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Annual reports

Australia's notifiable diseases status, 2006: Annual report of the National Notifiable Diseases Surveillance System

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Australian Meningococcal Surveillance Programme

Australian Sentinel Practice Research Network

Australian Quarantine Inspection Service

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Abstract

In 2006, 66 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 138,511 cases of communicable diseases to the National Notifiable Diseases Surveillance System: an increase of 10.4% on the number of notifications in 2005. In 2006, the most frequently notified diseases were sexually transmissible infections (57,941 notifications, 42% of total notifications), gastrointestinal diseases (27,931 notifications, 20% of total notifications) and vaccine preventable diseases (22,240 notifications, 16% of total notifications). There were 19,111 notifications of blood-borne diseases; 8,606 notifications of vectorborne diseases; 1,900 notifications of other bacterial infections; 767 notifications of zoonoses and 3 notifications of quarantinable diseases. *Commun Dis Intell* 2008;32:139–207.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status 2006, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, state and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

- identifying national trends;
- guidance for policy development and resource allocation at a national level;
- monitoring the need for and impact of national disease control programs;
- coordination of response to national or multi-jurisdictional outbreaks;
- description of the epidemiology of rare diseases, that occur infrequently at state and territory levels;

- meeting various international reporting requirements, such as providing disease statistics to the World Health Organization (WHO), and;
- support for quarantine activities, which are the responsibility of the national government.

Methods

Australia is a federation of 6 states (New South Wales, Queensland, South Australia, Tasmania, Victoria and Western Australia) and 2 territories (the Australian Capital Territory and the Northern Territory). State and territory health departments collect notifications of communicable diseases under their public health legislation. In 2006, the Australian Government Department of Health and Ageing (DoHA) did not have any legislated responsibility for public health apart from human quarantine. States and territories voluntarily forwarded data on a nationally agreed set of communicable diseases to DoHA for the purposes of national communicable disease surveillance.

Sixty-six communicable diseases (Table 1) agreed upon nationally through the Communicable Diseases Network Australia (CDNA) were reported to the National Notifiable Diseases Surveillance System (NNDSS). The system was complemented by other surveillance systems, which provided information on various diseases, including some that are not reported to NNDSS.

The national dataset included fields for unique record reference number; notifying state or territory; disease code; age; sex; indigenous status; postcode of residence; date of onset of the disease; death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case was collected. While not included in the national dataset, additional information concerning mortality and specific health risk factors for some diseases was obtained from states and territories.

Notification rates for each notifiable disease were calculated using 2006 mid-year resident population supplied by the Australian Bureau of Statistics (Appendixes 1 and 2). Where diseases were not notifiable in a state or territory, national rates were adjusted by excluding the population of that jurisdiction from the denominator. For some diseases, age adjusted rates were calculated using the indirect method of standardisation, with 2001 census data as the standard population.

The geographical distribution of selected diseases was mapped using ARCGIS software. Maps were based on the postcode of residence of each patient, aggregated to the appropriate Statistical Division (Map 1). Rates for the different Statistical Divisions were ordered into 6 groups — the highest value, the lowest value above zero, those equal to zero, and the intermediate values sorted into 3 equal-sized groups. The Statistical Divisions in the Australian Capital Territory were combined to calculate rates for the territory as a whole.

Information from communicable disease surveillance is disseminated through several avenues of communication. At the fortnightly teleconferences of the CDNA the most up-to-date information on topics of interest to the network is provided. The *Communicable Diseases Intelligence* (*CDI*) quarterly journal publishes surveillance data and reports of research studies on the epidemiology and control of various communicable diseases. Disease surveillance summaries from the NNDSS are published on the Communicable Diseases Surveillance section of DoHA's web site.

Notes on interpretation

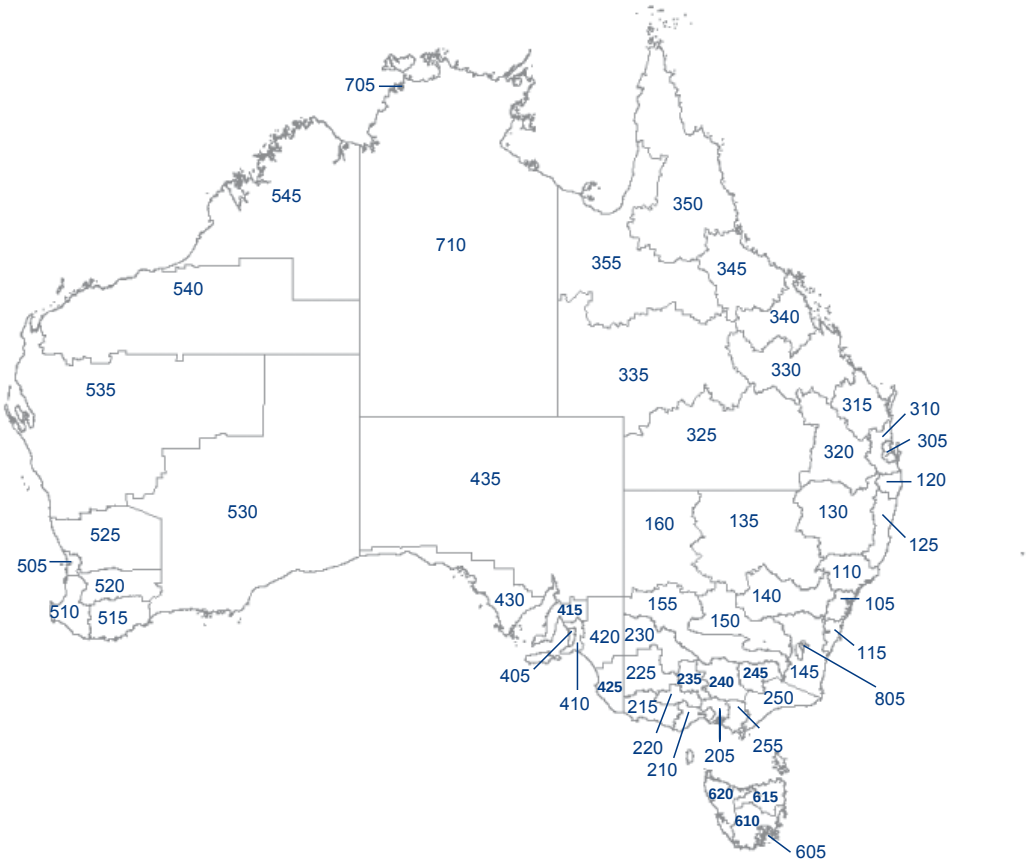
The present report is based on 2006 'finalised' data from each state and territory. States and territories transmitted data to NNDSS on average every other day, and the final dataset for the year was agreed upon in June 2007. The finalised annual dataset represents a snap shot of the year after duplicate records and incorrect or incomplete data have been removed. Therefore, totals in this report may vary slightly from the totals reported in *CDI* quarterly publications.

Analyses in this report were based on the date of disease onset in an attempt to estimate disease activity within the reporting period. Where the date of onset was not known however, the date of specimen collection or date of notification, whichever was earliest, was used. As considerable time may have lapsed between onset and diagnosis dates for hepatitis B (unspecified) and hepatitis C (unspecified), for these conditions the date of diagnosis, which is the earliest of specimen, notification or notification received dates supplied, was used.

Notified cases can only represent a proportion (the 'notified fraction') of the total incidence (Figure 1) and this has to be taken into account when interpreting NNDSS data. Moreover, the notified fraction varies by disease, by jurisdiction and by time.

Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories and hospitals. Although there is a list of national notifiable diseases, some diseases are not yet notifiable in some jurisdictions (Table 1).

Map 1. Australian Bureau of Statistics Statistical Divisions and population, Australia, by Statistical Division, 2006



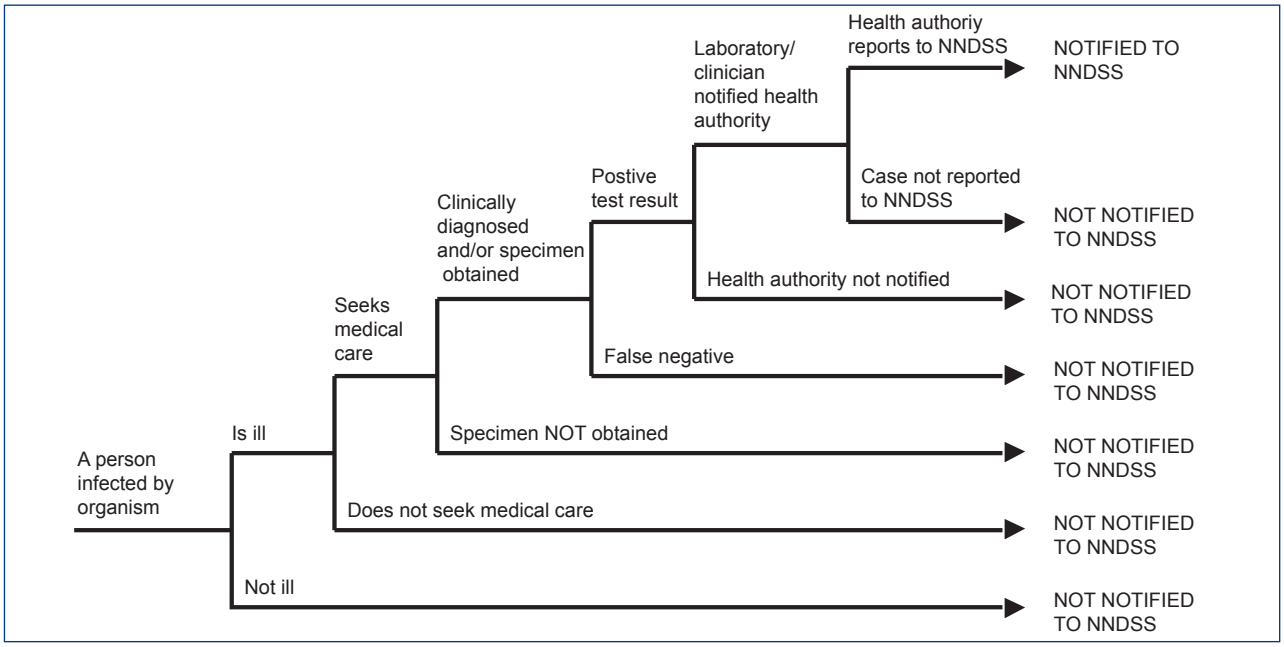
Statistical Division		Population
Australian Capital Territory		
805	Canberra*	328,817
New South Wales		
105	Sydney	4,293,105
110	Hunter	611,935
115	Illawarra	415,248
120	Richmond-Tweed	227,815
125	Mid-North Coast	297,409
130	Northern	181,078
135	North Western	119,276
140	Central West	181,374
145	South Eastern	204,854
150	Murrumbidgee	155,281
155	Murray	116,870
160	Far West	23,449
Northern Territory		
705	Darwin	113,955
710	NT – balance	92,733
Queensland		
305	Brisbane	1,820,375
310	Moreton	868,985
315	Wide Bay-Burnett	264,201

Statistical Division		Population
*Queensland, **continued***		
320	Darling Downs	226,430
325	South West	27,095
330	Fitzroy	193,182
335	Central West	12,155
340	Mackay	151,572
345	Northern	210,943
350	Far North	243,948
355	North West	34,558
South Australia		
405	Adelaide	1,138,833
410	Outer Adelaide	125,903
415	Yorke and Lower North	45,190
420	Murray Lands	69,066
425	South East	63,580
430	Eyre	34,979
435	Northern	77,105
Tasmania		
605	Greater Hobart	205,510
610	Southern	36,176
615	Northern	138,562
620	Mersey-Lyell	108,700
910	**Other territories**	**2,691**

Statistical Division		Population
Victoria		
205	Melbourne	3,684,461
210	Barwon	273,997
215	Western District	102,141
220	Central Highlands	150,412
225	Wimmera	50,920
230	Mallee	93,415
235	Loddon	178,091
240	Goulburn	207,377
245	Ovens-Murray	97,497
250	East Gippsland	84,222
255	Gippsland	169,133
Western Australia		
505	Perth	1,507,949
510	South West	227,981
515	Lower Great Southern	55,259
520	Upper Great Southern	17,609
525	Midlands	52,214
530	South Eastern	53,708
535	Central	60,167
540	Pilbara	40,132
545	Kimberley	35,865
Total Australia		20,605,488

* Includes Statistical Division 810 'Australian Capital Territory – balance'.

Figure 1. Communicable diseases notification fraction



Changes in surveillance practices introduced in some jurisdictions and not in others are additional factors that make comparison of data across jurisdictions difficult. In this report, information obtained from states and territories on any changes in surveillance practices including screening practices, laboratory practices, and major disease control or prevention initiatives undertaken in 2006, was used to interpret data.

Postcode information usually reflects the residential location of the case, but this does not necessarily represent the place where the disease was acquired. As no personal identifiers are collected in NNDSS, duplication in reporting may occur if patients move from one jurisdiction to another and were notified in both.

The completeness* of data in this report is summarised in Appendix 3. The case's sex was complete in 99.8% of notifications and date of birth in 100% of notifications. In 2006, indigenous status was complete in 45.8% of notifications, and varied by jurisdiction. Indigenous status† was complete for 91.4% of data reported in the Northern Territory, 84.1% in South Australia and 71.6% in Western Australia. In the remaining jurisdictions, less than 54% of data were complete for indigenous status.

Data completeness on indigenous status also varied by disease; in notifications of cholera, donovanosis, leprosy, tetanus, hepatitis (NEC) and Murray Valley encephalitis virus infection, reporting on indigenous status was 100% complete. Notifications for tuberculosis (TB), syphilis less than 2 years duration, meningococcal infection and haemolytic uraemic syndrome was more than 90% complete for indigenous status, while in notifications of other diseases such as pertussis, influenza (laboratory confirmed), Barmah Forest virus infection, hepatitis C (unspecified) and Ross River virus infection, data completeness was below 40%.

Notes on case definitions

In this report, each notifiable disease is introduced with a case definition, the 'CDNA case definition'. These case definitions were agreed upon by CDNA to be implemented nationally by January 2004.

CDNA case definitions are only intended for reporting to NNDSS. These definitions have been used by all jurisdictions from 2005 onwards. States and territories may also have case definitions which reflect their local public health needs. These may be the same as or more comprehensive than the CDNA case definitions.

* Data completeness = (total notifications – missing or unknown) / total notifications x 100

† Indigenous status' is a variable defined by the following values:

1=Indigenous – (Aboriginal but not Torres Strait Islander origin)
2=Indigenous – (Torres Strait Islander but not Aboriginal origin)
3=Indigenous – (Aboriginal and Torres Strait Islander origin)
4=Not indigenous – (not Aboriginal or Torres Strait Islander origin)
9=Not stated

Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2006

Disease	Data received from
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)*	All jurisdictions
Hepatitis C (incident)	All jurisdictions, except Queensland
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
STEC, VTEC§	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infections (NEC)‖	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
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 zoster (chickenpox)	All jurisdictions, except ACT, NSW and Victoria
Varicella zoster (shingles)	All jurisdictions, except ACT, NSW and Victoria
Varicella zoster (unspecified)	All jurisdictions, except ACT, NSW and Victoria

Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2006, *continued*

Disease	Data received from
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Flavivirus infection (NEC)‡‡	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection§§	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection¶¶	All jurisdictions
Tuberculosis	All jurisdictions

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection with Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC).

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Laboratory confirmed influenza is not a notifiable disease in South Australia but reports are forwarded to NNDSS.

‡‡ Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004.

§§ In the Australian Capital Territory, Murray Valley encephalitis virus infections and Kunjin virus infections are combined under Murray Valley encephalitis virus infections.

¶¶ Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NEC Not elsewhere classified

Results

Summary of 2006 data

There were 138,511 communicable diseases notifications received by NNDSS in 2006 (Table 2). Notifications rates per 100,000 population for each disease by state or territory are shown in Table 3. Trends in notifications and rates per 100,000 population for the period 2002 to 2006 are shown in Table 4a. The year in which diseases became notifiable to NNDSS in each jurisdiction is shown in Table 4b.

Table 2. Notifications of communicable diseases, Australia, 2006, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0	1	0	0	0	0	0	0	1
Hepatitis B (incident)*	7	54	11	50	7	9	107	50	295
Hepatitis B (unspecified)*,†	70	2,489	236	1,009	316	46	1,564	566	6,296
Hepatitis C (incident)	16	40	3	NN	54	10	200	108	431
Hepatitis C (unspecified)*	175	4,415	229	2,877	517	260	2,542	1,042	12,057
Hepatitis D	0	15	0	8	0	0	7	1	31
Gastrointestinal diseases									
Botulism	0	0	0	1	0	0	0	0	1
Campylobacteriosis‡	403	NN	263	3,967	2,514	596	5,718	1,937	15,398
Cryptosporidiosis	79	780	72	700	202	28	1,090	250	3,201
Haemolytic uraemic syndrome	0	11	0	0	1	0	1	0	13
Hepatitis A	1	95	30	31	8	4	44	67	280
Hepatitis E	2	10	0	2	0	0	8	1	23
Listeriosis	1	26		3	5		13	13	61
Salmonellosis	134	2,059	404	2,711	570	192	1,391	800	8,261
Shigellosis	2	75	125	97	37	3	76	128	543
STEC, VTEC§	0	10	2	15	36	0	4	3	70
Typhoid	0	35	3	6	3	1	19	11	78
Quarantinable diseases									
Cholera	0	3	0	0	0	0	0	0	3
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmitted infections									
Chlamydial infections (NEC)‖	821	11,819	2,056	12,223	3,128	1,044	9,966	5,897	46,954
Donovanosis	0	0	2	2	0	0	0	0	4
Gonococcal infection	33	1,695	1,777	1,559	499	18	1,300	1,666	8,547
Syphilis (all)¶	14	876	269	436	43	22	598	179	2,436
Syphilis – <2 years duration	2	210	150	165	2	5	231	48	813
Syphilis – >2 years or unspecified duration	12	666	119	271	41	17	366	131	1,623
Syphilis – congenital	0	5	8	1	0	0	0	0	14
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	11	2	7	0	0	2	0	22
Influenza (laboratory confirmed)**	80	614	40	1660	89	47	421	208	3,159
Measles	1	60	0	2	9	11	12	30	125
Mumps	1	154	7	58	22	0	16	17	275

Table 2. Notifications of communicable diseases, Australia, 2006, by state or territory, *continued*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases, *continued*									
Pertussis	258	4,916	96	2,178	2,179	41	1,066	264	10,998
Pneumococcal disease (invasive)	18	564	56	253	107	40	274	131	1,443
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	0	37	0	12	2	0	6	2	59
Rubella – congenital	0	0	0	0	0	0	0	0	0
Tetanus	0	2	0	0	0	0	1	0	3
Varicella zoster (chickenpox)	NDP	NN	193	380	760	16	NN	165	1,514
Varicella zoster (shingles)	NDP	NN	80	247	625	55	NN	70	1,077
Varicella zoster (unspecified)	NDP	NN	1	3,167	328	14	NN	55	3,565
Vectorborne diseases									
Barmah Forest virus infection	8	644	130	957	186	0	30	165	2,120
Dengue virus infection	6	50	21	78	11		5	16	187
Flavivirus infection (NEC)‡‡	0	0	0	23	0	0	10	0	33
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0
Kunjin virus infection	0	0	0	1	0	0	0	2	3
Malaria	11	140	66	268	34	26	115	115	775
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	1	1
Ross River virus infection	10	1,225	279	2,615	317	14	209	818	5,487
Zoonoses									
Anthrax	0	1	0	0	0	0	0	0	1
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	8	0	40	0	0	0	1	49
Leptospirosis	0	17	2	117	1	1	6	3	147
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	2	94	0	2	0	1	65	4	168
Q fever	0	174	5	164	18	0	36	5	402
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial infections									
Legionellosis	1	77	3	39	65	3	69	91	348
Leprosy	0	1	1		1	0	0	2	5
Meningococcal infection¶¶	5	107	6	71	18	5	85	21	318
Tuberculosis	14	472	32	149	72	9	367	114	1,229
Total	2,173	33,881	6,510	38,186	12,784	2,516	27,442	15,019	138,511

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection with Shiga toxin-/verotoxin-producing *Escherichia coli* (STEC/VTEC).

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Laboratory-confirmed influenza is not a notifiable disease in South Australia but reports are forwarded to NNDSS.

‡‡ Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004.

§§ In the Australian Capital Territory, Murray Valley encephalitis virus infections and Kunjin virus infections are combined under Murray Valley encephalitis virus 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.

NDP No data provided.

Table 3. Notifications rate for communicable diseases, Australia, 2006, by state and territory (per 100,000 population)

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (incident)*	2.1	0.8	5.3	1.2	0.5	1.8	2.1	2.4	1.4
Hepatitis B (unspecified)*,†	21.3	36.5	114.2	24.9	20.3	9.4	30.7	27.6	30.6
Hepatitis C (incident)	4.9	0.6	1.5	NN	3.5	2.0	3.9	5.3	2.6
Hepatitis C (unspecified)*	53.2	64.7	110.8	71.0	33.3	53.2	49.9	50.8	58.5
Hepatitis D	0.0	0.2	0.0	0.2	0.0	0.0	0.1	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis‡	122.6	NN	127.2	97.9	161.7	121.9	112.3	94.4	111.8
Cryptosporidiosis	24.0	11.4	34.8	17.3	13.0	5.7	21.4	12.2	15.5
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.0	0.1	0.0	0.0	0.0	0.1
Hepatitis A	0.3	1.4	14.5	0.8	0.5	0.8	0.9	3.3	1.4
Hepatitis E	0.6	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Listeriosis	0.3	0.4	0.0	0.1	0.3	0.0	0.3	0.6	0.3
Salmonellosis	40.8	30.2	195.5	66.9	36.7	39.3	27.3	39.0	40.1
Shigellosis	0.6	1.1	60.5	2.4	2.4	0.6	1.5	6.2	2.6
STEC, VTEC	0.0	0.1	1.0	0.4	2.3	0.0	0.1	0.1	0.3
Typhoid	0.0	0.5	1.5	0.1	0.2	0.2	0.4	0.5	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Highly pathogenic avian influenza	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	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
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 transmitted infections									
Chlamydial infections (NEC)‖	249.7	173.1	994.7	301.5	201.2	213.5	195.7	287.5	227.9
Donovanosis	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	10.0	24.8	859.7	38.5	32.1	3.7	25.5	81.2	41.5
Syphilis (all)¶	4.3	12.8	130.1	10.8	2.8	4.5	11.7	8.7	11.8
Syphilis – <2 years duration	0.6	3.1	72.6	4.1	0.1	1.0	4.5	2.3	3.9
Syphilis – >2 years or unspecified duration	3.6	9.8	57.6	6.7	2.6	3.5	7.2	6.4	7.9
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	0.2	1.0	0.2	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)**	24.3	9.0	19.4	41.0	5.7	9.6	8.3	10.1	15.3
Measles	0.3	0.9	0.0	0.0	0.6	2.2	0.2	1.5	0.6
Mumps	0.3	2.3	3.4	1.4	1.4	0.0	0.3	0.8	1.3
Pertussis	78.5	72.0	46.4	53.7	140.2	8.4	20.9	12.9	53.4
Pneumococcal disease (invasive)	5.5	8.3	27.1	6.2	6.9	8.2	5.4	6.4	7.0
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.5	0.0	0.3	0.1	0.0	0.1	0.1	0.3
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3. Notifications rate for communicable diseases, Australia, 2006, by state and territory (per 100,000 population), *continued*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases, *continued*									
Varicella zoster (chickenpox)	NDP	NN	93.4	9.4	48.9	3.3	NN	8.0	18.1
Varicella zoster (shingles)	NDP	NN	38.7	6.1	40.2	11.2	NN	3.4	5.2
Varicella zoster (unspecified)	NDP	NN	0.5	78.1	21.1	2.9	NN	2.7	17.3
Vectorborne diseases									
Barmah Forest virus infection	2.4	9.4	62.9	23.6	12.0	0.0	0.6	8.0	10.3
Dengue virus infection	1.8	0.7	10.2	1.9	0.7		0.1	0.8	0.9
Flavivirus infection (NEC)‡‡	0.0	0.0	0.0	0.6	0.0	0.0	0.2	0.0	0.2
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
Malaria	3.3	2.1	31.9	6.6	2.2	5.3	2.3	5.6	3.8
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.0	17.9	135.0	64.5	20.4	2.9	4.1	39.9	26.6
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	1.0	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.2	1.0	2.9	0.1	0.2	0.1	0.1	0.7
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.6	1.4	0.0	0.0	0.0	0.2	1.3	0.2	0.8
Q fever	0.0	2.5	2.4	4.0	1.2	0.0	0.7	0.2	2.0
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.3	1.1	1.5	1.0	4.2	0.6	1.4	4.4	1.7
Leprosy	0.0	0.0	0.5	0.0	0.1	0.0	0.0	0.1	0.0
Meningococcal infection¶¶	1.5	1.6	2.9	1.8	1.2	1.0	1.7	1.0	1.5
Tuberculosis	4.3	6.9	15.5	3.7	4.6	1.8	7.2	5.6	6.0

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection with Shiga toxin-/verotoxin-producing *Escherichia coli* (STEC/VTEC).

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Laboratory-confirmed influenza is not a notifiable disease in South Australia but reports are forwarded to NNDSS.

‡‡ Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004.

§§ In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

¶¶ 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.

NDP No data provided.

Table 4a. Notifications and notification rate for communicable diseases, Australia, 2002 to 2006, (per 100,000 population)

Disease	Notifications					Rates				
	2002	2003	2004	2005	2006	2002	2003	2004	2005	2006
Bloodborne diseases										
Hepatitis (NEC)	0	0	0	0	1	0.0	0.0	0.0	0.0	0.0
Hepatitis B (incident)*	391	347	283	251	295	2.0	1.7	1.4	1.2	1.4
Hepatitis B (unspecified)*,†	6,684	5,812	5,786	6,336	6,296	34.0	29.2	28.8	31.2	30.6
Hepatitis C (incident)	452	519	453	374	431	2.8	3.2	2.8	2.3	2.6
Hepatitis C (unspecified)*	15,618	13,674	12,760	12,023	12,057	79.5	68.8	63.4	59.1	58.5
Hepatitis D	22	27	29	30	31	0.1	0.1	0.1	0.1	0.2
Gastrointestinal diseases										
Botulism	0	1	1	3	1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	14,732	15,361	15,579	16,488	15,398	113.3	116.4	116.4	121.6	111.8
Cryptosporidiosis‡	3,273	1,223	1,684	3,211	3,201	16.7	6.2	8.4	15.8	15.5
Haemolytic uraemic syndrome	13	15	16	20	13	0.1	0.1	0.1	0.1	0.1
Hepatitis A	392	431	319	326	280	2.0	2.2	1.6	1.6	1.4
Hepatitis E	12	12	28	30	23	0.1	0.1	0.1	0.1	0.1
Listeriosis	62	69	67	54	61	0.3	0.3	0.3	0.3	0.3
Salmonellosis	7,880	7,008	7,838	8,425	8,261	40.1	35.2	39.0	41.4	40.1
Shigellosis	507	442	520	729	543	2.6	2.2	2.6	3.6	2.6
STEC, VTEC	59	52	49	86	70	0.3	0.3	0.2	0.4	0.3
Typhoid	69	51	76	52	78	0.4	0.3	0.4	0.3	0.4
Quarantinable diseases										
Cholera	5	1	5	3	3	0.0	0.0	0.0	0.0	0.0
Highly pathogenic avian influenza	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Plague	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Rabies	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	NN	NN	0	0	0	NN	NN	0.0	0.0	0.0
Smallpox	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
Yellow fever	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible diseases										
Chlamydial infections (NEC)‖	24,437	30,441	36,221	41,376	46,954	124.4	153.1	180.1	203.5	227.9
Donovanosis	17	16	10	13	4	0.1	0.1	0.0	0.1	0.0
Gonococcal infection	6,433	6,790	7,184	8,083	8,547	32.8	34.2	35.7	39.8	41.5
Syphilis (all)¶	2,010	2,017	2,341	2,222	2,436	10.2	10.1	11.6	10.9	11.8
Syphilis – <2 years duration	NN	NN	618	632	813	NN	NN	3.1	3.1	3.9
Syphilis – >2 years or unspecified duration	NN	NN	1723	1590	1,623	NN	NN	8.6	7.8	7.9
Syphilis – congenital	18	13	13	15	14	0.1	0.1	0.1	0.1	0.1
Vaccine preventable diseases										
Diphtheria	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	31	19	15	17	22	0.2	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)**	3,669	3,479	2,134	4,565	3,159	18.7	17.5	10.6	22.5	15.3
Measles	32	93	45	10	125	0.2	0.5	0.2	0.0	0.6
Mumps	69	77	102	241	275	0.4	0.4	0.5	1.2	1.3
Pertussis	5,564	5,096	8,755	11,197	10,998	28.3	25.6	43.5	55.1	53.4
Pneumococcal disease (invasive)	2,415	2,233	2,370	1,749	1,443	12.3	11.2	11.8	8.6	7.0
Poliomyelitis	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Rubella	253	54	31	31	59	1.3	0.3	0.2	0.2	0.3
Rubella – congenital	2	3	1	1	0	0.0	0.0	0.0	0.0	0.0
Tetanus	4	4	5	2	3	0.0	0.0	0.0	0.0	0.0

Table 4a. Notifications and notification rate for communicable diseases, Australia, 2002 to 2006, (per 100,000 population), *continued*

Disease	Notifications					Rates				
	2002	2003	2004	2005	2006	2002	2003	2004	2005	2006
Vaccine preventable diseases, *continued*										
Varicella zoster (chickenpox)	NN	NN	NN	NN	1,514	NN	NN	NN	NN	18.1
Varicella zoster (shingles)	NN	NN	NN	NN	1,077	NN	NN	NN	NN	12.9
Varicella zoster (unspecified)	NN	NN	NN	NN	3,565	NN	NN	NN	NN	42.7
Vectorborne diseases										
Barmah Forest virus infection	910	1,367	1,106	1,322	2,120	4.6	6.9	5.5	6.5	10.3
Dengue virus infection	171	860	351	221	187	0.9	4.3	1.7	1.1	0.9
Flavivirus infection (NEC)‡‡	73	60	61	29	33	0.4	0.3	0.3	0.1	0.2
Japanese encephalitis virus infection	0	1	1	0	0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0	18	12	1	3	0.0	0.1	0.1	0.0	0.0
Malaria	468	592	556	823	775	2.4	3.0	2.8	4.0	3.8
Murray Valley encephalitis virus infection	2	0	1	2	1	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	1,458	3,850	4,209	2,546	5,487	7.4	19.4	20.9	12.5	26.6
Zoonoses										
Anthrax	0	0	0	0	1	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
Brucellosis	40	20	38	41	49	0.2	0.1	0.2	0.2	0.2
Leptospirosis	160	126	177	129	147	0.8	0.6	0.9	0.6	0.7
Lyssavirus (NEC)	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Ornithosis	213	200	239	164	168	1.1	1.0	1.2	0.8	0.8
Q fever	796	563	464	355	402	4.1	2.8	2.3	1.7	2.0
Tularaemia	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections										
Legionellosis	315	333	312	334	348	1.6	1.7	1.6	1.6	1.7
Leprosy	6	5	7	10	5	0.0	0.0	0.0	0.0	0.0
Meningococcal infection¶¶	690	558	405	392	318	3.5	2.8	2.0	1.9	1.5
Tuberculosis	1,128	1,035	1,127	1,083	1,229	5.7	5.2	5.6	5.3	6.0
Total	101,555	104,968	113,786	125,415	138,511					

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection with Shiga toxin-/verotoxin-producing Escherichia coli (STEC/VTEC).

|| Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Laboratory-confirmed influenza is not a notifiable disease in South Australia but reports are forwarded to NNDSS.

‡‡ Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004.

§§ In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

¶¶ 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 4b. Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

Disease†	Earliest year for which NNDSS contains data*								Year from which NNDSS reporting commenced in annual reports	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Bloodborne diseases										
Hepatitis (NEC)	1991	1991	–	–	1990	–	1991	–	1991 to present	Includes reports of hepatitis D and E 1991–1998 WA did not report 1991–2000
Hepatitis B (incident)	1992	1992	1991	1991	1996	1995	1993	1993	1993 to present	
Hepatitis B (unspecified)	1991	1990	2005	1985	1996	1991	1991	1990	1991 to present	Includes reports of incident hepatitis B 1991, 1992, 1994, 1995
Hepatitis C (incident)	1991	1992	2003	NN	1995	1995	1997	1993	1993 to present	Not notifiable in Qld
Hepatitis C (unspecified)	1991	1991	1991	1990	1995	1991	1991	1993	1991 to present	Includes reports of incident hepatitis C 1991–1994 SA did not report 1991–1994 WA did not report 1991–1992
Hepatitis D	–	1992	1997	1992	1998	–	1995	2002	1999 to present	WA did not report 1991–2000
Gastrointestinal diseases										
Botulism	–	1999	2000	2001	1998	–	2000	–	1992 to present	
Campylobacteriosis	1991	NN	1991	1991	1990	1991	1991	1991	1991 to present	Not notifiable in NSW
Cryptosporidiosis	1995	1996	1998	1996	1993	1998	1998	2000	2001 to present	
Haemolytic uraemic syndrome	2007	1997	2002	1997	1995	1996	1998	1996	1999 to present	
Hepatitis A	1991	1990	1990	1991	1990	1991	1991	1991	1991 to present	
Hepatitis E	1999	1993	1993	1991	1997	1997	1995	2001	1999 to present	WA did not report 1991–2000
Listeriosis	1993	1991	1995	1991	1992	1991	1991	1991	1991 to present	ACT and SA did not report 1991 NT did not report 1991–1992
Salmonellosis	1991	1990	1991	1991	1990	1991	1991	1991	1991 to present	
Shigellosis	1991	2001	1991	1991	1990	1991	1991	1991	1991 to present	NSW reported only as 'foodborne disease' or 'gastroenteritis in an institution' 1991–2000
STEC, VTEC	2007	1998	1999	1996	1996	2005	1997	2001	1999 to present	WA did not report 1999–2000
Typhoid‡	1992	1991	1992	1991	1990	1994	1991	1991	1991 to present	
Quarantinable diseases										
Cholera	1995	1993	–	1992	1992	–	1994	1992	1991 to present	
Highly pathogenic avian influenza	–	–	–	–	–	–	–	–	2004 to present	
Plague	–	–	–	–	–	–	–	–	1991 to present	Tas did not report 1991–1995
Rabies	–	–	–	–	–	–	–	–	1991 to present	ACT did not report 1991 NSW did not report 1991–1998
Severe acute respiratory syndrome	–	–	–	–	–	–	–	–	2003 to present	
Smallpox	–	–	–	–	–	–	–	–	2004 to present	
Viral haemorrhagic fever	–	–	–	–	–	–	–	–	1991 to present	ACT did not report 1991
Yellow fever	–	–	–	–	–	–	–	–	1991 to present	
Sexually transmissible infections										
Chlamydial infection	1991	1991	1990	1991	1991	1991	1991	1993	1991 to present	
Donovanosis	–	–	1991	1991	–	–	–	1991	1991 to present	NSW and SA did not report 1991–2001 Tas did not report 1991–1992
Gonococcal infection§	1991	1991	1990	1991	1991	1991	1991	1991	1991 to present	

Table 4b. Earliest notification year for which NNDSS contains disease data, Australia, by state or territory, *continued*

Disease[†]	Earliest year for which NNDSS contains data*								Year from which NNDSS reporting commenced in annual reports	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Sexually transmissible diseases, *continued*										
Syphilis – all[‖]	1991	1991	1990	–	1991	–	1991	1991	1991 to present	
Syphilis < 2 years	1995	1992	2003	1991	1990	2004	1997	1995	2004 to present	
Syphilis > 2 years or unspecified duration	2004	1991	2001	1988	2004	1991	1997	1992	2004 to present	
Syphilis – congenital	–	1992	1995	1991	–	–	1995	1990	2003 to present	
Vaccine preventable diseases										
Diphtheria	–	–	1992	–	1993	–	1991	–	1991 to present	
Haemophilus influenzae type b	1991	1991	1992	1991	1990	1991	1991	1993	1991 to present	WA did not report 1991–1992
Influenza (laboratory confirmed)	1993	2001	1999	2001	2001	2002	2000	2000	2001 to present	Not notifiable in SA 2001–2007 however data has been provided since 2001
Measles	1991	1990	1991	1988	1990	1991	1991	1991	1991 to present	
Mumps	1992	1992	1994	1996	1993	1995	1994	1993	1992 to present	Qld did not report 1992–1995 and 2000 NT did not report 1992–1993 WA and SA did not report 1992 Tas did not report 1992–1994
Pertussis	1991	1990	1992	1991	1990	1991	1991	1990	1991 to present	
Pneumococcal disease (invasive)	1993	2001	1995	1991	2001	1991	2000	2000	2001 to present	
Poliomyelitis	–	–	–	–	–	–	2007	–	1991 to present	
Rubella[¶]	1991	1991	1991	1991	1990	1991	1991	1993	1991 to present	Tas did not report 1992–1994
Rubella – congenital	–	1993	–	2002	1990	–	2005	2002	2003 to present	
Tetanus	–	1991	1991	1997	1990	1992	1992	1991	1991 to present	Qld did not report 1991–1993
Varicella zoster (chickenpox)**	1993	NN	2006	2006	2006	2004	NN	2006	2006 to present	Not notifiable in NSW or Vic.
Varicella zoster (shingles)**	1997	NN	2006	2006	2006	2006	NN	2006	2006 to present	Not notifiable in NSW or Vic.
Varicella zoster (unspecified)**	2006	NN	2006	2005	2006	2006	NN	2006	2006 to present	Not notifiable in NSW or Vic.
Vectorborne diseases										
Barmah Forest virus infection	1995	1992	1992	1992	1993	1999	1995	1994	1995 to present	
Dengue virus infection	1993	1992	1992	1991	1992	1995	1991	1994	1991 to present	ACT did not report 1991–1992
Flavivirus infection (NEC)[††,‡‡]	2001	1991	1992	1991	1990	–	1991	–	1991 to present	Includes Japanese encephalitis, Murray Valley encephalitis and Kunjin 1991–2000
Japanese encephalitis virus infection	–	–	–	1995	–	–	–	1998	2001 to present	
Kunjin virus infection	–	1996	1992	1996	–	–	2001	1997	2001 to present	Reported under Murray Valley encephalitis in the ACT
Malaria	1991	1991	1991	1991	1990	1989	1991	1990	1991 to present	
Murray Valley encephalitis virus infection	–	2008	1991	1991	2001	–	–	1991	2001 to present	Combined with Kunjin in the ACT
Ross River virus infection	1992	1991	1991	1991	1992	1991	1991	1991	1991 to present	

Table 4b. Earliest notification year for which NNDSS contains disease data, Australia, by state or territory, *continued*

Disease†	Earliest year for which NNDSS contains data*								Year from which NNDSS reporting commenced in annual reports	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Zoonoses										
Anthrax	–	2006	–	1998	–	–	2007	–	2001 to present	
Australian bat lyssavirus	–	–	–	1998	–	–	–	–	2001 to present	
Brucellosis	1998	1991	–	1991	1995	2007	1991	1996	1991 to present	
Leptospirosis	1994	1990	1992	1991	1990	1991	1991	1991	1991 to present	
Lyssavirus (NEC)	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Ornithosis	1991	2001	1991	1992	1990	1993	1991	1991	1991 to present	NSW did not report 1991–2000 Qld did not report 1997–2001
Q fever	1991	1991	2002	1991	1990	2000	1991	1991	1991 to present	
Tularaemia	–	–	–	–	–	–	–	–	2004 to present	
Other bacterial infections										
Legionellosis	1991	1991	1992	1991	1990	1992	1991	1991	1991 to present	
Leprosy	1992	1992	1991	1997	1991	2007	1991	1991	1991 to present	
Meningococcal infection	1991	1991	1991	1991	1990	1991	1991	1990	1991 to present	
Tuberculosis	1991	1990	1991	1991	1991	1987	1992	1991	1991 to present	

* Data from NNDSS annual reports from 1991. First full year of reporting to the Commonwealth is shown. Some diseases may have been notifiable to state or territory health departments before the dates shown here.

† Prior to the implementation of the national case definitions in 2001, jurisdictions notified diseases according to their own case definition

‡ Includes paratyphoid in New South Wales, Queensland and Victoria.

§ Includes neonatal ophthalmia in the Northern Territory, Queensland, South Australia, and Victoria.

|| Includes syphilis – congenital from 1991 to 2002.

¶ Includes rubella – congenital from 1991 to 2002.

** Varicella data from the Australian Capital Territory were provided in 2008 and were not provided at the time data for the 2006 report were finalised.

†† Before 1997, includes Ross River virus infection, dengue virus infection and Barmah Forest virus infection.

‡‡ Flavivirus (NEC) replaced arbovirus (NEC) 1 January 2004.

NN Not notifiable.

– No cases reported to NNDSS.

In 2006, the total number of notifications was the highest recorded in NNDSS since the system began in 1991. There was an increase of 10.4% compared with the total number of notifications in 2005 (Figure 2).

In 2006, the most frequently notified diseases were sexually transmissible infection (57,941 notifications, 42% of total notifications), gastrointestinal diseases (27,931 notifications, 20% of total notifications) and vaccine preventable diseases (22,240 notifications, 16% of total notifications).

There were 19,111 notifications of bloodborne diseases; 8,606 notifications of vectorborne diseases; 1,900 notifications of other bacterial infections; 767 notifications of zoonoses and 3 notifications of quarantinable diseases (Figure 3).

Figure 2. Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2006

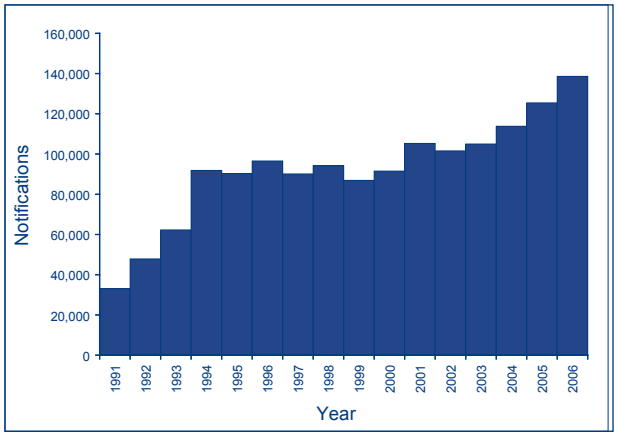
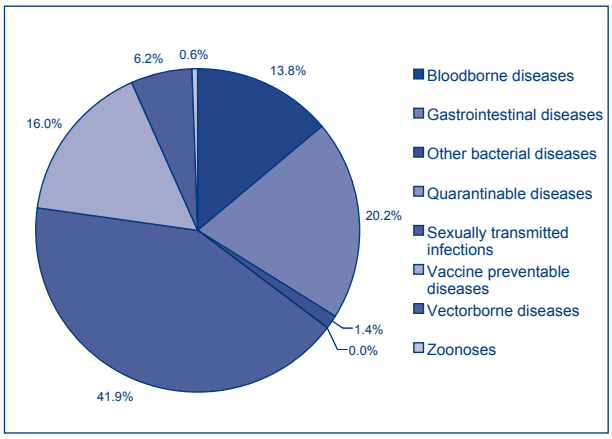
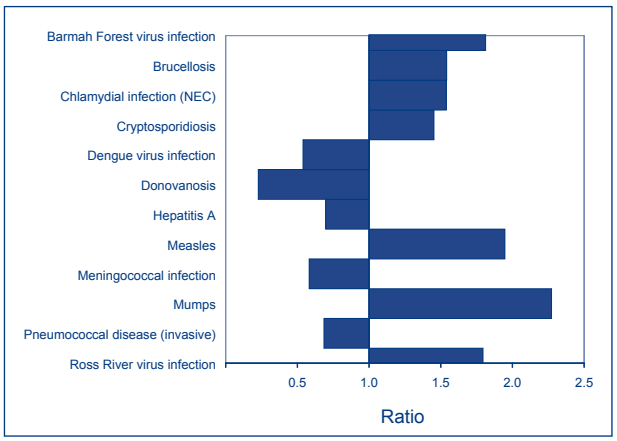


Figure 3. Notifications to the National Notifiable Disease Surveillance System, Australia, 2006, by disease category



The major changes in communicable disease notifications in 2006 are shown in Figure 4 as the ratio of notifications in 2006 to the mean number of notifications for the previous 5 years. Notifications of Barmah Forest virus infection, brucellosis, chlamydial infections, cryptosporidiosis, measles, mumps and Ross River virus infection were above the 5-year mean. Notifications below the 5-year mean were dengue virus infection, donovanosis, hepatitis A, meningococcal infection and pneumococcal disease (invasive). Notifications for the remaining diseases were within the historical range.

Figure 4. Comparison of total notifications of selected diseases reported to the National Notifiable Diseases System in 2006, with the previous 5-year mean



Bloodborne diseases

Bloodborne viruses reported to the NNDSS include hepatitis B, C and D. HIV and AIDS diagnoses are reported directly to the National Centre in HIV Epidemiology and Clinical Research (NCHECR). Information on national HIV/AIDS surveillance can be obtained through the NCHECR website at www.nchecr.unsw.edu.au

Hepatitis B

Incident hepatitis B notifications

Case definition – Incident hepatitis B

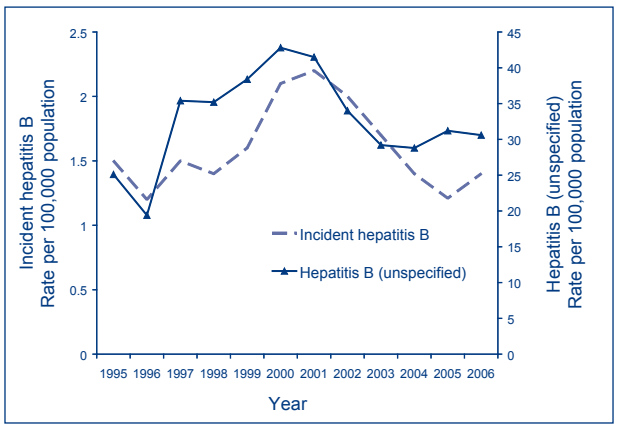
*Only **confirmed cases** are reported.*

***Confirmed case:** Detection of hepatitis B surface antigen (HBsAg) in a case shown to be negative within the last 24 months, OR detection of hepatitis HBsAg and IgM to hepatitis B core antigen in the absence of prior evidence of hepatitis B infection OR detection of hepatitis B virus by nucleic acid testing and IgM to hepatitis B core antigen in the absence of evidence of prior hepatitis B infection.*

In 2006, 295 cases of incident hepatitis B infection were reported to NNDSS, which was higher than in 2005 (251). The Northern Territory recorded the highest notification rate in 2006 with 5.3 cases per 100,000 population. Over the past 10 years, the rate of notification of incident hepatitis B infection increased from 1.5 cases per 100,000 population in 1996 to 2.2 cases per 100,000 population in 2001, and then declined to 1.2 cases per 100,000 population in 2005 and increased to 1.4 cases per 100,000 population in 2006 (Figure 5).

The increase in the number of incident hepatitis B notifications in 2006 may be a result of more complete case follow-up, as there was a corresponding decrease in hepatitis B (unspecified) notifications for the period.

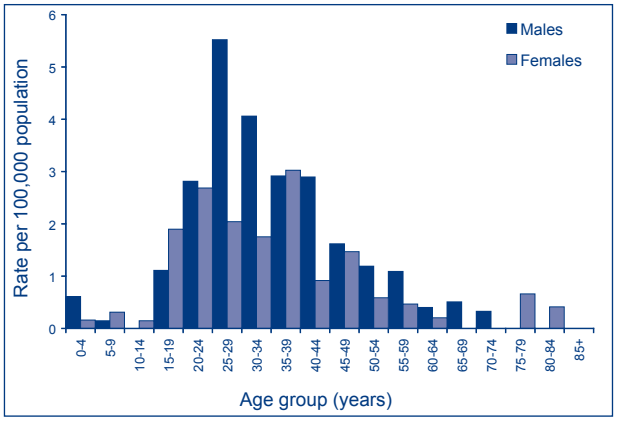
Figure 5. Notification rate of incident hepatitis B and hepatitis B (unspecified), Australia, 1995 to 2006, by year*



* Year of onset for incident hepatitis B and year of report for hepatitis B (unspecified) notifications.

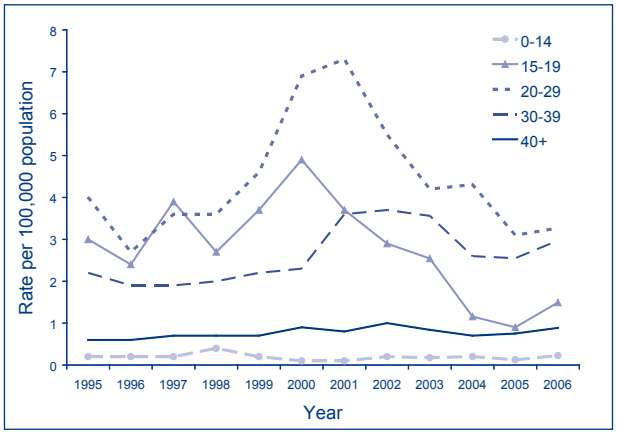
In 2006, the 25–29 years age group among males had the highest rate of incident hepatitis B infection (5.5 cases per 100,000 population), whereas the 35–39 years age group had the highest notification rate among females (3.0 cases per 100,000 population; Figure 6). Notifications of incident hepatitis B infection in males exceeded those in females, with a male to female ratio of 1.6:1 in 2006.

Figure 6. Notification rate for incident hepatitis B infections, Australia, 2006, by age group and sex



Trends in incident hepatitis B infection by year and age group are shown in Figure 7. In 2000–2006, the notification rate of incident hepatitis B fell by 69% among cases in the 15–19 years age group, and by 52% among cases in the 20–29 years age group. The adolescent hepatitis B vaccination program for children aged 10–13 years that was introduced in 1997,[1] may have played a role in this reduction for these age groups.

Figure 7. Notification rate of incident hepatitis B infections, Australia, 1995 to 2006, by year and age group



The source of exposure for cases of incident hepatitis B infection in 2006 was reported through health authorities in the Australian Capital Territory, South Australia, Tasmania and Victoria (Table 5). From 2002 to 2006, the proportion of notifications of incident hepatitis B infection associated with injecting drug use, remained relatively stable at approximately 51.0%. The proportion of diagnoses attributed to heterosexual contact decreased from about 21.0% between 2002 to 2005, to 11.4% in 2006. The source of exposure to hepatitis B was undetermined in approximately 26.0% of cases.

Table 5. Incident hepatitis B infection, Australia,* 2006, by exposure category†

Exposure category	Number	Percentage
Injecting drug use	68	51.5
Sexual contact	19	14.4
Male homosexual contact	3	2.3
Heterosexual contact	15	11.4
Not specified	1	0.8
Blood/tissue recipient	0	0.0
Skin penetration procedure	1	0.8
Healthcare exposure	0	0.0
Household contact	4	3.0
Other	5	3.8
Undetermined	35	26.5
Total exposures	132	100

Source: National Centre in HIV Epidemiology and Clinical Research 2007.

* Data include diagnosis in South Australia, Tasmania, Victoria and the Australian Capital Territory.

† More than one exposure category for each case could be recorded.

Hepatitis B (unspecified) notifications

Case definition – Hepatitis B (unspecified)

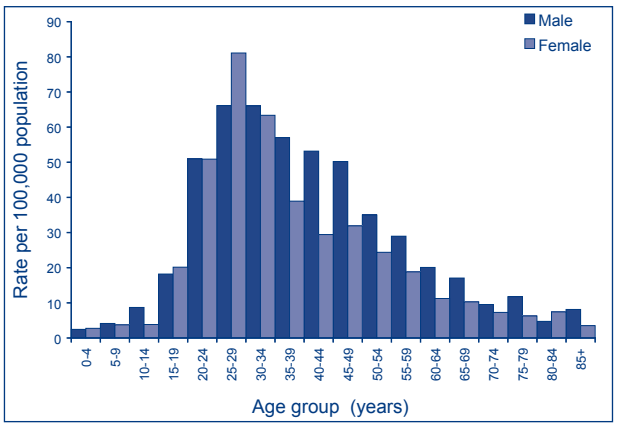
*Only **confirmed cases** are reported.*

***Confirmed case:** Detection of hepatitis B surface antigen or hepatitis B virus by nucleic acid testing in a case who does not meet any of the criteria for a newly acquired case.*

In 2006, a total of 6,296 cases of hepatitis B (unspecified) infection were notified to the NNDSS, compared with 6,336 in 2005. The Northern Territory recorded the highest notification rate (114.2 cases per 100 000 population), compared with other jurisdictions such as New South Wales (36.5 cases per 100,000 population) and Victoria (30.7 cases per

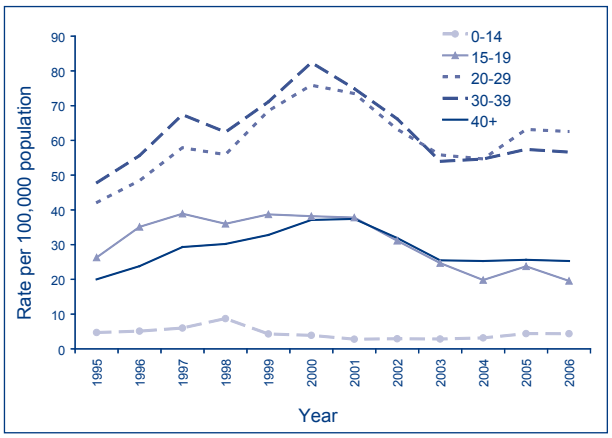
100,000 population). For 2006, the male to female ratio of notifications was 1.2:1. Among males, the highest notification rate was in the 25–29 and the 30–34 years age groups (66.1 and 66.0 cases per 100,000 population, respectively), whereas among females, the highest notification rate was in the 25–29 years age group (81.1 cases per 100,000 population), (Figure 8).

Figure 8. Notification rate for hepatitis B (unspecified) infection, Australia, 2006, by age group and sex



Notification rates of hepatitis B (unspecified) infection increased from 19.4 in 1996 to 42.8 in 2000 then declined to 30.6 cases per 100,000 population in 2006 (Figure 9). In 2006, rates of hepatitis B (unspecified) notifications continued to remain in the range of rates seen in 2003 to 2005 (29.2–31.2 cases per 100,000 population). Trends in hepatitis B (unspecified) infection by age group, and year are shown in Figure 9. Rates in the 15–19 years age group decreased in 2006 by 17.6% compared with 2005 (19.6 and 23.8 cases per 100,000 population, respectively).

Figure 9. Notification rate for hepatitis B (unspecified) infection, Australia, 1995 to 2006, by year and age group



In 2006, 5 cases of hepatitis B (incident) and 33 cases of hepatitis B (unspecified) infection were notified in children in the 0–4 years age group and represented 1.6% and 0.5% of all hepatitis cases notified respectively. Approximately 94% of infants born in Australia in 2006 received the hepatitis B vaccination.

Hepatitis C

Incident hepatitis C notifications

Case definition – Incident hepatitis C

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection of anti-hepatitis C antibody or detection of hepatitis C virus in a case with a negative test recorded in the last 24 months OR detection of anti-hepatitis C antibody in a case aged 18 to 24 months or detection of hepatitis C virus in a case aged 1 to 24 months OR detection of anti-hepatitis C antibody or hepatitis C virus AND clinical hepatitis within the last 24 months (defined as jaundice, urine bilirubin or ALT seven times the upper limit of normal) where other causes of acute hepatitis have been excluded.*

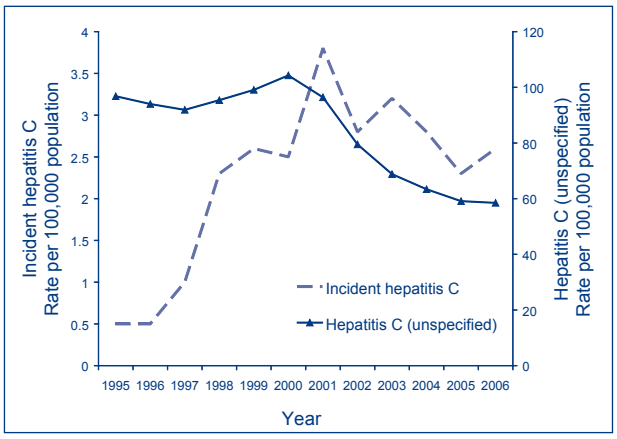
Notifications of incident hepatitis C were received from all jurisdictions except Queensland, where all cases of hepatitis C are reported as hepatitis C (unspecified). A total of 431 cases of incident hepatitis C were notified in 2006 (374 cases in 2005), giving a rate of notification of 2.6 cases per 100,000 population (Figure 10). The proportion of all hepatitis C notifications in 2006 that were documented as incident cases was 3.5%, compared with 3% in 2005. The highest rates of incident hepatitis C infection were reported from Western Australia (5.3 cases per 100,000 population) and the Australian Capital Territory (4.9 cases per 100,000 population).

The increase in the number of incident hepatitis C notifications in 2006 may be a result of more complete case follow-up, as there was a corresponding decrease in hepatitis C (unspecified) notifications for the period.

In 2006, as in 2005, the highest rates of incident hepatitis C notifications were in the 25–29 years age group in males (11.6 cases per 100,000 population) and in the 20–24 and 25–29 years age groups in females (6.5 cases per 100,000 population) (Figure 11).

Trends in the age distribution of incident hepatitis C infection are shown in Figure 12. From 2001 to 2006, notification rates declined by 56% in the

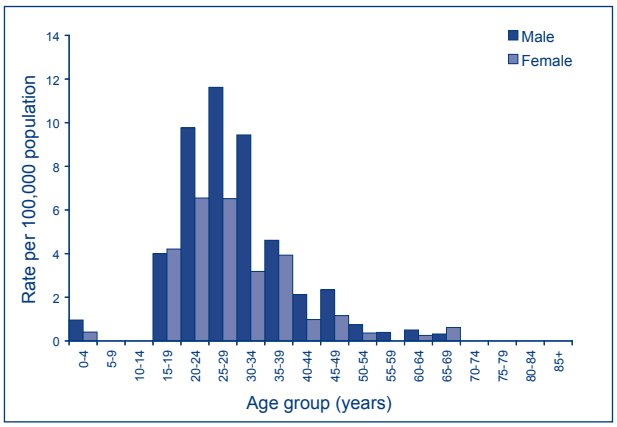
Figure 10. Notification rate of hepatitis C infection (incident* and unspecified†), Australia, 1995 to 2006



* Data from all states and territories except Queensland.

† Data provided from Queensland includes both incident and unspecified hepatitis C cases.

Figure 11. Notification rate of incident hepatitis C infection,* Australia, 2006, by age group and sex

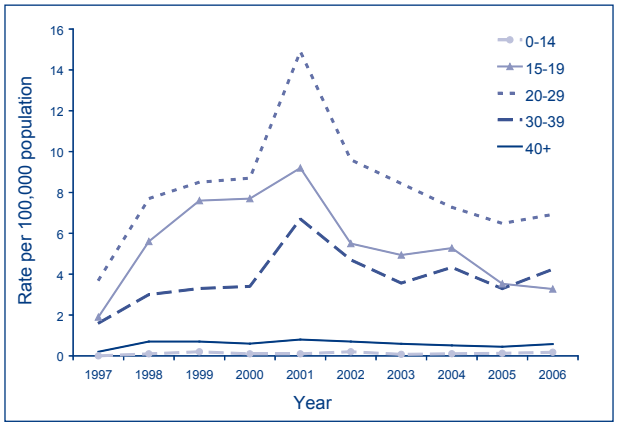


* Data from all states and territories except Queensland.

15–19 years age group, by 42% in the 20–29 years age range and by 21% in the 30–39 years age range. In 2005 to 2006, notification rates increased by 8.8% in the 20–29 years age range and by 29.6% in the 30–39 years age range.

The exposure history of cases of incident hepatitis C were collected in the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia in 2006 (Table 6). At least 62% of incident hepatitis C infections were among people with a history of injecting drug use.

Figure 12. Notification rate of incident hepatitis C infection,* Australia, 1997 to 2006, by age group and year



* Data from all states and territories except Queensland.

Table 6. Incident hepatitis C infection, Australia,* 2006, by exposure category†

Exposure category	Number	Percentage
Injecting drug use	295	62.2
Sexual contact	26	5.5
Blood/tissue recipient	2	0.4
Skin penetration procedure	37	7.8
Healthcare exposure	12	2.5
Household contact	2	0.4
Other‡	30	6.3
Undetermined	70	14.8
Total exposures	474	100

Source: National Centre in HIV Epidemiology and Clinical Research 2007.

* Data includes diagnoses in the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria, Western Australia and the Northern Territory.

† More than one exposure category for each case could be recorded.

‡ Includes cases for which the only reported risk factor was having been born to a woman with hepatitis C infection.

Hepatitis C (unspecified) notifications

Case definition – Hepatitis C (unspecified)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection of anti-hepatitis C antibody or detection of hepatitis C virus in a case who does not meet any of the criteria for a newly acquired case and is aged more than 24 months.*

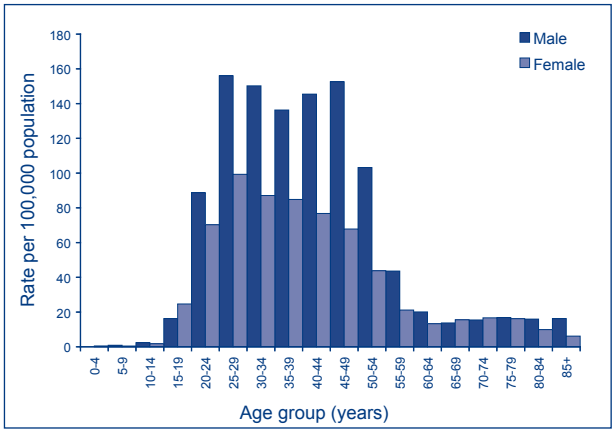
In 2006, 12,057 hepatitis C (unspecified) infections were notified to NNDSS (12,023 in 2005). This figure differs slightly from figures reported in the

National Centre in HIV Epidemiology and Clinical Research National Surveillance Report 2007[2] due to the late exclusion of 38 cases that did not meet the national case definition.

The national notification rate for hepatitis C (unspecified) infection declined from 104.0 cases per 100,000 population in 2001 to 58.5 cases per 100,000 population in 2006 (Figure 10). Improved surveillance practices, such as more complete follow-up and classification of incident cases and increased duplicate notification checks may account for some of the decrease in hepatitis C (unspecified) notifications.

In 2006, the Northern Territory continued to have the highest notification rate (110.8 cases per 100,000 population). Nationally, the male to female ratio was 1.7:1. The highest notification rates occurred in the 25–29, 30–34 and 45–49 years age groups (156.0, 150.2 and 152.6 cases per 100,000 population, respectively) among males and in the 25–29 years age group (99.3 cases per 100,000 population) among females (Figure 13).

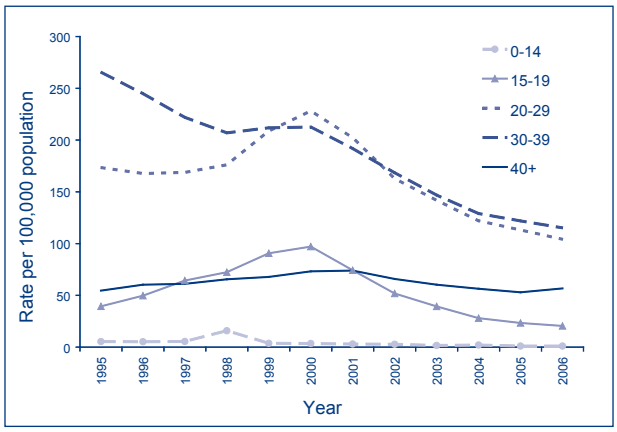
Figure 13. Notification rate for hepatitis C (unspecified) infection* Australia, 2006, by age group and sex



* Data provided from Queensland includes both incident and unspecified hepatitis C cases.

Trends in the age distribution of hepatitis C (unspecified) infection are shown in Figure 14. From 2000 to 2006, the notification rates of hepatitis C (unspecified) among the 15–19 years age group decreased by 78.8%. Notification rates also fell on average by 12% per year for the same period among cases in the 20–29 years age group and by 8.0% compared with 2005. In the 30–39 years age group, notification rates have also been declining on average by 9.7% per year since 2000. The decline in the population rate of notification of hepatitis C infection may be attributable to a reduction in risk behaviour related to injecting drug use, but changes in the rates of testing and percentage classified as incident cases may also have contributed to the decline.

Figure 14. Notification rate of hepatitis C (unspecified) infection,* Australia, 1995 to 2006, by age group



* Data provided from Queensland includes both incident and unspecified hepatitis C cases.

Although initial hepatitis C infection may be asymptomatic (more than 90% of cases) or mildly symptomatic, a high percentage (50%–80%) of cases develop a chronic infection. Of chronically infected persons, approximately 50% will eventually develop cirrhosis or cancer of the liver.[3] In 2006, it is estimated that 271,000 people, living in Australia, had been exposed to the hepatitis C infection. Of these cases approximately 157,000 had early liver disease (stage F0/1), and 40,000 had moderate liver disease (stage F2/3) associated with chronic hepatitis C infection; 5,400 were living with hepatitis C related cirrhosis; and 68,500 had cleared their infection.[2]

Hepatitis D

Case definition – Hepatitis D

*Only **confirmed cases** are reported.*

***Confirmed case:** Detection of IgM or IgG antibodies to hepatitis D virus or detection of hepatitis D on liver biopsy in a case known to be hepatitis B surface antigen positive.*

Hepatitis D is a defective single-stranded RNA virus that requires the presence of the hepatitis B virus to replicate. Hepatitis D infection can occur either as a co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection.[3] People co-infected with hepatitis B and hepatitis D may have more

severe acute disease and a higher risk of fulminant hepatitis compared with those with hepatitis B alone. The modes of hepatitis D transmission are similar to those for hepatitis B, and in countries with low hepatitis B prevalence, injecting drug users are the main risk group for hepatitis D.

There were 31 notifications of hepatitis D to the NNDSS in 2006, compared with 30 notifications in 2005, giving a notification rate of 0.15 cases per 100,000 population. The male to female ratio was 2.4:1. Of the 31 notifications, 15 were reported from New South Wales, 8 from Queensland, 7 from Victoria and 1 from Western Australia.

Gastrointestinal diseases

In 2006, gastrointestinal diseases notified to NNDSS were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga toxin-producing *Escherichia coli*/verotoxigenic *E. coli* (STEC/VTEC) infections and typhoid.

Notifications of gastrointestinal diseases in 2006 decreased to 27,924 from 29,424 in 2005 (Table 4a).

Compared with 2005, there was a decrease in the number of notifications of all gastrointestinal diseases except for listeriosis (an increase from 54 to 61 cases) and typhoid (an increase from 52 to 78 cases). Declines in the number of notifications in other diseases ranged from 1% in cryptosporidiosis to a 26% decline in shigellosis notifications (Table 4a).

The reported changes in the number of notifications were within the expected range (the 5-year mean plus or minus 2 standard deviations).

Botulism

Case definition – Botulism

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Clostridium botulinum *OR detection of* Clostridium botulinum *toxin in blood or faeces AND a clinically compatible illness (e.g. diplopia, blurred vision, muscle weakness, paralysis, death).*

In 2006, a single case of intestinal botulism that was not foodborne, was reported in 2006 in a 2-year-old child from Queensland. Intestinal botulism arises from the ingestion of *Clostridium botulinum* spores, which then germinate to produce and release toxin in the colon. Sources of intestinal botulism are poorly understood, but honey and dust have been suspected in the past.Cases of foodborne botulism are extremely rare. Since NNDSS commenced in 1991, there has only been 1 case of foodborne botulism, which was reported in 1999.

Campylobacteriosis

Case definition – Campylobacteriosis

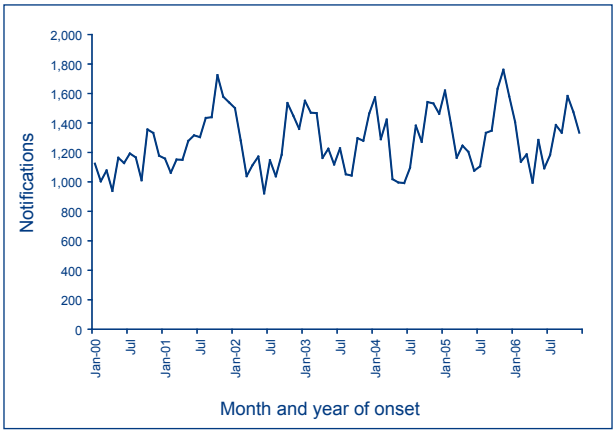
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation or detection of* Campylobacter *species.*

There were 15,398 notifications of campylobacteriosis in 2006, a 7% decline on the 16,488 notifications reported in 2005. Campylobacteriosis is notifiable in all jurisdictions except New South Wales. The national rate of notifications in 2006 was 111 cases per 100,000 population. The highest rate was reported in South Australia (161.7) and the lowest in Western Australia (94.4, Table 3).

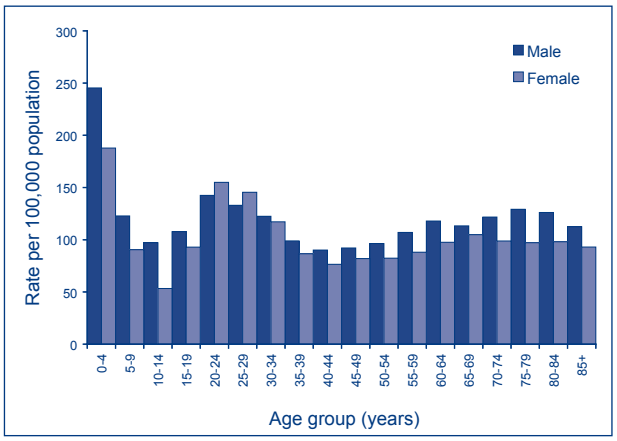
There was an increase in campylobacteriosis notifications in spring and summer, consistent with previous years (Figure 15).

Figure 15. Trends in notifications of campylobacteriosis, Australia, 2000 to 2006, by month of onset



Children aged 0–4 years had the highest notification rate of *Campylobacter* infection (218 cases per 100,000 population) with a secondary peak in the 20–24 years age group (148 cases per 100,000 population, Figure 16). In the 0–4 years age group notification rates were higher in males (245 cases per 100,000 population) than in females (188 cases per 100,000 population). The overall male to female ratio, as in previous years, was 1.15:1.

Figure 16. Notification rate of campylobacteriosis, Australia, 2006, by age group and sex



Cryptosporidiosis

Case definitions – Cryptosporidiosis

*Only **confirmed cases** are reported.*

Confirmed case: *Requires detection of* Cryptosporidium oocytes.

Laboratory definitive evidence: detection of Cryptosporidium oocytes.

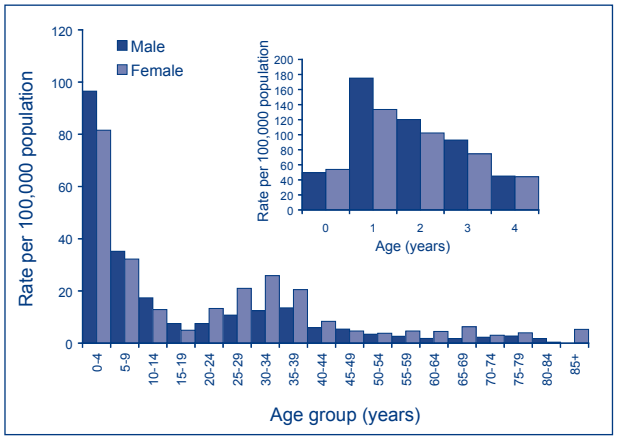
In 2006, a total of 3,201 cases of cryptosporidiosis were reported to NNDSS (15.5 cases per 100,000 population), a similar number and rate to 2005 (3,211 cases, 15.8 cases per 100,000 population).

The highest rates of cryptosporidiosis were reported in the Northern Territory (34.8 cases per 100,000 population) and the Australian Capital Territory (24 cases per 100,000 population).

Of the 3,201 cases of cryptosporidiosis notified to NNDSS in 2006, 1,142 (35%) were under the age of 5 years. Within this age group, boys aged 1 year had the highest notification rate at 175 cases per 100,000 population (Figure 17).

There was a prolonged increase in cryptosporidiosis notifications from New South Wales, Queensland and Victoria from November 2005 to May 2006. Rates of cryptosporidiosis were also elevated in the Australian Capital Territory and the Northern Territory. Interviews with Victorian cryptosporidiosis cases notified between January and May 2006 identified 36 swimming pools as a probable source for 2 or more cases and 2 outbreaks at a special-needs school associated with person to person spread. Hyperchlorination of the swimming pools and infection control procedures at the school brought these outbreaks under control.[4]

Figure 17. Notification rate of cryptosporidiosis, Australia, 2006, by age group and sex



Hepatitis A

Case definition – Hepatitis A

*Both **confirmed cases** and **probable cases** are reported.*

Confirmed case: *Requires detection of anti-hepatitis A IgM, in the absence of recent vaccination, OR detection of hepatitis A virus by nucleic acid testing.*

Probable case: *Requires clinical hepatitis (jaundice and/or bilirubin in urine) without a non-infectious cause AND contact between 2 people involving a plausible mode of transmission at a time when: (a) one of them is likely to be infectious (from 2 weeks before the onset of jaundice to a week after onset of jaundice), AND (b) the other has an illness that starts within 15 to 50 (average 28–30) days after this contact, AND at least 1 case in the chain of epidemiologically-linked cases (which may involve many cases) is laboratory confirmed.*

There were 280 notifications of hepatitis A in 2006 (1.4 cases per 100,000 population) a decrease of 14% on the 326 cases of hepatitis A reported to NNDSS in 2005. The number of notifications of hepatitis A decreased between 1998 and 2001 and have remained stable since 2002 (Figure 18).

The Northern Territory had the highest notification rate (14.5 cases per 100,000 population) followed by Western Australia (3.3 cases per 100 000 population) and New South Wales (1.4 cases per 100,000 population). Rates in all other jurisdictions were less than 1 case per 100,000 population (Table 3).

There were more notifications from males than females with a ratio of 1.2:1. Rates were highest in the 5–9 years age group (2.8 cases per 100,000 population, Figure 19).

In 2006, Indigenous Australians had higher notification rates of hepatitis A infections (6 cases per 10,000 population) compared with non-Indigenous

Figure 18. Trends in notifications of hepatitis A, Australia, 1991 to 2006, by month of notification

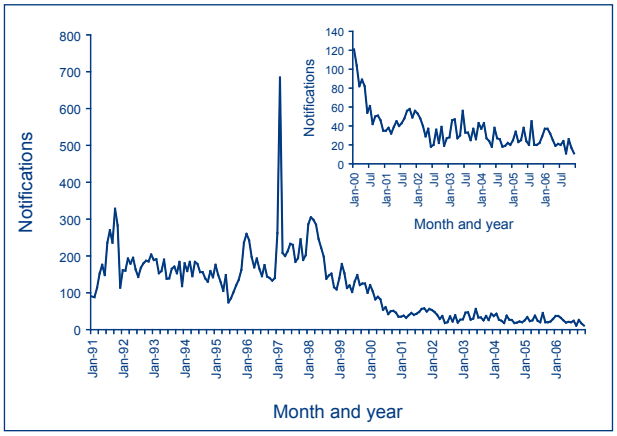
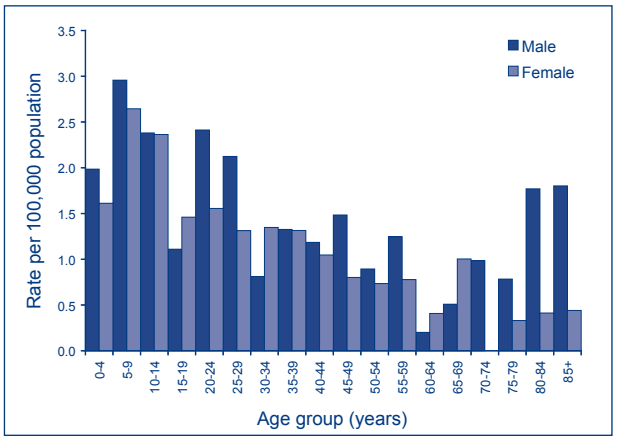


Figure 19. Notification rate of hepatitis A, Australia, 2006, by age group and sex



Australians (1.3 cases per 100,000 population). In 2006, indigenous status was complete in 86% of hepatitis A notifications and 10% overall were Indigenous (Table 7).

Hepatitis E

Case definition – Hepatitis E

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection of hepatitis E virus by nucleic acid testing OR, detection of hepatitis E virus in faeces by electron microscopy OR, detection of IgM or IgG to hepatitis E virus. If the person has not travelled outside Australia in the preceding 3 months, the antibody result must be confirmed by specific immunoblot.*

There were 23 cases of hepatitis E in 2006, a decrease of 23% on the 30 cases reported to NNDSS in 2005. Ten cases were reported from New South Wales, 8 from Victoria, 2 each in the Australian Capital Territory and Queensland and 1 from Western Australia.

There were 13 male and 10 female cases (male to female ratio 1.3:1). Cases were aged 16–61 years. Eleven of the cases acquired their infections overseas.

Listeriosis

Case definitions – Listeriosis

*Only **confirmed cases** are reported. Where a mother and foetus/neonate are both confirmed, both cases are reported.*

***Confirmed case:** Requires isolation or detection of* Listeria monocytogenes *from a site that is normally sterile, including foetal gastrointestinal contents.*

In 2006, 61 cases of listeriosis were notified to NNDSS, a 13% increase on the 54 cases reported to NNDSS in 2005.

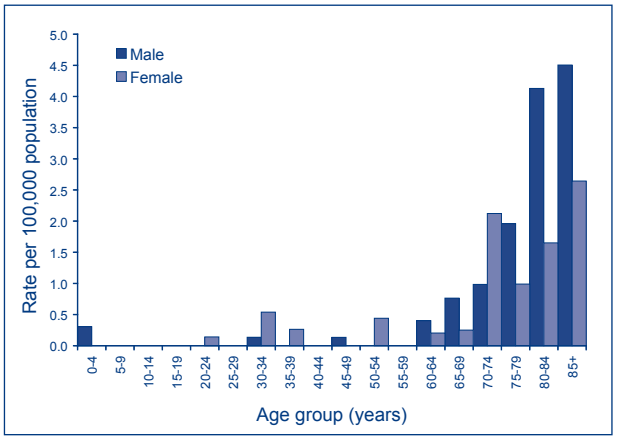
Table 7. Hepatitis A notifications, Australia, 2006, by indigenous status

State or territory	Indigenous		Non-Indigenous*		Total	
	Notifications	Rate	Notifications	Rate	Notifications	Rate
Australian Capital Territory	0	0.0	1	0.3	1	0.3
New South Wales	2	1.5	93	1.4	95	1.4
Northern Territory	10	16.5	20	13.7	30	14.5
Queensland	0	0.0	31	0.8	31	0.8
South Australia	3	11.3	5	0.3	8	0.5
Tasmania	0	0.0	4	0.8	4	0.8
Victoria	0	0.0	44	0.9	44	0.9
Western Australia	13	19.4	54	2.7	67	3.3
Total	28	6.0	252	1.3	280	1.4

* Notifications in non-Indigenous persons include diagnoses in persons whose indigenous status was not reported.

In 2006, 50 (82%) listeriosis cases were aged over 50 years, with the highest notification rate in the 85 years or over age group in males and females (Figure 20). Six cases (4 women and 2 men) aged between 76 and 87 years died. Eight cases of materno-foetal listeriosis were reported to OzFoodNet in 2006.[4] In 2 of these cases the infant died.

Figure 20. Notification rate of listeriosis, Australia, 2006, by age group and sex



Salmonellosis

Case definitions: – Salmonellosis

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation or detection of* Salmonella *species (excluding* Salmonella typhi *which is notified separately under typhoid).*

There were 8,261 notification of salmonellosis (40.1 cases per 100,000 population) to NNDSS in 2006, a 2% decline from the 8,425 notifications reported in 2005.

The highest notification rates were reported in the Northern Territory (196 cases per 100,000 population), Queensland (67 cases per 100,000 population) and the Australian Capital Territory (41 cases per 100,000 population, Table 3 and Map 2).

The highest rate of notification was in children aged between 0–4 years: 30% of salmonellosis notifications were in this age group (Figure 21). The male to female ratio was 1:1.

The 10 most frequently isolated serovars and phage types of *Salmonella,* which accounted for 39% of all isolates, are shown in Table 8. Nationally, *S.* Typhimurium 135 (including 135a), Saintpaul and Typhimurium 170/108 were the 3 most frequently isolated serovars/phage types.

Map 2. Notification rates of salmonellosis, Australia, 2006, by Statistical Division of residence

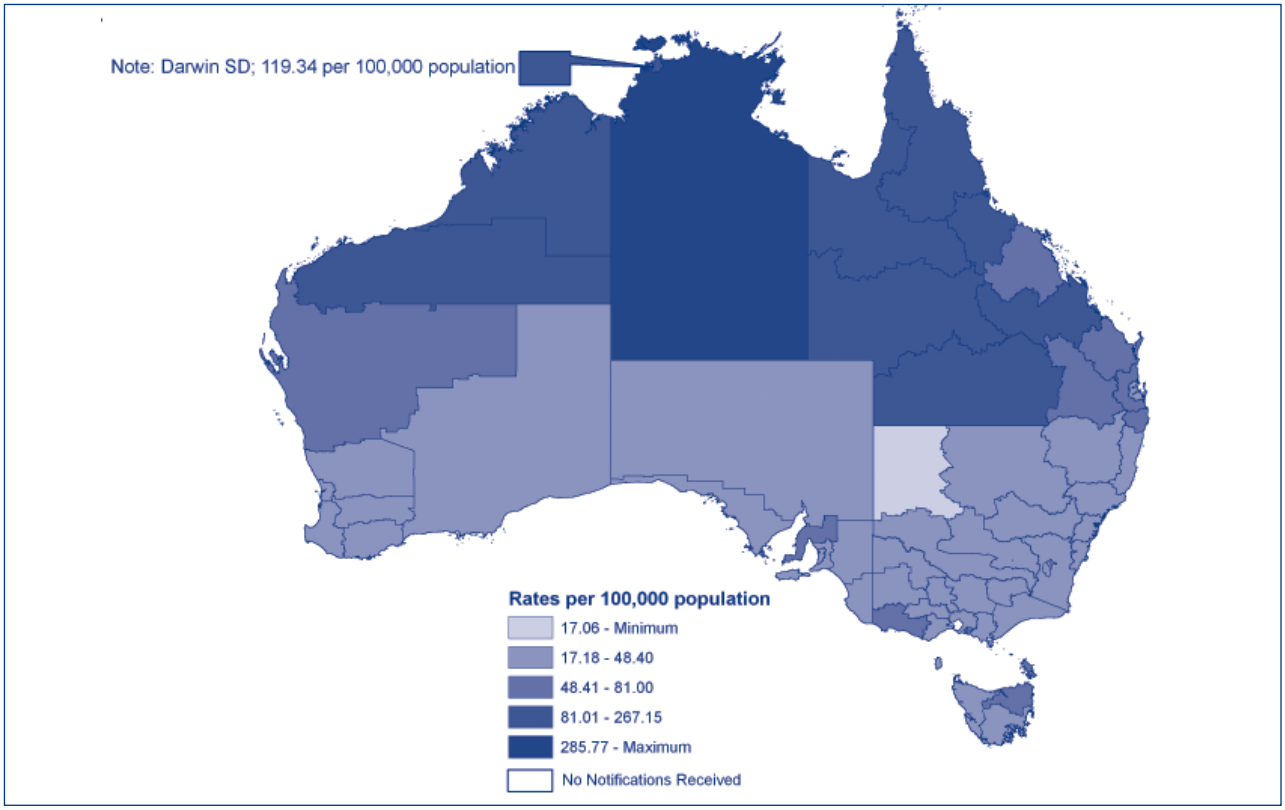
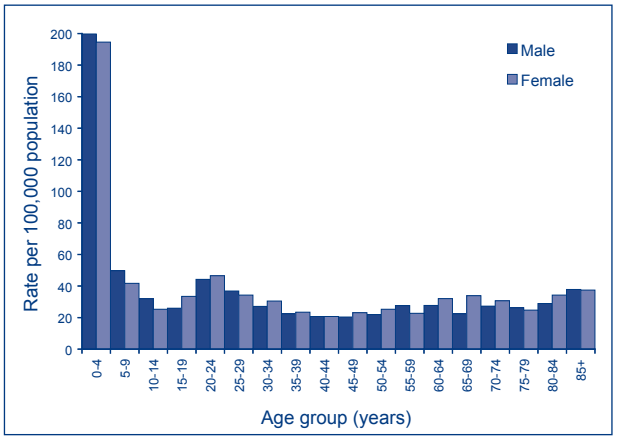


Figure 21. Notification rate of salmonellosis, Australia, 2006, by age group and sex



In 2006, OzFoodNet reported 41 outbreaks of foodborne salmonellosis. *S.* Typhimurium species were responsible for 25 of the 41 (61%) *Salmonella* outbreaks. Eggs and foods made with eggs were implicated in 16 outbreaks of salmonellosis.[4]

Shigellosis

Case definitions – Shigellosis

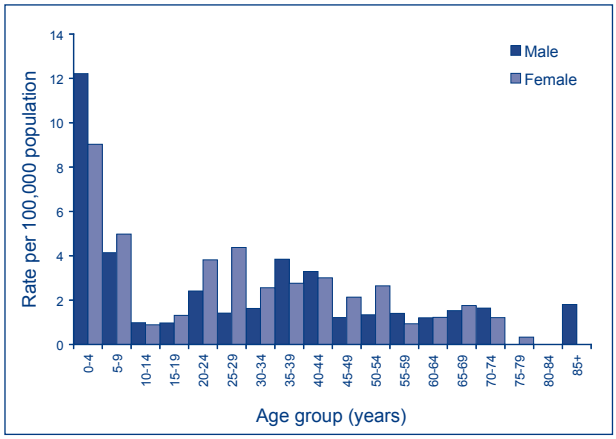
Only ***confirmed cases*** *are reported.*

Confirmed case: *Isolation or detection of* Shigella *species.*

In 2006 there were 543 cases of shigellosis reported to NNDSS, a decrease of 26% on the 729 cases reported in 2005. The 2006 notification rate was 2.6 cases per 100,000 population. The Northern Territory had the highest notification rate (61 cases per 100,000 population).

Children under the age of 5 years represented 25% of shigellosis notifications (136 cases, 10.7 per 100,000 population, Figure 22). The male to female rate ratio was 0.9:1.

Figure 22. Notification rate of shigellosis, Australia, 2006, by age group and sex



The highest burden of shigellosis continues to be in Indigenous populations. In 2006, of the notifications of shigellosis where indigenous status of cases was complete (71% of all cases) 38% were identified as Indigenous. In the Northern Territory (where in 97% of notifications the indigenous status of cases was complete), 91% of shigellosis cases were Indigenous and in South Australia (97% complete), 46% were Indigenous.

Shigella flexneri and *Shigella sonnei* infections accounted for 58.2% and 34.8% of shigellosis, respectively in 2006 (Table 9). Ninety-three per cent of *Shigella flexneri* infections were further typed, of which (104, 35%) were type 4a and 55 (18%) were

Table 8. Top 10 isolates of *Salmonella*, Australia, 2006

Salmonella type (sero/phage type)	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Salmonella Typhimurium 135*	11	195	20	169	79	39	143	53	709
Salmonella Saintpaul	14	100	31	264	13	6	74	57	559
Salmonella Typhimurium 170/108	11	212	0	54	58	14	99	5	453
Salmonella Typhimurium 9	7	76	1	62	58	14	109	11	338
Salmonella Virchow 8	2	27	13	207	0	1	9	4	263
Salmonella Birkenhead	1	101	0	150	0	0	4	0	256
Salmonella Typhimurium 44	6	41	1	29	16	3	109	6	211
Salmonella Infantis	2	59	15	17	36	1	25	10	165
Salmonella Chester	0	28	17	64	6	1	13	25	154
Salmonella Muenchen	0	27	16	67	6	0	6	31	153
Total	54	866	114	1,083	272	79	591	202	3,261

* Includes *Salmonella* Typhimurium 135a.

Table 9. Shigella infections, Australia, 2006, by serogroups and state or territory

Organism	State or territory									Per cent
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	
S. boydii	0	3	0	2	0	0	2	0	7	1.3
S. dysenteriae	0	1	0	2	0	0	1	2	6	1.1
S. flexneri	0	35	109	23	25	2	26	96	316	58.2
S. sonnei	0	34	13	59	11	1	46	25	189	34.8
Sub total	0	73	122	86	36	3	75	123	518	95.4
Unknown	2	2	3	11	1	0	1	5	25	4.6
Total	2	75	125	97	37	3	76	128	543	100.0

type 2A. Eighty-three per cent (158) of *Shigella sonnei* infections were further typed, of which 50% were type A.

Person to person transmission and acquisition of infection overseas are the major modes of *Shigella* infections. OzFoodNet did not identify any *Shigella* outbreaks associated with food in 2006.[4]

Shiga toxin producing/verotoxigenic *Escherichia coli*

Case definitions – Shiga toxin-producing/ verotoxin-producing Escherichia coli *(STEC/ VTEC)*

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Shiga-toxigenic/ verotoxigenic* Escherichia coli *from faeces, OR, isolation of Shiga toxin or verotoxin from a clinical isolate of* E. coli *OR, identification of the gene associated with the production of Shiga toxin or vero toxin in* E. coli *by nucleic acid testing on isolate or raw bloody diarrhoea.*

Note: Where STEC/VTEC is isolated in the context of haemolytic uraemic syndrome (HUS), it should be notified as STEC/VTEC and HUS.

There were 70 cases of STEC/VTEC notified to NNDSS in 2006 a reduction of 19% on the 86 cases reported in 2005.

As in previous years, South Australia routinely tested bloody stools by polymerase chain reaction (PCR) for genes coding for Shiga toxin. Consequently, 36 of the 70 cases (51%) were notified in South Australia, which also had the highest notification rate (2.3 cases per 100,000 population). There were no cases reported from the Australian Capital Territory or Tasmania.

Cases occurred in all age groups, with 11 (15%) cases in children aged less than 5 years. There were more cases reported among men (male to female ratio 1.4:1).

Typing information was available on only 18 cases. These included 7 cases of 0157, 4 of 011 and 3 of 026.

Haemolytic uraemic syndrome

Case definitions – Haemolytic uraemic syndrome (HUS)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires acute microangiopathic anaemia on peripheral blood smear (schistocytes, burr cells or helmet cells) AND AT LEAST ONE OF THE FOLLOWING: acute renal impairment (haematuria, proteinuria or elevated creatinine level), OR, thrombocytopaenia, particularly during the first seven days of illness.*

Note: Where STEC/VTEC is isolated in the context of HUS, it should be notified as both STEC/VTEC and HUS.

In 2006, 13 cases of HUS were reported to NNDSS (a 35% decrease on the 20 cases reported in 2005). Cases were reported mainly from New South Wales (11 cases) with single cases reported from South Australia and Victoria.

Of the 13 cases of HUS notified in 2006, 6 were males and 7 females. The median age for both sexes was 5 years with an age range of 1 to 60 years. STEC was isolated in 3 cases of HUS and the serotype of 1 (an 055) was identified. In New South Wales, all cases of HUS were interviewed but no common risk factors or links between cases were identified.[4]

Typhoid

Case definitions – Typhoid fever

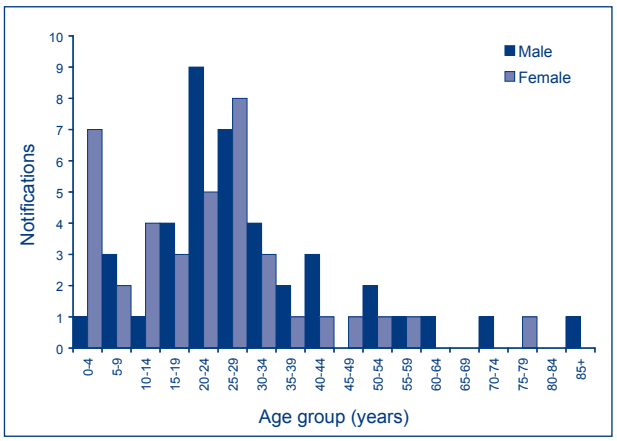
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation or detection of* Salmonella typhi.

There were 78 cases of typhoid reported in 2006, an increase of 50% on the 52 notifications in 2005. Nationally, the male to female ratio was 1.05:1, with the highest number of notifications in the 20–29 years age range (Figure 23).

OzFoodNet reported 74 cases of typhoid in 2006. Sixty-eight cases (93%) reported overseas travel; one-third of these travelled in India.[4]

Figure 23. Number of notifications of typhoid, Australia, 2006, by age group and sex



Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and nationally notifiable in Australia and to the World Health Organization in 2006 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean-Congo). HPAIH was declared a quarantinable disease on 23 March 2004 and consequently became subject to the routine quarantine powers available under the *Quarantine Act 1908*. SARS was declared a quarantinable disease under the *Quarantine Act 1908* on 7 April 2003.

In 2005, Australia committed to the International Health Regulation (IHR). These are requirements that will contribute significantly to enhancing national, regional and international public health security. During 2006, Australia was preparing for the IHR, which came into force on 15 June 2007. Under the IHR a 'decision instrument' must be utilised in order to identify whether a health-related event may constitute a public health emergency of internal concern and therefore requires formal notification to WHO.

Cholera, plague, rabies, smallpox, yellow fever, SARS, HPAIH and viral haemorrhagic fevers are of international public health importance as they continue to occur around the world. Travellers are advised to seek information on the risk of contracting these diseases in their destinations and take appropriate measures. More information on quarantinable diseases and travel health can be found on DoHA's web site at: http://www.health.gov.au/internet/main/Publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm

There were no cases of plague, rabies, smallpox, tularaemia, yellow fever, SARS, HPAIH or viral haemorrhagic fever reported in Australia in 2006.

Cholera

Case definition – Cholera

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of toxigenic* Vibrio cholerae *O1 or O139.*

In 2006, there were 3 cases of locally acquired cholera notified in Australia that were part of a local cluster in Sydney, New South Wales, in November. All cases were elderly women (aged 71, 71 and 84) infected with toxin-producing *Vibrio cholerae* O1 Ogawa El Tor. Investigations by the NSW Health Department found that the only common exposure among the 3 women was consumption of raw whitebait that was imported from Indonesia. As a result, a media release advising people to avoid eating raw whitebait was issued. No additional cases of cholera were discovered, and the 3 women all recovered.

Apart from 1 case of laboratory acquired cholera in 1996 and the 3 cases in 2006, all other cases of cholera reported since the commencement of the NNDSS in 1991 have been acquired outside Australia. There have been 17 cases notified over the last 5 years (ranging from 1 case in 2003 to 5 cases in both 2002 and 2004).

Sexually transmissible infections

In 2006, sexually transmissible infections (STIs) reported to NNDSS were chlamydial infection, donovanosis, gonococcal infections and syphilis. Two categories of adult syphilis have been reported since 2004: syphilis – infectious (primary, secondary and early latent) less than 2 years duration and syphilis – of greater than 2 years or unknown duration. Reports were also received by NNDSS on congenital syphilis. These conditions were notified in all states and territories.

Other national surveillance systems that monitor STI in Australia include the Australian Gonococcal Surveillance Programme, which is a network of specialist laboratories, and the National Centre in HIV Epidemiology and Clinical Research.

The national trends in the number and rates of STI notifications reported to NNDSS between 2001–2006 are shown in Table 4a. In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence. Increases in screening rates,[5,6] more targeted screening, the use of more sensitive diagnostic tests, as well as periodic public awareness campaigns may contribute to changes in the number of notifications over time.

Age standardised notification rates were calculated for Indigenous and non-Indigenous populations for jurisdictions that had indigenous status data completed in more than 50% of notifications. These data however, have to be interpreted cautiously as STI screening occurs disproportionately among Indigenous populations. Similarly, rates between females and males need to be interpreted cautiously as rates of testing for STI differ between the sexes.

Chlamydial infection

Case definition – Chlamydial infection

Only ***confirmed cases*** *are reported.*

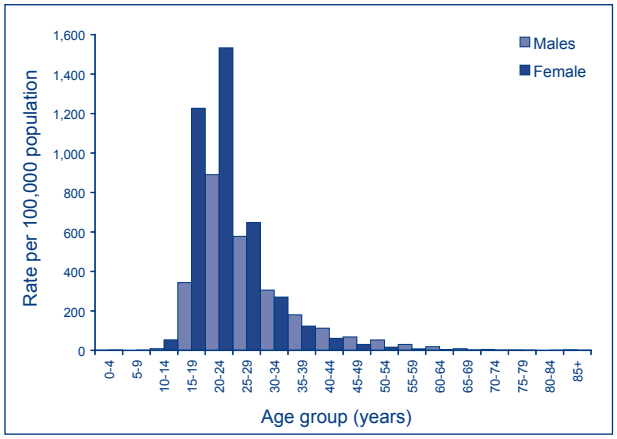
Confirmed case: *Isolation of* Chlamydia trachomatis *or detection of* Chlamydia trachomatis *by nucleic acid testing or detection of* Chlamydia trachomatis *antigen.*

In 2006, chlamydial infection continued to be the most commonly notified disease. A total of 46,954 notifications of chlamydial infection were received; a rate of 228 cases per 100,000 population. This represents an increase of 12% on the rate reported in 2005 (203 cases per 100,000 population). The rate of chlamydial infection notifications has continued to increase since surveillance of the condition commenced in 1991. Between 2002 and 2006, chlamydial infection notification rates increased from 124 to 228 cases per 100,000 population, an increase of 79% (Table 4a). This ongoing increase provided impetus for the launch of Australia's first National STI Strategy in July 2005.[7] While the prevalence of chlamydia varies by age group and other demographic and behavioural factors, no major section of the population is spared.[8]

Chlamydial infection notification rates were higher than the national average (228 cases per 100,000 population) in the Northern Territory (995 cases per 100,000 population), Queensland (302 cases per 100,000 population), Western Australia (288 cases per 100,000 population), and the Australian Capital Territory (250 cases per 100,000 population) (Table 3). At a regional level, the Northern Territory excluding Darwin had the highest chlamydial infection notification rate at 1,959 cases per 100,000 population (Map 3).

In 2006, notification rates of chlamydial infection in males and females were 185 and 270 cases per 100,000 population, respectively. In 2006, notification rates increased by 11% in males and by 12% in females when compared with 2005. The male to female ratio in 2006 was 1:1.5, which is similar to previous years. Rates in females exceeded those in males in the 0–29 years age range but were higher in males in the 30 years or more age range (Figure 24).

Figure 24. Notification rate of chlamydial infections, Australia, 2006, by age group and sex



Trends in age and sex notification rates between 2002 and 2006 show increases in all age groups between 10 and 39 years in both males and females (Figure 25). Between 2002 and 2006, the notification rate in males in the 20–24 years age group increased by 433.5 cases per 100,000 population. In females of the same age, the notification rate increased by 732.2 cases per 100,000 population.

In 2006, data on indigenous status was complete in 43% of cases of chlamydia infection and this is comparable to the preceding 5-year indigenous status completeness average of 43% (range: 40%–44%). The combined chlamydial infection notifications in 5 jurisdictions with greater than 50% completeness of indigenous status (Northern Territory, South Australia, Victoria, Tasmania and Western Australia) showed that in 2006, the age adjusted notification rate was 1,250 cases per 100,000 population, and 223 cases per 100,000 non-Indigenous population (Figure 26). During 2006, the age standardised ratio

Map 3. Notification rates of chlamydial infection, Australia, 2006, by Statistical Division

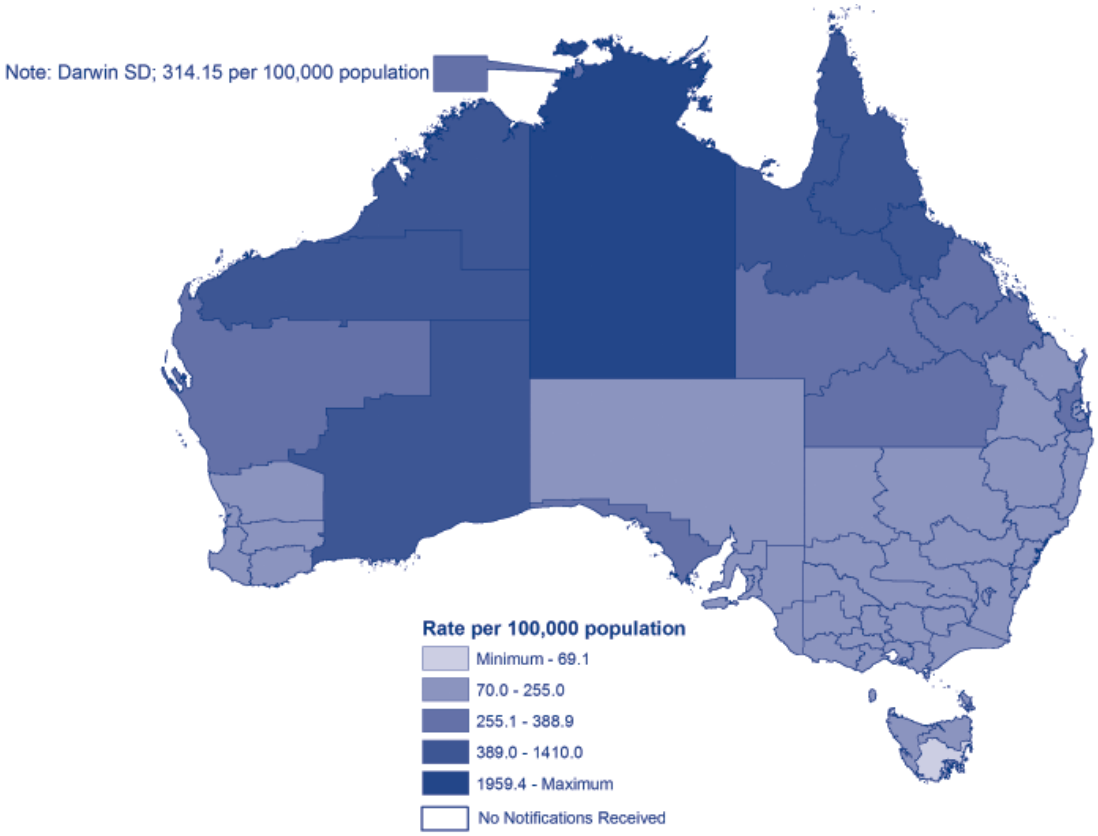


Figure 25. Trends in notification rates of chlamydial infection in persons aged 10–39 years, Australia, 2002 to 2006, by age group and sex

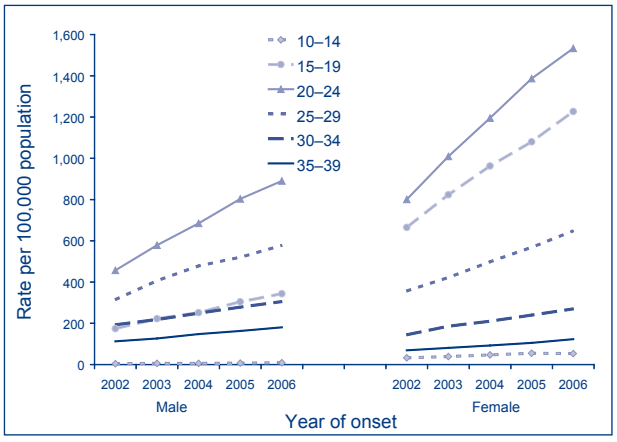
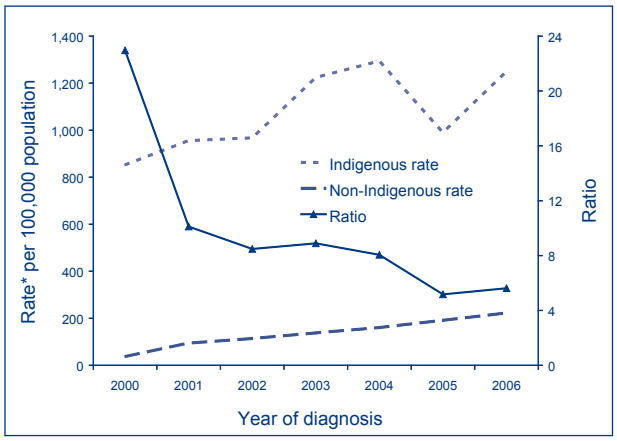


Figure 26. Trends in age standardised notification rate of chlamydial infections, the Northern Territory, South Australia, Tasmania, Western Australia, and Victoria, 2000 to 2006, by indigenous status



* The rates in non-Indigenous peoples include diagnoses in people whose Indigenous status was not reported.

of Indigenous to non-Indigenous chlamydial infection was 5.6:1 and this gap has increased slightly from 2005 (5.2:1), but since 2000, has improved significantly (ratio range: 8–23).

Donovanosis

Case definition – Donovanosis

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires demonstration of intracellular Donovan bodies on smears or biopsy specimens taken from a lesion or detection of* Calymmatobacterium granulomatis *by nucleic acid testing of a specimen taken from a lesion AND clinically compatible illness involving genital ulceration.*

***Probable case:** Requires compatible sexual risk history in a person from an endemic area or a compatible sexual risk history involving sexual contact with someone from an endemic area.*

Donovanosis is a sexually transmissible infection characterised by a chronic ulcerative genital disease. Although uncommon, it is a disease of public health importance in Australia because it predominantly occurs in Indigenous communities. It has been identified as a potential co-factor in HIV transmission, and it is preventable.[9] Donovanosis is targeted for elimination from Australia through the donovanosis elimination project. In 2006, 4 cases of donovanosis (3 male and 1 female) were reported to NNDSS. Cases were reported from the Northern Territory (2) and Queensland (2). All 4 cases were among Indigenous people. In 2005, a total of 13 cases, 11 Indigenous, 4 male and 9 female, were notified (Figure 27). Cases in 2006 were aged 30, 31, 37 and 58 years.

Gonococcal infections

Case definition – Gonococcal infection

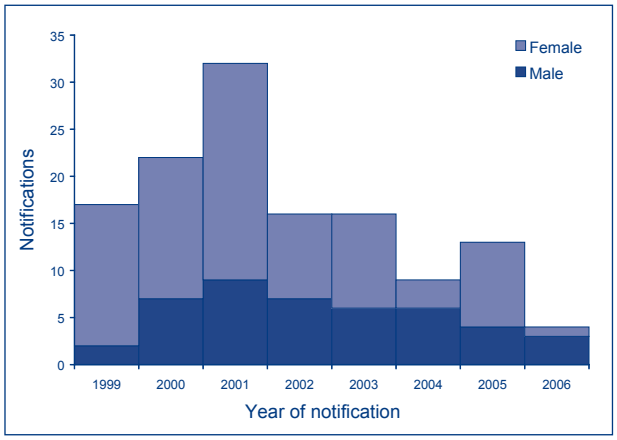
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Neisseria gonorrhoeae, *or detection of* Neisseria gonorrhoeae *by nucleic acid testing or detection of typical Gram-negative intracellular diplococci in a smear from a genital tract specimen.*

In 2006, 8,547 notifications of gonococcal infection were received by NNDSS. This represents a rate of 41.5 cases per 100,000 population, an increase of 4% from the rate reported in 2005 (39.8 cases per 100,000 population). The male to female ratio in 2006 was 2:1, unchanged in the previous 5 years (2001 to 2005) and reflecting ongoing transmission among men who have sex with men (MSM) in Australia's larger cities.[2]

The highest notification rate in 2006 was in the Northern Territory at 860 cases per 100,000 population (Table 3). The largest increase in the notification rate in 2006 (compared with 2005) occurred in South Australia, where a 24% overall increase in notification rates was reported. Notification rates in Tasmania decreased by 49% compared with 2005. In 2006 nationally, gonococcal infection rates for males and females were 57 and 26 cases per 100,000 population respectively. The exception to this pattern was the Northern Territory, where females had higher notification rates than males (929 versus 796 cases per 100,000 population). The regional distribution of gonococcal infection notification shows that the highest rate occurred in the Northern Territory excluding Darwin at 75 per 100,000 population.

Figure 27. Number of notifications of donovanosis, Australia, 1999 to 2006, by sex



Notification rates of gonococcal infection in males exceeded those in females in all age groups except in the 10–14 and 15–19 years age groups (Figure 28).

Trends in sex specific notification rates show that an increase in the rates in males in the 20–24 and 25–29 years age groups has continued. However, in 2006 the sex specific notification rates for the males in the 15–19 years age group decreased. In females, an increase occurred in the 20–24 and 30–34 years age groups, and there was a slight decrease in the 15–19 years age group (Figure 29).

In 2006, the data completeness of indigenous status of gonococcal infection notifications was 68%; the same as in 2005. The combined gonococcal infection notifications of 6 jurisdictions with more than 50% data completeness of indigenous status (Northern Territory, Queensland, South Australia, Western Australia, Tasmania and Victoria) shows that in 2006, the age adjusted notification rate in the Indigenous population was 1,206.1 cases per 100,000 population and 24.1 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 50:1.

Figure 28. Notification rate of gonococcal infection, Australia, 2006, by age group and sex

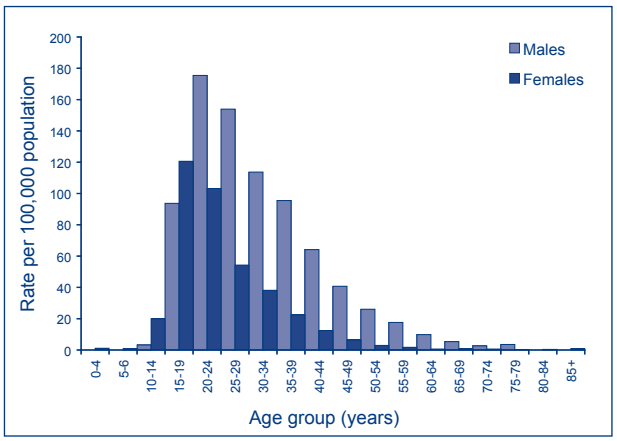
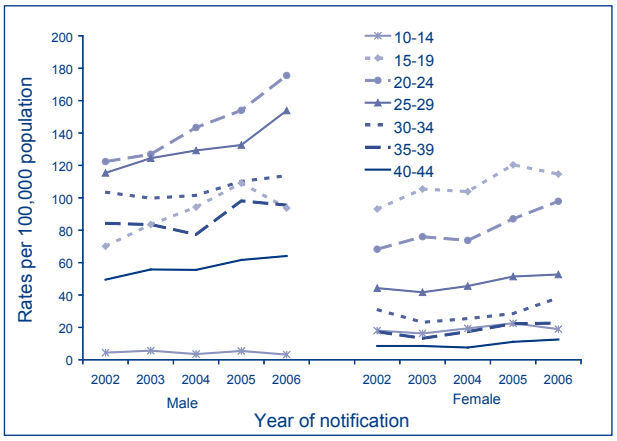


Figure 29. Trends in notification rates of gonococcal infection in persons aged 10–44 years, Australia, 2002 to 2006, by age group and sex



Other surveillance of gonococcal infections

The Australian Gonococcal Surveillance Programme (AGSP) is the national surveillance system of antibiotic susceptibility of gonococcal isolates. In each state and territory, a network of reference laboratories determines the susceptibility of isolates to a core group of antibiotics using a standard methodology. The following is the summary of their 2006 report.

In 2006, a total of 3,850 isolates of gonococci were tested for antibiotic susceptibility. Eighty-four per cent of isolates were from men (mainly MSM), of which 75% were obtained from the urethra, 15% from the rectum and 9% from the pharynx. In females, 93% of isolates were obtained from the cervix. Proportions for site of infection were similar to those reported in 2005.

Trends in the proportion of isolates resistant to penicillin, quinolones and tetracycline are shown in Table 10.

In 2006, the proportion of isolates resistant to penicillin by plasmid mediated resistance decreased by 5% and the proportion of isolates resistant to penicillin by chromosomally mediated increased by 24% compared with 2005. Quinolone resistance also increased by 23% to 37.8% from 30.6% in 2005 (Figure 30).

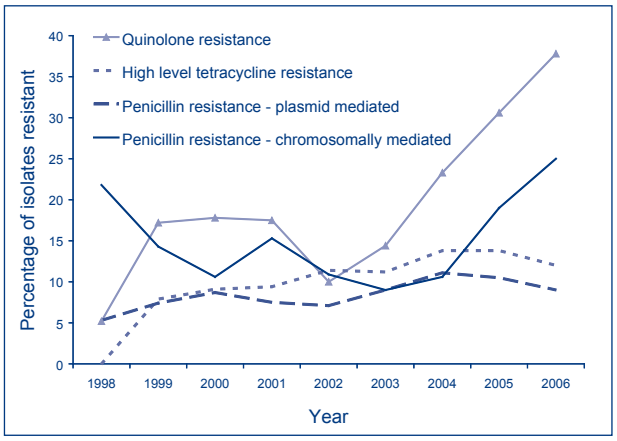
Information on the country where resistant strains were acquired were available in 23% of infections by strains with plasmid mediated resistance to penicillin and 22% of infections by strains resistant to quinolone. This showed that 43% (34/80) of plasmid mediated resistance were locally acquired with the rest acquired from Western Pacific countries and South East Asia. Eighty-one per cent of quinolone resistant strains were acquired locally and the remaining from overseas.

Table 10. Proportion of gonococcal isolates showing antibiotic resistance, Australia, 1998 to 2006

Year	Penicillin resistance (% resistant)		Quinolone resistance (% resistant)	High level tetracycline resistance (% resistant)
	Plasmid mediated	Chromosomally mediated		
1998	5.3	21.8	5.2	NR
1999	7.4	14.3	17.2	7.9
2000	8.7	10.6	17.8	9.1
2001	7.5	15.3	17.5	9.4
2002	7.1	10.9	10.0	11.4
2003	9.0	9.0	14.4	11.2
2004	11.1	10.6	23.3	13.8
2005	10.5	19.0	30.6	13.8
2006	9.0	25.0	37.8	12.0

NR Not resistant

Figure 30. Trend in percentage of gonococcal isolates showing antibiotic resistance, Australia, 1998 to 2006



Resistance to both the penicillin and quinolone groups of antibiotics has reached historical highs. Nationally, one third of gonococci were penicillin resistant by at least one mechanism, and a slightly higher proportion was quinolone resistant.

The distribution of infections with strains resistant to different antibiotic agents varies from jurisdiction to jurisdiction and urban to rural areas within each jurisdiction. The AGSP recommends that treatment regimes should be tailored to the local patterns of susceptibility.

Syphilis (all categories)

In 2004, all jurisdictions began reporting to NNDSS adult syphilis infections categorised as: infectious syphilis of less than 2 years duration, and syphilis of more than 2 years or unknown duration. Detailed analyses are reported for the 2 categories, as well as for syphilis of *all categories* for the purpose of showing trends in keeping with reports in previous years.

In 2006, a total of 2,436 cases of syphilis infection of all categories was reported, representing a notification rate of 11.8 cases per 100,000 population, an increase of 8.2% on the 10.9 cases per 100,000 population reported in 2005 (Table 4a, Figure 31). The Northern Territory continued to have the highest notification rate of syphilis (130 cases per 100,000 population), an increase of 14.7% from the previous year. South Australia reported an increase in the notification rate of syphilis of 133.0% compared with 2005. There were also increases in notification rates in Victoria (19.4%), Queensland (9.0%) and New South Wales (2.4%) and decreases in notification rates in Tasmania (27.2%) and Western Australia (by 11.2%) and the Australian Capital Territory (1.1%). At the regional level, the highest notification rate was in the Northern Territory excluding Darwin at 284 cases per 100,000 population (Map 4). As in other developed countries syphilis infection rates are rising in Australia among men who have sex with men.[10,11]

Map 4. Notification rates of syphilis infection, Australia, 2006, by Statistical Division of residence

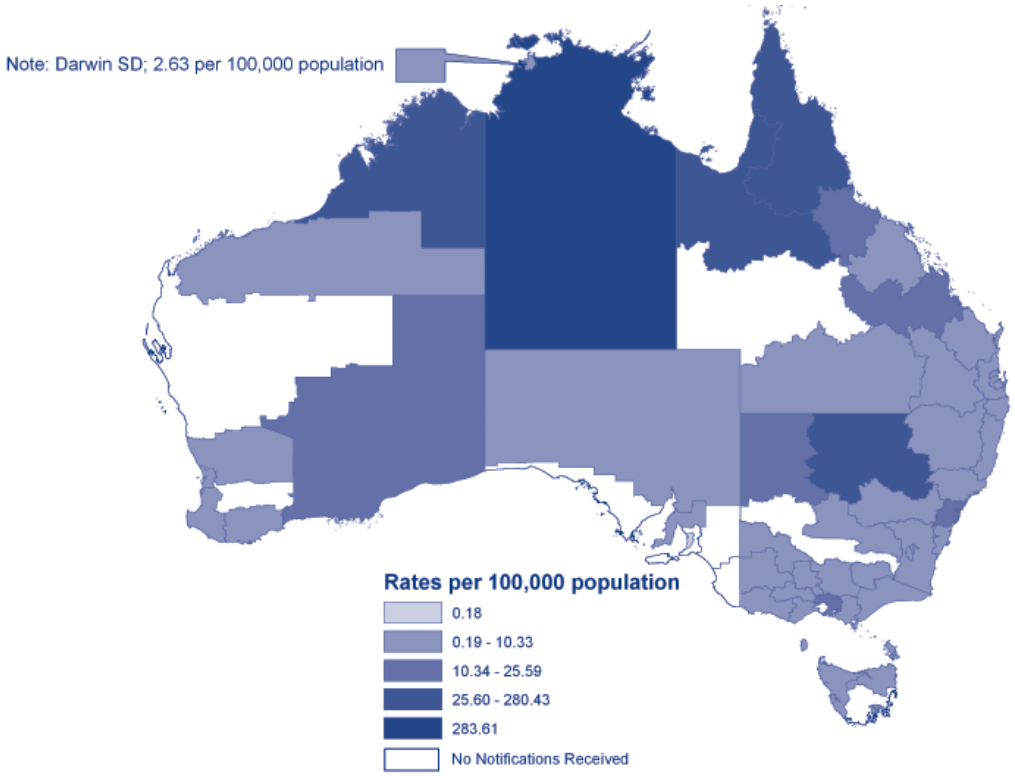
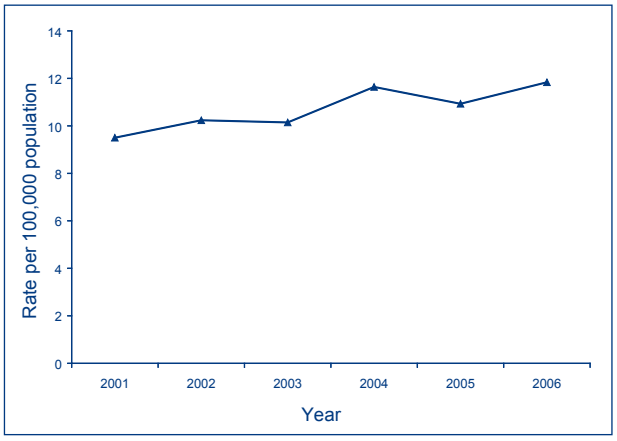


Figure 31. Notification rate of syphilis infection, Australia, 2001 to 2006



Syphilis – infectious (primary, secondary and early latent), less than 2 years duration

Case definition – Syphilis – infectious (primary, secondary and early latent), less than 2 years duration

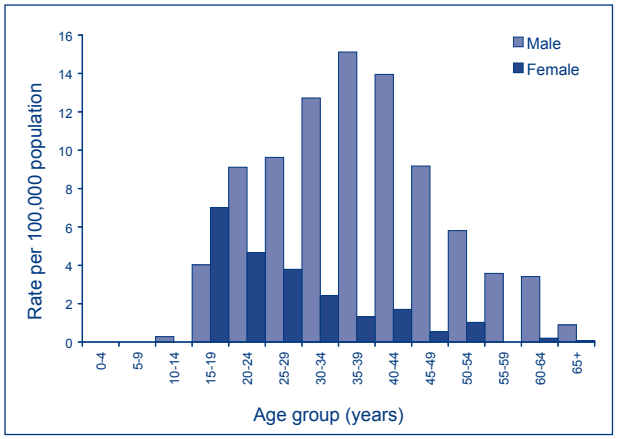
Only ***confirmed cases*** *are reported.*

Confirmed case: *Requires seroconversion in past 2 years (specific treponemal test (e.g. IgG enzyme immunoassay,* Treponema pallidum *haemagglutination assay,* Treponema palladium *particle agglutination,* Treponema pallidum *immobilisation assay), or fluorescent treponemal antibody absorption reactive when previous treponemal test non-reactive within past 2 years OR a fourfold or greater rise in non-specific treponemal antibody titre (e.g. Venereal Diseases Research Laboratory, Rapid Plasma Reagin) in the past 2 years, and a reactive specific treponemal test (e.g. IgG enzyme immunoassay,* Treponema pallidum *haemagglutination assay,* Treponema pallidum *particle agglutination,* Treponema pallidum *immobilisation assay, or fluorescent treponemal antibody absorption) OR demonstration of* Treponema pallidum *by darkfield microscopy (not oral lesions), direct fluorescent antibody tests, equivalent microscopic methods (e.g. silver stains), or nucleic acid testing or non-specific treponemal test (e.g. Venereal Diseases Research Laboratory, Rapid Plasma Reagin) reagin titre of greater than or equal to 1:8 AND presence of a primary chancre (or ulcer) or clinical signs of secondary syphilis.*

In 2006, a total of 813 cases of syphilis of less than 2 years duration were reported. This represents a notification rate of 3.9 cases per 100,000 population, an increase of 25.8% compared with 2005 (Table 4a). The Northern Territory had the highest notification rate at 72.6 cases per 100,000 population in 2006, an increase of 56.5% compared with 2005. Western Australia reported an increase in the notification rate for infectious syphilis of 142% compared with 2005. Increases in notifications also occurred in Victoria (88.3%), and Queensland (16.9%) and decreases occurred in South Australia (77.8%), the Australian Capital Territory (50.5%), Tasmania (17.3%) and New South Wales (14.2%) (Table 3).

The notification rates of syphilis of less than 2 years duration for males and females were 6.2 and 1.7 cases per 100,000 population respectively (Table 11). Notification rates were higher in males than in females in all jurisdictions, except in the Northern Territory where rates were higher in females (89.3 versus 57.7 cases per 100,000 population). Nationally, the male to female ratio was 3.6:1. Notification rates in males peaked in the 35–39 years age group (15.1 cases per 100,000 population) and in females in the 15–19 years age group (7 cases per 100,000 population) (Figure 32).

Figure 32. Rates of notification of syphilis of less than 2 years duration, Australia, 2006, by age group and sex



Over the period 2004 to 2006 notification rates have increased in most age groups for both males and females. In 2006, the largest increase in males occurred in the 40–49 years age group and in females in the 10–19 years age group (Figure 33). Increases in notifications of infectious syphilis occurred mainly in homosexual men.[2]

Data on indigenous status was complete in 95% of cases of syphilis of less than 2 years duration. The age adjusted notification rate was 47.9 cases per 100,000 Indigenous population, and 3.1 cases per 100,000 non-Indigenous population, a ratio of Indigenous to non-Indigenous of 15:1. Age specific notification rates show that compared with the non-Indigenous population, rates of syphilis of less than 2 years duration in the Indigenous population are in an order of magnitude higher and peak in a younger age group (Figure 34).

Table 11. Number and rates of notifications of syphilis of less than 2 years duration Australia, 2006, by state or territory and sex

	Male		Female		Total	
	n	Rate	n	Rate	n	Rate
ACT	1	0.6	1	0.6	2	0.6
NSW	190	5.6	19	0.6	210	3.1
NT	63	57.7	87	89.3	150	72.6
Qld	137	6.8	28	1.4	165	4.1
SA	2	0.3	0	0.0	4	0.3
Tas.	4	1.7	1	0.4	5	1.0
Vic.	208	8.3	23	0.9	231	4.5
WA	34	3.3	14	1.4	48	2.3
Total	639	6.2	173	1.7	813*	3.9

* Sex unknown for one case.

Figure 33. Rates of notification of syphilis of less than 2 years duration, Australia, 2004 to 2006, by age group and sex

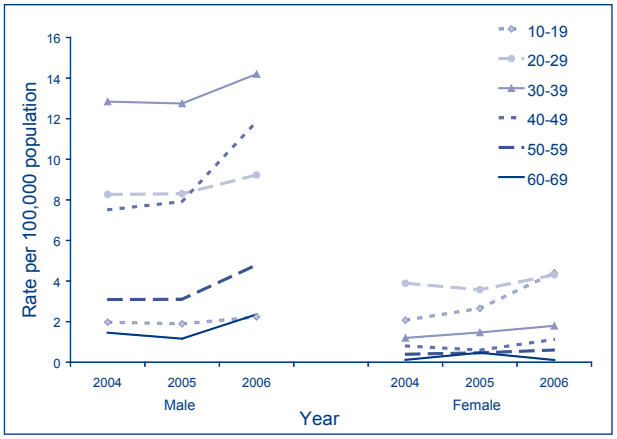
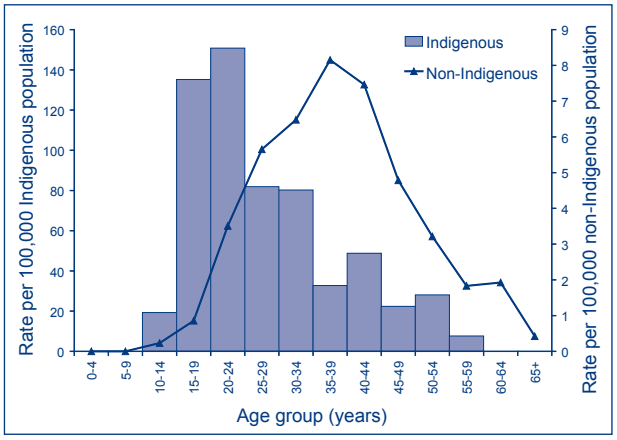


Figure 34. Notification rate of syphilis of less than 2 years duration, Australia, 2006, by indigenous status



Syphilis of more than 2 years or unknown duration

Case definition – Syphilis of more than two years or unknown duration

*Only **confirmed cases** are reported.*

***Confirmed case:** Does not meet the criteria for a case of less than 2 years duration AND either a reactive specific treponemal test (e.g. IgG enzyme immuno-assay,* Treponema pallidum *haemagglutination assay,* Treponema pallidum *particle agglutination,* Treponema pallidum *immobilisation assay, or fluorescent treponemal antibody absorption) which is confirmed either by a reactive non-specific treponemal test (e.g. Venereal Diseases Research Laboratory, Rapid Plasma Reagin) OR a different specific treponemal test if the non-specific treponemal test is non-reactive AND the absence of a history of documented previous adequate treatment of syphilis, or endemic treponemal disease (e.g. Yaws).*

In 2006, a total of 1,623 cases of syphilis of more than 2 years or unknown duration were reported: a notification rate of 7.9 cases per 100,000 population. The Northern Territory had the highest notification rate at 57.6 cases per 100,000 population.

In 2006, notification rates of syphilis of more than 2 years or unknown duration in males and females were 10.1 and 5.6 cases per 100,000 population, respectively (Table 12). Notification rates were higher in males than in females in all jurisdictions, except in the Northern Territory, where males had a higher rate than females (53.1 and 57.6 cases per 100,000 population, respectively). Nationally, the male to female ratio was 1.8:1. Notification rates in males and females were similar in the younger age groups up to 30–34 years. In females, the rate peaked in the 30–34 years age group while in males it remained high from 35 years (Figure 35).

Figure 35. Notification rate of syphilis of more than 2 years or unknown duration, Australia, 2006, by age group and sex

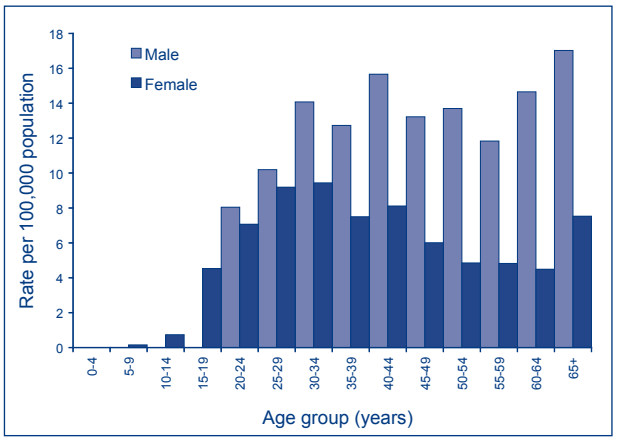
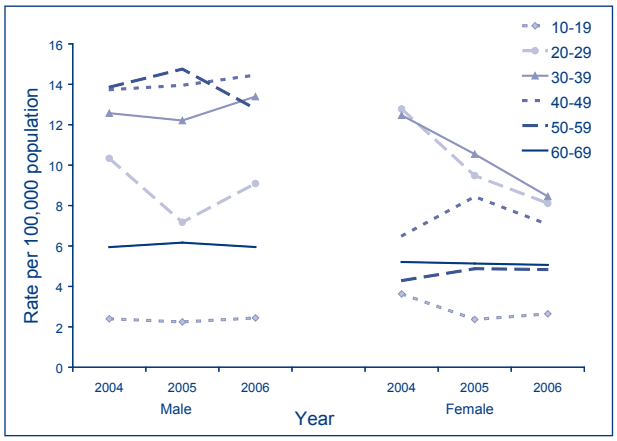


Figure 36. Rates of notification of syphilis of more than 2 years or unknown duration, Australia, 2004–2006, by age group and sex



Over the period 2004 to 2006 notification rates have remained stable in most age groups for both males and females, except in females aged 20 to 39 years, which have shown a large decrease. In 2006, the largest increase in rates occurred in males in the 20–29 and 30–39 years age groups (Figure 36).

Data on indigenous status were complete in 69.2% of cases of syphilis of more than 2 years or unknown duration. The combined age adjusted rate for the jurisdictions with greater than 50% data completeness of indigenous status (all jurisdictions except the Australian Capital Territory) was 78 cases per 100,000 Indigenous population, and 7 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 12:1. Age specific notification rates showed a similar pattern with age and no single distinct peak for either Indigenous or non-Indigenous groups. Overall, rates in the Indigenous population were higher than those in the non-Indigenous by an order of magnitude (Figure 37).

Figure 37. Notification rate of syphilis of more than 2 years or unknown duration, Australia, 2006, by indigenous status

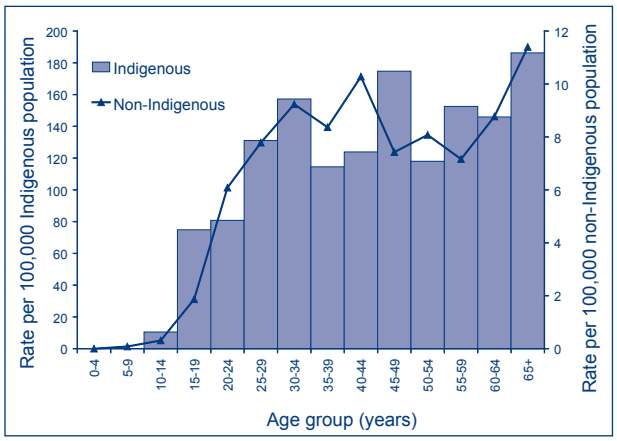


Table 12. Number and rates of notifications of syphilis of more than 2 years or unknown duration, Australia, 2006, by state or territory and sex

	Male		Female		Total	
	n	Rate	n	Rate	n	Rate
ACT	8	4.9	4	2.4	12	3.6
NSW	439	12.9	225	6.6	666	9.8
NT	58	53.1	61	62.6	119	57.6
Qld	166	8.2	105	5.2	271	6.7
SA	33	4.3	8	1.0	41	2.6
Tas.	11	4.6	6	2.4	17	3.5
Vic.	246	9.8	117	4.5	366	7.2
WA	74	7.2	57	5.6	131	6.4
Total	1,035	10.1	583	5.6	1,623*	7.9

* Sex unknown for 5 cases.

Syphilis – congenital

Case definition – Congenital syphilis

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires treponemal-specific antibody titres (e.g.* Treponema pallidum *haemagglutination assay, pallidum particle agglutination, fluorescent treponemal antibody absorption in infant serum greater than fourfold higher than in maternal serum OR treponemal specific antibody titres in infant serum comparable with those in maternal serum and specific treponemal IgM enzyme-linked immunosorbent assay or immunofluorescence assay positive OR* T. pallidum *DNA in normally sterile specimen from infant (CSF, tissue) by nucleic acid testing.*

OR dark field microscopy of infant lesion exudate or node aspirate smears (not oral lesions) to demonstrate characteristic morphology and motility of T. pallidum *OR demonstration of* T. pallidum *in infant tissues by special (e.g. silver) stains OR detection of* T. pallidum *DNA from an infant non-sterile site by nucleic acid testing OR reactive fluorescent treponemal absorbed-19S-IgM antibody test or IgM enzyme linked immunosorbent assay and Treponemal non-specific antibody titre (e.g. RPR) in infant serum greater than fourfold higher than in maternal serum AND asymptomatic infection (in the infant of an infected mother) OR foetal death in utero OR stillbirth, which is a foetal death that occurs after a 20-week gestation or in which the foetus weighs greater than 500 g and the mother is untreated or inadequately treated for syphilis at delivery. Inadequate treatment is a non-penicillin regimen or penicillin treatment given less than 30 days prior to delivery OR clinical evidence of congenital syphilis on examination on:*

a. age <2years: Hepatosplenomegaly, rash, condyloma lata, snuffles, jaundice (non-viral hepatitis), pseudoparalysis, anaemia, oedema

b. age >2 years: Interstitial keratitis, nerve deafness, anterior bowing of shins, frontal bossing, mulberry molar, Hutchinson teeth, saddle nose, rhagades or Clutton joints

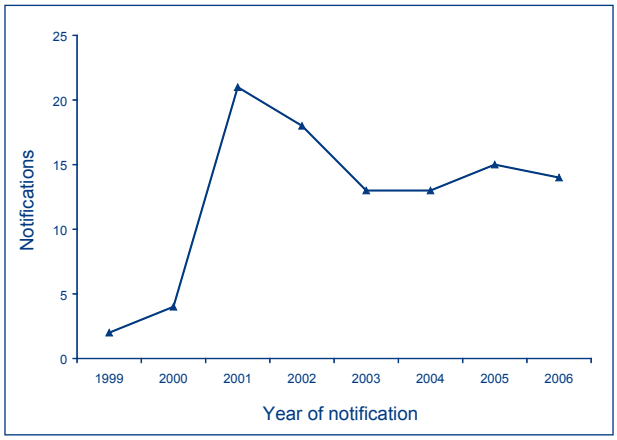
c. evidence of congenital syphilis on long bone X-ray

d. evidence of congenital syphilis on cerebrospinal fluid (CSF) examination

***Probable case:** An infant (regardless of clinical signs) whose mother has been inadequately treated for syphilis during pregnancy or an infant or child who has a reactive treponemal antibody test for syphilis and any one of the following: (1) any evidence of congenital syphilis on physical examination, (2) any evidence of congenital syphilis on radiographs of long bones, (3) a reactive cerebrospinal fluid Venereal Disease Research Laboratory Titre, (4) an elevated CSF cell count or protein (without other cause), (5) reactive fluorescent treponemal antibody absorbed assay –19S-IgM antibody test or IgM enzyme-linked immunosorbent assay.*

There were 14 cases of congenital syphilis notified in 2006, 6 males, 7 females and 1 unknown. Eight of the cases were reported in the Northern Territory, 5 in New South Wales, and 1 in Queensland. Six were Indigenous, 4 non-Indigenous and 4 were unknown. Notifications of congenital syphilis have plateaued over the last 4 years following a decline from a peak in 2001 (Figure 38). In the Northern Territory where rates of infectious syphilis of less than 2 years duration are highest, the highest number of cases of congenital syphilis continue to be reported.

Figure 38. Trends in notifications of congenital syphilis, Australia, 1999 to 2006



Vaccine preventable diseases

Introduction

This section summarises the national notification data for influenza and diseases targeted by the National Immunisation Program (NIP) in 2006. These include diphtheria, *Haemophilus influenzae* type b infection, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella, tetanus and varicella (chickenpox, shingles and unspecified). Data on hepatitis B and meningococcal disease, which are also targeted by the NIP, can be found in this report under 'Bloodborne diseases' and 'Other bacterial infections'. Other vaccine preventable diseases (VPDs) presented in this report include hepatitis A and Q fever.

Major changes to the funded Australian NIP Schedule in November 2005 included:

- inactivated poliovirus vaccine (IPV) replaced oral poliovirus vaccine (OPV) for all age groups. All IPV-containing combination vaccines include diphtheria-tetanus-acellular pertussis (DTPa) antigens (i.e. quadrivalent vaccines) and some also include hepatitis B and/or Hib antigens

(i.e. pentavalent and hexavalent vaccines). The specific combination vaccines administered at 2, 4, and 6 months of age vary between states and territories but all provide DTPa-IPV quadrivalent vaccine at 4 years of age.

- varicella vaccine was added to the NIPS as a single dose due at 18 months (for children born on or after 1 May 2004) or at 12–13 years of age.

In 2006, rotavirus (Rotateq® and Rotarix®) and human papilloma virus (HPV) (Gardasil®) vaccines were registered by the TGA and became available in the private market throughout Australia. In October 2006, the Northern Territory commenced a funded rotavirus immunisation program for infants. Both rotavirus and HPV vaccines were added to the funded NIP Schedule during 2007.

There were 22,240 notifications of vaccine preventable diseases in 2006 (16% of total notifications. This was significantly more than the 17,775 notifications of vaccine preventable diseases (VPDs) reported in 2005 due to the addition of varicella infections as notifiable diseases in 2006. Pertussis was the most commonly notified VPD (10,998, 49% of all VPD notifications). Numbers of notifications and notification rates for VPDs in Australia are shown in Tables 2 and 3.

Diphtheria

Case definition – Diphtheria

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolations of toxigenic* Corynebacterium diphtheriae *or toxigenic* C. ulcerans.

***Probable case:** Requires isolation of* Corynebacterium diphtheriae *or* C. ulcerans *(toxin production unknown) and pharyngitis/laryngitis or toxic symptoms OR clinical symptoms and epidemiological links with laboratory confirmed case.*

There were no cases of diphtheria reported in 2006. The last case of diphtheria reported in Australia was a case of cutaneous diphtheria in 2001, which was the only case reported since 1992. Immunity to diphtheria measured in a national serosurvey in the late 1990s in Australia, showed high levels in people aged less than 30 years and declining immunity with increasing age.[12] High levels of immunisation are needed to protect Australians against diphtheria when travelling in the 21 countries where the disease is still prevalent (http://www.who.int/immunization_monitoring/en/globalsummary/timeseries/tsincidencedip.htm)

Haemophilus influenzae type b disease

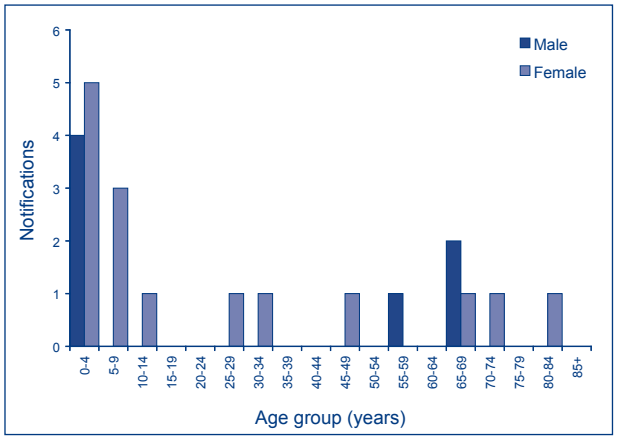
Case definition – Haemophilus influenzae *type b*

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Haemophilus influenzae *type b (Hib) from a sterile site OR detection of Hib antigen in cerebrospinal fluid consistent with meningitis.*

There were 22 notifications of *Haemophilus influenzae* type b (Hib) disease in 2006, a rate of 0.1 case per 100,000 population. This was 5 more cases than reported in 2005. Nine cases (41% of total) were in children aged less than 5 years and 3 were infants aged less than 1 year. There were 7 cases in males and 15 cases in females, (male:female ratio 0.46:1), unlike in 2005 when the ratio was 1.8:1 (Figure 39).

Figure 39. Number of notifications of *Haemophilus influenzae* type b infection, Australia, 2006, by age group and sex



Indigenous status was recorded for 18 of the 22 cases; 7 were Indigenous and 11 were non-Indigenous. The Hib notification rate was 1.4 cases per 100,000 in Indigenous people and 0.07 cases per 100,000 in non-Indigenous people; a ratio of 20:1. Between 2001 and 2005, Hib notification rates in Indigenous people have been between 4.6 and 8.6 times the rates in non-Indigenous people except in 2002 when the Indigenous rate was 25 times that of the non-Indigenous rate (Figure 40).

Cases under the age of 15 years were eligible for Hib vaccination in infancy. Of the 13 cases in 2006, 4 were unvaccinated, 1 partially vaccinated and 8 were fully vaccinated. Four of the fully vaccinated cases aged 5 years or less had received 2 or 3 validated doses of vaccine and met the case definition for vaccine failure.

Australia now has one of the lowest rates of Hib in the world after nearly 20 years of Hib vaccination.[13]

Figure 40. Notification rate of *Haemophilus influenzae* type b infection, Australia, 2001 to 2006, by indigenous status

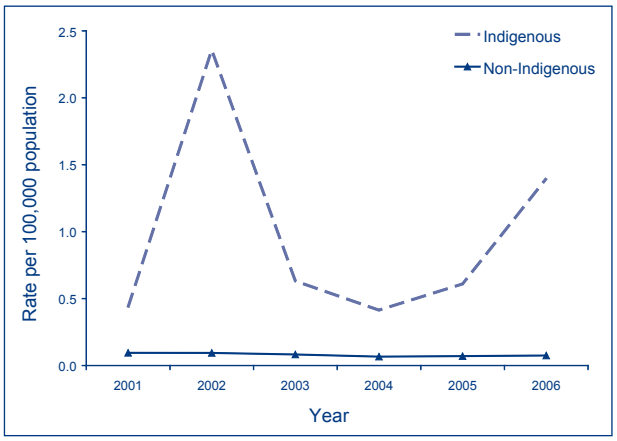
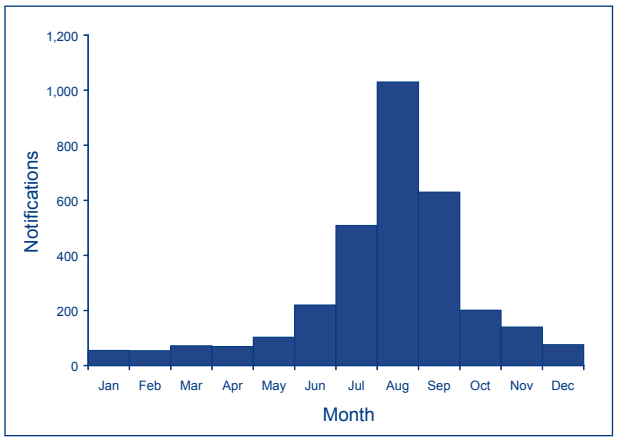


Figure 41. Number of notifications of laboratory confirmed influenza, Australia, 2006, by month of onset



Influenza (laboratory confirmed)

Case definition – Influenza

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of influenza virus by culture OR detection of influenza virus by nucleic acid testing OR detection of influenza virus antigen from an appropriate respiratory tract specimen OR a significant increase in antibody levels, or IgG sero-conversion or fourfold or greater rise in antibody titre or a single high titre antibody.*

Influenza notifications in 2006 were approximately one third lower than in 2005, and have been reported in detail separately.[14] There were 3,159 reports of laboratory-confirmed influenza in 2006, a rate of 15.3 cases per 100,000 population. Notifications of influenza showed a peak in August (Figure 41).

Peak rates in Queensland were substantially higher in August than in other states (174 cases per 100,000 population against 60 cases per 100,000 population for all Australia, Figure 42). Higher reporting rates in Queensland may be a product of the active promotion of influenza laboratory testing requests from general practitioners by public health authorities (Amy Sweeny, personal communication). There was an outbreak of influenza in an aged care facility in the Australian Capital Territory in November 2006[14] which accounts for the peak in notification rates (Figure 42).

There were 654 notifications in children aged less than 5 years (21% of all notifications). As in previous years, influenza notification rates were remarkably higher in children under 5 years compared with older age groups (notification rate of 51.3 cases per 100,000 population) (Figure 43). The rate was highest in those under 1 year of age (264 cases per 100,000 population) and declined progressively after that. The overall male to female ratio was 0.9:1.

Figure 42. Number of notifications of laboratory confirmed influenza, Australian Capital Territory, Queensland and Australia, 2006, by month of onset

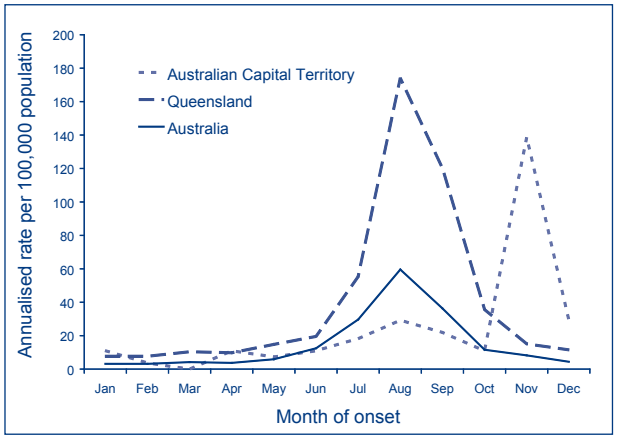


Figure 43. Notification rate of laboratory-confirmed influenza, Australia, 2006, by age group and sex

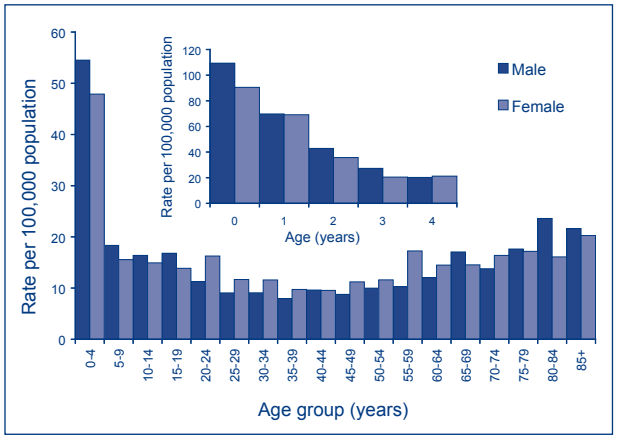


Table 13. Notification of laboratory confirmed influenza, Australia 2006, by state or territory and type

Influenza type	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Influenza A	72	420	28	1,223	34	36	354	71	2,238
Influenza B	8	150	12	406	55	10	66	102	809
Influenza A & B	0	35	0	5	0	0	1	0	41
Influenza type unknown	0	9	0	26	0	1	0	35	71
Total	80	614	40	1,660	89	47	421	208	3,159

In 2006, 3,088 (98%) influenza notifications had viral serotype data. Of these 72% (2,238) were influenza A, 26% (809) were influenza B and 1% (41) were mixed infections. A breakdown of influenza notification by virus type and jurisdiction is shown in Table 13.

Of 657 influenza virus isolates analysed at the WHO Collaborating Centre for Reference and Research on Influenza in 2006, 402 were A(H3N2), 24 were A(H1N1) strains and 231 were influenza B. Continued antigenic drift was seen within the A(H3N2) viruses from the previous reference strains (A/California/7/2004 and A/New York/55/2004) and drift was also noted in some of the A(H1N1)viruses from the reference strain A/New Caledonia/20/99. The influenza B viruses isolated were predominately of the B/Victoria lineage and similar to the reference vaccine strain B/Malaysia/2506/2004.[14]

Vaccination history was recorded in 405 cases; 50 were reported as vaccinated (31 of these were aged 65 years or older) and 355 were unvaccinated. Over 77% of Australians aged 65 years or older were vaccinated against influenza in 2006.[14]

Measles

Case definition – Measles

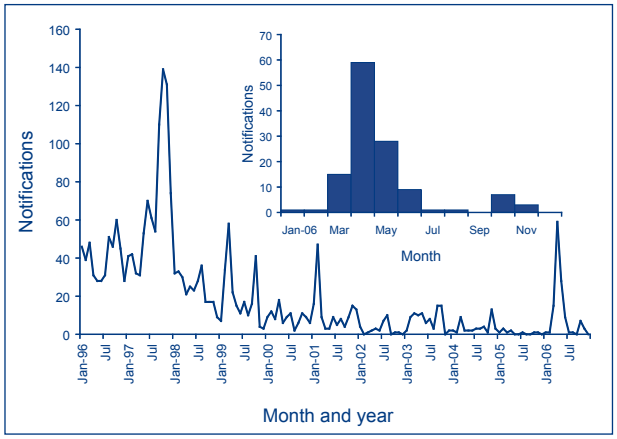
*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolation of measles virus or detection of measles virus by nucleic acid testing OR detection of measles virus antigen OR IgG seroconversion or significant increase in antibody level or fourfold or greater rise in titre or detection of measles specific IgM antibody in a reference laboratory (except when vaccinated 8 days to 8 weeks prior to testing) OR clinical illness characterised by a maculopapular rash and fever and cough, coryza, conjunctivitis or koplik spots and epidemiological link to a laboratory confirmed case.*

***Probable case:** Requires detection of measles IgM antibody in other than an approved reference laboratory and clinical illness.*

There were 125 cases of measles (0.6 cases per 100,000 population) notified in 2006; a dramatic increase on the 10 cases notified in 2005 (<0.1 cases per 100.000 population), which was the lowest annual rate for Australia since national surveillance began in 1991 (Figure 44). The increase was largely due to a multi-state outbreak in April 2006.

Figure 44. Number of notifications of measles, Australia, 1996 to 2006, by month of onset



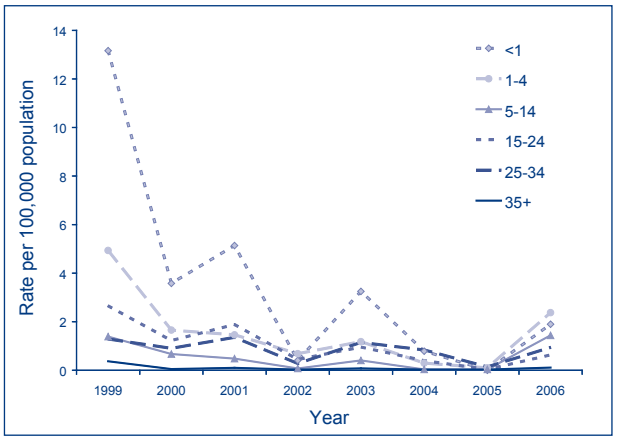
Cases were reported from all states and territories except the Northern Territory. There were 112 confirmed and 13 probable cases.

In 2006, there was a substantial increase in the number of cases in all age groups (Figure 45). There were 5 cases in children aged less than 1 year, 24 in those aged 1–4 years; 39 in the 5–14 years age group, 18 in the 15–24 years age group, 27 in the 25–34 years age group and 12 in those aged more than 35 years.

A multi-state outbreak of measles occurred in April 2006, and was associated with a touring Indian spiritual leader who visited Western Australia, New South Wales and Queensland. The index case(s) occurred in the unimmunised visitors and resulted in 82 cases, two-thirds of whom were unimmunised

and only 7% of whom were fully immunised against measles.[15] Measles virus genotyping indicated that the outbreak cases were all D8.

Figure 45. Trends in notification rates of measles, Australia, 1999 to 2006, by age group



The World Health Organization Western Pacific Region has set the year 2012 as the target for the elimination of measles. Researchers at the National Centre for Immunisation of Vaccine Preventable Diseases report that by a number of criteria, Australia is close to, or has, achieved endemic measles elimination.[16] These criteria include a low incidence of confirmed measles cases; a high proportion of the population receiving 2 doses of the measles-mumps-rubella (MMR) vaccine; serological evidence of high level immunity in the Australian population; absence of an endemic measles virus genotype; a high proportion of cases who acquired their infection outside Australia or linked to such cases; containment of outbreaks without re-establishing an endemic measles genotype and maintaining an effective reproductive number (Ro) of less than one.[16]

Case definition – Mumps

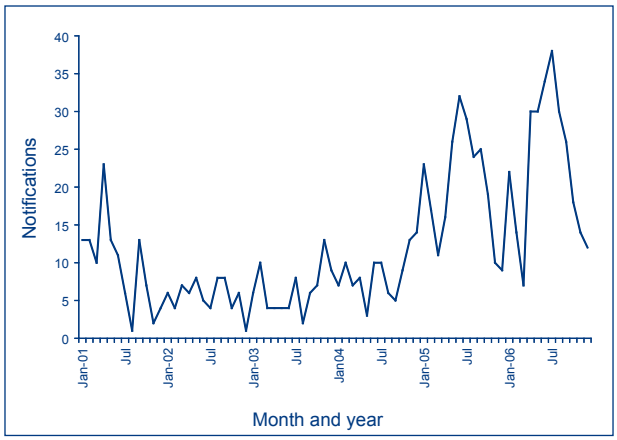
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of mumps virus or detection of mumps virus by nucleic acid testing or IgG seroconversion or significant increase in antibodies or a significant increase in antibody level, or a fourfold or greater rise in titre to mumps virus (except where there has been recent mumps vaccination) OR detection of mumps specific IgM antibody (in the absence of recent mumps vaccination) AND a clinically compatible illness characterised by swelling of the parotid or other salivary glands lasting 2 days or more without other apparent cause OR a clinically compatible illness AND an epidemiological link to a laboratory confirmed case.*

Mumps

In 2006, there were 275 notifications of mumps (1.2 cases per 100,000 population), a small increase on the 241 notifications of mumps (1.2 cases per 100,000 population), reported in 2005. The number of mumps notifications has been increasing since 2004 (Figure 46).

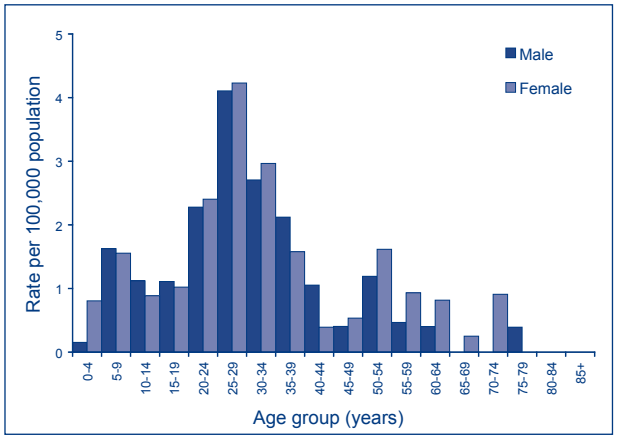
Figure 46. Number of notifications of mumps, Australia, 2001 to 2006, by month of onset



Cases were reported from all jurisdictions except Tasmania, with the largest number of cases (154) in New South Wales. There were clusters of mumps cases reported in New South Wales in 2006.

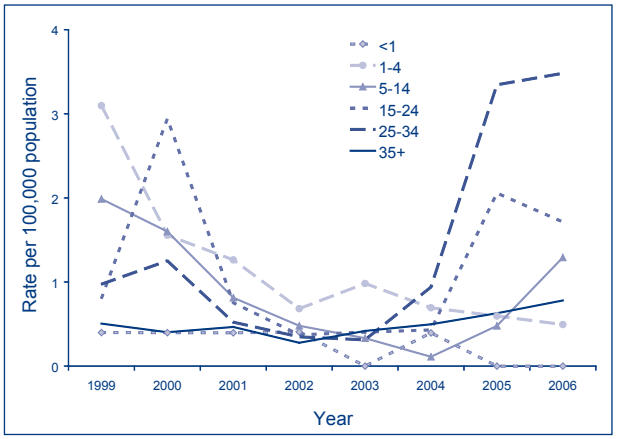
There were cases in all age groups with the highest rates in the 25–29 years age group (4.2 cases per 100,000 population). Rates in young children aged less than 5 years remained low (0.5 cases per 100,000 population, 6 cases). In 2006, the male to

Figure 47. Notification rate of mumps, Australia, 2006, by age group



female ratio of cases was 0.9:1 (Figure 47), which is a reversal of male preponderance seen in previous years, probably due to clustering of cases.

Figure 48. Trends in the notification rate of mumps, Australia, 1999 to 2006, by age group



Trends in notification rates by age group for mumps (Figure 48) show a continued increase in the rates for the 25–34 and 5–4 years age groups and a small decline in the 15–24 years age group.

Information on vaccination status was available for 177 (64%) cases; 32 were recorded as fully vaccinated; 13 as partially vaccinated; 132 as unvaccinated and there was no information on the vaccination status of the remaining 98 (36%) cases.

The high rate of mumps in the 25–34 years age group probably represents a susceptible cohort of individuals who have not been immunised. Mumps vaccine was made available in Australia in 1980 for use at 12–15 months of age and was combined with the measles vaccine in 1982. Therefore, no childhood doses of mumps vaccine were available to most individuals in the 25–34 years age group. This cohort was also not targeted in the Measles Control Campaign in 1998 where the 2nd dose of MMR was offered to primary school aged children (5–12 years). Uptake of vaccination in older individuals from the 15–24 years age group was likely to be poor.

A similar pattern is seen in the United Kingdom and the United States of America where under-immunised young adult populations led to outbreaks of mumps in the 18–24 years age group in 2004/05 and 2006, respectively.[17,18] The increase in notifications in 2005 and 2006 meant that the rates in Australia exceeded 1 cases per 100,000 population, a threshold for disease elimination and indicative of endemic mumps transmission in Australia.[13]

Pertussis

Case definition – Pertussis

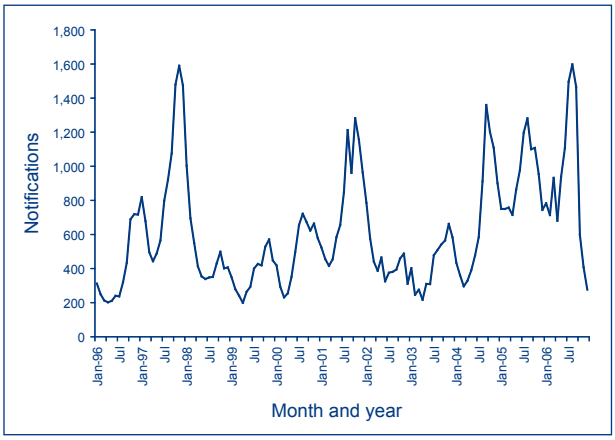
*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolation of* Bordetella pertussis *or detection of* B. pertussis *by nucleic acid testing OR seroconversion or significant increase in antibody level or fourfold or greater rise in titre (in the absence of pertussis vaccination) or a single high-titre IgA to whole cells or detection of B. pertussis by immunofluorescence AND **clinical evidence** (a coughing illness lasting 2 weeks or more or paroxysms of coughing or inspiratory whoop or post-tussive vomiting) OR **clinical evidence** AND epidemiological link to a confirmed case.*

***Probable case:** Requires clinically compatible illness.*

Pertussis is the most common vaccine preventable illness in Australia, with periodic epidemics occurring at intervals of 3 to 5 years on a background of endemic circulation. Rates are normally higher in late winter and spring, except from 2004 onward, when non-seasonal rates remained elevated compared with previous years (Figure 49).

Figure 49. Number of notifications of pertussis, Australia, 1996 to 2006, by month of onset



In 2006, 10,998 cases of pertussis were notified; a rate of 53.4 cases per 100,000 population. This was similar in number and rate to that reported in 2005 (11,197 cases, 55.1 cases per 100,000 population). In 2006, 10,559 (96%) were confirmed and 439 (4%) were probable cases.

Notification rates increased with age, with the highest notification rate in the 60–64 years age group (Figure 50). There were more cases among women

(6,624) than men (4,362) with a male to female ratio of 0.7:1. The highest rate among women was in the 60–64 years age group (114.9 cases per 100,000 population) and the highest rate in men was in the 65–69 years age group (83.2 cases per 100,000 population).

Trends in the pertussis notification rate in different age groups are shown in Figure 51. In 2006, pertussis notification rates declined in all age groups except for the 60 year and over age group, where rates increased from 61.2 cases in 2005 to 77.7 cases per 100,000 population. In particular, the decline seen in the 10–19 years age group following the introduction of adolescent vaccination in 2004, continued in 2006. In 2006, 89% of pertussis cases were aged 20 years or over compared with 50% in 2000.

Increases in rates of pertussis in Australia may be, in part, due to errors in diagnosis using serology. In October 2006, PanBio announced a major revision in the cut-off level for their pertussis serology tests. These kits were widely used in New South Wales. As a result, there was a sharp decline in pertussis notifications in the last months of 2006 (Figure 52).

Figure 50. Notification rate of pertussis, Australia, 2006, by age group and sex

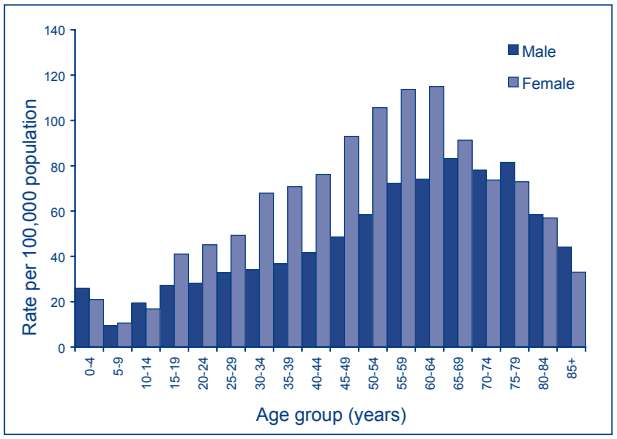
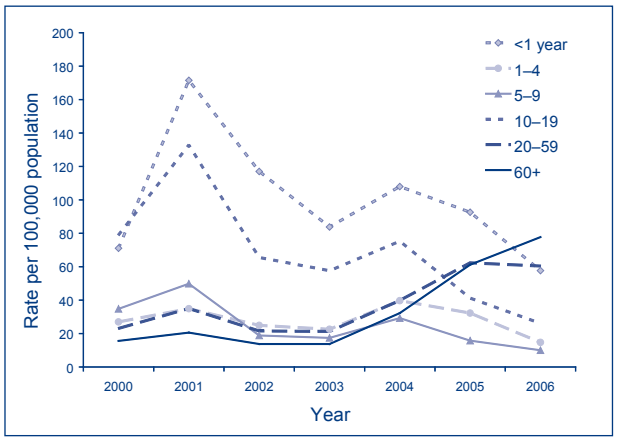


Figure 51. Trends in the notification rate of pertussis, Australia, 2000 to 2006, by age group



Map 5. Notification rates of pertussis, Australia, 2005, by Statistical Division of residence

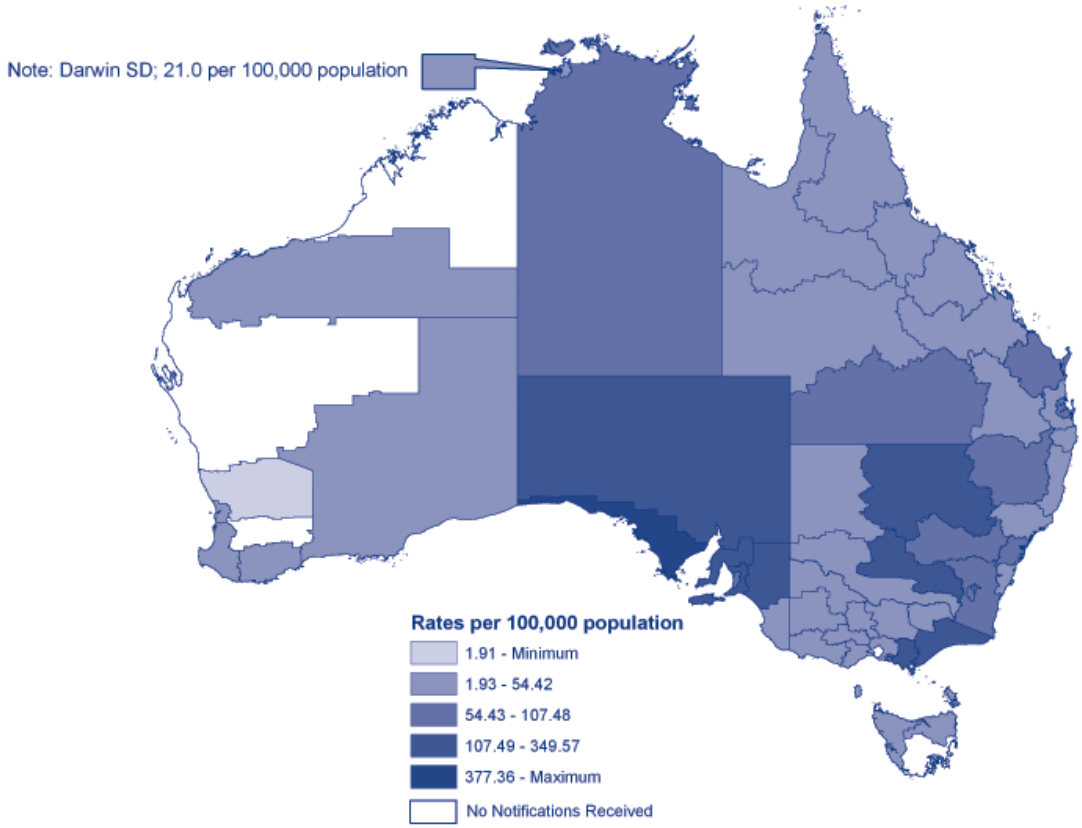
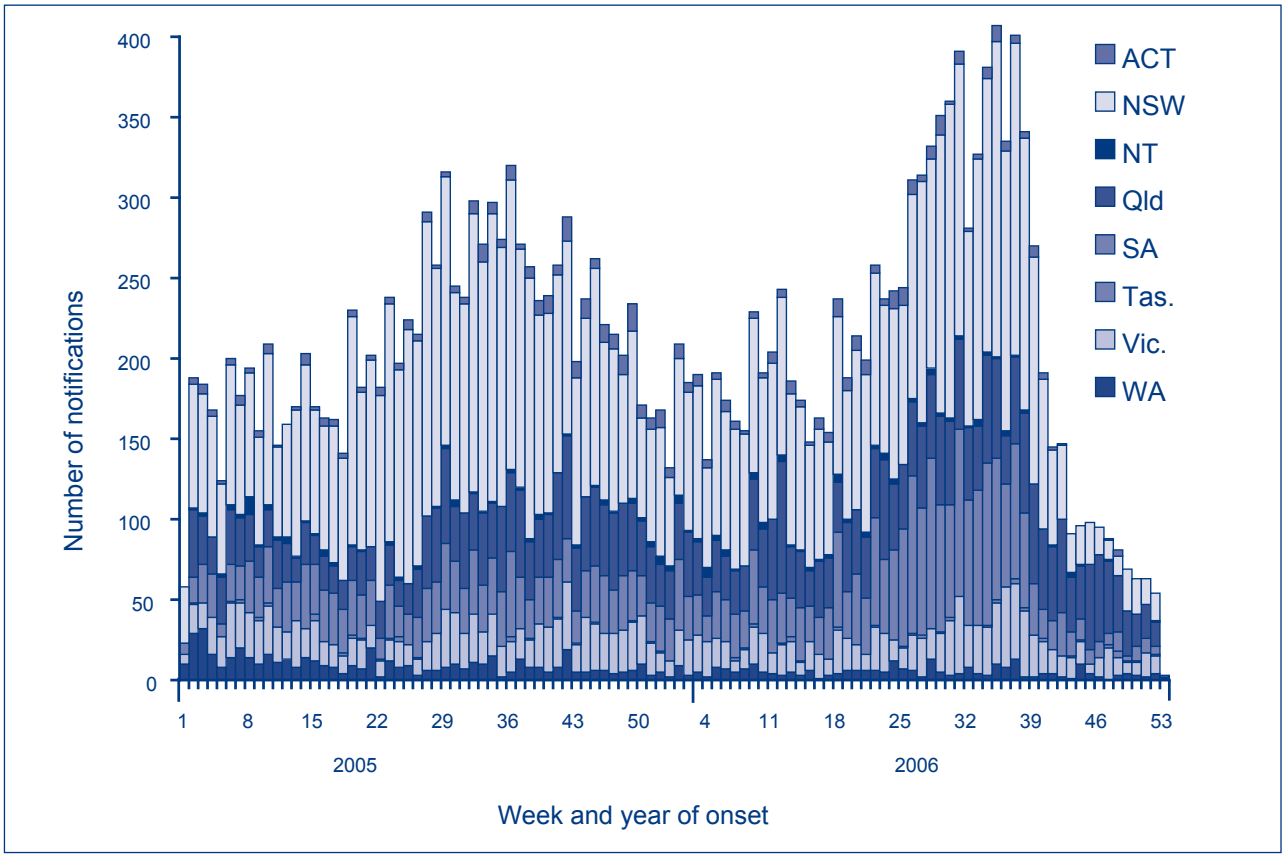
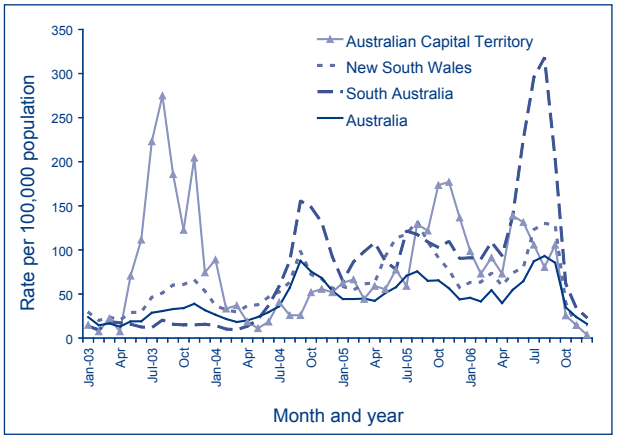


Figure 52. Number of notifications of pertussis, Australia, 2005 to 2006, by week of onset and state or territory



Notification rates of pertussis varied considerably by geographic location (Map 5). The highest rates were reported from South Australia, New South Wales and the Australian Capital Territory. The trends in pertussis notification rates by month of diagnosis are shown for these 3 states and for Australia in Figure 53.

Figure 53. Notification rate of pertussis, Australian Capital Territory, New South Wales, South Australia, and Australia, 2003 to 2006, by month of notification



Invasive pneumococcal disease

Case definition – Invasive pneumococcal disease

*Only **confirmed cases** are reported.*

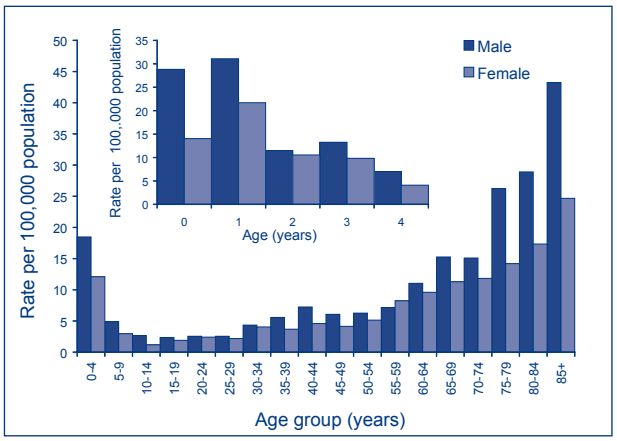
***Confirmed case:** Requires isolation of* Streptococcus pneumoniae *from a normally sterile site by culture or detection by nucleic acid testing.*

There were 1,443 notifications of invasive pneumococcal disease (IPD) in Australia in 2006, a rate of 7 cases per 100,000 population. Notification rates declined in 2006 by 14% nationally, with declines in all jurisdictions between 7% and 37%. The Northern Territory continued to have the highest notification rate (27 cases per 100,000 population) while Victoria had the lowest (5.4 cases per 100,000 population). The geographical distribution of IPD varied within states and territories, with the highest rates in central and northern Australia.

The highest rates of IPD notification in 2006 were in male adults aged 85 years or older (43.2 cases per 100,000 population, Figure 54). The male to female ratio of IPD cases was 1.3:1.

Additional data were collected on cases of invasive pneumococcal disease in all Australian states and territories during 2006. Analyses of these data are reported separately.[19]

Figure 54. Notification rate for invasive pneumococcal disease, Australia, 2006, by age group and sex



Poliomyelitis

Case definition – Poliomyelitis

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolation of wild-type poliovirus or detection of wild-type poliovirus by nucleic acid testing (confirmed in reference laboratory) and acute flaccid paralysis.*

***Probable case:** Requires acute flaccid paralysis not due to other causes as determined by the Polio Expert Committee.*

In 2006, no acute flaccid paralysis (AFP) cases due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated paralytic poliomyelitis (VAPP) were reported in Australia.

The WHO target for AFP surveillance in a polio non-endemic country is 1 case of AFP per 100,000 children aged less than 15 years. A total of 48 eligible AFP cases were notified in Australia between 1 January and 31 December 2006, giving an AFP rate of 1.2 cases per 100,000 population. The Polio Expert Committee (PEC) reviewed clinical and laboratory information on 43 of the 48 eligible AFP notifications. The PEC was unable to provide final classification for 5 AFP notifications due to insufficient clinical information. Hence the 2006 non-polio AFP rate, based on the 43 eligible cases classified by the PEC, was 1.1 per 100,000 children aged less than 15 years.

Since the inception of the Australian AFP surveillance system in 1995, the WHO AFP surveillance standard has only been achieved in 2000, 2001, 2004 and 2006. However, adequate faecal sampling remains well below the 80% target established by WHO with only 23% of eligible AFP notifications having 2 samples collected 24 to 48 hours apart and within 14 days of onset of paralysis.[20]

With the introduction of IPV into the standard immunisation schedule in Australia from November 2005, no further isolations of OPV strains of poliovirus are expected in Australian-born AFP cases without overseas travel. This was demonstrated in 2006, with the last reported laboratory isolations of a poliovirus occurring after 2 infants were vaccinated with OPV at the end of 2005.

In 2006, globally 2000 confirmed cases of wild poliovirus were reported to WHO.[21] Four countries: Nigeria, India, Pakistan and Afghanistan, were considered to be endemic. Imported wild poliovirus was detected in 10 countries and active transmission of imported poliovirus occurred in 4 of those (http://www.polioeradication.org/content/general/casecount.pdf). Australia is at risk of importation of polio through visitors and migrants from polio endemic areas and requires AFP and laboratory surveillance to be timely and comprehensive.[22]

Rubella

Case definition – Rubella

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolation of rubella virus OR detection of rubella virus by nucleic acid testing OR IgG seroconversion or significant increase in antibody level or fourfold or greater rise in titre to rubella virus in the absence of recent rubella vaccination, OR detection of rubella specific IgM in the absence of recent rubella vaccination and confirmed in a reference laboratory.*

Probable case:** Requires **clinical evidence** AND **laboratory suggestive evidence** OR **epidemiological evidence.

***Laboratory suggestive evidence:** In a pregnant patient, detection of rubella-specific IgM that has not been confirmed in a reference laboratory, in the absence of recent rubella vaccination.*

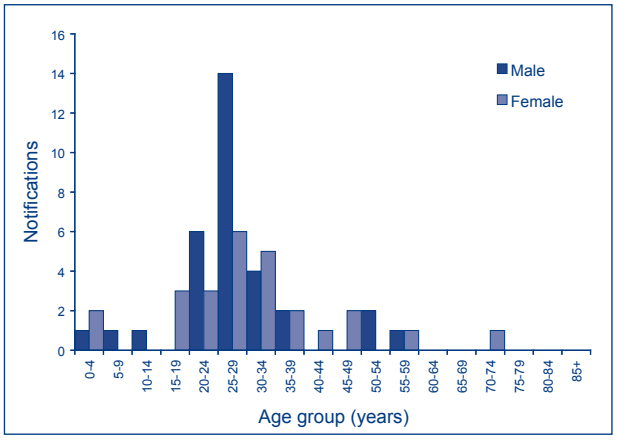
***Clinical evidence:** A generalised maculopapular rash AND fever AND arthralgia/arthritis OR lymphadenopathy OR conjunctivitis*

***Epidemiological evidence:** An epidemiological link is established when there is: 1. Contact between 2 people involving a plausible mode of transmission at a time when: a) one of them is likely to be infectious (about 1 week before to at least 4 days after appearance of rash) AND b) the other has an illness which starts within 14 and 23 days after this contact AND 2. At least 1 case in the chain of epidemiologically linked cases (which may involve many cases) is laboratory confirmed.*

In 2006, there were 59 notifications of rubella (0.3 cases per 100,000 population) an increase of 90% on the 31 notifications in 2005. In 2006, rubella cases were reported from New South Wales (37 cases), Queensland (12 cases), Victoria (6) and 2 cases each in South Australia and Western Australia. No cases were reported from other jurisdictions.

The overall male to female ratio of notified cases in 2006 was 1.2:1; but in the 25–29 years age group, the ratio was 2.3:1 (Figure 55). There was an overall predominance of males in notifications in 1999, 2002 and 2003.

Figure 55. Notification rate of rubella, Australia, 2006 by age group and sex



In Australia, populations at risk of rubella include young men who did not receive rubella immunisation in school based programs;[23] migrant women who did not receive rubella vaccines in their countries of birth;[24,25] and Indigenous women with inadequate immunity.[26]

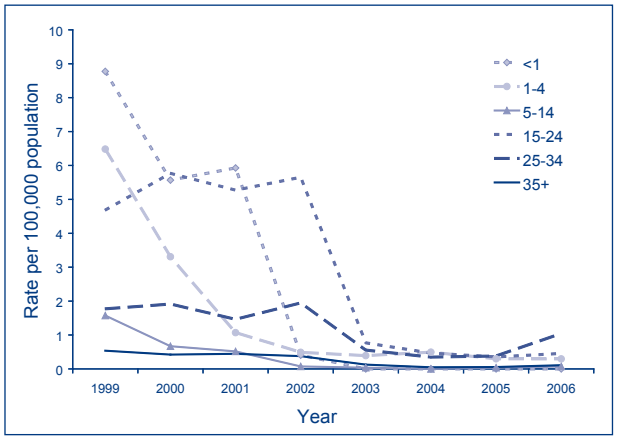
There were more than 28,000 cases of rubella reported to the WHO Western Pacific Region office in 2005, which implies that rubella infections could be acquired by Australian travellers to neighbouring rubella endemic countries.

Figure 56 shows trends in rubella notification rates in different age groups, with a slight increase in rates in young adults in 2006, but otherwise continuing at the low levels seen since 2003.

There were no cases of congenital rubella reported in 2006. Altogether there were 22 cases of rubella notified from women of child bearing age (15–49 years) in 2006.

As for measles and mumps, Australia is approaching the elimination of endemic rubella with rates of reported disease less than 1 case per 100,000 popu-

Figure 56. Trends in notification rates for rubella, Australia, 1999 to 2006, by age group



lation since 2002 (Table 4a). In 2004, the United States was declared free of endemic rubella based on epidemiological evidence; the absence of a circulating endemic rubella genotype; high rubella vaccine coverage; and serological evidence of high levels of population immunity and mass immunisation in the Pan American region.[27] The WHO Regional Committee for Europe agreed in 2005 to eliminate measles and rubella by 2010. http://www.euro.who.int/vaccine/20030808_4

Brotherton, et al[13] suggest that the achievement and confirmation of the elimination of locally acquired rubella circulation may require targeted immunisation of migrants from countries with low levels of rubella vaccination and the establishment of rubella genotyping in Australia.

Tetanus

Case definition – Tetanus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Clostridium tetani *from a wound in a compatible clinical setting and prevention of positive tetanospasm in mouse test using a specific tetanus antitoxin OR a clinically compatible illness without other apparent cause.*

In 2006, there were 3 notifications of tetanus. One case occurred in an 18-year-old (partially immunised) female from Victoria. The other 2 cases were aged 66 and 74 years.

Varicella infections

In November 2005, varicella vaccine was added to the NIP Schedule as a single dose due at 18 months (for children born on or after 1 May 2004) or at 12–13 years. In 2006, CDNA agreed to make varicella infections notifiable in Australian juris-

dictions. Three categories of varicella infection are notifiable: chickenpox, shingles and varicella infection (unspecified).

By the end of 2006, 5 jurisdictions, were sending data to NNDSS. New South Wales decided in 2006 not to make varicella infections notifiable. The legal processes to make varicella notifiable in the Australian Capital Territory and Victoria were still underway.

In 2006, there were 6,156 varicella notifications from those jurisdictions, 1,514 (25%) reported as chickenpox, 1,077 (17%) as shingles and 3,565 (58%) as unspecified varicella infection.

Varicella zoster infection (chickenpox)

Case definition – Varicella-zoster infection (chickenpox)

*Both **confirmed cases** and **probable cases** are reported.*

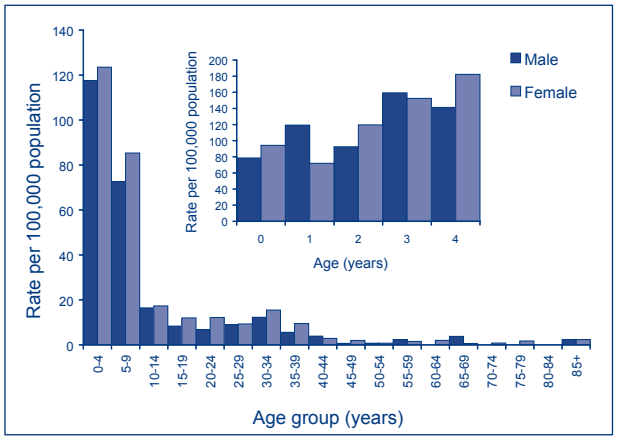
***Confirmed case:** Isolation of varicella-zoster virus from a skin or lesion swab OR detection of varicella-zoster virus from a skin or lesion swab by nucleic acid testing from a skin or lesion swab OR detection of varicella-zoster virus antigen from a skin or lesion swab by direct fluorescent antibody from a skin or lesion swab. If the case received varicella vaccine between 5 and 42 days prior to the onset of rash the virus must be confirmed to be a wild type strain OR detection of varicella-zoster virus-specific IgM in an unvaccinated person AND acute onset of a diffuse maculopapular rash developing into vesicles within 24–48 hours and forming crusts (or crusting over) within 5 days. OR acute onset of a diffuse maculopapular rash developing into vesicles within 24–48 hours and forming crusts (or crusting over) AND where an epidemiological link is established when there is: contact between 2 people involving a plausible mode of transmission at a time when one of them is likely to be infectious AND the other has illness 10 to 21 days after contact AND at least 1 case in the chain of epidemiologically-linked cases is laboratory confirmed.*

***Probable case:** Acute onset of a diffuse maculopapular rash developing into vesicles within 24–48 hours and forming crusts (or crusting over)*

In 2006, there were 1,514 notifications of chickenpox reported from 5 jurisdictions. The highest rates were reported from the Northern Territory (93.4 cases per 100,000, 193 cases) and South Australia (48.9 cases per 100,000 population, 760 cases). South Australia made varicella infections notifiable in 2002.

One thousand and sixty-four cases (70%) occurred in children aged less than 10 years. The highest rates were in the 0–4 years age group (120 cases per 100,000 population) and within this age group 3-year-olds had the highest rate (156 cases per 100,000 population, Figure 57) There were slightly more female than male cases notified (male:female ratio 0.9:1).

Figure 57. Notification rate of chickenpox, Australia,* 2006, by age group and sex



* Excluding the Australian Capital Territory, New South Wales and Victoria.

One thousand and sixty-two cases were confirmed and the remainder were probable cases.

Seventy-six were recorded as fully vaccinated for age; 7 partially vaccinated; 1,221 unvaccinated and there was no vaccination status information on the remainder of the notified cases.

Varicella zoster infection (shingles)

Case definition – Varicella-zoster infection shingles

*Both **confirmed cases** and **probable cases** are reported.*

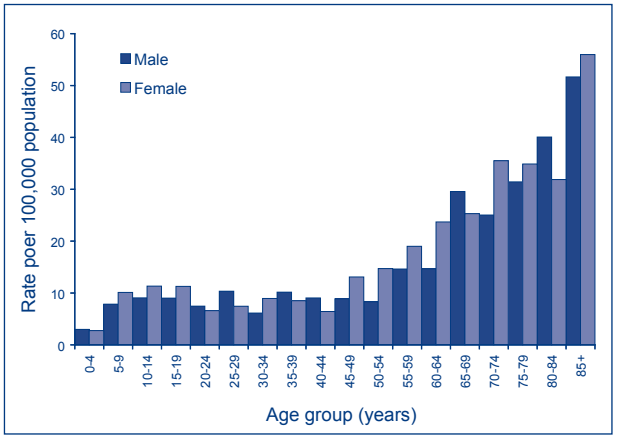
***Confirmed case:** Isolation of varicella-zoster virus from a skin or lesion swab OR detection of varicella-zoster virus from a skin or lesion swab by nucleic acid testing from a skin or lesion swab OR detection of varicella-zoster virus antigen from a skin or lesion swab by direct fluorescent antibody from a skin or lesion swab AND a vesicular skin rash with a dermatomal distribution that may be associated with pain in skin areas supplied by sensory nerves of the dorsal root ganglia.*

***Probable case:** A vesicular skin rash with a dermatomal distribution that may be associated with pain in skin areas supplied by sensory nerves of the dorsal root ganglia.*

There were 1,077 notifications of shingles reported to NNDSS in 2006 (a rate of 5.2 cases per 100,000 population). The highest rates were in South Australia (40.2 cases per 100,000 population, 625 cases) and the Northern Territory (38.7 cases per 100,000 population, 80 cases).

There was a predominance of female cases with a male to female ratio of 0.8:1. The highest rates were in the over 85 years age groups for both males and females (54.5 cases per 100,000 population, Figure 58).

Figure 58. Notification rate of shingles, Australia,* 2006, by age group and sex



* Excluding the Australian Capital Territory, New South Wales and Victoria.

Varicella zoster infection (unspecified)

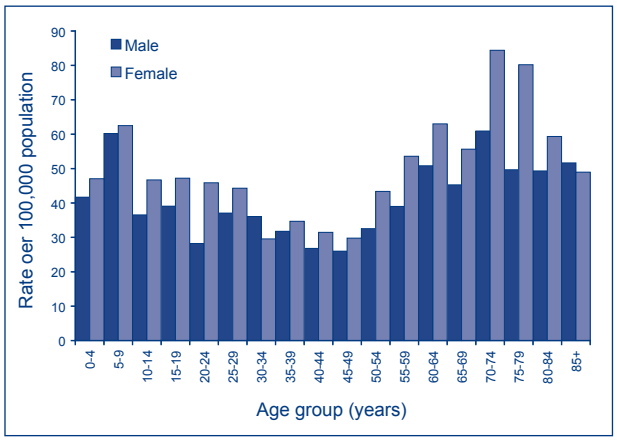
Case definition – Varicella-zoster infection unspecified

*Only **confirmed cases** are reported.*

***Confirmed case:** Isolation of varicella-zoster virus from a skin or lesion swab OR detection of varicella-zoster virus from a skin or lesion swab by nucleic acid testing from a skin or lesion swab OR detection of varicella-zoster virus antigen from a skin or lesion swab by direct fluorescent antibody from a skin or lesion swab OR detection of varicella-zoster virus-specific IgM in an unvaccinated person.*

There were 3,565 cases of varicella infections (unspecified) based on laboratory diagnosis, and largely from Queensland (3,167, 89%). There was a female predominance with a male to female ratio of 0.8:1. The age distribution of unspecified varicella infections is shown in Figure 59.

Figure 59. Notification rate of varicella zoster infection (unspecified), Australia,* 2006, by age group and sex



* Excluding the Australian Capital Territory, New South Wales and Victoria.

Childhood vaccination coverage reports

Estimates of vaccination coverage both overall and for individual vaccines for children at 1 year, 2 years and 6 years of age in 2006 are shown in Tables 14, 15 and 16 respectively. During 2006, there were no significant changes in coverage for children 'fully immunised' or individual vaccines for the 1 year and 2 year milestone ages. However, there was a significant change in coverage for children 'fully immunised' and individual vaccines for the 6 year milestone age. Coverage increased by 2 to 3.5 percentage points for all vaccines between the first and second quarter of 2006 and was maintained through the following 2 quarters. A possible factor in this increase in coverage at 6 years of age is the introduction of the multivalent combination vaccine Infanrix-IPV onto the schedule that occurred in November 2005, reducing the number of vaccines to be recorded from 3 to two. Other factors that may have had an impact at the local level include promotional campaigns centred around child care or school entry or data cleaning activities. Estimates at 6 years of age for all vaccines still remain significantly lower than estimates at the 1 year and 2 years milestones.

Vectorborne diseases

Notifications

During 2006, there were 8,606 notifications of mosquito-borne diseases reported to NNDSS. The notifiable mosquito-borne diseases include those caused by the alphaviruses (Barmah Forest virus

Table 14. Percentage of Australian children born in 2005 immunised according to data available on the Australian Childhood Immunisation Register, estimate at 1 year of age

Birth date Vaccine	1 Jan–31 Mar 2005 % immunised	1 Apr– 30 Jun 2005 % immunised	1 Jul–30 Sep 2005 % immunised	1 Oct–31 Dec 2005 % immunised
DTP	92.2	91.9	92.0	91.9
Polio	92.1	91.8	92.0	91.8
Hib	94.2	94.4	94.8	94.5
Hepatitis B	94.7	94.4	94.7	94.4
Fully immunised	90.7	90.8	91.2	91.0

Table 15. Percentage of Australian children born in 2004 immunised according to data available on the Australian Childhood Immunisation Register, estimate at 2 years of age

Birth date Vaccine	1 Jan–31 Mar 2004 % vaccinated	1 Apr–30 Jun 2004 % vaccinated	1 Jul–30 Sep 2004 % vaccinated	1 Oct–31 Dec 2004 % vaccinated
DTP	95.2	95.1	95.2	94.8
Polio	95.2	95.0	95.1	94.8
Hib	93.8	93.7	93.9	93.6
MMR	94.0	93.9	94.0	93.7
Hepatitis B	95.8	95.8	95.8	95.6
Fully immunised	92.4	92.2	92.4	92.0

Table 16. Percentage of Australian children born in 2000 immunised according to data available on the Australian Childhood Immunisation Register, estimate at 6 years of age

Birth date Vaccine	1 Jan–31 Mar 2000 % vaccinated	1 Apr–30 Jun 2000 % vaccinated	1 Jul–30 Sep 2000 % vaccinated	1 Oct–31 Dec 2000 % vaccinated
DTP	85.0	87.0	88.8	88.8
Polio	83.8	87.1	88.8	88.9
MMR	85.0	87.1	88.8	88.9
Fully immunised	82.7	86.2	88.0	88.0

and Ross River virus), flaviviruses (the viruses causing dengue, Murray Valley encephalitis, Kunjin and Japanese encephalitis) and malaria.

Alphaviruses

Alphaviruses are RNA viruses, which cause disease epidemics characterised by fever, rash and polyarthritis. In Australia, Barmah Forest virus and Ross River virus are the alphaviruses of major public health significance, accounting for 88% of the total mosquito-borne disease notifications for 2006. There are a variety of mosquito vectors for Barmah Forest virus and Ross River virus, which facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[28]

Barmah Forest virus infection

Case definition – Barmah Forest virus infection

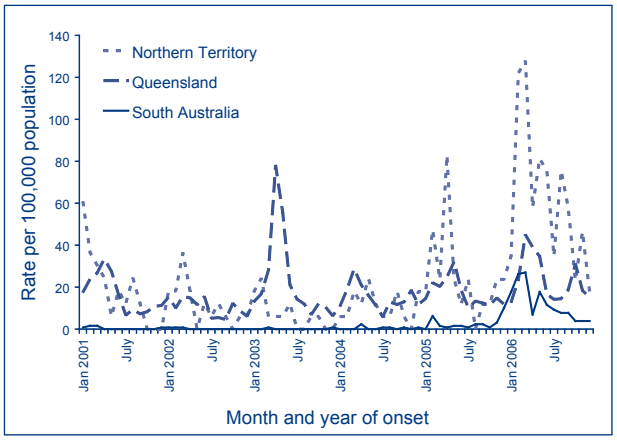
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Barmah Forest virus, OR detection of Barmah Forest virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Barmah Forest virus, OR detection of Barmah Forest virus-specific IgM.*

There were 2,120 notifications of Barmah Forest virus (BFV) infections notified to NNDSS in 2006, which accounted for 39% of total mosquito-borne disease notifications for the year. Forty-five per cent of BFV infection notifications were reported from Queensland (n=957) and 30% from New South Wales (644 cases). BFV infection notifications during 2006 were 1.8 times the mean for the previous 5 years.

The highest rates of BFV infection notifications were reported by the Northern Territory (62.9 cases per 100,000 population compared with 25.1 cases per 100,000 population in 2005), Queensland (23.6 cases per 100,000 compared with 17.2 cases per 100,000 in 2005), and South Australia (12 cases per 100,000 population compared with 2.6 cases per 100,000 in 2005), (Figure 60). The national BFV infection notification rate in 2006 was 10.3 cases per 100,000 population, compared with 6.5 cases per 100,000 population in 2005.

Figure 60. Notification rate of Barmah Forest virus infections, Northern Territory, Queensland, and South Australia, 2001 to 2006, by month and year of onset

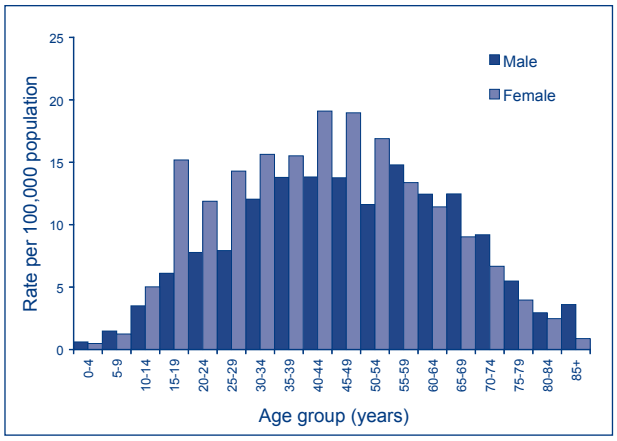


In the Northern Territory, the lowest BFV infection notification rate was 17.4 cases per 100,000 population during December and the peak occurred during March (127.7 cases per 100,000 population). Notification rates for the Northern Territory were significantly higher than the other states and territories from February through to November.

For 2006, the highest regional BFV infection notification rate was reported in South Australia's Murray Lands Statistical Division (86.8 cases per 100,000 population).

Figure 61 shows the age and sex distribution of BFV infection notifications. The BFV infection notification rate was highest amongst the 40–44 years age range (16.5 cases per 100,000 population), and the male to female ratio was 0.8:1. Males in the 35–39, 40–44 and 45–49 years age groups had the highest age-specific rate (13.8 cases per 100,000 population). The highest age specific BFV infection notification rate in females was in the 40–44 years age group (19.1 cases per 100,000 population). The notification rate in females for the 15–19 years age group was 2.5 times higher than males. The major contributing jurisdictions were the Northern Territory (154.3 cases per 100,000 population) and South Australia (40 cases per 100,000 population).

Figure 61. Notification rate of Barmah Forest virus infections, Australia, 2006, by age group and sex



Ross River virus infection

Case definition – Ross River virus infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Ross River virus, OR detection of Ross River virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Ross River virus, OR detection of Ross River virus-specific IgM.*

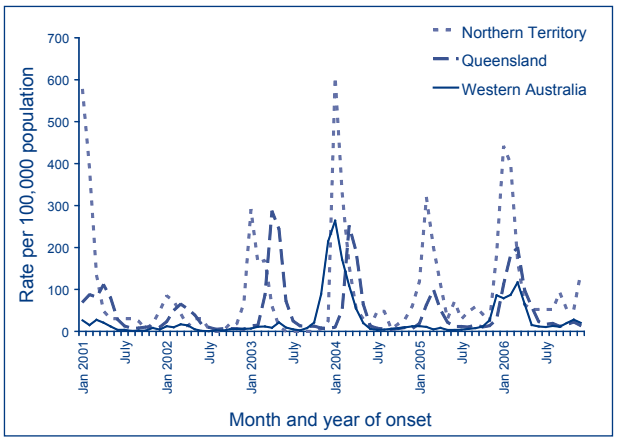
There were 5,487 notifications of Ross River virus (RRV) infections reported to NNDSS in 2006, which accounted for 63% of the total of mosquito-borne disease notifications received during this year.

The majority of RRV infection notifications in 2006 were from Queensland (48%, 2,615 cases) and New South Wales (22%, 1,225 cases). The highest rate of notifications was reported in the Kimberly Statistical Division of Western Australia (236.9 cases per 100,000 population). The national RRV infection notification rate for 2006 was 26.6 cases per 100,000 population.

RRV infection notifications in the Northern Territory peaked in January at 441.2 cases per 100,000 population (Figure 62). This was a 28% increase from the peak notification rate in 2005 (February, 319.5 cases per 100,000 population). Queensland reported a peak notification rate for RRV infection in March

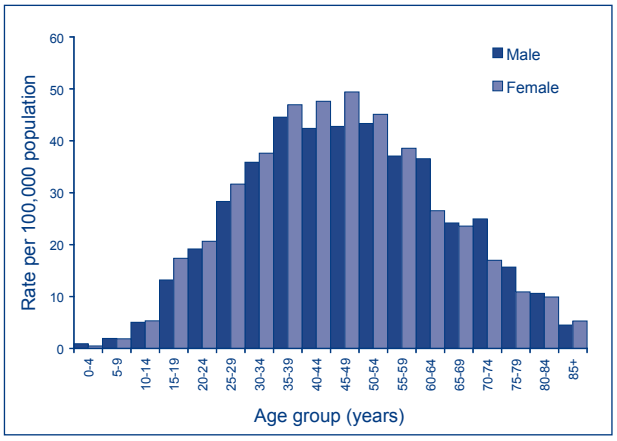
at 200.4 cases per 100,000 population, which was a 51% increase from the peak notification rate in 2005 (March, 99.6 cases per 100,000 population).

Figure 62. Notification rate of Ross River virus infections, Northern Territory, Queensland and Western Australia, 2001 to 2006, by month and season of onset



The age and sex distribution of RRV infection notifications is shown in Figure 63. The national notification rate was highest in the 45–49 years age group (46.1 cases per 100,000 population). The highest RRV infection notification rate in males (43.3 cases per 100,000 population) was observed in the 50–54 years age group and the highest notification rate in females was recorded in the 45–49 years age group (49.4 cases per 100,000 population).

Figure 63. Notification rate of Ross River virus infections, Australia, 2006, by age group and sex



Flaviviruses

Flaviviruses are single-stranded RNA viruses, some of which are associated with epidemic encephalitis in various regions of the world. In Australia, the flaviviruses of public health importance are Murray Valley encephalitis (MVEV), Kunjin (KUNV), Japanese encephalitis virus (JEV) and dengue viruses (DENV).

The Sentinel Chicken Program is a surveillance network involving New South Wales, the Northern Territory, Victoria and Western Australia. The flocks are located in strategic locations and are regularly tested for antibodies to MVEV infection, JEV infection and KUNV infection. This program is designed to provide early warning of flavivirus activity (excluding dengue).[29] Sentinel chicken surveillance reports from previous seasons have been published,[30,31,32] and the latest report has been published as part of the National Arbovirus and Malaria Advisory Committee annual report 2006–07.[33]

Murray Valley encephalitis virus infection

Case definition – Murray Valley encephalitis virus infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Murray Valley encephalitis virus, OR detection of Murray Valley encephalitis virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Murray Valley encephalitis virus, OR detection of Murray Valley encephalitis virus-specific IgM in cerebrospinal fluid in the absence of IgM to Kunjin, Japanese encephalitis or dengue viruses, OR detection of Murray Valley encephalitis virus-specific IgM in serum in the absence of IgM to Kunjin, Japanese encephalitis or dengue viruses. This is only accepted as laboratory evidence for encephalitic illnesses, AND non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash, OR encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following: 1. focal neurological disease or clearly impaired level of consciousness, 2. an abnormal computerised tomograph or magnetic resonance image or electrocardiograph, 3. presence of pleocytosis in cerebrospinal fluid, OR asymptomatic disease: Case detected as part of a serosurvey should not be notified.*

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case occurs in areas of Australia not known to have established enzootic/endemic activity or regular epidemic activity.

In 2006, Western Australia reported to NNDSS 1 case of Murray Valley encephalitis virus infection in an 8-year-old female, the case fully recovered.

Kunjin virus infection

Case definition – Kunjin virus infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Kunjin virus, OR detection of Kunjin virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Kunjin virus, OR detection of Kunjin virus-specific IgM in cerebrospinal fluid, OR detection of Kunjin virus-specific IgM in serum in the absence of IgM to Murray Valley encephalitis, Japanese encephalitis or dengue viruses. This is only accepted as laboratory evidence for encephalitic illnesses, AND non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash, OR encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following: 1. focal neurological disease or clearly impaired level of consciousness, 2. an abnormal computerised tomograph or magnetic resonance image or electrocardiograph, 3. presence of pleocytosis in cerebrospinal fluid, OR asymptomatic disease: case detected as part of a serosurvey should not be notified.*

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case occurs in areas of Australia not known to have established enzootic/endemic activity or regular epidemic activity.

During 2006 there were 3 notifications of KUNV reported to NNDSS, of which Queensland reported 1 notification (male, 44 years) and Western Australia reported 2 notifications (both females 20 and 27 years).

Dengue virus infection

Case definition – Dengue virus infection

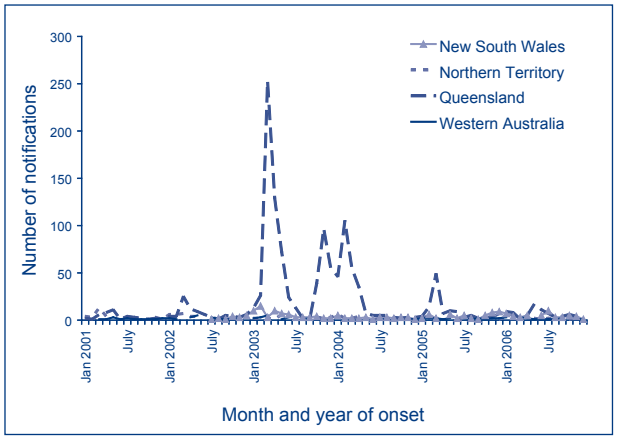
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of dengue virus, OR detection of dengue virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to dengue virus, proven by neutralisation or another specific test, OR detection of dengue virus-specific IgM in cerebrospinal fluid, in the absence of IgM to Murray Valley encephalitis, Kunjin, or Japanese encephalitis viruses, OR detection of dengue virus-specific IgM in serum, except in North Queensland. In North Queensland, dengue virus-specific IgM in serum is acceptable evidence ONLY when this occurs during a proven outbreak, AND a clinically compatible illness (e.g. fever, headache, arthralgia, myalgia, rash, nausea, and vomiting, with a possible progression to dengue haemorrhagic fever, dengue shock syndrome or meningoencephalitis).*

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case occurs in previously unaffected areas of Australia. Currently North Queensland is the only area with the potential for indigenous (epidemic) dengue virus in Australia.

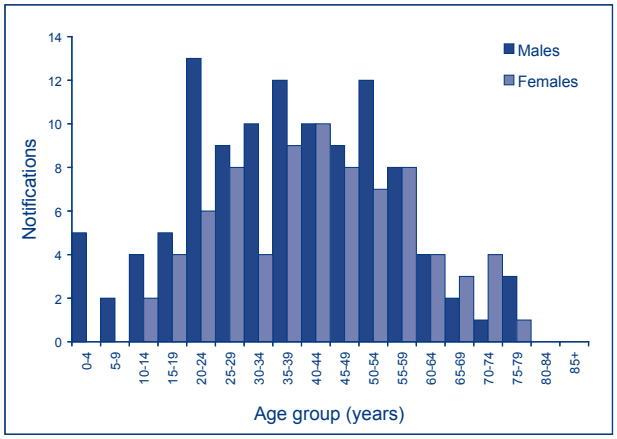
There were 187 notifications of dengue virus infection reported to NNDSS in 2006 (Figure 64), of which Queensland reported 78 notifications (42%). Of the 78 notifications, Queensland reported 28 notifications that were acquired locally.

Figure 64. Notifications of dengue virus infection (locally-acquired and imported cases), New South Wales, Northern Territory, Queensland and Western Australia, 2001 to 2006, by month and year of onset



The age and sex distribution of DENV notifications is shown in Figure 65. The highest rates occurred in the 20–24 years age group (13 cases) for males, and

Figure 65. Number of notifications of dengue virus infection (locally-acquired and imported cases), Australia, 2006, by age group and sex



in females in the 40–44 years age group (10 cases). The notification rate in males from the 20–24 years age group was 1.25 times higher than females, 8 of the 13 notifications in this group of males reported overseas acquisition.

Japanese encephalitis virus infections

Case definition – Japanese encephalitis virus infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Japanese encephalitis virus, OR detection of Japanese encephalitis virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre of Japanese encephalitis virus-specific IgG proven by neutralisation or another specific test, with no history of recent Japanese encephalitis or yellow fever vaccination, OR detection of Japanese encephalitis virus-specific IgM in cerebrospinal fluid, in the absence of IgM to Murray Valley encephalitis, Kunjin and dengue viruses, OR detection of Japanese encephalitis virus-specific IgM in serum in the absence of IgM to Murray Valley encephalitis, Kunjin and dengue viruses, with no history of recent Japanese encephalitis or yellow fever vaccination.*

AND a clinically compatible febrile illness of variable severity associated with neurological symptoms ranging from headache to meningitis or encephalitis. Symptoms may include headache, fever, meningeal signs, stupor, disorientation, coma, tremors, generalised paresis, hypertonia, and loss of coordination. The encephalitis cannot be distinguished clinically from other central nervous system infections.

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case appears to have been acquired in Australia.

There were no human cases of JEV infection notified in 2006. The last JEV infection notification was reported by Queensland in February 2004 when a 66-year-old male acquired JEV infection in Papua New Guinea. There have been 9 other cases of JEV infection reported to NNDSS since 1995, although JEV infection was not nationally notifiable until 2001. Four of these 9 notifications were reported in Torres Strait Islanders from the Badu Island community. The other locally acquired JEV infection case was reported in a resident from the Cape York Peninsula, Queensland. The remaining 4 cases were reported as acquired from overseas countries.

Flavivirus infection (NEC)

Case definition – Flavivirus infection (NEC)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of a flavivirus that cannot be identified in Australian reference laboratories or which is identified as one of the flaviviruses not otherwise classified, OR detection of a flavivirus, by nucleic acid testing, that cannot be identified in Australian reference laboratories or which is identified as one of the flaviviruses not otherwise classified, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre of flavivirus specific IgG that cannot be identified or which is identified as being specific for one of the flaviviruses not otherwise classified. There must be no history of recent Japanese encephalitis or yellow fever vaccination, OR detection of flavivirus IgM in cerebrospinal fluid, with reactivity to more than one flavivirus antigen (Murray Valley encephalitis, Kunjin, Japanese encephalitis and/or dengue) or with reactivity only to one or more of the flaviviruses not otherwise classified, OR detection of flavivirus IgM in the serum, with reactivity to more than one flavivirus antigen (Murray Valley encephalitis, Kunjin, Japanese encephalitis and/or dengue) or with reactivity only to one or more of the flaviviruses not otherwise classified. This is only accepted as laboratory evidence for encephalitic illnesses. There must be no history of recent Japanese encephalitis or yellow fever vaccination, AND non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash, OR encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following: 1. focal neurological disease or clearly impaired level of consciousness, 2. an abnormal computerised tomograph or magnetic resonance image or electrocardiograph, 3. presence of pleocytosis in cerebrospinal fluid.*

Confirmation by a second arbovirus reference laboratory is required if the case cannot be attributed to known flaviviruses.

There were 33 flavivirus infection (NEC) notifications during 2006; notified by Queensland (23 cases) and Victoria (10 cases).

There were 6 Kokobera virus and 1 Stratford virus infection notifications from Queensland in this category.

Malaria

Case definition – Malaria

*Only **confirmed cases** are reported.*

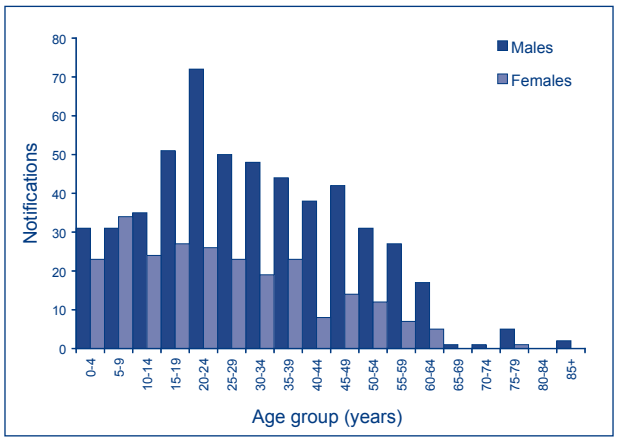
***Confirmed case:** Requires detection and specific identification of malaria parasites by microscopy on blood films with confirmation of species in a laboratory with appropriate expertise, OR detection of* Plasmodium *species by nucleic acid testing.*

There were 775 notifications of malaria in Australia in 2006. The majority of cases were reported by Queensland (35%, 268 cases), New South Wales (18%, 140 cases), Victoria (15%, 115 cases) and Western Australia (15%, 115 cases). Queensland reported that 135 of 268 notifications were acquired in Papua New Guinea.

The largest number (99 cases) of malaria notifications was in the 20–24 years age group (Figure 66). The male to female ratio was 1:0.5.

The infecting *Plasmodium* species was reported for 94% for malaria notifications in 2006 (Table 17). Of these 775 notifications, *P. falciparum* (46%, 354 cases) and *P. vivax* (42%, 324 cases) were the predominant species while untyped *Plasmodium* species accounted for 6% (48 cases). The remaining cases were *P. ovale* (2%, 17 cases), *P. malariae* (1%, 11 cases) and mixed *Plasmodium* species infections (3% 21 cases).

Figure 66. Number of notifications of malaria, Australia, 2006, by age group and sex



Zoonoses

A zoonosis is 'an infection or infectious disease transmissible under natural conditions from vertebrate animals to humans'.[34] Animal hosts play an essential role in maintaining the infection in nature, and humans are only incidental hosts.[35] Animals are thought to be the origin of approximately 75% of emerging human infectious diseases and wildlife contribute significantly to this threat.[3] The Australian Government, through the animal and human health agencies, is proactively addressing this threat by strengthening the link between animal and human health systems.

In 2006, zoonotic diseases notifiable to the NNDSS were anthrax, Australian bat lyssaviral or lyssaviral (unspecified) infection, brucellosis, leptospirosis,

Table 17. Malaria notifications in Australia, 2006, by parasite type and jurisdiction

Parasite type	Type (%)	State or territory								
		ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Plasmodium falciparum	46	4	47	46	115	21	18	37	68	354
Plasmodium malariae	1	0	5	0	6	0	0	0	0	11
Plasmodium ovale	2	0	2	0	4	1	1	8	1	17
Plasmodium vivax	42	7	78	19	126	8	6	62	18	324
Plasmodium species	6	0	5	0	16	1	1	0	23	48
Mixed *P. falciparum* and *P. vivax**	1	0	2	0	0	2	0	0	0	4
Mixed *P. falciparum* and other species*	2	0	0	1	0	1	0	8	4	14
Mixed *P. vivax* and other species*	0	0	1	0	1	0	0	0	1	3
Total	100	11	140	66	268	34	26	115	115	775

* New South Wales, South Australia, Tasmania, Victoria and Western Australia report mixed species infections per notified case. Queensland, the Northern Territory and the Australian Capital Territory report one notification for each species in a mixed infection.

ornithosis and Q fever. During 2006, a total of 767 notifications of zoonotic disease (0.5% of total notifications) were made to the NNDSS.

Anthrax

Case definition – Anthrax

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Bacillus anthracis*-like organisms or spores confirmed by a reference laboratory OR detection of* Bacillus anthracis *by microscopic examination of stained smears, OR detection of* Bacillus anthracis *by nucleic acid testing AND cutaneous: skin lesion evolving over 1–6 days from a papular through a vesicular stage, to a depressed black eschar invariably accompanied by oedema that may be mild to extensive, OR gastrointestinal: abdominal distress characterised by nausea, vomiting, anorexia and followed by fever, OR rapid onset of hypoxia, dyspnoea and high temperature, with radiological evidence of mediastinal widening, OR meningeal: acute onset of high fever, convulsions, loss of consciousness and meningeal signs and symptoms.*

Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts.[3] Anthrax has a low decreasing prevalence, and occurs only sporadically in Australia.[36] It can be an occupational hazard for veterinarians, agriculture and wildlife workers who handle infected animals.

One case of cutaneous anthrax in a 48-year-old man was reported to NNDSS in 2006. The case was from the western part of New South Wales and was associated with exposure to infected cattle in an area where anthrax was endemic.[37] Before this 2006 case, a human case of cutaneous anthrax had not been reported in Australia since 1998.

In 2006, 10 outbreaks of anthrax were reported in livestock. All cases occurred in central New South Wales, where cases have been known to occur in the past. In all cases, properties were subject to the recommended protocol of quarantine, carcass incineration, site disinfection and vaccination of in-contact animals. All movements from affected properties were traced to ensure that relevant product did not enter the export and domestic chains.[36]

Australian bat lyssaviral and lyssaviral (unspecified) infections

Case definition – Australian bat lyssavirus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Australian bat lyssavirus confirmed by sequence analysis, OR detection of Australian bat lyssavirus by nucleic acid testing.*

Case definition – Lyssavirus (unspecified)

*Only **confirmed cases** are notified AND only where there is insufficient evidence to meet a case definition for Australian bat lyssavirus or rabies.*

***Confirmed case:** Requires positive fluorescent antibody test result for lyssaviral antigen on fresh brain smears, OR specific immunostaining for lyssaviral antigen on formalin fixed paraffin sections of central nervous system tissue, OR presence of antibody to serotype 1 lyssavirus in the cerebrospinal fluid, OR detection of lyssavirus-specific RNA (other than to Australian bat lyssavirus or rabies).*

AND acute encephalomyelitis with or without altered sensorium or focal neurological signs.

No cases of either Australian bat lyssaviral or lyssaviral (unspecified) infections were notified during 2006. Previously, 2 known cases of human infection with Australian bat lyssavirus were fatal and occurred in 1996 and 1998 following close contact between bat-handlers and infected bats. One case was associated with a sub-order Megachiroptera (from a frugivorous bat) and the other was associated with sub-order Microchiroptera (found in smaller, mainly insectivorous bats).

Surveillance indicates Australian bat lyssavirus infection is and may have been present in Australian bats 15 years prior to its first detection. Sick and injured bats (opportunistic specimens) and change in seasonality and bat ecology pose an increased public health risk.

Brucellosis

Case definition – Brucellosis

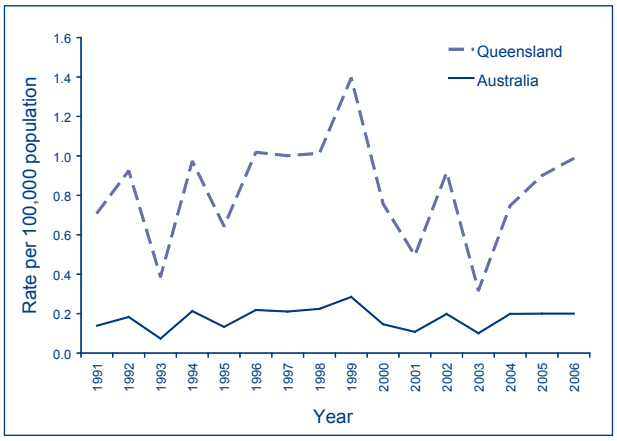
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Brucella *species, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre in* Brucella *agglutination titres or complement fixation titres between acute and convalescent phase serum samples. (Where possible both tests should be conducted at the same laboratory), OR a single high* Brucella *agglutination titre.*

Brucellosis is mainly an occupational disease for farm workers, veterinarians, and abattoir workers who work with infected animals or their tissue.[3]

In 2006, 49 cases of brucellosis were reported to the NNDSS, giving a national notification rate of 0.2 cases per 100,000 population. Cases were from Queensland (40 cases), New South Wales (8 cases) and Western Australia (1 case). The highest notification rate (74 cases per 100,000 population) was from the Central West region of Queensland. There is little evidence of change in the trend in the national or Queensland notification rates of brucellosis over the last 13 years (Figure 67). Most cases were male (43 cases, male to female ratio 7:1), and of these, 80% were aged between 20 and 64 years.

Figure 67. Trends in notifications rate for brucellosis, Australia and Queensland, 1991 to 2006



Species data was available for 31% of notifications (12 cases). Of these 6 were *Brucella suis*, 4 cases were *B. melitensis* (3 reported from New South Wales and 1 reported from Western Australia) and 2 cases were *B. abortus* (reported from New South Wales). All of these cases were acquired overseas.

Except for *B. suis*, cases are assumed to have had overseas exposure. Bovine brucellosis (*B. abortus*) was eradicated from the Australian cattle herd in 1989 and is presently considered an exotic animal disease in Australia.[38] Caprine and ovine brucellosis (caused by *B. melitensis*) has never been reported in Australian sheep or goats. Swine brucellosis (caused by *B. suis*) is confined to small areas of northern Australia, where it occurs in feral pigs, with human cases predominantly seen in recreational feral pig hunters.

Leptospirosis

Case definition – Leptospirosis

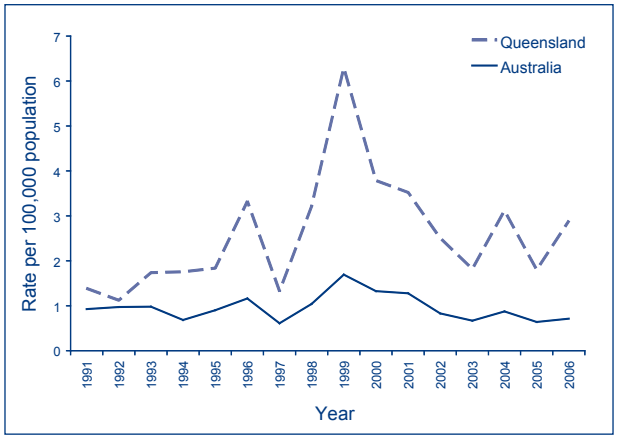
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of pathogenic* Leptospira *species, OR a fourfold or greater rise in* Leptospira *agglutination titre between acute and convalescent phase sera obtained at least 2 weeks apart and preferably conducted at the same laboratory, OR a single* Leptospira *micro agglutination titre greater than or equal to 400 supported by a positive enzyme-linked immunosorbent assay IgM result.*

Leptospirosis is caused by spirochaetes of the genus, *Leptospira*, which is found in the renal tubules of wild and domestic animals. In affected areas, where there is exposure to infected urine of domestic and wild animals, this disease can be an occupational and recreational hazard.[3]

Nationally, 147 notifications of leptospirosis were received during 2006 (0.7 cases per 100,000 population). During the last 13 years, notification rates peaked in 1999 and from 2000 onwards continued to decline (Figure 68).

Figure 68. Trends in notifications for leptospirosis, Australia and Queensland, 1991 to 2006



In 2006, the highest notification rate was in Queensland (117 notifications, 2.9 cases per 100,000 population). There were also notifications received from the Northern Territory (2 notifications, 1 case per 100,000 population), New South Wales (17 notifications, 0.2 cases per 100,000 population), Tasmania (1 notification, 0.2 cases per 100,000 population), Victoria (6 notifications, 0.1 cases per 100,000) and South Australia (1 notification 0.06 cases per 100,000 population). Sixty-six per cent of all notifications were from Far North Queensland; the notification rate in this region was 31 cases per 100,000 population.

Most leptospirosis cases were male (131 cases, male to female ratio 8.2:1), and the 20–24 years age group had the highest notification rate (1.7 cases per 100,000 population).

Ornithosis

Case definition – Ornithosis

*Both **confirmed cases** and **probable cases** are reported.*

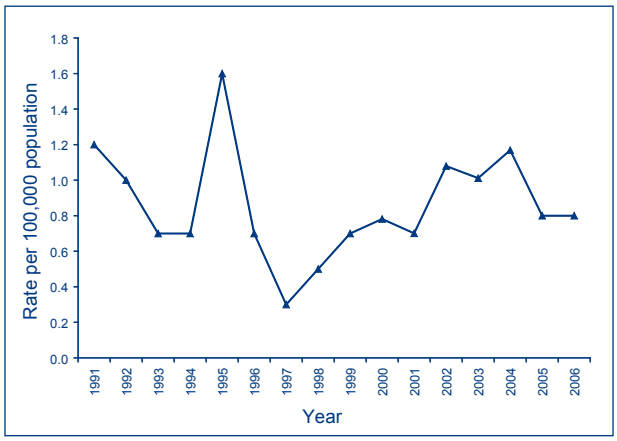
***Confirmed case:** Requires a fourfold rise or greater in antibody titre against* Chlamydia psittaci *as demonstrated by micro-immunofluorescence (MIF) on acute and convalescent sera (collected at least 2 weeks later) tested in parallel, OR detection of* C. psittaci *by nucleic acid testing or culture, AND pneumonia, OR AT LEAST TWO of the following: fever, headache, myalgia, rigours, dry cough or dyspnoea, AND exposure to birds or bird products, or proximity to an outbreak of ornithosis.*

***Probable case:** Requires a single high total antibody level or detection of IgM antibody to* C. psittaci *by MIF, OR a single high total antibody titre to* Chlamydia *species demonstrated by complement fixation (CF) in at least one sample obtained at least 2 weeks after onset of symptoms, OR a fourfold or greater rise in antibody titre against* Chlamydia *species as demonstrated by CF, AND pneumonia, OR AT LEAST TWO of the following: fever, headache, myalgia, rigours, dry cough or dyspnoea, AND exposure to birds or bird products, or proximity to an outbreak of ornithosis.*

Ornithosis is caused by *Chlamydophila psittaci* and is transmitted to humans by exposure to waterfowl, seabirds, shore birds, pigeons and doves and many psittacine birds. Birds can become carriers of the disease without becoming infected. The mode of transmission to humans is by inhaling bacteria usually from contaminated dried faeces, nasal or eye secretions and dust from infected birds.[3] Human-to-human transmission is rare.

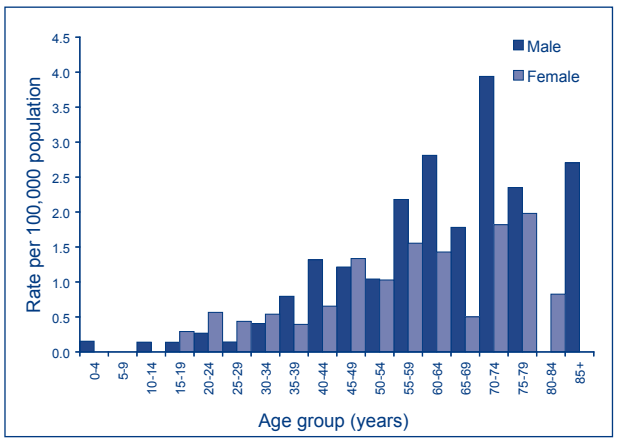
In 2006, there were 168 ornithosis infections notified to NNDSS, giving a national rate of 0.8 cases per 100,000 population. The national rate of notifications has steadily increased from 1997 to 2004, but in 2005 and 2006 decreased to 2001 levels (Figure 69).

Figure 69. Trends in notification rates for ornithosis, Australia, 1991 to 2006



New South Wales had the highest number of notifications (94 notifications, 1.4 cases per 100,000 population). Notifications were also received from Victoria (65 cases), Western Australia (4 cases), the Australian Capital Territory (2 cases), Queensland (2 cases), and Tasmania (1 case). The majority of cases were male (97 cases, male to female ratio 1.3:1). Eighty-one per cent of cases were aged 40 years or over, with the highest notification rate in males in the 70–74 years age group (12 notifications, 3.9 cases per 100,000 population) and in females in the 75–79 years age group (6 notifications, 2.0 cases per 100,000 population) (Figure 70).

Figure 70. Notification rate for ornithosis, Australia, 2006, by age group and sex



At risk groups of people contracting ornithosis include bird owners, pet shop employees, veterinarians, poultry processing workers, zoo workers and taxidermists. Older adults and pregnant women may have a more severe illness.[39] An outbreak in the Blue Mountains in June 2002 reinforced that infections in humans can be associated with wild birds, rather than with pet birds and aviaries.[40]

Q fever

Case definition – Q fever

*Only **confirmed cases** are reported.*

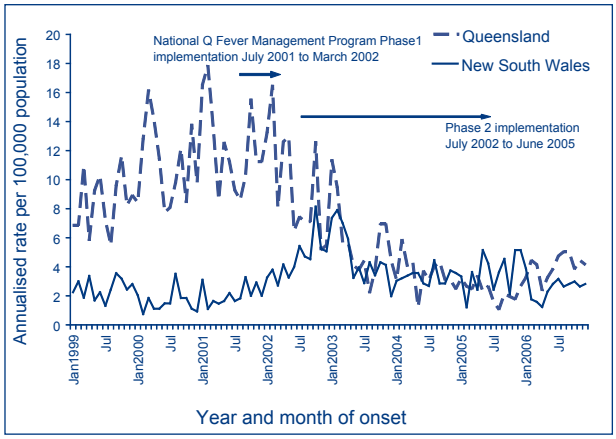
***Confirmed case:** Requires detection of* Coxiella burnetii *by nucleic acid testing, OR seroconversion or significant increase in antibody level to Phase II antigen in paired sera tested in parallel in absence of recent Q fever vaccination, OR detection of* C. burnetii *by culture (note this practice should be strongly discouraged except where appropriate facilities and training exist), OR detection of specific IgM in the absence of recent Q fever vaccination, AND a clinically compatible disease.*

Q fever is caused by *Coxiella burnetii*. Primary reservoirs of these bacteria are cattle, sheep and goats. The organisms are resistant to heat, drying and many common disinfectants, this enables the bacteria to survive for long periods in the environment. The mode of transmission to humans is commonly through the airborne route in dust, but it can also occur though direct contact with infected animals and other contaminated material. Humans are often very susceptible to the disease, and very few organisms may be required to cause infection. Person-to-person transmission is rare.[3]

In 2006, 402 cases of Q fever were notified to the NNDSS. The highest rates of notifications were from Queensland (164 notifications, 4 cases per 100,000 population) and New South Wales (174 notifications, 2 cases per 100,000 population) (Figure 71). The highest rates were in the 45–49 years age group for males (5.4 cases per 100,000 population), and in the 60–64 years age groups for females (1.8 cases per 100,000 population). There were 14 cases reported in people aged less than 14 years and 6 cases reported in adults aged over 75 years. The male to female ratio was 3.8:1.

Production of the Q fever vaccine ceased at the end of 2005 because of the manufacturers' inability to meet new regulations and other product pressures.[41] At the end of 2006, the Australian Ministers for Health and Agriculture announced funding for CSL Limited to recommence production of the Q fever vaccine.[41] Adults at risk, including abattoir workers, farmers, veterinarians, stockyard workers, shearers, animal transporters and many others exposed to cattle, sheep or goats or their products should be considered for vaccination.

Figure 71. Notification rate for Q fever, Queensland and New South Wales, 1999 to 2006, by month of onset



Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2006 and classified as 'other bacterial infections' in NNDSS. A total of 1,900 notifications were included in this group in 2006, which accounted for 1.37% of all the notifications to NNDSS, a similar total and proportion as in 2005 (1,826 notifications and 1.4% of total).

Legionellosis

Case definition – Legionellosis

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolation of* Legionella, *OR the presence of* Legionella *urinary antigen OR seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to* Legionella, *AND fever or cough or pneumonia.*

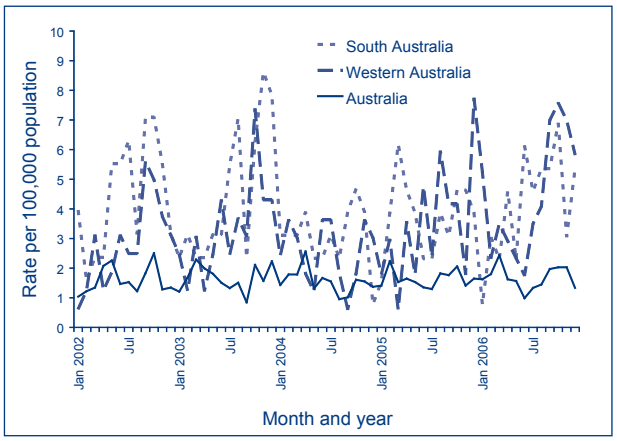
***Probable case:** Single high titre antibody titre to* Legionella, *OR detection of* Legionella *by nucleic acid testing, OR detection of* Legionella *by direct fluorescence assay, AND fever or cough or pneumonia.*

Legionellosis includes notifications of infections caused by all *Legionella* species. There were 348 notifications of legionellosis reported in 2006, giving a national rate of 1.7 cases per 100,000 population.

This was an increase over the 334 cases reported in 2005. In 2006, an increase in cases was seen in Western Australia (4.4 cases per 100,000 population, 91 cases) and South Australia (4.2 cases per 100,000 population, 65 cases).

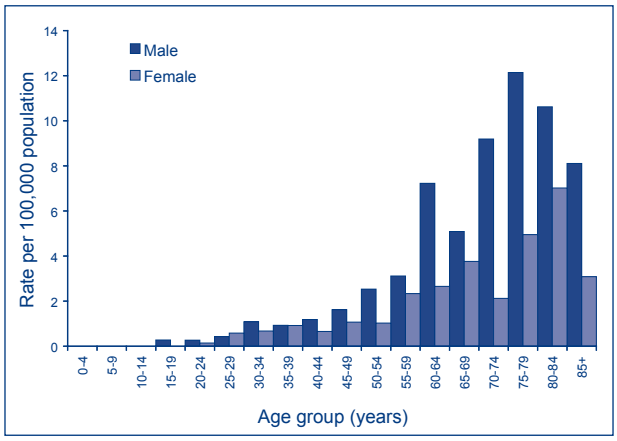
Legionellosis notifications showed a peak in autumn and spring, as in previous years (Figure 72). Rates of legionellosis have ranged between 0.8 and 2.6 cases per 100,000 population between 1999 and 2005, except in 2000, when rates reached 6.9 cases per 100,000 population as a result of the Melbourne aquarium outbreak with 125 cases.[42]

Figure 72. Trends in notification rates of legionellosis, South Australia, Western Australia and Australia, 2002 to 2006, by month of onset



In 2006, men accounted for 222 of the 348 notified cases of legionellosis resulting in a male to female ratio of 1.7:1. There were no cases in children under the age of 15 years. Overall, the highest rate of infection was 8.5 cases per 100,000 population in the 80–84 years age group. In men, the highest rate occurred in men in the 75–79 years age group (12.1 cases per 100,000 population, 31 cases) and women, in the 80–84 years age group (7 cases per 100,000 population, 17 cases, Figure 73).

Figure 73. Notification rate of legionellosis, Australia, 2006, by age group and sex



Data on the causative species were available for 336 (97%) of the 348 legionellosis cases. Of these, 178 (53%) were *Legionella longbeachae,* 154 (46%) cases were identified as *L. pneumophila* and 4 (1%) were *L. micdadei* or *L. bozemanii* (Table 18).

Of the 154 *L. pneumophila* notifications, serogroup data were available on 83 (54%) cases; 77 (92%) of serogrouped *L. pneumophila* were serogroup 1.

There are significant differences in the geographic distribution of *L. longbeachae* and *L. pneumophila*, with the *L. longbeachae* making up the majority of species in notifications from South Australia and Western Australia, while *L. pneumophila* are the most common infecting species in the eastern states (Queensland, New South Wales and Victoria).

Data on the death of legionellosis cases were available for 230 (66%) notifications. There were 9 reported deaths due to legionellosis in Australia in 2006, giving a case fatality rate of 3.9%. The break down of deaths by state or territory and infecting *Legionella* species is shown in Table 19. There were 6 deaths associated with *L. longbeachae* infection (all in Western Australia) giving a case fatality rate of 3.3%. Three patients with *L. pneumophila* infections

Table 18. Notifications of legionellosis, 2006, by state or territory and species

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Australia
Legionella longbeachae	0	22	2	7	46	2	13	86	178
Legionella pneumophila	1	54	1	26	18	1	51	2	154
Other species*	0	0	0	0	1	0	3	0	4
Unknown species	0	1	0	6	0	0	2	3	12
Total	1	77	3	39	65	3	69	91	348

* *Legionella micdadei, Legionella bozemanii*

died, giving a case fatality rate of 1.9%. Case fatality rates may be overestimated given the large proportion of cases without details of death outcomes.

The number of deaths decreased in 2006 relative to 2005 when there were 14 deaths. Decreases in deaths associated with legionellosis fell in all states and territories except Western Australia where there were 3 more deaths in 2006 than in 2005.

There were 3 outbreaks of legionellosis was reported in 2006. A cluster of 10 cases (including 1 death) was linked to a Melbourne metropolitan shopping centre. In Sydney, 6 linked cases were reported, while in Queensland a cluster of cases associated with a coal mine was also reported.

A case control study of *L. longbeachae* cases in South Australia was recently published, which clarified risk factors associated with this infection.[43] The organism has been isolated from potting mix [44] and inhalation of dust from potting mix has been thought to be a major route of infection. The recent study by O'Connor[43] demonstrated that other risk factors such as exposure to aerosolised bacteria from dripping hanging pots and possible ingestion of organisms due to failure to wash hands after gardening may be more significant. Long-term smokers were also shown to be at increased risk of infection.[43]

Leprosy

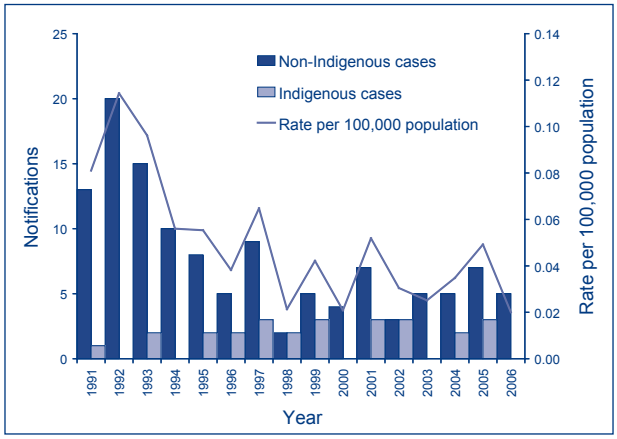
Case definition – Leprosy

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires demonstration of acid fast bacilli in split skin smears and biopsies prepared from ear lobe or other relevant sites or histopathological report from skin or nerve biopsy compatible with leprosy (Hansen's disease) examined by an anatomical pathologist or specialist microbiologist AND compatible nerve conduction studies or peripheral nerve enlargement or loss of neurological function not attributable to trauma or other disease process, or hypopigmented or reddish skin lesions with definite loss of sensation.*

Leprosy is a chronic infection of the skin and peripheral nerves with the bacterium *Mycobacterium leprae*. Leprosy is a rare disease in Australia, with the majority of cases occurring among migrants to Australia from leprosy endemic countries and occasional cases from Indigenous communities. Trends in the numbers of leprosy notification in Indigenous and non-Indigenous Australians and the overall rate are shown in Figure 74.

Figure 74. Number of notifications of leprosy in Indigenous and non-Indigenous Australians and the overall notification rate, 1991 to 2006



In 2006, 5 leprosy cases were notified to NNDSS compared with 10 cases in 2005. There were 2 cases in Western Australia, and a single case in New South Wales, the Northern Territory and South Australia. Two cases occurred in men and 3 in women. None of the cases were Indigenous Australians. The age range of cases was 26–42 years. Four of the 5 cases had multi-bacillary leprosy.

Table 19. Deaths due to legionellosis, Australia, 2006, by state or territory and species

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Australia
Legionella longbeachae	0	0	0	0	0	0	0	6	6
Legionella pneumophila	0	0	0	0	1	0	2	0	3
Other species*	0	0	0	0	0	0	0	0	0
Unknown species	0	0	0	0	0	0	0	0	0
Total	0	0	0	0	1	0	2	6	9

* *Legionella micdadei, Legionella bozemanii*

Invasive meningococcal disease

Case definition – Invasive meningococcal disease

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Defined as isolation of* Neisseria meningitidis *from a normally sterile site. Alternatively, detection of meningococcus by nucleic acid testing, or Gram negative diplococci in Gram stain in specimens from a normally sterile site or from a suspicious skin lesion, OR high titre IgM or a significant rise in IgM or IgG titres to outer membrane protein antigens, OR positive polysaccharide antigen test in cerebrospinal fluid AND disease compatible with invasive meningococcal disease.*

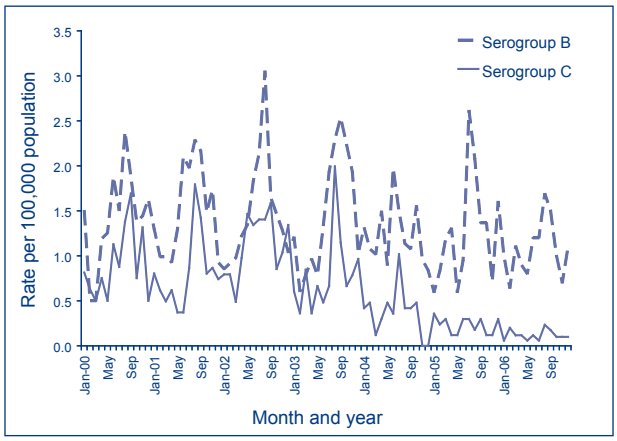
***Probable case:** Defined as the absence of evidence for other causes of clinical symptoms AND EITHER clinically compatible disease including haemorrhagic rash OR clinically compatible disease and close contact with a confirmed case within the previous 60 days.*

Historically in Australia, serogroups B and C have been the major cause of invasive meningococcal disease. The Australian Government commenced the National Meningococcal C Vaccination Program in January 2003.

In 2006, there were 318 notifications of invasive meningococcal disease in Australia, a decrease from 392 in 2005. A decline was seen in all states except South Australia and Victoria. The total in 2006 was the lowest since 1996. The national notification rate in 2006 was 1.5 cases per 100,000 population. The highest rate was reported from the Northern Territory (2.9 cases per 100,000 population, 6 cases).

Fifty-two per cent (165) of cases occurred in males, giving a male to female ratio of 1.1:1. As in previous years, the largest number of cases occurred in winter and spring (Figure 75). The majority of cases (294, 93%) were confirmed, and 24 (7%) had a probable diagnosis.

Figure 75. Trends in notification rates of meningococcal infection, Australia, 2002 to 2006, by month of onset and serogroup



Of the 318 meningococcal notifications in 2006, 267 (84%) were serogrouped. Of these, 223 (83.5%) were serogroup B, 24 (9%) were serogroup C and 20 (7.5%) were infections with serogroup Y (5), serogroup W-135 (14) or serogroup A (1) (Table 20). In comparison in 2005, 83% (326/393) of notified cases were serogrouped, 256 (79%) were serogroup B and 46 (14%) were serogroup C.

Serogroup C infections were largely confined to the eastern seaboard states; Victoria, New South Wales and Queensland, where serogroup C meningococcal disease has in previous years been more common than in other states.

The highest age specific meningococcal notification rate was in children aged 0–4 years with a rate of 8.8 cases per 100,000 population (112 cases). Eighty-four per cent of cases (94/112) were serogroup B infections, which is the highest age-specific rate for serogroup B infection, with 7.3 cases per 100,000 population.

In the 15–19 years age group, the overall rate of meningococcal infection was 3.9 cases per 100,000 population (55 cases), 42 (76%) of which were serogroup B (Figure 76).

Table 20. Notifications of meningococcal infection, 2006, by state or territory and serogroup

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Australia
Serogroup B	4	57	6	58	14	4	62	18	223
Serogroup C	1	14	0	4	0	1	3	1	24
Other serogroups*	0	6	0	3	3	0	7	1	20
Unknown serogroup	0	30	0	6	1	0	13	1	51
Total	5	107	6	71	18	5	85	21	318

* Serogroup Y (5 cases); serogroup W-135 (14 cases) and serogroup A (1 case).

Figure 76. Notification rate of meningococcal C infection, Australia, 2000 to 2006, by age group

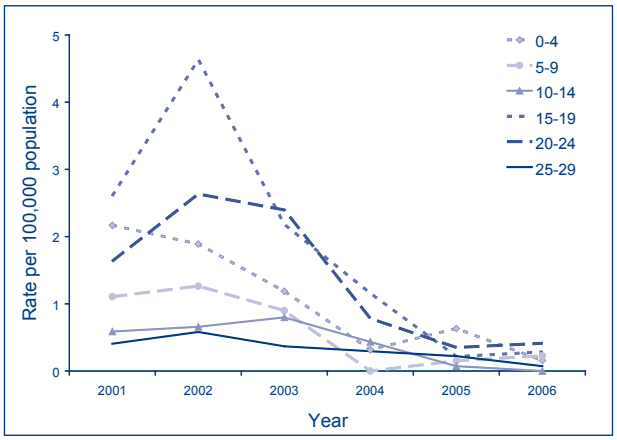
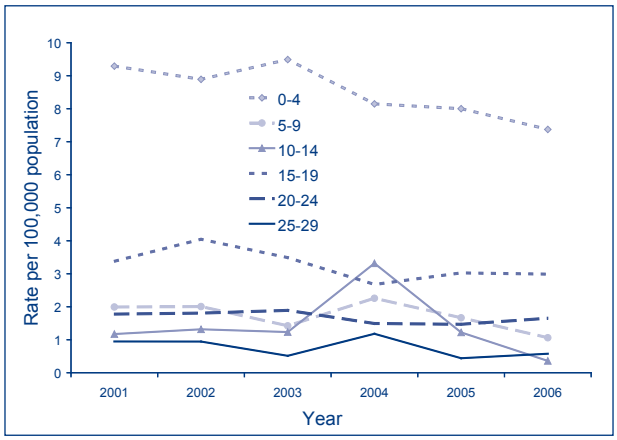


Figure 77. Notification rate of meningococcal B infection, Australia, 2001 to 2006, by age group



There has been a marked decrease in meningococcal C infection rates since 2003 when the National Meningococcal C Vaccination Program was introduced. In 2006, coverage meningococcal serogroup C vaccines in children aged 12 months reached 92.5% of Indigenous children and 93.4% of non-Indigenous children (data provided by the Australian Childhood Immunisation Register).

The greatest declines in the rate of serogroup C disease was in the 15–19 years age group from 6.6 cases per 100,000 population in 2002 (63 cases) to 0.3 cases per 100,000 population in 2006 (3 cases). The rate in the 20–24 years age group fell from 2.6 (35 cases) to 0.4 (6 cases) over the same period. Notification rates in the 0–4 years age group fell from 2.2 cases per 100,000 population in 2001 (28 cases) to 0.2 cases per 100,000 population (4 cases) in 2006.

Figure 77 shows that over the period 2001 to 2006 notification rates of serogroup B disease have declined in the 0–4 years age group by 21%; in the 5–9 years age group by 45%; and in the 10–14 years age group by 80%, while remaining stable in older age groups.

There were 12 deaths due to meningococcal disease in 2006 (a case fatality rate of 3.7%). Eight deaths were due to serogroup B (CFR= 3.6%), 3 due to W-135 (CFR = 21%) and only 1 death was due to serogroup C disease (CFR = 4.2%, Table 21). This was a decrease on the 20 deaths in 2005.

In contrast to previous years, there were only a few reports of small clusters of meningococcal disease (all serogroup B) in 2006. A case of meningococcal serogroup A on a passenger airline prompted a multi-state and international follow-up of potentially exposed fellow passengers. No secondary cases were reported.

Laboratory based meningococcal surveillance

The Australian Meningococcal Surveillance Programme (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using agreed standard methodology to determine the phenotype (serogroup, serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics. The results of laboratory surveillance in 2006 have recently been published.[45]

Table 21. Deaths due to meningococcal infection, Australia, 2006, by state or territory and serogroup

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Australia
Serogroup B	0	3	0	2	1	0	2	0	8
Serogroup C	0	1	0	0	0	0	0	0	1
Serogroup W-135	0	1	0	0	0	0	2	0	3
Total	0	5	0	2	1	0	4	0	12

In 2006, a total of 271 laboratory confirmed cases of invasive meningococcal disease were examined by the AMSP. Consistent with the NNDSS data, the AMSP reported that 80% were identified as serogroup B (217) and 9.6% were serogroup C (26). No evidence of meningococcal capsular 'switching' was detected. About two-thirds of all isolates showed decreased susceptibility to penicillin (MIC 0.06–0.5 mg/L). All isolates remained susceptible to rifampicin and ciprofloxacin.

The changing ecology of meningococcal disease in Australia has been recently reviewed.[46] The 'hypersporadic' period since the 1980s when hyper-virulent serogroup B and serogroup C clones dominated and incidence remained above 2 cases per 100,000 population, may be changing as the impact of the conjugate serogroup C meningococcal vaccine reduces the incidence of serogroup C disease.[47]

Tuberculosis

Case definition – Tuberculosis

Only ***confirmed cases*** *are reported.*

Confirmed case: *Defined as of* Mycobacterium tuberculosis *complex by culture, OR detection of* M. tuberculosis *complex by nucleic acid testing except which it is likely to be due to previously treated or inactive disease OR clinical diagnosis of tuberculosis including clinical follow-up assessment to ensure a consistent clinical course.*

While Australia has one of the lowest rates of tuberculosis in the world, the disease remains a public health problem in the overseas-born and Indigenous communities. In 2006, 1,229 TB notifications were received by NNDSS, a rate of 6 cases per 100,000 population compared with 1,083 cases notified nationally 2005. The notification rate of TB was higher than the national average in the Northern Territory (15 cases per 100, 000 population), while the lowest rate occurred in Tasmania (1.8 cases per 100, 000 population). Further details of TB notifications in 2006 have already been published.[48]

Other communicable disease surveillance

Laboratory Virology and Serology Reporting Scheme

The Laboratory Virology and Serology Reporting Scheme (LabVISE) is a passive surveillance scheme based on voluntary reports of infectious agents from sentinel virology and serology laboratories around Australia. LabVISE provides data on diagnoses of a number of infectious viruses, parasites and fungi. Interpretation of data from LabVISE is limited by uncertainties regarding its representativeness, lack of denominator data to calculate positivity rates, variable reporting coverage over time, and lack of consistent case definitions. LabVISE has an important role in supplementing information of diseases under surveillance in NNDSS and in monitoring infectious agents that are not reported by other surveillance systems.

In 2006, a total of 11 laboratories reported 19,384 infectious agents to LabVISE. This represents a 13% decrease in the number of reports received in 2005 (Table 22). Most of the reports were from South Australia (30%), Queensland (27%) and New South Wales (20%) (Table 22).

Fifty-nine per cent (11,517) of all reports received by LabVISE were viral infectious agents, and the remaining 41% (7,867) were bacterial or other infectious agents. Among viruses, herpes viruses (17%; 3,348) (including herpes virus type 6, cytomegalovirus, varicella-zoster virus, Epstein-Barr virus), RNA viruses (15%; 2,830) (including HTLV-1, rotavirus, astrovirus and Norwalk agent) and ortho/paramyxoviruses (14%; 2,626) (including influenza, parainfluenza and respiratory syncytial viruses) were the most commonly reported pathogens (Figure 78). Among non-viral infectious agents, *Chlamydia trachomatis* (20%; 3,883), *Bordetella pertussis* (7%; 1,313) and *Mycoplasma pneumoniae* (5%; 1,035) were the most commonly reported pathogens.

Figure 78. Reports of viral infections to the Laboratory Virology and Serology Reporting Scheme, 2006, by viral group

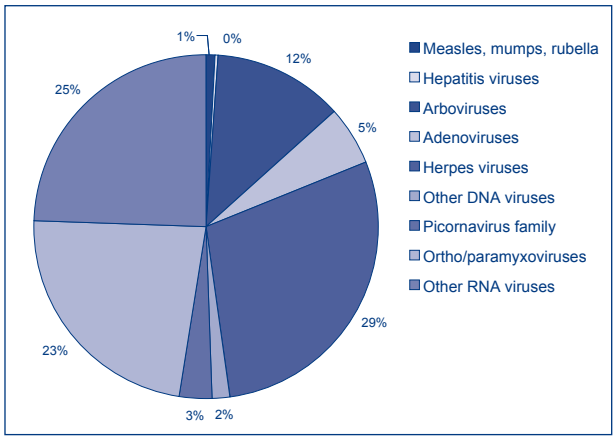


Table 22. Infectious agents reported to the Laboratory Virology and Serology Reporting Scheme (LabVISE) 2006, by state or territory

Organism	State or territory								Total 2006	Total 2005
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Measles virus	–	27	–	4	10	6	10	–	57	8
Mumps virus	1	–	1	7	4	–	14	–	27	38
Rubella virus	–	2	–	4	1	–	4	2	13	12
Hepatitis A virus	–	4	3	9	7	–	1	7	31	53
Hepatitis D virus	–	–	–	1	4	–	2	–	7	14
Hepatitis E virus	–	–	–	1	–	–	5	–	6	12
Ross River virus	–	41	45	662	222	1	31	60	1,062	452
Barmah Forest virus	–	10	–	127	146	–	6	–	289	185
Flavivirus (unspecified)	–	1	–	37	–	–	9	–	47	37
Adenovirus type 1	–	–	–	–	–	–	3	–	3	7
Adenovirus not typed/pending	10	284	1	38	163	–	126	–	622	680
Herpes virus type 6	–	–	–	–	–	–	4	–	4	2
Cytomegalovirus	4	221	1	95	410	17	111	3	862	1,042
Varicella-zoster virus	3	137	1	583	266	7	40	–	1,037	1,499
Epstein-Barr virus	–	16	93	483	428	7	34	384	1,445	2,148
Molluscum contagiosum	–	–	–	–	–	–	1	–	1	–
Poxvirus group not typed	–	–	–	–	–	–	3	–	3	2
Parvovirus	–	5	–	77	78	1	25	–	186	202
Coxsackievirus A9	–	16	–	–	–	–	–	–	16	3
Coxsackievirus A16	–	2	–	–	–	–	–	–	2	6
Echovirus type 34	–	1	–	–	–	–	–	–	1	–
Echovirus type 3	–	3	–	–	–	–	–	–	3	–
Echovirus type 5	–	2	–	–	–	–	–	–	2	2
Echovirus type 8	–	1	–	–	–	–	–	–	1	–
Echovirus type 11	–	2	–	–	–	–	–	–	2	4
Echovirus type 18	–	2	–	–	–	–	–	–	2	14
Echovirus type 22	–	4	–	–	–	–	–	–	4	1
Echovirus type 30	–	16	–	–	–	–	–	–	16	36
Rhinovirus (all types)	1	182	–	–	20	1	1	1	206	329
Enterovirus not typed/pending	6	73	–	11	2	3	7	–	102	187
Picornavirus not typed	–	–	–	–	–	1	1	–	2	1
Influenza A virus	3	79	–	69	68	4	114	–	337	708
Influenza A virus H3N2	–	1	–	–	–	–	–	–	1	2
Influenza B virus	–	42	–	15	81	–	34	–	172	257
Parainfluenza virus type 1	–	25	–	–	26	–	23	–	74	64
Parainfluenza virus type 2	–	9	–	1	5	–	–	–	15	49
Parainfluenza virus type 3	1	103	–	15	54	–	46	–	219	390
Respiratory syncytial virus	–	846	1	186	375	28	372	–	1,808	1,679
HTLV-1	–	–	–	–	6	–	–	–	6	9
Rotavirus	3	579	–	–	323	77	301	2	1,285	1,270
Astrovirus	–	–	–	–	–	–	1	–	1	4
Norwalk agent	–	25	–	–	–	–	1,513	–	1,538	267
Chlamydia trachomatis not typed	22	874	1	1,558	1,322	54	50	2	3,883	5,049
Chlamydia pneumoniae	–	–	–	–	–	–	1	–	1	8
Chlamydia psittaci	–	7	–	–	1	–	57	–	65	53
Chlamydia spp typing pending	–	1	–	–	–	–	–	–	1	–

Table 22. Infectious agents reported to the Laboratory Virology and Serology Reporting Scheme (LabVISE) 2006, by state or territory, *continued*

Organism	State or territory								Total 2006	Total 2005
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Chlamydia species	–	1	–	–	–	–	1	–	2	1
Mycoplasma pneumoniae	1	28	12	384	273	26	210	101	1,035	1,309
Mycoplasma hominis	–	23	–	–	–	–	–	–	23	7
Coxiella burnetii (Q fever)	–	4	3	32	44	1	20	–	104	162
Rickettsia prowazeki	–	–	–	–	24	–	–	–	24	161
Orentia tsutsugamushi	–	–	–	–	25	–	1	–	26	71
Rickettsia – spotted fever group	–	–	–	–	85	2	–	–	87	236
Streptococcus group A	–	6	–	294	–	–	77	–	377	609
Yersinia enterocolitica	–	4	–	1	–	–	–	–	5	6
Brucella species	–	2	–	3	–	–	–	–	5	14
Bordetella pertussis	2	45	3	157	1,003	1	102	–	1,313	1,573
Legionella pneumophila	–	7	–	–	13	–	8	–	28	23
Legionella longbeachae	1	–	–	–	10	–	10	–	21	51
Legionella species	–	–	–	–	–	–	1	–	1	1
Cryptococcus species	–	2	–	6	11	–	–	–	19	41
Leptospira species	–	2	–	9	7	–	–	–	18	33
Treponema pallidum	–	164	3	363	250	–	6	–	786	1,086
Entamoeba histolytica	–	–	–	1	–	–	–	–	1	14
Toxoplasma gondii	–	8	–	7	10	4	10	–	39	45
Echinococcus granulosus	–	–	–	–	3	–	–	–	3	10
Total	58	3,939	168	5,240	5,780	241	3,396	562	19,384	22,238

– No data received.

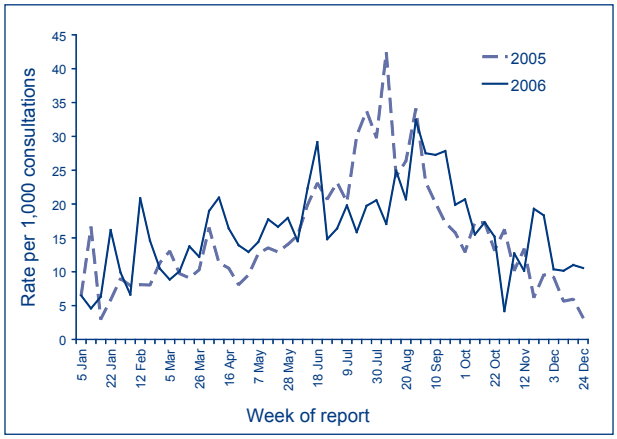
Australian Sentinel Practice Research Network

The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a national network of general practitioners that report each week on a number of conditions selected annually. Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia. The data provide an indicator of the burden of disease in the primary care setting and allow trends in consultation rates to be detected.

In 2006, influenza-like illnesses (ILI), gastroenteritis, and varicella infections (chickenpox and shingles) were the communicable diseases reported to ASPREN. Each week an average of 27 general practitioners (range 11–39) provided information from an average of 2,654 (range 934–3,999) consultations per week. The average number of participating practices and consultations has decreased since 2003.

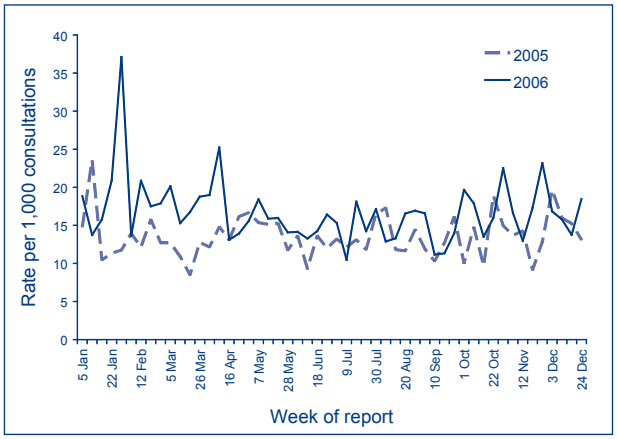
During 2006, influenza-like illness reports to ASPREN started increasing in week 24, with peaks in weeks 25 (29.2 cases per 1,000 consultations, mid-June) and 35 (32.4 cases per 1,000, late August). In 2005, ILI reports peaked in early August (42.4 cases per 1,000) (Figure 79).

Figure 79. Consultation rates for influenza-like illness, ASPREN 2006 compared with 2005, by week of report



Consultations for gastroenteritis fluctuated between 10.5 to 37.1 cases per 1,000 consultations. Rates reported for 2006 appeared to be slightly higher until April and then became very similar to 2005 (Figure 80).

Figure 80. Consultation rates for gastroenteritis, ASPREN, 2006 compared with 2005, by week of report



Consultations for varicella zoster (shingles) fluctuated between 0.4 to 6.2 cases per 1,000 consultations. Rates reported for 2006 appeared similar to 2005 from 16 April (Figure 81). Reports of varicella zoster (shingles) were available only from week 13 in 2005.

Consultations for varicella zoster (chickenpox) fluctuated between 0 to 3.3 cases per 1,000 consultations. Rates reported for 2006 appeared to be slightly less than 2005 from 16 April. Reports of varicella zoster (chickenpox) were available only from week 13 in 2005 (Figure 82).

Figure 81. Consultation rates for varicella zoster (shingles), ASPREN, 2006 compared with 2005, by week of report

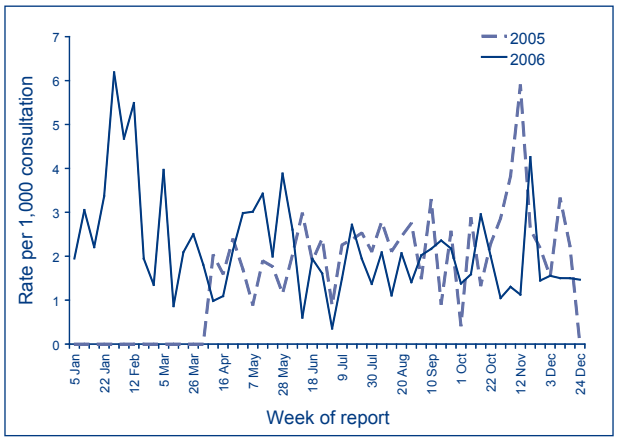
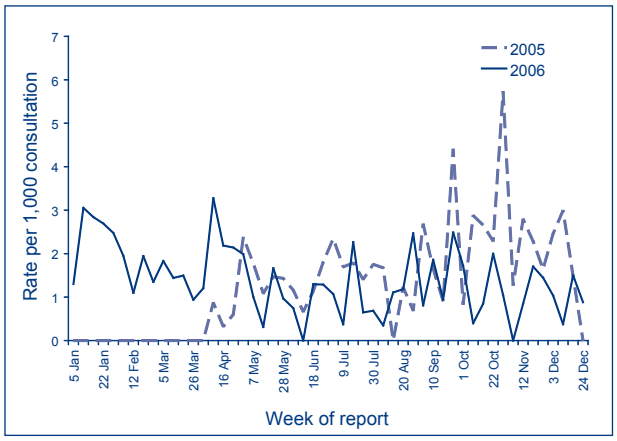


Figure 82. Consultation rates for varicella zoster (chickenpox), ASPREN, 2006 compared with 2005, by week of report



Appendices

Appendix 1. Mid-year estimate of Australian population, 2006, by state or territory

	State or territory								Aust.*
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Male	163,008	3,397,689	109,217	2,029,383	770,793	241,359	2,514,871	1,029,715	10,257,418
Female	165,809	3,430,005	97,471	2,024,061	783,863	247,589	2,576,795	1,021,169	10,348,070
Total	328,817	6,827,694	206,688	4,053,444	1,554,656	488,948	5,091,666	2,050,884	20,605,488

* Includes other territories.

Appendix 2. Mid-year estimate of Australian population, 2006, by state or territory and age group

Age	State or territory								Aust.*
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
0–4	20,583	419,143	17,746	258,643	88,725	30,341	311,755	128,008	1,275,165
5–9	20,341	435,279	16,492	271,248	93,971	31,845	315,725	134,121	1,319,250
10–14	21,506	454,682	16,436	286,675	101,067	34,132	333,930	142,220	1,390,910
15–19	23,713	458,434	15,211	284,088	103,019	34,184	339,951	146,554	1,405,419
20–24	28,406	467,748	16,698	294,079	106,552	31,461	361,189	147,154	1,453,429
25–29	26,275	464,560	17,170	271,463	95,566	26,927	352,002	137,870	1,391,964
30–34	25,174	498,740	17,979	290,904	100,381	29,247	371,984	146,203	1,480,776
35–39	24,613	493,828	17,407	296,793	109,703	32,958	385,208	153,876	1,514,579
40–44	24,281	502,157	16,289	299,270	114,406	35,204	376,518	155,808	1,524,169
45–49	24,005	490,975	14,620	290,993	114,967	36,823	365,395	152,132	1,490,120
50–54	22,410	443,890	12,835	263,103	105,859	34,401	330,423	139,070	1,352,213
55–59	20,921	422,039	10,821	253,560	102,967	33,373	312,573	128,905	1,285,321
60–64	14,300	330,540	6,769	196,400	79,600	26,146	240,209	94,003	988,066
65–69	10,185	268,111	4,407	151,903	64,304	21,461	196,318	75,006	791,770
70–74	7,576	219,188	2,399	115,823	53,497	16,757	161,003	58,083	634,371
75–79	6,155	195,190	1,729	98,565	49,927	14,326	143,560	48,778	558,247
80–84	4,768	144,501	887	71,408	38,377	10,765	106,571	34,446	411,726
85+	3,605	118,689	793	58,526	31,768	8,597	87,352	28,647	337,993
Total	328,817	6,827,694	206,688	4,053,444	1,554,656	488,948	5,091,666	2,050,884	20,605,488

* Includes other territories

Appendix 3. Completeness of National Notifiable Diseases Surveillance System data received, Australia, 2006, by state or territory

	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total notifications	2,173	33,926	6,512	38,201	12,786	2,520	27,456	15,019	138,593
Sex									
Unknown/missing	5	114	3	4	2	0	189	0	317
Per cent complete*	99.8	99.7	100.0	100.0	100.0	100.0	99.3	100.0	99.8
Age									
Unknown/missing	0	3	3	0	10	6	31	2	55
Per cent complete*	100.0	100.0	100.0	100.0	99.9	99.8	99.9	100.0	100.0
Indigenous status†									
Unknown/missing	2,011	26,932	557	25,233	2,030	1,210	12,840	4,264	75,077
Per cent complete*	7.5	20.6	91.4	33.9	84.1	52.0	53.2	71.6	45.8

* Data completeness = (Total – Unknown or missing)/Total x100

† 'Indigenous status' is a variable defined by the following values:

1=Indigenous – (Aboriginal but not Torres Strait Islander origin).

2=Indigenous – (Torres Strait Islander but not Aboriginal origin).

3=Indigenous – (Aboriginal and Torres Strait Islander origin).

4=Not indigenous – (not Aboriginal or Torres Strait Islander origin).

9=Not stated.

Blank/missing/null=No information provided.

Abbreviations

AFP	acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AMSP	Australian Meningococcal Surveillance Programme
ASPREN	Australian Sentinel Practice and Research Network
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
DoHA	Department of Health and Ageing
DTPa	diphtheria-tetanus-acellular pertussis
Hib	*Haemophilus influenzae* type b
HPAIH	highly pathogenic avian influenza in humans
HPV	human papilloma virus
HUS	haemolytic uraemic syndrome
IHR	International Health Regulations
ILI	influenza-like illness
IPD	invasive pneumococcal disease
IPV	inactivated poliovirus vaccine
LabVISE	Laboratory Virology and Serology Reporting Scheme
MMR	measles-mumps-rubella
MSM	men who have sex with men
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NIP	National Immunisation Program
NNDSS	National Notifiable Diseases System
OPV	oral poliovirus vaccine
PEC	Poliovirus Expert Committee
SARS	severe acute respiratory syndrome
STEC	Shiga toxin-producing *Escherichia coli*
STI	sexually transmissible infections
TB	tuberculosis
VAPP	vaccine associated paralytic poliomyelitis
VDPV	vaccine-derived poliovirus
VPD(s)	vaccine preventable disease(s)
VTEC	verotoxigenic *Escherichia coli*
WHO	World Health Organization

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Annual report of the National Influenza Surveillance Scheme, 2007

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Abstract

The year 2007 saw the most severe influenza season since national reporting of influenza began in 2001. Early in the season the National Incident Room was activated to provide effective national surveillance, reporting and management of the 2007 seasonal influenza outbreak. A surveillance team were tasked with establishing enhanced surveillance for the 2007 season and investigating unusual events in this outbreak. Key data required to comprehensively describe the number of cases, morbidity, mortality and virology of the influenza outbreak and the possible sources of these data were identified. In 2007 the number of laboratory-confirmed notifications for influenza was 3.1 times higher than the five-year mean. Forty-four per cent of notifications occurred in Queensland. High notification rates were reflected in an increase in presentations with influenza-like illness to sentinel general practices and Emergency Departments. Notifications and notification rates were highest in the 0–4 and 5–9 years age groups, possibly due to a bias towards testing in these age groups. The clinical morbidity of the infection in terms of complications or most affected groups cannot be determined but anecdotal reports indicate this season may have impacted young adults more than is usual. The available data suggest influenza has caused a significant burden on workplaces and the health care system as indicated by data on absenteeism and presentations for health care. The proportion of H1 strains of influenza circulating varied across Australia but was higher than 2006 in most jurisdictions. In 2007, 1,406 influenza isolates from Australia were antigenically analysed at the World Health Organization Collaborating Centre for Reference and Research on Influenza in Melbourne: 58.7% were A(H3N2), 34.4% were A(H1N1) and 6.9% were influenza B viruses. Antigenic drift away from the vaccine strain A/Wisconsin/67/2005 was observed with the A(H3N2) viruses and was also seen with most of the A(H1N1) viruses when compared with the vaccine strain A/New Caledonia/20/99. The small number of influenza B viruses examined were predominately of the B/Yamagata-lineage. Monitoring influenza through the National Incident Room during the 2007 season offered an excellent opportunity to conduct enhanced surveillance under conditions that were real and potentially serious but not an emergency. It enabled the current state of our surveillance systems to be assessed and opportunities for improvement to be identified. *Commun Dis Intell* 2008;32:208–226.

Keywords: influenza, surveillance, vaccine, influenza-like illness, sentinel surveillance

Introduction

Influenza or 'the flu' is a common, highly infectious respiratory viral disease. The virus spreads from person to person by airborne droplets of exhaled respiratory secretions, especially by coughing or sneezing.[1] Typical symptoms include sudden onset of fever, sore throat, runny nose, cough, fatigue, headache, and aches and pains.

Influenza causes annual epidemics of respiratory disease. Influenza epidemics usually occur during the winter months in temperate climates, causing an increase in hospitalisations for pneumonia, an exacerbation of chronic diseases and also contributing to increased mortality. Those most susceptible include the elderly and very young people, or people of any age who have a higher risk of complications (e.g. pneumonia, heart failure) due to certain chronic medical conditions, e.g. heart, lung, kidney, liver, immune, or metabolic diseases. Most healthy children and adults only have minor symptoms.

Laboratory-confirmed influenza is a nationally notifiable disease in all states and territories except South Australia and data are reported from each state or territory health department to the National Notifiable Disease Surveillance System (NNDSS). In temperate zones of Australia, the annual influenza season runs from May to October, with a peak in notifications around the middle of August. The severity of seasons varies from year to year. Australia experienced a moderate to severe season in 2003 but a mild season in 2006. The start of the annual influenza season is usually first detected by increased presentations at general practitioners (GPs) of 'influenza-like illness' (ILI) followed by increases in notifications of laboratory-confirmed influenza.

Surveillance methods

Data used to describe the 2007 influenza season were classified under the areas of epidemiology, morbidity, mortality and virology. Influenza surveillance was based on the following sources of data:

- notifications of laboratory-confirmed influenza required by legislation in most states and territories, and notified to the National Notifiable Diseases Surveillance System;

- subtype and strain data of circulating influenza viruses provided by the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza;
- consultation rates for ILI diagnosed by sentinel general practitioners;
- consultation rates for ILI diagnosed by New South Wales sentinel hospital emergency departments;
- testing rates for influenza by New South Wales sentinel laboratories;
- media monitoring of reports of influenza outbreaks and deaths or of influenza impacts on hospitals;
- absenteeism data from a national employer; and
- New South Wales and Australian Bureau of Statistics (ABS) mortality data.

National Notifiable Diseases Surveillance System

In 2007, laboratory-confirmed influenza was a notifiable disease under state and territory legislation in all jurisdictions except South Australia. Laboratory notifications were sent to NNDSS for national collation. Although influenza was not a notifiable condition in South Australia, laboratory notifications were generally provided to NNDSS. In this report, data were analysed by the date of onset, but when this was not available the earliest date from either the specimen collection date or notification date was used.

Age, sex, method of laboratory diagnosis and postcode or locality of patient residence was included in NNDSS notifications. Maps were produced using ArcGIS.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor consultations for ILI. In Australia, there are five such schemes: the Australian Sentinel Practice Research Network (ASPREN), which collects data at a national level from approximately 90 general practitioners from six states and territories (Australian Capital Territory, New South Wales, Queensland, South Australia, Victoria, Western Australia); the Queensland Influenza-like Illness Sentinel Surveillance in General Practice Program; the Victorian Influenza Surveillance Scheme; Western Australian sentinel general practices; and the Northern Territory Tropical Influenza Surveillance Scheme. ASPREN and the Northern Territory Tropical Influenza Surveillance Scheme report ILI rates throughout the year, while the other sentinel surveillance schemes report only from May to October each year. The national case definition of ILI is: presentation with fever, cough and fatigue. All sentinel surveillance schemes, including ASPREN, used the national case definition for ILI in 2007.

Emergency department surveillance

Rates for influenza-like illness presentation were collected from 30 emergency departments (ED) across New South Wales and data were provided to the Surveillance Branch, Office of Health Protection (OHP) within the Australian Government Department of Health and Ageing (DoHA), on a weekly basis, through the New South Wales Influenza Surveillance Report.

Laboratory surveillance

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centres for Reference and Research on Influenza located in Australia, Japan, the United Kingdom and the United States of America, are responsible for analysing influenza viruses collected through an international surveillance network involving 122 National Influenza Centres in 94 countries. The Melbourne Centre analyses viruses received from Australia and from laboratories throughout Oceania, the Asian region and beyond. All virus isolates are analysed antigenically and a geographically and temporally representative number of viruses, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation, are further analysed by genetic sequencing of the viral haemagglutinin gene and the neuraminidase gene. Together with serological and epidemiological data, this forms the basis from which WHO makes recommendations in February (for the Northern Hemisphere) and in September (for the Southern Hemisphere) for the vaccine formulation to be used in the following winter.

WHO vaccine formulation recommendations are made in the context of strains that are antigenically 'like' laboratory reference strains that are named according to a standard nomenclature for influenza viruses. For human isolates this nomenclature is based on type, the place of isolation, sequential number and year of isolation and for influenza A, the subtype of the HA and NA may also be included in brackets after the designation. An example of a human isolate is A/Sydney/5/97(H3N2), an influenza A(H3N2) virus that was the 5th sequential influenza A isolated in Sydney in the year 1997. The WHO recommendations[2] are then translated into actual virus strains acceptable to regulatory authorities and vaccine manufacturers by national and regional committees (such as the Australian Influenza Vaccine Committee.)[3]

The New South Wales sentinel laboratory network collects influenza virology testing data from six major public laboratories and influenza serology testing data from three. The number of laboratory requests for influenza laboratory tests were obtained weekly from New South Wales Influenza Surveillance Reports.

Absenteeism surveillance

A major nationwide employer, provided sick leave absenteeism data collected weekly for 2007. Absenteeism, defined as an absence for any reason for 3 or more consecutive days, was presented as a rate per 100 employees per week, on an average of 32,798 employees per week.

Media surveillance

Media information was sourced from Australian media clippings (Google News, Media Monitor) for the period 1 July to 30 August 2007. Clippings were scanned for articles relating to the 2007 influenza outbreak, particularly those relating to health system capacity.

Mortality

Death certificate data from the New South Wales Registry of Births, Deaths and Marriages provided an estimate of the number of deaths from pneumonia and influenza in New South Wales and compared the rate per 1,000 deaths with predicted seasonal mean plus a 95% confidence interval. These were obtained weekly from the New South Wales Influenza Surveillance Report.

Deaths data compiled by the ABS from information provided by the state and territory Registrars of Births, Deaths and Marriages, and coded using the tenth revision of the *International Classification of Diseases and Related Health Problems* (ICD-10) were used to estimate historical levels of influenza deaths. In this report, deaths for 2006 with an underlying cause of influenza and pneumonia (ICD-10 J10–J18) are presented.[4]

Information on the sudden child deaths which were a feature of this year's season, was obtained during teleconferences or through the media.

Morbidity data

There was no effective measure of morbidity of disease readily available during the influenza season. Instead, morbidity was assessed through a number of indicators including:

- absenteeism surveillance;
- Paediatric Intensive Care Unit admissions to intensive care units (ICUs) and deaths collected by the Australian Paediatrics Surveillance Unit (APSU);
- emergency department presentations for ILI in New South Wales;
- ILI presentations to GP surveillance and ED networks; and
- media reports.

Hospital admissions for influenza and pneumonia were not available during the season.

Results

The 2007 influenza season began in late May with a very gradual increase in notifications above non-seasonal levels from week 21 (week ending 27 May). At this time, consultation rates for ILI were similar to those of 2006 with small increases evident in most jurisdictions.

Increases in influenza notifications to the National Notifiable Diseases Surveillance System

At the end of July 2007, routine analysis of NNDSS data by epidemiologists in the Surveillance Branch of OHP identified a steep rise in influenza notifications diagnosed from week 28 (week ending 15 July), with levels reaching those of the peak in the 2003 season. Further analysis showed that the national increase was due to an increase in notifications in several jurisdictions, particularly Queensland and Western Australia. While it was thought likely that the increase in Western Australia was an effect of the increased media following the child deaths in that state, it was thought unlikely that this was the cause of the increase in Queensland. By this time influenza notifications in several jurisdictions had already exceeded the highest levels recorded in recent years.

Antigenic shift

Analysis of NNDSS influenza typing data indicated a change in the proportion of circulating influenza subtypes from that seen in previous years. Typing data for Queensland showed an increase in the proportion of type A(H1) relative to type A(H3). This change was reflected in the isolates typed by the World Health Organization Collaborating Centre for Reference and Research on Influenza (WHOCC) to that time, of which a significant proportion of cases were due to type A(H1).

The increase seen in influenza notifications was discussed with, and confirmed by Queensland Health. A Communicable Diseases Network Australia/

Jurisdictional Executive Group teleconference was held to discuss the epidemiology of influenza in each jurisdiction.

Jurisdictional representatives reported that influenza notifications were higher than recent influenza seasons in Queensland, New South Wales, Western Australia, the Australian Capital Territory and Tasmania. Notifications had started to rise in Victoria and the Northern Territory. South Australia was experiencing a moderate level of cases. Similar increases were seen in ILI consultation data from sentinel general practitioner sites. The age and sex distributions of influenza notifications were the same as in previous years.

The Queensland representative confirmed an increase in type A(H1) with 44% being type A(H1) compared with 1%–5% in previous years and 25% nationally.

The WHOCC reported that early testing showed a difference in the proportions of H1 and H3 strains across jurisdictions. Western Australian and Victorian isolates were mainly type A(H3) while Queensland and New South Wales isolates were a mixture of type A(H1) and type A(H3). In recent years, 2005 was the only season with a significant proportion of type A(H1) strains circulating with other seasons being predominantly type A(H3).

The WHOCC also reported that the circulating H1 strain appeared to be a genetic drift of the 2007 vaccine strain, but that the 2007 vaccine was expected to provide protection. The circulating A(H1) strains were a mixture of the vaccine strain A/New Caledonia/20/99, and the drift strain A/Solomon Islands/3/2006-like which was not in the vaccine. All Queensland H1 isolates were A/Solomon Islands/3/2006–like.

Laboratory confirmed cases

The first increases in notifications of laboratory-confirmed influenza in the 2007 season were registered in late May (week 21) when 35 cases were diagnosed. Notifications peaked in mid-August (week 33) and were almost back to inter-seasonal levels by the end of October (week 44) (Figure 1). The total number of notifications for the year was 10,577, which was 3.1 times the 5 year mean (Figure 2).

It should be noted that South Australia ceased sending influenza data to NNDSS on 31 August after changing to a new state database.

The median time from diagnosis to notification was estimated at 4 days in 2007, with 90% of notifications received within 14 days. Twenty-six per cent of notifications were received after seven days and 10% after 14 days. This time was calculated as the earliest recorded date (either onset date, diagnosis date

Figure 1. Laboratory-confirmed influenza notifications, 2007, by state or territory and week of diagnosis

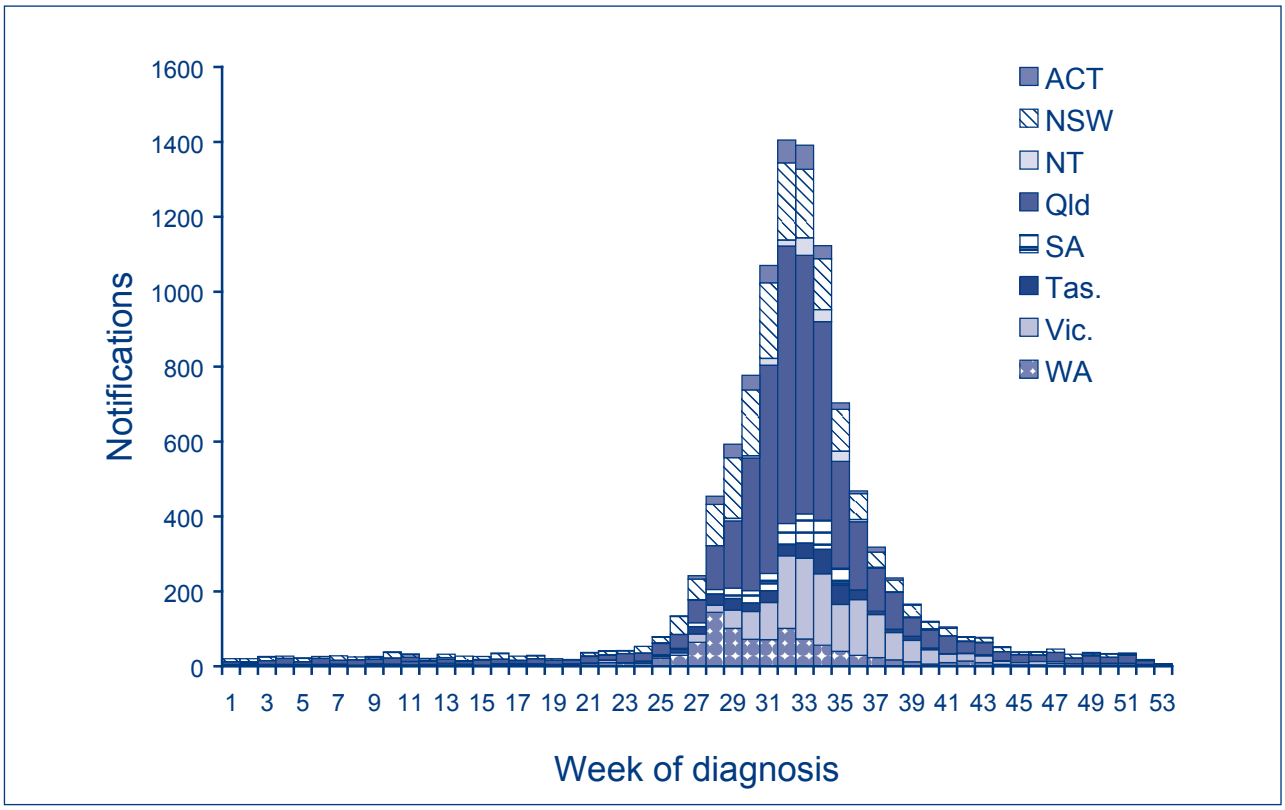


Figure 2. Laboratory-confirmed influenza notifications, 2003 to 2007, Australia, by month and year of diagnosis

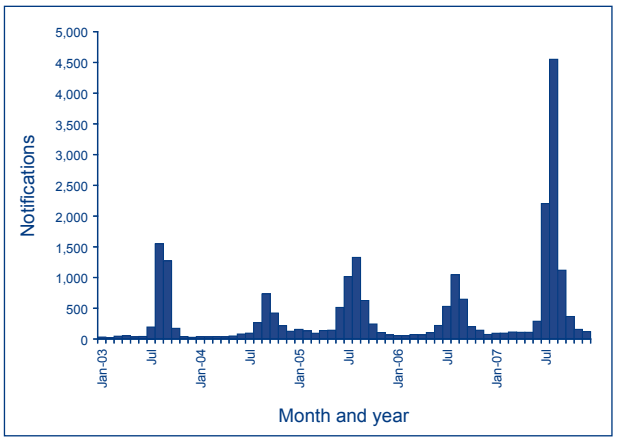
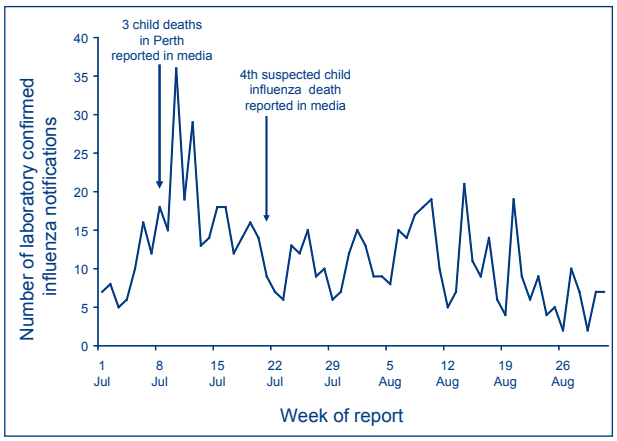


Figure 3. Number of laboratory confirmed notifications of influenza following paediatric deaths in Western Australia, National Notifiable Diseases Surveillance System, July to August 2007, by day of report



or laboratory date) to the date the information was received by the state or territory health department. In reality the timeliness was longer than this because of the additional time taken for notifications to be sent from the health department to NNDSS.

NNDSS laboratory confirmed notifications represent an unknown proportion of all influenza cases. Jurisdictions vary in the number and representativeness of samples they provide for laboratory confirmation and this affects the number of cases diagnosed and notified. The number of actual cases that each confirmed case represents varies between jurisdictions and between urban, rural and remote areas.

Media coverage of influenza deaths may also have increased the rate of presentations for health care and testing for influenza in children and thus laboratory diagnosis and notification. There appears to have been a large increase in influenza notifications from Western Australia following the deaths of 3 children, which were due to influenza (Figure 3).

Influenza-like illness consultations from sentinel general practitioner surveillance systems

Data from the ASPREN Sentinel GP Surveillance System showed that for 2007 there were 6,125 notifications for ILI. An average of 63 doctors reported to ASPREN each week, with an average of 6,437 consultations per week (range 2,530–9,356).

The rate of consultations for ILI began to increase in early June, peaking mid-July to mid-August and began to drop at the end of August (Figure 4). The rate of consultations for ILI mirrors that of laboratory-confirmed influenza notifications but increases in ILI consultation rates precedes increases in notifications by 1 to 2 weeks. The peak rate of ILI consultations to ASPREN in 2007 was approximately 50% higher than the peak rate reported in 2006.

Rates of ILI in individual state and territory sentinel GP surveillance systems, in general, mirrored the rates of influenza notifications in their jurisdictions (Figure 5). Western Australia was the only state where ILI consultation rates were lower in 2007 than in 2006.

Data provided by ASPREN were aggregated and did not allow for further analysis. A breakdown of sentinel GP data by state, postcode, age and sex would help to identify the demographics and geographic location of people presenting to general practitioners with ILI.

Figure 4. Consultation rates for influenza-like illness, ASPREN, 2006 and 2007, by week of report

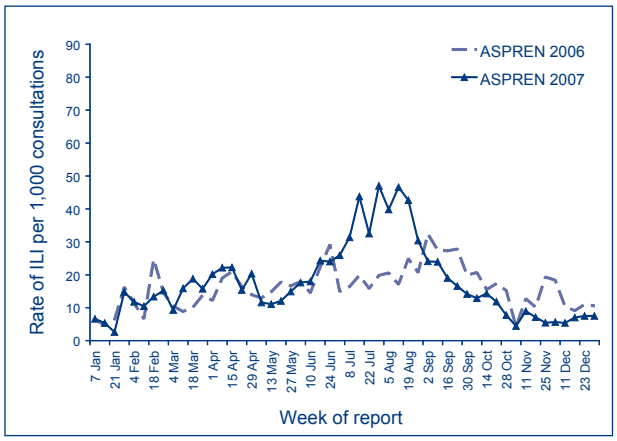
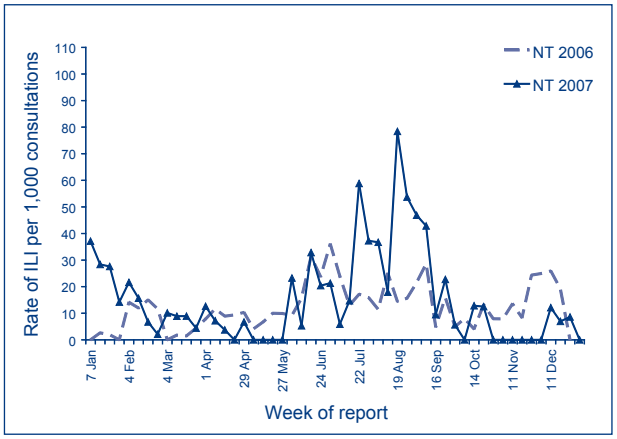
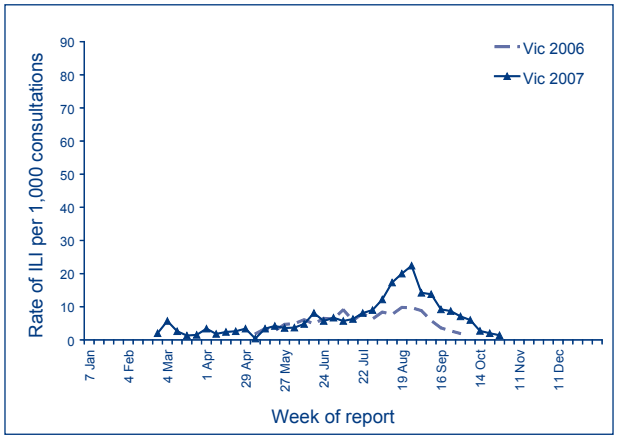


Figure 5. Consultation rates for influenza-like illness, 2006 and 2007, by sentinel surveillance scheme and week of report

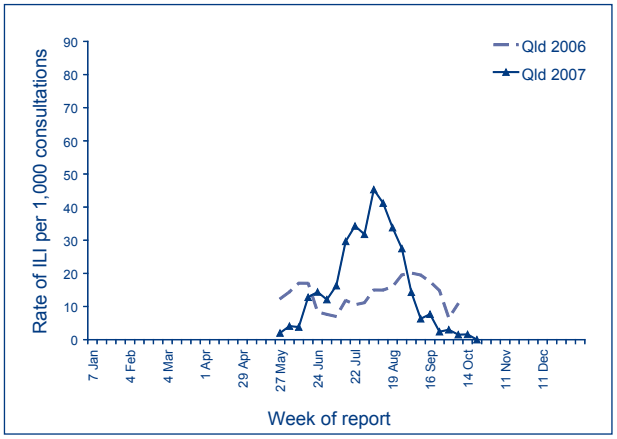
Northern Territory sentinel general practice



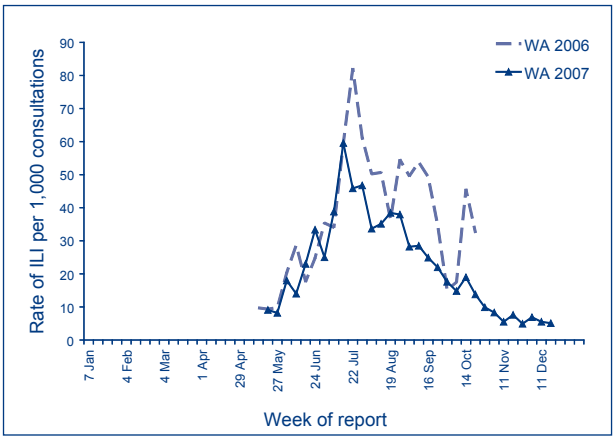
Victoria sentinel general practice



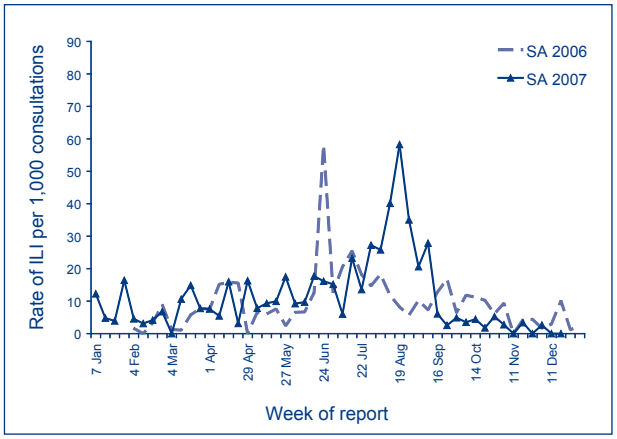
Queensland sentinel general practice



Western Australia sentinel general practice



South Australia sentinel general practice



While most ASPREN data are provided within a week of collection from GPs, some delay in reporting by some GPs mean that ILI rates are not stable for up to 3 weeks. Improvements to the timeliness of ASPREN data is important if increases in ILI are to be relied upon for early warning of the start of the influenza season.

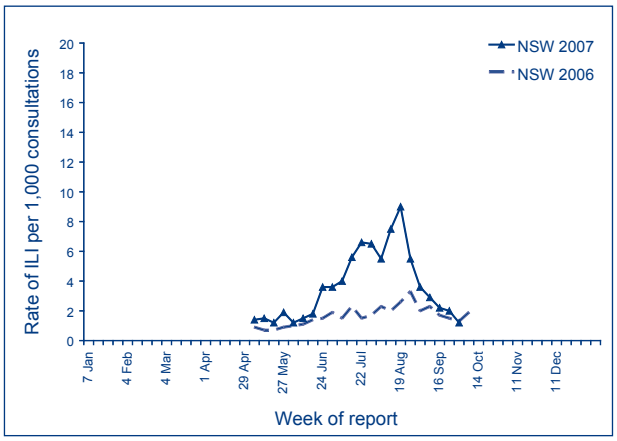
ASPREN data are not completely representative of the Australian population. There are no GPs from the Northern Territory or Tasmania that contribute data to ASPREN. Representativeness is also reduced with time due to declines in the number of reporting doctors.

No sampling of ILI patients for laboratory influenza testing occurred at a national level through the ASPREN scheme, although some state systems conducted random testing. The proportion positive of ILI presentations could not be determined.

Influenza-like illness – sentinel emergency department surveillance (New South Wales only)

Presentations to New South Wales emergency departments for ILI began to rise in mid-June and peaked at 9 per 1,000 consultations in mid-August (Figure 6). The increase in presentation rates reflected the rise in laboratory confirmed notifications of influenza to NNDSS. Presentation rates in 2007 exceeded those for any part of the 2006 influenza season.

Figure 6. Rate of influenza-like illness consultations from hospital emergency departments, New South Wales, April to September 2006 and 2007, by week of report



Sentinel ED surveillance data were timely but available from New South Wales only. ED surveillance systems operate in other jurisdictions but these do not routinely report data to the Australian Government Department of Health and Ageing.

Further work to identify how representative the ED data are and whether the demographics of people presenting with ILI to EDs are different to those who present to GPs will add to our understanding of this data set in future years.

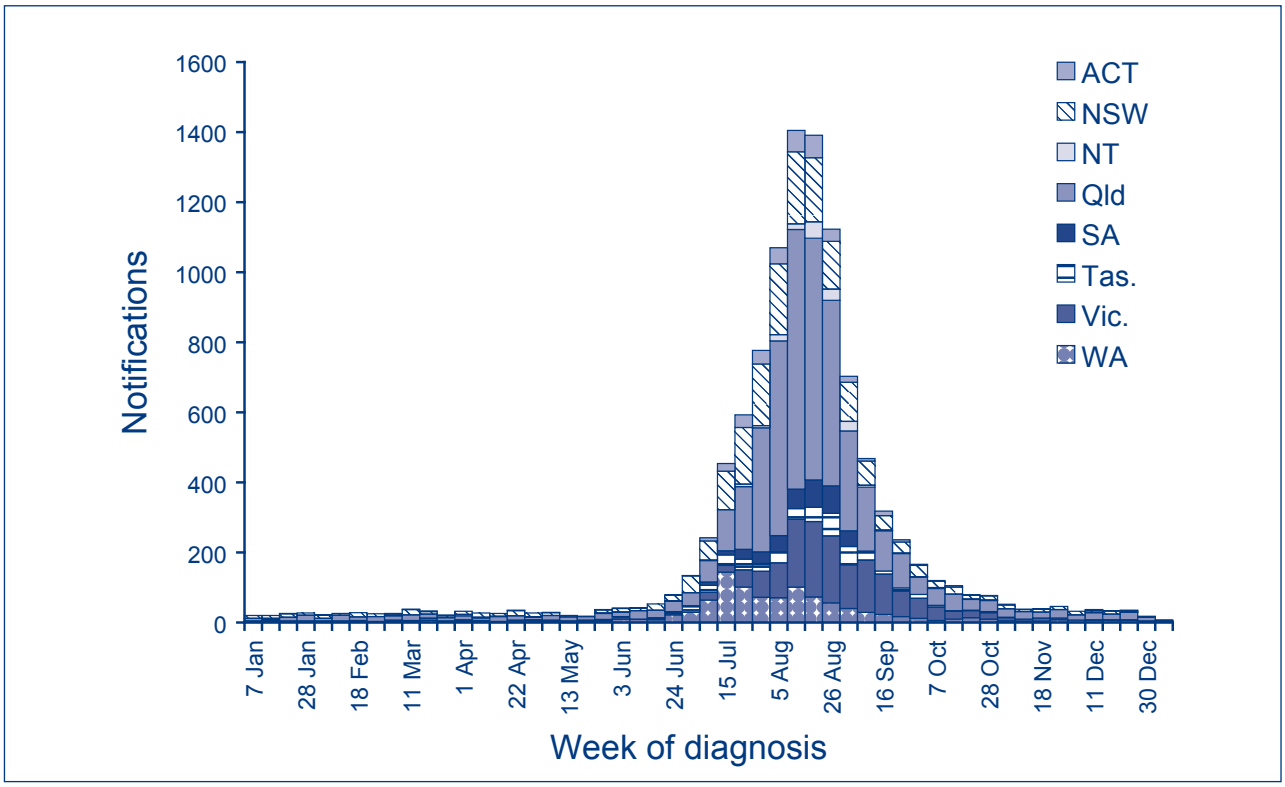
Geographic spread

In 2007, 44% of laboratory-confirmed influenza notifications occurred in Queensland, 18% in New South Wales, 15% in Victoria, 10% in Western Australia, 4% in South Australia, 4% in Tasmania, 4% in the Australian Capital Territory and 2% in the Northern Territory (Figure 7).

While the number of notifications peaked at a similar time in most jurisdictions, (at about week 33; week ending 19 August), the epidemic curve varies considerably by jurisdiction. Western Australia experienced a sharp, early increase in notifications at week 28 (week ending 15 July) likely caused by an increase in presentations following the influenza-related deaths of 3 children. A second peak occurred at about week 33. New South Wales also experienced an early increase in cases at week 28, while increases in Victoria commenced about four weeks later (week 32, week ending 12 August). In addition, notifications remained high for longer in Queensland, Western Australia and New South Wales (Figure 7).

Rates of notification for laboratory-confirmed influenza for 2007 varied across the country, ranging from 28 per 100,000 population in New South Wales to 115 per 100,000 population in the Australian Capital Territory. The rate of notification of influenza infec-

Figure 7. Laboratory-confirmed influenza notifications, May to October 2007, by state or territory and week of diagnosis



tion for Australia was 50 cases per 100,000 (Table 1). This was 3 times the mean rate over the previous 5 years (17.0 cases per 100,000 population).

The Map shows rates of laboratory-confirmed influenza in 2007 by Statistical Division. The highest rates of influenza occurred in statistical divisions, which encompassed the Northern Territory, northern Western Australia, parts of southern Western Australia, southern Queensland, Brisbane, the Australian Capital Territory and southern Tasmania.

Both state code (a mandatory code) and postcode of residence have high completion rates in NNDSS allowing the possibility of monitoring cases of influ-

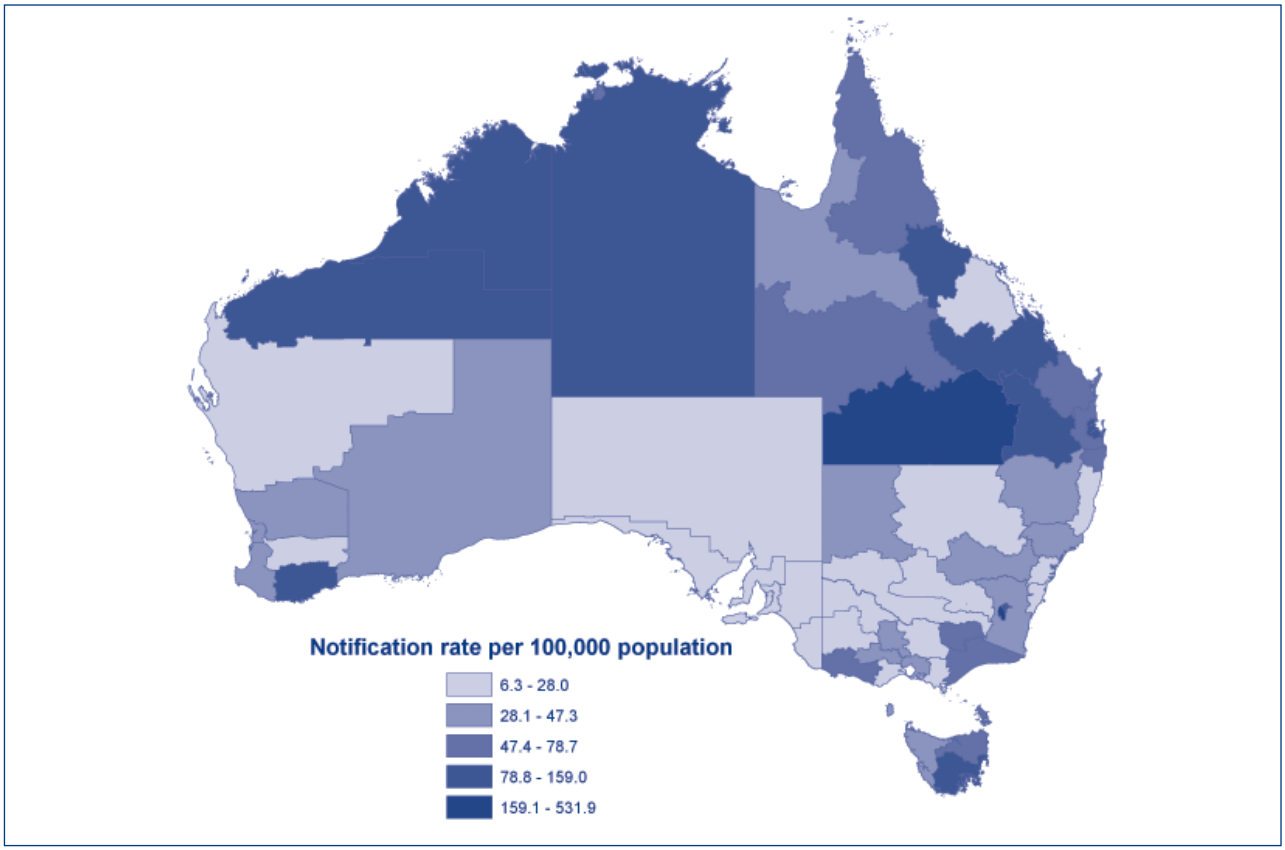
Table 1. Notifications and rates of laboratory-confirmed influenza notifications, 2007, by state or territory

State or territory	Total notifications	% of total	Notifications		Notification rate*		
			Male	Female	Male	Female	Total
Qld	4,644	43.9	2,215	2,429	106.0	116.0	111.0
NSW	1,918	18.1	991	923	29.0	26.6	27.8
Vic.	1,584	15.0	736	840	28.6	32.0	30.4
WA	1,038	9.8	522	516	49.0	49.6	49.3
SA	415†	3.9	180	234	74.0	93.6	84.1
Tas.	402	3.8	211	190	27.0	23.7	25.4
ACT	390	3.7	203	187	120.5	109.1	114.8
NT	186	1.8	109	77	97.6	74.5	86.5
Aus.	10,577	100.0	5,167	5,396	49.4	51.1	50.3

* Rate of notification per 100,000 population.

† South Australia ceased reporting notifications to the National Notifiable Diseases Surveillance System on 31 August 2007.

Map. Notification rates of laboratory-confirmed influenza, Australia, 2007, by Statistical Division of residence

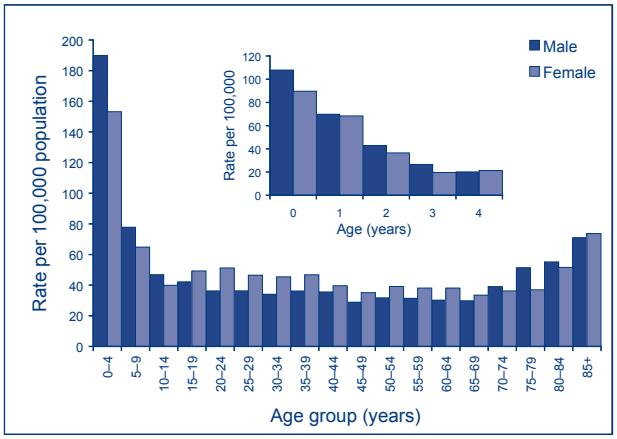


enza by time and location. Improved completion of typing data in NNDSS would also allow monitoring of virus type by location and time.

Age-sex profile

Age-specific notification rates for laboratory-confirmed influenza reported to the NNDSS in 2007 are shown in Figure 8. The highest notification rates were seen in those aged 0–4 years followed by the 5–9 years and 80–84 years age groups. Rates in the 0–4 years age group were around 3.4 times higher than for other age groups (170 cases per 100,000 population compared to an all-ages rate of 50 cases per 100,000 population).

Figure 8. Notification rate of laboratory-confirmed influenza, Australia, 2007, by age group and sex



People aged 65 years or over are the target for influenza vaccination as they are at an increased risk of complications from influenza. Notification rates for people in this age group were 43 cases per 100,000 population for both males and females. This compares with 2006 where influenza rates in 65 or over age group were 18 cases per 100,000 population for males and 17 cases per 100,000 population for females.

Forty-nine per cent of all notifications were male. Notifications were higher in females than in males for persons aged between 15 and 69 years. For children and the elderly, notifications for males exceeded those for females. The ratio of males to females in both the 0–4 years and 5–9 years age groups was 1.2:1.

Figure 9 shows rates of notifications for key age groups for the years 2003 to 2007. While rates increased in all age groups in 2007, the most marked were in the 0–4 and 5–9 year age groups.

Figure 9. Notification rate of laboratory-confirmed influenza reported to the National Notifiable Diseases Surveillance System, Australia, 2003 to 2007, by age group

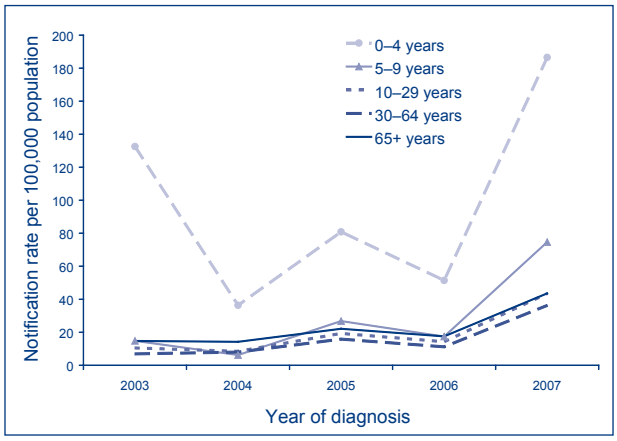
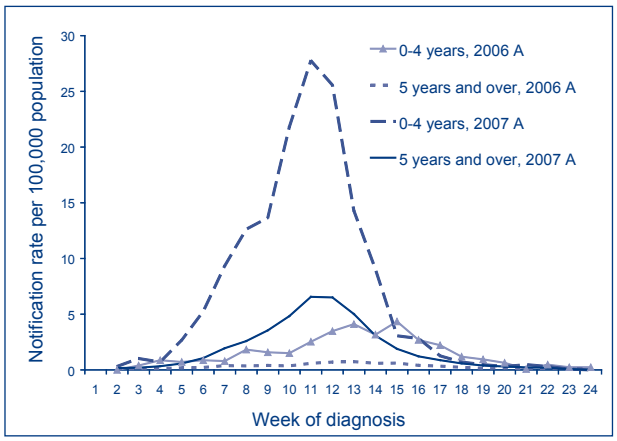


Figure 10 shows notification rates in key age groups in 2007 for influenza A. The data show that from mid-June, the notification rate for influenza A in children under 5 years of age increased significantly when compared to rates in other age groups. This increase was substantially higher than in 2006.

Figure 10. Notification rate of laboratory-confirmed influenza type A from week 23 to week 45, Australia, 2006 and 2007, by age group and week

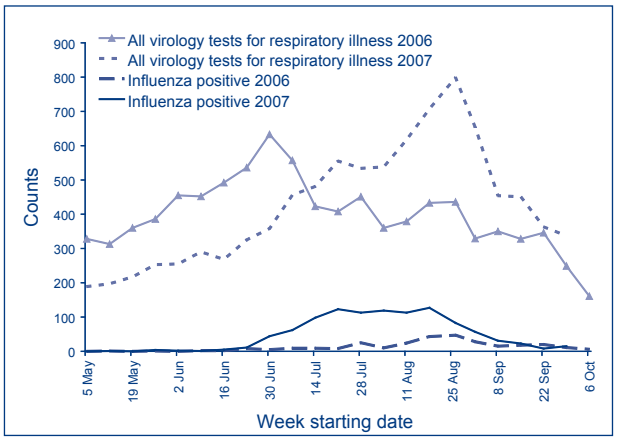


Laboratory surveillance

Data from New South Wales sentinel laboratory networks showed that the number of laboratory virology tests for respiratory illness (direct immunofluorescence, nucleic acid tests and viral culture) increased rapidly from July 2007, to over 800 tests per week in late August, exceeding the peak of approximately 600 tests per week in 2006 (Figure 11). The percentage of virological tests

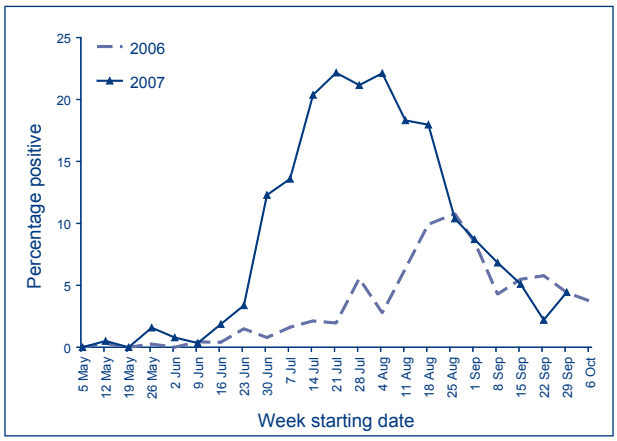
positive for influenza increased over the season from 1.9% in June to over 20% for the period between mid-July and early-August (Figure 12). Serology positive tests showed similar trends. This confirms that the increase in the number of cases of seasonal influenza was not an artefact of increased testing.

Figure 11. Total virology specimens tested and number positive for influenza A, New South Wales, May to October 2006 and 2007



Data source: NSW Influenza Surveillance Report, 22 to 28 September 2007

Figure 12. Percentage of virology specimens testing positive for influenza A, New South Wales, May to October 2006 and 2007



Data source: NSW Influenza Surveillance Report, 22 to 28 September 2007

Laboratory diagnosis method analysis

NNDSS notifications for influenza were analysed for laboratory diagnosis methods used.

Table 2 shows that during the period 1 June 2007 to 31 October 2007, 37% of laboratory-confirmed notifications were diagnosed by polymerase chain reaction (PCR) alone (n=3,545 of 9,672). Serology and culture-based diagnoses accounted for 21% and 9% of notifications respectively (n=3,545, n=880). Laboratory-confirmed influenza notifications based on antigen detection with and without confirmatory testing accounted for 16% of notifications (n=1,547) overall. The laboratory method was unknown in 14% of the total notifications for this period.

Trends in laboratory diagnosis methods used during the 2007 season are shown in Figure 13. At the peak of the season (week 33; week ending 19 August), 39% of notifications were diagnosed by PCR alone (n=559 of 1,441). Notifications based on antigen detection and antigen detection alone peaked in week 31 (16%, n=173). The proportion of weekly notifications based on less specific serological tests were low and use of these tests declined in the peak of the season.

Morbidity

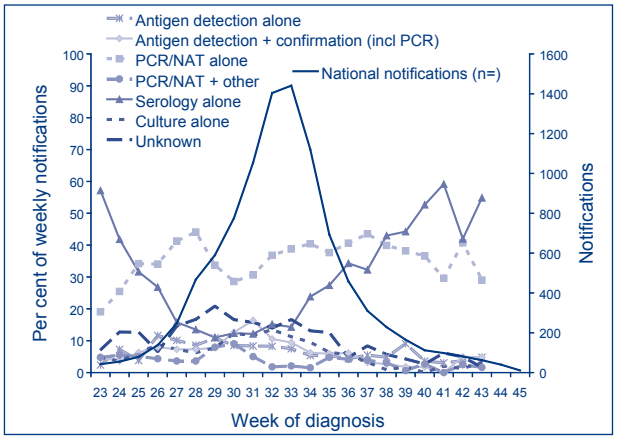
APSU surveillance

Results from the APSU survey commissioned by DoHA showed that for the month of September 2007, 15 cases aged under 5 years were admitted

Table 2. Laboratory diagnosis method of influenza notifications, Australia, 1 June to 31 October 2007

Laboratory diagnosis method	n	%
Antigen detection alone	704	7
Antigen detection + confirmation (including polymerase chain reaction)	843	9
Polymerase chain reaction or nucleic acid testing only	3,545	37
Polymerase chain reaction or nucleic acid testing + other laboratory test	361	4
Serology only	1,997	21
Culture only	880	9
Unknown laboratory method	1,342	14
Total	9,672	100

Figure 13. Percentage of weekly notifications reported to the National Notifiable Diseases Surveillance System, 4 June, 2007 (week 23) to 5 November (week 45), by method of laboratory diagnosis and week of diagnosis



to hospital with complications from influenza. Of the 15 cases, the ratio of male to female was 4:1 and 3 of the cases were identified as Aboriginal or Torres Strait Islander. Thirteen cases were influenza A and 2 were influenza B. The survey was not conducted for the whole of the influenza season.

Media reporting

Media reports discussed the clinical severity of the 2007 influenza strains by emphasising deaths (many descriptions of 'killer flu' in national print media) but also noted the impact of influenza on emergency department waiting times. Media clippings reported that during the 2007 influenza season:

- there were long waiting lists to see doctors;
- doctors were working longer hours;
- pharmacists saw increases in the demand for anti-virals and flu treatments;
- medical advice lines (such as Health Direct in Western Australia) had unprecedented demand with an 80% increase in calls;
- blood supplies were low because of illness in the donor population; and
- hospitals were regularly put on divert as the influenza season reached its peak.

In areas with a particularly severe influenza season, the media reported that hospitals were overwhelmed with both the sick and the worried well. In response to this, nurses worked extra shifts; holidays were cancelled; doctors were recruited from other departments to work evenings and weekends to relieve pressure; elective surgery was postponed; hospitals wards were closed down to enable staff to be freed up to work in other areas; and additional influenza clinics were opened next to the Emergency Departments.

Media also discussed absenteeism issues relating to the influenza season; recommendations that sick people stay at home and the use of masks by those who were infectious.

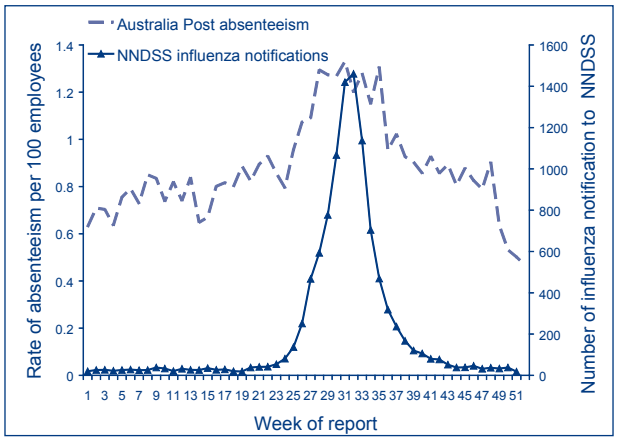
The media proved a useful mechanism for understanding public concerns relating to the influenza season. The media provides an early indicator of the perceived severity of the influenza season. Media can provide useful information that is not accessible through standard data sources. Following media attention on outbreaks, Emergency Departments may be overwhelmed with worried parents and the worried well.

Absenteeism surveillance

Absenteeism rates generally increase several weeks before notifications start increasing. This can provide an early warning system of the severity of the influenza season. Absenteeism is likely to be elevated by public health messages to stay home if unwell.

Absenteeism was higher during the 2007 influenza season than in the past five years. The absenteeism figures for 2007 (Figure 14) were the highest since collection commenced in 2002. The proportion of workers absent for more than 3 consecutive days peaked at 1.33% for the week ending 8 August 2007 (Week 31): the highest rate recorded since data has been collected.

Figure 14. National absenteeism (more than 3 consecutive days) rates and National Notifiable Diseases Surveillance System influenza notifications, 2007, by week of report



A comparison of absenteeism rates and laboratory-confirmed influenza notifications is shown in Figure 14. The 2007 figures show a broader peak than the laboratory confirmed notifications with the highest rates of absenteeism during the period 12 July to 5 September 2007.

The absenteeism rate was only measured in workers and does not reflect an 'incapacity' rate among the population in general. There is also a time lag as illness is only reported on return to work.

Mortality

Mortality from a primary influenza infection is rare and most of the deaths attributed to influenza occur from complications including pneumonia, obstructive airways disease and sudden cardiac deaths. These occur predominantly in identified risk groups such as those over 65 years or under 6 months of age; or those with chronic medical conditions.

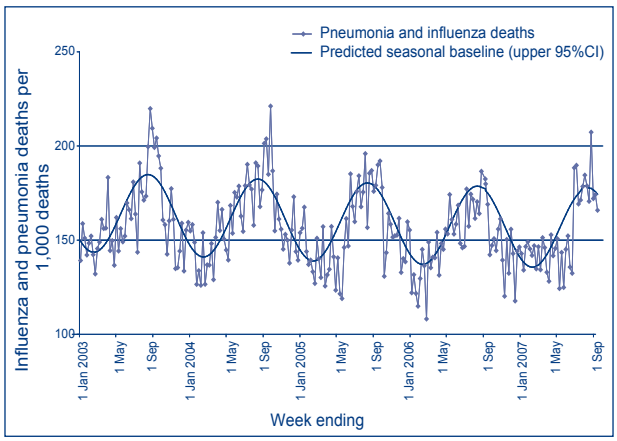
Deaths from pneumonia and influenza – New South Wales

Mortality rates from influenza in New South Wales reported by the Registry of Births, Deaths and Marriages showed that rates of deaths from influenza and pneumonia peaked in late August at 210 per 1,000 deaths (Figure 15). The combined pneumonia and influenza death rates were higher than those of 2006 and for several weeks were equal to or higher than the upper 95% confidence interval of the predicted seasonal baseline.

Australian Bureau of Statistics death data

The most recent data on causes of death in Australia are for 2006. Influenza and pneumonia (ICD-10 codes J10–J18) were noted as the underlying cause of death for 2,715 persons in 2006 (2.0% of all deaths). More females than males died of influenza or pneumonia (1,495 females compared to 1,220 males); however the standardised death rate for males was higher than for females (14.1 versus 10.2).[4]

Figure 15. Observed and predicted rate of influenza and pneumonia deaths as per New South Wales registered death certificates, January 2003 to September 2007



Source: New South Wales Influenza Surveillance Report, 22 to 28 September 2007.

Australian Institute of Health and Welfare death data

Australian Institute of Health and Welfare (AIHW) data show a mean of 2.6 deaths per year from influenza, in children aged 0 to 4 years, over the 9 years from 1997 to 2005 (Table 3). In 2007, 7 child deaths were reported by jurisdictions as associated with influenza.[5]

Virology

Antigenic characterisation

Of the 10,577 influenza cases notified to NNDSS in 2007, 95% included typing data. Influenza A was the predominant circulating type comprising 86% of isolates typed compared to 9% that were type B and 0.4% mixed type A&B (Figure 16). The finding of 0.4% of notifications having both types of influenza was lower than in previous years but is still higher than would be expected, as documented reports of dual infections are rare.[6] This may warrant further investigation in the future to confirm true dual influenza infections. In 2006, 75% of typed isolates were influenza A and 25% influenza B.

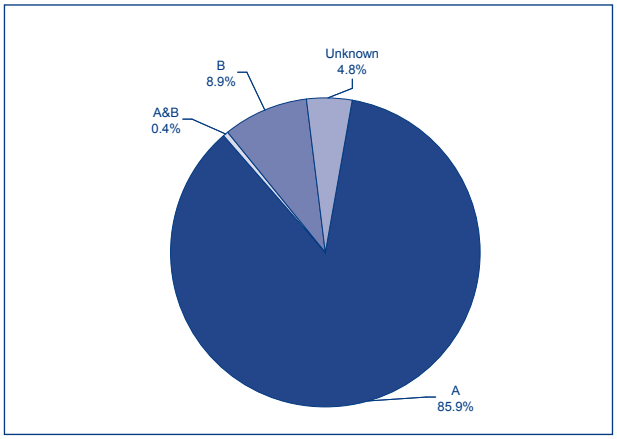
Table 3. Influenza deaths, Australia, 1997–2005, in children aged less than 5 years

	1997	1998	1999	2000	2001	2002	2003	2004	2005
Influenza deaths 0–4 years (J10, J11)	2	6	2	3	2	1	2	3	2

Source: Australian Institute of Health and Welfare 2007.

GRIM (General Record of Incidence of Mortality) Books. Australian Institute of Health and Welfare: Canberra.

Figure 16. Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2007, by type



The WHO Collaborating Centre for Reference and Research on Influenza received 1,406 isolates or clinical specimens from Australian laboratories in 2007 that yielded viable influenza viruses. This was the second highest number of isolates received over the last 10 years (the highest being in 2002). All of the 2007 viruses were analysed antigenically using the haemagglutination inhibition (H1) assay, which identified 826 (58.7%) as A(H3N2) strains, 483 (34.4%) as A(H1N1) strains and 97 (6.9%) as influenza B strains. The 2007 Australian A(H3N2) viruses were antigenically similar to either the 2007 vaccine strain A/Wisconsin/67/2005 or the newly emergent variant A/Brisbane/10/2007, with a few viruses not showing either of these HI patterns (Table 4). Consistent with the antigenic drift seen with a large number of the A(H3N2) isolates when tested with specific ferret antisera, serological studies conducted with pre– and post-vaccination human sera from recipients of 2006 vaccine which contained the A/Wisconsin/67/2005 strain, showed that there was a reduction in antibody titres to many 2007 A(H3N2) isolates. Antigenic analysis of the Australian 2007 A(H1) strains, also showed that there was significant drift away from the 2007 vaccine strain A/New Caledonia/20/99. Few influenza B viruses were isolated in 2007, but from those analysed, only 21% were antigenically related to the 2007 vaccine strain B/Malaysia/2506/2004 (B/Victoria/2/87-lineage viruses) while the remaining 79% were closely related to B/Florida/7/2004-like viruses (B/Yamagata/16/88-lineage viruses) representing the alternative B lineage.

Sequence analysis of the variable (HA1) region of the haemagglutinin (HA) gene was undertaken for 133 Australian 2007 viruses (40 A(H1), 72 A(H3) and 21 B) and for 73 the neuraminidase genes, (23 H1, 41 H3, 9 B). The phylogenetic analysis of the 2007 (H3) virus HA1 sequences showed that most Australian A(H3) viruses were closely related to A/Brisbane/10/2007-like viruses although some also grouped closely with related viruses recently isolated from South East Asia (such as A/Thailand/31/2007 and A/Macau/200/2007). Viruses isolated from fatalities involving 3 young children (<5 years) in Perth in July 2007 and 1 from Victoria also grouped in the A/Brisbane/10/2007 phylogenetic group, as did viruses from non-fatal childhood cases (e.g. A/Perth/26/2007) (Figure 17).

When the HA1 genes from A(H1) viruses isolated in Australia in 2007 were compared phylogenetically, they fell almost exclusively into the group of viruses represented by A/Brisbane/59/2007 (Figure 18). A few viruses fell into in other groups

Table 4. Antigenic comparisons of influenza A(H3) viruses by the haemagglutination-inhibition test

Virus antigen	Ferret antiserum Reciprocal haemagglutination-inhibition titre:	
	A/Wisconsin/67/2005	A/Brisbane/10/2007
A/Wisconsin/67/2005	**640***	1,280
A/Brisbane/10/2007	640	**640***
A/Perth/117/2007	640	1,280
A/Sydney/103/2007	320	320
A/South Australia/61/2007	80	160
A/Victoria/290/2007	80	320
A/Darwin/12/2007	80	320
A/Brisbane/188/2007	80	320
A/South Australia/41/2007	80	40
A/Canberra/1/2007	<40	160

* An A/California/7/2004-like strain (A/New York/55/2004) was the H3 strain used in the 2006 Australian influenza vaccine.

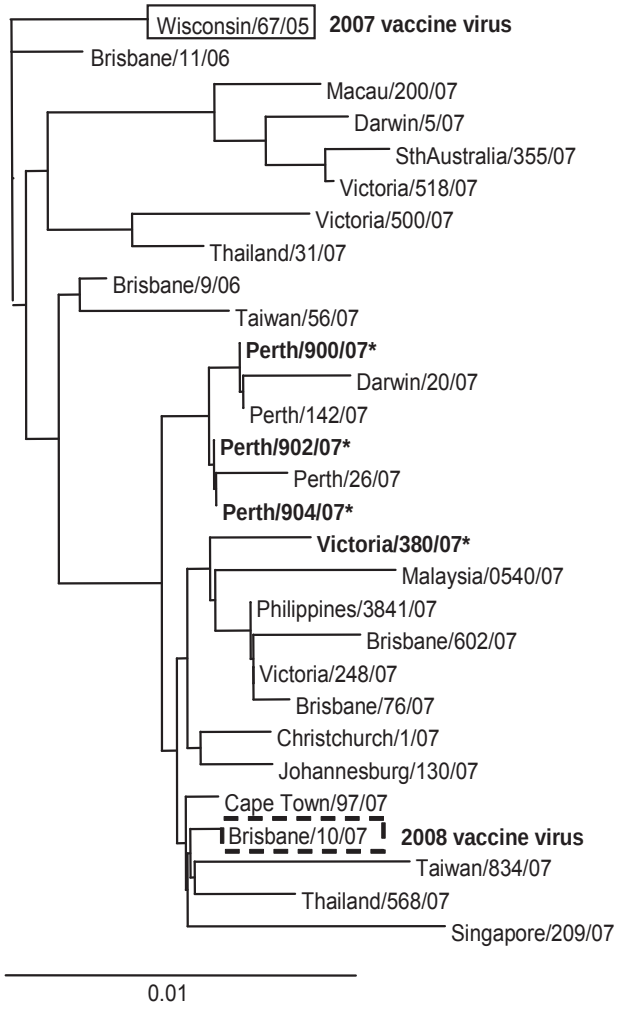
represented by the A/Hong Kong/2652/2006 or A/Brisbane/193/2004. Two A(H1) viruses associated with fatalities in young children also grouped with the A/Brisbane/59/2007-like viruses as did the 3 Tamiflu resistant A(H1) viruses isolated from Australian patients in late 2007 (Figure 18). The Australian 2007 influenza B viruses phylogenetically grouped into their respective lineages, either the B/Victoria or B/Yamagata lineage, with the B/Victoria-lineage viruses showing little change from the reference/vaccine strain B/Malaysia/2506/2004 while the B/Yamagata-like viruses showed only minor changes from the reference virus A/Florida/7/2004 but were somewhat better represented by the A/Brisbane/3/2007 virus (Figure 19).

International trends in influenza

In 2007 global influenza activity was generally moderate when compared with previous years. Influenza was detected in most countries and typically all 3 types/subtypes co-circulated (i.e. A(H1N1), A(H3N2) and influenza B). Levels of influenza A(H1) activity however, did increase in many countries compared with previous years. Outbreaks of A(H1) were reported in several countries, including Australia, Japan, Mexico, New Zealand, South Africa and the United States of Australia. In the Southern Hemisphere, influenza activity began in April and increased until July and then began to decline through August. In South America influenza A(H3N2) and B viruses co-circulated while influenza A(H1N1) and A(H3N2) (44 % and 31% respectively of all influenza viruses New Zealand and A(H1N1) viruses predominated in South Africa.

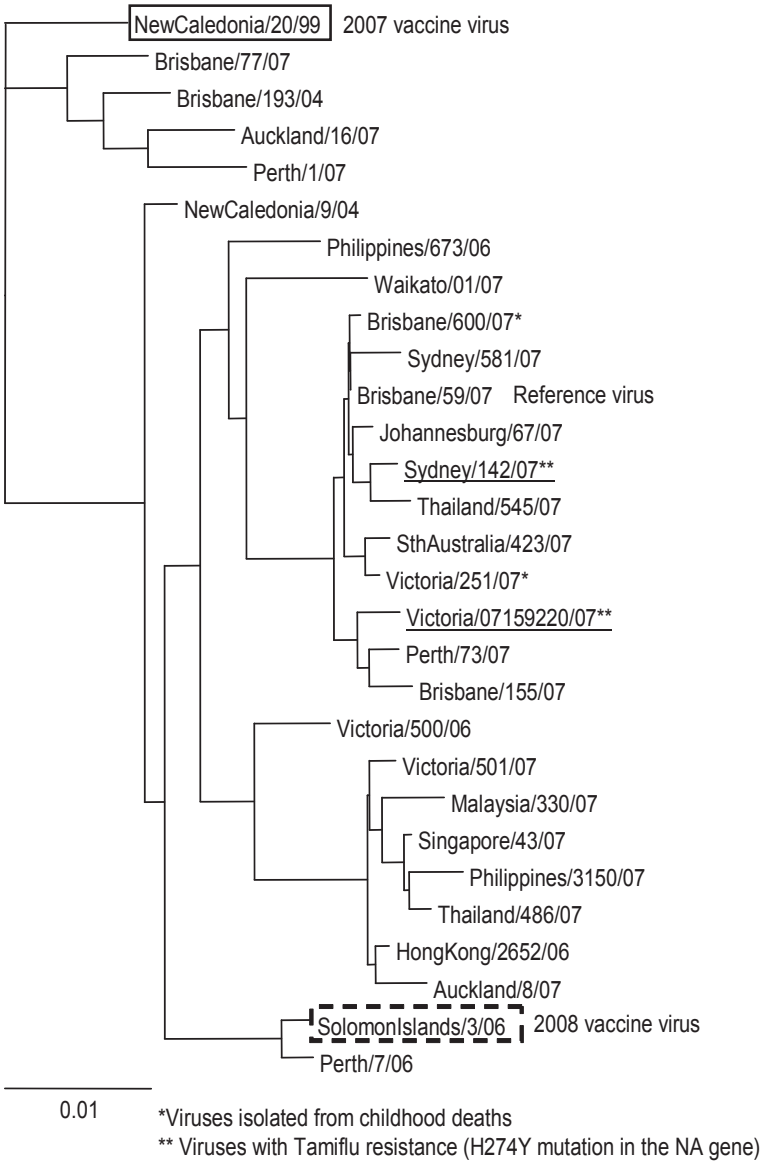
Outbreaks of A(H5N1) highly pathogenic avian influenza (HPAI) in poultry and wild birds still occurred in many parts of the world in 2007, with the continued exceptions of the Americas and Oceania. According to the official WHO figures, 88 H5N1 human infections occurred in 7 countries during 2007 resulting in 59 deaths. This was lower

Figure 17. Evolutionary relationships between influenza A(H3) haemagglutinins (HA1 region)



*Viruses isolated from childhood deaths

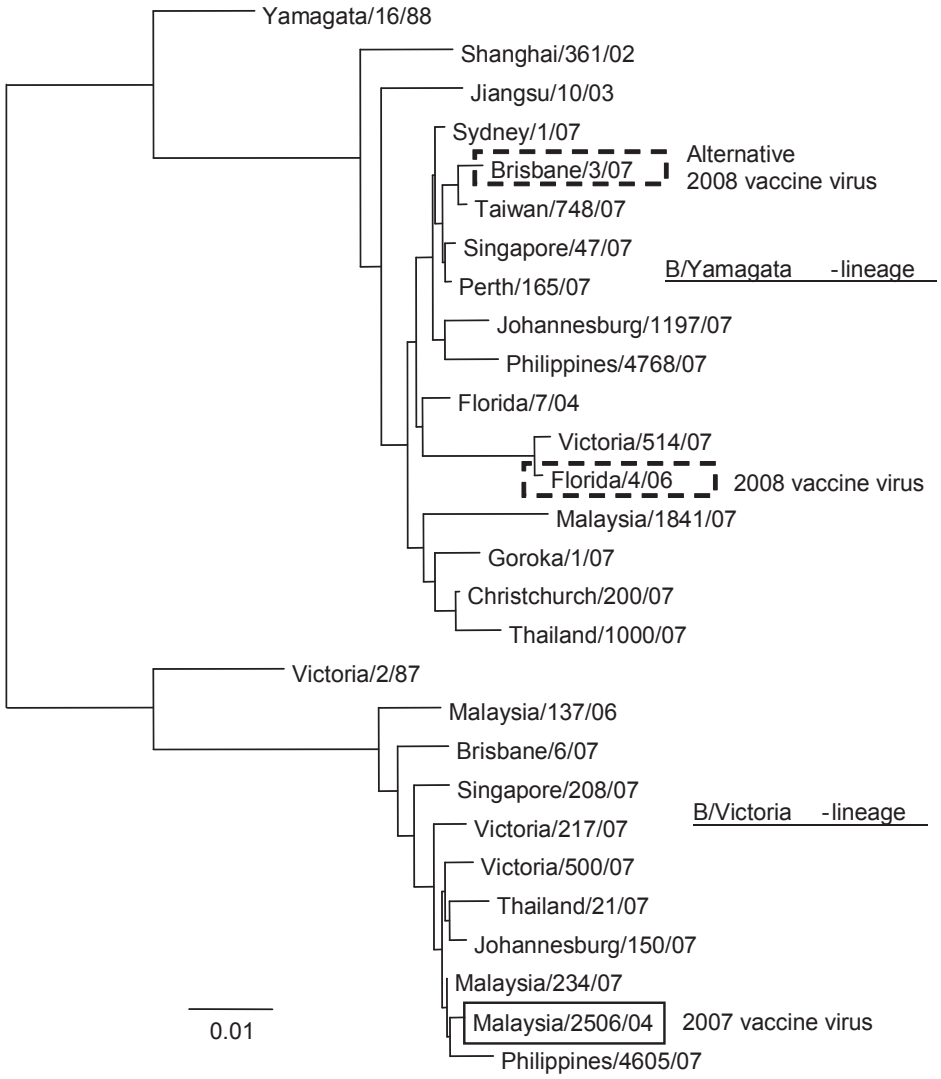
Figure 18. Evolutionary relationships between influenza A(H1) haemagglutinins (HA1 region)



than seen in either 2005 or 2006, (for details see the WHO avian influenza web site http://www.who.int/csr/disease/avian_influenza/en/). No highly pathogenic H5N1 infections were detected in humans or in birds in Australia in 2007.

While the temporal pattern of the annual influenza season in New Zealand is broadly similar to Australia, outbreaks often begin earlier in the year. In 2007 the New Zealand consultation rates for ILI started to increase in late May, with a minor peak at week 27 (first week of July) reaching a major peak at week 30–31 (late July) with levels then declining and returning to baseline during September. A lower level of hospitalisation with influenza was seen in 2007 (347 people) compared with previous years (2005; 390 people, 2006; 464 people). Of the 345 New Zealand isolates typed at the WHOCC, the majority were A(H1) (44.1%) with fewer A(H3) viruses (19.7%) and influenza B viruses (36.2%). This was in contrast to 2006 where the vast majority of New Zealand viruses tested at the Centre were influenza A(H3) viruses (80.4%). Interestingly, the majority of the New Zealand A(H1) viruses were antigenically closely related to the A/New Caledonia/20/99 vaccine strain with only one third typed as A/Solomon Islands/3/2006-like along with a few A/Brisbane/59/2007-like viruses. A(H3) viruses were mainly A/Brisbane/10/2007-like while the influenza B viruses were mainly of the B/Yamagata-lineage (B/Florida/7/2004-like) with very few B/Malaysia/2506/2004-like viruses (B/Victoria-lineage). Overall influenza activity in New Zealand in 2007 was low and below levels seen in 2003–2006. The full Environmental Science and Research report

Figure 19. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



on the 2007 influenza season in New Zealand is available on their web site at: http://www.surv.esr.cri.nz/virology/influenza_annual_report.php

Review of paediatric deaths from influenza, 2007

During the 2007 influenza season, eight children under five years of age died within 24 hours of developing mild and non-specific symptoms of respiratory infection. Although the cause of death has not been reported in many of these cases, these deaths appeared to be associated with influenza A. The apparent virulence of the infection led DoHA to attempt to collate a case-series to identify any common or predictive features of influenza A infection in these cases. Paediatric deaths are not routinely reported as a separate finding in seasonal influenza surveillance.

Surveillance of paediatric deaths was undertaken using several sources of data:

- jurisdictional communicable diseases units were asked to report any influenza associated deaths occurring in children aged 0–4 years, or any instances of children this age being admitted to ICU within 24 hours of the onset of influenza. A case report form was constructed in NetEpi and made available to the jurisdictions although information was accepted by DoHA in any format.
- The Australian Paediatric Surveillance Unit was requested to report any cases of children aged 0–4 years who had presented to ICU within 24 hours of the onset of influenza. A case report form was constructed in consultation with APSU. Reports of paediatric deaths from influenza appearing in the national media were reviewed and significant information noted.

The NetEpi and ASPU forms differed in some details, and the NetEpi form surveyed greater detail about the child's presenting illness and possible contacts. This was not considered problematic because both the jurisdictional public health units

and APSU should have been reporting the same cases, allowing the data received to be pooled into single case reports.

Case data

Details of a single case were received from NSW Health using the NetEpi system. Of the 15 cases aged 0–4 years presenting to ICUs within 24 hours of the onset of influenza, identified by APSU, there were no deaths. Other details were collated from teleconference notifications to the Communicable Diseases Network of Australia or media reports.

Sufficient data were available to include 8 paediatric deaths in the series. Minimal information was available on a further 3 children who were admitted to ICU and survived, and another possible further case of death from influenza B. Influenza was isolated in seven of the children who died, while adenovirus was isolated from the eighth child. Where data were provided on the duration of illness (6 of 7 influenza deaths), death occurred within 48 hours of the onset of symptoms. Influenza A subtype H3N2 was isolated from 3 cases and H1N1 from 4 cases in the 7 deaths where influenza was detected. Insufficient information was provided on medications, previous illnesses or exposure to animals to allow hypothesis generation of the cause of death. It was notable that the first 3 Western Australian deaths had group B streptococcus isolated from blood samples post-mortem but the significance of this is not clear.

All seven of the influenza deaths were boys. It is improbable that all 7 deaths would be male if the probability of a male and a female being in the sample was equal ($p=0.07$). This does not mean that the observed sex-bias was due to differential susceptibility to disease as the case series is not complete and the numbers are small. The finding is, however, interesting, given the higher preponderance of asthma and other possibly significant co-morbidities in boys aged 0–4 years.

Death from influenza is rare in children aged 0–4 years. The AIHW reported between 2 and 6 deaths per year from influenza in this age group between 1997 and 2005, with no data available for the past 2 years. Eleven child deaths in 2007 appears higher than expected, but this cannot be quantified without an estimate of the number of cases of infection among children occurring in different years.

Since enhanced surveillance was unable to identify definitively either the number of deaths in children 0–4 years of age which occurred during the 2007 season or many of the significant clinical details regarding these deaths, no epidemiological or clinical conclusions can be drawn from the data reported. It has not been possible to inform public health interventions based on an analysis of sudden deaths in this age group.

It was considered an important element of influenza surveillance to attempt to examine the number and circumstances of sudden deaths occurring in children. Deaths from influenza in this age group are rare, and therefore a measurable increase could reflect the emergence of a virulent strain of influenza. Acquiring a defined case series was assisted by the low 'background' rate of influenza deaths among children but this also necessitated active surveillance to avoid these deaths being lost in aggregate death data. Children are not usually a target of measures to prevent influenza, such as vaccination, and evidence of an increased level of risk may have helped inform decisions about management in the 2007 season. It was also not immediately apparent that the first 3 reported deaths were due to influenza, this diagnosis being made post-mortem, and accurate case reporting was necessary to exclude a novel emerging pathogen.

A more consistent national system is required for the early identification, collection and provision of unexplained paediatric deaths. Information should be collated nationally to detect any changes in the virulence, host-risk-factors or co-morbidities associated with death from circulating strains of influenza. If all deaths are reported and reported early this will provide better data on the number of deaths which are expected during an influenza season. Accurate baseline data would provide a quantitative measure against which to assess 'bad' influenza seasons, as well as form part of the surveillance for new or potentially pandemic strains of the virus.

Discussion

The impact of an influenza season and its severity is difficult to measure. Ideally a severity index would include the number of cases along with the burden of disease on individuals (such as hospitalisations) and the burden on the health system. At this time only the number of cases in an influenza season are able to indicate how severe the season was in Australia. In 2007, the largest number of notifications was seen since inception of influenza as a notifiable disease in 2001.

The 2007 influenza season was considered a moderate to severe season as measured by the number of cases. While the role of heightened media attention, differing diagnostic tests, and non-representative referral of samples makes an estimate of the true number of cases of influenza impossible, there were more than 3 times the number of laboratory-confirmed notifications compared with the five year mean. Furthermore,

an epidemic case load was recorded through ILI surveillance and absenteeism was higher than previous years.

The majority of the Australian isolates analysed at the WHO Influenza Centre were A(H3N2) viruses however, there was a substantial proportion of A(H1N1) viruses co-circulating along with a few B viruses. This was the highest proportion of A(H1N1) viruses that have circulated in Australia since 2001 when they were the predominant strain. Over the next 5 years from 2002 to 2006, the proportion of A(H1N1) strains seen annually in Australia has been <1%, <1%, 18.8%, and 3.5%, respectively. Accordingly, the extensive co-circulation of 2 influenza A strains may have contributed to the severity of the influenza season seen in Australia in 2007. Both A(H1N1) and A(H3N2) viruses were isolated from fatal cases involving children under 5 years of age as well as in adults, although any contribution that these influenza infections may have had in these deaths remains to be determined.

Antigenic drift away from the 2007 vaccine strains A/New Caledonia/20/99 and A/Wisconsin/67/2005 was seen for both the influenza A(H1N1) and the A(H3N2) viruses respectively. Antigenic and genetic analysis of these viruses showed the majority of A(H1N1) viruses isolated in Australia in 2007 were more closely related to the reference strain A/Brisbane/59/2007 while the A(H3N2) viruses were more closely related to the reference virus A/Brisbane/10/2007. Interestingly, in New Zealand the majority of the A(H1N1) strains were still similar to the vaccine strain A/New Caledonia/20/99. Three A(H1N1) strains with the H274Y mutation in the neuraminidase gene were detected in Australian 2007 isolates. This mutation confers oseltamivir (Tamiflu) resistance and appears to have arisen in Europe during the 2007–08 season[7] and was prevalent in a number of countries (e.g. Norway had 67% resistant A(H1) viruses). Few influenza B viruses were isolated in Australia in 2007 (97) and the majority of these (79%) were B/Florida/7/2004-like (B/Yamagata-lineage) while the remaining viruses (21%) were similar to the 2007 vaccine virus, B/Malaysia/2506/2004 (B/Victoria-lineage). This was in contrast to the 2006 season where B/Malaysia/2506/2004-like viruses predominated. Influenza A(H3N2) and B viruses in New Zealand were similar to those seen in Australia in 2007.

The WHO annual consultation on the composition of influenza vaccines for the Southern Hemisphere, 2008 took place in Geneva from 17–19 September 2007. The recommended composition of influenza virus vaccines for use in the 2008 Southern Hemisphere influenza season was:

— an A/Solomon Islands/3/2006 (H1N1)-like virus;

— an A/Brisbane/10/2007 (H3N2)-like virus;

— a B/Florida/4/2006-like virus.

The recommendation for the 2008 vaccine had changes to all 3 viruses compared to the previous year's Southern Hemisphere vaccine. A further change has been recommended for the 2008–09 Northern Hemisphere influenza vaccine, with the A(H1N1) component being updated to incorporate an A/Brisbane/59/2007-like virus. B/Brisbane/3/2007 is considered an B/Florida/4/2006-like virus and so can be used for the 2008 vaccine instead of B/Florida/4/2006.

Many sources of data were available to characterise the epidemiology of the 2007 influenza season. However, important opportunities to improve the surveillance data were also identified. These included enhanced data collection to maximise completeness; improved data quality; and opportunities to ease the collection of data. Improved and consistent protocols for data collection would also help to improve the representativeness of information across jurisdictions.

Potential sources of additional data and steps required to improve seasonal influenza surveillance data were identified. These should now be developed, in collaboration with key stakeholders, into specific actions plans with assigned responsibilities to ensure an ongoing improvement process for seasonal influenza surveillance.

Ongoing, continuous improvement of surveillance should be the goal each season, in the knowledge that effective seasonal surveillance will also be an essential precursor to the surveillance of pandemic influenza.

Monitoring influenza through the National Incident Room during the 2007 season offered an excellent opportunity to conduct enhanced surveillance under conditions which were real and potentially serious but not an emergency. It enabled the current state of our surveillance systems to be assessed and opportunities for improvement to be identified. Areas for improvement in the collection and presentation of influenza during outbreaks, as well as the ability to investigate emerging changes in the behaviour of infectious disease were recognised.

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Annual report of the Australian Gonococcal Surveillance Programme, 2007

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all states and territories. In 2007 the *in vitro* susceptibility of 3,042 isolates of gonococci from public and private sector sources was determined by standardised methods. The proportion of gonococci resistant to all antibiotics tested nationally was at historically high levels. Different antibiotic susceptibility patterns were again seen in the various jurisdictions and regions. Resistance to the penicillins nationally was at 38% and, with the exception of the Northern Territory, ranged between 24% in Western Australia and 54% in New South Wales. Quinolone resistance in gonococci also continued to increase so that nationally 49% of all isolates were ciprofloxacin-resistant, and most of this resistance was at high MIC levels. Again with the Northern Territory excepted, proportions of quinolone resistant gonococci ranged between 26% in Western Australia and 65% in New South Wales. All isolates remained sensitive to spectinomycin. Approximately 1% of isolates showed some decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more) and azithromycin resistance was present in low numbers of gonococci. A high proportion of gonococci examined in larger urban centres were from male patients and rectal and pharyngeal isolates were common. In other centres and in rural Australia the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2008;32:227–231.

Keywords: antimicrobial resistance, disease surveillance, gonococcal infection, *Neisseria gonorrhoeae*

Introduction

Gonorrhoea differs from the other major sexually transmitted diseases prevalent in Australia that are of bacterial origin in that treatment and disease control is compromised by antimicrobial resistance (AMR). AMR in *Neisseria gonorrhoeae* isolated in large urban centres in Australia is heavily influenced by the continuing introduction of multi-resistant gonococci. Treatment options have been severely limited by the increasing lack of efficacy of several major antibiotic groups.[1] In contrast, in remote Australia, traditional penicillin-based regimens retain their efficacy. Strategies for treating and controlling gonorrhoea are based on use of single dose treatments that cure a minimum of 95% of cases.[2] Formulation of these standard treatment regimens relies on data derived from continuous monitoring of the susceptibility of gonococci to recommended antibiotics.[2,3] The Australian Gonococcal Surveillance Programme (AGSP) has monitored the susceptibility of *N. gonorrhoeae* continuously since 1981.[4] The emergence and spread of penicillin and quinolone resistant gonococci in major cities has been well documented.[1] There are increasing concerns about the presence in Australia of gonococcal isolates showing resistance to multiple antibiotics including to the third generation cephalosporin ceftriaxone, which is used extensively in Australia.[1,5] This analysis of AMR in *N. gonorrhoeae* in Australia was derived from data generated by the AGSP during 2007.

Methods

Ongoing monitoring of AMR in gonococci in Australia is performed by the AGSP through a collaborative program conducted by reference laboratories in each state and territory. The AGSP is a component of the National Neisseria Network of Australia and comprises participating laboratories in each state and territory. This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible and both public and private sector laboratories refer isolates to regional testing centres. The increasing use of non-culture based methods of diagnosis has the potential to reduce the size of the sample of isolates available for testing. Details of the numbers of organisms examined are thus provided in order to indicate the AGSP sample size.

Gonococci, isolated in and referred to the participating laboratories, were examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins and for high-level resistance to the tetracyclines by a standardised methodology.[4,6] The AGSP also conducted a program-specific quality assurance (QA) program.[7] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory, which collated the results and also conducted the QA program. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates were included in analyses.

Results

Number of isolates

There were 3,103 gonococcal isolates referred to or else isolated in AGSP laboratories in 2007, approximately 20% fewer than the 3,937 examined in 2006. The source and site of infection of these isolates are shown in the Table. Nine hundred and seventy-three gonococci (31.4% of the Australian total) were isolated in New South Wales, 625 (20.1%) in Victoria, 455 (14.7%) in Queensland, 404 (13%) in the Northern Territory, 366 (11.8%) in Western Australia, and 240 (7.7%) in South Australia with small numbers in Tasmania (20) and the Australian Capital Territory (20). Three thousand and forty-two isolates remained viable for susceptibility testing.

Source of isolates

There were 2,560 strains from men and 541 from women, with a male to female (M:F) ratio of 4.7:1, slightly higher than the 5.3:1 ratio for 2006. The number of strains from men increased by 27 and there was a corresponding decrease in the number of isolates from women. The M:F ratio was again high in New South Wales (8.7:1) and Victoria (9:1) where strains were more often obtained from urban populations. The lower ratios in Queensland (3.3:1) Western Australia (3.6:1), South Australia (2.8:1) and the Northern Territory (2:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in Victoria (together 34% of isolates from men), and New South Wales (33%). One hundred and one isolates show the site as 'other' or 'not stated'. Twenty-six of these were pharyngeal and 10 were rectal isolates from women. Also included in this total were 23 cases of disseminated gonococcal infection, 13 in men (0.9% of infections) and 10 (1.8%) in women. Although not all infected sites were identified, isolates from urine samples were regarded as genital tract isolates and most of the other unidentified isolates were probably from this source, although they were not so specified. There were 10 isolates from the eyes of both newborn and older infants and also adults.

Antibiotic susceptibility patterns

In 2007 the AGSP reference laboratories examined 3,042 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin, and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

The categorisation of gonococci isolated in Australia in 2007 by penicillin MIC is shown in Figure 1. Infections unlikely to respond to the penicillin group of antibiotics (penicillin, ampicillin, amoxycillin, with or without clavulanic acid) are those caused by gonococci shown as 'penicillinase-producing' *N. gonorrhoeae* (PPNG) and 'RR – relatively resistant'. Resistance in the PPNG group results from the production of beta-lactamase, and in those 'relatively resistant' by the aggregation of chromosomally-controlled resistance mechanisms[1]

Table. Source and number of gonococcal isolates, Australia, by sex, site and region, 2007

Gender	Site	State or territory						
		NSW	NT	Qld	SA	Vic.	WA	Aust.*
Male	Urethra	572	262	294	148	366	259	1,922
	Rectal	178	0	38	13	117	11	364
	Pharynx	106	2	21	12	75	6	226
	Other/NS	17	3	6	4	5	11	48
	Total	873	267	359	177	563	287	2,560
Female	Cervix	82	132	89	50	57	72	488
	Other/NS	18	4	7	12	5	7	53
	Total	100	136	96	62	62	79	541
Unknown	Total	0	1	0	1	0	0	2
Total*		973	404	455	240	625	366	3,103

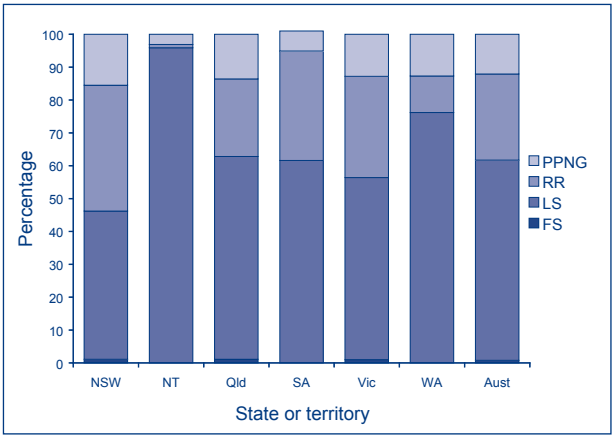
* Includes isolates from Tasmania (20) and the Australian Capital Territory (20).

NS Not stated.

The site of isolation and sex of some infected patients was not known.

(chromosomally mediated resistance to penicillin – CMRP). Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[1,6] (The minimal inhibitory concentration in mg/L (MIC) is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions.) Infections with gonococci classified as fully sensitive (FS, MIC ≤ 0.03 mg/L), less sensitive (LS, MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Figure 1. Penicillin resistance of gonococcal isolates, Australia, 2007, by state or territory



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.*

Nationally, 1,163 (38.2%) gonococci were penicillin resistant by one or more mechanisms in 2007, a further increase in the proportion of isolates resistant to this group of antibiotics recorded in 2006 (1,306 isolates, 34%), 2005 (1,148, 29.5%) and 2004 (770, 21.7%). Of these, 796 (26.2% of all isolates) were CMRP and 369 (12.1%) PPNG. The proportion of penicillin-resistant gonococci of all gonococcal isolates in New South Wales was 53.8% (PPNG 15.5%, CMRP 38.3%), Victoria 43.6% (PPNG 12.8%, CMRP 30.8%), South Australia 39.4% (PPNG 6.1%, CMRP 33.3%), Queensland 37.2% (PPNG 13.6%, CMRP 23.6%) and Western Australia 23.8% (PPNG 12.7%, CMRP11.1%). Four PPNG and 4 CMRP were identified in the Australian Capital Territory and in Tasmania there were 4 PPNG and 9 CMRP. In the Northern Territory, there were 16 penicillin resistant gonococci (13 from Darwin). Of these 12 were PPNG and 4 were CMRNG showing that 4.1% of strains were penicillin resistant (4.6% in 2006). Data on acquisition were available in 96 (23%) infections with PPNG. Half (48) of the infections with PPNG were acquired locally and half by overseas contact. These contacts were principally in Western Pacific or South East Asian countries with contacts reported from Thailand (8), the Philippines (6) and Indonesia (Bali) (5) the most numerous. Additionally, Chile, China, Ireland, Germany, Ghana, Korea, Malaysia, Singapore and Vietnam were named as countries of contact.

Ceftriaxone

From 2001 onwards, low numbers of isolates with slightly raised ceftriaxone MICs have been found in Australia. In 2006 there were 23 (0.6%) and in 2007, 25 (0.8%) gonococci with ceftriaxone MICs in the range 0.06 to 0.25 mg/L. Twelve of these were in New South Wales (1.2% of isolates there), 4 (0.9%) in Queensland, 3 (0.5%) in Victoria, 3 (1.2%) in South Australia, 1 (0.3%) in Western Australia and 2 in the Australian Capital Territory. These isolates were generally also penicillin and quinolone resistant.

Spectinomycin

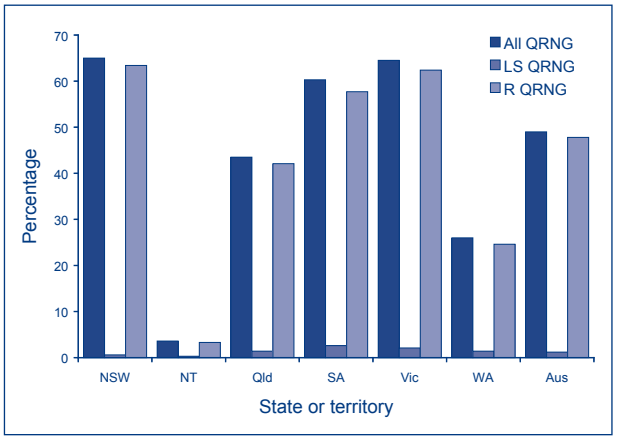
All isolates were again susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by state or territory. Thus far, resistance to the quinolone antibiotics in *N. gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as a MIC of 0.06 mg/L or more.[1,6] Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with a lower level of resistance, viz. 0.06–0.5 mg/L, in about 90% of cases, but lower doses of the antibiotic will result in treatment failure more often. At higher levels of resistance i.e. a MIC of 1 mg/L or more, rates of failed treatment rise rapidly. Currently, gonococci with MICs up to 16 and 32 mg/L are being seen in Australia. At MIC levels of 4 mg/L or more, treatment failure approaches 100%, even with higher ciprofloxacin doses.

Nationally in 2007, 1,493, (49%) of gonococci examined had some level of resistance to quinolones (QRNG), a substantive increase over the 1,455 (37.8%) detected in 2006 and maintaining a continuing and rapid increase in the proportion of QRNG detected. In 2005 there were 1,190 (30.6%) QRNG reported and 825 (23.3%) were found in 2004. Most of the QRNG (1,456 or 98.8%) had resistance at a higher level, i.e. MICs ≥1 mg/L, and many of these had MIC levels of the order of 8–64 mg/L. High proportions of QRNG were seen in New South Wales

Figure 2. Percentage of gonococcal isolates which were less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2007, by state or territory



LS QRNG MIC 0.06–0.5 mg/L.

R QRNG MIC 1 mg/L or more.

where 630 QRNG were 65% of all isolates examined, Victoria 398 QRNG (65.5%) and South Australia 140 QRNG (60.3%). Queensland (192 QRNG, 43.5%) and Western Australia (92 QRNG, 26%) also reported large rises in the number and proportion of QRNG detected. In other jurisdictions the number of QRNG remained low – Northern Territory, 15; Tasmania, 14; Australian Capital Territory, 12: but in the latter 2 jurisdictions these represented a high proportion of all isolates.

Information on acquisition of QRNG was available in 495 of the 1,493 (33%) cases. Four hundred and twenty-two of these (84%) were acquired locally and 73 (16%) were acquired overseas from sources referred to under PPNG acquisition with contacts also reported in Brazil, Greece, Spain, the United Kingdom and the United States of America.

High-level tetracycline resistance

The spread of high-level tetracycline resistance in *N. gonorrhoeae* is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea. There was an upsurge in TRNG isolation in 2002 when 11.4% of strains of this type were detected nationally with little further change in 2003. A further increase in TRNG numbers to 490 in 2004 saw them represent 13.8% of all gonococci. This proportion was unchanged in 2005 when 534 TRNG were detected. In 2006 there were slightly fewer TRNG (12%). In 2007, the highest proportion of TRNG detected in this series was recorded when 505 (16.6%) of gonococci examined were TRNG.

TRNG were present in all jurisdictions except Tasmania, with the highest proportion in Western Australia (110 TRNG, 31.1%). Lower proportions of TRNG were present in New South Wales (181, 18.7%), Queensland (76, 17%), Victoria (100, 16.2%) and South Australia (21, 9%). There were 13 (3.4%) TRNG found in the Northern Territory and 4 in the Australian Capital Territory.

Discussion

Urban Australia has seen continuing upward trends in the proportion of *N. gonorrhoeae* resistant to multiple antibiotics. In 2007 this trend continued with resistance to the penicillins and quinolones approximating 40% and 50% respectively of all isolates examined. There was also a historical high rate of gonococci with high-level tetracycline resistance. The 'rural–urban divide',[1] in gonococcal resistance rates was maintained, (Figures 1 and 2) illustrating the need for disaggregated information rather than pooled national data to define treatment regimens appropriate for the various jurisdictions. Remote areas in some jurisdictions with high disease rates continue to be able to use penicillin-based treatments, but effective use of this cheap and acceptable treatment requires close monitoring of resistance patterns.

Specific comment has been made in recent reports regarding gonococci with decreased susceptibility to ceftriaxone.[5] In 2007, the number of these isolates remained low at about 1% of all isolates tested, but they are almost always also resistant to quinolones and penicillins. Recent regional surveys and local studies have confirmed the wider distribution of these ceftriaxone-less sensitive gonococci[8,9] in countries in close proximity to Australia. The mechanism of resistance to ceftriaxone in these isolates is not fully elucidated, although alterations in the *penA* gene, including the presence of mosaic PBP2, are important.[9,10] The presence of mosaic PBP2 in gonococci can be detected by recently described molecular methods.[11] These changes appear to affect the efficacy of oral third generation agents such as cefixime and ceftibuten, disproportionately, but these antibiotics are not available for use in Australia. AGSP reports have also consistently emphasised that the local recommendation for a minimum dose of 250 mg of ceftriaxone is prudent given the presence of these isolates and the propensity for resistance to develop in *N. gonorrhoeae*.

All gonococci tested in Australia in 2007, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin. A low proportion of gonococci was also found to be resistant to azithromycin in 2007. Resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported with increasing frequency overseas.[12]

These data showing increasing and multiple problems with anti-microbial resistance in *N. gonorrhoeae* indicate a continuing need for surveillance of antimicrobial resistance in this organism. The declining number of gonococcal isolates available for testing in 2007, almost certainly in part as a consequence of the increasing use of non-culture based methods for the diagnosis of gonorrhoea, will be an important issue for surveillance in future years. While the number of gonococcal isolates available for testing in Australia under the AGSP remains satisfactory for surveillance purposes, a continuing commitment to maintenance of culture-based systems will be required while this surveillance is still based on testing of gonococcal isolates.[13]

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Surveillance of Creutzfeldt-Jakob disease in Australia: 2008

Genevieve M Klug, Alison Boyd, Victoria Lewis, Amelia R McGlade, Helene Roberts, Samantha L Douglass, Colin L Masters, Steven J Collins

Abstract

Australia-wide surveillance of all human transmissible spongiform encephalopathies (TSEs) is performed by the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR), since establishment in October 1993. During the surveillance period 1 April 2007 to 31 March 2008, the ANCJDR received 78 new suspect case notifications of TSEs (67 in 2007, 13 in 2008). This level of suspect case notification aligns with the previous 2006/2007 surveillance period, which was elevated in comparison to the previous 5 years. Based on the total number of probable and definite Creutzfeldt-Jakob disease (CJD) cases, encompassing retrospective cases to 1970 and prospectively ascertained cases from 1993 to 31 March 2008, the average age-adjusted mortality rate is 1.16 deaths per million per year. In this short report, we provide updated Australian TSE figures and describe recent changes in surveillance mechanisms and review their impact on case notifications and eventual CJD classification. *Commun Dis Intell* 2008;32:232–236.

Keywords: Australian National Creutzfeldt-Jakob Disease Registry, Creutzfeldt-Jakob disease, disease surveillance, mortality, transmissible spongiform encephalopathies

Introduction

Globally, the incidence of the rare, transmissible group of neurodegenerative disorders, known as transmissible spongiform encephalopathies (TSEs) is reported to be 1 case per million population per year. This group of invariably fatal diseases includes the most common phenotype, Creutzfeldt-Jakob disease (CJD) and the rarer forms of Gerstmann Sträussler-Sheinker syndrome, fatal familial insomnia and variant CJD (vCJD). While the disease is transmissible, the majority of cases occur with no discernible aetiology, arising sporadically. For the remainder of cases, a genetic basis of disease or an iatrogenic association through medical intervention has been determined as an underlying cause. Confirmation of definite cases is based upon neuropathologic assessment of biopsied or post-mortem brain tissue whereas probable cases must fulfil internationally recognised and validated clinical criteria for classification.[1] Possible case classification is also based on defined criteria where there is a suspicion of CJD, however, there is not sufficient clinical evidence to support a probable classification and for this reason these cases are excluded from the following statistical analysis. The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established in 1993 to provide nation-wide surveillance for all human TSE regardless of aetiology and offer specialised diagnostic services, including cerebrospinal fluid 14-3-3 protein analysis.

Australian National Creutzfeldt-Jakob Disease Registry surveillance

National TSE surveillance in Australia by the ANCJDR has involved the evaluation of 1,256 cases of suspect TSE of all aetiologies for the period 1 January 1970 to 31 March 2008. After detailed investigation, 531 cases were deemed non-CJD, 573 cases classified as definite (364) or probable (209) CJD and a further 12 cases fulfilling the possible case definition[1] (Table 1). For the 2007/08 period, 26 cases, consisting of 15 new suspect notifications

Table 1. Classification of cases by the ANCJDR, 1 January 1970 to 31 March 2008

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	323	36	5*	0	0	364
Probable	195	10	4	0	0	209
Possible	11	0	1	0	0	12
Incomplete	0	0	0	0	150†	150
Total	529	46	10	0	150	735

* Includes one definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

† Includes 101 living cases.

and 11 previously notified cases, were confirmed as probable (n=6) and definite (n=20) CJD and 1 further case classified as possible CJD. One hundred and fifty cases are currently under review, with 101 of these cases still living as at 31 March 2008.

The aetiologic proportions of all Australian CJD cases are consistent with previous observations.[2] Cases classified as sporadic CJD comprise 90.6% of all Australian cases, while 8% of cases are genetic and the remaining 1.4% are iatrogenic. No further cases of familial or iatrogenic CJD have been classified during the 2007/08 surveillance period and no cases of vCJD have been identified in Australia as at 31 March 2008.

For sporadic cases, the median age at death is 66 years (males, 65 years; females, 67 years). Females account for 53.5% of sporadic cases and their median duration of disease is 4 months. A slightly shorter disease duration is observed in males (median, 3 months) and the combined median is 4 months. In comparison to sporadic CJD, genetically determined TSEs typically have a younger age at death (median, overall 59 years; males, 51 years; females, 62 years) and longer illness duration (median, overall 6 months; males, 4 months; females, 7.5 months) but equal sex ratio, identical to the sporadic CJD cohort.

Based on all definite and probable CJD cases, the average, age-adjusted mortality rate in Australia for the 1970/08 period is 0.87 deaths per million per year. By restricting the time frame to the more accurate, prospective surveillance period of 1993 to 2008, the mortality rate is 1.16 deaths per million per year. This figure and individual state and territory mortality rates (Table 2) align with reported global figures[3] with the exception of some of the smaller states and territories, where it is likely that there is some case under-ascertainment.

Table 2. Transmissible spongiform encephalopathies deaths and mortality rate, 1993 to 2008,* by state or territory

State or territory	Total TSE deaths	Mean age-adjusted mortality rate (deaths/million/year)
ACT	5	1.06
NSW	127	1.20
NT	3	0.74
Qld	55	0.98
SA	25	1.05
Tas.	6	0.71
Vic.	104	1.35
WA	35	1.21
Aust.	360	1.16

* Includes all deaths occurring between 1 January 1993 and 31 March 2008.

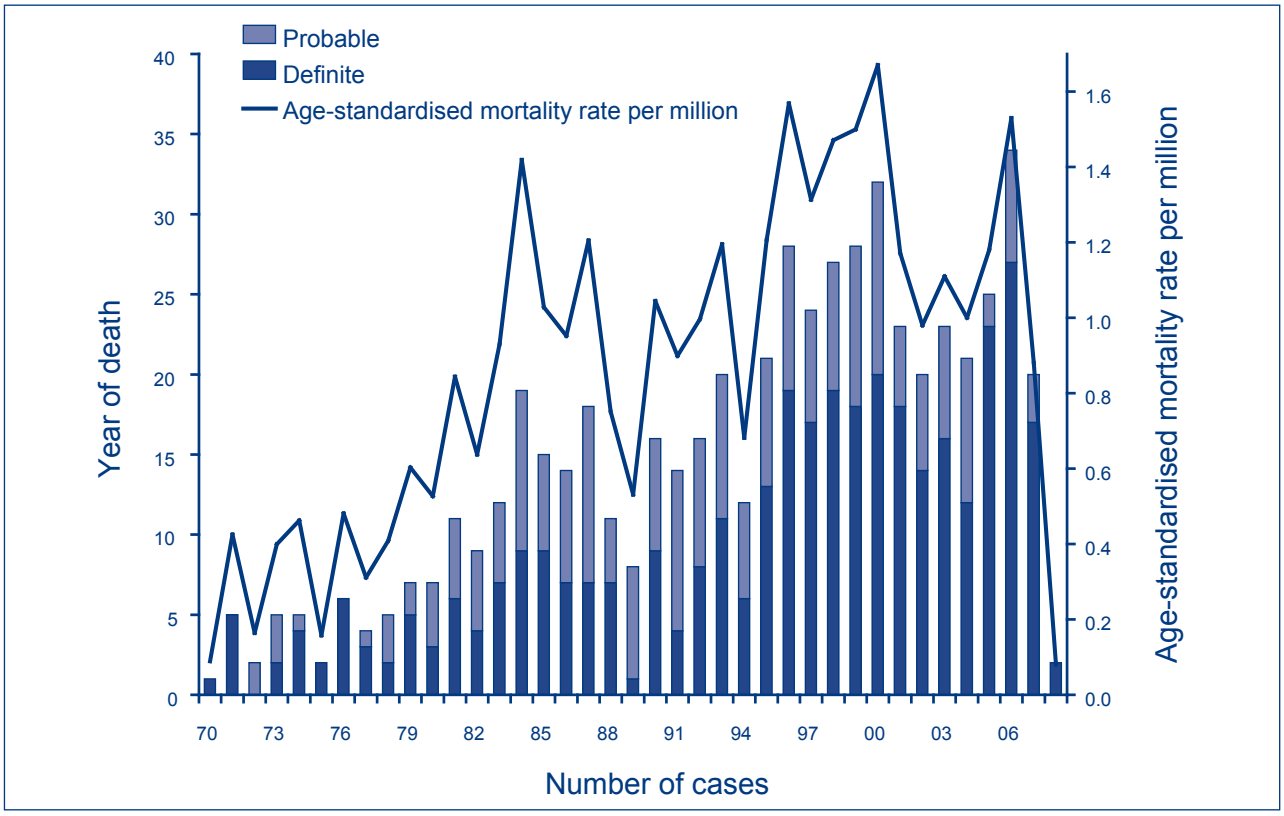
The temporal patterns of the annual mortality rates show distinct peak mortality rates in 2000 and 2006 (Figure 1). As described previously,[2] these peaks correlate with increased suspect case notifications in both 1999/00 and 2006 (Figure 2). In 2006, CJD was identified in every state and territory, which has not occurred in previous surveillance years and there was a marked increase in cases confirmed by neuropathologic examination, particularly in Queensland (Figure 1). A total of 6 cases (5 definite) were identified in Queensland in 2006 compared with 3 or fewer per year in the previous 5 years. This enhanced detection is attributable to the reorganisation and centralisation of Queensland CJD autopsy services in 2006, facilitating the timely neuropathological examination of cases. While temporal changes in annual CJD mortality rates are to be expected, the defined peaks in notifications and mortality need to be considered in relation to recent changes that have affected ANCJDR surveillance mechanisms.

Temporal changes in suspect Creutzfeldt-Jakob disease case notification

Since the ANCJDR began ascertaining both prospective and retrospective TSE cases in 1993, notification rates have fluctuated over time (Figure 2). By grouping all retrospective cases (where death is known to have occurred before 1993) and prospective cases, peaks in notification rates can be observed in 1993, 1995, 1996, and more recently in 1999–2000 and 2006. The earlier peaks largely comprise of retrospective cases identified by Australian Institute Health and Welfare death certificate searches, and national hospital and state morbidity searches. By excluding these cases, a clearer representation of the temporal trends in prospective ascertainment overall and in each of the states and territories can be observed (Figures 2 and 3).

In 1999/00, approximately 4.5 to 5.5 suspect cases per million population were notified per year in Australia. The peak during this period is attributable to 2 causes; firstly an increased awareness of CJD resulting from media coverage of vCJD and secondly, an increasing utilisation of the cerebrospinal fluid (CSF) 14-3-3 protein detection test by clinicians over this period. At that time all CSF referrals were treated as notifications and placed on the Register. As of March 2000, the ANCJDR increased screening of CSF 14-3-3 protein testing for the likelihood of clinical disease. From this time, only cases with positive 14-3-3 results or cases when clinicians had clinical information supporting notification, were entered on the Register for more detailed evalua-

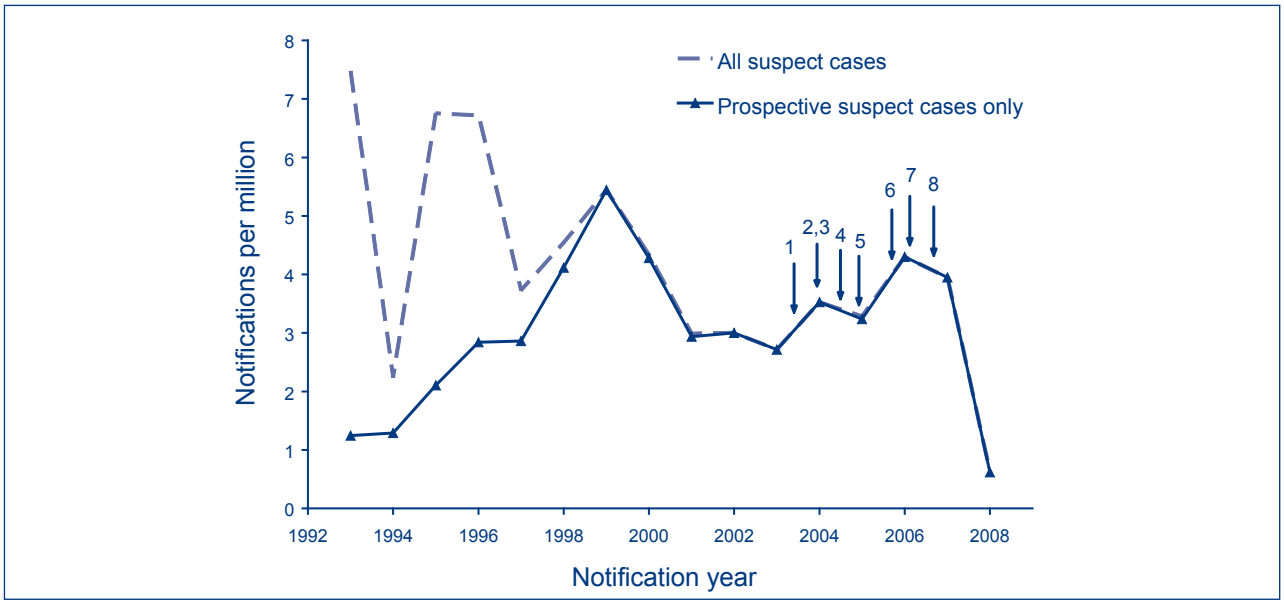
Figure 1. ANCJDR definite and probable cases, 1970 to 2008.* Number and age-standardised mortality rate



* To 31 March 2008.

Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australia.

Figure 2. Creutzfeldt-Jakob disease notification rates, 1993 to 2008,* all suspect cases and prospective cases only



* To 31 March 2008.

Numbers denote the point in time when Creutzfeldt-Jakob disease became notifiable in particular states and territories:
1 – Tasmania, 2 – Victoria, 3 – Western Australia, 4 – New South Wales, 5 – Northern Territory, 6 – Australian Capital Territory, 7 – Queensland, 8 – South Australia.

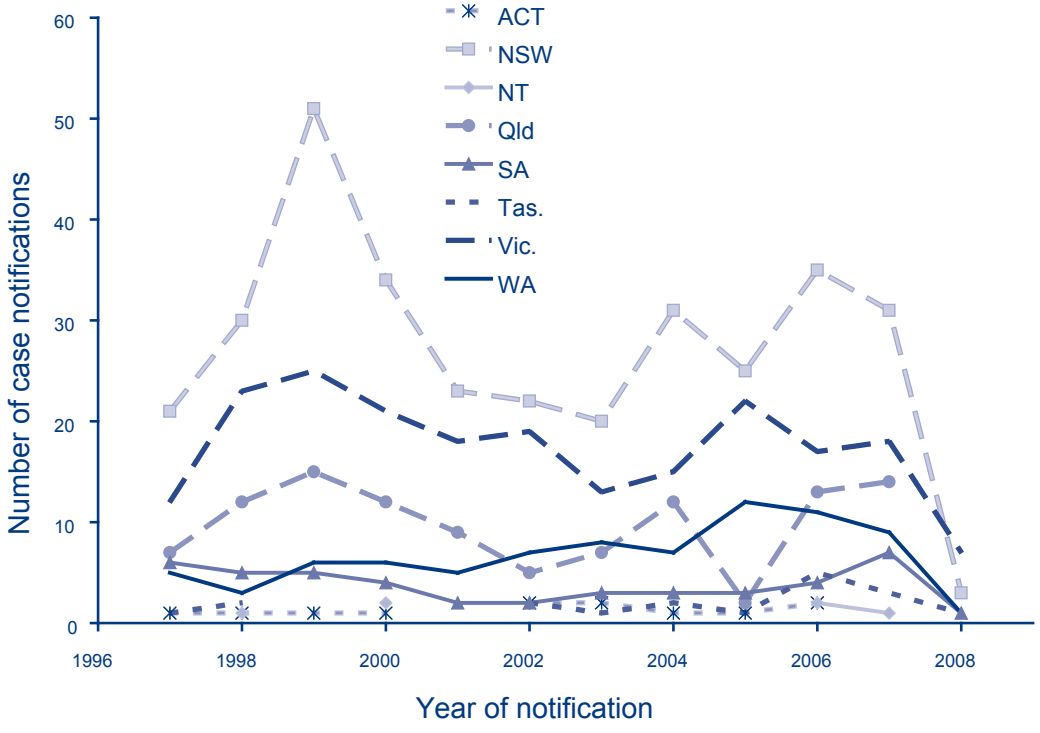
tion. All other referrals that were not entered on the Register but were found to have a negative, atypical or unsuitable result were followed up after 9 months and if found to be deceased, further evaluation was instigated. Consequently, formal suspect case notifications registered after this time declined in all states and territories (Figure 3) and we speculate that lower case ascertainment may have resulted from this new approach from 2000. Previous assessments have demonstrated that increased suspect CJD notifications to the ANCJDR appear to correlate with increased CJD case number confirmations.[2]

After the period of lower formal notifications registered during 2001 to 2003, the notification of suspect cases to the ANCJDR steadily increased (with the exception of Queensland in 2005), correlating with increased total CSF 14-3-3 protein testing referrals. By 2006 and 2007, the rate of suspect case notifications reached around 4 notifications per million per year (Figure 2). State notification rates generally reflect population distributions, particularly in 2006/07 (Figure 3). The increased notification rate of suspect cases for 2006/07 has occurred despite a change implemented by the ANCJDR from 1 January 2007. After careful consideration, the ANCJDR began charging a partial cost-recovery service fee of $75 for 14-3-3 protein CSF diagnostic testing.

There were concerns that charging may have negatively impacted on the single most important ascertainment method, the 14-3-3 protein CSF test. Referrals for this test is the source of the largest proportion of the initial suspect case notifications to the ANCJDR (42% of all notifications overall, 64% of all notifications since test was introduced in 1997). In contrast to expectation, total CSF referrals increased by 34% during 2007, suggesting that a fee for service had not deterred 14-3-3 test utilisation. It remains to be determined if there will be a negative impact over the longer term. There is no explanation at this time for the unexpected increase in CSF samples referred during 2007; however, it will be of interest to assess the 2008 CSF test referral rates in 2009.

An explanation for the increased suspect CJD notifications to the ANCJDR in 2006/07 may in part relate to the establishment of CJD as a notifiable disease in all states and territories of Australia. State by state scheduling of CJD as a notifiable disease occurred at different time points (Figure 2), but by June 2006, CJD was notifiable in all states and territories. The effect of this new notifiability status may have promoted disease awareness and facilitated increased notifications of suspect cases to the ANCJDR and respective health departments. In Victoria, the scheduling of CJD as a notifiable disease in 2004 was accompanied by the formalisation of a contrac-

Figure 3. Prospective, suspect Creutzfeldt-Jakob disease case notifications to the ANCJDR, 1997–2008,* by state or territory



* To 31 March 2008.

tual agreement between the Victorian Department of Human Services and the ANCJDR. While this agreement has not altered the number of suspect case notifications to the ANCJDR directly, it has improved communication between the ANCJDR and the DHS and led to the facilitation of a higher level of autopsy service in the State, resulting in an elevated autopsy rate and subsequently more timely classifications of Victorian suspect cases in recent years. Communication with health departments and organised autopsy services appear to be a key feature in effective CJD surveillance.

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Articles

Epidemiology of laboratory confirmed tuberculosis in Victoria, 1990 to 2004

Michelle E McPherson, David Leslie, Aina Sievers, Mahomed Patel, Heath Kelly

Abstract

In Australia, most cases of tuberculosis (TB) occur in migrants. To inform control strategies for this group, we investigated all laboratory confirmed tuberculosis cases diagnosed by the State TB reference laboratory in Victoria between 1990 and 2004. The laboratory data were matched to notification data to determine country of birth and a multivariate model was constructed to compare Australian and non-Australian-born patients. The proportion of non-Australian-born cases increased over the period of the study and a shift in cases from South East Asia to African countries was observed. Non-Australian-born cases were more likely to be young, female, have extrapulmonary disease and show first line TB drug resistance. The shift in country of birth of TB cases in Victoria reflects migration patterns and the corresponding epidemiology of TB in the country of origin of these migrants. Ongoing migration from countries with high TB incidence raises the question whether it is possible to eliminate TB from Australia and new control strategies should be considered. *Commun Dis Intell* 2008;32:237–241.

Keywords: tuberculosis, migrants, country of birth, incidence

Introduction

A third of the world's population is currently infected with tuberculosis (TB). An estimated 8 million new cases of TB disease and 2 million TB deaths occur every year.[1] Similar to other developed countries,[1] Australia has a low annual estimated incidence of TB, at 5–6 cases per 100,000 population.[2] Most TB cases in Australia now occur in people born in other countries, with the rate for non-Australian-born cases in 2005 being more than twenty times that of the non-Indigenous Australian-born national rate (20.6 cases per 100,000 population compared with 0.8 cases per 100,000 population respectively).[2] Control of TB in Australia is facilitated through entry screening of migrants using a chest x-ray and contact tracing people with active TB.[3]

The aim of this study was to investigate the population-based rate of laboratory confirmed TB in Victoria for the period 1990 to 2004. The characteristics of the non-Australian-born cases were also investigated to inform control strategies for this group.

Methods

Any person residing in Victoria between 1990 and 2004 with a laboratory confirmed diagnosis of TB was eligible for this study. Data were obtained from the Victorian Infectious Diseases Reference Laboratory (VIDRL), the state TB reference laboratory. Cases were matched against notification data of the Department of Human Services (DHS) to obtain further demographic details, including country of birth, using a purpose built Access database. Matches were made using combinations of name, date of birth, postcode and onset date fields. Of the 2,608 laboratory records included in the match (583 had this data already and were not included), 2,351 (90.1%) were matched to DHS notifications. The total number of TB notifications for the years 1990 to 2004 was also provided by DHS. The time between arrival in Australia and diagnosis of TB has been addressed in a separate study[4] and HIV status was unable to be analysed as it is not routinely collected by DHS. Given that TB is a notifiable disease in Victoria and analysing surveillance data is a core function of public health, ethics approval was not required.

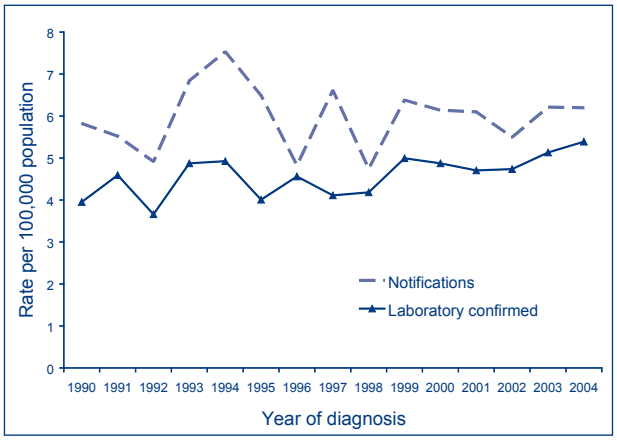
Region of birth categories, the Victorian population and population by country of birth were obtained from the Australian Bureau of Statistics.[5] As population by country of birth was available for census years only (1991, 1996 and 2001), data were analysed in three 4 year groups: 1990–1993, 1994–1998 and 1999–2003. Tests for trend were conducted using Poisson regression for notification and laboratory confirmed rates and linear regression for proportions. A categorical logistic regression model was constructed to compare Australian and non-Australian-born cases. Factors that were associated at the univariate level ($p<0.10$) were included in the model. Variables examined in the model were year of diagnosis, age-group, gender, clinical site (pulmonary or extra-pulmonary), drug resistance and country of birth. Country of birth information was incomplete in the DHS database for cases from 1996 and 1997 (43% unknown, compared to 9% for all other years, $p<0.001$). We therefore chose to

exclude these 2 years from the analysis; although the trend of the proportion of overseas-born cases was the same as we report in the results (p=0.001) when these 2 years were included. The Victorian totals include Indigenous residents as indigenous status has only been collected accurately since 2001 with only 4 Indigenous cases notified since then. Data were analysed using Stata Version 8.0.[6]

Results

There were 3,191 laboratory confirmed cases of tuberculosis between 1990 and 2004 in Victoria, representing 77% of notifications during this period. The rate of laboratory confirmed TB significantly increased over the study period from a low of 3.7 cases per 100,000 population in 1992 to a high of 5.4 cases per 100,000 population in 2004 (p<0.001). In comparison, notification rates, which included cases that were not laboratory confirmed, had a median annual rate of 6.2 per 100,000 and were relatively stable over the period (p=0.72) (Figure 1).

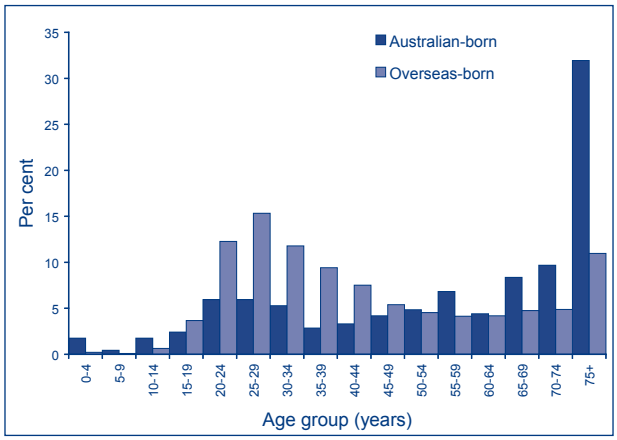
Figure 1. Annual notification rates and incidence of laboratory-confirmed tuberculosis, Victoria, 1990 to 2004



Half (51.9%) of the cases were male and the age specific rate of laboratory confirmed TB showed a bimodal distribution, with peaks at ages 20 to 34 and again at 55 years. There were 70 cases aged less than 15 years and the incidence in this group increased from a low of 0.1 case per 100,000 population in 1990 to a high of 1.1 case per 100,000 population in 1999 (p<0.001), corresponding to between 1 and 10 cases per year.

Of the 2,769 (87%) cases where country of birth information was available 2,315 (84%) were non-Australian-born. In the non-Australian-born group, the proportion of males and females was approximately equal, whereas males comprised 63% of the Australian-born group (p<0.001). For Australian-born patients the proportion of cases increased with increasing age, with the 75 years or over age group comprising 32% of all new cases. The highest proportion of non-Australian-born cases was in the 20–39 years age group (Figure 2).

Figure 2. Percentage of laboratory confirmed tuberculosis, Victoria, 1990 to 2004 by age group and country of birth



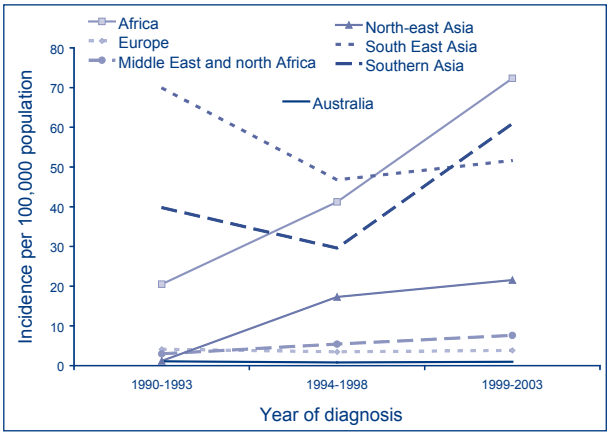
Laboratory confirmed tuberculosis rates by country of birth

The proportion of non-Australian-born cases increased over the study period from a low of 66% in 1994 to a high of 82% in 2004 (p=0.002). Of those with country of birth information 34% were born in South East Asia, 15% in Australia, 14% in Southern Asia, 11% in Europe and 8% in Africa.

Although the highest proportion of non-Australian-born cases was born in South East Asia, their laboratory confirmed TB rate declined between 1990–1993 and 1994–1998, after which it remained relatively constant (Figure 3). Laboratory confirmed rates for cases from all other regions increased, the most marked being in African migrants. Australian and European-born cases had low and stable rates over the 3 periods (1.0 case per 100,000 population and 4.0 cases per 100,000 population, respectively).

Laboratory confirmed TB rates in Victoria for migrants from the most high-risk countries were consistently over 30 per 100,000 (Table). In 1999–2003, migrants from Somalia, Ethiopia, India and Indonesia had the highest average annual rates, whereas in 1990–1993 the highest rates were observed for migrants from Vietnam, India, Cambodia and the Philippines. Combined with data illustrated in Figure 3, these data suggest there has been a shift in Victoria in recent years of cases of TB from South East Asia to African countries. An increase was also observed for cases from Southern Asia, especially from India.

Figure 3. Incidence of laboratory confirmed tuberculosis, Victoria, 1990–1993, 1994–1998 and 1999–2003, by region of birth



Resistance to tuberculosis drugs

Isolates from 331 (11%) laboratory confirmed cases were resistant to at least one of the first line TB drugs (isoniazid, rifampin, ethambutol and pyrazinimide), and 69% of these were from non-Australian-born cases. Although the trend over the entire study period was not significant (p=0.55), there was a significant increase between 2000 and 2004 from 3.4 to 5.8 per million (p=0.04) corresponding to 16 and 29 cases respectively. Of the 331 cases with drug resistance, 202 (61%) were resistant to isoniazid only, 62 (19%) were resistant to pyrazinimide only, 17 (5%) were resistant to isoniazid and pyrazinimide, 5 to rifampin only, 5 to isoniazid and ethambutol and 1 to ethambutol only.

Multi-drug resistance (MDR), defined as resistance to at least isoniazid and rifampicin, was uncommon, occurring in 39 cases (1.2%). All MDR cases with country of birth specified (73%) were non-Australian-born – 16 (41%) from South East Asia, 4 (10%) from both North-east and Southern Asia and a further 3 and 5 cases from Africa and Europe respectively. Again there was an increase in the rate of multi-drug resistance between 2000 and 2004 (p=0.005); corresponding to an increase from 1 to 7 cases.

Table. Number and rate of laboratory-confirmed tuberculosis, Victoria, 1990–1993, 1994–1998 and 1999–2003,* by country of birth and comparison to home countries incidence

Country	1990–1993		1994–1998		1999–2003		Incidence in home country‡
	Number per year	Rate†	Number per year	Rate†	Number per year	Rate†	
Australian-born§	35	1 (0.7–1.4)	26	1 (0.5–1.1)	32	1 (0.6–1.3)	6
Non-Australian-born	133	13 (11–15)	127	15 (13–18)	186	22 (19–24)	6
Africa							
Somalia	–	–	8	544 (157–931)	12	519 (226–813)	412
Ethiopia	–	–	2	163 (0–379)	7	335 (79–591)	356
Southern Asia							
India	11	56 (23–90)	10	39(15–65)	30	98 (63–133)	168
South East Asia							
Indonesia	3	38 (0–82)	6	48 (9–87)	9	80 (27–133)	285
Cambodia	4	52 (0–106)	3	41 (0–85)	6	62 (11–114)	508
Vietnam	40	89 (62–117)	36	65 (44–87)	34	60 (40–80)	178
Philippines	8	48 (15–82)	10	51 (20–82)	13	56 (25–87)	296
Northern Asia							
Hong Kong	1	4 (0–14)	3	19 (0–41)	5	34 (5–62)	92
China	10	47 (17–78)	9	31 (11–52)	12	32 (14–50)	102

* Countries were included in this table if they had more than 5 cases per year in the latest period (1999–2003) and rates were presented where the population denominator was higher than 1,000.

† Average annual rate per 100,000 population for the period. Population data sourced from the Australian Bureau of Statistics 2004.[5]

‡ Incidence for 2003 per 100,000 population. Sourced from the World Health Organization Global TB database[7] and Hong Kong Department of Health web site8 and based on notification data.

§ Includes Indigenous Australians.

Clinical site of tuberculosis

Over the 15 years reviewed, just over half of the diagnoses (55%) were pulmonary disease. Non-Australian-born cases had a significantly lower proportion of pulmonary disease than did the Australian-born for each year of this study (p=0.003) and for the whole period (52% compared with 76%). Over a quarter (27%) of non-Australian-born cases were diagnosed with TB in the lymphatic system, compared with 5% of Australian-born cases.

Comparison of non-Australian-born with Australian-born cases

In multivariate analysis non-Australian-born cases were more likely to be diagnosed in the later 2 diagnosis periods (OR=1.5, 95% CI 1.1–1.9 for 1995–1999 and OR=1.6, 95% CI 1.2–2.0 for 2000–2004). They were also more likely to be female (OR=1.4, 95% CI 1.1–1.8), aged 20–34 years (OR=3.1, 95% CI 1.9–5.1) or 35–49 years (OR=2.8, 95% CI 1.8–3.0), diagnosed with extrapulmonary disease (OR=2.3, 95% CI 1.1–1.9) and resistant to at least 1 of the 4 first line drugs (OR=3.1, 95% CI 1.7–5.7).

Discussion

TB in Victoria is a disease predominantly affecting migrants with a bimodal age distribution. Non-Australian-born females were likely to be diagnosed at a younger age, while Australian-born men were most likely to be diagnosed after the age of 70 years. Over time, the proportion of pulmonary disease has decreased, reflecting the increased prevalence of non-pulmonary disease in people born overseas. Drug resistance, including multi-drug resistance, was not common, and was mainly observed for non-Australian-born cases with all multi-drug resistance cases being non-Australian-born. The characteristics of non-Australian-born cases were similar to those from previous studies in Victoria[9] and New South Wales[10] and the notification rate by country of birth was similar to that reported nationally.[2,11] Other low incidence countries, such as the United Kingdom,[12] the United States of America,[13] and countries from Western Europe[14] also have TB incidence that reflects their migration patterns.

Laboratory confirmed TB by both region and country of birth changed during the period of this study with increasing rates from African countries and Southern Asia. This reflects Australia's immigration patterns as there have been increases in the number of arrivals from Africa, and to a lesser extent Southern Asia, in the latter part of the 20th Century,[15] and a decrease overall in arrivals from Asia.[16] The increase in rates for immigrants from African countries reflects the global picture of TB, as Africa is the only continent where TB rates are increasing.[1] It has been shown in Australia that the incidence of TB in the country of birth is the single most important group-level predictor of the rate of TB among migrants in Australia.[17] However, despite these changes in rates, South East Asia continued to contribute the highest number of cases for each period.

The rate of drug resistant isolates in this study was low, but the increase observed in the latter part of the study period may be cause for concern. Most cases with drug resistant isolates were from countries that have a high incidence of resistant strains of TB and also have a poor history of TB control.[18] All multi-drug resistance cases with country of birth information available were non-Australian-born. Improving TB control in high incidence countries may help to reduce the threat of drug resistance in Australia.

That the incidence of laboratory-confirmed TB increased over the study period while the notification rates were relatively stable reflects an improvement in specimen referral to the reference laboratory, and less reliance on diagnoses made on clinical and radiological findings only. Data on HIV status and treatment were not routinely available from clinical notes included with specimens received in the laboratory and could not be included in this review.

The results of this review raise the question of whether it is possible to eliminate TB from a low incidence country with ongoing immigration from high prevalence countries. The current TB control strategy in Australia of entry screening before arrival may not be adequate. Using chest x-ray as a screening tool will not help detect extra-pulmonary disease, which was more common in non-Australian-born cases in Victoria, and will miss many migrants who arrive with latent pulmonary infection. Other strategies, such as testing for latent TB on arrival or extending follow-up for a longer period post arrival, may be more effective in reducing the number of cases of TB in the non-Australian-born population, but will be more costly. Another control strategy, as recommended by the European framework for TB control, is to improve TB control in high incidence countries,[14] which could also be adopted by Australia to complement our own efforts.

Acknowledgments

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Prevalence of antimicrobial resistances in *Streptococcus pneumoniae* in Australia, 2005

Report from the Australian Group on Antimicrobial Resistance

Thomas Gottlieb, Peter J Collignon, Jennifer M Robson, Julie C Pearson, Jan M Bell and the Australian Group on Antimicrobial Resistance

Abstract

In 2005 the Australian Group for Antimicrobial Resistance (AGAR) conducted a survey of the prevalence of antimicrobial resistance in unique clinical isolates of *Streptococcus pneumoniae*. Twenty laboratories from the 5 mainland states and the Australian Capital Territory collected 1,776 isolates prospectively and tested them by disk diffusion, Etest® and/or agar dilution against a range of antimicrobials. Data from this survey were compared with AGAR surveys conducted in 1989, 1994, 1999 and 2002. Non-susceptibility to penicillin was detected in 28.0% of isolates, 22.7% were erythromycin resistant, 15.6% clindamycin resistant, 18.4% tetracycline resistant and 31.0% trimethoprim-sulphamethoxazole resistant. Levofloxacin resistance was detected in only 4 of 1,775 (0.2%) isolates tested. Intermediate resistance to levofloxacin was detected in another 4 isolates. Moxifloxacin resistance was present in 2 isolates with minimum inhibitory concentrations of 3 mg/L and 4 mg/L. Seventeen point three per cent of isolates were multi-resistant (acquired resistance to more than 2 drug classes). Trend data show an increase in penicillin non-susceptible strains in each survey from 1989 to 2005. Between 1999 and 2005 the proportion of invasive strains with high-level resistance increased from 2.6% to 5.4%. After a rapid emergence and rise in resistance between 1989 and 1999, recent studies have documented a continuing rise in resistance to all non-ß-lactams except trimethoprim-sulphamethoxazole. *Commun Dis Intell* 2008;32:242–249.

Keywords: antibiotic resistance, epidemiology, *Streptococcus pneumoniae*

Introduction

Worldwide *Streptococcus pneumoniae* is the most common bacterium causing pneumonia and meningitis. Invasive pneumococcal infection, primarily bacteraemia and meningitis, occurs most commonly in young children aged less than 5 years and older adults aged over 65 years. Bronchitis and sinusitis in adults and otitis media in children are frequently caused by *S. pneumoniae*. These conditions are responsible for a significant proportion of the antibiotic prescriptions in the community. Otitis media is a particular problem as few antibiotics reach therapeutic levels in the middle ear, particularly when *S. pneumoniae* have raised minimum inhibitory concentrations (MICs). Community antibiotic use in non-invasive infections is seen as an important driver for selection pressure of emerging resistance.[1] Emergence of multi-drug resistance varies among pneumococcal serotypes and correlates with clonal spread. Subsequent to completion of this survey the 7-valent pneumococcal conjugate vaccine (7vPCV), first introduced in 2001 for children at high risk of invasive pneumococcal disease, was funded for all children as part of the Australian Vaccination Schedule commencing 2005. The Australian Group on Antimicrobial Resistance (AGAR) is a group of laboratories that conducts regular antimicrobial susceptibility studies with funding from the Australian Government Department of Health and Ageing. We report the results of the 2005 *S. pneumoniae* survey and compare the changes in susceptibility with those of previous studies. Ongoing surveys, along with serotype studies, will serve as important baselines to monitor changes that may occur in response to the introduction of this vaccine.

Methods

Twenty institutions from the 5 mainland states and the Australian Capital Territory participated in the *S. pneumoniae* AGAR survey. Starting from 1 January 2005, each laboratory collected up to 100 consecutive significant clinical isolates. Only 1 isolate per patient was tested. If *S. pneumoniae* was isolated from more than 1 site, then the isolate from the most significant clinical site was tested.

Species identification

Alpha-haemolytic, optochin sensitive, and/or bile-soluble, Gram-positive cocci were identified as *S. pneumoniae*. Any strain with an optochin zone of inhibition of between 6 mm and 14 mm in CO_2 was tested for bile solubility.

Susceptibility testing methodology

Participating laboratories performed antimicrobial susceptibility tests according to each laboratory's routine standardised methodology[2–6] (CDS, CLSI or BSAC disc diffusion, Vitek2®, agar dilution or MIC testing). Clindamycin and erythromycin discs were placed side by side to look for clindamycin inducibility. Penicillin and moxifloxacin MICs were determined for all isolates using Etest® strips. Four hundred and seventy-one (95%) of the 497 isolates that were penicillin intermediate or resistant (MIC >0.064 mg/L) were also tested with either a ceftriaxone or cefotaxime Etest® strip.

Statistical analysis

P values were calculated using Fischer's Exact test (GraphPad® Prism Software).

Results

Source of isolates

The majority of isolates (54.9%) were from the respiratory tract. Invasive isolates (19.9%) include 341 isolates (19.2%) from blood cultures. Other common sites of isolation were ear and eye specimens (11.2% and 9.5% respectively). The ages of patients reflect the incidence of *S. pneumoniae* infection, with 25.1% of patients below the age of 5 years, and 27.4% in the elderly age group of ≥65 years.

Susceptibility testing results

Penicillin

In this report, the combined penicillin intermediate and resistant categories are referred to as non-susceptible (NS). For the purposes of determining antibiograms and multi-resistance, NS has been treated as resistant. Overall 28% of isolates were non-susceptible to penicillin (Table 1). Higher rates were detected in Queensland (33.8%) and New South Wales/Australian Capital Territory (31.7%) than in the other states combined (21.4%).

Overall, there was a significantly higher rate of non-susceptibility detected in non-invasive isolates compared with invasive isolates ($p<0.001$). Queensland and New South Wales/Australian Capital Territory also had the highest rates of non-susceptibility demonstrated in non-invasive strains (>36%). Although Victoria had the lowest rates of non-susceptibility in non-invasive strains, it had the highest rate in invasive strains (21.9% and 20.8% respectively). Nationally, there were trends to higher rates of resistance among the young (<5 years), and the elderly (>65 years) (data not shown).

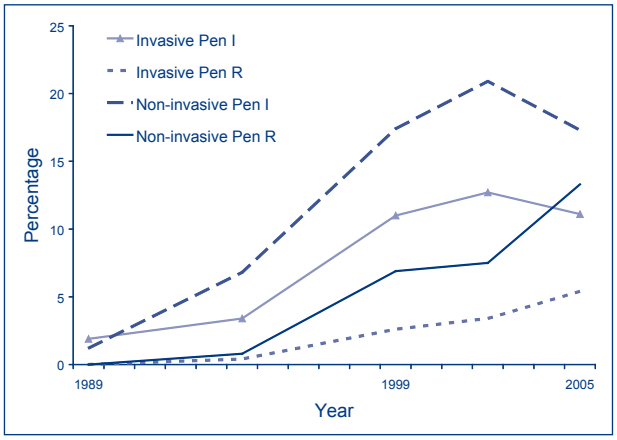
Penicillin 'high-level' resistance (MIC ≥2 mg/L) was present in 11.7% of isolates, with higher rates demonstrated in Queensland and New South Wales/Australian Capital Territory (range: 14.2%–17.3%) than in the other states (range: 7.1%–9.7%). Nationally, 29 isolates (1.6%) had a penicillin MIC ≥4 mg/L, a group that may better define the numbers that may fail penicillin treatment for non-meningeal infection. Overall, the most substantial rise in penicillin non-susceptibility was observed between 1994 and 1999, with a rise from 6.7% to 21.7%. Since 1999, this trend has continued, but less abruptly, with 28% non-susceptibility detected in the current study. Between the last 2 surveys, the rise seen has been predominantly in the proportion of strains demonstrating resistance (MIC ≥2 mg/L), rising from 6.8% to 11.7%, with a similar trend for both invasive (3.4% to 5.4%) and non-invasive (7.5% to 13.3%) isolates (Figure 1).

Because of changes in participating laboratories, these studies may not be fully comparable and hence p values have not been reported. However, an analysis of results for 8 core laboratories that have participated in all AGAR *S. pneumoniae* studies since 1994, have confirmed a similar trend to that described above. In the studies since 1994 a common methodology (Etest®) was used to measure penicillin MICs in all laboratories.

Table 1. Number and proportion of isolates non-susceptible to penicillin

Region	All Isolates		Invasive		Non-invasive	
	n	%	n	%	n	%
NSW/ACT	185/583	31.7	30/160	18.8	150/415	36.1
Qld	96/284	33.8	7/40	17.5	89/244	36.5
SA	101/392	25.8	9/73	12.3	92/319	28.8
Vic.	49/221	22.2	5/24	20.8	43/196	21.9
WA	66/296	22.3	7/54	13.0	59/242	24.4
Aus.	497/1,776	28.0	58/351	16.5	433/1,416	30.6

Figure 1. Trend in penicillin non-susceptible strains, AGAR surveys, 1989 to 2005



Pen I MIC 0.125–1 mg/L, Pen R MIC ≥2 mg/L

1989: Invasive n=105, non-invasive n=1,717, overall n=1,822.
1994: Invasive n=532, non-invasive n=1,835, overall n=2,385.
1999: Invasive n=381, non-invasive n=1,167, overall n=1,548.
2002: Invasive n=292, non-invasive n=717, overall n=1,009.
2005: Invasive n=351, non-invasive n=1,416, overall n=1,776

Cefotaxime/ceftriaxone

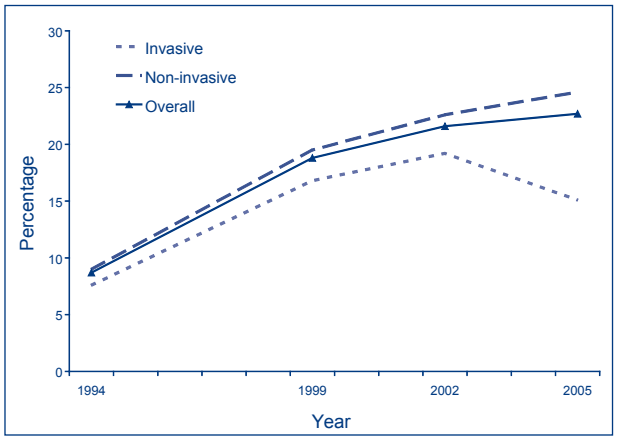
Cefotaxime or ceftriaxone MIC was determined on 471 (95%) penicillin non-susceptible isolates (MIC >0.064 mg/L). Non-susceptibility to the third generation cephalosporins was present in 14.9% of those strains tested. Of 14 cefotaxime/ceftriaxone resistant isolates (MIC >2 mg/L), 13 were also resistant to penicillin (2–8 mg/L), and 1 had intermediate resistance (0.38 mg/L). Of these, 13 were also resistant to macrolides, and 11 to tetracycline, but none were resistant to the fluoroquinolones. All 6 CSF samples were susceptible to ceftriaxone or cefotaxime using the lower (<0.5 mg/L) meningitis breakpoint.

Erythromycin and clindamycin

Macrolide (erythromycin) resistance was significantly higher ($p<0.001$) in non-invasive (24.6%) strains compared with invasive strains (15.1%) with the exception of Queensland, which had similar rates for both (28.3% and 27.5% respectively) (Table 2). The highest rate of resistance in non-invasive strains was seen in New South Wales/Australian Capital Territory, with a rate of 32%. The lowest rates of resistance for both invasive and non-invasive isolates were in Victoria (4.2% and 15.8% respectively). Resistance was highest among the elderly, and least in the 5–64 age range.

Erythromycin resistance increased from 1994 to 1999 for both invasive and non-invasive isolates. Comparison with the previous 2 studies revealed a rise in resistance for non-invasive isolates (from 19.5% to 24.6%), between 1999 and 2005, but this was not observed for invasive isolates (Figure 2).

Figure 2. Trends in erythromycin resistance, AGAR surveys, 1994 to 2005



1994: Invasive n=532, non-invasive n=1,835, overall n=2,385.
1999: Invasive n=381, non-invasive n=1,167, overall n=1,548.
2002: Invasive n=292, non-invasive n=717, overall n=1,009.
2005: Invasive n=351, non-invasive n=1,416, overall n=1,776

Clindamycin resistance was relatively uncommon among invasive strains (5.4% overall), with little variance across Australia (Table 3). A presumptive assessment of resistance genotype was made based

Table 2. Number and proportion of isolates with erythromycin resistance

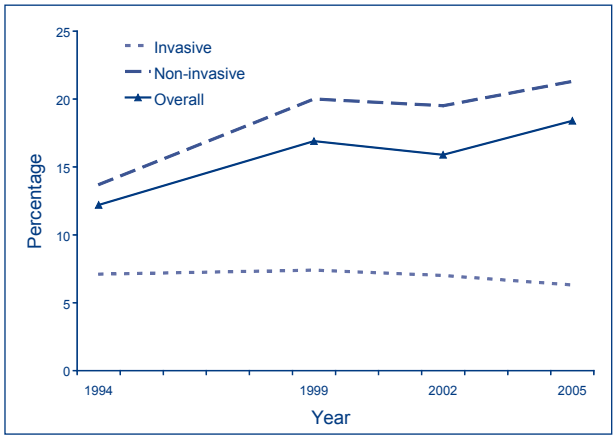
Region	All Isolates		Invasive		Non-invasive	
	n	%	n	%	n	%
NSW/ACT	162/583	27.8	26/160	16.2	133/415	32.0
Qld	80/284	28.2	11/40	27.5	69/244	28.3
SA	82/392	20.9	9/73	12.3	73/319	22.9
Vic.	35/221	14.5	1/24	4.2	31/196	15.8
WA	48/296	16.2	6/54	11.1	42/242	17.4
Aus.	404/1,776	22.7	53/351	15.1	348/1,416	24.6

on clindamycin resistance and the clindamycin disc induction test among erythromycin resistant isolates. Of 364 erythromycin resistant isolates, 222 (61%) had a MLS_B constitutive resistant phenotype and 6 (1.6%) had inducible resistance. These results suggest the presence of an *erm*B mechanism of resistance in 62.6% of Australian macrolide resistant isolates. All clindamycin resistant isolates were also resistant to erythromycin.

Tetracycline

There was a significant difference in tetracycline resistance among non-invasive (21.3%) and invasive (6.3%) strains ($p<0.001$). Compared with ß-lactam and macrolide resistance, tetracycline resistance in invasive isolates was consistently less than 10% across all Australian states (Table 4). Queensland and New South Wales/Australian Capital Territory had the highest rates of resistance demonstrated in non-invasive strains (24.6% and 25.5% respectively). Unlike rates for penicillins and macrolides, resistance was not higher in the young (<5 years), as would be predicted by lack of tetracycline use in this age group. Rates were again highest in the elderly. Tetracycline resistance increased from 1994 to 1999, all of the change being seen in non-invasive isolates (Figure 3). No significant rise in tetracycline resistance was demonstrated between 1999 and 2005 (16.9% and 18.4% respectively).

Figure 3. Trends in tetracycline resistance, AGAR surveys, 1994 to 2005



1994: Invasive n=532, non-invasive n=1,835, overall n=2,385.
1999: Invasive n=381, non-invasive n=1,167, overall n=1,548.
2002: Invasive n=292, non-invasive n=717, overall n=1,009.
2005: Invasive n=351, non-invasive n=1,416, overall n=1,776

Trimethoprim-sulphamethoxazole

Unlike other antibiotic classes, trimethoprim-sulphamethoxazole (TMP-SMX) resistance appears to have peaked in the previous decade. TMP-SMX resistance decreased from 1999 to 2005 for both invasive and non-invasive isolates (Figure 4). Nonetheless, rates of resistance of 25.6% for invasive and 32.2% for non-invasive strains limit the use of TMP-SMX in infections caused by *S. pneumoniae* (Table 5).

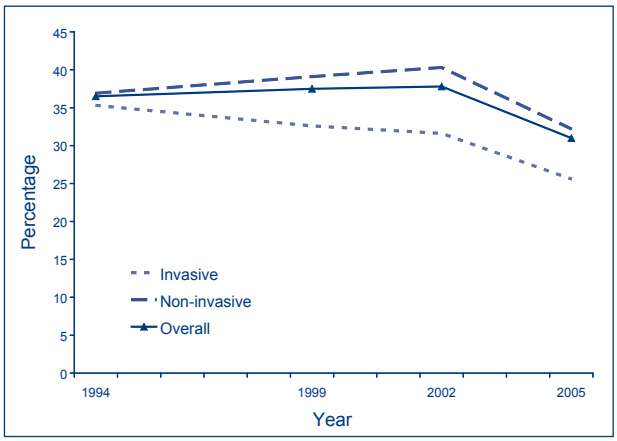
Table 3. Number and proportion of isolates with clindamycin resistance

Region	All Isolates		Invasive		Non-invasive	
	n	%	n	%	n	%
NSW/ACT	88/522	16.9	7/143	4.9	80/376	21.3
Qld	52/284	18.3	5/40	12.5	47/244	19.3
SA	53/309	17.2	2/67	3.0	51/242	21.1
Vic.	10/51	19.6	1/9	11.1	9/42	21.4
WA	25/296	8.4	2/54	3.7	23/242	9.5
Aus.	228/1,462	15.6	17/313	5.4	210/1,146	18.3

Table 4. Number and proportion of isolates with tetracycline resistance

Region	All Isolates		Invasive		Non-invasive	
	n	%	n	%	n	%
NSW/ACT	121/583	20.8	12/160	7.5	106/415	25.5
Qld	63/284	22.2	3/40	7.5	60/244	24.6
SA	60/391	15.3	2/73	2.7	58/318	18.2
Vic.	38/221	17.2	2/24	8.3	36/196	18.4
WA	44/296	14.9	3/54	5.6	41/242	16.9
Aus.	326/1,775	18.4	22/351	6.3	301/1,415	21.3

Figure 4. Trends in trimethoprim-sulphamethoxazole resistance, AGAR surveys, 1994 to 2005



1994: Invasive n=532, non-invasive n=1,835, overall n=2,385.
1999: Invasive n=381, non-invasive n=1,167, overall n=1,548.
2002: Invasive n=292, non-invasive n=717, overall n=1,009.
2005: Invasive n=351, non-invasive n=1,416, overall n=1,776

Fluoroquinolones

In 2005, fluoroquinolone resistance remained uncommon in Australia. By disc testing, levofloxacin resistance was detected in only 4 of 1,775 (0.2%) isolates tested. Intermediate resistance to levofloxacin was detected in another 4 isolates. All 8 were non-invasive isolates and were detected in New South Wales (4), South Australia (3) and Queensland (1). Moxifloxacin resistance was present in 2 isolates with MICs of 3 mg/L and 4 mg/L.

Multi-resistance

The most problematic strains of *S. pneumoniae* are those with multiple acquired resistances. Although there is no agreed benchmark for the definition of multi-resistance in *S. pneumoniae*, we have chosen acquired resistance to greater than 2 drug classes to define multi-resistance in this survey (Table 6). By this definition, 17.3% of isolates were multi-resistant.

Limitations of the study

There have been changes in participating laboratories in the AGAR *S. pneumoniae* surveys over time from 1989 through to 2005, with the more recent inclusion of a number of private pathology laboratories.

Table 5. Number and proportion of isolates with trimethoprim-sulphamethoxazole resistance

Region	All Isolates		Invasive		Non-invasive	
	n	%	n	%	n	%
NSW/ACT	198/583	34.0	43/160	26.9	151/415	36.4
Qld	101/284	35.6	8/40	20.0	93/244	38.1
SA	122/391	31.2	22/73	30.1	100/318	31.4
Vic.	65/221	29.4	7/24	29.2	58/196	29.6
WA	64/296	21.6	10/54	18.5	54/242	22.3
Aus.	550/1,775	31.0	90/351	25.6	456/1,415	32.2

Table 6. Multi-resistance in *Streptococcus pneumoniae*

Region	Number tested	Non-multi-resistant				Multi-resistant		
		0	1	2	%	3	4	%
NSW/ACT	583	307	90	62	78.7	42	82	21.3
Qld	284	144	48	31	78.5	13	48	22.5
SA	392	223	75	40	86.2	5	49	13.8
Vic.	221	140	31	16	84.6	15	19	15.4
WA	296	191	46	24	88.2	12	23	11.8
Australia	1,776	1,005	290	173	82.7	87	221	17.3
Invasive	351	216	77	37	94.0	12	9	6.0
Non-invasive	1,416	786	211	135	79.9	75	209	20.1

Antibiotics included: penicillin (intermediate or resistant); erythromycin, tetracycline, trimethoprim-sulphamethoxazole, levofloxacin.
Antibiotics excluded: clindamycin, cefotaxime, ceftriaxone.

Discussion

Internationally, changes in susceptibility in *S. pneumoniae* have evolved rapidly in the past 2 decades for both ß-lactam and other antibiotic classes, with potential to compromise treatment efficacy. Timely national surveillance studies that examine trends in antimicrobial resistance are essential for the formulation of appropriate, and up-to-date evidence-based therapeutic guidelines and in monitoring their ongoing relevance. This has implications for prescribing in situations where *S. pneumoniae* remains the major pathogen; primarily community-acquired lower and upper respiratory tract infections, including sinusitis and otitis media, as well as the empiric treatment of community-acquired meningitis. The AGAR has conducted studies in 1989, 1994, 1999, 2002 and 2005 to monitor changes in *S. pneumoniae* susceptibility.

In AGAR sponsored studies, penicillin non-susceptibility has continued to increase, albeit at a slower rate than first observed in the early 1990s, reaching a national level in this survey of 28%. This increase has occurred in both the penicillin intermediate and resistant categories. In 1989, only 1% of strains tested were penicillin non-susceptible and only intermediate resistance to penicillin was detected.[7] In 1994, 6.7% of isolates were non-susceptible, the majority with intermediate resistance. From 1999 to 2005, the rate of *S. pneumoniae* resistance doubled from 5.9% to 11.7%.[8,9]

Monitoring of invasive isolates is also performed by the Enhanced Surveillance Pneumococcal Working Group of the Communicable Diseases Network Australia and published yearly in *Communicable Diseases Intelligence*. In the 2005 report,[10] of 1,481 isolates tested, 11.9% had reduced susceptibility to penicillin, similar to but lower than the 16.5% reported in this survey. Monitoring of invasive strains is important in determining the burden of invasive infection and potential impact of this on vaccination strategies. The AGAR data also include non-invasive isolates. This provides a better estimate for the reservoirs and drivers of increasing community antibiotic resistance and for potential antibiotic prescribing failures in respiratory tract infections.

Increasing resistance rates will impact on empiric ß-lactam therapy but mainly for meningitis. In non-invasive pneumococcal infection in Australia, the currently recommended antibiotic of choice for oral therapy is amoxicillin.[11] When higher doses are employed, amoxicillin achieves adequate tissue levels and is active against penicillin-intermediate strains. Between 1999 and 2005 there was an almost twofold increase in the number of non-invasive isolates with a MIC ≥2 mg/L from 6.9% to 13.3%. These ongoing changes require continuing observation but amoxicillin prescribed at the upper dosing range, still continues to be appropriate therapy for the vast majority of non-invasive infections occurring in Australia.

Despite lower rates compared with non-invasive strains, increasing numbers of invasive strains also demonstrate high-level resistance to penicillin. The rates in invasive strains rose from 2.6% to 5.4% between 1999 and 2005. Reviews of the literature on the clinical implications of penicillin resistance[12,13] suggest that failures of therapy in non-meningeal infections are not predicted until MIC levels are ≥4 mg/L. As strains with MICs ≥4 mg/L are rare (1.6%) in Australia, data from the most recent AGAR study support current recommendations for high dose parenteral penicillin in non-meningeal invasive *S. pneumoniae* infection. Third generation cephalosporins in combination with vancomycin are recommended as empiric therapy for meningitis. When isolates have cefotaxime or ceftriaxone MICs ≥2 mg/L, even cephalosporins may be ineffective. However, of note in the 2005 study, all cerebrospinal fluid isolates were susceptible to the third generation cephalosporins using the CLSI meningeal breakpoints.

In 2005, erythromycin resistance was documented in 22.7% of isolates, a rise of 5% since 1999. Increases were primarily in non-invasive isolates. Of 364 erythromycin resistant isolates, 228 (62.6%) had a MLS_B phenotype suggestive of an *erm*B resistance mechanism, and associated with high-level erythromycin resistance. It has now been clearly documented that there is an association between macrolide resistance and therapeutic failures in bacteraemic *S. pneumoniae* infection.[14,15] The AGAR study data suggest that if macrolides are used as sole empiric therapy for respiratory tract infections when pneumococci may be implicated, many of these isolates (24.6% of non-invasive strains) are unlikely to respond to this class of antibiotics. The current Australian Antibiotic Guidelines[11] do not recommend macrolides as empiric therapy associated with otitis media, sinusitis, acute exacerbations of chronic bronchitis or as empiric therapy for community-acquired pneumonia except where additional cover for atypical organisms is required.

Tetracycline resistance in invasive isolates was below 10% across all Australian states. This may reflect the restricted use of oral tetracyclines for non-invasive infections in adults, and its restricted use for all infections in children.

A fall in resistance was demonstrated for trimethoprim-sulphamethoxazole from 37.8% in 2002 to 31.0% in 2005. Whilst it is considered an unsuitable drug for use in respiratory tract infection, the fall may reflect the reduction in antibiotic pressure

due to a decline in prescribing of this drug in the community. It is notable that the tetracycline and folate synthesis inhibitor drugs were the 2 classes of antibiotics to show significant reduction in community prescribing over the past 14 years, 53% and 54% reduction respectively (unpublished data – Drug Utilisation Subcommittee, Australian Government Department of Health and Ageing).

The new 'respiratory' fluoroquinolones, such as moxifloxacin are valuable drugs for the therapy of *S. pneumoniae* infections, particularly for infection by multi-drug resistant strains. However in countries where fluoroquinolones (particularly levofloxacin) have been used widely for respiratory tract infections, rising resistance levels are seen.[16–18] As for macrolides, quinolone resistance has been linked to failure of pneumonia therapy.[19] Because of the recent introduction of the new fluoroquinolones into clinical use, the AGAR 2005 *S. pneumoniae* study undertook an evaluation of resistance rates using levofloxacin discs and moxifloxacin Etest® MIC testing to detect early development of resistance. Of 1,776 isolates tested, only 2 moxifloxacin resistant non-invasive isolates were detected (0.1%). Both isolates were multi-resistant. The low fluoroquinolone resistance levels are likely to be related to the relatively low volume of these drugs that have so far been used in Australia because of restrictions placed on their use. Australian guidelines do not endorse fluoroquinolone use as first line therapy in community-acquired pneumonia.

Total antibiotic prescriptions in the Australian outpatient community have shown a steady and progressive fall from 1994 through to 2003 i.e. 25.6 to 19.8 DDD/1,000 population/day. In the last 2 years, this trend has reversed but still remains 15% below 1994 levels i.e. 21.7 DDD/1,000 population/day (unpublished data – Drug Utilisation Subcommittee, Australian Government Department of Health and Ageing). This overall reduction in antibiotic prescribing in the Australian community is likely to be a contributing factor to the relatively lower resistance levels seen in Australia compared with other regions such as Eastern Europe and East Asia.

Although rising levels of antibiotic resistance are often regarded as inevitable, a number of studies have shown a decline in resistance trends after implementation of community prescribing restrictions.

Regular AGAR surveillance studies of *S. pneumoniae* resistance have allowed a much clearer picture of changing susceptibility in this important community pathogen. After a rapid emergence and rise in resistance between 1989 and 1999, recent studies have documented a gradual rise or decrease for non-ß-lactam antimicrobials. Rates of 'high-level' penicillin resistance and multi-resistance continue to rise. Overall, based on AGAR studies, Australia has lower rates of resistance in *S. pneumoniae* than many other countries. Introduction and maintenance of effective vaccination programs as well as continuing promotion of prudent use of antibiotic prescribing in the community are crucial to maintaining effective control on resistance. If Australia can adopt these measures, there is an opportunity to curb this rise and to effect a reduction in resistance rates and preserve current antibiotics into the future. Ongoing surveillance studies such as the ones performed by the AGAR group are an important tool in measuring continuing trends in antibiotic resistance and as an indirect measure of the successes and failures of our health policies.

A full detailed report of this study may be found under 'AMR surveillance' on the Australian Group on Antimicrobial Resistance web site: http://www.antimicrobial-resistance.com/

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Western Australia

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Enhanced case detection for newly acquired hepatitis C infection: epidemiological findings and health service implications

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Abstract

Identifying newly acquired hepatitis C infections and describing their epidemiological characteristics has public health importance but can be resource intensive. We developed a new approach to conducting surveillance for newly acquired hepatitis C infection and analysed the epidemiological findings and health service implications. Doctors and laboratories in the Australian state of Victoria are required by law to notify all hepatitis C diagnoses to the Department of Human Services, but the routine report is limited to basic demographic information. For all cases reported as being aged 16–19 years or having clinical or laboratory indicators of newly acquired infection, during the period July 2004 to December 2005, additional information was sought from diagnosing doctors and used to classify cases as 'newly acquired' or 'unspecified' using a standard case definition. Of the 4,561 hepatitis C notifications received by the Department during the study period, 415 (9%) were selected for follow up and 148 of these (36%) were classified as newly acquired infections, compared with 4%–10% achieved from previous systems. Based on the enhanced data collection, the most common risk factor for transmission among newly acquired infections was injecting drug use (86%), the median age was 23 years, 59% were males and the predominant reason for testing was drug and alcohol screening (32%). This surveillance system was much more efficient at detecting newly acquired cases of hepatitis C infection than other approaches used in Victoria. Initial results show that injecting drug use continues to be by far the predominant mode of hepatitis C transmission in Victoria. *Commun Dis Intell* 2008;32:250–256.

Keywords: hepatitis C, surveillance, epidemiology, Australia

Introduction

Identifying and characterising cases of newly acquired (or incident) hepatitis C infection (HCV) enables public health officials to determine who is at risk, and assess and improve prevention efforts. It also provides a mechanism for monitoring the occurrence of transmission whether due to injecting drug use, or rarer pathways such as sexual contact[1] and skin-piercing, medical or cosmetic procedures.[2] Finally, diagnosis of hepatitis C in its acute or early stages provides the opportunity for treatment with a very high likelihood of clearance.[3]

Although active surveillance of clinical viral hepatitis can detect incident hepatitis C infections,[4] the yield is generally quite low, because the vast majority of these infections are asymptomatic.[5] The most commonly used approach for epidemiological monitoring of hepatitis C infection in most countries has been passive surveillance, based on legislatively mandated case reporting by doctors and laboratories to health departments,[6] but in its basic form it also has a limited ability to systematically detect incident infections. Therefore enhanced surveillance, involving the collection of information on prior hepatitis C testing and clinical history, has been undertaken to enhance the yield of incident cases.[6,7]

Doctors and laboratories in the Australian state of Victoria have been required to notify all diagnoses of hepatitis C infection to the Department of Human Services (DHS) since 1991. In 2000, DHS implemented a system in which all notifying doctors were followed up, and asked to provide enhanced information on risk factors and recency of infection. Because this approach was highly resource intensive, it was replaced in 2001 with a 10% random sample strategy[8,9] and in 2002, by an approach that targeted individuals aged 20 years and under, prisoners and military personnel.[10] A further major revision to the system was introduced in June 2004. This paper describes the new system, uses its output to identify predictors of recent hepatitis C infection, and compares its attributes to those of alternative systems.

Methods

Notification process

Doctors complete a standard notification form used for all notifiable diseases, including patient demographics, the disease being notified and, for hepatitis C, whether the disease was acute or not, its onset date and clinical symptoms and doctor contact details. Laboratories send a copy of the pathology report. Testing is based on routine laboratory diag-

nostic methods, which would generally begin with an antibody test for hepatitis C, and may include tests for hepatitis C RNA.

A duplicate search is then conducted on the DHS notifiable disease database. Cases with a history of a previous HCV notification (which may include re-infections) will not be counted or followed up.

Enhanced surveillance system

Cases of hepatitis C that were notified by a doctor or laboratory in people aged 16–19 years, or with specific indicators of recent acquisition, were selected for enhanced surveillance. The specific indicators that triggered enhanced surveillance were:

(a) doctor describes infection as acute;

(b) clinical indicator: any clinical suggestion of incident hepatitis C infection written on the doctor notification form i.e. bilirubin in urine, jaundice or elevated liver function tests;

(c) laboratory indicator: any evidence of a prior negative hepatitis C result or ALT 7 times the upper limit of normal.

The 16–19 years age group was selected to maximise the number of newly acquired infections detected, under the assumption that transmission among injecting drug users (IDUs), the primary hepatitis C risk group, is likely to occur most frequently within the first few years of initiation into injection,[11,12] an event that takes place at a median age around 18–19 years.[13,14]

For cases selected for enhanced data collection, surveillance officers contacted the doctor recorded on the notification form to obtain the information on reason for test, clinical indicators of acute hepatitis C, hepatitis C testing history (past negative and positive antibody and RNA tests), risk factors, results for hepatitis B serology and whether hepatitis A and B vaccines had been offered. The specific list of risk factors were consistent with those recommended as part of the National Hepatitis C Surveillance Strategy.[7] If the doctor could not provide the required information, permission to speak to the patient was sought and if obtained, the patient was contacted. Doctors were initially contacted by phone, however if repeatedly unavailable a letter and enhanced surveillance form were faxed. If the reported mode of transmission was other than injecting drug use, the case was referred for additional follow up.

A case was classified as either 'newly acquired' or 'unspecified' according to the Communicable Diseases Network Australia case definition for hepatitis C.[15] The Box shows the criteria used for the classification of hepatitis C newly acquired and unspecified cases.

No ethical approval was received for the enhanced surveillance as the activities were conducted on behalf of and in collaboration with DHS and the enhanced data collection procedures are covered under government legislation.

Statistical analyses

When more than one risk factor was reported a nationally recommended hierarchy was employed and the case classified according to the risk factor recorded as the most common.[16]

Cases classified as newly acquired on the basis of the follow up data collection (from either doctor or patient) were compared with those not classified as newly acquired, with regard to demographic and risk factor variables. Univariate analysis was conducted to identify variables associated with being classified as newly acquired. Identified predictors were then analysed using a multiple logistic regression model.

Box. Communicable Diseases Network Australia newly acquired hepatitis C case definition

Hepatitis C (newly acquired) – meets at least one of the following criteria

- Detection of anti-hepatitis C antibody from a person who has had a negative anti-hepatitis C antibody test recorded in the past 24 months
- detection of hepatitis C virus by nucleic acid testing from a person who has had a negative anti-hepatitis C antibody test result within the past 24 months
- detection of anti-hepatitis C antibody from a child aged 18–24 months
- detection of hepatitis C virus by nucleic acid testing in a child aged 1–24 months
- detection of anti-hepatitis C antibody or hepatitis C virus RNA and clinical evidence (jaundice or bilirubin in urine or ALT 7 times upper limit of normal)

Hepatitis C unspecified case

- Has laboratory definitive evidence (antibody or nucleic acid testing) and does not meet any of the above criteria for newly acquired case and is aged more than 24 months

Based on the final classification of cases (newly acquired or unspecified) we assessed the positive predictive value of the criteria that had been used to select cases for follow up.

Surveillance system attributes including resource utilisation

Attributes of various types of surveillance were compared. Calculation of resources used was based on an average 2.3 hours that was required to follow up each of the 415 notifications reported in this analysis. The number of staff days required per method was then calculated by multiplying the 2.3 hours per case by the expected number of cases followed up.

Data were managed and analysed using Stata Version 9.[17]

Results

Between July 2004 and December 2005, 4,561 notifications of hepatitis C were reported. On the basis of the selection criteria for additional data collection, 415 (9%) were followed up. The diagnosing doctor could not be contacted for 37 (9%) cases of which 3 were subsequently classified as newly acquired based on the receipt of laboratory results indicating a prior negative test.

Of the 415 cases, most (n=260, 63%) were followed up on the basis of clinical or laboratory indicators, with over half of these being reported as acute at the time of notification (n=146, 56%). A further 163 were followed up due to the age being recorded in the range 16–19 years and 29 satisfied the follow up criteria on the grounds of both age and clinical or laboratory indicators.

Newly acquired hepatitis C infection

Enhanced information allowed 148 of the 415 cases (36%) to be classified as newly acquired infections; the majority (70%, n=104) based on a record of having had a negative hepatitis C test within the 24 months prior to diagnosis and the remainder (30%, n=44) based on a record of clinical evidence consistent with the national case definition for acute hepatitis C infection.

A total of 182 hepatitis C notifications were followed up because they were recorded as being in a person aged 16–19, and of these cases, 27% (n=49) were ultimately classified as newly acquired. Based on clinical or laboratory indicators, 260 notifications were followed up with 44% (n=115) finally classified as newly acquired infections.

Predictors of newly acquired hepatitis C infection

The median age of infections classified as newly acquired was 23 years (range: 16–52 years) compared with 19 years (range: 2–70 years) for unspecified infections. A lower proportion of acute cases were aged 16–19 years (33%) compared with unspecified infections (51%) but a higher proportion were aged less than 40 years (94%) compared with unspecified infections (85%). Being younger than 40 years was a significant predictor (OR=6.0, 95% CI= 2.1–16.9) in multivariate analysis.

A higher proportion of acute cases were born in Australia (82%) compared with unspecified infections (60%). In univariate analysis being born in Australia was predictive of newly acquired infection (OR=2.4, 95%CI=1.1–4.4) but was non-significant in the multivariate model.

Cases ultimately classified as acute were most often tested as part of a drug and alcohol screen (32%), or for clinical signs and symptoms of acute hepatitis C (27%); the corresponding proportions for unspecified infections were 23% and 4% respectively with the latter reason found to be an independent risk factor (OR=9.1, 95% CI=3.9–21.5) for newly acquired infection in the multivariate analysis.

The most frequently reported risk exposure in the previous 2 years among newly acquired hepatitis C infection was injecting drug use, reported by 86%, compared with 60% in unspecified infections. In multivariate analysis, this factor was predictive of newly acquired infection (OR=2.9, 95%CI=1.4–6.0). Other exposures were each reported at low levels (less than 3%) among newly acquired cases, but some were slightly more frequent than in unspecified infections; resulting in moderate associations in univariate analysis for tattooing (OR=1.9, 95% CI=1.1–3.3); piercing (OR=2.2, 95% CI=1.2–4.3) and being in prison (OR=1.8, 95%, CI, 1.01–3.2), that were all non-significant in the multivariate model. (Table 1).

Positive predictive value of 'surveillance reason for follow up' for newly acquired infections

There was considerable variation in the degree to which the clinical and laboratory selection criteria were able to predict the likelihood of a case ultimately being classified as acute, and the positive predictive value of these criteria also varied with age. The predictive value of both clinical and laboratory criteria was high for those aged 16–19 years and then decreased sharply with age, whereas for cases followed up because the doctor had indicated that they were acute, the predictive value was generally lower and unrelated to age (Table 2).

Table 1. Characteristics of hepatitis C infections and predictors of hepatitis C newly acquired infections

Variable	Subgroup	Newly acquired		Unspecified		Univariate OR 95% CIs	Multivariate OR 95% CIs**
		n=148	%	n=267	%		
Age group	<40 years	139	93.9	222	85.4	**2.6 (1.2, 5.6)**	**6.0 (2.1, 16.9)**
	40+ years*	9	6.1	38	14.6	–	
Sex	Female	61	41.2	123	46.1	0.8 (0.6, 1.2)	
	Male*	87	58.8	144	53.9	–	
Region of birth	Australia	122	82.4	161	60.3	**2.4 (1.1, 4.4)**	
	Other*	11	10.3	35	13.1	–	
	Unknown	15	17.4	71	26.6	1.5 (0.6, 3.9)	
Surveillance reason for follow up*	Doctor described infection as acute	68	45.9	78	29.2	**2.0 (1.3, 3.1)**	**2.1 (1.2, 3.7)**
	Aged 16–19 years	49	33.1	135	50.6	0.3 (0.2, 0.5)	
	Clinical indicator	32	21.6	30	11.2	**2.2 (1.3, 3.7)**	**2.1 (1.01,4.5)**
	Laboratory indicator	27	18.2	21	7.9	**2.6 (1.4, 4.8)**	**4.3 (2.0, 9.2)**
Diagnosing clinic type	General practitioner	83	56.1	155	58.1	0.9 (0.6, 1.3)	
	Other*	62	42.0	100	37.4	–	
	Unknown	3	2.0	12	4.5	0.4 (0.11, 1.46)	
Reason for test*	Drug and alcohol screening	47	31.8	62	23.2	1.3 (0.9, 2.1)	**1.8 (1.04, 3.2)**
	Symptoms and signs of acute hepatitis	40	27.0	11	4.1	**7.8 (3.9, 15.9)**	**9.1 (3.9, 21.5)**
	Patient request	38	25.7	65	24.3	0.9 (0.6, 1.5)	
	Abnormal LFTs	35	23.6	30	11.2	**4.4 (2.4, 8.2)**	**2.5 (1.3, 5.0)**
	Other screening	24	16.2	30	11.2	**3.0 (1.6, 5.8)**	**2.6 (1.3, 5.2)**
	Other*	37	25.1	166	62.2	–	
Risk factor†,‡	Injecting drug use	127	85.8	161	60.3	**3.9 (2.3, 6.5)**	**2.9 (1.4, 6.0)**
	Tattoo	4	2.7	1	0.4	**1.9 (1.1, 3.3)**	
	Piercing	3	2.0	1	0.4	**2.2 (1.2, 4.3)**	
	Sexual partner hepatitis C positive	3	2.0	9	3.4	1.3 (0.8, 2.2)	
	Surgery	3	2.0	6	2.2	1.2 (0.5, 2.6)	
	Household contact hepatitis C positive	2	1.4	7	2.6	1.0 (0.5, 1.9)	
	Prison	1	0.7	3	1.1	**1.8 (1.01, 3.2)**	
	Other	6	4.0	24	9.0	0.8 (0.4, 1.5)	
	Risk factor not determined	3	2.0	11	4.1	0.4 (0.1, 1.3)	

Significant variables are in bold type.

OR Odds ratio

* The reference group used to calculate the odds ratio.

† Not mutually exclusive, multiple responses could be ticked on the enhanced surveillance form. For the univariate and multivariate analysis, the 'yes' response was compared to the 'no' response.

‡ When injecting drug use was reported only injecting drug use was reported. For cases with non-injecting drug use risk factors all risk factors were reported

Enhanced hepatitis C surveillance system attributes

We estimated that it would take 17.8 days per week (or more than 3 full time surveillance officers) to follow up all notifications received (Table 3). A 10% random sample would require 2.9 days per week for a yield of 14 cases per year. The strategy described in this paper of following up cases with clinical or laboratory indicators and all notifications in people aged 16–19 years would yield approximately 100 newly acquired infections per year with a surveillance officer working 2.5 days a week.

Table 2. Positive predictive value of 'surveillance reason for follow up' for newly acquired hepatitis C infections, by age group

Surveillance reason for follow up*	Age group (years)	Positive predictive value (%)
Doctor described infection as acute	16–19	54
	20–24	62
	25–29	53
	30+	36
Clinical indicator	16–19	100
	20–24	77
	25–29	38
	30+	38
Laboratory indicator	16–19	73
	20–24	60
	25–29	50
	30+	45

* Not mutually exclusive – multiple reasons may be selected.

Following up cases based on clinical or laboratory indicators only, produced a greater yield of newly acquired infections (44%) per case followed up, compared with the age-specific strategy alone (27%). It was estimated that it would require 2 days of work by the surveillance officer to detect 76 newly acquired infections per year. An age-specific strategy (16–19 years) would require 1.7 days work by the surveillance officer to follow up an estimated 33 newly acquired cases. A strategy based on a 22–24 years age group would yield an estimated 124 newly acquired infections, and widening the age group to 20–24 years would identify 186 cases per year and require an estimated 3.8 days per week.

Discussion

The selection of hepatitis C notifications for follow up based on either young age or clinical or laboratory indicators identified newly acquired hepatitis C infections more frequently (36%) than systems that involved follow up of all cases (10%)[18] or a 10% random sample (4%).[8]

Among newly acquired infections, the median age of cases was 23 years. This finding is consistent with the expected average age of hepatitis C seroconversion for IDUs; the reported average age of onset of injecting among IDUs in Australia is between 18 and 19 years[13,14] and the average time to seroconversion after beginning to inject was estimated to be approximately 3 years in a study conducted in the United States of America.[19]

Injecting drug use continued to be by far the predominant mode of hepatitis C transmission in Victoria, as it has been in the rest of Australia and other Western countries.[1] A very low percentage of newly acquired

Table 3. Specific attributes of different enhanced surveillance methods

Enhanced surveillance methods	Estimated cases per year	Estimated cases per week	Estimated surveillance days per week	Estimated proportion of cases identified as newly acquired infections (%)	Estimated Number of newly acquired infections per year	Demographically representative of newly acquired infections
'Clinical or laboratory indicators' or 'aged 16–19'	277	5.3	2.5	36	100	No
'Clinical or laboratory indicators'	173	3.3	2.0	44	76	Yes
Aged 16–19 years	121	2.3	1.7	27	33	No
Aged 19–21years	237	4.6	2.3	37*	81	No
Aged 22–24 years	336	6.5	2.8	37*	124	No
Aged 20–24 years	504	9.7	3.8	37*	186	No
10% random sample	341	6.6	2.9	4†	14	Yes
All cases	3,040	58.5	17.8	10‡	304	Yes
Passive surveillance	3,040			none	0	No

* Based on results from the 1996 New South Wales system.[1]

† Based on results from the 2001 Victorian system.[2]

‡ Based on the assumption that the proportion would be higher for those aged ≥19 years.

infections reported tattooing or sexual contact with a hepatitis C positive individual as a risk exposure; none of these individuals reported a history of IDU. It is possible that the reported occurrence of sexual risk may be an over-estimation because information on injecting drug use was not obtained from 2 of the 3 newly acquired cases. Although there are reports accumulating, which suggest that hepatitis C transmission can occur sexually among HIV positive men who have sex with men,[1] overall the occurrence of sexual transmission of hepatitis C remains rare.[20]

The enhanced surveillance system described here was not representative of all hepatitis C notifications, due to the incorporation of the age-specific strategy. The strength of this strategy is that it provides information on a well defined population that is at elevated risk of hepatitis C infection. The strategy could be extended to include a wider age range, but would require significantly more resources.

Although the follow up of cases with clinical or laboratory indicators of acute infection identified a higher proportion (44%) of newly acquired infections than the age-specific strategy it was somewhat surprising that more than half of these cases could not be confirmed as newly acquired. The confirmation rate would be substantially increased if follow up based on clinical or laboratory indicators was restricted to those aged less than 30 years. The lower positive predictive values in older cases may be due to the increase likelihood of chronic hepatitis infection with increasing age.[21]

There are several limitations that need to be considered when interpreting these findings. Firstly, a small number of doctors and laboratories were unable to be contacted to obtain enhanced surveillance information, resulting in these notifications being classified as unspecified. However, due to the small number it is unlikely that this 'loss to follow up' had any substantial impact on our results. Secondly, the assignment of risk factor is probably more accurate for newly acquired infections than it is for longer standing infections, as the risk occurred within a 2 year time frame and is hence less subject to recall bias.

Finally, any community wide surveillance system for hepatitis C has the inherent limitation that infections are rarely symptomatic in the early stages, and that most cases will therefore remain undetected.[5] Even if testing is conducted, it may be difficult to distinguish a newly diagnosed case as newly acquired, unless there is a history of a recent negative test prior to the positive diagnosis.

Considering the limitations of enhanced surveillance as a means of identifying and characterising newly acquired hepatitis C infections, alternative approaches to measuring hepatitis C incidence may also be necessary. Recruitment of people at risk of hepatitis into research cohorts is one option but it is very expensive and likely to be unrepresentative. Clinical services which offer regular testing to people at risk of hepatitis C provide another mechanism for monitoring incident cases.[16] The number of diagnoses detected in this way will be dependent on the regularity of client visits, but such cases may be more broadly representative of community patterns of infection than cases arising in a research cohort.

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Short reports

What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? A 2007 update

Brynley P Hull, Shelley Deeks, Robert Menzies, Peter B McIntyre

Abstract

In 2001, a publicly funded 7 valent pneumococcal conjugate vaccine (7vPCV) program commenced for Aboriginal and Torres Strait Islander children aged less than 2 years. This study updates early estimates of 7vPCV coverage in Aboriginal and Torres Strait Islander children using Australian Childhood Immunisation Register data between 31 December 2004 and 30 September 2007. We chose four 3-month birth cohorts of children and assessed their immunisation status at 12 months of age for pneumococcal conjugate vaccine and for 'fully immunised'. After the introduction of universal childhood conjugate pneumococcal vaccination in 2005, 7vPCV coverage increased substantially among Aboriginal and Torres Strait Islander children nationally, and in all jurisdictions but remained lower than among non-Indigenous children. The results re-emphasise the greater impact of universal, compared with targeted, programs on vaccine coverage among Indigenous children. *Commun Dis Intell* 2008;32:257–260.

Keywords: pneumococcal, vaccination coverage, immunisation register, Indigenous

Introduction

Invasive pneumococcal disease (IPD) became largely preventable in Australian children less than 2 years of age for the first time with the approval of pneumococcal conjugate vaccine in December 2000, which targeted the 7 most common IPD serotypes in non-Indigenous children. However, these 7 serotypes were responsible for a substantially lower proportion of IPD among Indigenous children in central and northern Australia who have the highest incidence of IPD.[1–3] In June 2001, a publicly funded 7 valent pneumococcal conjugate vaccine (7vPCV) program commenced for Aboriginal and Torres Strait Islander and other high risk children aged under 2 years and since May 2001, 7vPCV vaccination encounters have been recorded on the Australian Childhood Immunisation Register (ACIR). On 1 January 2005, the publicly funded program was expanded to include all Australian children under 2 years of age. In 2004, an initial evaluation of 7vPCV coverage among Indigenous children from ACIR data showed that estimated 7vPCV coverage increased over time but was still less than 50% for all jurisdictions except the Northern Territory, Queensland, and Western Australia. Importantly, since this time, the completeness of recording of Indigenous status has improved.[4]

The aim of this study was to evaluate trends in 7vPCV coverage in Indigenous children by jurisdiction since the introduction of universal pneumococcal conjugate vaccination and compare this to 'fully immunised' (not including 7vPCV) coverage for the same children.

Methods

Immunisation status assessment

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases receives downloads of ACIR immunisation data from Medicare Australia each quarter. This analysis was undertaken using ACIR data as of 31 December 2004 and 30 September 2007. We chose four 3-month birth cohorts, the first cohort included children born between 1 January 2003 and 31 March 2003 (using ACIR data as at 31 December 2004), the other 3 cohorts using ACIR data were as at 30 September 2007 (born between 1 April 2004 and 30 June 2004, 1 January 2006 and 31 March 2006, and 1 April 2006 and 30 June 2006). The immunisation status of children, recorded on the ACIR as Aboriginal and Torres Strait Islander, in the 4 birth cohorts was assessed at 12 months of age for pneumococcal conjugate vaccine and for 'fully immunised'. 'Fully immunised' was defined as receipt of all of 3 doses of diphtheria-tetanus-pertussis and poliomyelitis, and the second or third dose of *Haemophilus influenzae* type b and hepatitis B vaccines used to assess whether a child is completely immunised at 12 months of age in all years under study. Immunised for 7vPCV was defined as receipt of 3 doses of 7vPCV vaccine by 12 months of age in all years under study. The third dose assumption was applied in this analysis, so if the third dose was recorded as administered, it

was assumed that previous doses were administered.[6] The analysis was undertaken using the SAS software system.[7]

Aboriginal and Torres Strait Islander population denominators

In addition to ACIR Aboriginal and Torres Strait Islander denominator data, data on the number of Aboriginal and Torres Strait Islander births in all states and territories for the year 2006 was also obtained from the Australian Bureau of Statistics (ABS).[8] If the Aboriginal and Torres Strait Islander field on the ACIR was left blank, it was assumed that the child was not of Aboriginal or Torres Strait Island descent.

Results

Table 1 compares the number of Aboriginal and Torres Strait Islander children recorded on the ACIR (using the Indigenous indicator) and ABS (using Indigenous registered births) databases in Australia in 2002 and 2006. In 2002, the number of children in Australia identified as Aboriginal and Torres Strait Islander by the ACIR was 60% of the ABS estimates, with much variation between the states and territories (ranging from 98% in the Northern Territory to 14% in Queensland). However, by 2006 there was substantial improvement throughout the country and, in some jurisdictions, (the Australian Capital Territory, the Northern Territory, Queensland and Western Australia) the ACIR identified more children as Aboriginal and Torres Strait Islander than the ABS.

Table 2 shows a comparison of 7vPCV vaccine coverage estimates with 'fully immunised' coverage estimates for Aboriginal and Torres Strait Islander children for the 4 study birth cohorts. Prior to the introduction of universal childhood pneumococcal vaccination in Australia in 2005, 'fully immunised' and 7vPCV coverage improved marginally in Aboriginal and Torres Strait Islander children from the 2003 cohort to the 2004 cohort, with increases varying by jurisdiction. However, with the exception of the Northern Territory, 7vPCV vaccine coverage estimates in all jurisdictions remained considerably lower than 'fully immunised' coverage estimates for all scheduled vaccines at 12 months of age. After the introduction of universal pneumococcal vaccination for all Australian children, 7vPCV coverage increased substantially among Aboriginal and Torres Strait Islander children nationally and in all jurisdictions, although it remained below 80% in South Australia and Western Australia. Importantly, differential coverage between 7vPCV and other scheduled vaccines ('fully immunised') was absent after the introduction of the universal conjugate pneumococcal vaccination program.

Table 1. Comparison of the number of Aboriginal and Torres Strait Islander children, ACIR data versus ABS, 2002 versus 2006

State or territory	Indigenous population – ACIR data 2002*	Indigenous population (births) – ABS 2002 †	Ratio of ATSI identified by ACIR / ABS (%)‡	Indigenous population – ACIR data 2006§	Indigenous population (births) – ABS 2006‖	Ratio of ATSI identified by ACIR/ABS (%)¶
ACT	72	105	68.6	132	109	121.1
NSW	2,714	3,568	76.1	3,249	3,516	92.4
NT	1,422	1,445	98.4	1,615	1,565	103.2
Qld	489	3,493	14.0	3,852	3,463	111.2
SA	498	665	74.9	586	733	79.9
Tas.	109	482	22.6	327	413	79.2
Vic.	495	680	72.8	684	782	87.5
WA	1,411	1,653	85.4	1,970	1,910	103.1
Aust.	7,210	12,094	59.6	12,415	12,496	99.4

* Numbers for the 12 month birth cohort (born 1 January 2002 to 31 December 2002).

† Australian Bureau of Statistics. Births Australia 2002. Canberra: Australian Bureau of Statistics. Projected indigenous births from the 1996 Census.

‡ Accuracy of Australian Childhood Immunisation Register (ACIR) Indigenous data (The number of children estimated by the ACIR for 2002/the number of Census projection births in 2002)*100.

§ Numbers for the 12 month birth cohort (born 1 January 2006 to 31 December 2006).

‖ Australian Bureau of Statistics. Births Australia 2006 Canberra: Australian Bureau of Statistics. Projected indigenous births from the 2001 Census.

¶ Accuracy of ACIR Indigenous data (The number of children estimated by the ACIR for 2006/the number of Census projection births in 2006)*100.

Table 2. A comparison of 7vPCV and 'fully immunised' coverage estimates calculated from the ACIR for Aboriginal and Torres Strait Islander children born in four 3-month birth cohorts

State or territory	n*	% coverage for cohort born 1/1/03 – 31/3/03		% coverage for cohort born 1/4/04 – 30/6/04		% coverage for cohort born 1/1/06 – 31/3/06		% coverage for cohort born 1/4/06 – 30/6/06	
		7vPCV	Fully immunised†	7vPCV	Fully immunised	7vPCV	Fully immunised	7vPCV	Fully immunised
ACT	32	42.1	79.0	70.4	92.6	85.2	85.2	90.6	90.6
NSW	764	40.9	85.3	51.2	81.4	86.0	85.6	83.0	82.9
NT	422	79.2	80.3	82.6	84.5	86.7	85.2	87.7	87.9
Qld	954	60.8	85.6	62.9	82.1	86.5	85.2	87.1	86.2
SA	149	39.4	81.7	53.2	78.0	77.2	76.0	77.9	77.2
Tas.	67	13.0	87.0	31.1	92.2	95.4	95.4	85.1	88.1
Vic.	186	17.5	90.3	44.8	88.0	83.6	85.3	88.2	86.0
WA	527	51.5	80.2	55.1	75.9	79.2	77.7	78.6	78.0
Aust.	3,101	52.5	83.6	58.4	81.9	84.9	84.0	84.3	83.8

* Total number of Indigenous children in the cohort born 1 April 2006 to 30 June 2006.

† All of the third doses of diphtheria-tetanus-pertussis and poliomyelitis, and second or third dose of *Haemophilus influenzae* type b and hepatitis B vaccines used to assess whether a child is completely immunised at 12 months of age.

Table 3 compares 7vPCV coverage estimates between Aboriginal and Torres Strait Islander and non-Indigenous children in one cohort of children born after the introduction of the universal pneumococcal vaccination program in 2005. It shows that coverage for 7vPCV at 12 months is lower among Aboriginal and Torres Strait Islander children than among non-Indigenous children overall and in all jurisdictions.

Summary

This analysis has confirmed earlier reports[4] that the reporting of Indigenous status on the ACIR has improved substantially in recent years and is now high in almost all jurisdictions. The trend in increased reporting, previously shown to be 95% by 2005[4] has increased further to 99% in 2007. The ACIR can now be used with more confidence by vaccination program managers and other public health practitioners to estimate vaccine coverage in Aboriginal and Torres Strait Islander children.

The updated analysis of recent ACIR data allows a comparison of 7vPCV immunisation coverage among Aboriginal and Torres Strait Islander children in Australia before and after the introduction of the pneumococcal conjugate vaccine program to all Australian children in 2005. Prior to this date, the publicly funded 7vPCV program only included Aboriginal and Torres Strait Islander children. The main finding is that 7vPCV coverage among Aboriginal and Torres Strait Islander children in Australia increased substantially between the 2 periods, despite the fact that the program was publicly funded throughout for this population. This suggests that universal programs targeting all Australian children rather than solely Aboriginal and Torres Strait Islander children,[9] may positively impact on coverage for the latter group of children. While under-reporting of 7vPCV vaccinations to the ACIR may have been more pronounced prior to 2005, leading to an under-estimation of coverage in that period, a survey of Queensland Indigenous children also produced similar findings of lower coverage with non-standard vaccines, including 7vPCV.[10]

Table 3. A comparison of 7vPCV coverage estimates at 12 months of age for Aboriginal and Torres Strait Islander versus non-Indigenous children in the cohort born between 1 April and 30 July 2006

State or territory	% coverage for Indigenous	% coverage for non-Indigenous
ACT	90.6	94.2
NSW	83.0	91.5
NT	87.7	90.7
Qld	87.1	91.4
SA	77.9	91.8
Tas.	85.1	91.9
Vic.	88.2	91.5
WA	78.6	90.0
Aust.	84.3	91.4

It is also worthwhile to note that 7vPCV coverage among Indigenous children has now approached that of other vaccines on the immunisation schedule. While coverage for other vaccines at 12 months of age has been lower in Indigenous compared to non-Indigenous children, this disparity disappears

by 24 months of age, indicating that timeliness is the main obstacle to maintaining equity in coverage at 12 months of age.[11] Although there have been substantial gains throughout the time period under study, 7vPCV coverage among Aboriginal and Torres Strait Islander children is still less than that of non-Indigenous children. States and territories should continue to ensure that all children, especially Aboriginal and Torres Strait Islander children, are receiving all recommended vaccines in a timely manner.

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Likely impact of school and childcare closures on public health workforce during an influenza pandemic: a survey

Craig B Dalton, David N Durrheim, Michael A Conroy

Abstract

During an influenza pandemic, public health staff may not report to work due to illness, transport disruptions or care responsibilities, including care of children if school closures occur. A survey was conducted in a population health unit to estimate the impact of closure of schools and day care facilities on staff ability to work at their usual workplace or at home, and determine their access to the Internet for communication. Staff were also asked about concerns associated with working from home. Eighty-seven staff completed a paper based survey. Thirty-eight per cent (33/87) of staff may be absent from work due to the impact of childcare and school closure, however 24 (73%) of these staff would be able to work from home with most having access to dial-up (87%) and broadband Internet access (71%). Staff reported concerns about potential exposure to pandemic influenza, the need for personal protection and clearly defined roles and training, availability of adequate equipment and technology to work from home, and sick leave provisions during a pandemic. While school and childcare closures will have a significant impact on public health agency staff, they have the capacity and willingness to work from home. Their practical concerns should be addressed to optimise their participation. *Commun Dis Intell* 2008;32:261–262.

Keywords: disease outbreak, public health response, pandemic influenza

Background

Studies exploring the willingness of the clinical and public health workforce to report for duty during a pandemic suggest the need for public health agencies to plan for absenteeism and to develop methods for staff to work from home.[1,2] Staff may not report to work due to pandemic influenza or other illness, transport disruptions or care responsibilities, including care of children if school closures occur. Hunter New England Population Health is an integrated health protection and health promotion unit of NSW Health with 120 full time equivalent staff of which only 12 staff routinely perform health protection functions compatible with pandemic influenza response. All 120 staff may be called upon to provide support for case assessment, contact tracing and prophylaxis, and provision of public information during a pandemic.

A survey was conducted to estimate the impact of school and day care facility closures on staff ability to work at their usual workplace or at home and to determine their access to the Internet for communication. Staff were also asked to identify any concerns or questions associated with working from home.

Methods

Staff were provided with a standardised paper questionnaire in July 2006. They were asked to indicate whether they would be available to report to the workplace if schools or child care centres were closed due to a pandemic. Those unable to report to the workplace were asked if they would be available to work from home, how many hours per week they would be available and whether they had dial up or broadband Internet access. All responses were anonymous.

Results

Eighty-seven of 120 (72%) staff completed the survey. Up to 38% (33/72) of staff may be absent from work due to the impact of combined childcare and school closure, however 73% of these (24/33) would be able to work from home. Of these 24, most had access to dial-up (87%) or broadband (71%) Internet connections and 15 who estimated the amount of hours they could work from home, estimated 6 to 40 hours with a median of 30 hours per week.

Of 18 concerns reported by staff, 6 related to potential exposure to pandemic influenza or the need for personal protection, 4 to the requirement to have clear roles and training in a pandemic, 3 to the availability of adequate equipment and technology to work from home, and 3 to workers compensation or sick leave provisions during a pandemic.

Discussion

This survey of population health staff suggests that school and childcare closures will have a marked impact on absenteeism among workers responsible for public health disease control and surveillance functions. However, their ability and willingness to work from home would compensate, at least in part, for likely absenteeism.

The UK Influenza Contingency Plan used modelling to predict that 25% of employees would take between 5 and 8 days off in the 3 to 4 months of a pandemic.[3] At least 5% to 7% would be absent in the peak week of impact, with the proportion rising to 15% if the attack rate were to increase from 25% to 50%. The Australian Health Management Plan for Pandemic Influenza predicts peak workforce absenteeism rates of up to 50%.[4] It is likely that absenteeism to provide child care will be a significant contribution to total absenteeism if child care facilities and schools close. Arrangements that produce alternate child care-like facilities will be discouraged by authorities and may be resisted by parents and relatives due to concerns about the transmission of influenza if children are grouped in informal child care arrangements.

Public health disease control responses rely on access to and dissemination of information via email and shared access to databases. During the Australian National Cumpston Pandemic Exercise, NSW Health's NetEpi program was used via an online interface to manage all case assessment and contact tracing information, and surveillance reporting was conducted using an online interface. It may be difficult to procure new broadband connections once a pandemic has occurred, however, staff in many locations may be able to access broadband wireless Internet services through the use of plug-in wireless cards.

In addition to delays in gaining broadband Internet access, significant lead time may be required to establish secure methods for accessing internal network resources, such as virtual private networks, to ensure security of confidential patient information. Scenario modelling exercises performed within the financial sector in the United States of America suggested that the increased use of the Internet for distance-learning and recreation will result in decreased online performance in areas where schools were closed. By the peak of the pandemic, broad impacts across access networks, and residential Internet service throughput was reduced to 50% of normal due to congestion at the end, or 'last mile' of the network nodes.[5]

As might be expected, workers in a high technology environment, such as a health department, have higher rates of home Internet access (86%) compared to the general community (60%) and much higher rates of broadband access, 82% versus 31%.[6] Australia has similar household access to Internet and broadband to the United Kingdom, the United States of America and Germany.[6]

During the initial consultation with staff on the rationale for this survey, some employees without children responded that the survey was too narrow and that employees might be unable to work during a pandemic due to care for disabled or aged relatives as well. This survey specifically sought to estimate the impact of school and childcare closure, as this is predictable and described in the National Pandemic Plan.[4] It is recognised that absenteeism would rise above these levels due to illness among staff and due to their care role for family and friends and this should be acknowledged by planners.

Conclusions

Methods for rapidly connecting key public health staff to the Internet from home, such as wireless access, should be explored. Education and policy development for pandemic preparedness should transparently address personal protective equipment and workplace infection control, define specific roles for specific staff, and clarify workers compensation and sick leave provisions during a pandemic.

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Barmah Forest virus serology; implications for diagnosis and public health action

Patrick Cashman, Linda Hueston, David Durrheim, Peter Massey, Stephen Doggett, Richard C Russell

Abstract

Barmah Forest virus (BFV) is a commonly occurring arbovirus in Australia. Notifications of Barmah Forest infections diagnosed by a single positive IgM serology test have been increasing in coastal New South Wales north of Newcastle. We report on a 6 month prospective review of all routine notifications of BFV from the Lower Mid North Coast of New South Wales. Sera from 37 consecutive cases were sent for confirmatory testing by ELISA and neutralisation assays and 32 cases were interviewed. On confirmatory testing, 7 patients' sera (19%) was found to contain no BFV antibodies and 6 (16%) had BFV IgG only. Only 4 cases had antibody levels compatible with recent infection. A clinical presentation of fever with either rash or joint pain was associated with confirmation of recent BFV infection. On the basis of these findings, caution is advised in the interpretation of a single positive IgM for Barmah Forest disease and the clinical picture is an important factor in the diagnosis. Serological notifications of BFV alone should not prompt public health action such as public warning and targeted vector control in endemic areas. *Commun Dis Intell* 2008;32:263–266.

Keywords: Barmah Forest, arbovirus, serology, false positive

Introduction

Barmah Forest virus (BFV) is an alphavirus transmitted by mosquitoes that was first isolated in Australia in 1974.[1] Human infections have since been reported from all states and territories.[2] Symptoms of BFV infection include acute onset of fever, arthralgia and a florid maculopapular erythematous rash, after an incubation period of 5–21 days.[3–5] The disease has a seasonal occurrence with the highest incidence during the summer and autumn for most of Australia except south-east Western Australia where peak incidence is in spring.[6] There is no specific treatment available and so management is primarily directed at alleviation of symptoms.

The arthralgia, myalgia and accompanying lethargy, may be incapacitating. As 10% of cases remain symptomatic for more than 6 months, there would be attendant economic consequences yet to be quantified.[7]

BFV is most commonly transmitted in coastal New South Wales by the salt-marsh mosquito, *Aedes* (*Ochlerotatus*) *vigilax*.[8] As many breeding sites are in environmentally sensitive locations and occur over extensive geographical areas, source reduction is often not acceptable or practical, and opportunities for vector control are usually limited. This species' extensive flight range, which often exceeds 5 kilometres, also complicates vector control. The urban mosquito, *Aedes (Finlaya) notoscriptus,* which can use domestic containers for breeding, has also been implicated in transmission. *Aedes* (*Ochlerotatus*) *procax*, a freshwater species that breeds in forested ground-pools, has produced many isolates of BFV over recent years from the New South Wales Arbovirus Surveillance and Mosquito Monitoring Program and may also have a significant role in BFV transmission.[9]

The natural reservoir of BFV is yet to be determined. Circumstantial evidence suggests a more mobile (perhaps avian) host since epidemics spread rapidly, genetic analysis has found that isolates are homogeneous (a feature typical of bird-associated viruses), and studies on placental and marsupial mammals have shown these groups to be incompetent reservoirs.[10–13]

Of the nearly 2,500 BFV cases notified in the past 11 years in New South Wales, 80% occurred in the coastal region north of Newcastle.[14] BFV appears to be of increasing public health importance in this area, which is experiencing rapid population growth. In the first decade of BFV notification in New South Wales, outbreak years were followed by 2–3 seasons of low activity.[15] In the past 5 seasons, however, there have been sustained higher notification rates without a return to the previous baseline.[14]

BFV cases are notifiable in New South Wales under the *NSW Public Health Act, 1991*. A confirmed case requires only laboratory evidence either by:

- isolation of BFV; or
- detection of BF virus by nucleic acid testing; or
- IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to BFV; or
- detection of BFV- specific IgM.[16]

As a precursor to a possible case control study to explore the effectiveness of personal protective measures, we embarked on a 6 month prospective review of all routine notifications of BFV from the Lower Mid-North Coast (LMNC) of New South Wales. This region north of Newcastle encompasses 3 Local Government Areas roughly defined from Hawkes Nest in the South to Taree in the North along the coast and inland to the mountains west of Gloucester. The Mid-North Coast area of New South Wales has had the highest rate of BFV infection in Australia (67.5 cases per 100,000 population in 2005).[17] The purpose of the review was to investigate how many notified BFV cases during this 6 month period were actually recent infections.

Methods

All notifications of BFV in the LMNC area of New South Wales between 1 January and 30 June 2007 were investigated to describe the clinical presentation and confirm laboratory diagnosis.

Cases were interviewed by structured questionnaire for clinical presentation, particularly for onset date, prior medical history, exposure details and clinical features at presentation; specifically fever, rash, lethargy, arthralgia and myalgia.

Sera from the notifying laboratories were forwarded to the Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research (CIDMLS-ICPMR), Westmead Hospital for confirmatory testing as follows:

- BFV IgG (in-house indirect IgG ELISA standardised against neutralisation) sensitivity 98%, specificity 97.8% (personal communication, Linda Hueston, CIDMLS-ICPMR)
- BFV IgM (in-house antibody class capture ELISA standardised against neutralisation) sensitivity 97.9%, specificity 98.9% (personal communication, Linda Hueston, CIDMLS-ICPMR)
- neutralisation (micro-neutralisation using a 90% end-point).

Neutralisation titres of greater than or equal to 640 on single serum samples were considered diagnostic of recent infection. A seroconversion or fourfold or greater rise in neutralising antibody titre between acute and convalescent sera were considered to be diagnostic of recent infection.

Results

There were 37 BFV notifications from 1 January to 30 June 2007 for the LMNC. Five patients could not be contacted despite repeated attempts, and therefore 32 people were interviewed. All 37 cases were notified by laboratories following routine diagnosis as BFV IgM positive using the PanBio kit. Sera from all 37 BFV cases was sent for confirmatory testing.

On confirmatory testing, 7 patient's sera (19%) was found to contain no BFV antibodies and 6 (16%) had BFV IgG only (which was confirmed by neutralisation). The remaining notifications had both IgM and IgG antibodies (confirmed by neutralisation) but only 4 had antibody levels/titres compatible with recent infection. The antibody level/titre in 20 cases indicated prior infection.

Of the 32 patients who were interviewed, all were symptomatic with: 13/32 (41%) having fever, 5/32 (16%) having rash, 24/32 (75%) reporting fatigue, 16/32 (50%) arthralgia and 10/31 (32%) myalgia on initial presentation.

Self-reported fever was the only symptom statistically significantly associated with laboratory evidence of recent BFV infections (Table). All 4 cases that were laboratory-compatible with recent infection had fever and either rash or arthralgia at presentation. Of the 24 cases who presented with fatigue, only 3 were confirmed to have had recent BFV infection (Table).

Table. Clinical presentation and likelihood of confirmed recent Barmah Forest infection in this cohort of recent Barmah Forest virus notifications, Lower Mid North Coast, 1 January to 30 June 2007

Symptoms reported	ICPMR laboratory confirmed cases of recent BFV infection (n=4)		Other cases notified as BFV infection on initial laboratory result (n=28)		p-value (Two-tailed Fisher's Exact)
	n	%	n	%	
Fever	4	100	10	36	0.028
Rash	2	50	3	11	0.105
Fatigue	3	75	21	75	1.000
Arthralgia	2	50	14	50	1.000
Myalgia	0	0	10 (n=27)	37 (n=27)	0.277

Discussion

The high false positive rate of 19% (7/37) in this series of notifications would suggest that caution is advisable in interpreting a single positive BFV IgM result as diagnostic of recent BFV infection.

Our findings suggest that a clinical picture of fever with either rash or joint pain would be of assistance in the application of BFV serology in areas where BFV is endemic. Testing on the basis of a single symptom of fatigue would seem to be less reliable as a clinical criteria of recent infection. However it is important to distinguish between the typical macular rash of BFV disease and rashes with fever that could herald more ominous diagnoses, including measles and invasive meningococcal disease.

Amongst this small cohort of patients, a relatively large proportion had evidence of previous BFV infection, and thus clinicians need to be cautious in ascribing the 'classic' symptoms to recent BFV infection in an endemic area on the basis of a single positive test in the absence of fever.

The majority of BFV infections are subclinical or inapparent.[8,18] As BFV virus appears to be endemic in the LMNC, testing needs to be guided by the appropriate group of symptoms for BFV, as false positive results could lead to misdiagnosis and other necessary clinical investigations erroneously omitted.

Laboratory reporting of results may be aided by the treating clinician providing details of onset date and symptoms. Optical density readings, cut-off ratios or titres may assist clinicians and public health authorities in determining whether an infection is recent.

Obvious limitations of currently available commercial arboviral tests must be borne in mind when interpreting the results of epidemiological studies and understanding the distribution and trends of BFV notifications.[19] Sero-surveillance has the potential for directing and catalysing public health action, which could include public warning and targeted vector control in affected areas.[20] However, these LMNC findings make it advisable to conduct confirmatory testing before committing resources for responding to suspected incursions or increases in BFV disease.

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Quarterly reports

OzFoodNet quarterly report, 1 January to 31 March 2008

The OzFoodNet Working Group

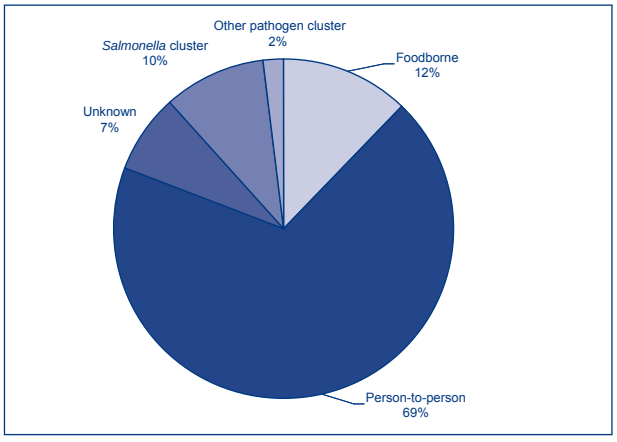
Introduction

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 January to 31 March 2008.

Data were received from OzFoodNet representatives in all Australian states and territories. The data in this report are provisional and subject to change as the results of outbreak investigations can take months to finalise.

During the first quarter of 2008, OzFoodNet sites reported 245 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 3,518 people, of which 118 were hospitalised and 13 died. The majority (68.7%, n=169) of outbreaks resulted from infections due to person-to-person transmission (Figure).

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 January to 31 March 2008



Foodborne disease outbreaks

There were 29 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 492 people and resulted in 38 people being admitted to hospital. There were no deaths. This compares with 40 outbreaks for the first quarter of 2007 and 28 outbreaks in the fourth quarter of 2007.

Salmonella was responsible for 11 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype. There were 3 outbreaks due to *S.* Typhimurium 135a and 2 each due to phage types 44, 126, 170 and 9.

There were 3 foodborne outbreaks of *Campylobacter* infection and one of norovirus during this quarter. There were 4 toxin-related outbreaks during the quarter, including 2 *Clostridium perfringens* outbreaks, one mixed outbreak of *C. perfringens* and *Bacillus cereus* and 1 ciguatera fish poisoning outbreak. The remaining 10 outbreaks were caused by unknown aetiological agents.

Twelve outbreaks reported in this quarter were associated with food prepared at restaurants, 7 from food prepared at private residences and 5 with food prepared by commercial caterers. There were 3 outbreaks associated with institutions, and one each with an aged care facility and with camping.

To investigate these outbreaks, sites conducted 9 cohort studies, 1 case control study, and collected case series data for 19 investigations. Investigators obtained analytical epidemiological evidence in 4 outbreaks and microbiological evidence in four. For the remaining 21 outbreaks, investigators obtained descriptive evidence implicating the food vehicle or suggesting foodborne transmission.

The following jurisdictional summaries describe key outbreaks and public health actions which occurred in this quarter.

Australian Capital Territory

The Australian Capital Territory did not report any foodborne outbreaks during the first quarter of 2008.

Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2008

State	Month of outbreak	Setting prepared	Agent responsible	Number affected	Evidence	Responsible vehicles
NSW	February	Commercial caterer	*C. perfringens/B. cereus*	75	M	Chicken curry, curry pumpkin, rice with lamb, plain rice
	January	Restaurant	Unknown	7	D	Suspected marinated mussels
	March	Private residence	*S.* Typhimurium 170	17	D	Custard cake
	January	Private residence	*S.* Typhimurium 126/126 var 1	41	M	Eggs
	March	Restaurant	*S.* Typhimurium 126/126 var 1	3	D	Raw egg dressing
	March	Institution	*C. perfringens*	48	D	Curry
	January	Restaurant	Unknown	3	D	Unknown
	January	Restaurant	Unknown	2	D	Unknown
	February	Institution	Unknown	6	D	Unknown
	February	Restaurant	Unknown	4	D	Unknown
	March	Restaurant	Unknown	3	D	Unknown
NT	March	Restaurant	*S.* Typhimurium 9	11	D	Unknown
Qld	February	Restaurant	*Campylobacter*	2	D	Chicken
	February	Restaurant	*Campylobacter*	4	D	Chicken liver pate
	March	Restaurant	Unknown	6	D	Unknown
	March	Private residence	Ciguatera Fish Poisoning	2	D	Yellowtail kingfish
	March	Institution	Norovirus	56	A	Deli meat & salad dish
Vic	January	Private residence	*S.* Typhimurium 135a	18	D	Mixed foods
	January	Private residence	*S.* Typhimurium 135a	7	M	Ice cream cake
	January	Private residence	*S.* Typhimurium 44	12	D	Lemon dessert
	February	Commercial caterer	*S.* Typhimurium 170	18	A	Chicken and pasta salad and ham
	February	Commercial caterer	*Campylobacter*	4	A	Chicken and pasta salad
	February	Commercial caterer	Unknown	21	A	Continental custard cake
	February	Aged care	*Clostridium perfringens*	6	D	Unknown
	March	Commercial caterer	*S.* Typhimurium 44	24	D	Unknown
Tas	January	Restaurant	*S.* Typhimurium 135a	78	M	Suspected eggs
SA	Jan	Camping	Unknown	5	D	Suspected milk
	March	Restaurant	Unknown	6	D	Unknown
WA	January	Private residence	*S.* Typhimurium 9	3	D	Suspected chicken

* No foodborne outbreaks were reported in 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.

New South Wales

Eleven outbreaks of foodborne illness were reported from New South Wales and the Hunter sites during this quarter.

Salmonella Typhimurium was associated with 3 outbreaks, all linked with consuming egg products. In the first egg-associated outbreak, there were 41 cases of *Salmonella* Typhimurium Multilocus Variable Number Tandem Repeat Analysis (MLVA) type 3-17-16-13-523 infection. Where phage typing was completed, 81.5% (22/27) were phage type 126 and 18.5% (5/27) were phage type 126 var 1. The median age of cases was 40 years and 61% were female. Most cases were resident of the Central Coast and Sydney areas. A local egg producer/retailer/wholesaler was consistently identified in hypothesis generating interviews and the NSW Food Authority conducted an environmental investigation. *S.* Typhimurium with the same MLVA type was isolated from manure samples collected in 3 of 5 egg sheds at the egg producer's property. Local media were engaged to warn the public of the potential risk of foodborne disease associated with egg and chicken products.

Three of 10 people became ill following a meal at a Central Coast restaurant in a second egg-associated outbreak of *S.* Typhimurium MLVA type 3-17-16-13-523 that was related to the community-wide one reported above. The restaurant used raw egg mayonnaise to dress Caesar salad eaten by those who were ill. The restaurant sourced their eggs from the implicated egg producer mentioned previously. The NSW Food Authority advised the proprietor not to use raw eggs in dressings.

In the third egg-associated outbreak, 17 of 21 people sharing a common meal at a private function became infected with *S.* Typhimurium phage type 170 MLVA type 3-9-8-12-523. Illness was associated with eating cake made with raw-egg custard filling. *S.* Typhimurium was not isolated from the farm where the eggs were sourced.

An outbreak of *Clostridium perfringens* enterotoxin type A affected 48 of 100 people who had a curry meal at an army training facility. Temperature abuse of food and inadequate catering equipment and management were likely factors in promoting the growth of spore forming bacteria that led to illness.

New South Wales also reported an outbreak of *C. perfringens* and *B. cereus* affecting 75 people who ate a catered meal, as well 6 small outbreaks of unknown aetiology.

New South Wales also reported 15 listeriosis notifications during the first quarter, which was an increase of 88% compared with the first quarter in 2007. Of the 15 cases, 3 were in pregnant women, one of whom miscarried. Despite an investigation, no epidemiological links or microbiological links between the cases were established. A listeriosis alert was sent out to all general practitioners in New South Wales informing them of the illness and prevention measures.

Northern Territory

The Northern Territory reported an outbreak of *S.* Typhimurium 9 amongst people eating at a buffet restaurant in March 2008. An epidemiological study did not identify a vehicle of transmission and all food samples taken were negative for *Salmonella*. It was suspected that cross contamination in the kitchen occurred.

Queensland

Queensland reported 5 outbreaks of foodborne illness during the first quarter of 2008.

Two separate outbreaks of gastroenteritis due to *Campylobacter* occurred in restaurant settings in Brisbane during February 2008. The first outbreak affected 4 people who ate chicken liver pate at a restaurant. The second outbreak involved 2 people from the same household who had consumed chicken dishes at a different Brisbane restaurant. No food items were available for testing from either outbreak.

Two people became intoxicated with suspected ciguatera fish poisoning after eating yellowtail kingfish at a private residence. The fish was purchased from a local seafood business in Brisbane and was thought to have been caught off the New South Wales coast.

A large outbreak of norovirus gastroenteritis affected 40.6% (56/138) of attendees on a 5 day training course at a Brisbane academy. A retrospective cohort study identified an association between a cold meat and salad dish, provided by an outside caterer, and illness (RR=2.0, 95%CI: 1.5–2.7, p=0.004). Eight faecal specimens were submitted for testing and all were positive for norovirus. Transmission was suspected to be from person-to-food-to-person.

An outbreak of gastroenteritis of unknown aetiology occurred at a wedding reception held at a local golf club. Faecal specimens were negative for viral and bacterial pathogens and no food vehicle was identified.

South Australia

In January 2008, a suspected foodborne outbreak was reported in 5 children who were camping. The children reported drinking milk that was off and

developed sudden onset of violent vomiting, lethargy and dehydration. No pathogens were detected in faecal specimens.

Tasmania

Tasmania reported an outbreak of 78 cases of *S.* Typhimurium affecting people eating food from the same food business over a 4 day period. Forty-one isolates were typed as phage type 135a and one as an untypable phage type. Illness was strongly associated with eating products containing aioli made with raw eggs (OR 511, 95%CI 90–4709, $p<0.000$). *S.* Typhimurium 135a was isolated from 4 food items: aioli, tartare sauce, sweet potato salad and guacamole. All these foods contained raw egg ingredients, except guacamole that was commercially produced and may have become cross contaminated. The food business used eggs from the same producer implicated in similar outbreaks of *S.* Typhimurium 135a in Tasmania during 2005.[1]

Victoria

Victoria reported 8 outbreaks of foodborne illness during this quarter.

An outbreak of *Salmonella* Typhimurium 135a affected 18 of 30 guests at a family gathering on Christmas day. *S.* Typhimurium 135a was isolated from faecal specimens collected from 5 cases. Several different foods were associated with illness, although no definitive food vehicle was identified.

Another outbreak of *S.* Typhimurium 135a affected 6 people following consumption of ice cream cake prepared with raw eggs and served on Christmas day. Five cases had *S.* Typhimurium 135a isolated from faecal specimens and *S.* Typhimurium 135a was isolated from a sample of leftover ice cream cake. The eggs used to make the ice cream cake were sourced from a family member's private flock.

Twelve of 14 people at a family function were infected with *S.* Typhimurium 44, which was isolated from 6 faecal specimens. Lemon dessert made with raw eggs sourced from a backyard flock was suspected as the source of this outbreak.

An outbreak of S. Typhimurium 170 affected 32% (18/56) of a group of workers following a catered lunch. Eight cases had *S.* Typhimurium 170 isolated from a faecal specimen. Both chicken and pasta salad (RR 5; 95% CI 1.3–19.7) and ham (RR 3.7; 95% CI 1.1–11.4) were associated with illness. The chicken and pasta salad was prepared with a raw egg mayonnaise. The eggs were traced back to a farm that was investigated by the Victorian Department of Primary Industries.

Four people became infected with *Campylobacter* after a Christmas work function at a private residence. Analysis of food histories from 17 people showed that consumption of chicken and pasta salad prepared in a butcher shop was associated with illness.

Gastroenteritis of unknown aetiology affected 21 people attending a catered private party. Analysis of food exposures for 40 guests identified that illness was associated with eating a continental custard cake. One person who did not attend the party but ate leftover cake also became ill. Eleven faecal specimens were collected but no bacterial or viral pathogens were isolated.

An outbreak of diarrhoea due to *C. perfringens* occurred in an aged care facility in February. Five residents and 1 staff member were affected and 2 cases had *C. perfringens* enterotoxin isolated from faecal specimens. No source for the outbreak was identified.

In March 2008, an outbreak of *S.* Typhimurium 44 occurred amongst a film crew. Foods provided to the film crew were prepared differently each day making it difficult to conduct an investigation to identify the source of the outbreak. Twenty-seven cases were identified through an employee illness register.

Western Australia

Western Australia reported 1 foodborne outbreak during the quarter. In January, 3 members of a family from a regional town were admitted to hospital with bloody diarrhoea, vomiting and fever. Two faecal specimens were positive for *Salmonella* Typhimurium 9 with a Pulsed Field Gel Electrophoresis (PFGE) profile designated STYMAV.0108. There were no other reports of isolates with similar profiles in Western Australia at this time. Undercooked chicken was suspected as the source of the infection.

The National Gastroenteritis Survey II

During the quarter, OzFoodNet and the New South Wales Food Authority launched the National Gastroenteritis Survey II (NGSII). The NGSII is a national cross sectional survey to assess changes in the prevalence of gastroenteritis in Australia. The NGSII is a telephone survey conducted over 12 months in all states and territories, with an oversample in New South Wales.

There were 1,263 interviews completed between 4 February and 9 April 2008. Fifty-six point seven per cent of people contacted agreed to participate in the survey. In total, the crude proportion of survey respondents reporting gastroenteritis in the 4 weeks

prior to interview was 6.0% (76/1,263). OzFoodNet will be comparing the results of this survey to the previous national survey conducted in 2001–02 to provide important information on the changing nature of gastroenteritis and foodborne disease in Australia.[2]

Food recalls linked to microbiological contamination, January to March 2008

During the period January to March 2008 there were 8 food recalls that occurred due to microbiological contamination. Of these 8 microbial food recalls, 4 were due to contamination with *Listeria*, 1 was due to the presence of *Salmonella* and 3 were due to other microbial contamination. Where there was potential for human cases linked to these recalls jurisdictions carried out epidemiological investigations in response. Tuna steaks imported from Indonesia were recalled after testing revealed high levels of histamine. The steaks were tested as a result of consumer complaints.

Comments

During this quarter, 28% (8/29) of foodborne outbreaks were suspected to be due to eggs or egg-based dishes. Similar to previous reports, a wide range of foods were identified as a cause of these outbreaks, including desserts, salad dressings, sauces, and undercooked eggs.[3] In this report, 2 of the egg-associated outbreaks were attributed to eggs laid by backyard chickens.

During the quarter, there was a marked reduction in the reported number of person-to-person outbreaks. In the last 6 months of 2007, there were 1,179 outbreaks of gastroenteritis spread from person-to-person compared to 169 for this quarter. The majority of these outbreaks were due to norovirus. In 2006–2007, international reports of large numbers of gastroenteritis outbreaks due to norovirus highlighted the global nature of this virus.[4,5]

In this report, state and territory health departments used different molecular testing methods to further characterise strains of *S.* Typhimurium during outbreaks, with Queensland and New South Wales using MLVA and Western Australia using PFGE. These typing systems are highly discriminatory and very useful in foodborne outbreaks. Based on laboratory experience, jurisdictions have begun to use results of molecular tests of isolates for the purposes of epidemiological investigation prior to phage type results coming back from reference laboratories. To ensure that results are comparable between different jurisdictions, Australia needs to move towards a harmonised system of molecular typing salmonellas and other organisms for public health surveillance.[6]

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Communicable diseases surveillance

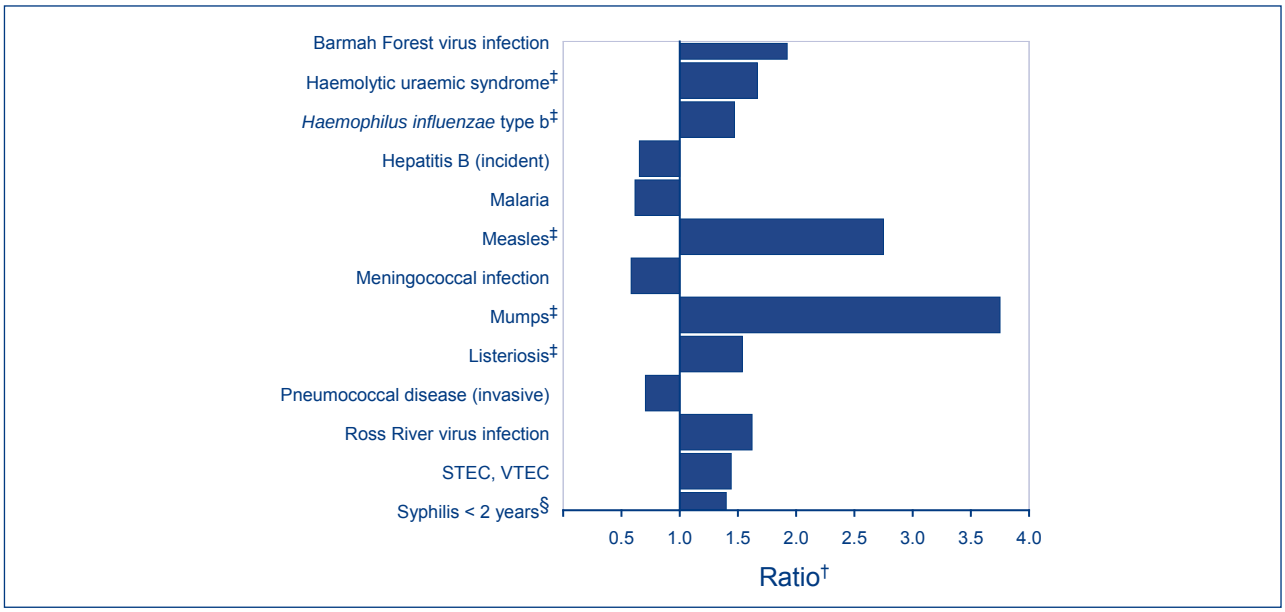
Highlights for 1st quarter, 2008

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 to the National Notifiable Diseases Surveillance System (NNDSS) with an onset in the first quarter (January to March) 2008, in comparison with the 5-year mean for the same period. Notifications were above the 5-year mean for the same period and exceeded 2 standard deviations from the 5-year mean for: Barmah Forest virus infection, haemolytic uraemic syndrome, listeriosis, measles and mumps. Notifications were above the 5-year mean, but less than 2 standard deviations from the 5-year mean, for *Haemophilus influenzae* type b, Ross River virus infection, Shiga toxin-producing/verotoxin-producing *Escherichia coli* (STEC/VTEC) and syphilis (less than 2 years duration). Notifications were below the 5-year mean for hepatitis B (incident), malaria, meningococcal infection and invasive pneumococcal disease.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2008 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 5 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 5 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 5 years.

‡ Where the mean of the current quarter exceeds the mean of the corresponding quarter for the previous 5 years by more than 2 standard deviations.

§ Ratio for syphilis of less than 2 years duration is based on 4 years data.

Vectorborne diseases

There are currently 8 notifiable mosquito-borne diseases under national surveillance in Australia. These include alphaviruses (Barmah Forest virus and Ross River virus), flaviviruses (dengue, Japanese encephalitis, Kunjin, Murray Valley encephalitis and flavivirus infection not elsewhere classified) and malaria.

In Australia the alphaviruses, Barmah Forest virus and Ross River virus, are of major public health significance, causing annual epidemics with seasonal peaks occurring between January and May each year. Infection with either of these diseases is characterised by rash, fever, fatigue and joint pain.

Barmah Forest virus infection

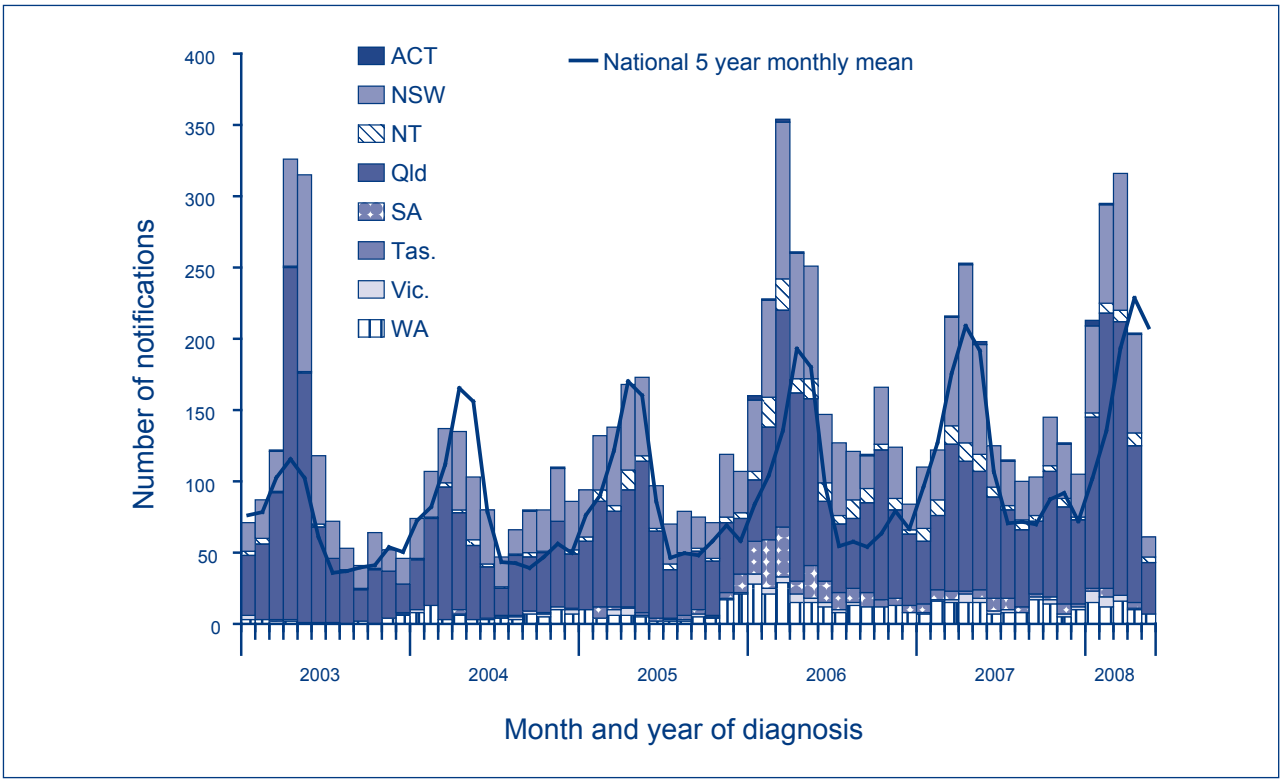
During the first quarter of 2008 there were 824 notifications of Barmah Forest virus infection, which was 93% higher than the 5-year mean for the previous corresponding quarters. All jurisdictions, except Tasmania, reported cases with the majority of cases notified from Queensland (n=505, 61%) and New South Wales (n=226, 28%).

The notification rate was highest in Queensland at 48.3 cases per 100,000 population (annualised), while in the Northern Territory, although only 18 notifications of Barmah Forest virus infection were received, the rate was 33.5 cases per 100,000 population. The rates in these jurisdictions were substantially higher compared with the other jurisdictions where rates were around 13.1 cases per 100,000 population in New South Wales, 8.2 cases per 100,000 population in Western Australia and 5.9 cases per 100,000 population in the Australian Capital Territory.

The total number of cases for the quarter represented a substantive increase from the previous quarter (n=373) and the same quarter in 2007 (n=436). The increase in cases in this quarter may be attributable to increased seasonal rainfall, especially in Queensland, higher than average overnight temperatures and other environmental conditions which promote mosquito breeding and the transmission of the virus.

Figure 2 shows the number of notifications for Barmah Forest virus infection received by NNDSS against the 5-year monthly mean for the period between 2003 and the first quarter of 2008. The entire period from June 2007 to March 2008 was above the 5-year monthly rolling mean, highlighting increased inter-seasonal and seasonal activity, with a higher number of notifications received in the first quarter of 2008 (n=824), compared with the seasonal peak within the first quarter of 2006 (n=742).

Figure 2. Notifications of Barmah Forest virus infection, Australia, 1 January 2003 to 31 March 2008, by month of diagnosis



Ross River virus infection

There were 2,756 notifications of Ross River virus infection reported in the first quarter of 2008, nearly 2.5 times higher than the corresponding period in 2007, however in comparison to the 5-year mean for the previous corresponding quarters it was only 1.6 times higher. Over half of the cases notified to NNDSS (n=1,560, 57%) were from Queensland. New South Wales reported 607 cases (22%) and Western Australia reported 258 cases (9%).

Although only 92 notifications of Ross River virus infection were reported from the Northern Territory, the notification rate was 171.2 cases per 100,000 (annualised), and in Queensland the rate was 149.2 cases per 100,000 population. The notification rates in the Northern Territory and Queensland were substantially higher than in other jurisdictions (49.0 cases per 100,000 population in Western Australia, 43.0 cases per 100,000 population in Tasmania and 35.2 cases per 100,000 population in New South Wales).

Figure 3 shows the number of notifications for Ross River virus infection received by NNDSS nationally against the 5-year rolling mean for the period between 2003 and the first quarter of 2008. As highlighted in this figure, the seasonal peak has shifted from March–April to February in the first quarter of 2008 (n=2,756) and appears to be moderately high and above the 5-year rolling mean, however it is not as high as the seasonal peak in the first quarter of 2006 (n=3,433).

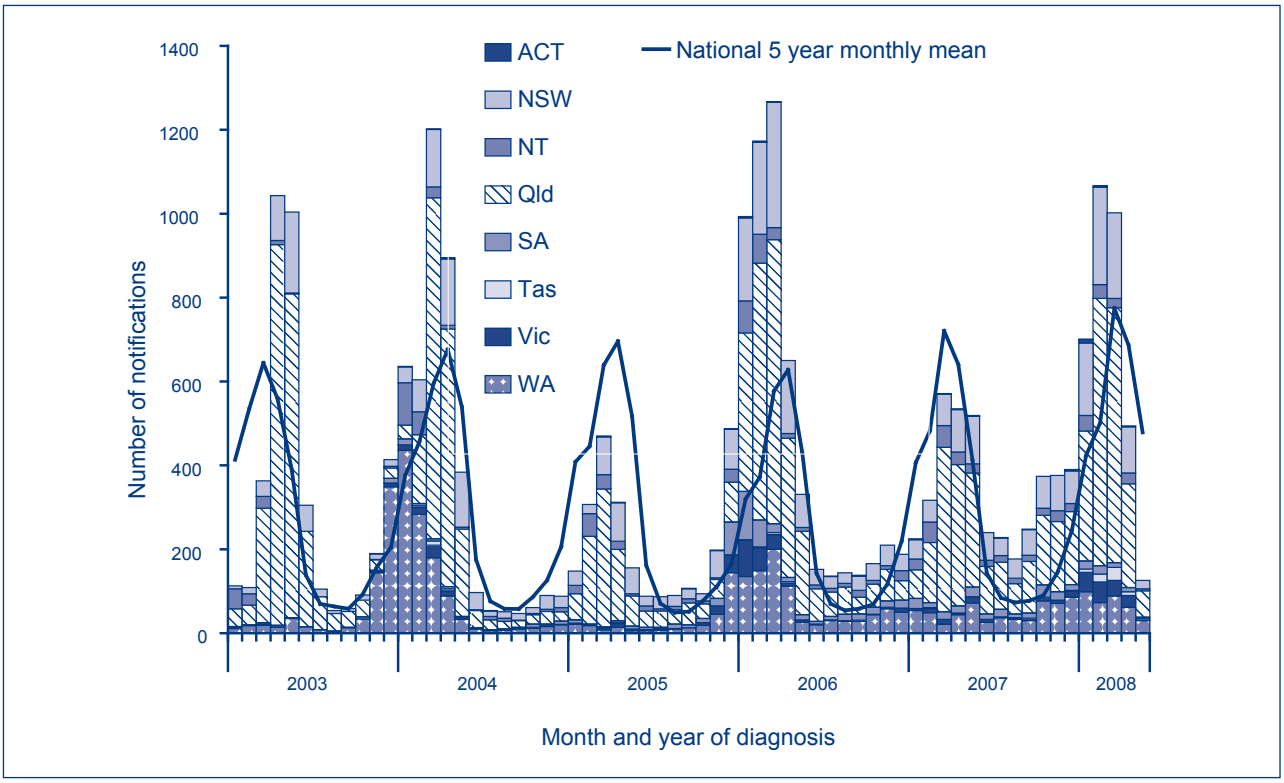
Vaccine preventable diseases

Measles

Measles is an acute, highly communicable viral disease that can lead to serious complications such as pneumonia (lung infection), encephalitis (inflammation of the brain) or otitis media (middle ear infection). In the past measles infection was a common childhood illness, but as a result of national immunisation campaigns measles is now rare in Australia, except for occasional outbreaks of limited duration that are generally linked to an imported case.[1]

Between 1 January and 31 March 2008, 33 cases of measles were reported to the NNDSS, three times the number of notifications received for the whole of 2007 (n=11). Cases were reported from New South Wales (n=15), Queensland (n=8), Western Australia (n=4), the Northern Territory (n=3), Victoria (n=2) and South Australia (n=1). The number of cases for the first quarter of 2008 (n=33) was 2.8 times higher than the 5-year mean (n=12). Figure 4 shows the epidemic curve of measles cases since 2003 by jurisdiction. The high number of

Figure 3. Notifications of Ross River virus infection, Australia, 1 January 2003 to 31 March 2008, by month of diagnosis



notifications reported in 2006 were associated with a touring Indian spiritual leader, which led to a multi-jurisdictional outbreak of measles in April 2006.

This quarter there was an annualised notification rate of 6.3 cases per 1,000,000 population, with 4.8 cases per 1,000,000 being locally acquired cases of measles and 1.5 cases per 1,000,000 reported as acquiring measles outside Australia. The annualised notification rate for this quarter is higher than in previous years, including the multi-jurisdictional outbreak that occurred in 2006 (6.1 cases per 1,000,000 population). This is due to several localised clusters and outbreaks in New South Wales and Queensland respectively, with secondary epidemiologically linked cases associated with cases who acquired measles outside Australia.

Fifty-eight per cent of cases were male and 42% were female. The age of cases ranged from less than 1 year to 48 years. Of the 33 cases, vaccination status was known for 26 cases. Three of the cases (9%) were reported as being fully vaccinated for age, 4 (12%) cases were reported as being partially vaccinated for age and 19 (58%) cases were reported as not vaccinated.

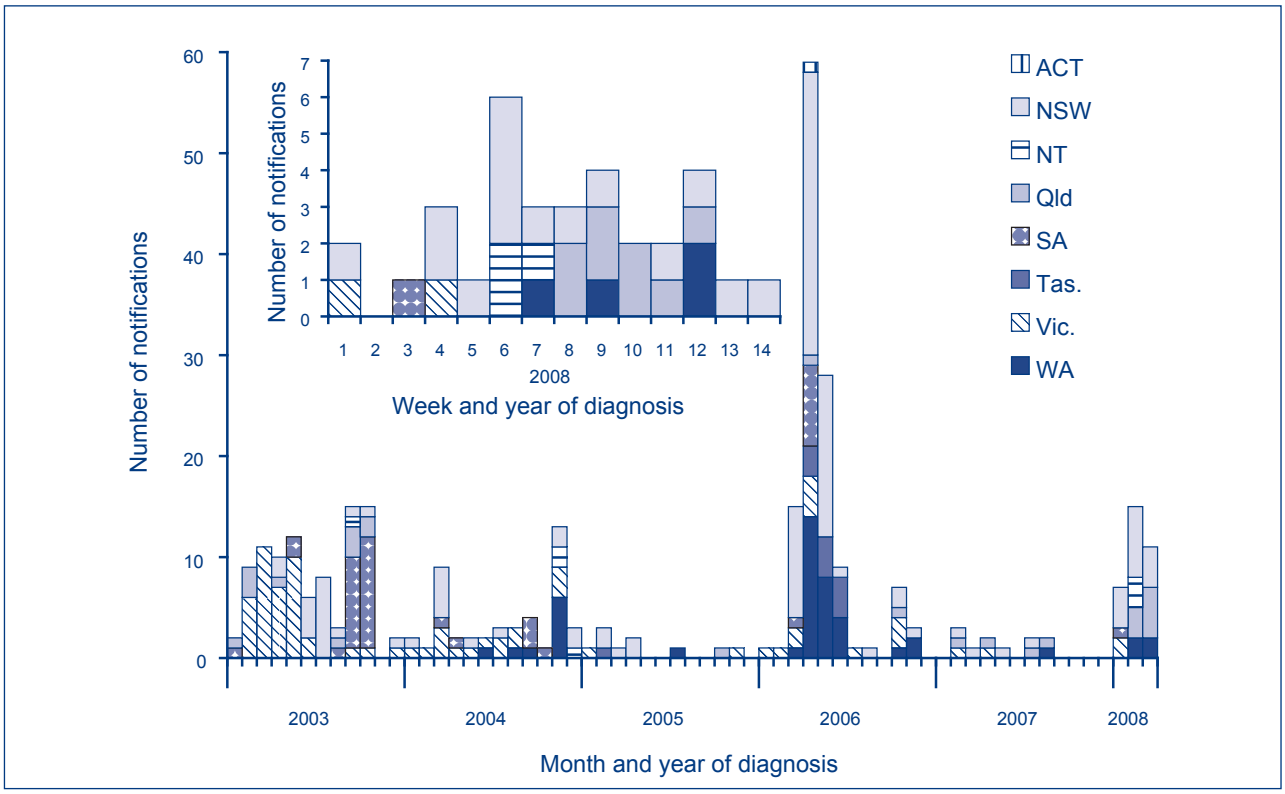
Eight of the cases (24%) were reported as acquiring measles outside Australia from countries including Thailand, India, China and the United Arab Emirates. Four secondary cases (12%) were epidemiologically linked to these imported cases; 2 of these reported from the Northern Territory were linked to an overseas acquired case through travel on the same flight from China to Melbourne via Darwin; a third case was exposed to the same overseas acquired case in the Darwin airport terminal.

An outbreak of measles in the Cairns area of Queensland was identified in late March. Eight cases were reported in two parallel chains of transmission, consisting of 4 cases each. All cases were unvaccinated. There were no recent overseas travel histories or contact with overseas travellers reported.

The virus genotype associated with both the Queensland outbreak and the Darwin cases was confirmed as H1 (personal communication, Dr Mike Catton, Victorian Infectious Diseases Reference Laboratory, CDNA Teleconference, 14 May 2008). The genetic material from these cases shared identical sequence within the nucleoprotein and was identical in this region to measles viruses from cases in New York in 2006 and Shanghai in 2003.

The only other genotyping data reported in 2008 was a D4 from a case in January who had returned from India. There is evidence that Australia currently has no endemic measles genotypes in circulation, with interruption of the endemic transmission of measles having occurred in the 1990s.[2]

Figure 4. Epidemic curve of notifications of measles, Australia, 1 January 2003 to 31 March 2008, by week of diagnosis and state or territory



The current National Immunisation Program Schedule recommends two doses of the measles, mumps and rubella vaccine (MMR) at 12 months of age and at 4 years of age, unless there is a contraindication. High-level vaccination coverage is imperative to enable measles elimination, requiring rates for each new birth cohort of >95% for a single dose and >90% for 2 doses.[3] Coverage data from March 2007 indicates that 93.7% of Australian children for the birth cohort 1 October to 31 December 2004 were immunised for MMR at age 2 years. The proportion of children immunised at 6 years of age for MMR was 88.9%.[4]

Other bacterial infections

Meningococcal disease

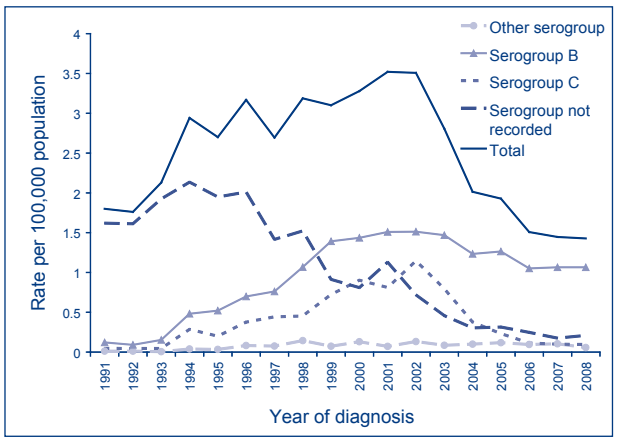
Meningococcal disease is caused by bacterial infection with *Neisseria meningitidis*, which is a gram-negative diplococcus carried and transmitted by humans.[5] The disease is characterised by the sudden onset of fever, intense headache, nausea, stiff neck and photophobia.[6] There are 13 known serogroups and globally serogroups A, B, C, W-135 and Y are the serogroups most commonly associated with disease. In January 2003, the National Meningococcal C Vaccination Program commenced to provide meningococcal C vaccine, to all Australian children aged 1 to 19 years, and the vaccine was also added to the National Immunisation Program schedule.[5]

Between 1 January and 31 March 2008 there were 44 notifications of meningococcal infection reported nationally, half the number of cases reported in the previous quarter (n=78) and similar to the number reported for the corresponding period in 2007 (n=45). Cases were aged between less than 1 year and 77 years, with 32% of cases aged 0–4 years (n=14), 23% aged 5–14 years (n=10) and 18% aged 15–24 years (n=8).

Serogroup data were available on 35 (80%) of the notified cases in the quarter. Twenty-nine (66%) were serogroup B, 5 (11%) were serogroup C, 1 case (2%) was serogroup Y, and in the remaining cases the serogroup was either not typed or no data were provided (n=9, 21%).

Since the introduction of the National Meningococcal C Vaccination Program in 2003, there has been a decrease in the overall number of cases of meningococcal disease, and a steady decline in the number of serogroup C infections among the 0–18 years age group. As shown in Figure 5, although there has been an increase in the proportion of meningococcal infections due to serogroup B, the overall rates of meningococcal infections continue to decline.

Figure 5. Meningococcal disease notifications, Australia, 1 January 1991 to 31 March 2008, by serogroup (annualised rate per 100,000 population)



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Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 38,405 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2008 (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 (NEC)	All jurisdictions
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
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
STEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	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

Table 1. Reporting of notifiable diseases by jurisdiction, *continued*

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)*	All jurisdictions except South Australia
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 zoster (chickenpox)	All jurisdictions except New South Wales and Victoria
Varicella zoster (shingles)	All jurisdictions except New South Wales and Victoria
Varicella zoster (unspecified)	All jurisdictions except New South Wales and Victoria
Vectorborne diseases	
Arbovirus infection (NEC)†	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularemia	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., Arbovirus (NEC) replaced Flavivirus (NEC from 2008

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 January to 31 March 2008, by date of onset*

Disease	State or territory								Total 1st quarter 2008†	Total 4th quarter 2007	Total 1st quarter 2007	Last 5 years mean 1st quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0.0
Hepatitis B (incident)	1	5	1	14	7	2	15	6	51	69	81	78.0	51	78.0	0.7
Hepatitis B (unspecified)	14	623	19	194	83	19	445	166	1,563	1,639	1,823	1,571.4	1,563	1,571.4	1.0
Hepatitis C (incident)	2	3	4	NN	13	3	25	18	68	72	96	108.4	68	108.4	0.6
Hepatitis C (unspecified)	43	1,236	56	690	121	74	549	355	3,124	2,876	3,227	3,289.6	3,122	3,289.6	1.0
Hepatitis D	0	1	0	4	0	0	5	1	11	7	10	6.8	11	6.8	1.6
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	1	0.6	0	0.6	0.0
Campylobacteriosis§	118	NN	71	1,490	481	153	1,773	496	4,582	4,699	4,865	4,309.6	4,582	4,309.6	1.1
Cryptosporidiosis	7	209	37	240	26	0	158	71	748	809	1,136	992.2	748	992.2	0.8
Haemolytic uraemic syndrome	0	2	0	3	0	0	3	0	8	7	7	4.8	8	4.8	1.7
Hepatitis A	1	22	1	25	11	0	17	7	84	35	47	92.6	84	92.6	0.9
Hepatitis E	0	4	0	1	0	0	6	2	13	3	6	8.8	13	8.8	1.5
Listeriosis	0	16	0	5	1	1	4	1	28	16	16	18.2	28	18.2	1.5
Salmonellosis	34	692	144	755	194	127	587	272	2,805	2,199	3,500	2,952.8	2,805	2,952.8	1.0
Shigellosis	0	22	65	28	50	2	16	55	238	162	144	173.8	238	173.8	1.4
STEC, VTEC‖	0	6	0	8	12	0	4	0	30	42	40	20.8	30	20.8	1.4
Typhoid	0	10	0	11	1	0	8	4	34	14	39	26.2	34	26.2	1.3
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	1	0.8	0	0.8	0.0
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Severe acute respiratory syndrome	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
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 January to 31 March 2008, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2008†	Total 4th quarter 2007	Total 1st quarter 2007	Last 5 years mean 1st quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	256	3,227	506	3,929	883	383	2,885	2,052	14,121	12,892	13,484	10,541.2	14,121	10,541.2	1.3
Donovanosis	0	0	0	0	0	0	0	0	0	0	2	3.2	0	3.2	0.0
Gonococcal infection	7	306	379	442	111	10	212	417	1,884	1,809	2,024	2,016.6	1,884	2,016.6	0.9
Syphilis (all)	8	268	56	87	11	7	200	61	698	761	730	637.8	698	637.8	1.1
Syphilis < two years duration	1	66	28	53	0	2	97	33	280	297	302	197.3	280	197.3	1.4
Syphilis >two years or unspecified duration	7	202	28	34	11	5	103	28	418	464	428	440.5	418	440.5	1.0
Syphilis - congenital	0	1	0	0	0	0	0	0	1	2	3	3.4	1	3.4	0.3
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	1	1	0	0	0	3	0	5	6	2	3.4	5	3.4	1.5
Influenza (laboratory confirmed)	16	78	30	203	NDP	4	27	56	414	715	344	223.2	414	223.2	1.9
Measles	0	15	3	8	1	0	2	4	33	0	4	12.0	33	12.0	2.8
Mumps	0	46	11	13	9	0	6	53	138	296	46	36.8	138	36.8	3.8
Pertussis	17	665	35	322	77	3	258	32	1,409	1,475	1,039	1,550.6	1,409	1,550.6	0.9
Pneumococcal disease (invasive)	2	50	15	43	6	5	44	26	191	296	193	270.6	191	270.6	0.7
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	0	0	0	1	0	0	3	4	7	9	12.4	4	12.4	0.3
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0.0
Tetanus	0	1	0	0	0	0	1	1	3	1	0	1.0	3	1.0	3.0
Varicella zoster (chickenpox)	3	NN	26	41	93	4	NN	49	216	488	358	289.0	216	289.0	0.8
Varicella zoster (shingles)	1	NN	20	115	148	24	NN	124	432	360	472	324.0	432	324.0	1.4
Varicella zoster (unspecified)	20	NN	1	734	116	15	NN	180	1,066	1,202	1,069	954.5	1,066	954.5	1.2
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	5	0	0	2	0	7	3	10	17.4	7	17.4	0.4
Barmah Forest virus infection	5	226	18	505	8	0	19	43	824	373	436	428.0	824	428.0	1.9
Dengue virus infection	1	37	14	51	9	0	2	37	151	75	93	165.4	151	165.4	0.9
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0.0
Kunjin virus infection	0	0	0	0	0	0	0	0	0	1	0	4.6	0	4.6	0.0
Malaria	4	27	5	41	5	1	19	22	124	139	153	202.8	124	202.8	0.6
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.6	0	0.6	0.0
Ross River virus infection	11	607	92	1,560	44	53	131	258	2,756	1,136	1,113	1,699.6	2,756	1,699.6	1.6

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 January to 31 March 2008, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2008†	Total 4th quarter 2007	Total 1st quarter 2007	Last 5 years mean 1st quarter	Year to date 2008	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	1	0.4	0	0.4	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	1	0	6	0	0	0	0	7	6	15	10.4	7	10.4	0.7
Leptospirosis	0	3	0	38	0	0	0	0	41	18	50	51.0	41	51.0	0.8
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	7	0	0	0	0	11	2	20	27	28	42.0	20	42.0	0.5
Q fever	0	44	0	49	6	0	3	0	102	111	115	124.4	102	124.4	0.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Other bacterial infections															
Legionellosis	0	25	0	7	5	0	11	10	58	97	75	86.8	58	86.8	0.7
Leprosy	0	1	0	2	0	0	0	0	3	2	5	3.2	3	3.2	0.9
Meningococcal infection**	0	10	2	20	2	0	6	4	44	78	45	75.4	44	75.4	0.6
Tuberculosis	2	97	6	28	9	2	102	19	265	370	273	259.0	271	259.0	1.0
Total	573	8,594	1,618	11,717	2,544	892	7,559	4,908	38,405	35,396	37,230	32,747	38,405	32,747	1.2

* Date of onset = the true onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified were analysed by the date of notification.

† Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. 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* (STEC/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.

NDP No data provided

Table 3. Notification rates of diseases, 1 January to 31 March 2008, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (incident)	1.2	0.3	1.9	1.3	1.8	1.6	1.2	1.1	1.0
Hepatitis B (unspecified)	16.5	36.2	35.4	18.6	21.0	15.4	34.2	31.5	29.7
Hepatitis C (incident)	2.4	0.2	7.4	NN	3.3	2.4	1.9	3.4	1.3
Hepatitis C (unspecified)	50.6	71.8	104.2	66.0	30.5	60.0	42.2	67.4	59.5
Hepatitis D	0.0	0.1	0.0	0.4	0.0	0.0	0.4	0.2	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	138.9	NN	132.1	142.5	121.4	124.1	136.2	94.2	87.2
Cryptosporidiosis	8.2	12.1	68.8	23.0	6.6	0.0	12.1	13.5	14.2
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.3	0.0	0.0	0.2	0.0	0.2
Hepatitis A	1.2	1.3	1.9	2.4	2.8	0.0	1.3	1.3	1.6
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.5	0.4	0.2
Listeriosis	0.0	0.9	0.0	0.5	0.3	0.8	0.3	0.2	0.5
Salmonellosis	40.0	40.2	267.9	72.2	49.0	103.0	45.1	51.7	53.4
Shigellosis	0.0	1.3	120.9	2.7	12.6	1.6	1.2	10.4	4.5
STEC, VTEC‡	0.0	0.3	0.0	0.8	3.0	0.0	0.3	0.0	0.6
Typhoid	0.0	0.6	0.0	1.1	0.3	0.0	0.6	0.8	0.6
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Highly pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	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
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§	301.3	187.4	941.5	375.8	222.9	310.5	221.7	389.8	268.8
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	8.2	17.8	705.2	42.3	28.0	8.1	16.3	79.2	35.9
Syphilis (all)	9.4	15.6	104.2	8.3	2.8	5.7	15.4	11.6	13.3
Syphilis <2 years duration	1.2	3.8	52.1	5.1	0.0	1.6	7.5	6.3	5.3
Syphilis >2 years or unspecified duration	8.2	11.7	52.1	3.3	2.8	4.1	7.9	5.3	8.0
Syphilis - congenital	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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	1.9	0.0	0.0	0.0	0.2	0.0	0.1
Influenza (laboratory confirmed)	18.8	4.5	55.8	19.4	NDP	3.2	2.1	10.6	7.9
Measles	0.0	0.9	5.6	0.8	0.3	0.0	0.2	0.8	0.6
Mumps	0.0	2.7	20.5	1.2	2.3	0.0	0.5	10.1	2.6
Pertussis	20.0	38.6	65.1	30.8	19.4	2.4	19.8	6.1	26.8
Pneumococcal disease (invasive)	2.4	2.9	27.9	4.1	1.5	4.1	3.4	4.9	3.6
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 January to 31 March 2008, 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	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.6	0.1
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.2	0.1
Varicella zoster (chickenpox)	3.5	NN	48.4	3.9	23.5	3.2	NN	9.3	4.1
Varicella zoster (shingles)	1.2	NN	37.2	11.0	37.4	19.5	NN	23.6	8.2
Varicella zoster (unspecified)	23.5	NN	1.9	70.2	29.3	12.2	NN	34.2	20.3
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.5	0.0	0.0	0.2	0.0	0.1
Barmah Forest virus infection	5.9	13.1	33.5	48.3	2.0	0.0	1.5	8.2	15.7
Dengue virus infection	1.2	2.1	26.0	4.9	2.3	0.0	0.2	7.0	2.9
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	4.7	1.6	9.3	3.9	1.3	0.8	1.5	4.2	2.4
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	12.9	35.2	171.2	149.2	11.1	43.0	10.1	49.0	52.5
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	0.1	0.0	0.6	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	0.0	3.6	0.0	0.0	0.0	0.0	0.8
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.4	0.0	0.0	0.0	0.0	0.8	0.4	0.4
Q fever	0.0	2.6	0.0	4.7	1.5	0.0	0.2	0.0	1.9
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.0	1.5	0.0	0.7	1.3	0.0	0.8	1.9	1.1
Leprosy	0.0	0.1	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Meningococcal infection‖	0.0	0.6	3.7	1.9	0.5	0.0	0.5	0.8	0.8
Tuberculosis	2.4	5.6	11.2	2.7	2.3	1.6	7.8	3.6	5.0

* 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* (STEC/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.

NDP No data provided

Laboratory Serology and Virology Reporting Scheme

There were 7,637 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2008 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 January to 31 March 2008, and total reports for the year†

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	7	1	–	2	–	3	–	13	6	13	6
Mumps virus	–	–	2	3	6	–	5	2	18	9	18	9
Rubella virus	–	–	–	4	–	–	1	–	5	8	5	8
Hepatitis viruses												
Hepatitis A virus	–	3	1	10	6	–	–	1	21	10	21	10
Hepatitis D virus	–	–	–	1	5	–	1	–	7	7	7	7
Hepatitis E virus	–	1	–	–	–	–	–	–	1	1	1	1
Arboviruses												
Barmah Forest virus	–	14	–	223	11	1	1	–	250	139	250	139
Flavivirus (unspecified)	–	–	–	29	–	–	1	–	30	25	30	25
Ross River virus	–	31	20	675	42	4	2	18	792	301	792	301
Adenoviruses												
Adenovirus not typed/pending	–	73	–	70	182	2	17	1	345	180	345	180
Herpes viruses												
Cytomegalovirus	–	44	1	175	142	–	3	–	365	287	365	287
Epstein-Barr virus	–	4	40	305	138	2	13	149	651	677	651	677
Varicella-zoster virus	2	132	–	442	153	–	27	–	756	697	756	697
Other DNA viruses												
Parvovirus	–	4	1	63	2	–	7	–	77	92	77	92
Picornavirus family												
Enterovirus not typed/pending	2	40	–	14	23	–	2	–	81	27	81	27
Picornavirus not typed	–	–	–	–	–	1	–	–	1	–	1	–
Rhinovirus (all types)	1	28	–	–	–	–	1	–	30	65	30	65
Coxsackievirus A9	–	1	–	–	–	–	–	–	1	–	1	–
Echovirus type 7	–	1	–	–	–	–	–	–	1	–	1	–
Ortho/paramyxoviruses												
Influenza A virus	–	2	2	26	16	–	1	–	47	52	47	52
Influenza B virus	–	1	–	5	13	–	2	–	21	5	21	5
Parainfluenza virus type 1	–	40	–	14	1	–	5	–	60	4	60	4
Parainfluenza virus type 2	–	2	–	6	–	–	2	–	10	5	10	5
Parainfluenza virus type 3	–	1	–	1	2	–	3	–	7	33	7	33
Respiratory syncytial virus	–	94	–	94	10	–	5	–	203	171	203	171
Other RNA viruses												
HTLV-1	–	–	–	–	3	–	–	–	3	3	3	3
Norwalk agent	–	16	–	–	–	–	–	–	16	140	16	140
Rotavirus	–	29	–	–	63	3	13	–	108	45	108	45

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 January to 31 March 2008, and total reports for the year,† *continued*

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia psittaci	–	–	–	1	–	1	9	–	11	21	11	21
Chlamydia trachomatis - not typed	5	329	2	1,465	524	9	11	2	2,347	2,164	2,347	2,164
Chlamydia species	–	–	–	–	–	–	2	–	2	–	2	–
Coxiella burnetii (Q fever)	3	6	–	42	13	–	11	–	75	28	75	28
Mycoplasma pneumoniae	–	10	10	66	37	5	39	9	176	313	176	313
Mycoplasma hominis	–	2	–	–	–	–	–	–	2	3	2	3
Orientia tsutsuganushi	–	–	1	–	2	–	–	–	3	3	3	3
Rickettsia - spotted fever group	–	4	–	2	1	1	3	–	11	3	11	3
Streptococcus group A	–	7	40	193	–	–	31	–	271	232	271	232
Bordetella pertussis	–	10	1	110	63	–	11	–	195	180	195	180
Brucella species	–	–	–	8	–	–	–	–	8	1	8	1
Legionella longbeachae	–	–	–	–	1	1	–	–	2	3	2	3
Legionella pneumophila	–	4	–	–	–	–	1	–	5	4	5	4
Yersinia enterocolitica	–	4	–	1	–	–	–	–	5	1	5	1
Cryptococcus species	–	2	–	3	2	–	–	–	7	9	7	9
Leptospira species	–	–	–	29	1	–	–	–	30	23	30	23
Treponema pallidum	–	61	11	264	221	–	2	1	560	535	560	535
Toxoplasma gondii	–	–	–	–	–	1	–	–	1	8	1	8
Echinococcus granulosus	–	–	–	–	6	–	–	–	6	3	6	3
Total	13	1,007	133	4,344	1,691	31	235	183	7,637	6,523	7,637	6,523

* 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 January to 31 March 2008*

State or territory	Laboratory	January 2008	February 2008	March 2008	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	160	115	105	380
	New Children's Hospital, Westmead	48	55	58	161
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	16	19	33	68
	South West Area Pathology Service, Liverpool	31	39	59	129
Queensland	Queensland Medical Laboratory, West End	1,409	1,745	1,559	4,713
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	609	596	484	1,689
Tasmania	Northern Tasmanian Pathology Service, Launceston	10	7	12	29
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	16	20	9	45
	Monash Medical Centre, Melbourne	19	12	13	44
	Royal Children's Hospital, Melbourne	17	20	16	53
	Victorian Infectious Diseases Reference Laboratory, Fairfield	58	31	–	89
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	79	77	81	237
Total		2,472	2,736	2,429	7,637

* The complete list of laboratories reporting for the 12 months, January to December 2008, 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 2008, 4 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 2008;32:134–135.

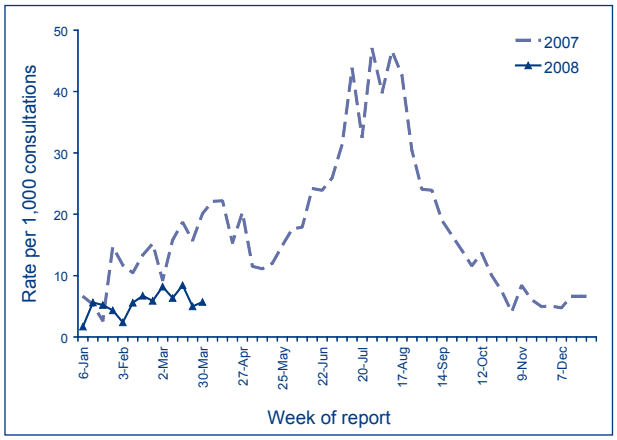
Data on influenza-like illness, gastroenteritis, chickenpox and shingles from 1 January to 31 March 2008 compared with 2007, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 and 4, respectively.

Reporting period 1 January to 31 March 2008

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 94 general practitioners contributed data to ASPREN in the first quarter of 2008. Each week an average of 78 general practitioners provided information to ASPREN at an average of 7,013 (range 5,307 to 8,257) consultations per week.

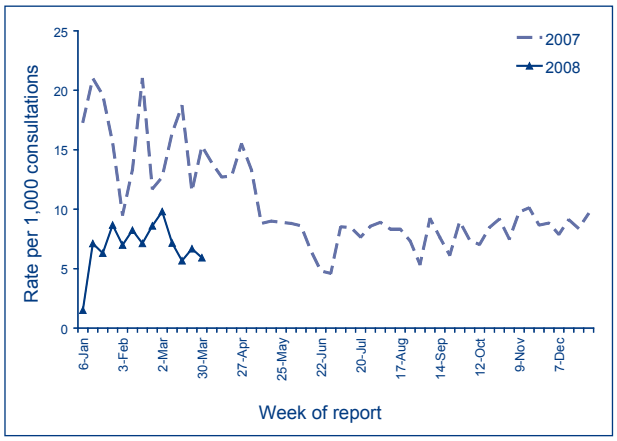
In the first quarter of 2008, influenza like illness (ILI) rates remained low from 1.7 to 8 cases per 1.000 consultations. For the same reporting period in 2007 reported rates were higher at 2 to 20 cases per 1,000 consultations (Figure 1).

Figure 1. Consultation rates for influenza-like illness, ASPREN, 2007 to 31 March 2008, by week of report



Reports of gastroenteritis from 1 January to 31 March 2008 were lower compared with the same period in 2007 (Figure 2). During this reporting period, consultation rates for gastroenteritis ranged from 2 to 10 cases per 1,000.

Figure 2. Consultation rates for gastroenteritis, ASPREN, 2007 to 31 March 2008, by week of report



Reports of varicella infections were reported at a lower rate for the first quarter of 2008 compared with the same period in 2007. From 1 January to 31 March 2008, recorded rates for chickenpox were between 0 to 0.7 case per 1,000 consultations (Figure 3).

In the first quarter of 2008, rates for shingles fluctuated between less than 1 to 1.5 cases per 1,000 consultations (Figure 4).

Figure 3. Consultation rates for chickenpox, ASPREN, 2007 to 31 March 2008, by week of report

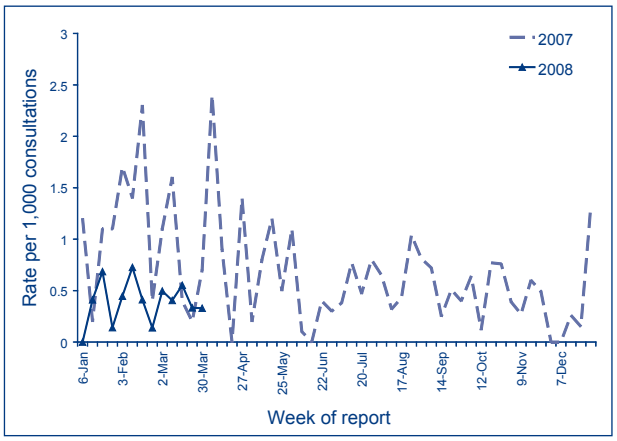
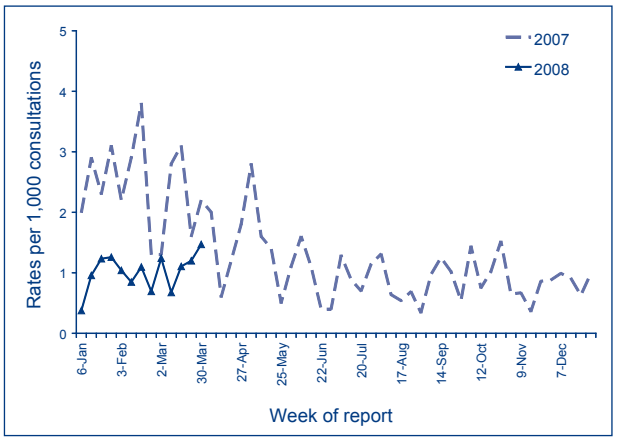


Figure 4. Consultation rates for shingles, ASPREN, 2007 to 31 March 2008, by week of report



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 October and 31 December 2006, at 24 months of age for the cohort born between 1 October and 31 December 2005, and at 6 years of age for the cohort born between 1 October and 31 December 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 *2008;32:134–135 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 decreased marginally by 0.2 percentage points to 91.3% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months or by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia also decreased marginally by 0.2 percentage points to 92.8% (Table 2). There were also no important changes in coverage for any individual vaccines due at 24 months or by jurisdiction.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2006; assessment date 31 March 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,114	23,355	906	14,103	4,600	1,589	17,310	7,036	70,013
Diphtheria, tetanus, pertussis (%)	94.1	92.0	91.0	92.0	91.1	92.9	92.8	89.7	91.9
Poliomyelitis (%)	94.2	91.9	91.0	92.0	91.0	92.8	92.8	89.7	91.9
Haemophilus influenzae type b (%)	96.3	94.9	94.4	94.0	93.9	95.2	95.0	93.5	94.5
Hepatitis B (%)	96.3	94.9	95.3	93.8	93.9	95.2	94.8	93.3	94.4
Fully immunised (%)	93.9	91.6	90.1	91.2	90.4	92.7	91.9	88.9	91.3
Change in fully immunised since last quarter (%)	+1.1	-0.1	-0.6	-0.2	-1.2	-0.8	-0.3	+0.1	-0.2

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2005;* assessment date 31 March 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,156	22,580	868	13,916	4,357	1,576	16,715	6,697	67,865
Diphtheria, tetanus, pertussis (%)	95.6	94.8	95.9	94.6	95.0	95.8	95.7	94.4	95.0
Poliomyelitis (%)	95.6	94.8	95.9	94.6	95.0	95.8	95.7	94.4	95.0
Haemophilus influenzae type b (%)	95.6	95.1	94.2	93.7	93.6	95.7	94.5	94.1	94.5
Measles, mumps, rubella (%)	95.1	93.9	95.7	94.1	93.9	94.7	94.8	93.4	94.2
Hepatitis B (%)	96.8	95.5	96.9	95.4	95.5	96.4	96.3	95.3	95.7
Fully immunised (%)	93.9	92.6	93.9	92.5	92.7	94.1	93.6	91.6	92.8
Change in fully immunised since last quarter (%)	+0.1	-0.3	-0.2	+0.4	-0.2	-1.6	-0.5	+0.2	-0.1

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2007;31:252.

Immunisation coverage for children 'fully immunised' at 6 years of age for Australia decreased by 0.6 percentage points from the previous quarter to 88.2% (Table 3). For 'fully immunised' and all individual vaccines, there were important decreases of greater than 1 percentage point in South Australia, Tasmania, Victoria and Western Australia.

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.

Figure 5. Trends in vaccination coverage, Australia, 1997 to 31 December 2007, by age cohorts

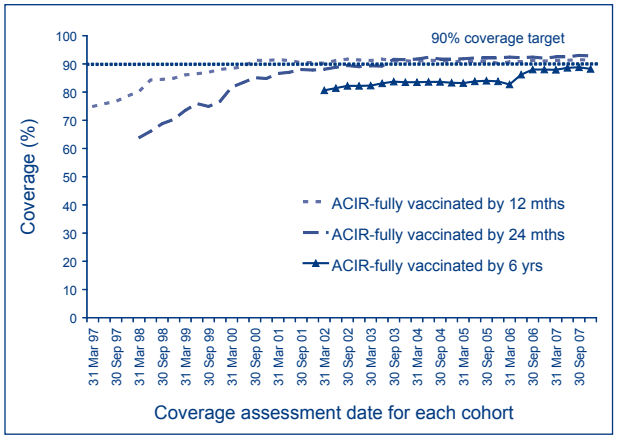


Table 3. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2001;* assessment date 31 March 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,061	22,048	872	13,095	4,352	1,483	16,088	6,552	65,551
Diphtheria, tetanus, pertussis (%)	91.5	89.2	88.8	89.2	86.6	87.4	91.0	84.9	89.0
Poliomyelitis (%)	91.3	88.9	88.8	89.0	86.3	87.1	90.9	84.6	88.8
Measles, mumps, rubella (%)	91.0	88.9	89.3	89.0	86.2	87.0	90.8	84.7	88.8
Fully immunised (%)	90.6	88.3	88.3	88.4	85.7	86.4	90.3	83.9	88.2
Change in fully immunised since last quarter (%)	+1.8	-0.8	-0.1	+0.8	-1.9	-1.8	-1.0	-1.3	-0.6

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2003;27:302.

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 5* Salmonella *typing 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 January to 31 March 2008 are included in Tables 4 and 5. Data include cases reported and entered by 29 April 2008. 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 *2008;32:136.*

There were 2,258 reports to NEPSS of human *Salmonella* infection in the first quarter of 2008, approximately 25% more than in the fourth quarter of 2007. Limited first quarter data from Western Australia were available at the time of preparing this report. We anticipate these data will be included in NEPSS in time for the next quarterly report. Taking into account the limited Western Australia data, the overall count of cases for the remainder of Australia was similar to the recent historical mean incidence of salmonellosis at this time of each year. However, the incidence in 3 states (South Australia, Victoria and Tasmania) was at least 20% higher than the recent historical mean for the period.

During the first quarter of 2008, the 25 most common *Salmonella* types in Australia accounted for 1,478 cases, 65% of all reported human *Salmonella* infections. Eighteen of the 25 most common *Salmonella* infections in the first quarter of 2008 were also among those most commonly reported in the preceding quarter.

The most prominent feature of the current data is an increase in *S.* Typhimurium phage type 135, particularly in Victoria and Tasmania. *S.* Typhimurium phage types 44, 170 and 8 were also increased in Victoria, while *S.* Typhimurium phage type 126 was increased in New South Wales and Victoria. *S.* Montevideo and *S.* Singapore were moderately increased in New South Wales. An increase in *S.* Infantis involved New South Wales, South Australia and Victoria.

S. Virchow phage type 8 has historically been largely confined to Queensland. The recent Australia-wide count of *S.* Virchow phage type 8 is less than the historical average. However, during the first quarter of 2008 there was an increase in reports of *S.* Virchow phage type 8 outside its usual range, involving New South Wales, South Australia, the Northern Territory, Tasmania, Victoria and the Australian Capital Territory.

Table 4. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2008, as reported to 29 April 2008

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA*	Australia
Total all *Salmonella* for quarter	31	573	109	640	174	122	592	17	2,258
Total contributing *Salmonella* types	20	115	45	118	56	14	118	10	218

* Limited first quarter data from Western Australia were available at the time of preparing this report.

Table 5. Top 25 *Salmonella* types identified in Australia, 1 January to 31 March 2008, by state or territory

National rank	*Salmonella* type	State or territory								Total 1st quarter 2008	Last 10 years mean 1st quarter	Year to date 2008	Year to date 2007
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA*				
1	*S.* Typhimurium PT 135	4	60	0	40	8	52	143	6	313	246	313	250
2	*S.* Typhimurium PT 9	3	20	0	7	20	3	57	2	112	199	112	336
3	*S.* Typhimurium PT 44	1	5	0	7	4	7	71	0	95	41	95	183
4	*S.* Typhimurium PT 170	3	40	0	9	0	0	40	0	92	105	92	124
5	*S.* Saintpaul	0	15	12	59	3	0	2	0	91	136	91	137
6	*S.* Virchow PT 8	2	24	7	36	12	5	3	0	89	113	89	87
7	*S.* Birkenhead	0	35	0	45	0	0	3	0	83	101	83	93
8	*S.* Infantis	4	30	3	3	13	0	13	0	66	50	66	60
9	*S.* Mississippi	0	2	0	6	0	43	7	0	58	47	58	76
10	*S.* Chester	1	7	3	26	8	0	3	0	48	70	48	71
11	*S.* Typhimurium PT 197	0	18	0	21	2	0	5	1	47	69	47	93
12	*S.* Typhimurium PT 126	0	25	0	3	3	0	14	0	45	30	45	9
13	*S.* Muenchen	0	10	1	19	2	0	3	0	35	54	35	58
14	*S.* Waycross	1	14	0	19	0	0	1	0	35	46	35	36
15	*S.* Singapore	0	16	0	9	2	0	5	0	32	21	32	24
16	*S.* Hvittingfoss	0	3	3	25	0	0	0	0	31	43	31	41
17	*S.* Montevideo	1	18	0	5	0	0	5	0	29	16	29	42
18	*S.* Aberdeen	1	2	0	21	0	0	1	0	25	47	25	50
19	*S.* Typhimurium PT 8	1	3	0	3	1	0	16	0	24	12	24	13
20	*S.* Weltevreden	0	4	7	11	1	0	0	0	23	16	23	26
21	*S.* Stanley	0	4	0	6	2	0	10	0	22	17	22	37
22	*S.* Typhimurium PT 29	0	4	0	3	11	0	4	0	22	11	22	49
23	*S.* Zanzibar	0	4	2	14	1	0	0	0	21	12	21	12
24	*S.* Potsdam	0	4	0	12	1	0	2	1	20	23	20	26
25	*S.* Reading	0	2	3	14	0	0	1	0	20	12	20	18

* Limited first quarter data from Western Australia were available at the time of preparing this report.

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.

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 *2008;32:135.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2008, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2008, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W-135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	08													0	0
	07			1	1					1	1			2	2
New South Wales	08			4	4	1	1	1	1					6	6
	07			12	12	3	1			1		2	2	17	17
Northern Territory	08					1	1							1	1
	07			1	1									1	1
Queensland	08			16	16	2	2							18	18
	07			11	11									11	11
South Australia	08			2	2									2	2
	07			1	1									1	1
Tasmania	08													0	0
	07									1	1			1	1
Victoria	08			4	4									4	4
	07			6	6									6	6
Western Australia	08			3	3							1	1	4	4
	07			3	3									3	3
Total	08			30	30	4	4	1	1			1	1	36	36
	07			35	35	3	3	0	0		1	2	2	41	41

OVERSEAS BRIEF

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.

Reporting period 1 January to 31 March 2008

Chikungunya

Indonesia

Outbreaks of chikungunya in Indonesia have continued throughout the first quarter of 2008, particularly in the city of Bandar Lampang, Lampung Province, on the south east end of the island of Sumatra.[1]

Chikungunya is difficult to distinguish from dengue fever without laboratory confirmation, so that some cases in the outbreak may have been due to dengue fever. Community health centres do not routinely report their cases to the health office, which may lead to widespread under-reporting.[1]

Sri Lanka

In February 2008, military officials reported an outbreak of chikungunya among troops in the northern war zone of Sri Lanka. Blood samples sent to the Medical Research Institute confirmed chikungunya in the majority of cases, however dengue fever infection may also be involved. Approximately 500 troops were treated at local hospitals in the Anuradhapura district. The outbreak was attributed to heavy rains and resultant floods leading to the proliferation of mosquito breeding sites.[2]

Dengue and dengue haemorrhagic fever

Global update

Dengue virus infection has become the most common arboviral disease of humans with more than 2.5 billion people living in areas where dengue virus infection can be locally acquired. Epidemics caused by all 4 virus serotypes have progressively increased in size and frequency over the last 25 years. Since 2005, dengue virus infection has become endemic in most tropical countries of the South Pacific, Asia, the Caribbean, the Americas, and Africa.

The presence of multiple dengue virus serotypes circulating simultaneously (hyperendemicity) in most tropical urban centres in the endemic regions is of particular concern. Hyperendemicity is associated with increased transmission rates and increased rates of dengue haemorrhagic fever (DHF). The incidence of DHF over the past 25 years has increased significantly in South East Asia, the South Pacific and the American tropics with major epidemics now occurring in many countries every 3 to 5 years. DHF is more commonly seen in children under the age of 15 years in Asia. In contrast, in the Americas and the Pacific, primary infection at a young age is not common. International travellers from non-endemic countries who have not had a primary infection are usually at low risk for DHF.[3]

New Caledonia

On 22 February 2008 New Caledonian health authorities declared a dengue virus infection epidemic. The declaration was made following an outbreak that initially involved the townships of greater Noumea but then extended to the north-eastern island of Lifou and the Isle of Pines, south of the capital Noumea. Up to 31 March 2008, 284 positive cases of dengue virus infection had been reported to the Department of Health.[4] Local transmission of serotype DEN 1 had been confirmed in the majority of cases. The last dengue virus infection epidemic in New Caledonia was in 2003 (also serotype DEN 1) with approximately 20 deaths.[5]

Tonga

Health officials in Tonga have reported more than 200 cases of dengue virus infection[6] in the first 2 months of 2008, including 3 deaths as of 19 March 2008.[7] The deaths included a 23-year-old and an infant who both died as a result of the more severe DHF.[6] An Auckland medical health officer reported on 2 April 2008 at least 23 cases of dengue virus infection imported from Tonga to New Zealand, confirming that this outbreak is continuing.[8]

South East Asia

A number of South East Asian countries including East Timor, Malaysia, the Philippines, Singapore, Sri Lanka and Thailand reported continuing outbreaks of dengue virus infection during the reporting period. While dengue virus infection is endemic in these countries and high case numbers are expected for this time of year (due to the onset of the rainy season) increased incidences were reported compared with 2007, particularly in Singapore and Thailand.[9]

Malaysia

The Malaysian Ministry of Health reported 9,889 cases of dengue virus infection including 26deaths in the first 3 months of 2008. While this is less than the 13,949 cases (including 34 deaths) reported for the same time for 2007, case numbers are continuing to rise. The majority of cases to date have occurred in the central Selangor State (262 cases) followed by the capital Kuala Lumpur and the southern State of Johor.[9]

Singapore

The Singapore Ministry of Health reported 1,228 dengue virus infection cases in the first quarter of 2008 (a 68% increase compared with the same period in 2007) but no increase in the number of DHF cases.[10]

Thailand

The Thai Public Health Minister reported 7,413 cases of dengue virus infection (including 9 deaths across 8 provinces) between 1 January and 31 March 2008, a 76% increase compared with the same period in 2007.[9]

Human immunodeficiency virus

Global update

The number of people globally who are living with human immunodeficiency virus (HIV) is increasing due to the continued accumulation of new infections in a growing population and longer survival times, but prevalence has remained steady. The estimated number of people living with HIV worldwide in 2007 was 33.2 million (30.6–36.1 million).[11]

South East Asia and Pacific Region

South East Asia continues to have the highest prevalence of HIV in the Asian region. Myanmar, Thailand and Cambodia all demonstrate evidence of decreasing prevalence in contrast to Indonesia and Vietnam where prevalence is increasing.[11]

Indonesia

The HIV epidemic in Indonesia now affects 32 of its 33 provinces and has expanded from localised populations of injecting drug users in Bali, Jakarta and West Java to their non-injecting partners, sex workers and their clients and prisoners. In Papua province the adult HIV prevalence was estimated as 2.4% (ranging from 3.2% in remote highlands and 2.9% in less-accessible lowland areas) and 3% among 15–24 year olds. Unprotected sex is the main mode of transmission in Papua.[11]

Papua New Guinea

Seventy per cent of the 75,000 people living with HIV in the Oceania region in 2007 were from Papua New Guinea (PNG) where the epidemic continues to expand. Unsafe heterosexual intercourse remains the main mode of transmission in PNG with the majority of infections occurring in rural areas.[11]

New Zealand

In New Zealand unsafe sex between men remains the predominate mode of HIV transmission. However, there has been an increase in diagnoses through unsafe heterosexual intercourse with the majority of these infections contracted in Asia and sub-Saharan Africa.[11]

Pacific nations (other)

While no other country or territory within Oceania has reported more than 300 HIV cases since the start of testing and reporting, risk factors (such as low uptake of condom use in the small island states of Fiji, Kiribati and Tonga) associated with HIV infection are prevalent across the region.[11]

Influenza (avian)

During the first quarter of 2008 the World Health Organization (WHO) has confirmed 28 cases of human H5N1,[12] 22 of which were fatal giving a case-fatality rate (CFR) of 78%. This is lower than the number of cases confirmed during the same period in 2007 (39 cases, CFR 61%).[13] These WHO-confirmed cases were from 4 countries; China (3 cases), Egypt (5 cases), Indonesia (15 cases), and Vietnam (5 cases).[13] There was no evidence of human-to-human transmission of avian influenza during the reporting period.

In April 2008 the WHO released a final report on test results for the family cluster of H5N1 cases in Peshawar, Pakistan in late 2007.[14] In addition to the 1 human H5N1 case confirmed by the WHO in December 2007, 2 additional cases (1 fatal) were confirmed by serological testing. The report stated that the results support the epidemiological findings that limited human-to-human transmission was likely among some of the family members. The report also stated that there was a further probable human case, but that no sample was available for testing.

Influenza (seasonal)

During the 2007–2008 Northern Hemisphere influenza season in Europe,[15] Canada[16] and the United States of America (USA)[17] there was a mismatch between the majority of influenza A (H3) and influenza B virus strains typed from respiratory specimens and those recommended by the WHO for inclusion in the Northern Hemisphere 2007–2008 influenza vaccine. The strain of influenza A (H1) virus typed from respiratory specimens and that recommended by the WHO for inclusion in the 2007–2008 influenza vaccine were more closely matched.

The composition of the WHO recommended influenza vaccine for the upcoming 2008 Southern Hemisphere influenza season includes those virus strains that made up the majority of strains circulating in the 2007–2008 European and USA influenza seasons. The Australian Influenza Vaccine Committee has agreed to adopt the WHO recommendations for the composition of the vaccine for the 2008 season (influenza A: (H1N1): an A/Solomon Islands/3/2006 (H1N1)-like strain, influenza A: (H3N2): an A/Brisbane/10/2007 (H3N2)-like strain, and influenza B: B/Florida/4/2006-like strain).[18]

Following similar results during the 2007 Southern Hemisphere season, a significant proportion of influenza A (H1N1) virus isolates from the 2007–2008 Northern Hemisphere influenza season were resistant to oseltamivir. In the USA 10.2% of influenza A (H1N1) viruses tested have been found to be resistant to oseltamivir,[19] while in Europe influenza A (H1N1) viruses resistant to oseltamivir have been found in 19 countries with an overall prevalence of 23%[20] and in Canada[16] 20.3% of the H1N1 isolates tested were resistant to oseltamivir.

Measles

Despite the success of the Measles Initiative, particularly in Africa where measles deaths have decreased by more than 91% between 2000 and 2006, large outbreaks continue to occur across the world.[21] During the first 3 months of 2008 many countries reported new or continuing outbreaks of measles with several of these linked via imported cases to outbreaks in other countries. Viral genotyping is an increasingly important measles surveillance tool in non-endemic countries. Countries where measles remains endemic tend to have one predominant genotype or a small number of genotypes and so genotyping can facilitate the identification of imported measles virus infections.

European Region

Despite an overall decrease of 53% in measles cases (from 31 countries that report to EUVAC.NET) in 2007 compared with 2006, concern remains about the high incidence of measles reported in some countries. The World Health Organization Regional Office for Europe reported that in 2007 the majority (60%) of measles cases in the WHO European Region occurred in Western European countries. The highest incidence rates in Western Europe were in Switzerland with 14.06 cases per 100,000 population, followed by the United Kingdom (UK) with 1.64 cases per 100,000 population. The majority of all measles cases (87%) across the European Region were unvaccinated.[21]

Switzerland

An outbreak of measles in Switzerland, which began in November 2006, is continuing with 2,250 cases reported between November 2006 and 14 April 2008.[22] It is the largest outbreak registered in the country since measles notification began in Switzerland in 1999 and could be attributed to low vaccination coverage rates, 86% for first dose and 70% for second dose.[22] Cases have been predominantly among school aged children with 98% of cases unvaccinated or only partially vaccinated. Measles virus genotype D5 has been continuously transmitted within Switzerland over at least the past 15 months and has been linked to outbreaks in neighbouring countries and the USA.[23]

United States of America

In 2000, the WHO declared measles eliminated in the USA with successful interruption of endemic transmission at this time. Measles case numbers have declined to fewer than 150 cases annually since 1997. However, case numbers so far this year (up to 25 April 2008) are the highest for any year since 2001. The US CDC reported 61 confirmed cases of measles attributed to outbreaks, which began within this reporting period with 48 (79%) of these linked to imported cases as follows: San Diego, California (11 cases between 25 January and 16 February 2008, ex Switzerland, genotype D5); Pima County, Arizona (15 cases between 13 February and 23 April 2008, ex Switzerland, genotype D5); Honolulu, Hawaii (3 cases spread from San Diego and an additional 2 cases between 5 and 25 February, ex Italy); Fairfax, Virginia (1 case in February, ex India); New York City (14 cases between 7 February and 20 April, ex Israel and Belgium, genotype D4) and Milwaukee County, Wisconsin (4 cases between 19 March and 9 April, likely ex China, genotype H1).[24,25]

Nipah virus

The Institute of Epidemiology Disease Control and Research in Dhaka, Bangladesh reported an outbreak of Nipah virus in the Manikgonj and Rajbari districts of west-central Bangladesh with 8 suspected cases (all of them fatal) in February 2008. Two of the cases (siblings) tested positive for Nipah antibody. The index case became sick on 20 February 2008 and died on 29 February 2008. All cases in the Rajbari district were clustered in time and place (all from the same village). Seven of the 8 cases from this village had consumed date palm juice 10 to 15 days prior to becoming ill, as had the 3 cases (all from the same family) from Bishnupur village in Manikgonj district.[26]

Nipah outbreaks have occurred repeatedly in Bangladesh since 2001. The outbreaks occur seasonally between January and May and are associated with the harvesting and consumption of date palm juice during this time. Fruit bats are the animal reservoir for Nipah virus in Bangladesh with date palm juice a plausible agent for its transmission.[26]

Plague

Global trends

While the number of human cases of plague remain steady in Asia, there has been an increase in both the proportion of global cases of plague that are reported from Africa and the number of African countries that are reporting cases over the past 20 years with 90% of all cases over the last 5 years reported from Madagascar, Tanzania, Malawi, Uganda and the Democratic Republic of Congo.[27] In the last 15 years 45% of all human cases of plague in Africa have been reported from Madagascar.[28]

Poliomyelitis

Between 1 January and 2 April 2008, the Global Polio Eradication Initiative reported a total of 256 cases of wild poliovirus infection (with confirmations and dates of onset in 2008) from the endemic countries of Afghanistan (4), India (165), Nigeria (84) and Pakistan (3). In addition, 1 case of wild poliovirus was reported during this time from the re-infected country of the Democratic Republic of Congo and 2 from Angola. Chad and Angola are the only 2 re-infected countries in which the transmission of the originally imported poliovirus has never been interrupted and from where the virus has spread internationally. Somalia was declared polio-free on 25 March 2008 with no cases reported in the previous 12 months.[29]

India

Recent control efforts in India have focussed on the interruption of wild poliovirus type 1 (WPV1) transmission and the maintenance of high levels of population immunity against WPV1 in areas free of this serotype. This strategy appears to be successful with only 2 of the 165 cases reported in India with onset in 2008 being WPV1. There have been no WPV1 cases reported from the previously highly endemic area of Uttar Pradesh State since 10 November 2007. Low levels of WPV1 transmission are only likely to continue in remote areas of Bihar where access is restricted. An outbreak of WPV3 in mid-2007 is responsible for the higher numbers of this serotype in 2008, but this is being addressed via the implementation of 2 rounds of monovalent OPV type 3 in 2008.[29]

Nigeria

A significant increase in WPV1 cases have been reported from Nigeria in 2008 (75 cases) compared with the same time last year (8 cases). Fifty per cent of the cases in 2008 are geographically localised to 3 states: Kano, Jigawa and Sokoto. The next immunisation activity will occur in the high risk northern states from 5 to 8 April 2008.[29]

Chad

Large numbers of refugees from Chad moving across the border to neighbouring Cameroon and the Central African Republic has increased the risk of spreading polio infection (a case reported in February from Cameroon with date of onset in November 2007 was genetically linked to the virus circulating in Chad).[29]

Tuberculosis

The World Health Organization's fourth report on *Anti-Tuberculosis Drug Resistance in the World* states that levels of multi-drug resistant tuberculosis (MDR-TB) are much higher than expected and that extensively drug resistant TB (XDR-TB) is present and virtually untreatable in 45 countries. The report concludes that there is an urgent need to address the problem, including improving diagnostic capacity in some countries (including PNG) which cannot diagnose or treat MDR cases.[30]

Africa

Outbreaks of drug-resistant TB in Africa continue unabated and are often further complicated by co-infection with HIV/AIDS.[31] During this report-

ing period the Ministry of Health in Botswana reported the first ever cases (2) of XDR-TB in the country and 100 cases of MDR-TB.

United Kingdom ex Somalia

In March 2008, UK Health officials confirmed the first case of XDR-TB in the UK. The case is a 30-year-old immigrant from Somalia who was found to have inactive pulmonary TB when screened on arrival in the UK in November 2007. Cultures later identified the XDR strain of tuberculosis and public health officials began to trace and monitor those potentially exposed.[32]

Yellow fever South America

In this quarter there have been major outbreaks of yellow fever in Argentina , Brazil and Paraguay.

The Argentinan Ministry of Health reported the country's first official case of human yellow fever since 1967, on 3 March 2008 in Misiones Province in the north-eastern corner of Argentina. As of 17 March 2008, the Argentinan MoH has reported a total of 5 cases of human yellow fever, including 1 death, all from Misiones Province.[33,34]

Between 8 January and 4 April 2008, the Brazilian Ministry of Health reported 70 human cases (40 of them confirmed) including 21 deaths from sylvan (jungle) yellow fever. Ninety per cent of the confirmed cases had no proven vaccination history or were vaccinated greater than 10 years previously.[35]

The Paraguayan Ministry of Health reported the first cases of yellow fever in the country in more than 30 years. Between 15 January and 31 March 2008, the MoH reported 26 confirmed cases of human yellow fever (including 8 deaths).

While some rural, forested areas of Paraguay have previously been known risk areas for yellow fever this recent outbreak has demonstrated the presence of yellow fever in other areas including possible urban transmission.[36,37]

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