of increased awareness and testing, for the following reasons:

- Comparing the epidemic periods to the preceding inter-epidemic periods, the proportion of positive tests increased, or at least stayed constant, overall, and in both children and adults.
- The proportion of positive tests during the epidemics was statistically significantly higher in the affected age group.
- The upsurges in notifications were not preceded by increases in testing activity.
- In adults although there was a similar amount of testing in 2005 compared to 2003, the notification rate was considerably higher in 2005 and the proportion of positive tests was statistically significantly higher in the 2005 epidemic, suggesting that the disease that was detected in 2005 was not present in 2003.
- Although the number of hospital separations is small, there was an increase in the proportion of adult separations in 2005.

However, manufacturer A was not able to accurately determine when the false positive issue first arose. If it was at some point earlier in the study period, the apparent changes in disease activity could be the result of a test artifact.

Excluding the serology data, there was an increase in positive tests in 2000 and 2003, but not 2005. The use of PCR in adults increased during the study period. The proportion of positive test results via PCR was highest in 2000 and 2003 and declined to its lowest level in 2005. The proportion of positive culture results was also the lowest in 2005. In contrast, the proportion of positive serology results reached a peak in 2005. These results suggest that the false positive issue arose in 2005 and may have been responsible for the apparent increase in disease in adults during that period. However, conclusions are limited by the small number of positive PCR and culture results. Furthermore, as adults may present and therefore be tested later in the course of their illness than children, resulting in a lower chance of a positive result with these tests, and the sensitivity of PCR has shown to decrease with increasing age,[19] an increase in positive PCRs and cultures may not be expected during an epidemic that predominantly affects adults.

Increases in notifications during the study period are not explained by the greater use of multiple tests on patients. The use of multiple tests on patients did increase during the study period, however, instances of patients having a positive result when tested via more than one method were few.

A study from the Australian Capital Territory showed that information alerts issued by ACT Health in 2003 in response to the increased notifications at the time, were associated with an increase in the proportion of cases notified within the infectious period of 21 days.[20] Potentially, such information alerts could have led to testing of patients with a clinical spectrum that did not previously lead to pertussis testing. For example, testing people whose cough was not protracted at presentation (and therefore may have been due to a variety of causes). However, apart from being slightly elevated following the end of the 2003 epidemic, the testing and notification activity were very closely related, suggesting that the testing that was occurring was discriminate and in response to underlying disease activity.

There were very few notified cases that did not meet the surveillance case definition for pertussis and no obvious trend in the proportion of notified cases that did over time. Even if the false positive issue did arise at some point during the study period, it would appear that the majority of cases detected via the faulty kits did have an illness similar to pertussis. A recent evaluation has showed that the majority of the false positive results were due to non-specific cross-reactions with the filamentous haemagglutinin (FHA) as demonstrated by Western

blot.[21] Cross reactions with the FHA antigen are known to occur in respiratory illnesses such as influenza and *Mycoplasma pneumoniae*.

A study from British Columbia in Canada examined notifications, hospital separations, and laboratory data during successive outbreaks in the 1990s and 2000, and concluded that the incidence of pertussis increased in 2000 in pre-teens and teens and decreased in infants and younger children.[5] The proportion of positive cultures was maintained among pre-teens and teens during the 2000 outbreak compared to earlier outbreaks, despite the rate dropping among young children. In addition, the greatest proportion of positive tests was in the older age group. Our study demonstrates a similar increase in the proportion of positive PCR results during an epidemic year (2003) compared to earlier years.

A New South Wales study also concluded that the observed increase in pertussis notifications from 1988 to 2002 in adults reflected a real increase in disease.[8] There was a significant increase in both pertussis notification and hospitalisation rates among those aged 15 years or more, whereas in other age groups, there was an increase in notification rates only. Although the number of separations in our study is small, and hospitalisations attributable to pertussis may also be over-estimated by false positive serology results, the increase in the proportion of adult separations in 2005 is also suggestive of a true increase in disease activity in this age group.

To our knowledge, this study is the first published analysis of population based pertussis testing data in Australia, and one of only a few in the international literature. This study used testing data from all of the major laboratories that serviced the Australian Capital Territory during the study period, with testing data from only one minor laboratory missing. Although there was some missing culture data (both results and the test request), the quantity of this was estimated to be insignificant. Although some PCR results were missing, the test requests were available for the calculation of testing rates. These PCR tests were not included in the analysis of the proportion of positive test results. Although only one laboratory was able to provide data grouped by patient, these data were used to adjust the testing rates for the combined data to account for the increased use of multiple tests during the study period.

This study highlights the current difficulties in diagnosing pertussis. Diagnosis in Australia is usually based on a single positive serological test for IgA antibody against whole cell *B. pertussis* antigen. Based on comparison with a clinical case definition, this method was previously shown to be highly specific and thought to be more likely to under-estimate rather than over-estimate the true incidence of disease.[22] However, a recent evaluation, prompted by the product recall, has demonstrated that even the new version of manufacturer A's kit has a specificity of only 86.7% compared to a test panel of sera using complement fixation, immunofluorescence and Western blot. In addition, the sensitivity and specificity of all the currently available serology kits were shown to be variable.[23] Our experience demonstrates the importance of continuous review of laboratory testing methods and close laboratory liaison regarding apparent changes in surveillance data.

Were the apparent epidemics of pertussis in the Australian Capital Territory in 2000, 2003 and 2005 the result of real disease activity, increased and less discriminate testing or an artifact of false positive serology results? As conclusions are limited by the uncertainty surrounding when the false positive serology problem began, the combination of an increase in adult notifications, separations and proportion of positive tests in 2005, is only weak evidence of an increase in underlying disease activity. However, the increase in the proportion of positive PCR results during 2003 is strong evidence that this apparent epidemic was due to a real increase in disease rather than increased testing, and provides support for the current policy in Australia of providing a booster dose of the pertussis vaccine to adolescents.

Acknowledgements

We would like to thank the Australian Capital Territory Health Protection Service, ACT Pathology, Capital Pathology and Symbion Pathology for extracting the data.

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Prevalence of antimicrobial resistance in *Enterococcus* isolates in Australia, 2005:

Report from the Australian Group on Antimicrobial Resistance

Keryn J Christiansen, John D Turnidge, Jan M Bell, Narelle M George, Julie C Pearson

Abstract

Antibiotic resistance in *Enterococcus* species causing clinical disease was examined in a point-prevalence study in 2005. Twenty-two sites around Australia collected up to 100 consecutive isolates and tested them for susceptibility to ampicillin, vancomycin, high-level gentamicin and/or high-level streptomycin using standardised methods. Results were compared to similar surveys conducted in 1995, 1999 and 2003. In the 2005 survey, *Enterococcus faecalis* (1,987 strains) and *E. faecium* (180 strains) made up 98.6% of the 2,197 isolates tested. Ampicillin resistance was common (77%) in *E. faecium*, but rare still in *E. faecalis* (0.2%). Resistance to vancomycin was 7.2% in *E. faecium* and 0.2% in *E. faecalis*; the *van*B gene was detected in all vancomycin-resistant isolates. High-level resistance to gentamicin was 35.8% in *E. faecalis* and 52.2% in *E. faecium*; the figures for high-level streptomycin resistance were 10.3% and 60.2% respectively. Compared to previous Australian Group on Antimicrobial Resistance surveys in 1995, 1999 and 2003, the proportions of vancomycin resistance and high-level gentamicin resistance in enterococci are increasing. It is important to have an understanding of the occurrence of vancomycin resistant enterococci and high level aminoglycoside resistance in Australia to guide infection control practices, antibiotic prescribing policies and drug regulatory decisions. *Commun Dis Intell* 2007;31:392–397.

Keywords: antibiotic resistance, enterococcus, vancomycin

Introduction

Enterococci are part of the normal flora of the gastrointestinal tract. They can give rise to endogenous infections such as urinary tract infections outside of hospitals. In hospitals they can be transmitted through suboptimal infection control practices and can give rise to a wide variety of infections, usually in patients with co-morbidities. The two main species causing infections in humans are *Enterococcus faecalis* (80%–90%) and *Enterococcus faecium* (5%–10%) with only a very small number of other species being isolated from clinical specimens. Enterococci are recognised as significant nosocomial pathogens causing urinary tract, blood stream, sterile site and wound infections. Enterococci, although resistant to many antibiotics, have been generally susceptible to amoxycillin and vancomycin. *E. faecium* has become increasingly resistant to ampicillin/amoxycillin making vancomycin the treatment of choice for severe infections caused by this organism. Since 1988 resistance to vancomycin has emerged and increased worldwide and is widespread in Europe and the United States of America (USA). The National Nosocomial Infections Surveillance System in the USA has demonstrated a rising resistance rate for enterococci causing infections in ICU patients with a 2003 rate of 28.5%.[1] The first vancomycin resistant enterococcal (VRE) isolate was reported in Australia in 1994[2] and a report on the emergence and epidemiology of VRE in Australia was described in 1998[3] when 69 isolates had been documented. Prevalence or incidence rates of VRE in Australian hospitals are not routinely collected although there have been reports of individual hospital outbreaks of VRE infections and associated colonisation of other patients.[4–8] The clinical impact of vancomycin resistance in enterococci has been reported to increase mortality, length of stay and hospital costs.[9–11] Intensive infection control measures can be used to eradicate the organism from a hospital population or to prevent it from becoming established.[4]

Enterococci cause 5%–18% of all cases of endocarditis, both on prosthetic and normal heart valves.[12–14] Combination therapy of a ß-lactam and an aminoglycoside (gentamicin or streptomycin)[15–17] has been the standard treatment for at least 50 years as use of ß-lactams alone are associated with high relapse rates (30%–60%). Aminoglycosides are not routinely used to treat other enterococcal infections but in endocarditis the synergy between the two agents greatly increases the likelihood of a cure. Synergy does not occur if the organism has high level gentamicin or streptomycin resistance (MIC > 500 mg/L).

It is important to have an understanding of the occurrence of VRE and high level aminoglycoside resistance in Australia to guide infection control practices, antibiotic prescribing policies and drug regulatory decisions.

Methods

Institutions

Participating laboratories were located in New South Wales (6), the Australian Capital Territory (1), Queensland (3), Victoria (4), South Australia (3), Western Australia (4) and Tasmania (1). To ensure institutional anonymity, data from New South Wales and the Australian Capital Territory and from Tasmania and Victoria have been combined.

Commencing on 1 January 2005, each participating laboratory collected up to 100 consecutive, significant, clinical isolates of enterococci. Only one isolate per patient was tested unless a different antibiogram was observed from routine susceptibility results. Two thousand, one hundred and ninety-seven isolates were included in the survey. Results were compared with previous surveys conducted by the Australian Group on Antimicrobial Resistance (AGAR) in 1995, 1999 and 2003.

Laboratory methods

Participating laboratories were required to meet standards for species identification. All isolates were tested for pyrrolidonyl arylamidase and esculin hydrolysis in the presence of bile with optional testing for growth in 6.5% NaCl, Group D antigen and growth at 45°C. Isolates were identified to species level by one of the following methods: API 20S, rID32Strep, Vitek or Vitek 2, Microscan, polymerase chain reaction (PCR), or conventional biochemical tests. If biochemical testing was performed, the minimum tests necessary for identification were: motility, pigment production, methyl-α-D-glucopyranoside, fermentation of 1% raffinose, 1% arabinose, 1% xylose and pyruvate utilisation. Participating laboratories performed antimicrobial susceptibility tests according to each laboratory's routine standardised methodology[18–22] (CLSI, CDS or BSAC disc diffusion, Vitek, Vitek 2, agar dilution or CLSI broth microdilution). Antimicrobials that were tested by all laboratories included ampicillin and vancomycin. In addition, all isolates were screened for high level gentamicin and 1,201 (55%) isolates were screened for high level streptomycin resistance. Isolates were tested for ß-lactamase production using nitrocefin. All isolates that were resistant to vancomycin were referred to the appropriate state National VRE Network laboratory for molecular testing to confirm organism identification and resistance phenotype.

Results

Specimen source

The majority of isolates (73.6%) were from the urinary tract. These were predominantly *E. faecalis* (93.7%). Invasive (primarily blood, cerebrospinal fluid and sterile cavity) isolates comprised 10.3% of the total number collected (Table 1). *E. faecium* was disproportionately represented in the invasive group (18.9%). Of the *E. faecalis* isolates, 8.7% were invasive compared to 23.9% of *E. faecium*. Isolation of enterococci was more common in women, in keeping with the greater incidence of urinary tract infections in that sex. Of note however, is the greater proportion of *E. faecium* (63.9%) from women compared to men (36.1%).

Susceptibility results

Ampicillin

Resistance to ampicillin was predominantly in the *E. faecium* isolates where the proportion of resistance was similar across all the states except Queensland, where the rate was lower (Table 2). Resistance in all species was due to penicillin binding protein

Table 1. Source of isolates

Source	*E. faecalis*	*E. faecium*	Other spp.	Total	%
Urine	1,514	96	6	1,616	73.6
Wound	157	22	9	188	8.6
Blood/CSF	110	27	8	145	6.6
Sterile site	62	16	4	82	3.7
Other	144	19	3	166	7.6
Total	1,987	180	30	2,197	
Invasive	172	43	12	227	10.3
Non-invasive	1,815	137	18	1,970	89.7
Sex					
Female	1,041	115	9	1,165	53.0
Male	946	65	21	1,032	47.0

CSF Cerebrospinal fluid.

Table 2. Ampicillin resistance

	Qld		NSW/ACT		Vic/Tas		SA		WA		Aus	
	n	%	n	%	n	%	n	%	n	%	n	%
E. faecalis	0/286	0.0	1/619	0.2	0/449	0.0	0/280	0.0	2/353	0.6	3/1,987	0.2
invasive	0/22	0.0	0/76	0.0	0/35	0.0	0/8	0.0	0/31	0.0	0/172	0.0
E. faecium	7/12	58.3	57/72	79.2	36/47	76.6	10/13	76.9	28/36	77.8	138/180	76.7
invasive	2/4	50.0	18/20	80.0	8/12	66.7	0/0	0.0	4/7	57.1	30/43	69.8

changes. Two thousand and seventy-seven (94.5%) isolates were tested for ß-lactamase; none were positive. Trend data for *E. faecium* show an initial increase in ampicillin resistance between 1995 and 1999 with a plateau from 1999 to 2005 (Figure 1).

Vancomycin

Vancomycin resistance was uncommon in *E. faecalis* (0.2%). A total of 7.2% of *E. faecium* were vancomycin resistant with a greater proportion isolated from invasive infections. Resistant organisms were detected in New South Wales/Australian Capital Territory, Victoria/Tasmania and Western Australia. The 16 vancomycin resistant enterococci were all confirmed by PCR and were of the *van*B genotype. Thirteen (81.2%) were *E. faecium* (Table 3). Trend data for *E. faecium* show that after no vancomycin resistance was detected in 1995 there has been a marked increase, particularly for the invasive category (Figure 2) during the study periods. Vancomycin resistant *E. faecium* have occurred in all five regions over the four survey periods, with Victoria/Tasmania showing the greatest increases in VRE over time (Figure 3).

Figure 1. Ampicillin resistance: *Enterococcus faecium*

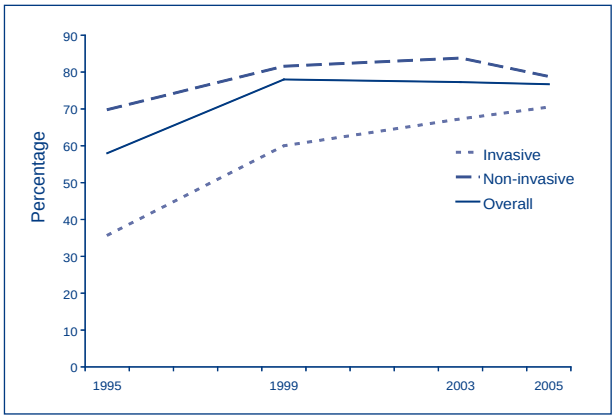


Table 3. Vancomycin resistant enterococci

Specimen source	*E. faecalis*	*E. faecium*	Genotype
Urine	3	5	*van*B
Wound		3	*van*B
Blood		1	*van*B
Sterile site		3	*van*B
Other		1	*van*B
Total	3	13	

Figure 2. Vancomycin resistance: *Enterococcus faecium*

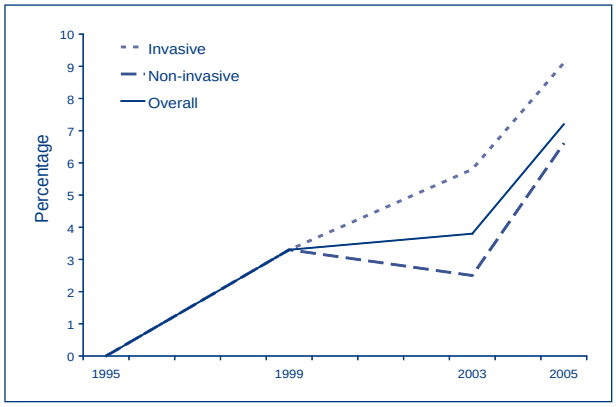
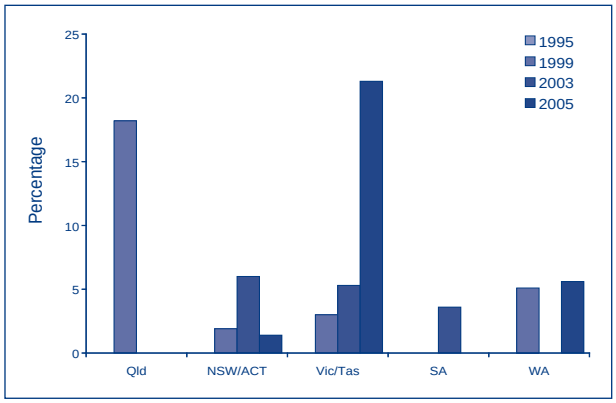


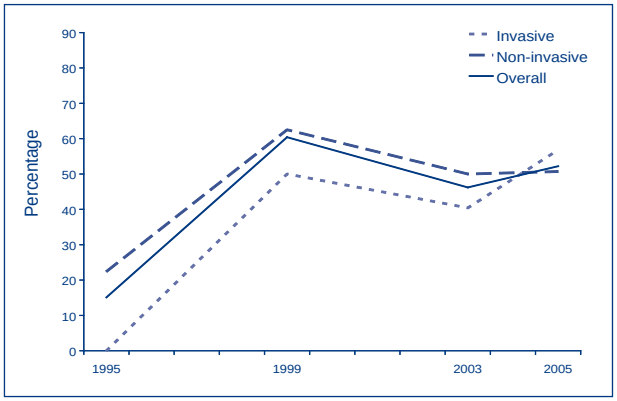
Figure 3. Regional location of vancomycin resistant *Enterococcus faecium*, 1995, 1999, 2003, 2005



Gentamicin

High level gentamicin resistance (HLG) was seen in both *E. faecalis* (35.8%) and *E. faecium* (52.2%) with comparable proportions in most regions (Table 4). Trend data for 1995 to 2005 (Figures 4 and 5) show an increase in HLG resistance over the last 10 years. However, in *E. faecium*, HLG has reached a plateau whilst in *E. faecalis* resistance is continuing to increase.

Figure 4. High level gentamicin resistance: *Enterococcus faecium*



Streptomycin

High level streptomycin resistance (HLS) as with HLG resistance is more common for *E. faecium* than *E. faecalis* (Table 5). The trend since 1995 is for increasing resistance particularly for invasive isolates of *E. faecium* (Figure 6). The rate of increase in HLS is similar to that for HLG for *E. faecium*. In *E. faecalis*, the HLS is relatively stable with lower rates of expression than HLG (Figure 7).

Figure 5. High level gentamicin resistance: *Enterococcus faecalis*

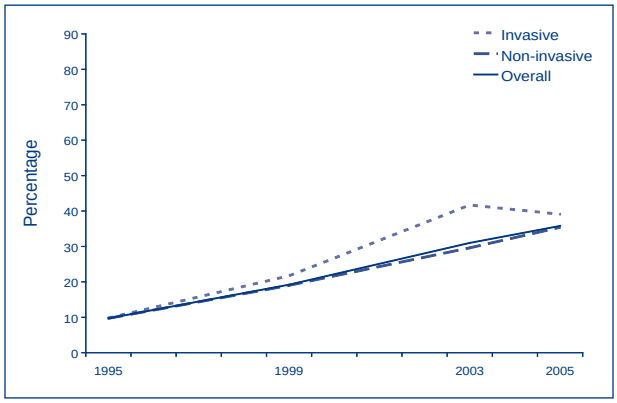


Figure 6. High level streptomycin: *Enterococcus faecium*

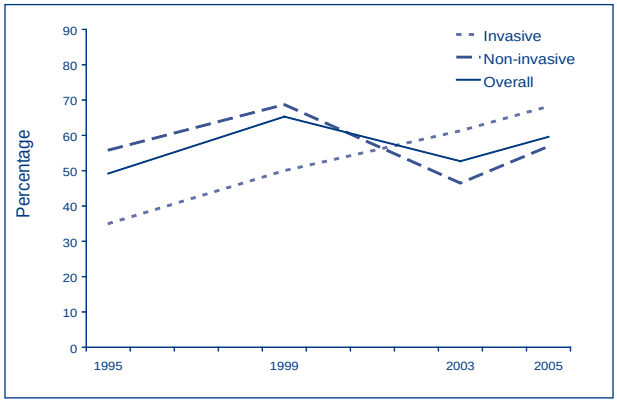


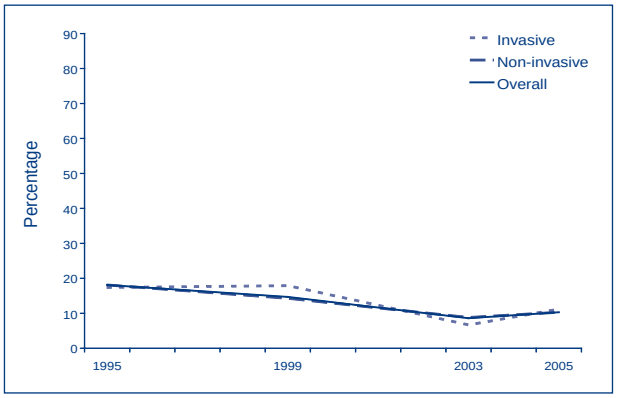
Table 4. High level gentamicin resistance

	Qld		NSW/ACT		Vic/Tas		SA		WA		Aus	
	n	%	n	%	n	%	n	%	n	%	n	%
E. faecalis	101/286	35.3	243/619	39.4	145/448	32.4	58/280	20.7	163/353	46.2	710/1,986	35.8
invasive	7/22	31.8	34/76	44.7	10/35	28.6	2/8	25.0	15/31	48.4	68/172	39.5
E. faecium	7/12	58.3	48/72	66.2	12/47	25.5	9/13	69.2	18/36	50.0	94/180	52.2
invasive	2/4	50.0	16/20	80.0	2/12	16.7	0/0	0.0	5/7	71.4	25/43	58.1

Table 5. High level streptomycin resistance

	Qld		NSW/ACT		Vic/Tas		SA		WA		Aus	
	n	%	n	%	n	%	n	%	n	%	n	%
E. faecalis	40/286	14.0	32/348	9.2	11/90	12.2	22/280	7.9	8/88	9.1	113/1,092	10.3
invasive	2/22	9.1	5/36	13.9	1/9	11.1	0/8	0.0	1/5	20.0	9/80	11.2
E. faecium	6/12	50.0	25/50	50.0	7/8	87.5	9/13	69.2	9/11	81.8	56/94	60.2
invasive	3/4	75.0	8/13	61.5	2/2	100	0/0	0.0	2/3	66.7	15/22	68.2

Figure 7. High level streptomycin: *Enterococcus faecalis*



Limitations of the study

The enterococci in this study were tested against a limited range of antimicrobials. In part this was driven by the presence of intrinsic resistances in this genus. As only a maximum of 100 isolates were collected per institution only a portion of actual clinical isolates are represented. There have been changes in participating laboratories in the AGAR *Enterococcus* surveys over time from 1995 through to 2005 with the more recent inclusion of a number of private pathology laboratories. This may have influenced trend data.

Discussion

It is clear from this study and the examination of trends over the last 10 years that resistance problems are increasing significantly in *E. faecium*. Furthermore, this species is accounting for an increasing proportion of invasive disease. Treatment options for this species are becoming ever more limited as resistance to ampicillin and other penicillins is now very high, and glycopeptide resistance is increasing (7% across Australia, range 0%–21% in 2005).

In *E. faecium*, ampicillin resistance is the result of changes in penicillin-binding proteins. This is also true for most strains of *E. faecalis*, although ß-lactamase production has been seen rarely (3 known instances in Australia in the last decade).[23] No ß-lactamase-producing strains of enterococci were detected in this survey. This survey has shown that ampicillin resistance is now the norm in *E. faecium* but is still uncommon in *E. faecalis*. Ampicillin resistance in enterococci presents considerable challenges when infections are serious, as the strains will not be susceptible to any ß-lactam, and the drug of choice becomes vancomycin, which is only slowly bactericidal. Further, for endocarditis the combination of vancomycin with an aminoglycoside creates significant toxicity problems.

Unfortunately vancomycin resistance in enterococci is slowly increasing in Australia. It has been seen in all states and territories although rates in each region seem to vary considerably. It is widely recognised that rates of colonisation far exceed the rates of infection with VRE, and thus the amount of VRE seen in our survey does not truly reflect the size of the VRE reservoir. The survey results are also consistent with the previous Australian experience that the dominant type of resistance is encoded by the *van*B complex,[24] in contrast with the situation in Europe and the USA where *van*A dominates. Vancomycin-resistant strains causing serious infection are very challenging to treat. The choices are linezolid, quinupristin-dalfopristin and the recently released tigecycline. Each of these agents presents its own challenges for treatment as well.

The increasing rates of high-level resistance to aminoglycosides (except for streptomycin resistance in *E. faecalis*) is surprising. It is not clear what is driving this increase. For *E. faecium* it may well be the increase in resistant clones that are becoming established in some hospitals. Loss of susceptibility to high levels of aminoglycosides greatly compromises the ability to effectively treat enterococcal endocarditis.

The data provided by this survey will be useful in informing microbiologists, infectious diseases physicians and infection control practitioners about the increasing importance of VRE in Australia. It will help to guide prescribers treating presumptive enterococcal infections in empirical choices; e.g. ampicillin/amoxycillin still being active against the vast majority of strains of *E. faecalis* when treating infections caused by this organism. Finally, the data will assist regulators and the pharmaceutical industry on the growing importance of VRE in Australia, and guide decision makers about controls that might be required on reserve antibiotics.

A full detailed report of this study may be found on the Australian group on Antimicrobial Resistance website: http://www.antimicrobial-resistance.com under 'AMR Surveillance'.

Acknowledgements

This study was fully supported by a grant from the Australian Government Department of Health and Ageing. The participating laboratories were from: Alfred Hospital, Austin Hospital, The Canberra Hospital, Concord Hospital, Gribbles Pathology (SA), Institute of Medical and Veterinary Science, John Hunter Hospital, Nepean Hospital, PathWest Fremantle Hospital, PathWest QEII Medical Centre, PathWest Royal Perth Hospital, Queensland Health Pathology Service, Princess Alexandra Hospital, QHPS Royal Brisbane Hospital, Royal Children's Hospital, Royal Hobart Hospital, Royal North

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Epidemiology and outcomes for *Staphylococcus aureus* bacteraemia in Australian hospitals, 2005–06:

Report from the Australian Group on Antimicrobial Resistance

John D Turnidge, Graeme R Nimmo, Julie Pearson, Thomas Gottlieb, Peter J Collignon and the Australian Group on Antimicrobial Resistance

Abstract

The Australian Group on Antimicrobial Resistance studied the epidemiology and outcomes of *Staphylococcus aureus* bacteraemia in selected Australian hospitals in 2005–06. Seventeen hospital-based laboratories collected basic demographic, susceptibility and patient outcome data on all cases of *S. aureus* bacteraemia for 5 to 24 months during the study period. There were 1,511 cases of bacteraemia documented, of which 66% occurred in males and 32% originated from vascular access devices. Bacteraemia had a community onset in 60% of cases, although 31% of these were health-care associated. Overall, 57% of episodes were health-care related. Methicillin-resistant *Staphylococcus aureus* (MRSA) was the responsible pathogen in 24% of instances; of these 53% were of the typical multi-resistant hospital type, and 29% were of the community-associated type. Seven per cent of all staphylococcal bacteraemias were caused by community-associated MRSA strain types, attesting to the growing size of this problem in Australia. Outcomes were available for 51% of cases and in those the all-cause mortality at 7 days or discharge (whichever came earlier) was 11.2%. Age was strongly associated with mortality; the rate for patients aged more than 60 years was 18%. Sepsis originating from intravascular access devices had a lower mortality rate of 5%. *S. aureus* bacteraemia is a common community and hospital infection with a significant mortality. A nationally co-ordinated program documenting the incidence and outcomes of this disease would likely lead to measures designed to reduce the incidence and improve outcomes of this disease. *Commun Dis Intell* 2007;31:398–403.

Keywords: *Staphylococcus aureus,* bacteraemia, epidemiology, outcomes

Introduction

Staphylococcus aureus ranks as one of the most common and important bacterial pathogens of humans.[1] It is a commensal organism which, with the right conditions and pathogenic factors, can invade the host and cause a range of diseases from minor skin and soft tissue infections to osteomyelitis, endocarditis and life-threatening septicaemia. It is prevalent as a cause of infection both in the community and in hospital practice, and is one of the most common species found in positive blood cultures. Its versatility is further enhanced by its ability to acquire resistance and multiple resistance, exemplified by the emergence over time of penicillin resistance, methicillin resistance and multi-resistance, initially in hospitals and later in the community. There are currently no vaccines effective against this common pathogen.

Although it is recognised as an important cause of morbidity and mortality by infectious disease practitioners, there are limited data on the incidence of serious *S. aureus* sepsis in Australia, and only two regional studies on patient outcomes in patients with methicillin-resistant *S. aureus* sepsis.[2,3] The Australian Group on Antimicrobial Resistance has been monitoring resistance in *S. aureus* since 1986,[4,5] and has recently presented information on the large burden of bacteraemia in Australia.[6] The present study was designed to provide preliminary information on the outcomes of *S. aureus* bacteraemia in Australia.

Methods

Institutions

As members of the Australian Group on Antimicrobial Resistance, 17 hospital laboratories from each state and territory of Australia participated in the collection of anonymous data on cases of *S. aureus* bacteraemia from January 2005 to December 2006 over periods ranging from 5 to 24 months. The laboratories were in Queensland (3), New South Wales/Australian Capital Territory (3), Victoria/Tasmania (4), South Australia/Northern Territory (3), and Western Australia (4). With one exception, each laboratory serviced either a single hospital or submitted data from only one hospital.

Data collection methods

Cases of *S. aureus* bacteraemia were identified with the first positive blood culture from a patient with a

compatible illness. Demographic data (age and sex), disease data (date of admission, onset in community or hospital, health-care association, source of infection and mortality) were collected prospectively. Cases were in general considered to have a hospital onset of infection if the time of collection of the first positive blood culture for *S. aureus* was more than 48 hours after admission. Mortality was measured at either 7 days after the time of blood culture collection or at discharge if sooner. Participants were requested to make a judgement about the relationship between mortality and staphylococcal sepsis. No attempts were made to follow up patients after this time. The susceptibility test results were tabulated for each strain.

Data analysis

Where relevant, dichotomous outcome measures (died, survived) were compared using Chi-squared tests (for contingency tables and for trend).

Antibiograms

Strains of *S. aureus* were categorised according to their susceptibilities to a range of antibiotics as penicillin-susceptible, methicillin-susceptible or methicillin-resistant. Methicillin-resistant strains were further presumptively identified as being of the hospital-associated multi-resistant type (AUS-2/3-like) because of resistance to at least three of the following characterising agents: erythromycin, gentamicin, tetracycline, ciprofloxacin, and trimethoprim; hospital-associated United Kingdom type (EMRSA-15-like) due to resistance to ciprofloxacin ± erythromycin but none of the other three agents; or community-associated type (WA-1, South West Pacific, Queensland, and others) if susceptible to all characterising agents or resistant to erythromycin only among those agents.

Results

Data were available on 1,511 cases of *S. aureus* bacteraemia. Two thirds of cases (66.2%) were in males, and males predominated in all age groups (Figure 1).

Associated infections

Information on the type of infection with which the bacteraemia was associated was available on 709 cases (Table 1). The most common infection overall was bacteraemia from an intravascular line, either central or peripheral, or other form of vascular access (e.g. haemodialysis shunt). These accounted for 32% of all infections seen. As expected skin/skin structure infections and bone/joint infections accounted for significant proportions of the associated infections. Endocarditis was the underlying infection in nearly 8% of all cases. Between the ages of 20 and 50, a higher proportion of bacteraemias, 8%, were due to endocarditis.

Table 1. *Staphylococcus aureus* infection types associated with bacteraemia

Infections	Number	Percentage (n = 709)
Intravascular access	258	36.4
IV line infection	226	31.9
Infected AV fistula	24	3.4
Other vascular	8	1.1
Skin and skin structure	143	20.2
Cellulitis/soft tissue infection	131	18.5
Infected burns	6	0.8
Infected dermatological disease	5	0.7
Furunculosis	1	0.1
Orthopaedic	107	15.1
Septic arthritis	52	7.3
Osteomyelitis	46	6.5
Discitis	9	1.3
Cardiac	54	7.6
Endocarditis	54	7.6
Respiratory tract	53	7.4
Pneumonia	50	7.1
URTI unspecified	2	0.3
Orbital cellulitis/sinusitis	1	0.1
Surgical	51	7.2
Post-operative wound infection	38	5.4
Infected vascular prosthesis	11	1.6
Infected implanted device	2	0.3
Other	43	6.1
Urinary tract infection	18	2.5
Deep abscess	10	1.4
Cholangitis	3	0.4
Meningitis	3	0.4
Febrile neutropenia	2	0.3
Gastroenteritis	2	0.3
Peritonitis	2	0.3
Post-partum endometritis	2	0.3
Early onset neonatal sepsis	1	0.1
Unknown/not stated	802	

Site of onset and health-care association

Bacteraemia had its onset in the community in 59.6% (865/1,449) of cases (Table 2). Of these, 30.9% were health-care associated (216/700 instances where information on this association was provided). In 35 instances, even though the onset was in hospital, the infection was assessed as not being associated with health care. Such cases included examples such as neonatal sepsis following acquisition from the mother, or the documentation of *S. aureus* from another site at the time of admission without any medical intervention that could have provoked a bacteraemia.

Susceptibilities

Methicillin-resistant strains (MRSA) were responsible for 359 or 23.8% of infections. Of these, 191 (53%) were presumptively the typical multi-resistant hospital type (AUS-2/3-like), 44 (12%) were of the hospital type prominent in the United Kingdom (EMRSA-15-like), and 103 (29% and 6.9% overall) were of the community MRSA type (caMRSA-like). In a further 21 episodes, too few antibiotic susceptibilities were reported to be able to assign a presumptive type of MRSA. The remainder of the 1,511 strains were either penicillin susceptible (PSSA, 192 = 12.7%) or penicillin-resistant and methicillin-susceptible (MSSA, 961 = 63.6%).

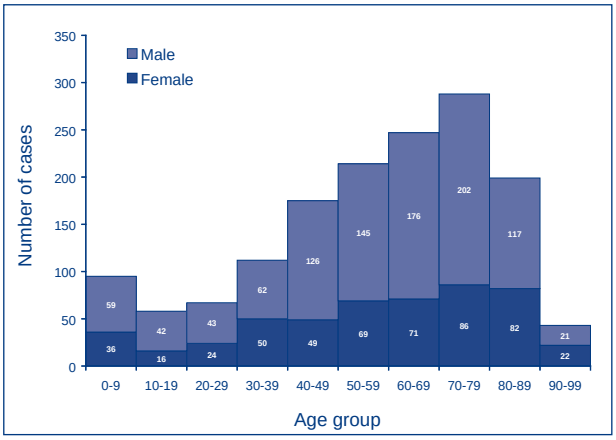
For those strains where the information was provided, 87% (CI = 81%–93%) of AUS-2/3-like MRSA were health-care associated, compared to 74% (CI = 59%–88%) of EMRSA-15-like strains, 74% (CI = 55%–73%) of caMRSA-like strains, 50% (CI = 46%–53%) of MSSA and 54% (CI = 47%–62%) of PSSA strains (Table 3). Overall, methicillin-resistant strains were more likely to be health-care-associated than methicillin-susceptible strains (MSSA plus PSSA) (78% v. 51%, P <0.0001), and hospital-type MRSA (AUS-2/3-like and EMRSA-15-like) were more likely to be associated with health-care than caMRSA (84% v. 64%, P = 0.0002).

Outcomes

Outcomes were available for 768 cases (51%). The all-cause mortality in this group was 11.2% (86 cases) (Table 4). The documented attributable mortality was 39/768 or 5.1%, although this is likely to be an underestimate as the cause of death was not documented in 29 of the 86 cases. Given that most of the data were collected from a laboratory base, the reliability of attribution of cause for mortality was not considered high, and thus subsequent analyses were undertaken with the all-cause mortality data.

The most significant factor associated with death was age, as highlighted in Figure 2 (and Table 5). Mortality was greater than 20% in patients aged over 80 years, with an overall trend to lower percentages the younger the patient. Survival was not influenced by sex, place of onset of sepsis, health-care versus

Figure 1. Age and sex distribution of *Staphylococcus aureus* bacteraemia cases, (n = 1511)



Numbers on the bars represent the exact numbers of females and males in each decade of life.

Table 2. Site on onset of bacteraemia and its association with health care

Onset	Health-care associated			Total
	Yes	No	Unknown/ not stated	
Community	216	484	165	865
Hospital	452	35	97	584
Unknown/not stated	32	18	12	62
Total	700	537	274	1,511

Table 3. Proportion of types of *Staphylococcus aureus* that were health-care associated

Type of *S. aureus*	Health-care associated		Total	Proportion health-care associated (95% Confidence interval)
	Yes	No		
AUS2/3-like	115	17	132	87.1 (81.4–92.8)
EMRSA-15-like	25	9	34	73.5 (54.6–74.3)
caMRSA*-like	57	32	89	64.0 (54.1–74.0)
MRSA-unclear type	16	2	18	88.9 (74.4–100)
MSSA	401	405	806	49.8 (46.3–53.2)
PSSA	86	72	158	54.4 (46.7–62.2)
Total	700	537	1,237	56.6 (53.8–59.4)

* Community-associated MRSA.

non-health-care association, or β-lactam resistance of any type. Mortality was significantly reduced when the source of the infection was an intravascular line.

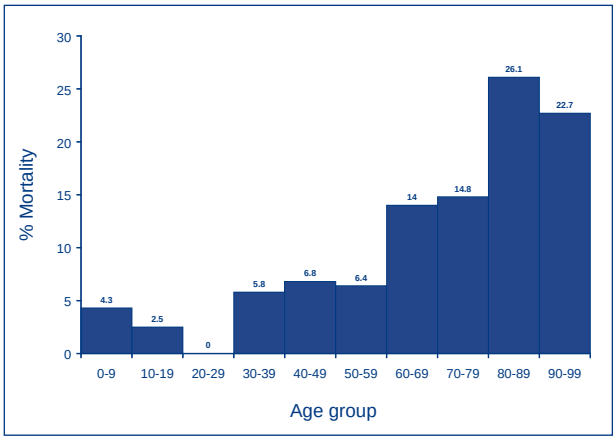
Discussion

Our data show than *S. aureus* bacteraemia remains a common problem in Australia. Unfortunately, we do not have accurate information on what proportion of the Australian population was served by the participating sites, so we could not estimate the true rates of sepsis in our population. However, our previous study showed that 0.15% of hospital admissions in Australia were for *S. aureus* bacteraemia and estimated that about 6,900 episodes occur

Table 4. Outcomes on cases

Outcome at 7d or at discharge if earlier	Number of cases
Death due to sepsis	9
Death due to sepsis and other causes	30
Death from other causes	18
Death from undocumented cause	29
Subtotal	86
Patient alive but on-going sepsis	79
Patient recovered	510
Patient recovered but with significant new morbidity	41
Patient survived at 7 days	52
Subtotal	682
Unknown/Not stated	743

Figure 2. Age stratified mortality rates *Staphylococcus aureus* bacteraemia cases, (n = 768)



Numbers on the bars represent the exact percentages in each decade of life.

Table 5. Potential risk factors for mortality

Factor	Group	Died	Survived	% Mortality	P
Age	< 60 years	20	378	5.0	< 0.0001
	≥60 years	66	304	17.8	
Sex	Female	34	245	12.2	NS*
	Male	52	437	10.6	
Health-care associated	Yes	41	345	10.6	NS
	No	41	299	12.1	
Place of onset	Community	49	413	10.6	NS
	Hospital	36	269	11.8	
Source	IV line†	9	168	5.1	0.003
	Not an IV line	77	514	13.0	
Methicillin-resistant strain	Yes	14	135	9.4	NS
	No	72	547	11.6	
β-lactam resistance	Penicillin-susceptible	13	103	11.2	NS
	Methicillin-susceptible	59	444	11.7	
	Methicillin-resistant	14	135	9.4	NS
Methicillin-resistant type	AUS-2/3-like	8	51	13.6	NS
	EMRSA-15-like	3	17	15.0	
	caMRSA-like	3	56	5.1	

* Not significant.

† Intravascular line or access.

annually in Australia.[6] Based on the average of 13.8 months of data collected from the 17 laboratories, we estimate that we captured about one fifth (approximately 1,300 per year) of all bacteraemias occurring nationally during the study period, and therefore our study provides at least an indicative sample of the problem.

The proportion of cases (24%) caused by methicillin-resistant strains is slightly higher than our previous observations (19%).[6] This may relate to the lower number of laboratories serving private hospitals captured in this study compared to the previous study or a genuine increase in the prevalence of MRSA types. More importantly, we were able to estimate what proportion was due to strains with a resistance profile resembling community-associated MRSA. The finding of 7% of all bacteraemias being due to community-associated MRSA attests to the growing size of this problem in Australia.[7] More surprising was the finding that the major proportion of caMRSA were the cause of health-care associated infections. Outbreaks of caMRSA in hospitals in Australia have been reported,[8] but are not common, and it is more likely that the health-care association is related to increasing rates of colonisation in the community. One seminal study has shown that nasal carriage, most of it present at the time of initiation of health care, accounts for about 80% of subsequent health-care associated bacteraemias.[9]

The crude mortality rate is in the range observed in recent studies from Australia and other countries in adults [10–15] and children.[16–19] Because direct follow-up after 7 days or discharge was not required as part of data collection, we believe that the mortality rate observed is lower than the true figure. We confirmed the very strong association between age and outcome. Mortality rates were significantly lower when the source of infection was an intravascular line or from other vascular access, but no other factor that we examined influenced mortality significantly. In particular, we did not show increased mortality in patients with MRSA infection, which is seen in some series and not others.[20]

Unfortunately, despite the incidence, importance and severity of staphylococcal bacteraemia, there is currently no mechanism in place nationally to monitor incidence and outcomes.[21] This infection is substantially more common and has a higher mortality rate than meningococcal sepsis,[22,23] and yet remains a 'disease in the background'. This is in part because outbreaks in the community have been difficult to detect due to the substantial incidence of sporadic cases. Their substantial impact has therefore been overlooked by the community, the media or public health authorities. However, the recent acquisition of methicillin-resistance by virulent strains has provided a prominent phenotypic marker (Panton-Valentine leukocidin) that has made the epidemic nature of these infections obvious. Their association with deaths in young otherwise healthy children and adults[24,25] has emphasised their importance as a potential target for public health and clinical intervention even more. Emergence of community-associated MRSA highlights the need for a national approach to a growing problem, and our study supports the call for mandatory central reporting of *S. aureus* bacteraemia, but one that also includes community-onset disease, as happens in the United Kingdom.[26] Only then will we be in a position to design better intervention tools.

We recognise that our methods for measuring outcomes had limitations and were subject to possible bias. Not all participating laboratories were able to provide outcome data. Those who did provide data were not audited for accuracy of data capture, and judgements about attributable mortality are acknowledged to be subjective. Nevertheless, we feel that our data provide the first national indication of the importance of serious *S. aureus* infection in the Australian community, and they should drive the future development of robust systems for measuring and improving outcomes of this common infection.

Acknowledgements

This study was supported in part by a grant from the Australian Government Department of Health and Ageing.

The participating laboratories were from: The Alfred Hospital (D Spelman, C Franklin); Concord Hospital (T Gottlieb, G Funnell); Fremantle Hospital–PathWest (D McGechie, G Francis); Royal Adelaide Hospital–Institute for Medical and Veterinary Science (I Bastian, R Pratt); John Hunter Hospital (J Ferguson, J Anderson); Launceston General Hospital (E Cox, K Wilcox); Monash Medical Centre (T Korman, D Kotsanas); Princess Alexandra Hospital (J Faoagali, G Lye), Royal Brisbane and Women's Hospital–Pathology Queensland, (G Nimmo, N George); The Prince Charles Hospital–Pathology Queensland (C Coulter, S Gribble), Queen Elizabeth II Medical Centre–PathWest (C Gollege, B Henderson); Royal Darwin Hospital (G Lum, P Southwell); Royal Perth Hospital–PathWest (J Pearson, K Christiansen, G Coombs); St John of God Hospital, Perth (S Benson, J Fenton); St Vincent's Hospital, Melbourne (MJ Waters, L Joyce); The Canberra Hospital (P Collignon, S Bradbury, J Roberts); Women's and Children's Hospital (J Turnidge, J Bell).

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Short reports

Position statement on interferon-γ release immunoassays in the detection of latent tuberculosis infection, October 2007

National Tuberculosis Advisory Committee

Detection and treatment of latent tuberculosis infection (LTBI) is considered to be an increasingly important element of tuberculosis (TB) control efforts in Australia and other low incidence countries. *In vitro* T-cell based interferon-γ release immunoassays (IGRAs) are marketed as a substitute for the tuberculin skin test (TST) for the detection of LTBI. The specificity of these immunoassays has been optimised by utilising pooled synthetic antigens, such as early secretory protein 6 [ESAT-6] and culture filtrate protein 10 [CFP-10], from the *Mycobacterium tuberculosis*-specific region of difference 1 (RD1) region and has been recently reviewed (Pai et al, 2004; Menzies et al, 2007).

Data suggest that IGRAs using these antigens are more specific than TST, having less cross-reactivity with previous Bacille Calmette-Guérin (BCG) immunisation or exposure to non-tuberculous mycobacteria, potentially offering distinct advantages for the detection of LTBI. However, the assessment of the sensitivity of IGRAs for diagnosing LTBI in differing environments and countries is complicated by the lack of a gold standard for diagnosing LTBI, the varied methodology across studies in the performance of TST and the interpretation of TST reactions, and the limited long-term follow-up of those subjects tested with IGRAs compared with the historical data available on those populations tested with TST. There is also limited data on the use of these immunoassays in certain subpopulations such as immunocompromised patients, children, and populations from TB-endemic countries, although such data on these populations are emerging for one or both of the two commercial IGRA *in vitro* tests currently available. Additionally, long-term follow-up studies are underway and will help clarify issues relating to the performance characteristics of IGRAs. As such information is carefully reviewed, the performance characteristics and clinical interpretation of these immunoassays will become better defined. Furthermore, the National Tuberculosis Advisory Committee (NTAC) feels that the performance, utility and cost effectiveness of IGRAs remain to be defined under Australasian TB program conditions. Finally, populations most in need of access to accurate diagnosis and potential treatment of LTBI are often in remote and other community centres distant from laboratory services, or are the groups for which the IGRA tests are currently assessed to be least reliable, i.e. children and the immunosuppressed (although for the latter group, TST is also unreliable).

Both NTAC and state-based TB services encourage further clinical and economic evaluation of IGRAs. NTAC considers that the role of IGRAs in diagnosing LTBI will be better defined by:

- ongoing comparative studies of TST and interferon-γ assays undertaken by staff specially trained in the standardised application of the TST, where results can be compared as both continuous and dichotomous variables to assess suitable positive/negative cut-off scores, as well as to further investigate sensitivity, specificity and discordant results;
- sequential testing of IGRAs on various patient groups to characterise and quantify conversion and reversion reactions;
- further research on the use of IGRAs in children;
- independent cost-benefit analysis on the use of IGRAs using states' and territories' preferred protocols of investigating LTBI in Australia. Such analysis is needed to investigate the relative economic outcomes of changing from TST to immunoassays taking into account the structure of TB services and program delivery in Australia; and
- comparison of alternative IGRAs to determine differences between the assays.

NTAC suggests the research and rollout questions summarised in the December 2005 Morbidity and Mortality Weekly Report (Mazurek et al, 2005) paper from the United States Centers for Disease Control and Prevention and recent review article by Pai (Pai et al, 2007) could act as a basis for future investigations.

In summary, NTAC makes the following recommendations:

- currently TST remains the preferred method of screening for LTBI pending further evaluation of IGRAs;
- TST and IGRAs have almost no place in the diagnosis of active TB disease;
- state-based TB services should be encouraged to participate in the evaluation of the role of IGRAs for the investigation of LTBI; and
- IGRAs may be used as a supplementary test in individualised clinical assessment for LTBI where increased specificity is valuable in reducing the confounding effect from prior BCG vaccination or prior exposure to non-tuberculous mycobacteria.

In making these recommendations, NTAC recognises that IGRAs are a novel test for a disease with a delayed onset where the 'gold standard' comparator test (i.e. TST) is imperfect. The NTAC position statement and recommendations will be under ongoing review and will be revised as new peer-reviewed published data becomes available. NTAC is committed to ongoing monitoring of new diagnostic tests that may be of value in TB control.

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Endorsed by the National Tuberculosis Advisory Committee 24 August 2007. Endorsed by the Communicable Disease Network Australia 3 September 2007. Endorsed by the Australian Health Protection Committee 12 October 2007. This statement supersedes all previous such statements.

Some members of the Committee declared receipt of limited funding assistance from Oxford Immunotec, Cellestis Limited and CSL Limited to support research and attendance at a conference.

Disclaimer

National Tuberculosis Advisory Committee multi-drug resistant tuberculosis Information Paper (October 2007)

Definition

Multi-drug resistant tuberculosis (MDRTB) is defined as a strain of *Mycobacterium tuberculosis* with resistance to at least isoniazid (H) and rifampicin (R), the two key drugs in TB treatment. Very recently, extensively drug-resistant tuberculosis (XDRTB) has gained notoriety and is defined as MDRTB with additional resistance to any fluoroquinolone, and to at least one of three injectable second-line anti-TB drugs (capreomycin, kanamycin, and amikacin) used in MDRTB treatment.

Geographic distribution

Drug resistance data have been collected from 90 countries since the launch of the Global Project on Anti-Tuberculosis Drug Resistance by the World Health Organization (WHO) and International Union Against Tuberculosis and Lung Disease in 1994. The estimated total number of MDRTB cases in 2004 was 424,203 (95% CI, 376,019–620,061), or 4.3% (95% CI, 3.8%–6.1%). High burden countries, such as India and China, have significant absolute numbers of MDRTB cases. Some other countries are recognised as 'hot spots' for MDRTB, including Kazakhstan, Uzbekistan and other former Soviet bloc countries. Unfortunately, drug resistance surveys are incomplete with greater than 100 countries (e.g. Indonesia, Nigeria) not included and only 1–2 provinces of large high-burden countries (such as India and China) screened.

Australia has a very low incidence of MDRTB. The Mycobacterium Reference Laboratory Network (MRLN) reports the susceptibility patterns for approximately 800 *M. tuberculosis* isolates obtained in Australia each year from the approximately 1,000 annual notified TB cases; the non-microbiologically-confirmed patients representing clinical, radiological and/or histological diagnoses. The annual incidence of MDRTB has varied between 0.3%–2.0% between 1995 and 2005 with no clear increasing or decreasing trend. Nearly 95% of patients with drug-resistant TB are overseas-born migrants. For example, 21 of 24 MDRTB cases in 2004–2005 were overseas-born migrants from India (n=3), China (n=3), Papua New Guinea (PNG, n=6), Vietnam (n=4), Eritrea, Sudan, Pakistan, South Africa and the Philippines. Preliminary data collected by the MRLN suggest an increase in the absolute number of MDRTB cases in 2006 (n=23) compared with 2004 (n=12) and 2005 (n=12). The MRLN and NTAC are determining the migrant status of the MDRTB cases from 2006.

With the recent World Health Organization change to the XDRTB case definition, Australian authorities have reviewed previous Australian MDRTB cases and reclassified two as XDRTB over the last five years.

One region in Australia, Far North Queensland, is particularly impacted by the influx of people with TB (with a high proportion of multi-drug resistant cases). A treaty between Australia and PNG allows free movement of local inhabitants of the outer Torres Strait Islands of Australia and of selected coastal villages of the Western Province of PNG, for traditional cultural practices. Between 2001 and 2006, 15 of 57 (26%) bacteriologically-proven TB cases were MDR (Konstantinos A, Queensland TB Control Centre, personal communication).

Avoiding the production of multi-drug resistant tuberculosis

The estimated 4.3% prevalence of MDRTB has a corollary; the vast majority of TB is not multi-drug resistant and is treatable with standard short-course chemotherapy – H, R, ethambutol (E) and pyrazinamide (Z) for 2 months followed by H and R for 4 months (i.e. 2HRZE/4HR). Correctly applied, this multi-drug regimen produces cure rates greater than 97% and prevents the emergence of resistance. Unfortunately, *M. tuberculosis* can accumulate mutations as sub-populations of resistant organisms are selected by incomplete or inappropriate drug therapies.

Multi-drug resistant TB is therefore an iatrogenic disease produced by: prescribing errors, poor case supervision, drug malabsorption or unreliable drug supplies. The common prescribing errors are:

- addition of a single drug to a failing regimen;
- failure to identify drug resistance;
- provision of an initial regimen that was inadequate in content (i.e. only HR when resistance was likely) or duration; and

- failure to recognise or address patient non-compliance.

Detection of multi-drug resistant tuberculosis

The early recognition of TB and MDRTB patients is becoming more problematic in Australia where the incidence of TB is very low (i.e. about 5 cases per 100,000 population) and a generation of doctors is now unfamiliar with the disease. Tuberculosis must be considered in the differential diagnosis of any patient with a cough lasting more than three weeks with associated risk factors. Groups at high risk of TB in Australia include migrants, the elderly, indigenous populations and other disadvantaged groups.

The key predictor of MDRTB is a history of previous treatment for TB especially in those with cavitary pulmonary disease. However, diagnosing MDRTB on the basis of clinical prediction alone risks misdiagnosis and unnecessary use of less effective, more toxic and prolonged treatment. In new TB cases treated with a well supervised standard regimen, treatment failure most commonly reflects insufficient treatment rather than the presence of drug resistance.

Culture and drug susceptibility testing (DST) is the principal method of detecting MDRTB. Positive culture and drug susceptibility results should be available within 30 days of specimen receipt using modern broth-based culture methods, which are now the 'standard of practice' in Australian mycobacteriology laboratories. Drug susceptibility tests must be performed in the following circumstances:

- all initial isolates of *M. tuberculosis;*
- isolates from patients who remain culture-positive after 3 months of treatment;
- isolates from patients who are clinically failing treatment; or
- an initial isolate from a patient relapsing after previously successful TB treatment.

Some laboratories may also offer direct molecular detection of R resistance on polymerase chain reaction-positive specimens from patients strongly suspected of having MDRTB. These molecular methods may be 'in-house' amplification and sequencing of the *rpoB* gene or a commercial reverse-hybridisation assay. The rationale for these molecular tests is that about 95% of R-resistant isolates contain mutations in an 81-bp segment of the *rpoB* gene, and R resistance is a marker for MDRTB. Risk factors that might prompt a molecular test for R resistance are:

- contact with a known MDRTB case;
- previous treatment for tuberculosis;
- migration from or residence in a country with a high prevalence of MDRTB; and/or
- HIV infection.

Approaches to treatment

There is limited clinical data to define precisely the best approach to the management of a MDRTB case in terms of the most appropriate drug combination and the duration of therapy. However, various guidelines have been developed based on expert opinion and, although differences exist, it is recommended that a MDRTB treatment regimen should be individually tailored based on the results of DST (providing that the results are timely) and, where previous treatment has occurred, a thorough history of previous drug usage.

With the loss of H and R, drug options are limited. WHO have classified anti-tuberculous agents into five categories to guide the selection process (Table). 'Second line' agents are invariably less effective and potentially more toxic. The initial regimen should include at least four new agents based on drug susceptibility testing but, depending on the severity of disease and level of resistance, more agents may be required. The best outcomes appear to be in patients with limited disease and where the organism is susceptible to an injectable agent and a quinolone (which are the key agents in treating MDRTB).

The duration of the initial phase (that includes an injectable agent) is usually decided by when culture conversion occurs. Recommendations vary from a minimum of 6 months use of the injectable agent to 4–6 months beyond the time of sputum conversion. Ultimately the decision will depend on the effectiveness of other drugs used, the sputum status of the patient, and treatment tolerance.

Again, recommendations vary regarding the total duration of therapy. The minimum standard suggested by the WHO is 18 months after culture conversion extending to 24 months in those with more extensive disease.

Surgery as an adjunct should be considered in those with a significant risk of failing medical treatment based on the level of drug resistance and disease severity. Suitability for surgery depends on disease being localised, adequacy of lung function and a sufficient period of completed treatment to reduce the bacillary burden as much as possible.

Case management

Given the small number of cases of MDRTB in Australia and the complexities involved, management should be by a 'team approach' and coordinated by those with TB expertise. All treatment should

World Health Organization anti-tuberculosis drug classification

Group	Drugs
Group1 – First line (oral)	Isoniazid, Rifampicin, Ethambutol, Pyrazinamide
Group 2 – Injectable agents	Streptomycin, Kanamycin, Amikacin, Capreomycin
Group 3 – Quinolones	Ciprofloxacin, Ofloxacin, Moxifloxacin, Gatifloxacin
Group 4 – Other second line agents (bacteriostatic)	Ethionamide, Protionamide, Cycloserine, Para-aminosalicylic acid, Thioacetazone
Group 5 – Agents of uncertain efficacy (not routinely recommended)	Clofazimine, Amoxicillin-clavulanate, Clarithromycin, Linezolid

be administered by direct observation and patients isolated until sputum cultures have converted to negative. Patients should be reviewed for at least two years after successful completion of treatment. Longer follow-up may be indicated depending on the level of drug resistance and the complexity of the treatment course.

Management of contacts of the multi-drug-resistant tuberculosis case

The risk of infection in contacts of an infectious MDRTB case is not significantly different than for contacts of drug susceptible cases. However, unlike the high level of proven efficacy of preventive treatment in the individual recently infected with a drug susceptible organism, treatment of individuals likely infected with an MDRTB strain is problematic. Their management should therefore be undertaken by those with appropriate TB clinical expertise.

Assessment of the individual exposed to an MDRTB case should consider the probability of recent infection and the subsequent risk of progression to active disease. The US Centers for Disease Control and Prevention guidelines recommend that treatment should be considered in those with a high probability of infection and an added risk factor (such as HIV co-infection) predisposing to progression to active TB disease. In those with a lower probability of recent infection, an observation alone approach or treatment as for the contact of a drug susceptible case was advised.

When preventive treatment is indicated, the consensus is that at least two drugs be used daily for a 6–12 month period based on the drug susceptibility results of the source case. The combination most commonly recommended is pyrazinamide plus a quinolone.

Avoiding the transmission of tuberculosis and multi-drug-resistant tuberculosis

The problem of TB and MDRTB can be exacerbated by transmission of infection to other patients and staff. A TB infection control program contains three principal strategies:

- administrative measures;
- engineering controls (e.g. negative pressure ventilated rooms); and
- personal respiratory protection (PRP, e.g. N95 masks, powered air-purifying respirator).

Recommendations from the Centers for Disease Control and other publications listed at the end of this document fully describe these three strategies. Recognising there has been an emphasis on recruiting healthcare workers from high-prevalence TB and MDRTB countries who have a higher risk of infection and disease, it is important that infection control services in Australia provide appropriate screening and health services to assist these recruits and to protect Australian healthcare services.

Administrative measures are the most important and cost-effective interventions for TB control. These measures aim to facilitate the early recognition and treatment of TB, and hence to prevent subsequent nosocomial transmission. Engineering controls and PRPs, while important, are expensive interventions and cannot compensate for imperfect administrative controls.

The key administrative measure is the prompt recognition of TB patients. Various algorithms have been developed to identify patients requiring isolation and investigation for TB. These algorithms consider the patient's symptoms (e.g. chronic cough, fever ≥ 3 weeks, loss of $> 10\%$ body weight) and epidemiological risk factors (e.g. contact with TB, migrant from a TB-endemic country). Hospitals and other health services must adapt these algorithms to their local circumstances, balancing the likelihood of TB in their patient populations, the availability of isolation rooms, and the 'costs' of TB transmission from undiagnosed patients.

Effective TB control in Australia is also dependent on what is happening in neighbouring countries; this is especially true for MDRTB. There is some evidence that movement of people from high risk areas, including the 'Torres Strait Protected Zone', represents a high risk for transmission of MDRTB to the Australian community.

Acknowledgement

We would like to thank the preparatory writers of this document, Rick Stapledon and Ivan Bastian and the current NTAC members.

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Current status

- NTAC endorsed the MDRTB Position Paper via teleconference on 9 March 2007;
- CDNA endorsed the MDRTB Position Paper on 18 April 2007;
- AHPC endorsed the MDRTB Position Paper on 12 October 2007.

Disclaimer

This paper captures the knowledge of experienced professionals, builds on past research efforts, and provides advice on best practice based upon the best available evidence at the time of completion.

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Observations on managing an outbreak of influenza A infection in an aged care facility

Bradley J McCall, Christine M Mohr, Kari AJ Jarvinen

Abstract

Influenza outbreaks in aged care facilities (ACFs) can be associated with high morbidity and mortality. National guidance includes the use of antiviral medication for residents and staff and other measures to prevent serious health outcomes. An outbreak of influenza in an ACF was reported to the Brisbane Southside Population Health Unit (BSPHU) on 10 August 2007. The BSPHU assisted the ACF and local general practitioners in the provision of oseltamivir to staff and residents on 11 August 2007. The onset of illness in the last case was 13 August 2007. Antiviral prophylaxis was ceased and the outbreak declared over on 22 August 2007. This paper describes some of the practical issues encountered in the public health response in this setting. Vaccination of ACF residents and staff remains the key preventive strategy for the future. *Commun Dis Intell* 2007;31:410–412.

Keywords: influenza, disease outbreak

Background

Influenza infection in aged care facilities (ACFs) is associated with an increased risk of poor health outcomes among residents, including death.[1] Consequently, residents and those who care for them are recommended to have annual influenza vaccinations to reduce the likely impact of seasonal influenza epidemics.[2] In recent years a number of influenza outbreaks in ACFs have led to the development of *Guidelines for the prevention and control of influenza in aged care facilities in Australia.*[3] We report on our experience with one outbreak and the deployment of a public health team to coordinate the provision of antiviral medication.

Influenza notifications increased in South East Queensland in July 2007 and peaked in mid-late August 2007. In total, there were 4,097 notifications of laboratory-confirmed influenza reported in Queensland in the calendar year up to 27 September 2007.[4] Two cases of rapid test kit confirmed influenza among residents of an ACF were reported to the Brisbane Southside Population Health Unit (BSPHU) on the afternoon of Friday 10 August 2007. At the time of reporting another nine residents were recognised with symptoms that met a working case definition for influenza-like illness (fever ≥ 38°C, cough and one of: myalgia; headache; sore throat; fatigue; or chills). The ACF had commenced isolation of sick residents on 9 August 2007.

Methods

An Outbreak Control Team (OCT) was formed to manage the public health response. Reference was made to the State Outbreak Control Team for guidance on the extent of provision of antiviral medication and management of associated issues including media. It was decided to offer antiviral medication (as treatment or prophylaxis) to all staff and residents regardless of vaccination status as both of the confirmed cases and most of the suspected cases had already received this years' influenza vaccine. Vaccination was recommended for those who had not previously received it. Throat swabs were collected from 11 suspected cases. Antiviral medication (oseltamivir) was obtained from state supplies and a public health team visited the ACF on the afternoon of 11 August to coordinate the provision of antiviral medication to staff and residents. Vaccine effectiveness (VE) was calculated using the cohort method in Epi Info 6.[5]

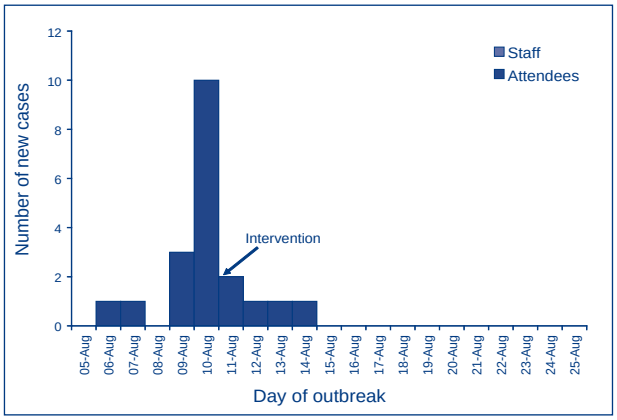
Results

In total, 79 residents (77% already vaccinated) and 45 staff (46% already vaccinated) were provided with oseltamivir on 11 August. Twenty residents (including two under treatment by their general practitioner) received treatment courses and 59 received chemoprophylaxis courses. A proportion received reduced doses on account of poor renal function, calculated from the most recent routine pathology tests held at the ACF.[3] A local general practitioner (GP) rendered generous assistance to the public health team and provided additional shiftwork staff with prophylaxis. Ten days of antiviral medication was supplied for each person. New cases were isolated and tested in accordance with standard procedures for controlling influenza in this setting.[3]

Twenty patients met the clinical case definition. Fourteen of these were noted to have received the 2007 influenza vaccine. Eleven patients had throat swabs collected with seven returning posi-

tive results for influenza A, subsequently typed as Influenza A Wisconsin/67/2005 (H3N2) in two patients. Confirmed cases were confined to hostel residents with no confirmed cases among residents in the immediately adjacent nursing home. The last case was recorded with onset on 14 August 2007 (Figure). There were no fatalities or hospital admissions recorded.

Number of cases in an influenza outbreak in an aged care facility, Queensland, August 2007



Forty-five staff (90%) were provided with antiviral prophylaxis. One staff member was unable to take either form of prophylaxis and was excluded. No staff reported symptoms. Public health measures were lifted and antiviral medication was ceased on 22 August 2007 after no new cases had been reported since 14 August.

Vaccine effectiveness for all residents in the facility using the clinical case definition was calculated as 31%. This increased to 44% when only laboratory confirmed cases were included. Among the hostel cohort using the clinical case definition VE was measured as 33%. This increased to 46% when only laboratory confirmed cases were included.

Discussion

This was the first time this intervention had been carried out in Queensland. We offer the following observations on our experience as potential learning points.

It is difficult to determine the precise impact of each of the measures on the progression of this outbreak. Although the VE was relatively low, the observation that no cases were very unwell or required hospitalisation suggests that the match with the current vaccine strain may have afforded protection from more serious outcomes. Isolation of cases, hygiene and other social distancing measures were an important part of the response and from our observation there appeared to be good compliance among staff and residents. It is tempting but not wholly justifiable to attribute more significance to the role of antiviral medication in terminating this outbreak.

On a weekend, BSPHU with the invaluable assistance of a local GP, was the only agency with the ability to conduct this intervention. However, even during the week, this intervention is of sufficient complexity that BSPHU staff would have to attend in person to provide support to the ACF staff, residents and families. The nature of shift work meant that all ACF staff could not be contacted or provided with treatment at one 'clinic'. Some staff were working in other ACFs which created additional infection control concerns.

Our intervention consolidated and gave consistency to the outbreak response. A less directly supportive approach may have resulted in delayed intervention, potentially significant leakage of antivirals to staff family members (with medical conditions) and staff attending a myriad of GPs with understandable differences in management.

Consent may be difficult to obtain in this setting. There were not sufficient resources to contact every legal guardian, so prophylaxis was provided after consent of each attending GP was obtained, and drug orders were written in medication charts.

Most residents had recent pathology tests, which allowed review of serum creatinine levels to guide antiviral dosage decisions in an elderly population.

The inclusion of a (influenza-vaccinated) pharmacist should be mandatory in any team approach to assist nursing home staff with dispensing. (This and the serum creatinine survey were the most time consuming parts of the exercise.)

One staff member required repeated counselling on the risks of taking the influenza back home to family members; another unvaccinated staff member had contraindications for both oseltamivir and zanimivir and was excluded from work until the outbreak was declared over. There remains substantial room for improvement in ensuring high rates of influenza vaccination among staff working in ACFs.

Agency staff required additional counselling and feedback to their agency about the intervention and the importance of vaccinating agency staff for the influenza as one unvaccinated staff member could not work in another facility as she was previously

rostered. Agencies should actively promote and provide influenza vaccination for their staff as part of their responsibility for workplace health and safety.

Facility management required support on managing the expectations of our response team. This was a huge intervention from the nursing home's perspective and required considerable flexibility in rostering staff, changing shifts and managing the medication issues. ACF staff required additional guidance and support on recording of temperatures and symptoms to meet the case definition.

A number of documents were sourced from other jurisdictions and formatted to suit this intervention. We gratefully acknowledge the work of other jurisdictions and the Australian Government Department of Health and Ageing and the Communicable Diseases Network Australia in developing essential forms and templates which facilitated the management of this outbreak.

The laboratory system supported this intervention well. Results were obtained on the day of the intervention and this was useful in determining the scope and direction of the response.

Personal Protective Equipment was available and all BSPHU staff deployed as part of this intervention had received the influenza vaccine.

The BSPHU supports more than 100 ACFs in the Brisbane Southside area. During a severe influenza season (as just experienced) it is likely that other outbreaks of influenza occurred in ACFs and were not reported to the BSPHU. The potential for a public health intervention in numerous ACFs would require deployment of considerable resources to support such a response. Vaccination of ACF residents and staff remains the key preventive strategy for the future.

Acknowledgements

Management and staff of the ACF, local medical practitioners, BSPHU staff members, Dr Christine Selvey (Communicable Disease Branch, Queensland Health) and members of the Queensland Health State Outbreak Control Team, Public Health Virology Staff, Forensic and Scientific Services, Queensland Health.

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Influenza surveillance within hospitals: what is the world doing?

Kylie S Carville, Heath A Kelly

Abstract

Influenza within hospitals is receiving increasing attention as a result of planning for an influenza pandemic and the magnitude and severity of the 2007 influenza season in Australia. This article reviews current approaches to influenza surveillance of admitted patients, as opposed to surveillance of emergency departments, in hospitals internationally. Most examples came from the United States of America and Canada, although systems have been described in the United Kingdom and Japan. In-hospital surveillance of influenza occurs within broader surveillance systems established by national governments, and through other systems established by sub-national governments and individual hospitals. Systems vary in focus, i.e. laboratory confirmed influenza or influenza-like illness, and some are labour intensive while others incorporate differing degrees of automation. The approach to influenza surveillance within hospitals will depend on objectives and available resources, although an automated approach is likely to have greater longevity as labour requirements are reduced. *Commun Dis Intell* 2007;31:413–418.

Keywords: Influenza, surveillance, hospital, admission, pandemic

Introduction

The avian influenza epidemic and preparedness for pandemic influenza, along with a number of publicised influenza-associated deaths in the 2007 influenza season,[1] have focused considerable attention on surveillance of influenza. In all Australian jurisdictions except South Australia laboratory-confirmed influenza is a notifiable disease, and surveillance for influenza-like illness (ILI) in the community, using general practitioner (GP) sentinel surveillance, is established in many Australian states and territories.[2] Syndromic surveillance of Emergency Departments (ED) has also been established in a number of Australian jurisdictions. In New South Wales, the well established ED surveillance system is utilised in the place of a sentinel GP system.[3] ED syndromic surveillance has often been established as part of bioterrorism preparedness, and a wealth of literature is available, predominantly from the United States of America (USA).[4] Syndromic surveillance can provide timely alerts of increased incidence of influenza in the population through identification and counts of triage text indicative of ILI, and counts of diagnosis codes related to influenza.[5] However, data collection does not generally extend into the hospital for admitted patients. Currently, assessment of discharge diagnosis codes for ILI occurs retrospectively as data are not available in a timely fashion.[2,6]

Thus our understanding of influenza admission rates and burden on hospitals is limited. International studies have analysed retrospective hospitalisation data to document the burden of influenza, however many focus on laboratory-confirmed influenza in children, which will underestimate the burden of disease.[7–9] Some studies, including one from New South Wales,[10] have sought to estimate the true burden of paediatric influenza related hospitalisation, however different methods provide different results. Routine sentinel surveillance in hospitals not only has the potential to increase our understanding of the burden of influenza and/or ILI-related hospitalisations, but to provide timely data for action for infection control practitioners (ICP) and to provide surveillance experience and systems that may be of use during a pandemic. We sought to review inpatient surveillance for influenza or ILI in comparable countries in order to inform approaches to this surveillance in Victoria.

Methodology

Searches of MEDLINE were last conducted in September 2007 utilising the terms 'surveillance AND (hospital OR hospitalisation) AND influenza'. Articles were excluded if it was clear that the focus of the title or abstract was not influenza surveillance (or syndromic surveillance including influenza-like illness) or hospital-based. Articles under the 'Related Links' heading were examined where the title and abstract suggested the article could be relevant. Searches were carried out with Google using the same terms to identify any surveillance measures detailed on the Internet but not yet published in peer-reviewed literature.

Approaches to in-hospital surveillance for influenza

Twenty-three articles describing surveillance of influenza, or influenza-like illness, in hospitalised patients were found. Importantly, most articles do not evaluate attributes of system operation such as timeliness, completeness of reporting, or actual costs.

Active influenza surveillance systems with specific study personnel

In Canada, surveillance of laboratory-confirmed influenza among both paediatric and adult admissions has utilised two systems, Immunization Monitoring Program ACTive (IMPACT) and the Toronto Invasive Bacterial Diseases Network (TIBDN). IMPACT is a national paediatric hospital-based active surveillance network for adverse events following immunisation, vaccine failures and selected vaccine preventable diseases in children aged less than 16 years.[11] The network involves 12 Canadian centres, representing about 90% of all tertiary care paediatric beds in Canada. These hospitals routinely perform viral diagnostic tests on children admitted with acute respiratory symptoms. Each IMPACT centre has a designated part time nurse who reviews medical records of children with laboratory-confirmed influenza to determine whether influenza was the cause of admission and, if so, to collect specific data.

TIBDN is a collaboration of all hospitals, microbiology laboratories, infection control practitioners, physicians and public health units serving the population of metropolitan Toronto and Peel Regions (population 3.7 million).[12] Surveillance for hospital admissions associated with laboratory-confirmed influenza, or positive rapid test, was conducted from 1 July 2004 to 30 June 2007. It was felt increased influenza testing may occur over the period under study, due to the advent of routine rapid testing for influenza and the attention focused on viral respiratory illnesses post SARS. Microbiology laboratories contacted TIBDN when an isolate of influenza was identified from an in-patient unit or the ED (where the patient was admitted), consent was sought by a study nurse and data collected by interview and chart review.

Data from TIBDN on adults indicated that a majority of patients (79%) had at least one underlying illness. Testing for influenza among adults was rare and a variety of laboratory approaches were used (some laboratories were using culture only, which is not a timely measure), ultimately impacting on clinical care, surveillance and costs.[13] IMPACT data can be assessed by season, region and age. Data indicate that half of the children admitted with influenza were otherwise healthy. Nearly half required supplemental oxygen, around 12% of admissions were to the ICU, and half of these required ventilation.[14–17] The need to evaluate impacts of changes to paediatric influenza immunisation recommendations in Canada (vaccination of all children aged 6–24 months) were used to promote surveillance.[15] Data from IMPACT are incorporated into FluWatch, the Canadian national influenza surveillance network, and are reported alongside viral detection and strain identification data and sentinel practitioner ILI consultations.[18,19]

In the USA two Centers for Disease Control and Prevention associated systems, the New Vaccine Surveillance Network (NVSN) and, to a lesser extent, the Emerging Infections Program (EIP) Network, have been used to conduct influenza surveillance. The NVSN, established in 1999, evaluates the impact of new vaccines and vaccine policies through a network of sites that conduct population-based surveillance, among other research.[20] Active surveillance of hospitalisation with acute respiratory illness is conducted in children aged under five years in three urban counties. Study nurses identify children admitted over 4 days of the week (96 hours) (increased to 7 days in 2004–2005) with a diagnosis (by admitting physician) that fits the broad case definition of acute respiratory infection. When informed consent is obtained, swabs are taken for respiratory virus polymerase chain reaction (PCR) testing, and medical record review and parent interview are conducted.

The EIP is designed to assess the public health impact of emerging infections and evaluate methods for surveillance, prevention and control.[21] Some EIP sites began identifying cases of laboratory-confirmed influenza-associated hospitalisations in patients aged under 18 years in 2003, chiefly through review of hospital laboratory lists of influenza positive results. EIP surveillance is thus cheaper and logistically simpler to implement than NVSN, although EIP depends on whether practitioners order influenza tests and can be affected by the lower sensitivities of rapid diagnostic tests.

NVSN data have shown that older children are more likely to require oxygen than younger children, and that 72% of children whose hospitalisation for acute respiratory infection or fever in 2000–2004 was attributable to laboratory confirmed influenza, were not assigned a discharge diagnosis of influenza.[22] Admission rates have been seen to vary across seasons, institutions and ages. In 2000–2001 one third of children had one or more underlying medical conditions, 80% of influenza associated paediatric hospitalisations were in children under two years, and 3% of children enrolled had a positive influenza test.[23,24]

EIP data from 2003-2004 showed that 25% of children hospitalised with laboratory-confirmed influenza received antiviral therapy and that 35% of children aged over 6 months had received at least one influenza vaccination, although these figures vary across hospitals.[25] Surveillance data from EIP were compared with a retrospective audit of discharge data for a range of ICD codes previously shown to reflect influenza in children. This showed that the incidence of hospitalisations for influenza based on these codes was around 10 times higher than those with laboratory evidence.[25]

Data from NVSN and EIP were used to perform a capture-recapture analysis to better estimate the number of children hospitalised with influenza.[26,27] The NVSN identified a greater proportion of children with influenza than did the EIP (69% and 39%, respectively, using capture-recapture estimates as a reference), however, it did not achieve complete ascertainment despite the resources invested in the program. This was largely due to atypical presentations that did not meet enrolment criteria. The authors state that capture-recapture can be used to obtain better estimates about the total number of influenza cases from these two imperfect systems, and that the more expensive, sensitive system (NVSN) would thus not need to operate full time.

A pilot was conducted in the West Midlands region of the United Kingdom over two winter seasons (2001–2002 and 2002–2003) to determine the burden of influenza and other respiratory infections among respiratory patients and to assess the feasibility of their approach as a surveillance tool.[28] Nurses were employed to conduct a daily review of admissions, enrol patients, and take samples for PCR testing. There was little influenza activity in the seasons studied, limiting assessment of the burden of influenza and other respiratory viruses on winter bed pressures. The authors did not comment on the potential of the overall system to function beyond suggesting (potentially expensive) routine diagnostic assessment of respiratory patients using PCR.

Active influenza/ILI surveillance systems that utilise existing hospital staff

The International Medical Centre of Japan conducted syndromic surveillance for acute respiratory infections for three winters, as preparation for any future re-emergence of SARS or a novel influenza pandemic.[29,30] The system encompassed patients and staff. A case was defined as a patient who had a fever and one or more symptoms of respiratory tract infection. The system was labour intensive, requiring surveillance forms to be completed by section heads with daily follow up by ICP. Rapid tests for influenza were recommended for cases; use of rapid tests increased over subsequent seasons. Results were documented weekly on the hospital intranet. The authors state that the system clearly documented sudden outbreaks of influenza in the hospital, but did not specify whether this system assisted with outbreak identification. They did state that staff with influenza were instructed to undergo treatment at home, which they believe assisted in control of nosocomial infection. As no additional study staff were utilised (unlike IMPACT or NVSN), cooperation of general hospital personnel and effective functioning of the infection control team was essential. The authors reported a decrease in the number of reports after the seasonal peak compared with before, which they attributed to a sense of 'impending crisis' in physicians and nurses prior to peak, which then decreased.

Some USA states have developed their own influenza surveillance systems. Colorado has established a laboratory-confirmed surveillance system for influenza hospitalisations, as influenza-associated hospitalisation was made notifiable in the state in 2004.[31] ICP review laboratory and admission information and report over the Internet or via facsimile. While underestimating the burden of influenza as it is based on positive tests (including less sensitive rapid diagnostic tests), it does provide data on all ages (NVSN is children only). Reported cases peaked in the same week as reports from sentinel health-care providers in the state.

In California, where influenza is not a notifiable disease, the Department of Health Services initiated enhanced surveillance of paediatric intensive care units (ICU) in December 2003 following reports of severe impacts from the new Influenza A/H3N2 strain.[32] ICP collected data on children aged under 18 years with a clinical syndrome consistent with influenza; laboratory confirmation; and paediatric ICU admission; or death anywhere in the hospital. A report on the first two seasons of the program indicated no incentive was offered for collection of data, but did not provide information on completeness or timeliness of reporting. Data produced by the system included age profile (more than 80% under 5 years), underlying medical conditions (suffered by 53%), and vaccination (only 16% of patients were vaccinated).

The Connecticut Department of Public Health established hospital admissions syndromic surveillance (HASS) in 2001. In this partially automated system, hospital staff conduct a daily review of the previous day's admissions, categorise admissions into 11 syndrome categories and submit aggregate data via a secure website. The report states that this requires only 10–15 minutes per day. The use of case counts simplifies the system but without case-based demographic data further analyses are not possible. Excess pneumonia admissions (over annual weekly average) paralleled laboratory confirmation of influenza and sentinel GP reporting, however there was a slight lag.[33, 34]

Automated influenza-like illness surveillance systems

In California, in addition to the data generated by the paediatric ICU surveillance system described above, hospitalisation data from the main health maintenance organisation in the state (providing care to over one sixth of Californian residents) on 'flu admits' ('pneumonia', 'influenza' or 'flu' in hospital

admission diagnosis field) are also collected. Data are electronically extracted and transmitted daily to the California Department of Health Services; data are compiled weekly as the proportion of hospitalisations that were 'flu admits'.[35] Data from both paediatric ICU surveillance and automated hospitalisation surveillance are compiled into a comprehensive Californian influenza surveillance system, which includes outpatient ILI visits, school-based ILI surveillance, antiviral prescription data, sentinel laboratories and the state reference laboratory. Louie et al, state that these strategies are simple, flexible, stable and acceptable, and cover a range of unique populations in order to contribute to a more complete picture of influenza activity in the state.[35]

An attempt to automate surveillance of pneumonia in two neonatal ICUs in New York used a natural language processor, which created coded clinical information from computerised laboratory and radiology reports.[36] This system was evaluated by comparison with prospective identification of cases by ICP. The system had a positive predictive value of 8% but a negative predictive value of 99%, leading the authors to suggest it could be used to screen out negatives and enable ICP to focus on the highest risk cases.

The University of Utah Hospital in Salt Lake City established an automated surveillance system within the University Hospital, based on electronic medical records, for the Winter Olympics in 2002.[37] Project staff aimed to develop a system with access to real-time medical record information, as it was felt that ED surveillance systems were limited by the lack of immediate access to detailed patient level data. The approach was intended to make it easier for ICP to assist public health agencies with timely surveillance by decreasing the number of false positive alerts sent to public health authorities, without using substantial ICP time. ICP led a team that developed a rule-based system used to identify patients who fit within certain infection syndromes, including 'hospitalised influenza'. The electronic system considered items such as patient contact data (including ICU admission and death), test ordering and results, and used a statistical technique called CUSUM to determine an upper limit for the number of cases expected. Alerts were generated when this upper limit was exceeded. ICP had intranet access to the system to view both aggregated and individual patient data, enabling review of the detailed electronic medical records. Increased influenza activity (largely resulting from a separate project for influenza surveillance in the athletes' village) was the only confirmed public health event of significance reported to local public health authorities. While the use of such a system would depend on the type of data entered into patient management, laboratory and other electronic data systems within a given hospital, this approach could be applied in other institutions.

Conducting in-hospital surveillance in Australia

Beyond pilot programs in two hospitals in Victoria, we are unaware of routine influenza or ILI surveillance of admitted patients in Australia. The needs and resources of hospitals and health departments will shape routine influenza/ILI surveillance objectives and thus the type of system, if any, to be developed. Surveillance systems such as IMPACT and NVSN require ongoing investment in labour and resources. In contrast, while the initial establishment of an automated system may require substantial resources, ongoing requirements would be less. The utility of automated systems will depend on the existence and quality of data that can be obtained electronically. Laboratory requests, results and burden information (such as length of stay, admission to ICU, ventilation, and death) should be accessible electronically in many hospitals. Manual record review may be needed for some data unless hospitals have extensive medical records. Other factors, such as the case definition of ILI (influenza presentations to the ED have been shown to exhibit confusing symptoms[38]) and use of diagnostic testing for at least some cases, would need to be addressed in the development of an ILI surveillance system. An effective routine system could rely in part on automation of case identification and data extraction from ED, patient management and laboratory data systems. Individual hospitals could value-add by manual collection of any additional information required for their own purposes, with a substantially reduced workload compared to a completely manual system. Both labour intensive and automated systems can provide timely data to enable ICP to enact infection prevention measures however, electronic systems may be more likely to function during a pandemic when high workforce absenteeism is likely. Within hospital response may be the responsibility of ICP, who then need to be resourced appropriately.

Retrospective reviews of influenza-associated hospitalisations can provide influenza burden data with which to inform policy and practice, but are estimates limited by lack of routine testing and discrepancies between discharge coding and test results, and data may not be reviewed at individual hospital level. Real time measures of the burden on hospitals may assist in timely reallocation of resources during years of high seasonal activity. Beyond alerting ICP and providing information on the burden in hospitals, data from routine sentinel influenza/ILI surveillance in hospitals could contribute to existing influenza surveillance systems, as use of more than one surveillance system improves the age range of

patients captured by surveillance and allows validation of findings.[6] A pilot program modelled on the IMPACT system, Paediatric Active Enhanced Disease Surveillance (PAEDS) commenced in Australia in August 2007.[39] This program focuses on acute flaccid paralysis, intussusception, severe varicella and seizures in children aged one month to less than 8 months. It may be worth considering whether there is scope for PAEDS to expand to influenza in children if the pilot is successful. Indeed, during the 2007 influenza season, following reports of child deaths attributed to influenza, the Department of Health and Ageing engaged the Australian Paediatric Surveillance Unit to conduct weekly active surveillance for the month of September on cases of severe complicated influenza in children aged under five years.[40] Development and refinement of ED syndromic surveillance systems around the country could consider extending the work to alert ICP and other relevant hospital staff of the admission of infectious patients, and collection of useful electronic data for admitted patients. Depending on resources, hospitals may be able to conduct stand alone influenza surveillance to some extent, but a commitment to the establishment of electronic systems that would serve routinely and in a pandemic may represent a better use of resources.

Acknowledgements

This work was funded by the Australian Government Department of Health and Ageing.

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Letters

Letter to the Editor

Submitted in response to: Craig A, Armstrong P. *Exercise Paton:* A simulation exercise to test New South Wales emergency departments' response to pandemic influenza. *Commun Dis Intell* 2007;31:310-313.

Editor,

Re: *Exercise Paton*: A simulation exercise to test New South Wales Emergency Departments' Response to Pandemic Influenza

The short report on Exercise Paton clearly demonstrates Australian jurisdictions' commitment to preparing for an influenza pandemic.[1] The exercise focused on containment activities, which will form the critical first phase of any Australian response.[2] In order for containment to be effective, public health staff need to be able to rapidly identify suspected cases of pandemic influenza, that is, people with a history of recent contact with pandemic influenza who have onset of fever within the previous 24 hours.[3] Furthermore, successful home quarantine for contacts of pandemic influenza cases will depend on their ability to reliably monitor themselves for symptoms of influenza, including fever. The interim case definition for pandemic influenza includes a specific criterion for fever of ≥38ºC.[4] It is assumed that most community contacts of pandemic influenza will have a thermometer at home to perform daily or twice daily temperature monitoring.[2] However, there is scant information regarding the availability of thermometers in Australian households.

Following the June 2007 long-weekend natural disaster in the Hunter region of New South Wales, we conducted a random survey of 227 households in the local government areas of Newcastle and Lake Macquarie in New South Wales, to assess household disaster preparedness.[5] Our response rate was 71% and households were representative of recent census demographics. We found that only 48% (95% confidence interval 41–54%) of households had a thermometer available at home. This finding indicates that many community contacts of pandemic influenza cases would have difficulty monitoring their temperature at home and be unable to accurately report the development of fever while under home quarantine.

In order to support essential public health activities, including screening, surveillance and home quarantine, Commonwealth and State Governments should include household thermometers in their medical stockpiles. Careful thought should also be given to other essential components of a 'home quarantine starter pack' containing basic supplies that will assist contacts to remain at home. Such preparations may prove vital for successful containment of pandemic influenza or other future infectious disease epidemics that Australia may face.

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Quarterly reports

OzFoodNet quarterly report, 1 July to 30 September 2007

The OzFoodNet Working Group

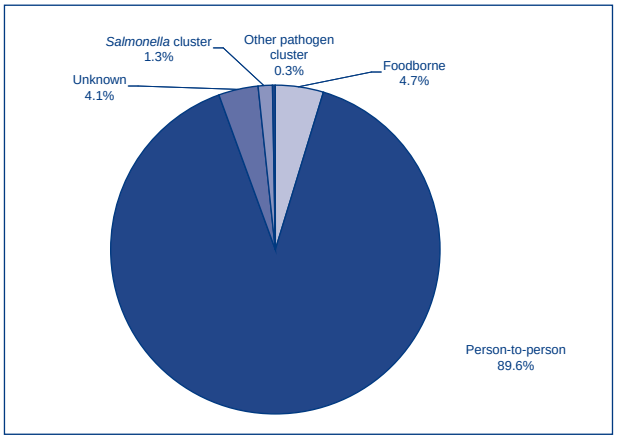
Introduction

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 investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 July to 30 September 2007.

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 third quarter of 2007, OzFoodNet sites reported 761 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 under-represent the true burden of enteric illness. In total, these outbreaks affected 16,058 people, of which 281 were hospitalised and 53 people died. The majority (90%, n=682) of outbreaks resulted from infections due to person-to-person transmission (Figure 1).

Figure 1. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 July to 30 September 2007



Foodborne disease outbreaks

There were 36 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 502 people and resulted in 12 people being admitted to hospital. There were no deaths. This compares with 23 outbreaks for the third quarter of 2006 and 34 outbreaks in the previous quarter of 2007.

Salmonella was responsible for eight outbreaks during this quarter, with *Salmonella* Typhimurium being the most common serotype. *S.* Typhimurium 135a was responsible for two outbreaks, *S.* Typhimurium 44 and *S.* Typhimurium 193 were each responsible for one outbreak. The other *Salmonella* serotypes causing outbreaks were *S.* Virchow 45, *S.* Dublin, *S.* Oslo and *S.* Singapore.

Norovirus was associated with eight foodborne outbreaks during this quarter. *Campylobacter* was identified in three outbreaks and there was one outbreak of *Shigella sonnei* biotype g. There were three toxin-related outbreaks during the quarter including two ciguatera fish poisoning outbreaks and a *Clostridium perfringens* intoxication outbreak. The remaining 13 outbreaks were caused by unknown aetiological agents.

Thirteen outbreaks reported in this quarter were associated with food prepared by restaurants, six from food prepared in aged care facilities, six from food prepared by commercial caterers, five from food prepared by takeaway outlets, and three outbreaks were from contaminated primary produce. Single outbreaks were associated with food prepared in an institution and private residence. There was one outbreak where the food preparation setting was unknown.

To investigate these outbreaks, sites conducted seven cohort studies and one case control study, and collected case series data on 22 outbreaks. There were six outbreaks where no individual patient data were collected. Investigators obtained analytical epidemiological evidence in four outbreaks and microbiological evidence in one outbreak. For the remaining 31 outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2007

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
NSW	July	Restaurant	Unknown	6	D	Suspected mushrooms and cos lettuce
	July	Restaurant	Unknown	5	D	Suspected chicken schnitzel
	July	Restaurant	Unknown	3	D	Suspected bruschetta and parmesan cheese
	August	Takeaway	Unknown	2	D	Suspected cooked rice
	August	Takeaway	Unknown	4	D	Beef and chicken kebabs
	August	Takeaway	Unknown	5	D	Unknown
	August	Restaurant	Unknown	3	D	Unknown
	August	Aged care facility	Unknown	9	D	Suspected beef sausages
	September	Restaurant	Norovirus	19	A	Oysters
	September	Takeaway	Unknown	3	D	Unknown
	September	Restaurant	*Salmonella* Singapore	5	D	Unknown
	September	Unknown	Unknown	2	D	Unknown
	September	Aged care facility	Unknown	6	D	Suspected tiramisu and cream, fruit salad, strudel and custard
	September	Commercial caterer	Unknown	17	D	Unknown
NT	July	Contaminated primary produce	Ciguatera fish poisoning	2	D	Reef cod
	August	Commercial caterer	*Salmonella* Oslo	3	D	Suspected roast pork
	September	Commercial caterer	Norovirus	8	D	Ill food handler suspected
Qld	August	Restaurant	Norovirus	24	A	Ill food handler suspected
	August	Restaurant	*S.* Typhimurium 135a	8	D	Duck pate
	August	Contaminated primary produce	*Shigella sonnei* biotype g	55	M	Baby corn
	September	Contaminated primary produce	Ciguatera fish poisoning	5	D	Coral trout
	September	Institution – other	Norovirus	35	D	Ill food handler suspected
SA	July	Private residence	*S.* Typhimurium 193	13	A	Unknown
	July	Restaurant	Norovirus	14	D	Unknown
	August	Aged care facility	*Campylobacter*	6	D	Unknown
	September	Commercial caterer	Norovirus	24	D	Unknown
Tas	September	Restaurant	*S.* Typhimurium 135a	2	D	Sushi
Vic	July	Restaurant	Norovirus	21	D	Ill food handler suspected
	July	Aged care facility	*Campylobacter*	6	D	Unknown
	July	Aged care facility	*Clostridium perfringens*	30	D	Several foods were suspected
	August	Commercial caterer	Unknown	20	A	Roast chicken and/or stuffing
	August	Aged care facility	*Campylobacter*	6	D	Unknown
	August	Restaurant	*Salmonella* Dublin	6	D	Unknown
	September	Restaurant	Norovirus	96	D	Ill food handler suspected
WA	August	Commercial caterer	*S.* Typhimurium 44	7	D	Unknown
	September	Takeaway	*S.* Virchow 45	22	D	Suspected sushi

* No foodborne outbreaks were reported in the Australian Capital Territory 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.

The following jurisdictional summaries describe key outbreaks which occurred in this quarter.

New South Wales

New South Wales reported 14 outbreaks of foodborne illness during this quarter. Norovirus caused 19 restaurant patrons to be ill in one outbreak during September. A cohort investigation showed a strong association between illness and oyster consumption (estimated RR11.2, 95%CI, 1.6–77.3). *Salmonella* Singapore affected five people over a 6-week period and all cases implicated a single restaurant. Four cases had a positive stool result for *S.*Singapore. Investigators were unable to identify a common food source. An aetiological agent was not identified for the remaining 12 outbreaks, which affected between two and 17 people.

Northern Territory

The Northern Territory reported three outbreaks during the quarter. Norovirus caused an outbreak at a remote mine site where food was provided by a commercial catering company on site. The spread of illness was likely to have been foodborne as a seconded staff member, not trained in food handling, worked while symptomatic with gastroenteritis illness. Norovirus was detected in a clinical specimen from the ill food handler while in hospital. *Salmonella* Oslo was identified in two people who were ill after eating roast pork prepared by a catering company and eaten at a private party. The roast pork was reportedly undercooked and the catering business was unregistered. Another case became ill after they consumed left over roast pork during a picnic the next day.

Queensland

Queensland reported five outbreaks during the quarter. Norovirus caused two outbreaks of gastrointestinal illness and the spread of illness for both was due to food handlers working while they were unwell. There was an outbreak of norovirus where salad was significantly associated with illness among 24 patrons who had dined at a restaurant, and there was an outbreak of norovirus associated with a breakfast meal that caused illness among 35 students of a residential college.

Salmonella Typhimurium 135a contaminated a duck liver pate that caused illness among eight restaurant patrons. The making of the pate did not include a satisfactory cooking or cleaning process of the duck livers before preparation. *S.* Typhimurium (not 135a) was detected in a sample of raw duck liver from the restaurant.

Shigella sonnei biotype g caused a community-wide outbreak of foodborne illness during August. Initially, this outbreak was identified in a film production crew with 43 epidemiologically linked cases reported to Queensland Health. Further cases were subsequently reported from the wider community. A concurrent outbreak of *Shigella sonnei* biotype g associated with fresh baby corn was reported in Denmark.[1] Clinical specimens from cases in Australia and Denmark had indistinguishable pulsed-field gel electrophoresis (PFGE) patterns and identical antibiograms.[2] All Australian cases, and a New Zealand case that had stayed at a Queensland resort, reported consumption of fresh baby corn prior to illness onset. The fresh baby corn was imported from Thailand in a consignment during late July by a single wholesaler in Queensland. Investigators were able to establish a common source for the fresh baby corn in both the Danish and Queensland outbreaks.[3]

South Australia

South Australia reported four outbreaks during the quarter. There was an outbreak of *Campylobacter* in six residents from an aged care facility. The food causing this outbreak was not identified despite a food and environmental investigation of the facility.

Norovirus is suspected to have caused two groups of people to develop gastroenteritis after eating at the same restaurant on the same day. A faecal specimen from one of the cases tested positive for norovirus. All other food and environmental sampling did not detect norovirus or other pathogens. Norovirus is also suspected to have caused illness on a film set operating in rural South Australia. Foodborne transmission was suspected because 24 of 55 participants had an onset of illness within a two hour period. Two clinical samples were positive for norovirus but a food and environmental investigation was unable to identify the source of infection.

South Australia also investigated an outbreak of *Salmonella* Typhimurium 193 among 13 people associated with a meal at a private residence. The aetiological agent was detected in clinical specimens from eight of these cases. The food vehicle for this outbreak was not identified despite an investigation that included food and environmental sampling.

Tasmania

Tasmania reported a single outbreak of two cases of *Salmonella* Typhimurium 135a during the quarter. The onsets of infection for the two cases were one day apart, in late September, and food histories from both cases included eating at a common

sushi restaurant. Investigators found no links from the restaurant to businesses associated with recent *S.* Typhimurium 135a outbreaks in Tasmania [4].

Victoria

Victoria reported seven outbreaks of foodborne illness during the third quarter. Norovirus caused two outbreaks where the food was likely to have been contaminated with norovirus by food handlers working while they were infectious. In one of these outbreaks, 96 people from 13 different groups (total 290 people) reported gastroenteritis after eating at the restaurant. In the second norovirus outbreak, illness was identified in at least four different groups (21 cases) who ate at the same restaurant on the same day.

Salmonella Dublin caused illness in three separate groups (6 cases) that dined at the same restaurant. The restaurant was located in a rural area and was connected to tank water. Eggs were sourced from the proprietor's own chickens and also from a commercial brand. Raw eggs were used in a tiramisu dessert served on the day that cases dined. Water, eggs, and various animal faecal specimens from the proprietor's farm were tested and all were negative for *Salmonella*. Food handlers were interviewed and none reported illness—they were all screened and were negative for *Salmonella*. The source of the outbreak was not identified.

Victoria investigated two separate outbreaks of gastroenteritis among residents of aged care facilities. In each outbreak there were six cases, two of whom were confirmed with *Campylobacter* infection. The mode of transmission was suspected to have been foodborne for both of these outbreaks due to clustering of illness onsets but a specific food source could not be identified for either.

Clostridium perfringens caused 30 cases of illness among residents of a Victorian aged care facility. *C. perfringens* enterotoxin was detected in faecal specimens of 13 cases. It is suspected that inappropriate use of leftover foods and inadequate cooling and reheating of foods were the contributing factors in the outbreak.

Victoria investigated an outbreak of unknown aetiology among 20 of 85 guests attending a wedding. A commercial caterer provided foods that included roast chicken with stuffing. It is suspected that either *C. perfringens* enterotoxin or *Bacillus cereus* diarrhoeal enterotoxin was the aetiological agent for this outbreak due to the incubation period, the duration of illness and symptoms. One specimen from a case was positive for *C. perfringens* enterotoxin and grew *B. cereus* in culture. Inadequate cooling and reheating of chicken and its stuffing was thought to have caused the outbreak.

Western Australia

Western Australia reported two outbreaks of foodborne illness during the quarter. *Salmonella* Virchow 45 affected 22 people in an outbreak associated with sushi. Cases reported eating from two sushi outlets that were owned and operated by the same people. The mayonnaise used in the sushi at both outlets was prepared by one person, and was made using raw eggs from a Queensland supplier. The PFGE profile of the *S.* Virchow isolates from WA outbreak cases was indistinguishable from three clinical and two egg pulp isolates collected from Queensland during 2007. *Salmonella* Typhimurium 44 affected five people in an outbreak associated with a university college. However, a further two cases of *S.* Typhimurium 44 with indistinguishable PFGE type did not eat at the college. Environmental samples and faecal samples from food handling staff were negative for *Salmonella*. The source of the outbreak was not identified.

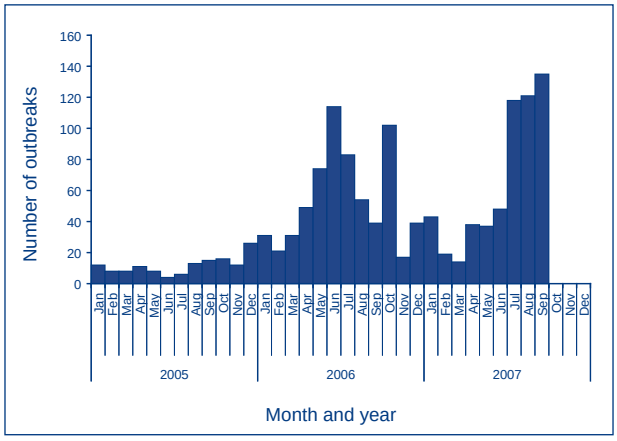
Australian Capital Territory

The Australian Capital Territory did not report any foodborne outbreaks during this quarter of 2007.

Comments

OzFoodNet sites reported 374 outbreaks due to person-to-person transmission of norovirus during this quarter of 2007 and 573 outbreaks of person-to-person norovirus for the first 9 months of 2007 (Figure 2). This compares with 176 person-to-person norovirus outbreaks for the third quarter of 2006.

Figure 2. Outbreaks of non-foodborne norovirus, Australia, January 2005 to September 2007, by month of notification to OzFoodNet sites



Source: OzFoodNet sites

During this quarter of 2007 a new strain of norovirus, designated 2006b, which had previously affected Europe during 2006,[5] caused widespread outbreaks of disease in eastern states of Australia (personal communication, W Rawlinson, October 2007).

Gastroenteritis outbreaks caused by norovirus occur all year round and are more commonly reported where people are in 'communal arrangements', for example, aged care homes, hospitals, schools, and cruise ships. Norovirus is highly infectious and easily spread from one infected person to another. The onset of illness often includes sudden vomiting, where infectious airborne particles can be easily spread to surfaces where virus survive for long periods of time.[6] Outbreaks of non-foodborne gastroenteritis caused by norovirus are common with hundreds of outbreaks reported to state and territory health departments each year.[7] Guidelines for managing gastroenteritis outbreaks due to norovirus are available from state and territory health departments.

Food handlers, who worked while infectious are suspected to have contaminated food in more than half of the foodborne outbreaks of norovirus (five of eight outbreaks) during this quarter of 2007. These outbreaks highlight the need to maintain procedures that prevent the contamination of food during preparation.[6] Some states require food handlers to be excluded from food handling for at least 48 hours after the resolution of symptoms. Norovirus can be excreted for some time after symptoms resolve, therefore it is important that food handlers maintain good personal hygiene on returning to work to protect food from contamination.

The outbreak of shigellosis associated with baby corn highlighted the increasing importance of imported food as a potential source of disease. There have been 14 outbreaks due to imported food since 2001, many of which are due to novel infections, such as multi-drug resistant *Shigella sonnei* biotype g (OzFoodNet unpublished data). The global nature of foodborne illness highlights the importance of rapid communication tools, such as Eurosurveillance and Promed for alerting countries to potential multi-country spread of disease.[3]

Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would also like to thank laboratories conducting serotyping and phage typing of *Salmonella* for their ongoing work during the quarter.

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Communicable diseases surveillance

Highlights for 3rd quarter, 2007

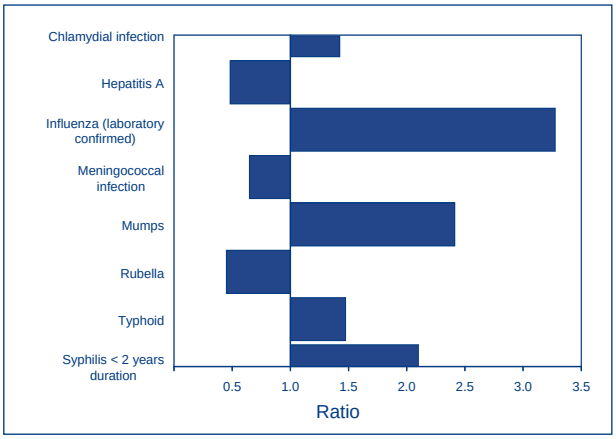
Communicable diseases surveillance highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. These national data collections 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 and 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' 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 third quarter of 2007 (July to September), compared with the 5-year mean for the same period.

Notifications were above the 5-year mean for chlamydia, influenza (laboratory confirmed), mumps, typhoid and syphilis of less than 2 years duration. Notifications were below the 5-year mean for hepatitis A, meningococcal infection and rubella.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2007 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.

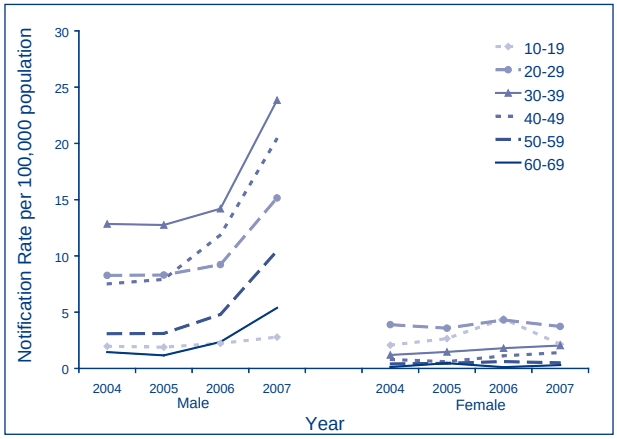
Sexually transmissible infections

Syphilis infections

There were 288 cases of syphilis (less than 2 years duration) reported to NNDSS in the third quarter of 2007, giving a national notification rate of five cases per 100,000 population (Figure 2). Males in the 35–39 year age group (29 cases per 100,000 population) and females in the 20–24 year age group (4 cases per 100,000 population) had the highest rates of notification. The Northern Territory recorded the highest notification rate with 29 cases per 100,000 population, however this was 18% less notifications compared with the same period in 2006.

Compared to the same period in 2006, the number of syphilis (less than 2 years duration notifications) have increased nationally by 38%. The major increases have been in Victoria (57%) and New South Wales (30%).

Figure 2. Notification rates of syphilis (less than 2 years duration) in persons aged 10–69 years, Australia, 2004 to 2006, by age group and sex



Vaccine preventable diseases

Influenza

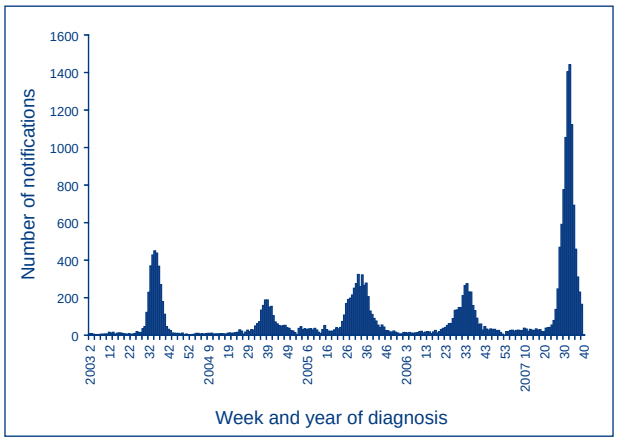
Laboratory-confirmed influenza is a nationally notifiable disease in all states and territories except South Australia, however data are reported from all state or territory health departments to the NNDSS.

The 2007 influenza season began in late May with a very gradual increase in notifications. From 15 July there was a steep rise in influenza notifications in several jurisdictions, particularly Queensland and Western Australia. Nationally, notifications peaked in mid August.

The total number of laboratory-confirmed influenza notifications to NNDSS for the third quarter was 8,958 cases (91% of year-to-date notifications); this was 3.3 times the 5-year mean for the same period. The number of notifications was more than three times the number reported for the same period in the previous four seasons (Figure 3). The majority of notifications were from Queensland with 3,861 cases (43%).

During the third quarter of 2007, the highest rate of notifications occurred in the Australian Capital Territory with 427 cases per 100,000 population, followed by Queensland (374 cases per 100,000), the Northern Territory (316 cases per 100,000), Tasmania (302 cases per 100,000), South Australia (163 cases per 100,000), Western Australia (153 cases per 100,000), Victoria (107 cases per 100,000) and New South Wales (78 cases per 100,000). The rate of notification of influenza infection for Australia was 170 cases per 100,000 population.

Figure 3. Number of influenza notifications, Australia, 1 January 2003 to 30 September 2007, by date of diagnosis



Measles

Four notifications of measles were reported in the third quarter of 2007. There were two males and two females reported aged between 1 and 22 years. One case was a student from Japan, two cases had returned from overseas (from Indonesia and the Middle East), and one case had no history of travel. One case was unvaccinated and three cases had an unknown vaccination history.

Acknowledgments

Thanks go to staff of the Surveillance Policy and Systems Section of the Australian Government Department of Health and Ageing and all our state and territory data managers.

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 41,649 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2007 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

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
Varicella infections (chickenpox)	All jurisdictions except NSW
Varicella infections (unspecified)	All jurisdictions except NSW
Varicella zoster infections	All jurisdictions except NSW
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 July to 30 September 2007, by date of onset*

Disease	State or territory								Total 3rd quarter 2007†	Total 2nd quarter 2007	Total 3rd quarter 2006	Last 5 years mean 3rd quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	3	7	1	16	5	0	12	9	53	76	67	83.8	208	245.6	0.6
Hepatitis B (unspecified)	30	1,148	36	216	115	7	478	157	2,187	1,970	1,682	1,518.4	6,153	4,657.2	1.3
Hepatitis C (incident)	2	7	1	NN	8	8	29	20	75	74	94	110.0	242	341.6	0.7
Hepatitis C (unspecified)	57	1,707	59	653	131	63	657	323	3,650	3,471	3,127	3,210.2	10,888	10,010.0	1.1
Hepatitis D	0	3	0	1	0	0	2	4	10	7	10	6.4	26	23.0	1.1
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.1	1	1.0	1.0
Campylobacteriosis§	69	NN	77	911	825	157	1,262	505	3,806	4,430	3,903	3,325.6	13,168	11,055.4	1.2
Cryptosporidiosis	0	50	14	23	28	12	78	62	267	672	313	625.4	2,098	2,078.0	1.0
Haemolytic uraemic syndrome	0	2	0	0	0	0	0	0	2	3	0	2.4	13	9.4	1.4
Hepatitis A	1	17	1	7	2	0	13	3	44	41	55	86.0	132	273.6	0.5
Hepatitis E	0	2	0	0	0	0	0	0	2	7	6	4.6	15	17.8	0.8
Listeriosis	0	5	0	2	2	0	1	0	10	8	15	16.2	34	46.8	0.7
Salmonellosis (NEC)	21	336	89	303	185	17	312	279	1,542	2,521	1,244	1,879.4	7,585	5,885.8	1.3
Shigellosis	1	21	34	34	25	0	27	27	169	148	103	138.8	460	424.4	1.1
SLTEC, VTEC‖	0	0	0	4	3	0	3	2	12	18	16	16.8	72	46.8	1.5
Typhoid	0	7	2	1	1	2	6	0	19	18	15	12.0	74	50.2	1.5
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	0	0.8	2	2.6	0.8
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 July to 30 September 2007, by date of onset,* *continued*

Disease	State or territory								Total 3rd quarter 2007†	Total 2nd quarter 2007	Total 3rd quarter 2006	Last 5 years mean 3rd quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	263	2,872	467	3,106	787	271	2,750	1,729	12,245	12,951	11,628	9,069.4	38,496	27,024.8	1.4
Donovanosis	0	0	0	1	0	0	0	0	1	0	0	2.8	3	9.0	0.3
Gonococcal infection	5	253	353	317	56	13	235	443	1,675	2,108	1,925	1,957.0	5,791	5,645.8	1.0
Syphilis (all)	12	315	50	85	13	10	267	47	799	793	658	571.2	2,313	1,713.2	1.4
Syphilis < 2 years duration	1	66	15	48	2	4	127	25	288	365	180	166.0	946	482.3	2.1
Syphilis >2 years or unspecified duration	11	249	35	37	11	6	140	22	511	428	478	414.0	1,367	1,207.3	1.1
Syphilis - congenital	0	2	0	0	0	0	0	0	2	3	2	5.8	7	12.0	0.6
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	3	0	1	1	0	0	0	5	4	11	5.2	11	16.8	0.7
Influenza (laboratory confirmed)	356	1,359	164	3,861	650	378	1,389	801	8,958	576	2,256	444.6	9,878	3,014.2	3.3
Measles	0	2	0	3	0	0	0	1	6	3	2	27.8	13	51.2	0.3
Mumps	1	80	16	17	14	2	3	14	147	99	93	44.0	291	120.6	2.4
Pertussis	35	548	5	482	170	6	292	21	1,559	1,360	4,559	1,704.2	3,981	6,181.4	0.6
Pneumococcal disease (invasive)	11	204	24	157	43	15	97	46	597	405	534	538.2	1,194	1,616.8	0.7
Poliomyelitis	0	0	0	0	0	0	1	0	1	0	0	0.0	1	0.0	0.0
Rubella	0	0	0	4	1	0	1	0	6	15	25	20.4	30	66.6	0.5
Rubella - congenital	0	0	0	0	0	0	0	0	0	1	0	0.4	1	1.2	0.8
Tetanus	0	1	0	0	0	1	0	0	2	0	0	0.6	2	2.6	0.8
Varicella infections (chickenpox)	NDP	NN	88	105	295	5	NN	82	575	432	466	NA	1,366	NA	NA
Varicella infections (unspecified)	NDP	NN	23	68	179	11	NN	76	357	493	322	NA	1,312	NA	NA
Varicella zoster infections	NDP	NN	1	843	168	6	NN	154	1,172	998	931	NA	3,249	NA	NA
Vectorborne diseases															
Barmah Forest virus infection	1	87	14	167	25	0	3	16	313	578	356	507.2	1,331	1,116.0	1.2
Dengue	2	17	4	19	13	1	3	13	72	89	39	95.0	254	287.8	0.9
Flavivirus infection (NEC)	0	0	0	2	0	0	1	0	3	6	4	12.2	19	42.6	0.4
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	0	0	0	0	0	0	0	0	1.4	0	6.2	0.0
Malaria	4	26	2	39	8	2	21	12	114	168	202	157.6	435	504.2	0.9
Murray Valley encephalitis virus	0	0	0	0	0	0	0	0	0	0	0	0.2	0	1.2	0.0
Ross River virus infection	2	149	44	306	56	0	6	59	622	1,325	391	1,196.8	3,051	3,036.6	1.0

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2007, by date of onset,* *continued*

Disease	State or territory								Total 3rd quarter 2007†	Total 2nd quarter 2007	Total 3rd quarter 2006	Last 5 years mean 3rd quarter	Year to date 2007	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	0	0	0.0	1	0.2	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	5	1	1	1	0	8	4	15	6.4	26	25.2	1.0
Leptospirosis	0	1	0	7	0	0	2	0	10	30	20	44.2	87	124.0	0.7
Lyssavirus unspecified	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	1	0	1	0	0	7	1	10	26	46	52.2	64	154.2	0.4
Q fever	2	43	0	43	7	0	8	1	104	124	119	130.6	344	389.8	0.9
Other bacterial infections															
Legionellosis	0	13	0	9	1	0	9	10	42	90	82	85.2	207	240.6	0.9
Leprosy	0	0	0	0	0	0	0	0	0	4	2	1.6	9	6.2	1.5
Meningococcal infection**	1	43	1	32	12	3	26	8	126	62	111	107.0	234	361.0	0.6
Tuberculosis	7	90	4	36	11	0	109	14	271	232	326	257.4	774	801.2	1.0
Total	886	9,421	1,574	11,886	3,841	991	8,111	4,939	41,649	36,444	35,775	28,316.5	115,929	88,540.4	1.4

* 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. Note: Ratios for syphilis <2 years; syphilis >2 years or unspecified duration based on 2 years data

§ 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 3. Notification rates of diseases, 1 July to 30 September 2007, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	3.6	1.1	9.6	1.5	0.0	1.6	1.5	2.5	1.4
Hepatitis B (unspecified)	25.2	49.3	111.8	26.7	38.7	3.2	33.5	31.3	37.5
Hepatitis C (incident)	1.2	0.5	0.0	NN	3.8	1.6	2.3	3.3	1.8
Hepatitis C (unspecified)	45.6	92.1	111.8	65.7	29.9	45.6	50.3	50.3	66.1
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.2	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†	125.9	NN	188.8	96.8	301.3	137.7	108.2	86.5	126.2
Cryptosporidiosis	2.4	4.9	69.4	8.3	34.7	5.6	11.5	32.4	12.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Hepatitis A	0.0	0.7	3.9	0.9	0.3	0.8	0.4	1.9	0.8
Hepatitis E	1.2	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.1
Listeriosis	0.0	0.3	0.0	0.0	0.0	0.8	0.2	0.0	0.2
Salmonellosis (NEC)	38.4	35.7	233.2	64.2	86.6	37.6	36.4	41.8	48.0
Shigellosis	0.0	1.1	75.2	1.0	6.0	1.6	2.1	5.2	2.8
SLTEC, VTEC‡	0.0	0.1	0.0	0.4	2.5	0.0	0.2	0.0	0.3
Typhoid	0.0	0.7	0.0	0.1	0.0	0.0	0.4	0.0	0.3
Quarantinable diseases									
Cholera	0.0	0.1	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§	268.6	169.9	1,360.5	305.2	237.3	229.7	220.5	348.0	246.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	10.8	20.8	1,040.6	33.6	46.2	7.2	18.1	80.9	40.1
Syphilis (all)	0.0	0.0	0.0	0.0	5.0	0.0	0.0	0.0	0.4
Syphilis <2 years duration	0.0	6.2	125.3	6.2	0.3	3.2	7.1	5.9	6.9
Syphilis >2 years or unspecified duration	7.2	10.0	67.4	4.8	0.0	4.8	8.3	5.6	7.8
Syphilis - congenital	0.0	0.1	3.9	0.0	0.0	0.0	0.0	0.0	0.1
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	0.1	0.0	0.0	0.0	0.4	0.1
Influenza (laboratory confirmed)	8.4	9.6	13.5	20.6	5.0	16.8	2.9	19.9	11.0
Measles	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Mumps	0.0	3.3	23.1	1.0	2.5	0.0	0.6	0.2	1.9
Pertussis	33.6	25.8	13.5	33.4	48.5	6.4	22.9	6.3	25.9
Pneumococcal disease (invasive)	10.8	7.4	32.8	7.1	16.1	2.4	5.7	6.9	7.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3. Notification rates of diseases, 1 July to 30 September 2007, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, continued									
Rubella	1.2	0.2	0.0	0.4	0.5	0.0	0.2	0.2	0.3
Rubella - congenital	0.0	0.1	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
Varicella infections (chickenpox)	NDP	NN	40.5	4.5	78.6	0.8	NN	9.8	NA
Varicella infections (unspecified)	NDP	NN	30.8	8.0	75.3	16.8	NN	14.2	NA
Varicella zoster infections	NDP	NN	3.9	67.5	34.1	4.0	NN	30.5	NA
Vectorborne diseases									
Barmah Forest virus infection	3.6	13.5	61.7	24.0	8.3	0.0	0.7	3.5	11.0
Dengue	2.4	0.9	5.8	4.5	2.0	0.0	0.2	2.3	1.7
Flavivirus infection (NEC)	0.0	0.0	0.0	0.5	0.0	0.0	0.1	0.0	0.1
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	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.2	1.1	17.3	4.9	4.5	1.6	3.0	5.6	3.2
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	4.8	16.3	129.1	70.2	25.1	1.6	1.8	23.2	25.2
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	0.3	0.0	0.0	0.0	0.2	0.1
Leptospirosis	0.0	0.1	0.0	2.3	0.3	0.0	0.2	0.4	0.6
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.6	0.0	0.0	0.0	0.0	1.2	0.0	0.5
Q fever	0.0	2.9	1.9	3.5	5.8	0.0	0.9	0.4	2.4
Other bacterial infections									
Legionellosis	2.4	1.7	3.9	1.4	3.5	0.8	1.2	2.3	1.7
Leprosy	0.0	0.0	0.0	0.1	0.5	0.0	0.0	0.2	0.1
Meningococcal infection‖	0.0	1.3	3.9	0.7	1.3	0.8	1.8	0.4	1.2
Tuberculosis	4.8	5.2	13.5	3.1	4.3	1.6	5.2	2.5	4.4

* 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.

Laboratory Virology and Serology Reporting Scheme

There were 10,198 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2007 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2007, and total reports for the year†

	State or territory								This period 2007	This period 2006	Year to date 2007	Year to date 2006
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	1	–	4	1	–	–	–	6	2	18	54
Mumps virus	–	–	–	2	14	–	3	–	19	2	41	25
Rubella virus	–	–	–	1	–	–	–	–	1	5	15	13
Hepatitis viruses									0			
Hepatitis A virus	–	1	–	5	1	–	1	–	8	8	31	24
Hepatitis D virus	–	–	–	–	4	–	–	–	4	1	20	5
Arboviruses												
Ross River virus	–	4	1	120	38	–	2	–	165	44	888	1,021
Barmah Forest virus	–	2	1	93	16	–	–	–	112	36	409	265
Flavivirus (unspecified)	–	–	–	25	–	–	–	–	25	4	81	43
Adenoviruses												
Adenovirus not typed/ pending	1	90	1	139	150	–	8	–	389	229	772	489
Herpes viruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1		2	2
Cytomegalovirus	1	52	–	115	116	3	8	–	295	217	888	735
Varicella-zoster virus	5	98	1	518	145	2	5	–	774	287	2,088	897
Epstein-Barr virus	–	16	–	360	197	2	10	–	585	365	1,962	1,184
Other DNA viruses												
Parvovirus	–	1	–	103	5	1	12	–	122	61	287	149
Picornavirus family									0			
Rhinovirus (all types)	1	44	–	–	8	–	–	–	53	100	214	142
Enterovirus type 69	–	–	–	–	–	–	1	–	1		1	
Enterovirus not typed/ pending	–	13	–	6	8	3	–	–	30	18	107	94
Picornavirus not typed	–	–	–	–	–	3	–	–	3	1	4	2
Ortho/paramyxoviruses												
Influenza A virus	1	299	15	1,106	440	24	82	7	1,974	229	2,098	301
Influenza B virus	–	10	–	11	54	–	19	–	94	126	109	170
Influenza virus - typing pending	–	1	–	–	–	–	–	–	1		1	
Parainfluenza virus type 1	–	7	–	3	7	–	–	–	17	16	28	74
Parainfluenza virus type 2	–	8	–	1	7	–	–	–	16	5	57	12
Parainfluenza virus type 3	–	78	2	78	56	1	6	–	221	89	318	114
Respiratory syncytial virus	2	324	–	262	360	42	41	–	1,031	1,187	1,833	1,748
Other RNA viruses												
HTLV-1	–	–	–	–	3	–	–	–	3		12	4
Rotavirus	–	70	–	–	135	–	3	–	208	738	315	870
Norwalk agent	–	11	–	–	–	–	381	–	392	429	614	1,110

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2007, and total reports for the year,† *continued*

	State or territory								This period 2007	This period 2006	Year to date 2007	Year to date 2006
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other pathogens												
Chlamydia trachomatis not typed	4	226	–	1,301	513	10	10	1	2,065	932	6,274	3,404
Chlamydia pneumoniae	–	–	–	–	–	–	1	–	1	1	1	1
Chlamydia psittaci	–	–	–	–	–	–	3	–	3	17	39	43
Chlamydia species	–	–	–	–	–	–	1	–	1	1	2	2
Mycoplasma pneumoniae	–	9	3	221	67	8	58	–	366	294	988	901
Mycoplasma hominis	–	1	–	–	–	–	–	–	1	10	5	20
Coxiella burnetii (Q fever)	1	1	–	26	16	–	7	–	51	23	143	94
Orientia tsutsugamushi	–	–	–	–	1	–	–	–	1	2	7	23
Rickettsia - spotted fever group	–	–	–	–	5	–	–	–	5	19	66	85
Streptococcus group A	–	6	53	230	–	–	39	–	328	65	823	329
Yersinia enterocolitica	–	2	–	2	–	–	–	–	4	1	7	5
Brucella abortus	–	–	–	–	–	–	1	–	1	–	2	–
Brucella species	–	–	–	4	–	–	–	–	4	2	7	5
Bordetella pertussis	–	5	–	145	98	1	13	1	263	552	659	1,223
Legionella pneumophila	–	2	–	–	–	–	5	–	7	6	28	25
Legionella species	–	–	–	–	–	–	1	–	1	–	3	–
Cryptococcus species	–	–	–	7	14	–	–	–	21	3	41	17
Leptospira species	–	1	–	11	2	–	–	–	14	5	52	16
Treponema pallidum	–	29	8	270	183	–	11	–	501	191	1,767	686
Entamoeba histolytica	–	–	–	1	–	–	–	–	1	1	6	1
Toxoplasma gondii	–	1	–	–	3	1	1	–	6	3	21	36
Echinococcus granulosus	–	–	–	–	2	–	–	–	2	–	16	3
Total	16	1,413	85	5,170	2,669	101	734	9	10,198	6,327	24,170	16,466

* 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 July to 30 September 2007*

State or territory	Laboratory	July 2007	August 2007	September 2007	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	115	137	57	309
	New Children's Hospital, Westmead	212	190	101	503
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	28	39	17	84
	South West Area Pathology Service, Liverpool	161	80	–	241
Queensland	Queensland Medical Laboratory, West End	1,604	2,373	1,615	5,592
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	841	1,112	709	2,662
Tasmania	Northern Tasmanian Pathology Service, Launceston	23	50	24	97
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	40	39	13	92
	Royal Children's Hospital, Melbourne	21	31	18	70
	Victorian Infectious Diseases Reference Laboratory, Fairfield	85	264	199	548
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	–	–	–	–
Total		3,130	4,315	2,753	10,198

* The complete list of laboratories reporting for the 12 months, January to December 2007, 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 Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. The aim of ASPREN is to also provide an indicator of the burden of disease in the primary health care setting and to detect trends in consultation rates.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2007, four conditions are being monitored all of which are related to communicable diseases. They include influenza like illness (ILI), gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in Commun Dis Intell 2007;31:158.

Reporting period 1 July to 30 September 2007

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory and Tasmania. A total of 98 general practitioners contributed data to ASPREN in the third quarter of 2007. Each week an average of 74 general practitioners provided information to ASPREN at an average of 8,389 (range 7,354 to 9,356) consultations per week.

From July to the end of August 2007, influenza-like illness (ILI) rates were high (30 to 47 cases per 1,000 consultations) compared with the same reporting period in 2006 (16 to 32 cases per 1,000 consultations) (Figure 1). ILI rates peaked to 47 cases per 1,000 consultations at the end of July and began to decrease from mid-September (14 to 19 cases per 1,000 consultations) compared with 20 to 28 cases per 1,000 consultations for the same period in 2006.

Reports of gastroenteritis from 1 July to 30 September 2007 were lower compared to the same period in 2006 (Figure 2). During this reporting period, consultation rates for gastroenteritis remained constant (between 5 to 9 cases per 1,000 consultations).

Reports of varicella infections were reported at a lower rate for the third quarter of 2007 compared with the same period in 2006, but there was no recognisable seasonal pattern. From 1 July to 30 September 2007, rates for chickenpox fluctuated between 0.4 to 1 case per 1,000 consultations (Figure 3).

In the third quarter of 2007, rates for shingles fluctuated between less than 1 to 1.3 cases per 1,000 consultations (Figure 4).

Figure 1. Consultation rates for influenza like illness, ASPREN, 2006 to 30 September 2007, by week of report

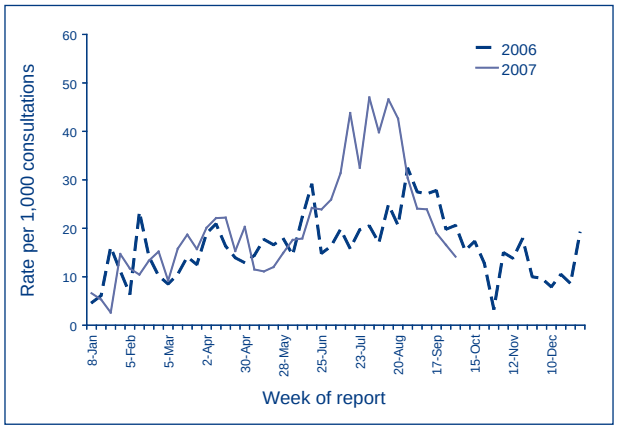


Figure 2. Consultation rates for gastroenteritis, ASPREN, 2006 to 30 September 2007, by week of report

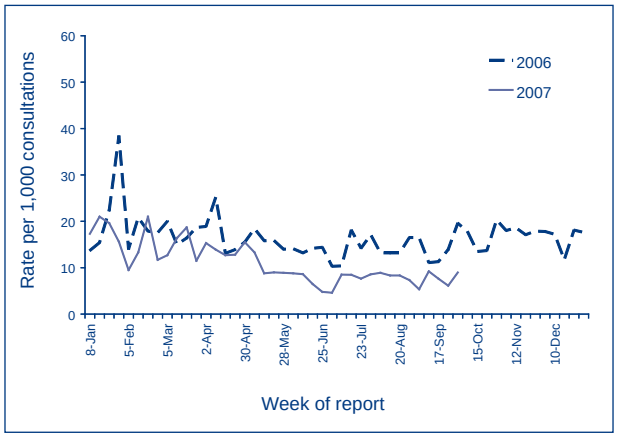


Figure 3. Consultation rates for chickenpox, ASPREN, 2006 to 30 September 2007, by week of report

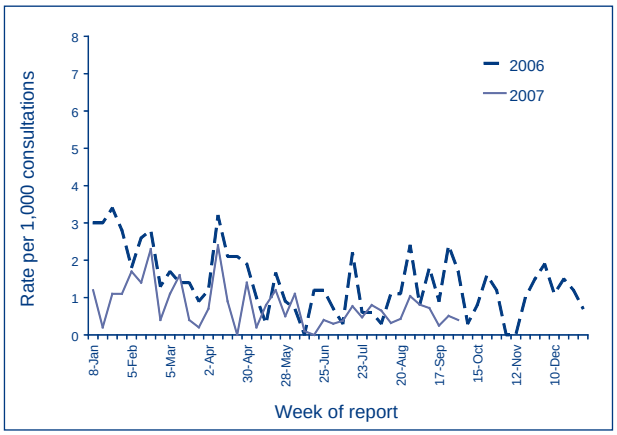
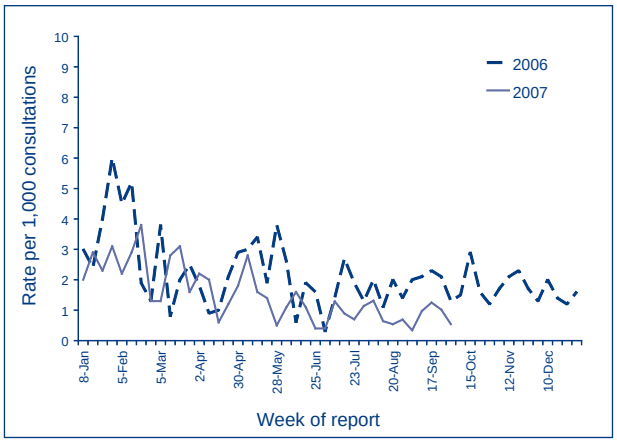


Figure 4. Consultation rates for shingles, ASPREN, 2006 to 30 September 2007, by week of report



Childhood immunisation coverage

Tables 1, 2 and 3 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 April and 30 June 2006, at 24 months of age for the cohort born between 1 April and 30 June 2005, and at 6 years of age for the cohort born between 1 April and 30 June 2001 according to the National Immunisation Program.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2007;31:163–164 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 0.1 percentage points to 91.3% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia remained at 92.5%, identical to the previous quarter (Table 2). There were no significant changes in any jurisdiction or in coverage for individual vaccines. However, it is important to note that, for the two vaccines where no further doses are due between 6 months and 24 months (diphtheria-tetanus-pertussis and polio), coverage at the national level was 95.0% and 94.9%, respectively at 24 months versus 91.9% at 12 months. This suggests that delayed notification or delayed vaccination is substantially decreasing coverage estimates at 12 months of age.

Immunisation coverage for children 'fully immunised' at 6 years of age for Australia increased from the last quarter by 0.7 percentage points to 88.6% to reach its highest recorded level (Table 3). Coverage for all three individual vaccines measured at 6 years of age increased by 0.5–0.6 percentage points and for each of them is now greater than 89% for the first time. Significant increases in coverage in the Northern Territory and South Australia appear to be the main driver of the increases nationally.

Figure 5 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 few years for all age groups. It should be noted that currently, coverage for the vaccines added to the National Immunisation Program since 2003 (varicella at 18 months, 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.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2006; assessment date 30 September 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,115	22,747	951	14,371	4,518	1,380	16,428	6,996	68,506
Diphtheria, tetanus, pertussis (%)	94.5	92.0	90.8	91.8	91.8	92.0	92.6	90.2	91.9
Poliomyelitis (%)	94.6	92.0	90.8	91.7	91.8	92.0	92.5	90.1	91.9
Haemophilus influenzae type b (%)	96.0	95.0	94.6	93.8	94.4	94.9	94.6	93.8	94.5
Hepatitis B (%)	95.9	94.9	95.3	93.6	94.3	94.6	94.6	93.7	94.4
Fully immunised (%)	94.4	91.7	90.6	90.9	91.2	91.7	91.5	89.6	91.3
Change in fully immunised since last quarter (%)	+0.1	+0.2	-0.5	-0.0	+0.7	+0.3	-0.3	+0.7	+0.1

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2005; assessment date 30 September 2007*

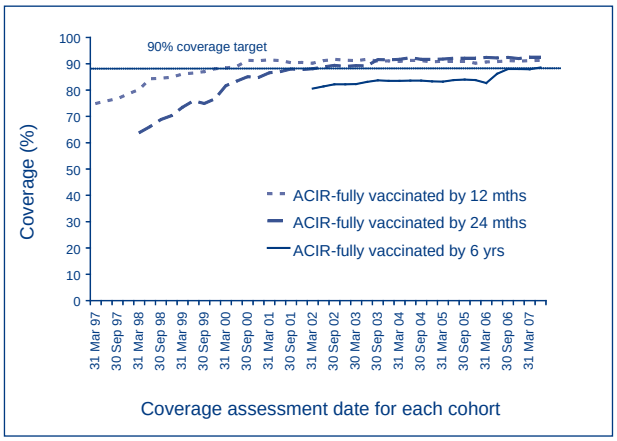
Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,034	22,762	934	14,745	4,498	1,501	16,369	6,893	68,736
Diphtheria, tetanus, pertussis (%)	95.8	95.1	96.0	94.5	94.9	96.5	95.7	93.7	95.0
Poliomyelitis (%)	95.7	95.0	96.2	94.4	94.9	96.4	95.6	93.7	94.9
Haemophilus influenzae type b (%)	95.8	94.9	95.0	93.6	93.7	96.2	94.5	93.2	94.3
Measles, mumps, rubella (%)	95.5	93.7	95.9	93.5	94.1	95.8	94.6	92.5	93.9
Hepatitis B (%)	96.1	95.8	97.1	95.6	95.7	97.0	96.2	94.6	95.8
Fully immunised (%)	93.9	92.3	93.8	91.9	92.6	94.9	93.5	90.5	92.5
Change in fully immunised since last quarter (%)	+2.0	+0.0	+1.3	-0.3	+1.1	-0.2	-0.3	-0.1	-0.1

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2006;30:488.

Table 3. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2001; assessment date 30 September 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	992	21,705	928	14,180	4,484	1,441	15,461	6,613	65,804
Diphtheria, tetanus, pertussis (%)	90.2	89.0	88.0	89.1	88.3	90.7	91.7	85.5	89.3
Poliomyelitis (%)	90.2	88.8	87.6	89.2	88.4	90.8	91.9	85.6	89.3
Measles, mumps, rubella (%)	90.0	88.9	87.9	89.2	88.1	90.8	91.7	85.6	89.3
Fully immunised (%)	89.1	88.2	87.3	88.5	87.7	90.3	91.1	84.7	88.6
Change in fully immunised since last quarter (%)	-0.3	+0.5	+2.5	+0.7	+2.0	+0.6	+0.5	+0.5	+0.7

Figure 5. Trends in vaccination coverage, Australia, 1997 to 30 June 2007, by age cohorts



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 *2007;31:162.*

Reporting period 1 April to 30 June 2007

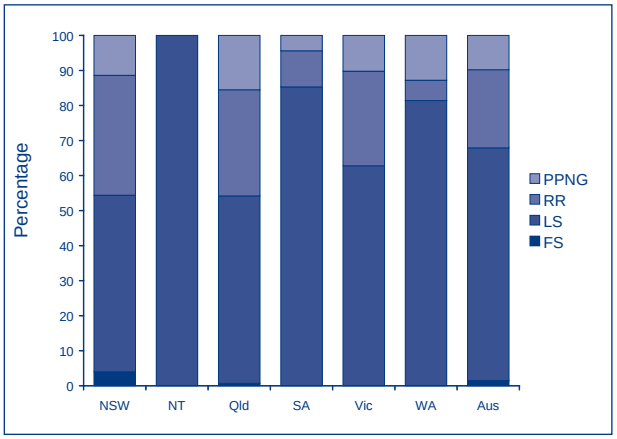
The AGSP laboratories received a total of 823 isolates in this quarter of which 806 underwent susceptibility testing. About 30% of this total was from New South Wales, 18% each from Victoria and Queensland, 14% from the Northern Territory, 11% from Western Australia and 8% from South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter, 259 (32.1%) of all isolates examined were penicillin resistant by one or more mechanisms. Seventy-nine (9.8%) were penicillinase-producing *Neisseria gonorrhoeae* (PPNG) and 180 (22.3%) resistant by chromosomal mechanisms, (CMRP). These proportions are little different from those recorded in this quarter in 2006. The proportion of all strains resistant to the penicillins by any mechanism ranged from nil in the Northern Territory to 45% in New South Wales and Queensland. High rates of penicillin resistance were also found in Victoria (37%), Western Australia (18.6%) and South Australia 14.7%).

Figure 6 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 PPNG aggregated for Australia and by state and territory. A high proportion of those strains classified as PPNG or CMRP fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 6. Categorisation of gonococci isolated in Australia, 1 April to 30 June 2007, 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.*

In New South Wales and Victoria most of the penicillin resistance was due to CMRP. In New South Wales 84 (34%) were CMRP with 28 PPNG (11.4%) and in Victoria 40 (27%) were CMRP and 15 (10%) PPNG. In Queensland 43 CMRP comprised 30.3% of isolates and 22 PPNG comprised 15.5% of isolates. In Western Australia PPNG were more prominent (12.8%, 11 isolates) with 5.8% CMRP. Of 10 resistant strains in South Australia, seven were CMRP and

three were PPNG. One CMRP was reported from Tasmania but there were no PPNG. There were no penicillin resistant gonococci in the Northern Territory or the Australian Capital Territory.

Ceftriaxone

Eleven isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected: six in New South Wales, three in Victoria and one each in Queensland and South Australia.

Spectinomycin

All isolates were susceptible to this injectable agent.

Quinolone antibiotics

A total of 359 quinolone resistant *N. gonorrhoeae* (QRNG) was present in this quarter and represented 44.5% of all gonococci tested, compared with 33.7% in this quarter in 2006. In 2005, 30% of all gonococci were QRNG. The majority of QRNG in the current period (348, 97%) exhibited higher-level resistance (ciprofloxacin MICs 1 mg/L or more). 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 detected in all jurisdictions except Tasmania, the Northern Territory and the Australian Capital Territory (Figure 7). The highest number (152) and proportion (62%) of QRNG were found in New South Wales. QRNG were also prominent in Victoria where 80 QRNG represented 54% of isolates, Queensland 74 QRNG (52%), South Australia 30 QRNG (44%) and Western Australia 22 QRNG (25.6%).

High level tetracycline resistance

The number (121) of high level tetracycline resistance (TRNG) detected approximated that found in this quarter in 2006 (117) and represented 15% of all isolates. The highest proportion of TRNG in any jurisdiction (38%) was in Western Australia and the highest number (42) was in New South Wales. TRNG were present in all states except Tasmania. No TRNG were found in the Northern Territory or the Australian Capital Territory.

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Figure 7. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae*, Australia, 1 April to 30 June 2007, by state or territory

LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

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 *2007;31:162.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2007, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2007, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD
Australian Capital Territory	07			1	3						1			1	4
	06			1	1	0	1	0		0		0		1	2
New South Wales	07			35	52	1	7	2	4	0	1	3	7	41	70
	06			24	46	9	13	0	1	1	3	2	5	36	68
Northern Territory	07			0	1	0	1							0	2
	06			1	3									1	3
Queensland	07			24	43	4	5	1	1	2	2		1	31	52
	06			20	45	0	4			1	1			21	52
South Australia	07			5	9	1	1					1	1	7	11
	06			3	9			0	1	1	1			4	11
Tasmania	07			2	2			1	1		1			3	5
	06			0	3	0	1							0	4
Victoria	07			14	35	0	2	1	4	1	2	3	4	19	47
	06			18	47	1	3	0	1	3	5	1	1	23	57
Western Australia	07			8	15									8	15
	06			6	15					1	1			7	16
Total	07			89	160	6	16	5	10	3	6	7	13	110	205
	06			73	169	10	22	0	3	7	10	3	6	93	210

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 6 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 July to 30 September 2007 are included in Tables 7 and 8. Data include cases reported and entered by 19 October 2007. 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 *2007;31:163–164.*

Reporting period 1 July to 30 September 2007

There were 1,284 reports to NEPSS of human *Salmonella* infection in the third quarter of 2007. The annual cycle of *Salmonella* incidence typically reaches a nadir in the third quarter. Although this count represents a marked decline in the incidence of salmonellosis from the first and second quarters this year (when a total of 5,749 reports were received) it still represents the highest count in the

third quarter for more than 15 years, and is approximately 20% greater than the 10-year historical mean for this quarter.

During the third quarter of 2007, the 25 most common *Salmonella* types in Australia accounted for 721 cases, 56% of all reported human *Salmonella* infections. Fifteen of the 25 most common *Salmonella* infections in the third quarter of 2007 were also amongst those most commonly reported in the preceding quarter.

The most notable feature of the current data is a large outbreak of *S.* Typhimurium in Western Australia. Some isolates from this outbreak have been characterised as phage type 12, the remainder have not been phage typed.

Other increases above the historical average for the period include *S.* Infantis (South Australia and New South Wales), *S.* Typhimurium phage type U290 (New South Wales), *S.* Virchow phage type 45 (Western Australia) and *S.* Typhimurium phage type 44 (Victoria and Queensland). More modest increases include *S.* Typhimurium phage type 22 and *S.* Anatum (both mostly in Queensland), *S.* Typhimurium phage type 193 (South Australia, New South Wales and Victoria), and *S.* Newport and *S.* Typhimurium phage type U302 (both mostly 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 7. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2007, as reported to 19 October 2007

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	20	289	63	254	117	20	288	233	1,284
Total contributing *Salmonella* types	16	105	39	97	57	13	103	39	218

Table 8. Top 25 *Salmonella* types identified in Australia, 1 July to 30 September 2007, by state or territory

National rank	*Salmonella* type	State or territory								Total 3rd quarter 2007	Last 10 years mean 3rd quarter	Year to date 2007	Year to date 2006
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium (not phage typed)	0	0	0	0	0	0	0	128	128	0.5	131	0
2	*S.* Typhimurium PT 135	1	21	1	19	2	3	29	0	76	95	520	546
3	*S.* Typhimurium PT 9	0	15	1	4	6	1	24	0	51	69	610	273
4	*S.* Infantis	1	15	4	4	12	0	5	3	44	24	141	146
5	*S.* Saintpaul	0	10	3	20	0	0	6	4	43	48	270	324
6	*S.* Typhimurium PT 44	0	6	0	9	1	0	17	2	35	9	326	125
7	*S.* Stanley	1	8	0	5	1	0	13	4	32	19	99	73
8	*S.* Typhimurium PT 170	0	10	0	4	0	2	10	0	26	26	222	274
9	*S.* Virchow PT 8	1	3	1	16	1	0	0	0	22	27	178	217
10	*S.* Typhimurium PT 197	0	6	0	9	1	1	5	0	22	17	159	84
11	*S.* Enteritidis (not phage typed)	0	0	0	0	0	0	2	20	22	0	23	0
12	*S.* Typhimurium PT 193	0	5	1	2	8	0	5	0	21	1.8	38	10
13	*S.* Typhimurium PT U290	1	15	0	1	0	0	3	0	20	9	46	25
14	*S.* Typhimurium RDNC	1	12	0	3	1	0	2	0	19	18	94	85
15	*S.* Birkenhead	0	10	0	5	0	0	3	0	18	23	162	219
16	*S.* Anatum	0	3	2	7	0	0	2	3	17	12	58	89
17	*S.* Muenchen	2	2	2	3	1	0	2	4	16	17	104	122
18	*S.* Chester	0	0	2	6	0	0	5	2	15	22	126	119
19	*S.* Enteritidis PT 6a	0	3	0	4	0	0	7	0	14	10	54	37
20	*S.* Singapore	0	9	0	1	0	0	3	1	14	8	54	38
21	*S.* Virchow PT 45	0	1	0	1	0	0	0	12	14	0.1	16	4
22	*S.* Aberdeen	0	1	0	12	0	0	0	0	13	11	104	124
23	*S.* Typhimurium untypable	2	6	0	1	1	0	3	0	13	10	70	55
24	*S.* Newport	0	0	0	1	2	2	8	0	13	9	48	39
25	*S.* Montevideo	0	7	1	3	0	0	1	1	13	3.7	96	30

OVERSEAS BRIEF

Reporting period 1 July to 30 September 2007

The Overseas brief highlights disease outbreaks during the quarter that were of major public health significance world-wide or those that may have important implications for Australia.

Chikungunya

Between 1 July and 21 September 2007, the World Health Organization (WHO) reported 292 suspected cases of chikungunya (125 of them laboratory-confirmed) in the Ravenna region in north-eastern Italy.[1] This is the first ever recorded local vectorborne transmission of chikungunya in Europe. Cases of returned travellers with chikungunya viraemia have previously been reported in Europe, usually during the European winter, when seasonal outbreaks of chikungunya in the Southern Hemisphere are at their peak, reducing the risk of local transmission from these imported cases.

The probable index case returned from travel to Kerala State in India in early June 2007, with onset of symptoms on 15 June 2007. The outbreak peaked in the third week of August 2007.[2] The majority of cases were reported from the villages of Castiglione di Cervia and Castiglione di Ravenna, at first involving cases with epidemiological links to the index case. However, after the end of August 2007, cases were reported that had no epidemiological links to the first cases, or exposure in the villages of Castiglione di Cervia and Castiglione di Ravenna,[1] suggesting that indigenous transmission was likely to have occurred in five separate localities all in north-eastern Italy.[1,2]

The two villages of Castiglione di Cervia and Castiglione di Ravenna are known to have established populations of the tiger mosquito, *Aedes albopictus,* which is a competent vector for chikungunya.[1] This vector is present in a number of areas of Europe including some areas of southern France, Spain, the Netherlands and some areas around the Adriatic seas.[3] From 18 August 2007, vector control measures were implemented in the two villages, including the removal of breeding sites and the use of insecticides.[1] There is a possibility that transmission of the virus may resume in 2008 when mosquito eggs laid in the 2007 season hatch in the northern spring and summer (research has shown that these larvae could be infected with the chikungunya virus).[1]

Cholera

Between 14 August and 7 October 2007, the WHO reported 3,857 laboratory-confirmed cases (case-fatality rate, [CFR] 0.5%) of *Vibrio cholerae* in Iraq. The outbreak was first reported from Kirkuk Province in northern Iraq, and subsequently from 9 of 18 provinces (mostly northern and some central provinces) in the country, including cases in the capital, Bagdad. The WHO estimates that 30,000 people became ill with acute watery diarrhoea during the outbreak. The number of new confirmed cholera cases peaked between 2 and 9 September 2007, but the number of new cases of diarrhoea continued to climb until the end of September 2007.[4]

The outbreak presented a significant risk to neighbouring countries and a cholera outbreak in neighbouring Iran (with 43 cases between 19 September and 6 October 2007, most in the western Kurdistan province, bordering Iraq[5]) was thought to have been related to movement of people or goods across the border from Iraq.[6] It is not clear whether the infection was spread by Iraqi refugees or local Iranians.[6] The WHO did not recommend restrictions to travel or trade between Iraq and neighbouring countries, but recommended the strengthening of surveillance and response systems.[7]

Dengue fever

South East Asia

Outbreaks of dengue were reported across South East Asia during the reporting period, coinciding with the rainy season that occurs between June and August. A seasonal rise in incidence is to be expected during the rainy season, but there were indications that the 2007 outbreaks could be more severe than usual. The Western Pacific Regional Office of the WHO warned that South East Asia was heading for a major dengue outbreak following an early start to the dengue season.[8]

The extent and range of dengue fever worldwide has expanded markedly over the last 30 years. Factors leading to the expansion are thought to be rapid urbanisation (water supplies are inadequate leading people to store water in open containers where mosquitoes can breed), increased population mobility (leading to increased transmission in new areas) and population explosion (putting a strain on health care services). Information on the extent of dengue fever in South East Asia is unreliable, with many cases and outbreaks not reported. The WHO has called for improved surveillance and reporting of dengue fever to enable better planning of control efforts.

Latin America and the Caribbean

The Pan American Health Organization (PAHO) reported 643,123 cases of dengue fever across Latin America and the Caribbean between 1 January and 13 October 2007, 13,087 of which have developed into dengue haemorrhagic fever (including 183 deaths). The PAHO reported that, with the current wet conditions caused by the La Niña climatic condition, the total number of cases across the region this year could exceed one million.

Central and Andean America (which includes the countries of Belize, Costa Rica, El Salvador, Guatemala, Honduras, Mexico, Nicaragua, Panama, Bolivia, Colombia, Ecuador, Peru and Venezuela) has been particularly badly affected, with a high proportion of haemorrhagic fever cases. While only 24% of the cases of classical dengue fever in 2007 between 1 January and 13 October 2007 were reported from Central and Andean America, 91.4% of the haemorrhagic fever cases were from this region.

Ebola haemorrhagic fever

Between 8 June and 2 October 2007, the WHO reported 76 suspected cases (25 of them laboratory confirmed) of Ebola haemorrhagic fever from the Democratic Republic of the Congo.[9] All of the confirmed cases were located in the Mweka and Luebo health zones in one small area of Kasai Occidental Province. Concurrent outbreaks of typhoid and *Shigella dysenteriae* type 1 were occurring in the same areas during this outbreak, which may have inflated suspected case numbers. The WHO had earlier reported suspected cases as 372 (including 166 deaths) as of 11 September 2007,[10] but this was later revised downwards following epidemiological investigations (see above).

On 29 August 2007, African media reported an outbreak of an unknown disease in the Mweka area of the Kasai Occidental Province of the Democratic Republic of the Congo.[11] The Ministry of Health confirmed an outbreak of Ebola haemorrhagic fever following laboratory confirmation from the Centers of Disease Control and Prevention (CDC), Atlanta and the Centre International de Recherches Médicales de Franceville. The first suspected cases became ill on 8 June 2007 following the funeral of a village chief, which all of the early cases attended. The most recent case was confirmed on 30 September 2007.

The Ministry of Health and members of WHO Global Outbreak Alert and Response Network and other networks worked in partnership during this outbreak to investigate cases; establish field isolation and testing units; mobilise resources; and improve local infrastructure and infection control procedures.[9,10] The effective response to this outbreak highlights the importance of international networks in supporting the control efforts of ministries of health during major disease outbreaks.

Influenza (avian)

Global update

The WHO confirmed nine human cases of H5N1 with dates of onset between 1 July and 30 September 2007.[12] Seven of the nine cases were fatal, (CFR 78%).[12] The WHO reported 16 cases including 12 deaths (CFR 75%) with dates of onset during the same period of 2006.[13]

Indonesia continues to report the most cases, with six of the nine cases during the quarter.[12] Vietnam reported two cases (one of them fatal) and Egypt reported one non-fatal case.[12]

The source of infection for four of these nine cases was established as exposure to sick and dead poultry, while the source for five others was not reported, but there was no evidence of human-to-human transmission in any of these cases.[12]

On 31 August 2007, the WHO introduced an External Quality Assessment Project for national reference laboratories for the detection of subtype influenza A viruses. The WHO has therefore amended the criteria for accepting confirmed cases of A (H5) infection. Based on the amended criteria, the Ministry of Health of Vietnam confirmed three additional cases of human infection with H5N1 avian influenza, including two deaths with dates of onset between 1 May and 30 June 2007.[14]

Marburg haemorrhagic fever

On 30 July 2007, the Ministry of Health in Uganda reported a fatal case of Marburg haemorrhagic fever (following laboratory confirmation by the CDC, Atlanta) in a 29-year-old man from the Kamwenge district.[15] The man had onset of symptoms on 4 July 2007 and died on 14 July 2007.[15] This fatal case had close contact with two earlier probable cases (non-fatal), one of whom became ill in early June and the other on 27 June 2007. Both subsequently tested positive for anti-Marburg virus IgG. These three cases were co-workers at a mine in western Uganda (that mine had recently been re-opened after 50 years of closure).

The Ministry of Health declared that the outbreak was contained on 9 August 2007,[16] but in late September 2007 a further suspected case (subsequently laboratory confirmed by the CDC, Atlanta) was reported in a man who had re-entered the mine (which was closed) where the earlier cases were thought to have contracted the infection.[17,18]

The reservoir of Marburg virus is unclear (possibly non-human primates or bats) and the mode of transmission to humans is not well described.[19,20] Three possible sources of the infection in the current outbreak are being investigated: a Colobus monkey that the probable index case slaughtered in the week prior to becoming ill (there is no laboratory evidence on whether the monkey was infected with the virus); a large bat colony resident in the mine where the men worked (with limited, if any, human contact before the mine was re-opened); and ticks (workers at the mine complained of tick bites).[21]

Between 1967 and 2007, a number of outbreaks of Marburg haemorrhagic fever have occurred in, or been linked to central and southern Africa (including Uganda). The most extensive outbreaks occurred in the Democratic Republic of the Congo (which borders Uganda) in 1998–99, with 103 cases (CFR 67%) and Angola in 2004–05 with 374 cases (CFR 88%).[22]

Poliomyelitis

Global update

Between 3 July and 10 October 2007, the Global Polio Eradication Initiative reported 288 cases of wild poliovirus infection from the endemic countries of Afghanistan (8), India (191), Nigeria (83) and Pakistan (6) and 29 cases of wild poliovirus from the re-infected countries of Angola (7) Chad (5), the Democratic Republic of the Congo (15), Myanmar (1) and Niger (1).[23]

Fewer cases of wild poliovirus have been reported between 1 January and 10 October 2007 than during the same period of 2006, with a 41% reduction in the number of cases reported from endemic countries and a 65% reduction in the number reported from re-infected countries.[23] Transmission of wild poliovirus has been interrupted in 10 of the 13 re-infected countries where it had been circulating in 2006.[24] Myanmar and Niger reported new outbreaks in 2007 following importations of the virus.

Nigeria

The WHO has now reported on a rare outbreak of vaccine-derived polio in Nigeria that occurred between 2006 and August 2007 with 69 children contracting the infection from others who had been immunised.[25,26] The weakened form of live poliovirus used in the oral polio vaccine (OPV) is thought to have mutated and been excreted, infecting the others through faecal-oral contact. Of the 69 children affected, 60 were either not vaccinated or insufficiently vaccinated.[26] Similar OPV-associated outbreaks have occurred in nine countries in the past 10 years, all in communities with low immunisation coverage, resulting in approximately 200 vaccine associated polio cases, while more than 6.5 million wild polio cases were prevented by the polio vaccine.[26]

West Nile virus infection

North America

Canada reported its worst ever West Nile virus (WNV) season in 2007. Between 1 January and 13 October 2007, the Public Health Agency of Canada reported 2,290 cases (including mild infections and asymptomatic cases), of which none were fatal, compared with 123 cases (none fatal) during the same period of 2006.[27] The presence of WNV in Canada was first confirmed in birds in 2001, with the first human cases reported from Quebec in 2002.[27,28]

The United States of America (USA) reported a similar WNV season in 2007 to the previous 3 years.[29] Between 1 January and 16 October 2007, the CDC reported 3,022 human cases of WNV infection including 76 deaths, compared with 3,498 cases including 108 deaths during the same period of 2006.[30] The first outbreaks of WNV in the USA were in New York in 1999, with widening spread across the country since then.

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Notice to readers

CHANGE OF CONTACT DETAILS

The Surveillance Branch of the Office of Health Protection has recently moved to 1 Bowes Place, WODEN ACT 2606. Due to the move it was not possible to retain the same phone and facsimile numbers. Please note the new contact details for *CDI* below.

Surveillance Branch
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Australian Government Department of Health and Ageing
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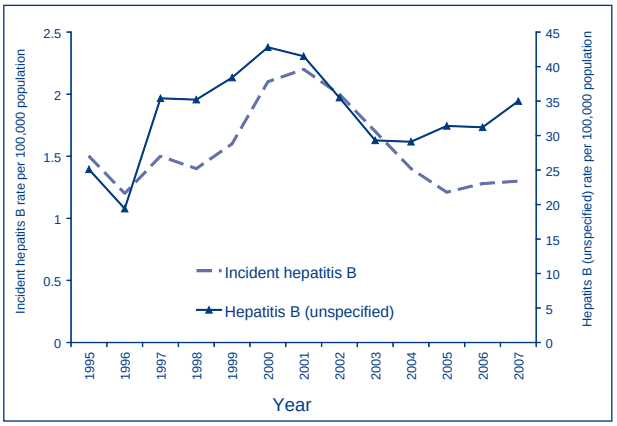
Facsimile: +61 2 6289 2600
Email: cdi.editor@health.gov.au

ERRATA

Communicable Diseases Surveillance Highlights

Figures 2 and 3 published in the Communicable Diseases Surveillance Highlights in the last issue of *CDI* (*Commun Dis Intell* 2007;31:320) were incorrect. The correct figures are published below. *CDI* apologises for this error.

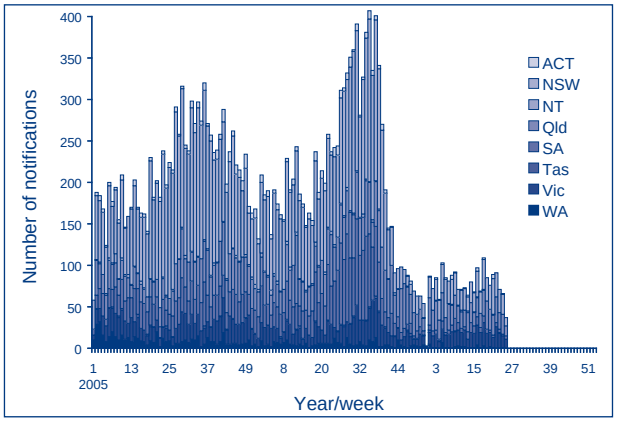
Figure 2. Notification rates of incident hepatitis B and hepatitis B (unspecified), Australia, 1995 to 2007* by year†



* Annualised rate to 30 June 2007.

† Year of diagnosis for incident hepatitis B; year of notification for unspecified hepatitis B.

Figure 3. Notifications of pertussis, Australia, 1 January 2005 to 30 June 2007, by week of onset



Australia's notifiable disease status, 2005

The report Australia's notifiable disease status, 2005: annual report of the National Notifiable Diseases Surveillance System published in March 2007 (*Commun Dis Intell* 2007;31:69) contained an error in the table at Appendix 3. Completeness of National Notifiable Diseases Surveillance System data received.

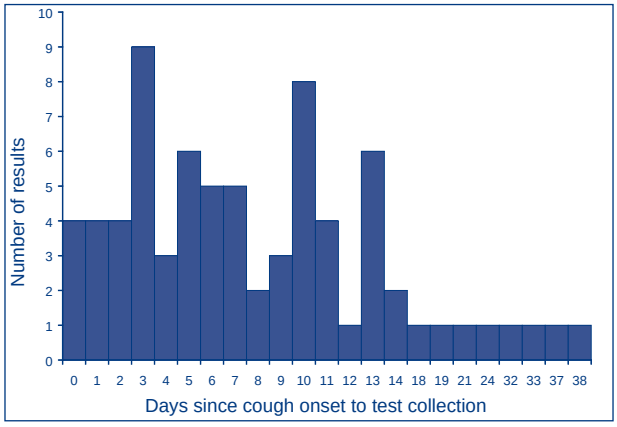
While Western Australia aim for 100% completeness of data on indigenous status, the correct figure for 2005 was actually 70%.

Bordetella pertussis PCR positivity, following onset of illness in children under 5 years of age

The article on *Bordetella pertussis* published in the March 2007 issue of *CDI* (Palmer CM, McCall B, Jarvinen K, Nissen MD. *Bordetella pertussis* PCR positivity, following onset of illness in children under 5 years of age. *Commun Dis Intell* 2007;31:202–205) contained an error.

There was an inconsistency in the figure heading and the X axis title for Figure 4. The corrected figure is reproduced below.

Figure 4. Polymerase chain reaction positive results by time (days) since cough onset (< 5 years age) n = 74



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CDI REVIEWERS, 2007

The *CDI* staff wish to thank the following reviewers for their valued assistance throughout the year.

Anthony Keil, Avner Misrachi, Brad McCall, Charles Watson, Chris Bunn, David Looke, David Lyle, David Smith, Frank Beard, Gillian Scott, Heather Gidding, Jeanette Pham, Jennifer Robson, Jenny Musto, John Bates, Kari Jarvinen, Kevin Doyle, Mark Ferson, Mark Veitch, Patrick Charles, Peter Markey, Peter McIntyre, Rosemary Lester, Scott Bowden, Sue Campbell-Lloyd, Thomas Riley, Vicki Krause, William Rawlinson.