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Annual reports

Australia's notifiable disease status, 2008: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

In 2008, 65 communicable diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 160,508 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, an increase of 9% on the number of notifications in 2007. In 2008, the most frequently notified diseases were sexually transmissible infections (69,459 notifications, 43% of total notifications), vaccine preventable diseases (34,225 notifications, 21% of total notifications) and gastrointestinal diseases (27,308 notifications, 17% of total notifications). There were 18,207 notifications of bloodborne diseases; 8,876 notifications of vectorborne diseases; 1,796 notifications of other bacterial infections; 633 notifications of zoonoses and 4 notifications of quarantinable diseases. *Commun Dis Intell* 2010;34(3):157–225.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2008, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, state and territory, and local levels. Primary responsibility for public health action lies with the state and territory health departments. The purpose of communicable disease surveillance at a national level is to:

- identify national trends and compare the rates of specific diseases across Australia with national averages;
- guide policy development and resource allocation at a national level;
- monitor the need for and impact of national disease control programs;
- identify national or multi-jurisdictional outbreaks and coordinate a national response;
- describe the epidemiology of rare diseases in Australia;
- meet international reporting requirements, such as providing disease statistics to the World Health Organization (WHO); and
- support 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 September 2007, the *National Health Security Act 2007*[1] received royal assent. This Act provides a legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List,[2] which specifies the diseases for which personal information can be shared. The *National Health Security Agreement 2008*[3] establishes operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Agreement.

Under the Agreement, in 2008 states and territories forwarded de-identified data on the nationally agreed set of 65 communicable diseases to the Department of Health and Aging for the purposes of national communicable disease surveillance, although not all 65 diseases were notifiable in each jurisdiction. States and territories provided data

to the National Notifiable Diseases Surveillance System (NNDSS) electronically, daily or several times a week. The system was complemented by other surveillance systems, which provided information on various diseases, including four that are not reported to NNDSS, namely human immunodeficiency virus (HIV), acquired immune deficiency (AIDS) and the classical and variant forms of Creutzfeldt-Jakob disease (CJD).

In 2008, the NNDSS core dataset included the following 5 mandatory data fields: unique record reference number; notifying state or territory; disease code; confirmation status and the date when the public health unit was notified (notification receive date). In addition, the following core but non-mandatory data fields were supplied where possible: date of birth; age at onset; sex; indigenous status; postcode of residence; disease onset date; date when the medical practitioner signed the notification form (notification date), death status, date of specimen collection and outbreak reference number (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 were collected and reported to NNDSS. Data quality was monitored by the Office of Health Protection and the National Surveillance Committee (NSC) and there was a continual process of improving the national consistency of communicable disease surveillance through the daily, fortnightly and quarterly review of these data.

While not included in the core national dataset, enhanced surveillance information for some diseases (invasive pneumococcal disease, hepatitis C, tuberculosis and some sexually transmissible infections) were reported from states and territories to NNDSS but not included in this report. Additional information concerning mortality and specific health risk factors for some diseases were obtained from states and territories and included in this annual report.

Newly diagnosed HIV infection and AIDS were notifiable conditions in each state or territory health jurisdiction in 2008 and these data were forwarded directly to the National HIV Registry and National AIDS Registry at the National Centre in HIV Epidemiology and Clinical Research (NCHECR). Further information can be found in NCHECR's annual surveillance report.[4]

Surveillance of the classical and variant forms of CJD in Australia has been conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) since its establishment in October 2003. CJD is a nationally notifiable disease and by June 2006, CJD was notifiable in all states and territories. Further surveillance information on CJD can be found in surveillance reports from the ANCJDR.[5]

Information from communicable disease surveillance is communicated through several avenues. The most up-to-date information on topics of interest is provided at fortnightly teleconferences of the Communicable Diseases Network Australia (CDNA) and a summary of these reports is available online from http://www.health.gov.au/cdnareport[6] The *Communicable Diseases Intelligence* (*CDI*) quarterly journal publishes surveillance data and reports of research studies on the epidemiology and control of various communicable diseases.

Notification rates for each notifiable disease were calculated using the estimated 2008 mid-year resident population supplied by the Australian Bureau of Statistics[7] (ABS) (Appendix 1 and Appendix 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 either the direct method of standardisation for gastrointestinal diseases, or indirect method for sexually transmissible infections, with 2006 census data as the standard population.

The 4 maps produced for this report (chlamydia, influenza, pertussis, Q fever) were created with ArcGIS mapping software (ESRI, Redlands, CA) and based on the NNDSS notifications' residential postcode recorded in the NNDSS.

With one exception, maps were based on Statistical Divisions (SDs), as defined by the Australian Standard Geographical Classification (AGSC) (Map 1, Table 1), for all states and territories. The Northern Territory was represented by Statistical Subdivisions (SSD) and in the case of Greater Darwin, by the combination of the Tiwi Islands, Darwin, Palmerston and Litchfield SSD. This combination helped preserve confidentiality while improving legibility at the scale the maps to be printed. The geocode 77777 for Greater Darwin is only nominal.

Notifications were summed by the postcode weighting calculated by the Australian Bureau of Statistics Postcode Concordance.[8] These ABS concordance data were used to proportionally allocate notifications into SDs/SSDs according to the percentage of the population of the postcode living in the region. The total notifications per region are displayed in the relevant area.

Disease rates were calculated per 100,000 population for the relevant areas using ABS population

data.[7] Rates were mapped for different SDs and ordered into 5 groups using the Jenks Natural Breaks method (http://resources.arcgis.com/content/kbase?fa=articleShow&d=26442) whereby the largest breaks between natural clusters of ordered data were identified and used as class boundaries. A class '0' was added to account for areas with no notifications, resulting in a total of 6 rate classes per map. Note that the classification is data dependent and changes from map to map.

Notes on interpretation

The present report is based on 2008 'finalised' data from each state or territory agreed upon in September 2009 and represents a snapshot of the year after duplicate records and incorrect or incomplete data were 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 diagnosis in an attempt to estimate disease activity within the reporting period. The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. As considerable time may have elapsed between the onset and diagnosis dates for hepatitis B (unspecified), hepatitis C (unspecified) and tuberculosis, the earliest specimen date, health professional notification date or public health unit notification receive date was used for these conditions.

Notified cases 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.

A survey of jurisdictional public health departments was conducted in 2009 to ascertain the source of each notification (Table 2). Five jurisdictions reported notifications in their jurisdictions originating from laboratory only, of greater than or equal to 95%. South Australia and Western Australia reported notifications in their jurisdictions originating from laboratory and doctor of 77% and 66.2% respectively, whilst Victoria reported 46%. South Australia reported the greatest percentage of notifications in their jurisdictions originating from doctors only, at 9%.

Whilst most jurisdictions have data on laboratory notifications, the percentage of notifications attributed to doctor only and laboratory and doctor for each state and territory are based on estimates deduced from the data that are available, noting that fields for these data may be incomplete. Western Australia is the only jurisdiction that maintains data on the source of notifications from laboratories and/or doctors.

Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories and hospitals. Although the National Notifiable Diseases List[2] was established under the *National Health Securities Act, 2007*, some diseases are not yet notifiable in all 8 jurisdictions (Table 3).

Figure 1: Communicable diseases notifiable fraction

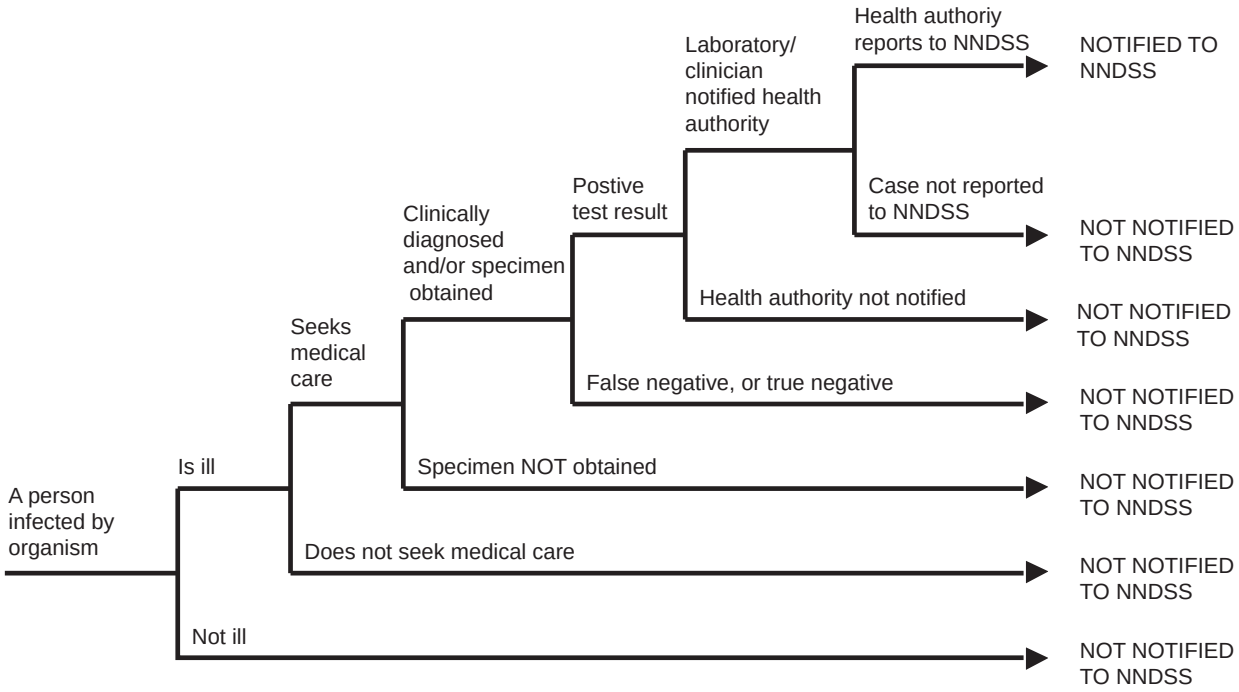
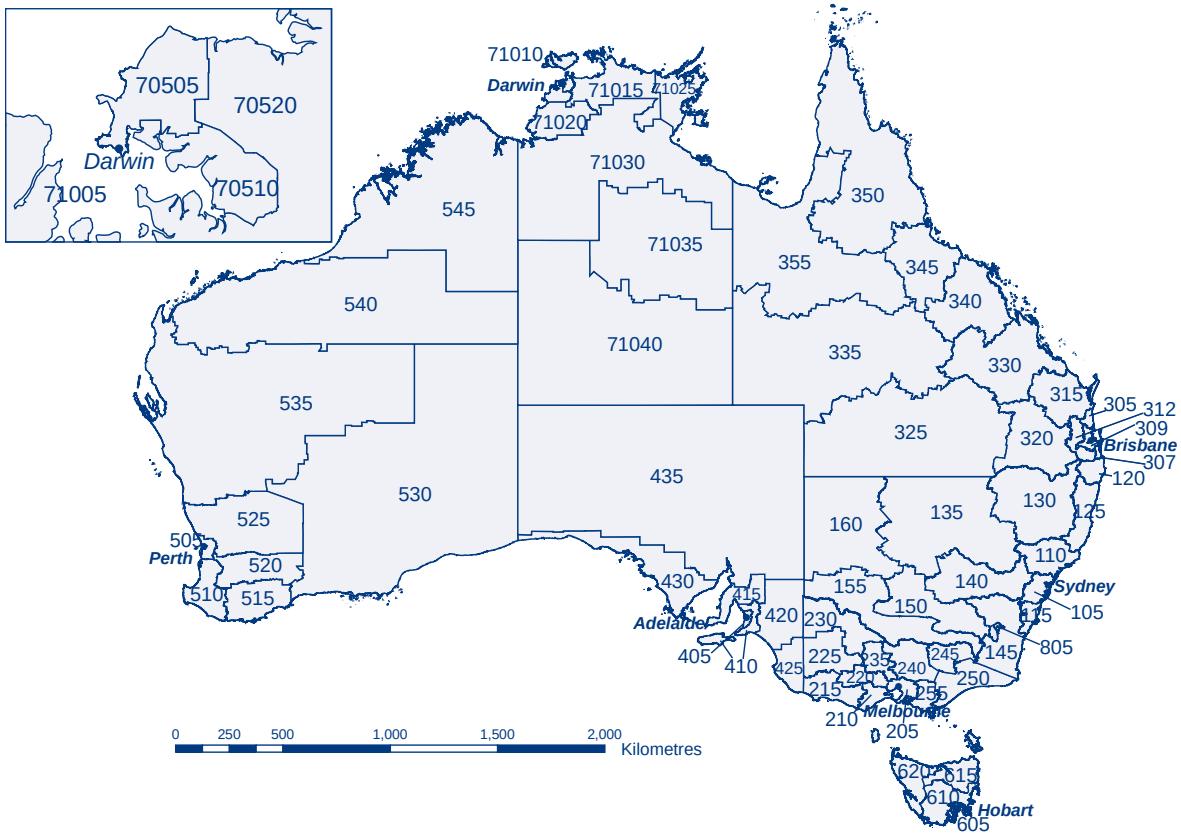


Table 1: Australian population by Statistical Division and Statistical Subdivision for the Northern Territory, 2008

SD code	Statistical Division	Population
Australian Capital Territory		
805	Canberra	345,257
810	ACT balance	294
New South Wales		
105	Sydney	4,399,722
110	Hunter	632,851
115	Illawarra	423,487
120	Richmond–Tweed	237,361
125	Mid-North Coast	304,323
130	Northern	181,667
135	North Western	116,736
140	Central West	180,074
145	South Eastern	212,238
150	Murrumbidgee	155,868
155	Murray	117,108
160	Far West	22,737
Northern Territory (Subdivisions)		
71005	Finniss	2,214
71010	Bathurst–Melville	2,501
71015	Alligator	6,913
71020	Daly	4,353
71025	East Arnhem	16,077
71030	Lower Top End NT	18,894
71040	Central NT	40,299
77777	Greater Darwin	123,139
Queensland		
305	Brisbane	1,945,639
307	Gold Coast	497,848
309	Sunshine Coast	312,804
312	West Moreton	90,738
315	Wide Bay–Burnett	277,965
320	Darling Downs	231,599
325	South West	26,150
330	Fitzroy	214,753
335	Central West	12,256
340	Mackay	167,666
345	Northern	220,656
350	Far North	262,095
355	North West	33,746
South Australia		
405	Adelaide	1,172,105
410	Outer Adelaide	134,085
415	Yorke and Lower North	46,396
420	Murray Lands	70,125
425	South East	65,402
430	Eyre	35,174
435	Northern	80,074
Tasmania		
605	Greater Hobart	209,287
610	Southern	36,875
615	Northern	140,275
620	Mersey–Lyell	111,092
Victoria		
205	Melbourne	3,892,419
210	Barwon	278,668
215	Western District	104,709
220	Central Highlands	152,075
225	Wimmera	50,404
230	Mallee	93,568
235	Loddon	179,948
240	Goulburn	207,685
245	Ovens–Murray	98,250
250	East Gippsland	85,318
255	Gippsland	170,779
Western Australia		
505	Perth	1,602,559
510	South West	236,058
515	Lower Great Southern	57,439
520	Upper Great Southern	18,887
525	Midlands	54,603
530	South Eastern	58,074
535	Central	63,409
540	Pilbara	45,983
545	Kimberley	34,185
Other territories		–
Total		21,423,938

Source: ABS 3218.0 Regional Population Growth, Australia, 23 April 2009 (http://abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3218.02007–08).

Map 1: Australian Bureau of Statistics Statistical Division codes, Australia, and Statistical Subdivision codes, Northern Territory, 2008



Changes in surveillance practices have been introduced in some jurisdictions and not in others, and making the comparison of data across jurisdictions difficult. In this report, some information was obtained from states and territories, including changes in surveillance practices, screening practices, laboratory practices, and major disease control or prevention initiatives to assist in the interpretation of the 2008 data.

Postcode information usually reflects the residential location of the case, but this does not necessarily represent the place where the disease was acquired. In December 2008, the CDNA endorsed the NNDSS cross-border notification protocol, which determines that the jurisdiction of residence of a case has the responsibility of reporting the notification to NNDSS. This was implemented from 1 January 2009, and may also affect some retrospective notifications by removing duplicates and preventing the loss of notification data in NNDSS.

Table 2: Percentage of notifications from different sources in each jurisdiction, 2008

State or territory	Source of notifications		
	Laboratory only	Doctor only	Laboratory and doctor
ACT	98.0	1.0	1.0
NSW*	95.0	1.5	1.2
NT	99.0	1.0	<1.0
Qld	97.5	0.5	2.0
SA*	8.5	9.0	77
Tas	98.0	2.0	<1.0
Vic	48.0	6.0	46.0
WA	30.5	3.3	66.2

* Not all percentages add up to 100% due to other sources of notifications and/or incomplete data for laboratory and medical notification fields.

Table 3: Diseases notified to the National Notifiable Diseases Surveillance System, Australia 2008

Disease	Data received from
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)*	All jurisdictions
Hepatitis B (unspecified)†	All jurisdictions
Hepatitis C (newly acquired)*	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 infections¶	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection**	All jurisdictions
Syphilis – < 2 years duration†	All jurisdictions
Syphilis – > 2 years or unspecified duration†	All jurisdictions, except South Australia
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

Table 3: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, *continued*

Disease	Data received from
Vaccine preventable diseases, *continued*	
Varicella zoster (chickenpox)‡‡	All jurisdictions, except New South Wales
Varicella zoster (shingles)‡‡	All jurisdictions, except New South Wales
Varicella zoster (unspecified)‡‡	All jurisdictions, except New South Wales
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
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

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

‡ In Queensland, includes incident hepatitis C 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 and Western Australia excludes ocular infections. Where data fields were complete, infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

** Where data fields were complete, gonococcal infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

†† Influenza (laboratory confirmed) became notifiable in South Australia on 1 May 2008.

‡‡ Varicella zoster became notifiable in Victoria on 21 September 2008.

§§ Arbovirus (NEC) replaced Flavivirus (NEC) in 2008.

‖‖ 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.

NEC Not elsewhere classified.

Data completeness was assessed for the notification's sex, age at onset, and indigenous status, and reported as the proportion of complete notifications. The completeness of data in this report is summarised in the Results.

The percentage of data completeness was defined as:

Percentage of data completeness = (total notifications – missing or unknown)/total notifications x 100

The indigenous status was defined by the following nationally accepted values:[10]

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

Notes on cases definitions

All notifiable diseases reported to the NNDSS must meet their respective national surveillance case definitions. These case definitions were agreed by CDNA and implemented nationally from January 2004 and were used by all jurisdictions for the first time in 2005. The national surveillance case definitions and their status are available from http://www.health.gov.au/casedefinitions

Results

There were 160,508 communicable disease notifications received by NNDSS in 2008 (Table 4).

In 2008, the most frequently notified diseases were sexually transmissible infections (69,459 notifications, 43.3% of total notifications), vaccine preventable diseases (34,225 notifications, 21.3% of total notifications) and gastrointestinal diseases (27,308 notifications, 17% of total notifications).

There were 18,207 notifications of bloodborne diseases; 8,876 notifications of vectorborne diseases; 1,796 notifications of other bacterial infections; 633 notifications of zoonoses and 4 notifications of quarantinable diseases. In 2008, the total number of notifications was the highest recorded in the NNDSS since the surveillance system commenced data collection in 1991. There was an increase of 9% compared with the total number of notifications in 2007 (Figure 2).

Notifications and notification rates per 100,000 population for each disease by state or territory, in 2008, are shown in Table 5 and Table 6 respectively. Trends in notifications and rates per 100,000 population for the period 2003 to 2008 are shown in Table 7.

The year in which diseases became notifiable to NNDSS in each jurisdiction is shown in Table 8.

Table 4: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2008, by disease category rank order

Disease category	Number	%
Sexually transmitted infections	69,459	43.3
Vaccine preventable diseases	34,225	21.3
Gastrointestinal diseases	27,308	17.0
Bloodborne diseases	18,207	11.3
Vectorborne diseases	8,876	5.5
Other bacterial diseases	1,796	1.1
Zoonoses	633	0.4
Quarantinable diseases	4	<0.1
Total	160,508	100.0

Figure 2: Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2008

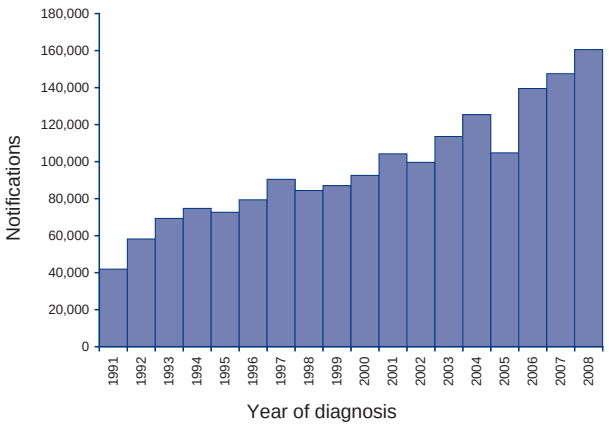


Table 5: Notifications of communicable diseases, Australia, 2008, by state or territory

Disease	State or territory								
	ACT	NSW	NT*	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0	0	0	0	0	0	1	0	1
Hepatitis B (newly acquired)†	1	46	8	45	11	12	88	34	245
Hepatitis B (unspecified)	67	2,555	197	843	417	58	1,832	631	6,600
Hepatitis C (newly acquired)†	5	24	6	NN	65	24	154	103	381
Hepatitis C (unspecified)‡,§	195	3,555	222	2,634	515	324	2,252	1,241	10,938
Hepatitis D	0	14	1	7	0	0	14	6	42
Gastrointestinal diseases									
Botulism	0	0	0	0	0	0	0	0	0
Campylobacteriosis‖	381	NN	257	4,821	1,992	475	5,780	1,829	15,535
Cryptosporidiosis	11	484	102	695	63	36	449	165	2,005
Haemolytic uraemic syndrome	0	17	1	7	2	0	4	0	31
Hepatitis A	5	69	3	71	20	1	85	22	276
Hepatitis E	0	14	3	7	0	0	14	6	44
Listeriosis	1	34	0	12	1	1	11	8	68
Salmonellosis	132	2,261	497	2,047	661	206	1,651	855	8,310
Shigellosis	3	109	175	97	137	4	134	169	828
STEC,VTEC¶	0	19	0	37	39	0	11	0	106
Typhoid	0	43	1	18	3	0	32	8	105
Quarantinable diseases									
Cholera	0	2	0	0	0	0	0	2	4
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 infection**	988	14,019	2,296	15,197	3,653	1,481	12,210	8,640	58,484
Donovanosis	0	0	1	1	0	0	0	0	2
Gonococcal infection††	21	1,332	1,567	1,638	521	25	926	1,693	7,723
Syphilis – all‡‡	36	1,407	253	390	52	22	793	290	3,243
Syphilis < 2 years duration‡	4	416	83	187	52	7	374	180	1,303
Syphilis > 2 years or unspecified duration‡	32	991	170	203	NDP	15	419	110	1,940
Syphilis – congenital	0	3	1	3	0	0	0	0	7
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	9	2	6	1	1	6	0	25
Influenza (laboratory confirmed)§§	244	1,814	199	3,703	473	388	1,300	1,016	9,137
Measles	0	39	3	11	2	0	2	8	65
Mumps	0	77	53	29	17	2	13	95	286
Pertussis	145	7,818	477	2,260	1,459	200	1,694	463	14,516
Pneumococcal disease (invasive)	20	547	60	326	120	39	355	162	1,629
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	0	17	0	4	1	0	8	7	37
Rubella – congenital	0	0	0	0	0	0	0	0	0
Tetanus	0	1	0	1	0	0	1	1	4

Table 5: Notifications of communicable diseases, Australia, 2008, by state or territory, *cont'd*

Disease	State or territory								
	ACT	NSW	NT*	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *continued*									
Varicella zoster (chickenpox)‖‖	12	NN	115	429	620	29	230	355	1,790
Varicella zoster (shingles)‖‖	7	NN	106	447	931	125	185	508	2,309
Varicella zoster (unspecified)‖‖	102	NN	2	3,138	223	46	162	754	4,427
Vectorborne diseases									
Arbovirus infection (NEC)¶¶	0	1	0	21	0	0	6	0	28
Barmah Forest virus infection	7	533	76	1,242	37	1	32	174	2,102
Dengue virus infection	6	154	23	232	31	6	8	98	558
Japanese encephalitis virus infection	0	1	0	0	0	0	0	0	1
Kunjin virus infection***	0	0	0	1	0	0	0	0	1
Malaria	15	116	20	167	17	8	105	85	533
Murray Valley encephalitis virus infection***	0	1	0	0	0	0	0	1	2
Ross River virus infection	21	1,152	261	2,838	197	77	231	874	5,651
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australia bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	2	0	46	0	0	0	0	48
Leptospirosis	0	17	1	89	0	0	4	1	112
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	41	0	3	0	0	53	6	103
Q fever	2	164	3	158	17	0	20	6	370
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial diseases									
Legionellosis	4	89	1	31	21	1	54	70	271
Leprosy	0	4	1	2	0	0	2	2	11
Meningococcal infection†††	3	81	8	85	20	1	64	24	286
Tuberculosis	12	501	32	144	54	8	379	98	1,228
Total	**2,446**	**39,186**	**7,034**	**43,983**	**12,393**	**3,601**	**31,355**	**20,510**	**160,508**

* Due to delays in data quality checks, data for Northern Territory was preliminary at the time of analysis.

† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

§ In Queensland, includes incident hepatitis C 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 and Western Australia excludes ocular infections. Where data fields were complete, infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

†† Where data fields were complete, gonococcal infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

‡‡ Does not include congenital syphilis.

§§ Influenza (laboratory confirmed) became notifiable in South Australia on 1 May 2008.

‖‖ Varicella zoster became notifiable in Victoria on 21 September 2008.

¶¶ Arbovirus (NEC) replaced Flavivirus (NEC) in 2008.

*** 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.

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Table 6: Notification rates of nationally notifiable communicable diseases, Australia, 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 (newly acquired)†	0.3	0.7	3.6	1.0	0.7	2.4	1.7	1.6	1.1
Hepatitis B (unspecified)	19.4	36.6	89.6	19.6	26.0	11.7	34.5	29.1	30.8
Hepatitis C (newly acquired)†	1.4	0.3	2.7	NN	4.1	4.8	2.9	4.7	2.2
Hepatitis C (unspecified)‡,§	56.4	50.9	101.0	61.3	32.1	65.1	42.4	57.2	51.0
Hepatitis D	0.0	0.2	0.5	0.2	0.0	0.0	0.3	0.3	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis‖	110.3	NN	116.9	112.3	124.2	95.5	108.8	84.2	107.5
Cryptosporidiosis	3.2	6.9	46.4	16.2	3.9	7.2	8.4	7.6	9.4
Haemolytic uraemic syndrome	0.0	0.2	0.5	0.2	0.1	0.0	0.1	0.0	0.1
Hepatitis A	1.4	1.0	1.4	1.7	1.2	0.2	1.6	1.0	1.3
Hepatitis E	0.0	0.2	1.4	0.2	0.0	0.0	0.3	0.3	0.2
Listeriosis	0.3	0.5	0.0	0.3	0.1	0.2	0.2	0.4	0.3
Salmonellosis	38.2	32.4	226.1	47.7	41.2	41.4	31.1	39.4	38.8
Shigellosis	0.9	1.6	79.6	2.3	8.5	0.8	2.5	7.8	3.9
STEC,VTEC¶	0.0	0.3	0.0	0.9	2.4	0.0	0.2	0.0	0.5
Typhoid	0.0	0.6	0.5	0.4	0.2	0.0	0.6	0.4	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	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 transmitted infections									
Chlamydial infection**	285.9	200.7	1044.5	353.9	227.8	297.7	229.8	397.9	272.9
Donovanosis	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection††	6.1	19.1	712.9	38.1	32.5	5.0	17.4	78.0	36.0
Syphilis – all‡‡	10.4	20.2	115.1	9.1	3.2	4.4	14.9	13.4	15.1
Syphilis < 2 years duration‡	1.2	6.0	37.8	4.4	3.2	1.4	7.0	8.3	6.1
Syphilis > 2 years or unspecified duration‡	9.3	14.2	77.3	4.7	NDP	3.0	7.9	5.1	9.8
Syphilis – congenital	0.0	0.0	0.5	0.1	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	0.9	0.1	0.1	0.2	0.1	0.0	0.1
Influenza (laboratory confirmed)§§	70.6	26.0	90.5	86.2	29.5	78.0	24.5	46.8	42.6
Measles	0.0	0.6	1.4	0.3	0.1	0.0	0.0	0.4	0.3
Mumps	0.0	1.1	24.1	0.7	1.1	0.4	0.2	4.4	1.3
Pertussis	42.0	111.9	217.0	52.6	91.0	40.2	31.9	21.3	67.7
Pneumococcal disease (invasive)	5.8	7.8	27.3	7.6	7.5	7.8	6.7	7.5	7.6
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.1	0.1	0.0	0.2	0.3	0.2
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 6: Notification rates of nationally notifiable communicable diseases, Australia, 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*									
Varicella zoster (chickenpox)‖‖	3.5	NN	52.3	10.0	38.7	5.8	NRC	16.4	19.6
Varicella zoster (shingles)‖‖	2.0	NN	48.2	10.4	58.1	25.1	NRC	23.4	25.3
Varicella zoster (unspecified)‖‖	29.5	NN	0.9	73.1	13.9	9.2	NRC	34.7	48.5
Vectorborne diseases									
Arbovirus infection (NEC)¶¶	0.0	0.0	0.0	0.5	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	2.0	7.6	34.6	28.9	2.3	0.2	0.6	8.0	9.8
Dengue virus infection	1.7	2.2	10.5	5.4	1.9	1.2	0.2	4.5	2.6
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	4.3	1.7	9.1	3.9	1.1	1.6	2.0	3.9	2.5
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	6.1	16.5	118.7	66.1	12.3	15.5	4.3	40.3	26.4
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	1.1	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.2	0.5	2.1	0.0	0.0	0.1	0.0	0.5
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.6	0.0	0.1	0.0	0.0	1.0	0.3	0.5
Q fever	0.6	2.3	1.4	3.7	1.1	0.0	0.4	0.3	1.7
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	1.2	1.3	0.5	0.7	1.3	0.2	1.0	3.2	1.3
Leprosy	0.0	0.1	0.5	0.0	0.0	0.0	0.0	0.1	0.1
Meningococcal infection†††	0.9	1.2	3.6	2.0	1.2	0.2	1.2	1.1	1.3
Tuberculosis	3.5	7.2	14.6	3.4	3.4	1.6	7.1	4.5	5.7

* Due to delays in data quality checks, data for Northern Territory was preliminary at the time of analysis.

† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

§ In Queensland, includes incident hepatitis C 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 and Western Australia excludes ocular infections. Where data fields were complete, infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

†† Where data fields were complete, gonococcal infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

‡‡ Does not include congenital syphilis.

§§ Influenza (laboratory confirmed) became notifiable in South Australia on 1 May 2008.

‖‖ Varicella zoster became notifiable in Victoria on 21 September 2008.

¶¶ Arbovirus (NEC) replaced Flavivirus (NEC) in 2008.

*** 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.

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

NRC No rate calculated – due to part year reporting. Varicella zoster became notifiable in Victoria on 21 September 2008.

Table 7: Notifications and notification rate per 100,000 population for communicable diseases, Australia, 2003 to 2008

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2003	2004	2005	2006	2007	2008			2003	2004	2005	2006	2007	2008
Bloodborne diseases														
Hepatitis (NEC)	0	0	1	1	0	1	0.4	2.5	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	347	283	251	291	294	245	293.2	0.8	1.7	1.4	1.2	1.4	1.4	1.1
Hepatitis B (unspecified)	5,803	5,781	6,291	6,254	6,887	6,600	6,203.2	1.1	29.2	28.7	30.8	30.2	32.7	30.8
Hepatitis C (newly acquired)*	514	456	373	436	385	381	432.8	0.9	3.2	2.8	2.3	2.6	2.3	2.2
Hepatitis C (unspecified)†,‡	13,606	12,661	11,955	11,931	11,905	10,938	12,411.6	0.9	68.4	62.9	58.6	57.6	56.5	51.0
Hepatitis D	26	29	32	30	34	42	30.2	1.4	0.1	0.1	0.2	0.1	0.2	0.2
Gastrointestinal diseases														
Botulism	1	1	3	1	1	0	1.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	15,360	15,589	16,497	15,423	16,996	15,535	15,973.0	1.0	116.2	116.2	121.0	111.1	120.0	107.5
Cryptosporidiosis	1,222	1,685	3,215	3,203	2,812	2,005	2,427.4	0.8	6.1	8.4	15.8	15.5	13.3	9.4
Haemolytic uraemic syndrome	15	16	20	14	19	31	16.8	1.8	0.1	0.1	0.1	0.1	0.1	0.1
Hepatitis A	431	319	327	281	165	276	304.6	0.9	2.2	1.6	1.6	1.4	0.8	1.3
Hepatitis E	12	28	30	24	18	44	22.4	2.0	0.1	0.1	0.1	0.1	0.1	0.2
Listeriosis	69	67	54	61	50	68	60.2	1.1	0.3	0.3	0.3	0.3	0.2	0.3
Salmonellosis	7,001	7,839	8,424	8,255	9,533	8,310	8,210.4	1.0	35.2	38.9	41.3	39.9	45.2	38.8
Shigellosis	442	520	729	546	602	828	567.8	1.5	2.2	2.6	3.6	2.6	2.9	3.9
STEC,VTEC‖	52	49	86	70	107	106	72.8	1.5	0.3	0.2	0.4	0.3	0.5	0.5
Typhoid	51	76	52	77	91	105	69.4	1.5	0.3	0.4	0.3	0.4	0.4	0.5
Quarantinable diseases														
Cholera	1	5	3	3	4	4	3.2	1.3	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	0.0	0.0
Plague	0	0	0	0	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	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	0.0	0.0
Smallpox	0	0	0	0	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	0.0	0.0
Yellow fever	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 7: Notifications and notification rate per 100,000 population for communicable diseases, Australia, 2003 to 2008, *continued*

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2003	2004	2005	2006	2007	2008			2003	2004	2005	2006	2007	2008
Sexually transmissible infections														
Chlamydial infection[¶]	30,419	36,212	41,346	47,458	52,022	58,484	41,491.4	1.4	152.9	179.9	202.7	229.3	246.9	272.9
Donovanosis	16	10	13	6	3	2	9.6	0.2	0.1	0.0	0.1	0.0	0.0	0.0
Gonococcal infection**	6,779	7,175	8,070	8,562	7,676	7,723	7,652.4	1.0	34.1	35.6	39.6	41.4	36.4	36.0
Syphilis – all[††]	2,004	2,347	2,234	2,687	3,161	3,243	2,486.6	1.3	10.1	11.7	11.0	13.0	15.0	15.1
Syphilis < 2 years duration[†]	NN	634	650	878	1,422	1,303	716.8[‡‡]	1.8	NN	3.1	3.2	4.2	6.7	6.1
Syphilis > 2 years or unspecified duration[†]	NN	1,713	1,584	1,809	1,739	1,940	1,369.0[‡‡]	1.4	NN	9.2	8.4	9.5	8.9	9.8
Syphilis – congenital	13	13	15	14	7	7	12.4	0.6	0.1	0.1	0.1	0.1	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	0.0	0.0
Haemophilus influenzae type b	19	15	17	22	17	25	18.0	1.4	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)[§§]	3,479	2,138	4,561	3,254	10,449	9,137	4,776.2	1.9	17.5	10.6	22.4	15.7	49.6	42.6
Measles	93	45	10	125	12	65	57.0	1.1	0.5	0.2	0.0	0.6	0.1	0.3
Mumps	77	102	240	275	586	286	256.0	1.1	0.4	0.5	1.2	1.3	2.8	1.3
Pertussis	5,096	8,750	11,200	10,995	5,345	14,516	8,277.2	1.8	25.6	43.5	54.9	53.1	25.4	67.7
Pneumococcal disease (invasive)	2,226	2,373	1,706	1,463	1,483	1,629	1,850.2	0.9	11.2	11.8	8.4	7.1	7.0	7.6
Poliomyelitis	0	0	0	0	1	0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	55	31	31	59	34	37	42.0	0.9	0.3	0.2	0.2	0.3	0.2	0.2
Rubella – congenital	3	1	1	0	2	0	1.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	4	5	2	3	3	4	3.4	1.2	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)[¶¶]	NN	NN	NN	1,558	1,667	1,790	1,080.3[¶¶]	1.7	NN	NN	NN	17.8	18.6	19.6
Varicella zoster (shingles)[¶¶]	NN	NN	NN	1,092	1,561	2,309	886.7[¶¶]	2.6	NN	NN	NN	12.5	17.4	25.3
Varicella zoster (unspecified)[¶¶]	NN	NN	NN	3,677	4,286	4,427	2,701.3[¶¶]	1.6	NN	NN	NN	42.0	47.9	48.5
Vectorborne diseases														
Arbovirus infection (NEC)***	58	60	28	32	22	28	40.0	0.7	0.3	0.3	0.1	0.2	0.1	0.1
Barmah Forest virus infection	1,367	1,103	1,323	2,140	1,716	2,102	1,529.8	1.4	6.9	5.5	6.5	10.3	8.1	9.8
Dengue virus infection	861	351	220	187	314	558	386.6	1.4	4.3	1.7	1.1	0.9	1.5	2.6
Japanese encephalitis virus infection	1	1	0	0	0	1	0.4	2.5	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection[†††]	9	6	1	3	1	1	4.0	0.3	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	585	547	817	770	568	533	657.4	0.8	2.9	2.7	4.0	3.7	2.7	2.5
Murray Valley encephalitis virus infection[†††]	0	1	2	1	0	2	0.8	2.5	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3,844	4,209	2,540	5,545	4,207	5,651	4,069.0	1.4	19.3	20.9	12.5	26.8	20.0	26.4

Table 7: Notifications and notification rate per 100,000 population for communicable diseases, Australia, 2003 to 2008, *continued*

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2003	2004	2005	2006	2007	2008			2003	2004	2005	2006	2007	2008
Zoonoses														
Anthrax	0	0	0	1	1	0	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	20	38	41	51	38	48	37.6	1.3	0.1	0.2	0.2	0.2	0.2	0.2
Leptospirosis	127	177	129	145	108	112	137.2	0.8	0.6	0.9	0.6	0.7	0.5	0.5
Ornithosis	200	239	164	165	93	103	172.2	0.6	1.0	1.2	0.8	0.8	0.4	0.5
Q fever	560	463	351	408	445	370	445.4	0.8	2.8	2.3	1.7	2.0	2.1	1.7
Other bacterial infections														
Legionellosis	333	312	331	349	306	271	326.2	0.8	1.7	1.6	1.6	1.7	1.5	1.3
Leprosy	7	6	10	7	13	11	8.6	1.3	0.0	0.0	0.0	0.0	0.1	0.1
Meningococcal infection‡‡‡	558	405	391	318	306	286	395.6	0.7	2.8	2.0	1.9	1.5	1.5	1.3
Tuberculosis	986	1,052	1,083	1,208	1,174	1,228	1,100.6	1.1	5.0	5.2	5.3	5.8	5.6	5.7
Total	**102,748**	**113,593**	**125,384**	**139,481**	**147,530**	**160,508**	**125,747.2**							

* Newly acquired hepatitis includes cases in whom the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases in whom the duration of infection could not be determined.

‡ Data provided from Queensland (2003–2008) and the Northern Territory (2003–2004) includes both newly-acquired and unspecified hepatitis C notifications.

§ 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 and Western Australia excludes ocular infections. Where data fields were complete, infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

** Where data fields were complete, infections defined as non-sexually acquired (e.g. perinatal) in individuals aged less than 13 years, were excluded from the data.

†† Does not include congenital syphilis.

‡‡ Ratios for Syphilis < 2 years; syphilis > 2 years or unspecified duration are based on 4 years data.

§§ Influenza (laboratory confirmed) became notifiable in South Australia on 1 May 2008.

‖‖ Nationally notifiable in 2006 and first full year of national reporting from 2007. Varicella zoster became notifiable in Victoria on 21 September 2008.

¶¶ Ratios for varicella zoster (chickenpox); varicella zoster (shingles); and varicella zoster (unspecified) are based on 2 years data.

*** Arbovirus (NEC) replaced Flavivirus (NEC) in 2008.

††† 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.

NEC Not elsewhere classified.

NN Not notifiable.

Table 8: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Bloodborne diseases										
Hepatitis (NEC)	1991	1991	1991	1991	1991	1991	1991	NN	1991 to present	WA do not report
Hepatitis B (newly acquired)	1995	1993	1993	1994	1993	1993	1993	1994	1995 to present	
Hepatitis B (unspecified)	1991	1991	2004	1994	1991	1991	1991	1991	1991 to present	
Hepatitis C (newly acquired)	1995	1993	2005	NN	1993	1995	1997	1995	1993 to present	All jurisdictions except Qld
Hepatitis C (unspecified)	1991	1991	1991	1991	1994	1991	1991	1993	1995 to present	Includes reports of incident hepatitis C, 1991 to 1994
Hepatitis D	1999	1999	1999	1997	1999	1999	1999	2001	1999 to present	WA did not report 1999–2000
Gastrointestinal diseases										
Botulism	1992	1998	1998	1997	1993	1992	1992	2001	1992 to present	State reporting started as shown
Campylobacteriosis	1991	NN	1991	1991	1991	1991	1991	1991	1991 to present	NSW do not report
Cryptosporidiosis	2001	2001	2001	1996	2001	2001	2001	2001	2001 to present	
Haemolytic uraemic syndrome	1999	1999	1999	1997	1999	1999	1999	1999	1999 to present	
Hepatitis A	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Hepatitis E	1999	1999	1999	1999	1999	1999	1999	2001	1999 to present	WA did not report 1999–2000
Listeriosis	1991	1991	1994	1991	1992	1991	1991	1991	1991 to present	SA did not report 1991 NT did not report 1991–1993
Salmonellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Shigellosis	1991	2001	1991	1997	1991	1991	1991	1991	1991 to present	NSW did not report 1991–2000 Qld did not report 1991–1996
STEC, VTEC	1999	1999	1999	2002	1999	1999	1999	2001	1999 to present	Qld did not report 1991–2001 WA did not report 1999–2000
Typhoid†	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Quarantinable diseases										
Cholera	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Highly pathogenic avian influenza in humans	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Plague	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rabies	1993	1997	1991	1991	1991	1991	1991	1991	1991 to present	
Severe acute respiratory syndrome	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Smallpox	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Viral haemorrhagic fever	1993	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Yellow fever	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Sexually transmissible infections										
Chlamydial infection (NEC)	1993	1991	1991	1991	1993	1991	1991	1993	1994 to present	NSW did not report 1994–1998
Donovanosis	1991	2002	1991	1991	2002	1993	1991	1991	1991 to present	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Gonococcal infection‡	1991	1993	1991	1991	1991	1991	1991	1991	1991 to present	

Table 8: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory,* *continued*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Sexually transmissible infections, *continued*										
Syphilis – all§	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis < 2 years	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis > 2 years or unspecified duration	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis – congenital	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Vaccine preventable diseases										
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Haemophilus influenzae type b	1991	1991	1991	1991	1991	1991	1991	1994	1991 to present	WA did not report 1991–1993
Influenza (laboratory confirmed)	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Measles	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Mumps	1992	1992	1995	1997	1994	1995	1992	1994	1995 to present	Qld did not report (1992–1996 and 2000)
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Pneumococcal disease (invasive)	2001	2001	2001	1997	2001	2001	2001	2001	2001 to present	
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rubella‖	1991	1991	1993	1991	1993	1995	1992	1994	1993 to present	Tasmania did not report 1993–1994
Rubella – congenital	2003	2003	2003	1997	2003	2003	2003	2003	2003 to present	
Tetanus	1991	1991	1991	1997	1991	1991	1991	1991	1991 to present	Qld did not report 1991–1996
Varicella zoster (chickenpox)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Varicella zoster (shingles)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Varicella zoster (unspecified)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Vectorborne diseases										
Barmah Forest virus infection	1995	1995	1997	1995	1995	1995	1995	1995	1995 to present	
Dengue virus infection	1993	1991	1991	1991	1991	1991	1991	1995	1991 to present	ACT did not report 1991–1992
Arbovirus infection (NEC)¶,**	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	Includes JEV, MVEV and Kunjin 1991–2000
Japanese encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Kunjin virus	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Reported under MVEV in ACT
Malaria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Murray Valley encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Combined with Kunjin in ACT
Ross River virus infection	1993	1993	1991	1991	1993	1993	1991	1991	1993 to present	

Table 8: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory,* *continued*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Zoonoses										
Anthrax	2001	2001	2001	1991	2002	2001	2001	2001	2001 to present	
Australian bat lyssavirus	2001	2001	2001	1998	2001	2001	2001	2001	2001 to present	
Brucellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Leptospirosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Lyssavirus (NEC)	2001	2001	2001	1998	2001	2001	2001	2001	2001 to present	
Ornithosis	1991	2001	1991	1992	1991	1991	1991	1991	1991 to present	NSW did not report 1991–2000 Qld did not report 1997–2001
Q fever	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Tularaemia	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Other bacterial infections										
Legionellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Leprosy	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Meningococcal infection	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Tuberculosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

* Data from the National Notifiable Diseases Surveillance System annual reports from 1991. First full year of reporting to the Department of health and Ageing is shown. Some diseases may have been notifiable to state or territory health departments before the dates shown here.

† 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.

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

** Flavivirus (NEC) replaced arbovirus (NEC) 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) in 2008.

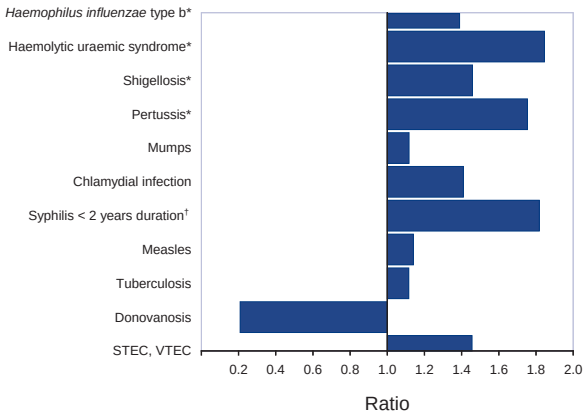
NN Not Notifiable

The major changes in communicable disease notifications in 2008 are shown in Figure 3 as the ratio of notifications in 2008 to the mean number of notifications for the previous 5 years, or in the case of infectious syphilis < 2 year duration, 4 years. Notifications of Murray Valley encephalitis virus infection, *Haemophilus influenzae* type b, haemolytic uraemic syndrome (HUS), shigellosis and pertussis were highest since 2003 and surpassed the expected range (5-year mean plus 2 standard deviations). Notifications of mumps, chlamydial infection, syphilis < 2 years duration, measles, tuberculosis, donovanosis and Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC) were within the historical range.

Data completeness

The case's sex was complete for 99.8% of notifications and age at onset for close to 100% of notifications (Table 9). In 2008, indigenous status was complete for 49.9% of notifications, and varied by jurisdiction. Indigenous status was complete for

Figure 3: Comparison of total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2008, with the previous 5-year mean



* Exceeded 2 standard deviations above the 5 year mean.

† Syphilis < 2 years duration based on a 4-year mean.

Table 9: Completeness of National Notifiable Diseases Surveillance System data received, Australia, 2008, by state or territory*

	State or territory								
	ACT	NSW	NT†	Qld	SA	Tas	Vic	WA	Aust
Total notifications	2,446	39,186	7,034	43,983	12,393	3,601	31,355	20,510	160,508
Sex									
Unknown/ missing	2	121	5	2	1	1	193	1	326
Per cent complete	99.9	99.7	99.9	100.0	100.0	100.0	99.4	100.0	99.8
Age at onset									
Unknown/ missing	0	0	0	0	1	0	47	0	48
Per cent complete	100.0	100.0	100.0	100.0	100.0	100.0	99.9	100.0	100.0
Indigenous status									
Unknown/ missing	2,162	29,290	531	25,841	1,854	1,572	14,592	4,565	80,407
Per cent complete	11.6	25.3	92.5	41.2	85.0	56.3	53.5	77.7	49.9

* Indigenous status is usually obtained from medical notification and completeness varies by disease and by state and territory. This reflects differences in notification requirements (i.e. depending on the jurisdiction, some diseases are primarily or completely notified by pathology laboratories rather than clinicians) and the fact that it is not possible to follow-up all cases for diseases with a large volume of notifications and/or not requiring specific case-based public health action.

† Due to delays in data quality checks, data for the Northern Territory were preliminary at the time of analysis.

92.5% of data reported in the Northern Territory, 85.0% in South Australia and 77.7% in Western Australia. In the remaining jurisdictions, less than 57% of data were complete for indigenous status.

Data completeness on indigenous status also varied by disease as summarised in Appendix 3. There were 5 diseases for which notifications were 100% complete for indigenous status.[10] A further 5 diseases equalled or exceeded 90% completeness for indigenous status. Of the 18 priority diseases agreed to by CDNA and the NSC in 2008 for improving Indigenous identification, seven had an indigenous completeness that exceeded 90% (donovanosis, leprosy, measles, tuberculosis, meningococcal infection, *Haemophilus influenzae* type b, syphilis < 2 years duration). The diseases for which there was less than 90% Indigenous completeness included hepatitis A, pneumococcal disease (invasive), shigellosis, gonococcal infection, and locally-acquired dengue virus infection. HIV, which is one of the priority diseases, is not reported to the NNDSS. In 2008, CDNA set target thresholds of 95% completeness for key diseases and 80% completeness for the remainder of the notifiable diseases.

Bloodborne diseases

Bloodborne viruses reported to the NNDSS include hepatitis B, C, and D. HIV and AIDS diagnoses are reported directly to NCHECR. Information on national HIV/AIDS surveillance can be obtained through the NCHECR web site at www.nchecr.unsw.edu.au

Hepatitis B

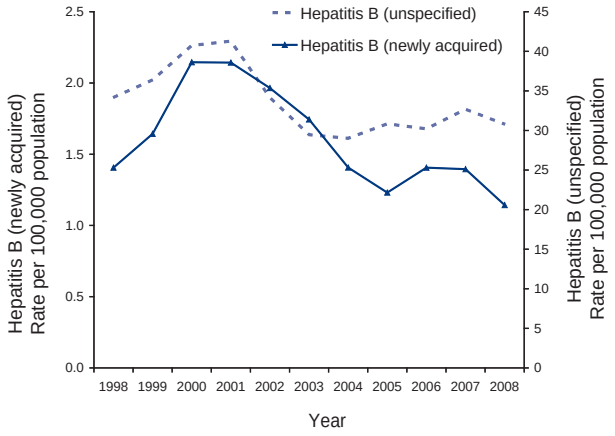
Hepatitis B notifications are classified as either 'newly acquired' (infection acquired within 24 months prior to diagnosis) or 'unspecified' (infection acquired greater than 24 months prior to diagnosis or not able to be specified). The classification of hepatitis B cases is primarily based on serological evidence or evidence of a previously negative test within the 24 months prior to diagnosis. In 2008, there were 6,845 notifications of hepatitis B (both newly acquired and unspecified), corresponding to a rate of 31.9 notifications per 100,000 population. Following a peak of notifications between 2000 to 2001 (42.5 and 43.0 per 100,000 population, respectively), the overall hepatitis B notification rate has declined and remained stable at around 32 notifications per 100,000 population between 2003 and 2008. In 2008, the Northern Territory recorded the highest rate of hepatitis B notifications at 93.3 per 100,000 population, followed by New South Wales (37.2 per 100,000 population) and Victoria (36.1 per 100,000 population).

Since the introduction of the adolescent hepatitis B vaccination program for children aged between 10 and 13 years in 1997,[11] there has been a general decline in overall hepatitis B notification rates amongst the 15–19 and 20–29 year age groups. In 2008, 2 notifications of newly acquired hepatitis B and 24 notifications of hepatitis B (unspecified) were reported in children in the 0–4 year age group, representing 0.8% and 0.4% of hepatitis notifications in these categories respectively. Approximately 93% of the 2008 Australian birth cohort received the full-course of the hepatitis B vaccine.[9, 12–14]

Newly acquired hepatitis B notifications

In 2008, 245 newly acquired hepatitis B notifications (1.1 per 100,000 population) were reported to NNDSS, which was lower than in 2007 (294 notifications; 1.4 per 100,000 population). The 2008 notification rate was the lowest identified over the past 10 years, following a peak of 2.1 notifications per 100,000 population between 2000 and 2001 (Figure 4).

Figure 4: Notification rate for newly acquired hepatitis B* and unspecified hepatitis B,† Australia, 1998 to 2008, by year‡



* Data for newly acquired hepatitis B for the Northern Territory (1998–2004) includes some unspecified hepatitis B cases.

† Data for unspecified hepatitis B for all jurisdictions except the Northern Territory between 1998 and 2004.

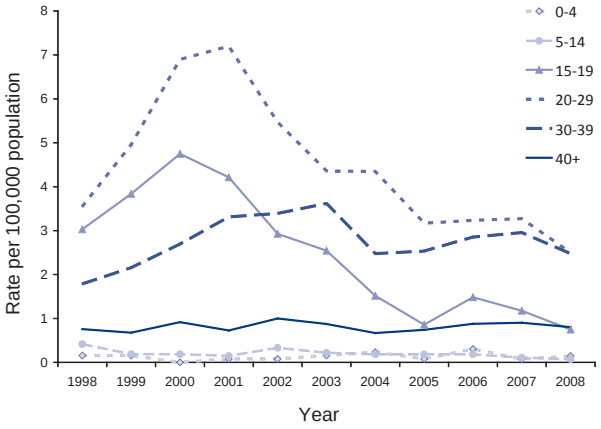
‡ Year of diagnosis for newly acquired hepatitis B and for hepatitis B (unspecified) notifications, and not necessarily year of infection.

Nationally, the proportion of all hepatitis B notifications in 2008 that were documented as newly acquired cases was 3.6%, compared with 4.1% in 2007. The proportion of newly acquired infections compared to total hepatitis B infections varied substantially – Tasmania (17%); Queensland, Victoria and Western Australia (5%); the Northern Territory (4%); South Australia (3%); and the Australia Capital Territory and New South Wales (2%). The highest rates of newly acquired hepatitis B infection were reported from the Northern Territory with 3.6 per 100,000 population and Tasmania (2.4 per 100,000 population). The identification and classification of newly acquired hepatitis B is reliant upon public health follow-up, and the level at which this occurs varies between jurisdictions and over time.

Trends for newly acquired hepatitis B infection by year and age group are shown in Figure 5.

Between 2000 and 2008, the notification rate of newly acquired hepatitis B fell by 85% in the 15–19 year age group. In the 20–29 year age group, there was a steady decline of 66% following a peak of 7.2 notifications per 100,000 population in 2001. The trends in these age groups were seen for both sexes.

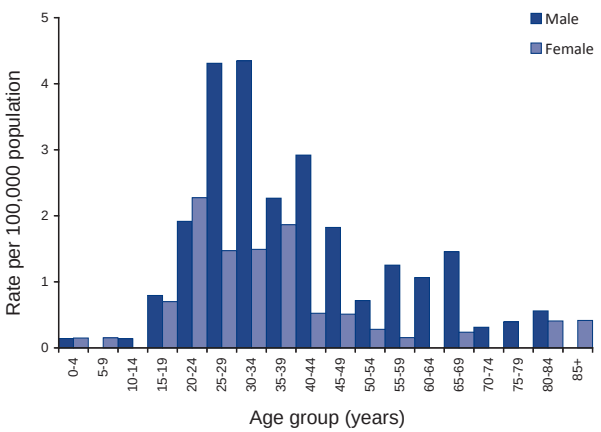
Figure 5: Notification rate of newly acquired hepatitis B,* Australia, 1998 to 2008, by year and age group



* Data for newly acquired hepatitis B for the Northern Territory (1998–2004) includes some unspecified hepatitis B cases.

In 2008, the highest notification rate of newly acquired hepatitis B infection was observed in the 25–29 and 30–34 year age groups amongst males (4.3 per 100,000 population each). Among females, the highest notification rate was in the 20–24 year age group (2.3 per 100,000 population) (Figure 6). Notifications of newly acquired hepatitis B infection were higher amongst males, with a male to female ratio of 2.2:1.

Figure 6: Notification rate for newly acquired hepatitis B, Australia, 2008, by age group and sex



In 2008, the exposure history for notifications of newly acquired hepatitis B was collected by health authorities in South Australia, Tasmania and Victoria and reported to the NCHECR. From 2003 to 2008, approximately half of the annual newly acquired hepatitis B notifications reported injecting drug use. The proportion of diagnoses reporting a history of heterosexual contact with a hepatitis B positive partner decreased from 21% in 2004 to 11% in 2006 and increased to 18% in 2008. The source of exposure to hepatitis B was undetermined in around 20% of cases.[4]

Unspecified hepatitis B notifications

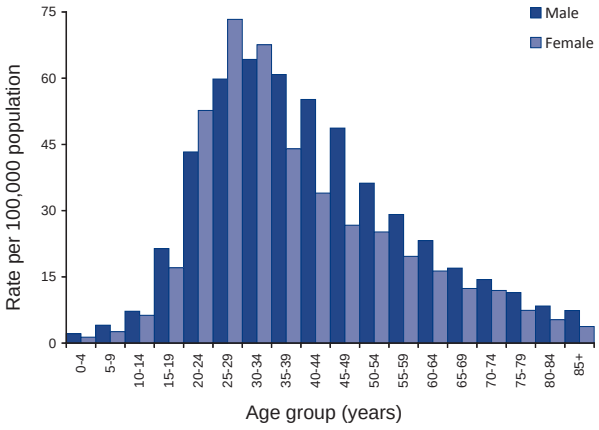
In 2008, a total of 6,600 notifications of unspecified hepatitis B infection were reported to the NNDSS, compared with 6,887 notifications in 2007. The Northern Territory recorded the highest notification rate (89.6 per 100,000 population), compared with other jurisdictions such as New South Wales (36.6 per 100,000 population) and Victoria (34.5 per 100,000 population).

In 2008, sex was recorded in 6,528 of the 6,600 notifications (99%). The male to female ratio of notifications was 1.2:1. Among males, the highest notification rate was amongst the 30–34 year age group (64.3 per 100,000 population) followed by the 35–39 and 25–29 year age groups with rates of 60.8 and 59.8 per 100,000 population respectively. Among females, the highest notification rate was amongst the 25–29 year age group (73.3 per 100,000 population), followed by the 30–34 year age group (67.6 notifications per 100,000 population) (Figure 7).

The notification rates of hepatitis B (unspecified) have generally declined over the past 10 years, despite a peak of 41.3 notifications per 100,000 population in 2001 and a low point of 29.0 per 100,000 population in 2004 (Figure 4). In 2008, the rate of hepatitis B (unspecified) notifications (30.8 per 100,000) was approximately the same as those for 2005–2007 (range 30.2–32.7 per 100,000 population).

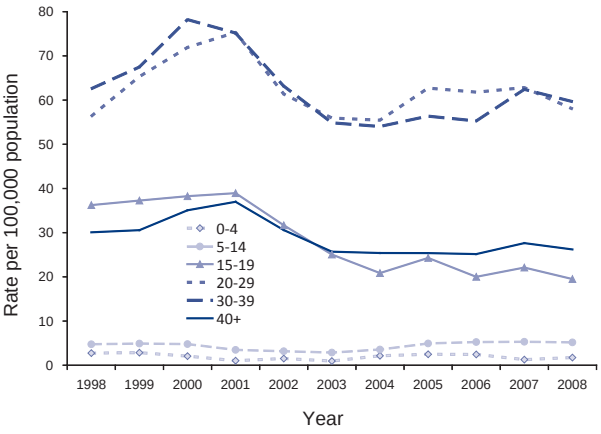
Trends in hepatitis B (unspecified) infection by age group, sex and year are shown in Figure 8. Rates across most age groups decreased in 2008, compared with 2007, with the 15–19 year age group declining by 11.8% (22.1 to 19.5 notifications per 100,000 population). The highest notification rates were amongst the 25–29 and 30–34 year age groups (67.4 and 66.7 per 100,000 population respectively).

Figure 7: Notification rate for unspecified hepatitis B, Australia, 2008, by age group and sex*



* Excluding 72 cases whose sex or age were not reported.

Figure 8: Notification rate for unspecified hepatitis B,* Australia, 1998 to 2008, by year and age group



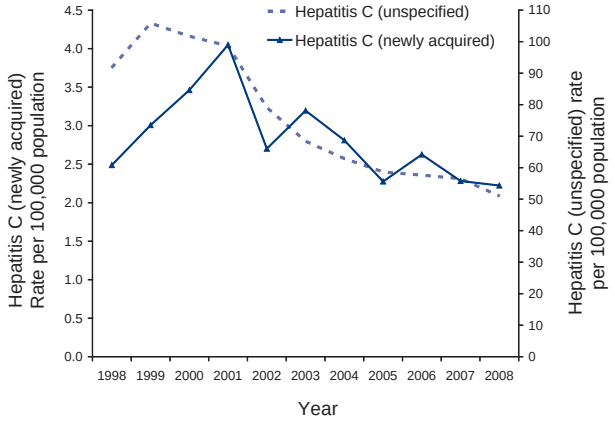
* Data for hepatitis B (unspecified) from all states except the Northern Territory between 1998 and 2004.

Hepatitis C

Hepatitis C notifications are classified as either 'newly acquired' (infection acquired within 24 months prior to diagnosis) or 'unspecified' (infection acquired greater than 24 months prior to diagnosis or not able to be specified). Current testing methods cannot distinguish between newly acquired (incident) and chronic infections (greater than 2 years or unspecified). The identification of newly acquired cases is therefore dependent on evidence of a previously negative test result within 24 months prior to their diagnosis (i.e. seroconversion). Ascertainment of hepatitis C testing histories usually requires active follow-up by public health units.

From 1999 to 2008, total hepatitis C notification rates declined by 51.2% (108.3 to 52.8 notifications per 100,000 population). The greatest reductions were between 2001 and 2002 (20% decline), and are believed to be associated with the detection and accounting of prevalent cases that occurred in the late 1990s through the expansion of testing in high risk groups[15] (Figure 9). The continuing decline in the notification rate may be attributable to reductions in the prevalence of injecting drug use, and risk behaviours related to injecting practices, especially amongst young people, and the implementation of needle exchange programs.[4,15] Changes in hepatitis C laboratory testing practices may have also contributed to the observed decline.

Figure 9: Notification rates for newly acquired hepatitis C* and unspecified hepatitis C,† Australia, 1998 to 2008



* Data for newly acquired hepatitis C from all states and territories except Queensland 1998–2008 and the Northern Territory 1998–2004.

† Data for unspecified hepatitis C provided from Queensland (1998–2008) and the Northern Territory (1998–2004) include both newly acquired and unspecified hepatitis C notifications.

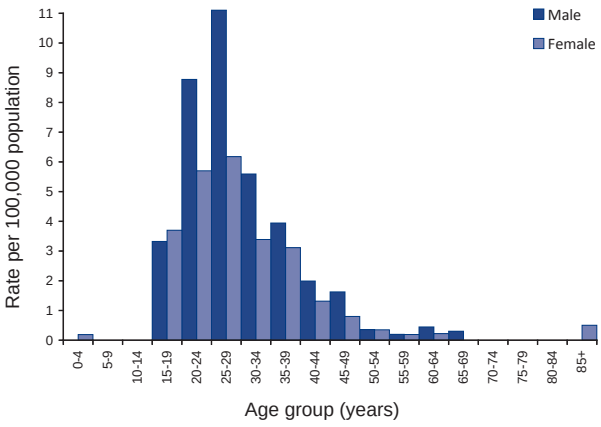
Newly acquired hepatitis C notifications

Notifications of newly acquired hepatitis C were received from all jurisdictions except Queensland, where all cases of hepatitis C, regardless of whether they are newly acquired, are reported as unspecified hepatitis C. There were 381 newly acquired hepatitis C notifications reported in 2008 (385 notifications in 2007), giving a notification rate of 2.2 per 100,000 population (Figure 9).

As a proportion of all hepatitis C notifications in 2008, 3.4% were identified as newly acquired infections, compared with 3.1% in 2007. Amongst jurisdictions, the proportion of newly acquired infections compared with total hepatitis C infections varied substantially – South Australia (11%), Western Australia (8%), Tasmania (7%), Victoria (6%), the Australia Capital Territory and the Northern Territory (3%), and New South Wales (1%). The highest rates of newly acquired hepatitis C infection were reported in Tasmania (4.8 per 100,000 population), Western Australia (4.7 per 100,000 population) and South Australia (4.1 per 100,000 population). The identification and classification of newly acquired hepatitis C is reliant upon public health follow-up to identify testing and clinical histories. The level of case follow-up and method varies among jurisdictions.

Notification rates of newly acquired hepatitis C were highest in males in the 25–29 and 20–24 year age groups (11.1 and 8.8 per 100,000 population respectively), with peaks in females also occurring for the same 5 year age groups (6.2 and 5.7 per 100,000 population respectively) (Figure 10).

Figure 10: Notification rate for newly acquired hepatitis C, Australia,* 2008, by age group and sex†



* Data from all states and territories except Queensland.

† Excludes 1 case whose sex was not reported.

Trends in the age distribution of newly acquired hepatitis C infection are shown in Figure 11. While rates for individual age groups can vary markedly from year to year, there is a general downward trend in the 15–19 and 20–29 year age groups. Overall, the annual rates in the other age groups are similar to trends in previous years.

Enhanced surveillance data for newly acquired infections in 2008 were collected in all jurisdictions except Queensland. Of the newly acquired hepatitis C notifications within these jurisdictions, 88% had exposure history information recorded (335 of 381) (Table 10). Approximately 78% of these hepatitis cases were amongst people with a

Table 10: Newly acquired hepatitis C notifications, selected jurisdictions,* 2008, by sex and exposure category in the 24 months prior to diagnosis

Exposure category	Number of exposure factors reported			Percentage§ of total cases‖ (*n*=335)
	Male	Female	Total‡	
Injecting drug use	95	54	150	44.8
Imprisonment	72	12	84	25.1
Skin penetration procedure	50	39	89	26.6
Tattoos	*35*	*19*	*54*	*16.1*
Ear or body piercing	*14*	*18*	*32*	*9.6*
Acupuncture	*1*	*2*	*3*	*0.9*
Healthcare exposure	6	10	16	4.8
Surgical work	*5*	*5*	*10*	*3.0*
Major dental surgery	*1*	*4*	*5*	*1.5*
Blood/tissue recipient	*0*	*1*	*1*	*0.3*
Sexual contact	15	25	40	11.9
Household contact	11	9	20	6.0
Needlestick or biohazardous injury¶	3	3	6	1.8
Other	3	3	6	1.8
Risk factor unable to be determined	4	3	7	2.1
Total number of exposure factors reported†	259	158	418	–

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

† More than 1 exposure category for each case could be recorded.

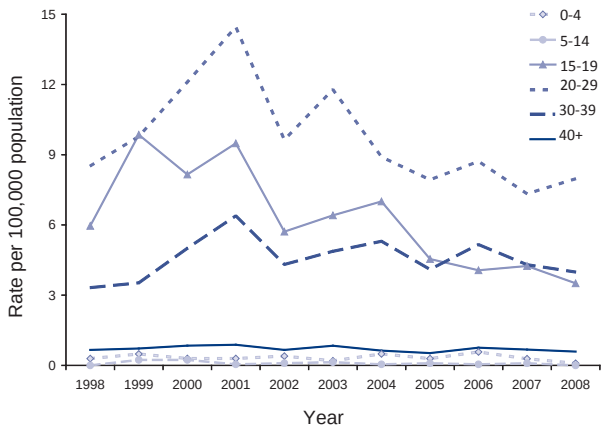
‡ Total includes notifications in cases whose sex was not reported.

§ The denominator used to calculate the percentage is based on the total number of cases with exposure information recorded and as more than 1 exposure category for each case could be recorded, the total percentage does not equate to 100%.

‖ Total number of cases where exposure history reported.

¶ Includes both occupational and non-occupational exposures.

Figure 11: Notification rate for newly acquired hepatitis C, Australia,* 1998 to 2008, by age group and year



* Data from all states and territories except Queensland (1998–2008) and the Northern Territory (1998–2004).

history of injecting drug use (45% with injecting drug use in the 24 months prior to diagnosis), and 25% were amongst persons detained in a correctional facility within the 24 months prior to their diagnosis. Screening rates are higher in the prison entry population than the general population. A screening survey of prison entrants conducted over a 2-week period in 2007 found that the prevalence of hepatitis C, based on hepatitis C antibody detection, was 35% amongst this population.[16]

Unspecified hepatitis C notifications

In 2008, 10,938 unspecified hepatitis C infections were notified to the NNDSS (51.0 notifications per 100,000 population) compared with 11,905 notifications in 2007 (56.5 notifications per 100,000 population).

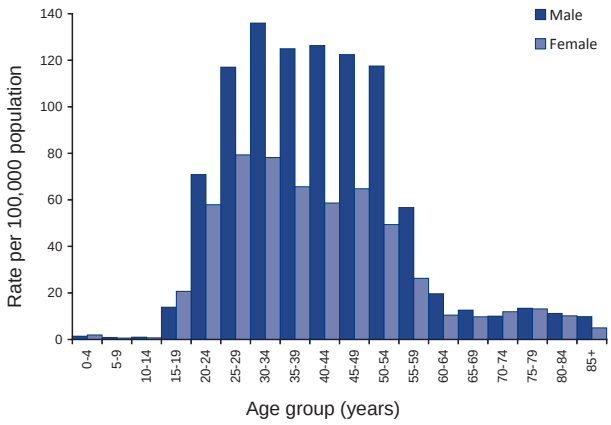
The national notification rate for unspecified hepatitis C infection declined from 105.8 per 100,000 population in 1999 to 51.0 per 100,000 population in 2008 (Figure 9). Changes in surveillance practices; increased duplicate notification checks;

changes in rates of testing; and the Northern Territory separately reporting newly acquired hepatitis C notifications from 2003, may account for some of the decrease in unspecified hepatitis C notifications since 2000, in addition to broader reductions in the prevalence of injecting drug use.[4,15]

In 2008, the Northern Territory continued to have the highest notification rate (101.0 per 100,000 population) followed by Tasmania (65.1 per 100,000 population), Western Australia (57.2 per 100,000 population) and the Australian Capital Territory (56.4 per 100,000 population). Queensland's rate was also high, at 61.3 per 100,000 population, however this included both newly acquired and unspecified cases.

The male to female ratio remained consistent with historical trends at 1.7:1. The highest notification rate occurred in the 30–34 year age group (136.0 per 100,000 population) amongst males and in the 25–29 and 30–34 year age groups (79.4 and 78.2 per 100,000 population respectively) amongst females (Figure 12).

Figure 12: Notification rate for unspecified hepatitis C,* Australia, 2008, by age group and sex†

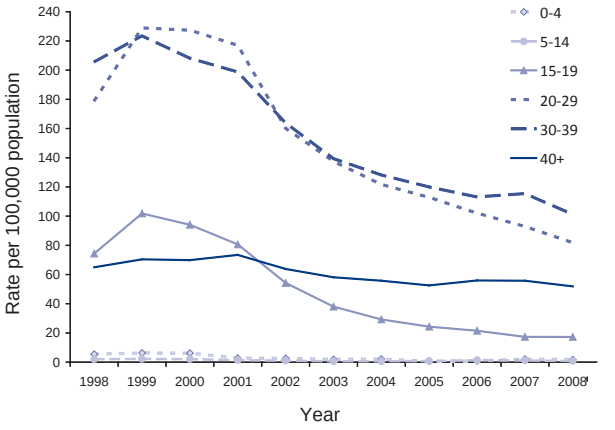


* Data provided from Queensland includes both newly acquired and unspecified hepatitis C notifications.

† Excludes 38 cases whose sex was not reported.

Trends in the age distribution of unspecified hepatitis C infection are shown in Figure 13. From 2001 to 2008, the notification rates of unspecified hepatitis C declined by 79% amongst the 15–19 year age group, by 62% amongst the 20–29 year age group and by 49% in the 30–39 year age group. Trends in the 0–4 and the 40 years and over age groups have remained relatively stable over the past 10 years.

Figure 13: Notification rate for unspecified hepatitis C,* Australia, 1998 to 2008, by age group



* Data provided from Queensland (1998–2008) and the Northern Territory (1998–2004) include both newly acquired and unspecified hepatitis C notifications.

Although initial infection with the hepatitis C virus is asymptomatic or mildly symptomatic in more than 90% of cases, approximately 50%–80% of cases will go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.[17] In 2008, it was estimated that 284,000 people living in Australia had been exposed to the hepatitis C virus. Of these, approximately 162,000 had chronic hepatitis C infection and early liver disease, and 44,000 had chronic hepatitis C infection and moderate liver disease associated with chronic hepatitis C infection; 5,700 were living with hepatitis C related cirrhosis; and 72,100 had cleared their infection.[4]

Hepatitis D

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.[17] 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 group at risk for hepatitis D.

In Australia, the rate of hepatitis D remains low. In 2008, there were 42 notifications of hepatitis D, compared with 34 notifications in 2007, giving a notification rate of 0.2 per 100,000 population. The male to female ratio was 4.3:1. Of the 42 notifications, 14 were reported from New South Wales, 13 from Victoria, 7 from Queensland, 6 from Western Australia and 1 case from the Northern Territory.

Gastrointestinal diseases

In 2008, 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* (STEC) infections and typhoid.

Overall notifications of gastrointestinal diseases in 2008 decreased 10% from 30,325 in 2007 to 27,308 in 2008. However, notifications of hepatitis E, HUS, shigellosis and typhoid were notably increased compared with the 5-year mean (exceeded the mean by more than 2 standard deviations).

OzFoodNet, Australia's enhanced foodborne disease surveillance network, monitors the incidence of diseases caused by pathogens commonly transmitted by food using population-based passive and enhanced surveillance for notifiable gastrointestinal diseases and for outbreaks of gastroenteritis and enteric disease. In 2008, OzFoodNet aggregated and analysed data from the NNDSS supplemented by enhanced surveillance data from OzFoodNet sites on the following 9 diseases or conditions, a proportion of which may be transmitted by food: non-typhoidal salmonellosis; campylobacteriosis infections (except in New South Wales); listeriosis; shigellosis; typhoid; STEC infections; botulism; HUS; and hepatitis A. The data and results from these analyses are summarised in the following sections but are reported in more detail elsewhere. [18]

Botulism

Foodborne botulism arises from the consumption of a food that is contaminated with pre-formed *Clostridium botulinum* toxin.

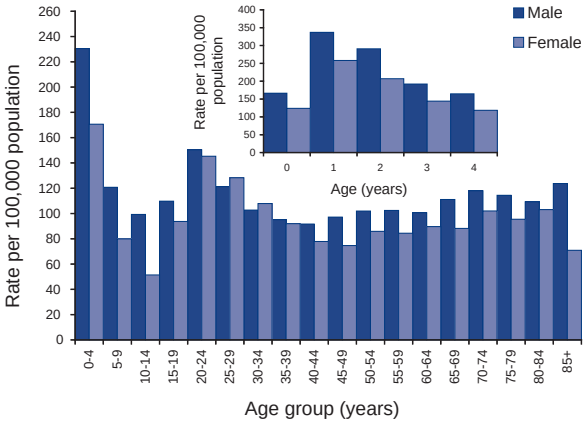
No cases of botulism were reported to NNDSS in 2008, compared with 1 case in 2007.

Campylobacteriosis

Campylobacteriosis is notifiable in all Australian jurisdictions, except New South Wales.

In 2008, there were 15,535 notifications of campylobacteriosis, a 9% decrease compared with the 16,996 notifications reported in 2007. The national rate of campylobacteriosis notifications in 2008 was 107.5 per 100,000 population. The lowest and highest rates of *Campylobacter* notification were in Western Australia (84.2 per 100,000 population) and in South Australia (124.2 per 100,000 population) respectively. The highest age specific notification rates of *Campylobacter* were amongst males and females aged 0–4 years. Amongst children aged under 5 years, the highest notification rate was in boys aged 1 year (336.9 per 100,000 population) (Figure 14).

Figure 14: Notification rate for campylobacteriosis, Australia, 2008, by age group and sex, and inset: age and sex in children aged under 5 years



Cryptosporidiosis

In 2008, 2,005 notifications of cryptosporidiosis were reported to the NNDSS, with a national notification rate of 9.4 per 100,000 population, a 29% decrease over the number of notifications reported in 2007.

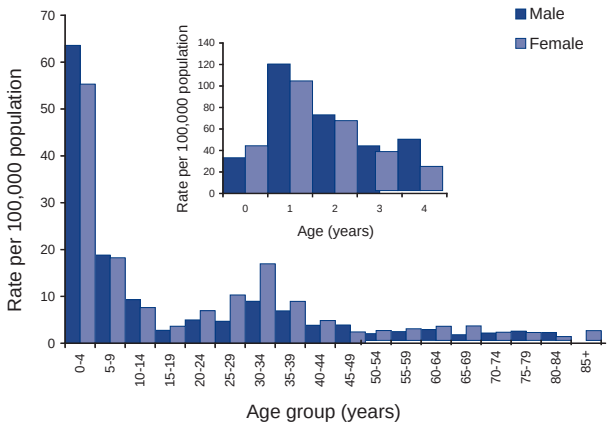
The highest notification rates of cryptosporidiosis were reported in the Northern Territory (46.4 per 100,000 population) and Queensland (16.2 per 100,000 population).

Fifty-three per cent of all cryptosporidiosis notifications in 2008 were in children aged under 10 years, the majority of which were male (54%) (Figure 15). Overall, the number of cryptosporidiosis notifications were similar between males (49%) and females (51%), however, the number of notifications was higher among females (54%) (Figure 15), while in the 20–39 year age range (62%) than in males of the same age.

Haemolytic uraemic syndrome

During 2008, there were 31 cases of HUS notified to NNDSS, with a rate of 0.1 per 100,000 population, which is the same as the mean annual notification rate between 2003 and 2007. Over

Figure 15: Notification rate for cryptosporidiosis, Australia, 2008, by age group and sex, and inset: age and sex in children aged under 5 years



half of these notifications were reported from New South Wales (17 notifications). The median age of notifications was 14 years, with a range of 0–83 years. Similar to previous years, the highest notification rate was in children aged 0–4 years, with 11 of the 31 notifications in this age group (0.8 notifications per 100,000 population). [18]

Cases of HUS may be due to causes other than Shiga toxin-producing *E. coli*, including other non-foodborne pathogens and genetic predisposition. In 2008, an antecedent STEC infection was reported for 52% (16/31) of notifications. In 2008, 1 case of HUS was known to be due to a non-bacterial cause, 2 cases resulted from *Streptococcus pneumoniae* infection, and in the remaining 11 cases no aetiology was reported.

Hepatitis A

Notifications of hepatitis A declined in 2008, with 276 notifications compared with a mean of 306 per year between 2003 and 2007 (Table 11 and Figure 16).

In 2008, the median age of notifications was 24 years (range 1–97 years) of which 57% (158/276) of notifications were male.

Overseas travel was the most frequently reported risk factor for infection with hepatitis A in 2008, with 56% (154/276) of notifications reporting overseas travel (Table 11). The most commonly reported overseas travel destinations were India (29), Indonesia (11) and Pakistan (8).

Indigenous status was known for 89% of notifications in 2008. The proportion of cases of hepatitis A amongst Indigenous persons declined from a mean of 14% (167/1,193) of notifications for the years 2003–2006 to 1.2% (3/245) of notifications in 2008 (Table 12). This marked decrease in the number and proportion of cases that were Indigenous is likely to be due in part to targeted vaccination programs for Indigenous children commencing in Queensland in 1999, and the provision of free hepatitis A vaccine for all Indigenous children in South Australia, Western Australia and the Northern Territory from 2006 (Figure 16).[19]

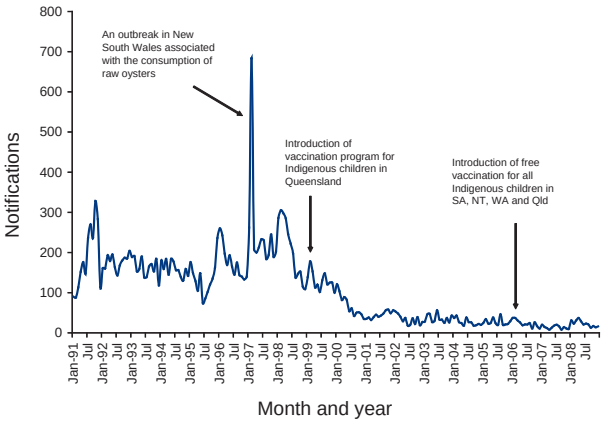
Table 11: Notifications of hepatitis A, Australia, 2008, by state or territory

State or territory	Number of cases	Number acquired overseas	Per cent acquired overseas
ACT	5	3	60
NSW	69	53	77
NT	3	2	67
Qld	71	30	42
SA	20	13	65
Tas	1	0	0
Vic	85	46	54
WA	22	7	32
Total	276	154	56

Table 12: Hepatitis A notifications, Australia, 2003 to 2008, by indigenous status

Year	Indigenous		Non-Indigenous		Unknown	
	n	%	n	%	n	%
2003	53	12	325	76	53	12
2004	37	12	251	79	31	10
2005	48	15	232	71	46	14
2006	28	10	218	78	35	12
2007	0	0	146	88	19	12
2008	3	1	243	88	30	11

Figure 16: Trends in notifications of hepatitis A, Australia, 1991 to 2008, by month of diagnosis



Hepatitis E

In 2008, there were 44 notifications of hepatitis E, compared with 18 notifications in 2007 and a mean of 22 cases per year between 2003 and 2007. Fourteen cases were reported from both New South Wales and Victoria, 7 cases from Queensland, 6 cases from Western Australia and three from the Northern Territory.

In 2008, 68% (30/44) of cases were known to have been acquired overseas. The median age of cases was 28 years (range 12–78 years), possibly reflecting higher rates of overseas travel in younger adults.

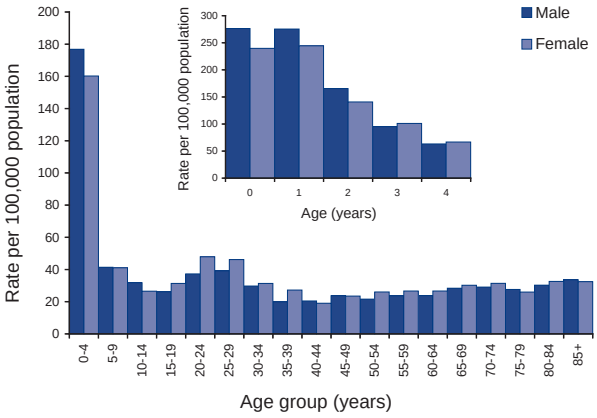
Listeriosis

In 2008, 68 cases of *Listeria monocytogenes* infection were reported to the NNDSS, a crude notification rate of 0.3 per 100,000 population including 12 deaths. The 2008 notification rate was consistent with the 5-year historical mean annual notification rate (0.3 per 100,000 population). Similar to previous years, 22% of cases (15/68) were pregnancy-associated infections, occurring in pregnant women or newborn babies. In 2008, 47% (25/53) of the non-pregnancy related cases were female. Forty-nine per cent (33/68) of notifications were in people aged 60 years or more. The highest age specific notification rate was in people aged 85 years or more (1.9 per 100,000 population, 7 cases). Seven per cent (1/15) of pregnancy related cases and 21% (11/53) of non-pregnancy associated cases in 2008 were fatal.[18]

Salmonellosis (non-typhoidal)

In 2008, there were 8,310 cases of *Salmonella* infection corresponding to a notification rate of 38.8 per 100,000 population and similar to the 5-year mean of 8,210 cases per year. Notification rates amongst jurisdictions ranged from 31.1 per 100,000 population in Victoria to 226.1 per 100,000 population in the Northern Territory. Approximately half (49%) of *Salmonella* notifications were in males. The highest age specific rate of *Salmonella* infection was 169.3 per 100,000 population in children aged from 0–4 years, with the highest rates in those aged 2 years or over (Figure 17).

Figure 17: Notification rate for *Salmonella* infection, Australia, 2008, by age and sex



In 2008, the most commonly notified *Salmonella* serotype was *S.* Typhimurium, which was responsible for approximately 42% of all notified infections. *S.* Typhimurium phage types 135, 44, 170/108 and 9 were commonly reported, representing four of the top 5 *Salmonella* infections nationally.[18]

In 2008, OzFoodNet reported 35 outbreaks of foodborne salmonellosis affecting 486 people. Individual notifications of salmonellosis are very rarely attributed to a food vehicle.

Shigellosis

In 2008, there were 828 cases of shigellosis reported to the NNDSS compared with 602 in 2007. The 2008 notification rate was 3.9 per 100,000 population, which was higher than the mean annual notification rate of 2.8 notifications per 100,000 between 2003 and 2007. As in previous years, the highest notification rate was in the Northern Territory, with 79.6 per 100,000 population compared with an average rate of 71.9 per 100,000 population per year between the years 2003 and 2007.[18]

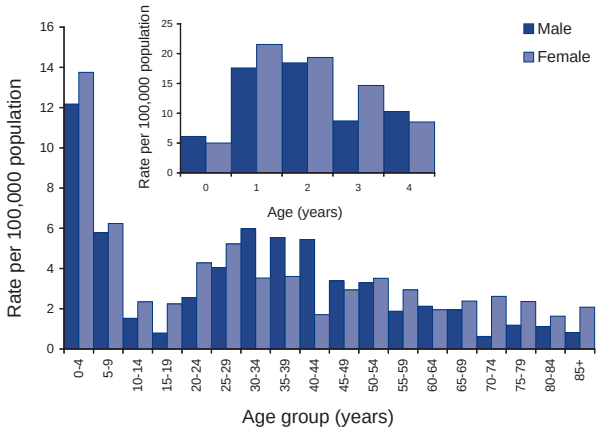
The highest age specific notification rates were amongst males and females aged 0–4 years, with age specific rates of 12.5 and 13.9 notifications per 100,000 population, respectively (Figure 18). Overall in 2008, 50% of all shigellosis notifications were male.

Notification rates were higher amongst men aged between 30 and 44 years than in females of the same age, which may in part be explained by the outbreak of shigellosis amongst men who reported sex with other men as a risk factor in 2008.[18]

Rates of shigellosis in Australia are higher amongst Indigenous people than in non-Indigenous people. In 2008, there were 318 notifications of shigellosis amongst Indigenous people (38% of notifications), with an age standardised rate of 58.9 per 100,000 population. Indigenous status information in 2008 was 81% complete. Shigellosis is one of the 18 priority diseases for which the NSC has agreed to improve Indigenous status reporting.

The most common biotypes of shigellosis in 2008 were *Shigella sonnei* biotype a (28%) and *Shigella sonnei* biotype g (22%). These 2 biotypes were also the most common in 2007, but different to 2006 when the most common biotype was *Shigella flexneri* 4a mannitol negative.[18]

Figure 18: Notification rate for shigellosis, Australia, 2008, by age and sex



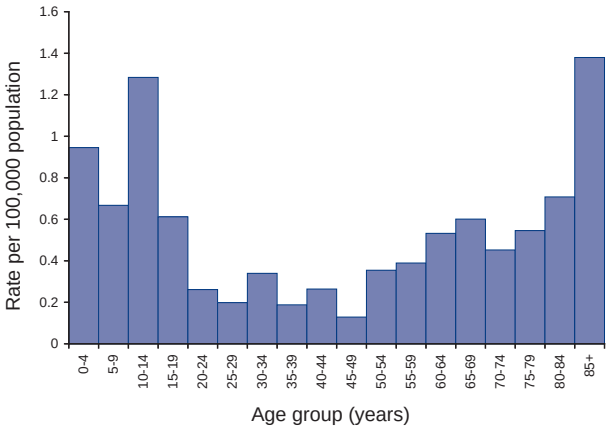
Shiga toxin-producing *Escherichia coli*

In 2008, there were 106 cases of STEC, corresponding to a rate of 0.5 notifications per 100,000 population. This was similar to the mean annual notification rate of 0.4 notifications per 100,00 population between 2003 and 2007.[18]

In 2008, 51.9% of cases were female and the median age of cases was 24 years (range 0–89 years). The highest age specific notification rate for STEC was amongst people over the age of 85 years. Other peaks were observed in the 0–4 and 10–14 year age groups (Figure 19).

South Australia reported 36% (39/106) of all STEC notifications, followed by Queensland

Figure 19: Notifications of Shiga toxin-producing *Escherichia coli*, Australia, 2008, by age group



(35%, 37/106), New South Wales (18%, 19/106) and Victoria (10%, 11/106). There were no cases notified in the Australian Capital Territory, the Northern Territory, Tasmania or Western Australia in 2008.

Rates of STEC infection are strongly influenced by jurisdictional practices regarding the screening of stool specimens.[20] In particular, South Australia routinely tests all bloody stools by polymerase chain reaction (PCR) for gene coding for Shiga toxins and other virulence factors, contributing to the higher rates of detection of infection for this State. Queensland conducts routine culture on bloody stools. If there is no growth in culture, PCR is not performed, instead, enzyme-linked immunosorbent assay for Shiga toxin is conducted on the specimen. In New South Wales, some routine screening for STEC genes in stools containing microscopic blood is conducted in the Hunter–New England region, but not elsewhere. In Western Australia, 2 pathology laboratories routinely screen bloody stools with either sorbitol Maconkey agar culture or tissue culture. Other jurisdictions do not routinely screen for STEC.

Typhoid

There were 105 cases of *Salmonella* Typhi infection (typhoid) notified during 2008. This equated to a notification rate of 0.5 per 100,000 population, slightly higher than the annual rate of 0.3 per 100,000 between 2003 and 2007. Cases were reported from all Australian states and territories except for the Australian Capital Territory and Tasmania.

Overseas travel was the primary risk factor for typhoid in Australia in 2008 with 92% (97/105) of notifications known to have been acquired

overseas. India was the most frequently reported country for overseas acquired cases, with 49% (48/97) of notifications, followed by Bangladesh, Indonesia, and Pakistan, each of which were reported as a travel destination for 9% (9/97) of overseas-acquired notifications. The highest typhoid notification rates were in the 20–24 year age group (1.4 per 100,000 population) and the 25–29 year age group (1.1 per 100,000 population) (Figure 20), reflecting higher rates of overseas travel in these age groups.

Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and notifiable in Australia and to the WHO in 2008 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).

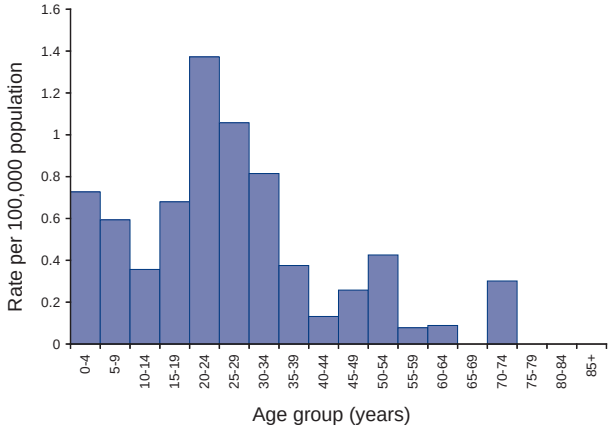
Cholera, plague, rabies, smallpox, yellow fever, SARS, HPAIH and viral haemorrhagic fevers are of international public health importance as they continue to occur around the world. Travellers are advised to seek information on the risk of contracting these diseases at their destinations and to take appropriate measures. More information on quarantinable diseases and travel health can be found on the following web sites:

Australian Government Department of Health and Ageing web site at: http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm

Smartraveller: The Australian Government's travel advisory and consular assistance service at: http://www.smartraveller.gov.au/

There were no cases of plague, rabies, smallpox, yellow fever, SARS, HPAIH or viral haemorrhagic fevers reported in Australia in 2008. Table 13 provides information on the occurrence of quarantinable diseases in Australia.

Figure 20: Notifications of typhoid, Australia, 2008, by age group



Cholera

In 2008, 4 cases of cholera were notified in Australia, two from New South Wales and two from Western Australia and all were acquired overseas. One case had travelled to Singapore, 1 case had travelled to the Philippines and 2 cases had travelled to India. All cases of cholera reported since the commencement of the NNDSS in 1991 have

Table 13: Australia's status for human quarantinable diseases, 2008

Disease	Status	Date of last record and notes
Cholera	Free	Small number of cases are reported annually[22]
Plague	Free	Last case recorded in Australia in 1923[23]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990[24]
Smallpox	Free	Last case recorded in Australia in 1938[25]
Yellow fever	Free	No cases recorded on shore in Australia – 5 occasions on which vessels arrived in Australian ports 1892–1915[23]
Severe acute respiratory syndrome	Free	Last case recorded in Australia in 2003[26]
Highly pathogenic avian influenza in humans	Free	No cases recorded[27]
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded[28]
Marburg	Free	No cases recorded[28]
Lassa	Free	No cases recorded[28]
Crimean–Congo	Free	No cases recorded[28]

been acquired outside Australia except for 1 case of laboratory-acquired cholera in 1996 and 3 cases in 2006. There have been 16 cases of cholera notified between 2003 and 2007.[28]

Sexually transmissible infections

In 2008, the sexually transmissible infections (STIs) reported to NNDSS were chlamydial infection, donovanosis, gonococcal infection and syphilis. Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance Programme (AGSP), which is a network of specialist laboratories monitoring antimicrobial susceptibility patterns of infection; and the NCHECR, which maintains the National HIV Registry and the National AIDS Registry.

Since 2004, 2 categories of non-congenital syphilis have been reported: infectious syphilis (primary, secondary and early latent) of less than 2 years duration; and syphilis of greater than 2 years or unknown duration. The NNDSS also received reports on cases of congenital syphilis. These conditions were notified in all states and territories, except in South Australia where cases of syphilis of greater than 2 years or unknown duration were not reported to the NNDSS.

The national trends in the number and rates of STI notifications reported to the NNDSS between 2003 and 2008 are shown in Table 7. 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,[29, 30] more targeted screening, the use of less invasive and more sensitive diagnostic tests, as well as periodic public awareness campaigns may contribute to changes in the number of notifications over time. For some diseases, changes in surveillance practices may also need to be taken into account when interpreting national trends.

Indirect age standardised notification rates, using the method described by the Australian Institute of Health and Welfare,[31] were calculated for Indigenous and non-Indigenous notifications for jurisdictions that had indigenous status data completed in more than 50% of notifications. Where the indigenous status was not completed, notifications were counted as non-Indigenous when analysing these notifications. These data however, need to be interpreted with caution as STI screening occurs predominately in specific high risk groups, including in Indigenous populations; and Indigenous and non-Indigenous population distributions and proportions vary widely for each jurisdiction. Previous research into high rates of STIs amongst the Indigenous population in the Northern Territory established that the disparity in notification rates could be attributed to more targeted screening programs and to poorer access to primary health care services, rather than increased levels of sexual activity amongst Indigenous people.[32, 33] Similarly, rates between females and males need to be interpreted with caution as rates of testing for STIs and health care-seeking behaviours differ between the sexes.

Notifications of chlamydial, gonococcal and non-congenital syphilis infections were excluded from analysis where the case was aged 13 years or less and the infection was deemed to be non-sexually acquired, e.g. perinatally acquired infections.

Chlamydial infection

Chlamydial infection continues to be the most commonly notified disease in 2008. A total of 58,484 notifications of chlamydial infection were received, corresponding to a rate of 273 per 100,000 population. This represents an increase of 10% on the rate reported in 2007 (247 per 100,000 population). The rate of chlamydial notifications has continued to increase since surveillance of the condition commenced in 1991 in all jurisdictions, except New South Wales where it became notifiable in 1997. Between 2003 and 2008, chlamydial infection notification rates increased from 152.9 to 272.9 per 100,000 population, an increase of 78% (Table 7). While the prevalence of chlamydial infection varies by age group and other demographic and behavioural factors, no major section of the population is spared.[34]

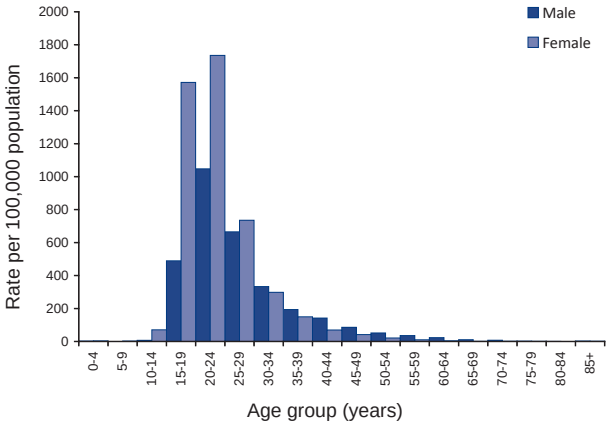
Chlamydial infection notification rates were substantially higher than the national average in the Northern Territory (1,044 per 100,000 population), Western Australia (397.9 per 100,000 population) and Queensland (353.9 per 100,000 population) (Table 6). At a regional level, chlamydial notification rates were highest in the Barkly and Central NT Statistical Subdivisions of the Northern Territory (range: 1144.6 to 2121.1 notifications per 100,000 population), noting that notification rates in geographic areas where the estimated residential population and case numbers are small, should be interpreted with caution. In the Statistical Divisions of Far North in Queensland and Pilbara in Western Australia and the Northern Territory Statistical Subdivisions of Alligator, East Arnhem, Finniss and the Lower Top End NT, notifications rates were also substantially higher than the national rate (range: 740.9 to 1144.5 notifications per 100,000 population) (Map 2).

In 2008, notification rates of chlamydial infection in males and females were 221.4 and 322.8 per 100,000 population respectively. When compared with 2007, notification rates increased by 11% in males and 10% in females. The male to female ratio in 2008 was 0.7:1, which is similar to previous years. Rates in females markedly exceeded those in males, especially in the 15–19 and 20–24 year age groups with ratios of 0.3:1 and 0.6:1 respectively (Figure 21).

Trends in age and sex specific notification rates between 2003 and 2008 show increases across all age ranges, especially between 15 and 29 years in both males and females (Figure 22). Since 2003, the highest notification rate increases occurred in males in the 20–24 year age group (80%) and amongst females in the 15–19 (90%) and 20–24 year age groups (70%).

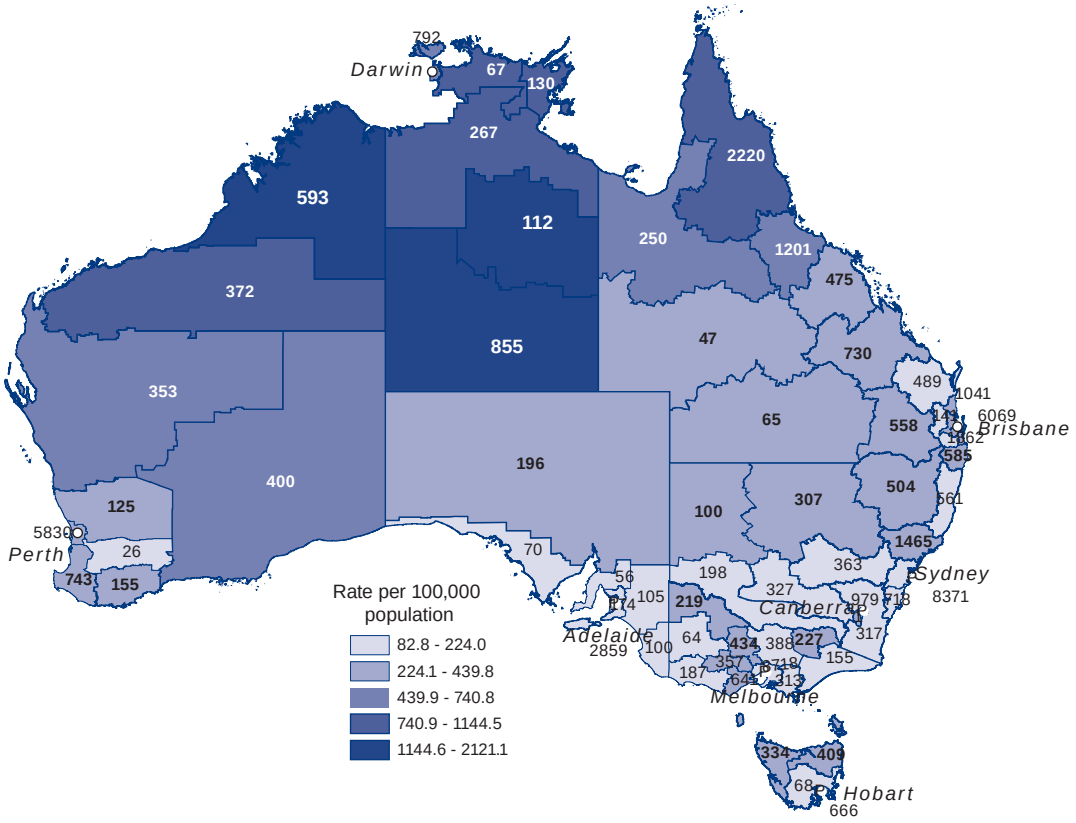
From 2003 to 2008 the rates of chlamydial infection diagnosis have increased in both Indigenous and non-Indigenous populations. Nationally in 2008, data on indigenous status were complete in 48% of notifications, higher than the preceding 5-year average of 43% (range: 40%–45%). Six jurisdictions had greater than 50% completeness of the indigenous status field: the Northern

Figure 21: Notification rate of chlamydial infection, Australia, 2008, by age group and sex*



* Excludes 114 notifications whose age or sex was not reported.

Map 2: Notification rates and counts* for chlamydial infection, Australia, 2008, by Statistical Division of residence and Statistical Subdivision of residence for the Northern Territory



* Numbers shown in the Statistical Divisions and Statistical Subdivisions represent the count of notifications.

Notification rates in geographic areas where estimated residential population and case numbers are small should be interpreted with caution.

Territory, Queensland, South Australia, Victoria, Tasmania and Western Australia. Among these jurisdictions, the combined age standardised notification rate was 1,134 per 100,000 in the Indigenous population and 279 per 100,000 in the non-Indigenous population.

The age standardised rate ratio of Indigenous to non-Indigenous chlamydial infection notifications across these jurisdictions was 4:1. Between 2006 and 2008, rates of chlamydial infection notifications in the Indigenous population increased by 7% in the Northern Territory and decreased by 34% in South Australia for the same period (Figure 23). Nationally, the disparity in notification rates between Indigenous and non-Indigenous populations has improved substantially since 2000. It should be noted that indigenous status identification completeness in the notification data varies both across years and by jurisdiction.

Although surveillance data continue to show substantial increases in chlamydial infection notifications nationally, a large proportion of cases with genital chlamydial infections are asymptomatic.[17] Enhanced surveillance of chlamydial notifications undertaken in Tasmania during 2008 identified that 57% of males presented as asymptomatic compared with 70% of females (personal communication, David Coleman, Tasmanian Department of Health and Human Services, 2 July 2010). A paper published on enhanced chlamydial surveillance data in Tasmania for the period 2001 to 2007 also noted that females were more likely to have been tested for chlamydial infection as a result of screening, and males were more likely to have been tested when presenting with symptoms or as a result of contact tracing.[35] Therefore, notification rates for this disease are particularly susceptible to overall rates of testing as well as targeted testing in certain high risk population sub-groups.

Figure 22: Trends in notification rates of chlamydial infection in persons aged 10–39 years, Australia, 2003 to 2008, by age group and sex

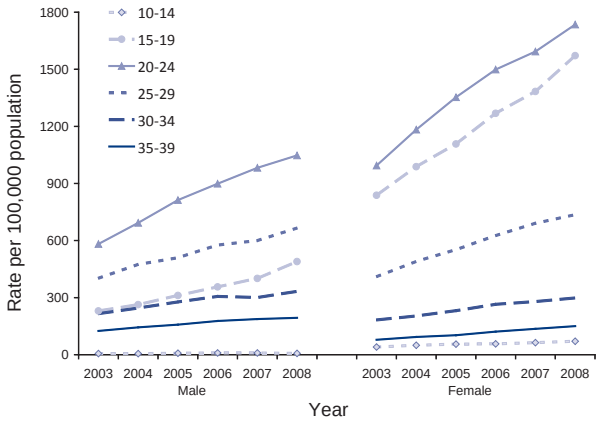
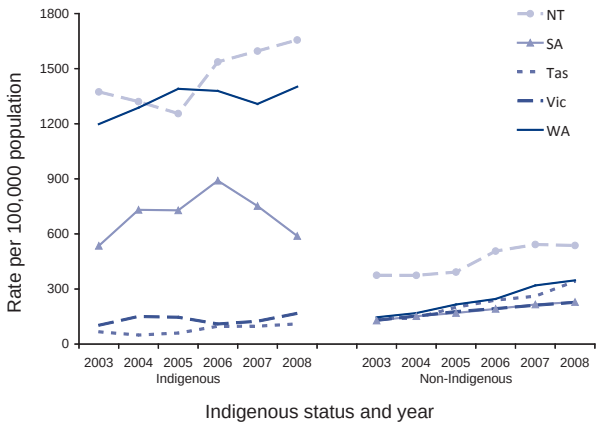


Figure 23: Trends in notification rates of chlamydial infection, selected states and territories,* 2003 to 2008, by indigenous status

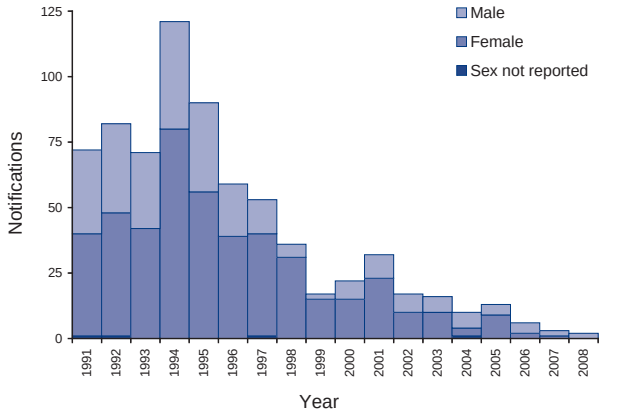


* States and territories in which Indigenous status completeness was reported for more than 50% of cases over a 5 year period.

Donovanosis

Donovanosis is a sexually transmissible infection characterised by a chronic ulcerative genital disease. Although it is now relatively uncommon, it is a disease of public health importance in Australia because it predominantly occurs in Indigenous communities and has been identified as a potential co-factor in HIV transmission. Donovanosis has been targeted for elimination in Australia through the National Donovanosis Elimination Project.[36] In 2008, 2 notifications in Indigenous males, one from Queensland and one from the Northern Territory, were reported to the NNDSS, one fewer than in 2007 (Figure 24).

Figure 24: Number of notifications of donovanosis, Australia, 1991 to 2008, by sex



Gonococcal infections

In 2008, 7,723 notifications of gonococcal infection were received by the NNDSS corresponding to a rate of 36.0 per 100,000 population, a slight decrease compared with 2007 (36.4 per 100,000 population).

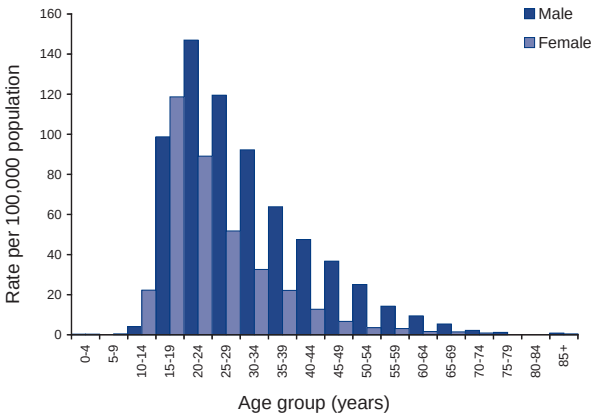
The highest notification rate in 2008 was in the Northern Territory (713 per 100,000 population), substantially higher compared with Western Australia, Queensland and South Australia (78.0, 38.1 and 32.5 per 100,000 population respectively) (Table 6). Considerable declines in notification rates between 2007 and 2008 were observed in the Australian Capital Territory (54%), Tasmania (35%) and Victoria (12%). Increases in notification rates for the same period were observed in South Australia (20%) and Queensland (16%).

Nationally, there was a decrease in the gonococcal infection notification rates in males (3%) and an increase in the notification rates in females (3%). Gonococcal infection notification rates were substantially higher amongst males than females, 47.1 and 25.0 per 100,000 population respectively. The male to female rate ratio in 2008 was 2:1, similar to the previous 5 years (2003 to 2007). As in previous years, the exception to this pattern was the Northern Territory, where females had an overall higher notification rate than males (748 versus 677 per 100,000 population). Nationally, notification rates of gonococcal infection in males exceeded those in females in all age groups except in the 10–14 and 15–19 year age groups (Figure 25).

Trends in sex specific notification rates show that in 2008 there has been an abatement of the declines seen in 2007 amongst males in the 20–34 year age range. In females, there were no marked change in notification rates; trends for all age groups appeared to remain relatively stable with a small increase occurring in the 15–19 year age group and a decrease continuing to occur in the 20–24 year age group (Figure 26).

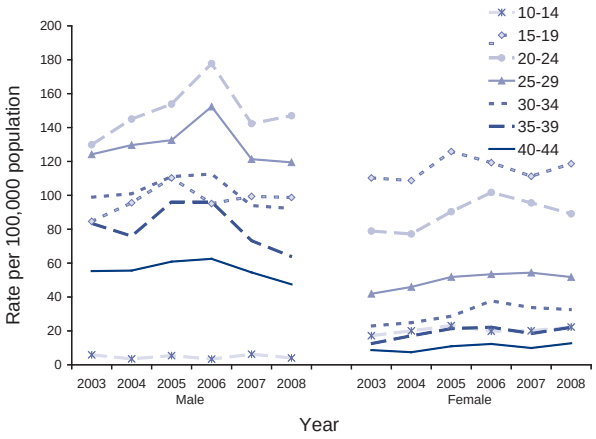
In 2008, the data completeness of the indigenous status field for gonococcal infection notifications was 72%, which is a slight increase compared with previous years. Six jurisdictions had greater than 50% completeness of the indigenous status field: the Northern Territory, Queensland, South Australia, Tasmania, Victoria and Western Australia. Among these jurisdictions the combined age standardised notification rate for gonococcal infection was 791 per 100,000 in the Indigenous population and 21 per 100,000 in the non-Indigenous population. The age standardised rate ratio of Indigenous compared with non-Indigenous gonococcal infection notifications across these respective jurisdictions was 37:1. Between 2007 and 2008, rates of gonococcal infection notifications in the Indigenous population declined by 32% in South Australia, with declines also being seen in the Northern Territory, Tasmania and Western Australia. For the same period, increases in the notification rate of gonococcal infections were seen in Queensland (17%). In Victoria, there was a doubling of the rate, however this effect was due to changes in very small notification numbers in this population (Figure 27).

Figure 25: Notification rate of gonococcal infections, Australia, 2008, by age group and sex*



* Excludes 12 notifications whose age or sex was not reported.

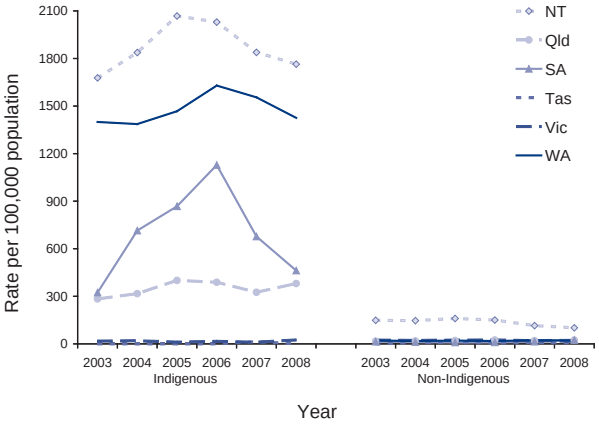
Figure 26: Trends in notification rates of gonococcal infection in persons aged 10–44 years, Australia, 2003 to 2008, by age group and sex



Other surveillance of gonococcal infections

The AGSP is the national surveillance system for monitoring the antimicrobial resistance of *Neisseria gonorrhoeae* isolates, via a network of public

Figure 27: Trends in notification rates of gonococcal infection, selected states and territories,* 2003 to 2008, by indigenous status



* States and territories in which indigenous status completeness was reported for more than 50% of cases over a 5 year period.

and private reference laboratories located in each jurisdiction. Susceptibility testing is performed on gonococcal isolates to a core group of antibiotics: penicillin, ceftriaxone, spectinomycin, quinolone and tetracycline, using a standard methodology. The following is a summary of the AGSP 2008 report.[37]

In 2008, a total of 3,192 gonococcal isolates were tested for antibiotic susceptibility, representing approximately 41% of gonococcal infection notifications. The number of gonococcal isolates available for susceptibility testing is affected by the increasing use of non-culture based diagnosis methods.

Of the total number of isolates collected through the AGSP in 2008, there were 2,509 isolates from males, 682 isolates from females (male to female ratio 4.7:1) and there was 1 isolate where the sex was not reported. In males, 73% of isolates were obtained from the urethra, 15% from the rectum and 9% from the pharynx. In females, the majority of isolates (88%) were obtained from the cervix.

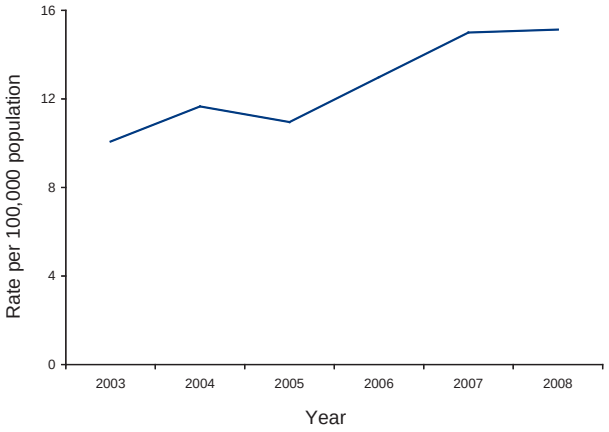
In 2008, approximately 44% of gonococcal isolates were resistant to penicillins and 54% to the quinolone antibiotic group. The number of isolates with high level tetracycline resistance continued to be at a historically high level. As in previous years, the pattern of gonococcal antibiotic susceptibility differed between states and territories, and rural and urban areas within each jurisdiction,[38] where for example in remote areas of some jurisdictions with high disease rates, penicillin based treatments continue to be effective.

Syphilis (all categories)

In 2004, all jurisdictions began reporting to the NNDSS non-congenital syphilis infections categorised as: infectious syphilis (primary, secondary or early latent) of less than 2 years duration; and syphilis of more than 2 years or unknown duration. However, in South Australia only notifications of infectious syphilis are reported to the NNDSS. Detailed analyses are reported for these 2 categories, as well as for syphilis of the combined categories (syphilis – all categories) for the purpose of showing trends in previous years.

In 2008, a total of 3,243 notifications of syphilis infection of all categories was reported, representing a notification rate of 15.1 per 100,000 population, a slight increase compared with 2007 (Table 7, Figure 28). The Northern Territory continued to have the highest notification rate of syphilis (115 per 100,000 population), although in 2008 the rate was 17% lower than in 2007. In 2008, there were increases in notification rates in Western Australia (30%), the Australian Capital Territory (27%), New South Wales (14%) and South Australia (5%). As in other developed countries syphilis infection rates have continued to rise in Australia amongst men who have sex with men.[39,40]

Figure 28: Notification rate of non-congenital syphilis infection (all categories), Australia, 2003 to 2008



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

In 2008, a total of 1,303 cases of infectious syphilis (primary, secondary and early latent), less than 2 years duration, were reported. This represents a notification rate of 6.1 per 100,000 population, a decrease of 9% compared with 2007 (6.7 per 100,000 population) (Table 7). The

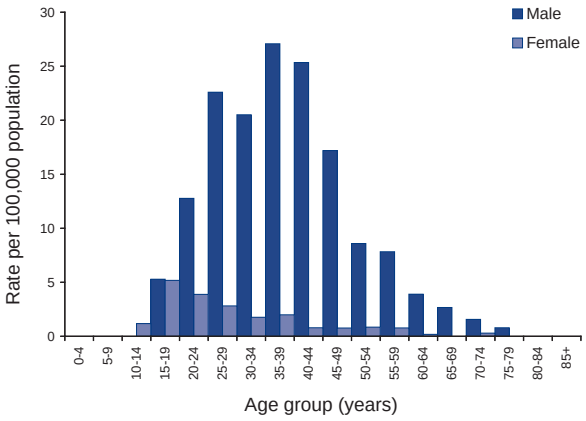
Northern Territory had the highest notification rate at 37.8 per 100,000 population in 2008, a decrease of 32% compared with 2007. Decreases in notification rates per 100,000 population compared with 2007 occurred across all jurisdictions, except Western Australian and South Australia, which increased by 69% (4.9 to 8.3) and 5% (3.1 to 3.2) respectively.

Nationally, the notification rates of infectious syphilis for males and females were 10.8 and 1.4 per 100,000 population respectively, and represented a male to female ratio of 8:1 (Table 14). Notification rates in males were highest in the 35–39 year age group (27.1 per 100,000 population), closely followed by the 40–44 year age group (25.3 per 100,000), whereas in females the highest notification rate was observed in the 15–19 year age group (5.2 per 100,000 population). In all jurisdictions and across all age groups, notification rates were higher in males than in females, except the 10–14 year age group where the rate was 1.2 per 100,000 for females compared with no notifications for males (Figure 29).

Over the period 2004 to 2008 notification rates amongst males increased substantially until 2007, especially in the 20–29, 30–34 and 40–49 year age groups, and then decreased or were similar in 2008. The overall increases observed during this period occurred mainly in men who have sex with men.[4] In females, for the 2004 to 2008 period, rates remained relatively steady, except in the 15–19 and 20–29 year age groups where they decreased by 21% and 41%, respectively, compared with 2007 (Figure 30).

In 2008, data on indigenous status were complete in 96% of notifications of infectious syphilis and all jurisdictions had greater than 50% completeness of the indigenous status field. The age standardised notification rate was 37.1 per 100,000 in the Indigenous population and 5.3 per 100,000 in the non-Indigenous population, representing a ratio of 7:1. These age standardised notification rates ranged substantially across jurisdictions. Over the past 5 years, the disparity in notification rates between Indigenous and non-Indigenous populations continued to decrease across all jurisdictions except the Australian Capital Territory (indigenous status less than 50% complete 2004–2007) (Figure 31). Analysis of age specific notification rates show that compared with the non-Indigenous population, rates of infectious syphilis in the Indigenous population are highest in a younger age group, 15–19 years, compared with the non-Indigenous population where notification rates are highest in the 35–39 year age group.

Figure 29: Notification rate of infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2008, by age group and sex



* Excludes 2 notifications whose sex was not reported.

Table 14: Number and rates* of notifications of infectious syphilis (less than 2 years duration), Australia, 2008, by state or territory and sex†

State or territory	Male		Female		Total	
	Count	Rate*	Count	Rate*	Count	Rate*
ACT/NSW	398	11.0	22	0.6	420	5.7
NT	49	43.0	34	32.1	83	37.8
Qld	167	7.8	20	0.9	187	4.4
SA	45	5.7	7	0.9	52	3.2
Tas	5	2.0	2	0.8	7	1.4
Vic	355	13.5	17	0.6	374	7.0
WA	133	12.1	47	4.4	180	3.4
Total	1,152	10.8	149	1.4	1,303	6.1

* Notification rate per 100,000 population.

† Total includes 2 notifications whose sex was not reported.

Figure 30: Trends in notification rates of infectious syphilis (primary, secondary and early latent), less than 2 years duration, in persons aged 10 years or over, Australia, 2004 to 2008, by age group and sex

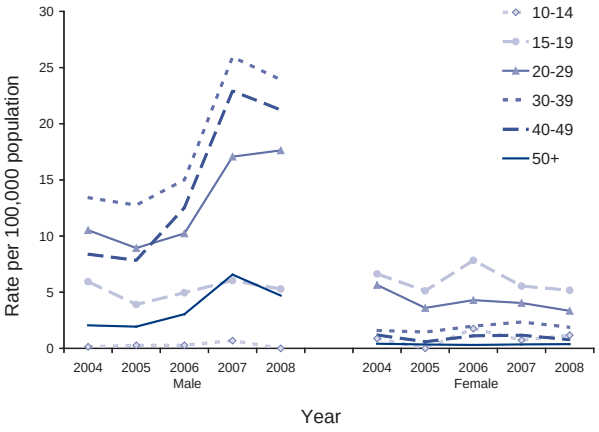
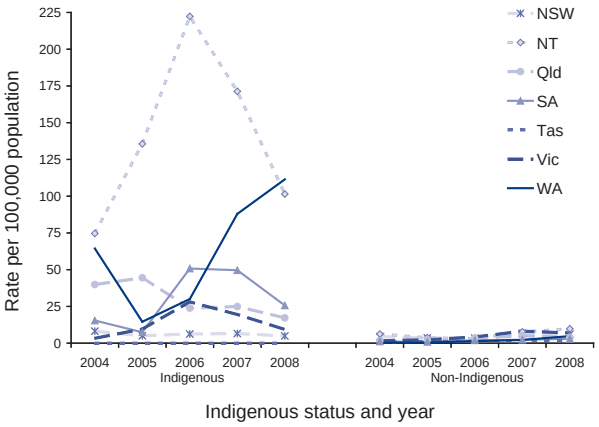


Figure 31: Trends in notification rates of infectious syphilis, selected states and territories,* 2003 to 2008, by indigenous status



* States and territories in which Indigenous status completeness was reported for more than 50% of cases over a 5 year period.

Syphilis of more than 2 years or unknown duration

In 2008, a total of 1,940 notifications of syphilis of more than 2 years or unknown duration were reported, a notification rate of 9.8 per 100,000 population. This rate represents an increase of 10% compared with 2007 (8.9 per 100,000 population). The Northern Territory continued to have the highest notification rate at 77.3 per 100,000 population, however, this was a decrease of 6% compared with 2007 (81.9 per 100,000 population).

In 2008, notification rates of syphilis of more than 2 years or unknown duration in males and females were 12.7 and 6.7 per 100,000 population, respectively (Table 15). Notification rates were higher in males than in females in all jurisdictions, except the Northern Territory, where males had a lower rate than females (74 and 81 per 100,000 population, respectively). Nationally, the male to female ratio was 1.9:1. The distributions of notification rates across age groups were similar in males and females with a bimodal distribution, noting however, that rates in males were substantially higher compared with females, especially in the older age groups. In males, the rate remained high from 35 years and over, peaking in the 35–49 year age range and again in the 85 or over year age group. Whilst amongst females, a younger peak was seen in the 30–34 year age group, with a second peak again in the 85 years or over age group (Figure 32).

Over the period 2004 to 2008, notification rates increased substantially between 2005 and 2008 amongst males aged 30 years or over. In females for the same period, notification rates have remained relatively stable, except in females aged 20–29 years where the rates have decreased from 14 per 100,000 population in 2004 to 8 per 100,000 population in 2008 (Figure 33).

Table 15: Number and rates* of notifications of syphilis of more than 2 years or unknown duration, Australia,† 2008, by state or territory and sex

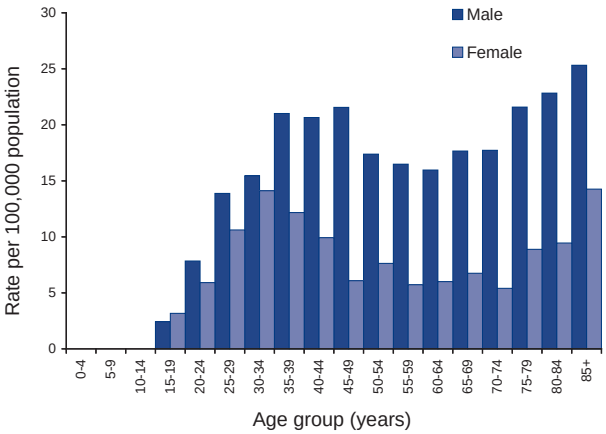
State or territory	Male		Female		Total‡	
	Count	Rate*	Count	Rate*	Count	Rate*
ACT/NSW	715	19.7	304	8.2	1,023	14.0
NT	84	73.7	86	81.3	170	77.3
Qld	115	5.4	88	4.1	203	4.7
Tas	11	4.5	4	1.6	15	3.0
Vic	273	10.4	139	5.2	419	7.9
WA	59	5.4	51	4.8	110	2.1
Total	1,257	12.7	672	6.7	1,940	9.8

* Notification rate per 100,000 population.

† Data from all states and territories except South Australia.

‡ Total includes 10 notifications whose sex was not reported.

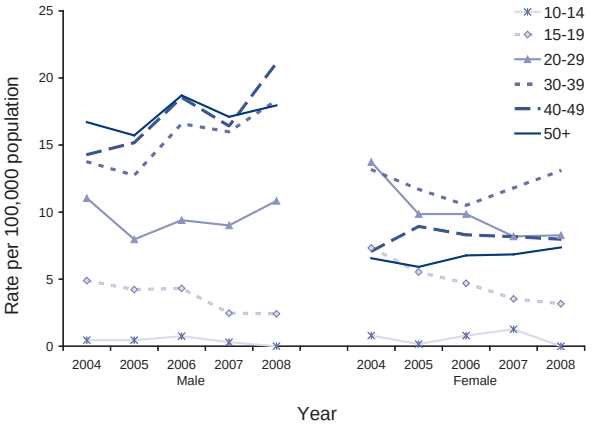
Figure 32: Notification rate of syphilis of more than 2 years or unknown duration, Australia,* 2008, by age group and sex†



* Data from all states and territories except South Australia.

† Excludes 11 notifications where sex was not reported.

Figure 33: Rates of notification of syphilis of more than 2 years or unknown duration, Australia,* 2004 to 2008, by age group and sex

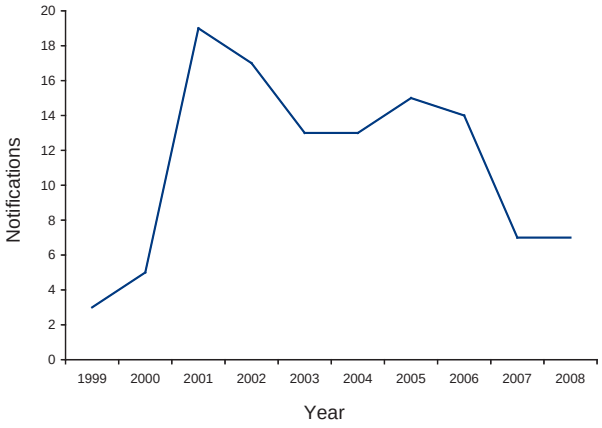


* Data from all states and territories except South Australia.

Congenital syphilis

There were 7 notifications of congenital syphilis reported in 2008, 3 males and 4 females. Three notifications each were reported from New South Wales and Queensland, and one from the Northern Territory. Two of the notifications were Indigenous, four non-Indigenous and one was reported as unknown indigenous status. Following a peak of 19 notifications in 2001, notifications of congenital syphilis have continued to decline (Figure 34).

Figure 34: Trends in notifications of congenital syphilis, Australia, 1999 to 2008



Vaccine preventable diseases

Introduction

This section summarises the national notification surveillance data for laboratory-confirmed influenza and notifiable diseases targeted by the National Immunisation Program (NIP) in 2008. These include diphtheria, *Haemophilus influenzae* type b (Hib) infection, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella, tetanus and varicella zoster infections (chickenpox, shingles and unspecified). Data on hepatitis B and invasive meningococcal disease, which are also targeted by the NIP, can be found in this report under 'Bloodborne diseases' and 'Other bacterial infections' respectively. Other vaccine preventable diseases (VPDs) presented in this report include hepatitis A and Q fever under the 'Gastrointestinal diseases' and 'Zoonoses' sections respectively.

In 2008, there were 34,225 notifications of VPDs (20% of total notifications). This is 25% more than the 27,332 notifications of VPDs reported in 2007. Pertussis was the most commonly notified VPD (14,516, 42% of all VPD notifications). The number of notifications and notification rates for VPDs in Australia are shown in Tables 5 and 6.

There were no new vaccines added to the NIP in 2008. However, due to an international shortage of some Hib vaccines (monovalent Hib *PedvaxHib*® and Hib-hepatitis B *Comvax*®) those vaccines were replaced by the hexavalent DTP-IPV-HepB-Hib vaccine at 2, 4 and 6 months and another monovalent Hib vaccine (Hiberix®) at 12 months in March 2008 in Victoria, Queensland and South

Australia. For the remainder of 2008, Comvax® and PedvaxHib® were used only in Western Australia for Indigenous children and for all children in the Northern Territory.

Information on receipt of vaccines has been recorded on the NNDSS using the 'vaccination status' field (full, partial or unvaccinated), plus a field capturing number of doses. In January 2008, new more detailed fields were added to record 'vaccine type' and vaccination date for each dose. The new fields were intended to replace the old fields, with a transition period allowing either type of vaccination details. In 2008, 2 jurisdictions commenced using the new fields (Northern Territory and Queensland), while the remaining jurisdictions continued using the old fields. In this report data on receipt of vaccines are presented for each disease combining data from the 2 different formats.

Diphtheria

There were no notifications of diphtheria reported to the NNDSS in 2008. The last notification of diphtheria reported in Australia was a case of cutaneous diphtheria in 2001, the only notification reported since 1992.

Haemophilus influenzae type b disease

There were 25 notifications of Hib disease in 2008 corresponding to a rate of 0.1 notifications per 100,000 population. There were eight more notifications than reported in 2007. Thirty-six per cent (9/25) of notifications were amongst children aged less than 10 years, with the remainder being distributed between those aged between 30 and 84 years. Sixty per cent (15/25) of the notifications were in males with a male to female ratio of 1.5:1, unlike in 2007 when the ratio was 0.9:1 (Figure 35).

Indigenous status was recorded for 24 of the 25 notifications; three were Indigenous and 21 were non-Indigenous. The Hib notification rate in 2008 was 0.6 per 100,000 in the Indigenous population and 0.1 per 100,000 in the non-Indigenous population, equating to a ratio of 6:1. Between 2003 and 2007, Hib notification rates in the Indigenous population were 6.6 to 30.3 times higher than the rates in the non-Indigenous population. However the figures vary dramatically because of the low number of notifications (Figure 36). This analysis excludes those notifications with an unreported or unknown indigenous status (6 for 2003, 4 for 2006 and one for each of the remaining years).

Figure 35: Notifications of *Haemophilus influenzae* type b infection, Australia, 2008, by age group and sex

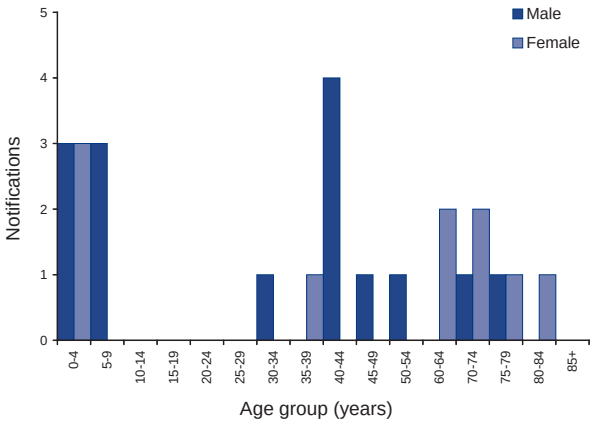
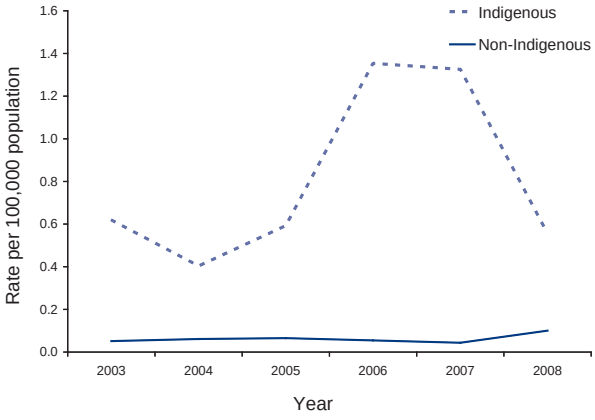


Figure 36: Notification rate for *Haemophilus influenzae* type b infection, Australia, 2003 to 2008, by indigenous status



Children under the age of 16 years were eligible for Hib vaccination in infancy in 2008, as Hib vaccines were introduced to the NIP for all children born after February 1993. Of the 9 notifications aged less than 16 years in 2008, five were vaccinated and four were unvaccinated. Of the five that were vaccinated, two had received their age appropriate vaccinations and three had not been fully vaccinated for age. Vaccination status for a total of 3 notifications across all ages was unknown or not supplied.

After nearly 2 decades of Hib vaccination, Australia now has one of the lowest rates of Hib in the world.[41] A recent study on the trends of invasive Hib in Australia between 1995 and 2005 concluded that almost 60% of invasive Hib cases in children are preventable.[42]

Influenza

The Australian 2008 influenza season was less severe than the 2007 season, but the number of notifications was higher than in each of the years 2004 to 2006 (Figure 37). Notifications were 1.9 times greater than the 5-year mean and peaked in the first week of September. There were 9,137 notifications of laboratory-confirmed influenza in 2008, corresponding to a rate of 43 per 100,000 population. Queensland accounted for 41% of all confirmed influenza notifications to the NNDSS (Figure 38), but this proportion may in part reflect different testing and laboratory practices rather than real differences in the incidence of infection.[43] Notifications in the non-seasonal period were higher than in previous years.

The highest notification rates occurred in the Northern Territory with 91 per 100,000 population, followed by Queensland (86 per 100,000 population), Tasmania (78 per 100,000 population) and the Australian Capital Territory (71 per 100,000 population) (Table 5).

There were 1,351 notifications of laboratory-confirmed influenza in children aged less than 5 years (14.8% of all notifications). As in previous years, influenza notification rates were markedly higher in children aged under 5 years (98 per 100,000 population) compared with those aged 5 years or over (39 per 100,000 population) (Figure 39). Within this age group, the highest rate was in children under 1 year of age (162 per 100,000 population).

In 2008, 8,906 (98.5%) influenza notifications in the NNDSS included typing data. Influenza B was predominant in the 2008 season; the first year this has been observed since influenza became nationally notifiable in 2001. Of typed notifications, 55% (4,924) were influenza B, 44% (3,894) were influenza A and 1% of notifications were

Figure 37: Notifications of laboratory-confirmed influenza, Australia, 2008, by month of diagnosis

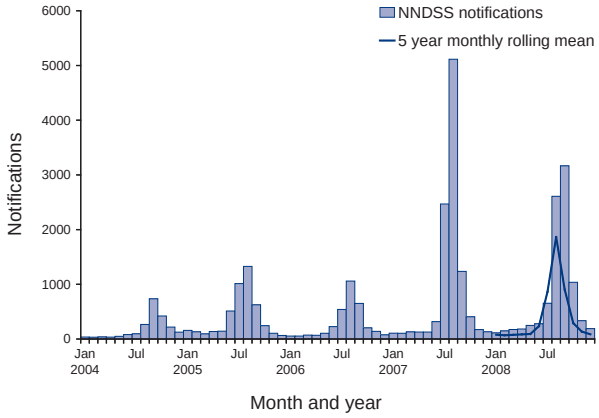
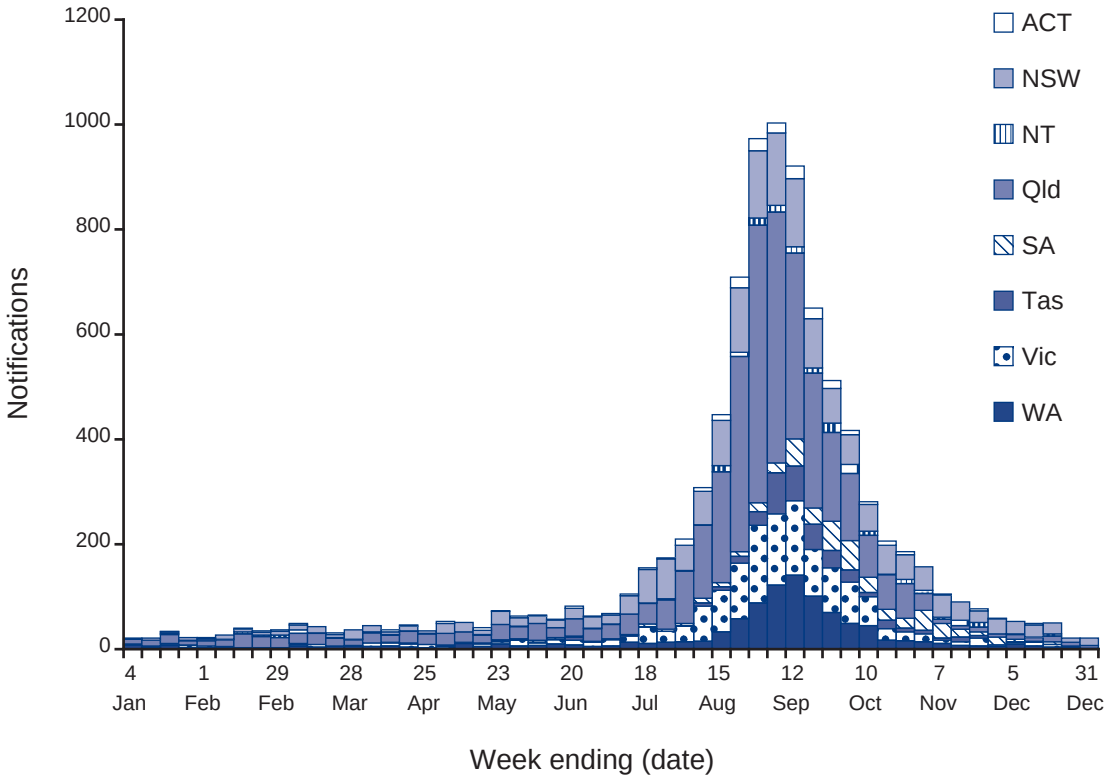


Figure 38: Notifications of laboratory-confirmed influenza, Australia, 2008, by state or territory and week of diagnosis

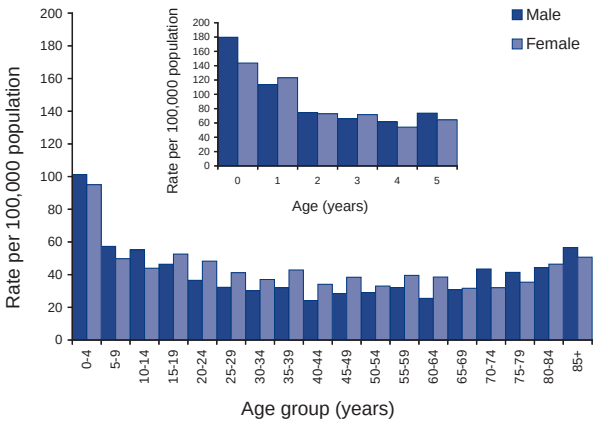


notified as 'A&B' (86) or type C (2). Prior to the start of the season, influenza notifications were predominantly influenza A, however influenza B predominated during the peak of the season (Figure 40).

In 2008, 1,224 influenza virus isolates were analysed at the WHO Collaborating Centre for Reference and Research on Influenza. There were approximately equal proportions of viruses from the 2 influenza B lineages (B/Victoria and B/Yamagata), however B/Yamagata viruses (B/Florida/4/2006-like included in the 2008 influenza vaccine) were predominant at the start of the season, while B/Victoria (B/Malaysia/2506/2004-like) viruses predominated at the end of the season. Of circulating A(H3) viruses, most were antigenically similar to A/Brisbane/10/2007; the 2008 A(H3) vaccine strain. Circulating A(H1) strains showed significant drift away from the 2008 vaccine strain A/Solomon Islands/3/2006 to the A/Brisbane/59/2007-like viruses.

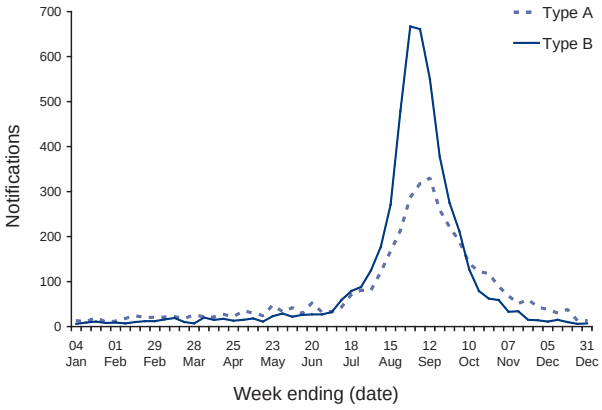
The recommendation for the 2009 Southern Hemisphere vaccine had only one change compared with the 2008 Southern Hemisphere vaccine: a change to the A(H1) virus from a A/Solomon Islands/3/2006-like virus to A/Brisbane/59/2007-like virus. The other 2 recommended strains: A/Brisbane/10/2007-like virus (H3N2) and B/Florida/4/2006-like virus, were left unchanged.

Figure 39: Notification rate for laboratory-confirmed influenza, Australia, 2008, by age group and sex*



* Excludes 14 notifications whose age or sex was not reported.

Figure 40: Notifications of laboratory-confirmed influenza, Australia, 2008, by type and week of diagnosis*



* Notifications of influenza type 'A&B' (n=86), 'C' (n=2) and 'untyped' (n=231) were excluded from analysis.

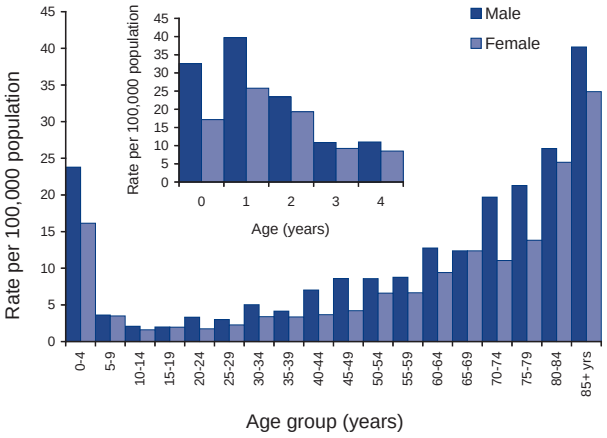
Invasive pneumococcal disease

There were 1,629 notifications of invasive pneumococcal disease (IPD) in Australia in 2008, a rate of 7.6 notifications per 100,000 population. This was a small increase of 10% from the 1,483 notifications reported in 2007 (7.0 notifications per 100,000 population). An increase in notification rates between 2007 and 2008 was seen in New South Wales (547 notifications, 7.8 per 100,000 population), South Australia (120 notifications, 7.5 per 100,000 population), Tasmania (39 notifications, 7.8 per 100,000 population), Victoria (355 notifications, 6.7 per 100,000 population) and Western Australia (162 notifications, 7.5 per population). The lowest notification rate in 2008 was seen in the Australian Capital Territory (20 notifications, 5.8 per 100,000 population).

In 2008, males accounted for 913 (56%) of the 1,629 notifications of IPD. In most age groups there were more male than female notifications, resulting in a male to female ratio of 1.3:1. Figure 41 shows that the highest rates of IPD in 2008 were notified in persons aged 85 years or over (36.1 notifications per 100,000 population) and in children aged 1 year (32.9 notifications per 100,000 population).

The 7 valent pneumococcal conjugate vaccine (7vPCV) became available for infants and children at high risk of IPD in 2001. In 2005 it was added to the NIP for all children up to 2 years of age.[11] Notification rates of IPD disease caused by 7vPCV serotypes in the Indigenous population have declined over the past 5 years, from 7.8 to 3.2 notifications per 100,000 population (38 to 17 notifications) between 2003 and 2008. In the non-Indigenous population, notification rates of 7vPCV serotype disease have also declined from 5.8 to 1.2 notifications per 100,000 population (1,132 to 235 notifications) between 2004 and 2008.

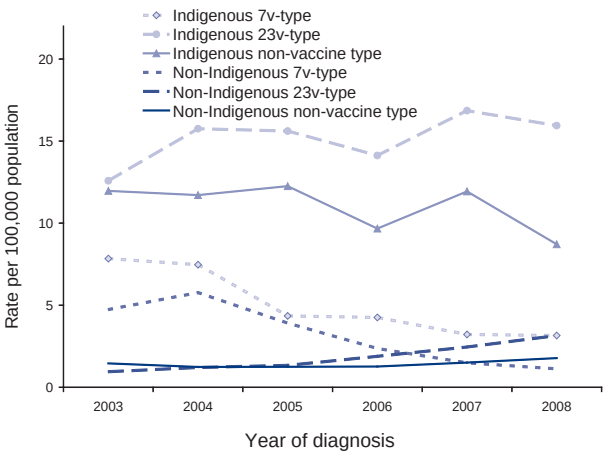
Figure 41: Notification rate for invasive pneumococcal disease, Australia, 2008, by age group and sex



The 23 valent pneumococcal polysaccharide vaccine (23vPPV) has been on the NIP since 1999 for all Indigenous Australians over 50 years of age and for those 15 to 49 years of age with high risk conditions. Since 2005, 23vPPV has also been on the NIP for all Australians over the age of 65 years. The number of notifications of IPD in both Indigenous and non-Indigenous populations due to 23vPPV serotypes increased between 2003 and 2008 from 61 to 86 notifications (12.6 to 15.9 notifications per 100,000 population) and 184 to 658 notifications (0.9 to 3.1 notifications per 100,000 population) respectively (Figure 42).

Additional data were collected on notifications of IPD in all Australian jurisdictions during 2008. Details can be found in the invasive pneumococcal disease annual report series published in *CDI*, at www.health.gov.au/cdi

Figure 42: Notification rate for invasive pneumococcal disease, Australia, 2003 to 2008, by serotype



Measles

There were 65 notifications of measles reported to NNDSS in 2008 corresponding to a rate of 0.3 notifications per 100,000 population. This was a large increase compared with the 12 notifications reported in 2007 (0.1 per 100,000 population) (Figure 43). In 2008, notifications were reported from New South Wales (39), Queensland (11), Western Australia (8), Northern Territory (3), Victoria (2), and South Australia (2).

In 2008, 55% (36/65) of measles notifications were male. The age at diagnosis ranged from 7 months to 48 years with the median age being 17 years. There was an increase in notifications in all age groups compared with 2007. This increase was highest in those 25–34 years of age (19 in 2008 compared with 0 in 2007) (Figure 44).

Of the 54 notifications with information on the place of acquisition, 26% (14/54) were reported as being acquired from overseas including the United Kingdom, Dubai, Thailand, Japan, China

Figure 43: Measles notifications, Australia, 2003 to 2008, by month of diagnosis

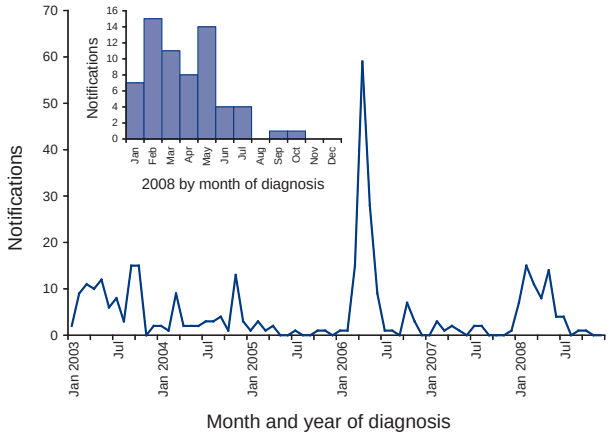
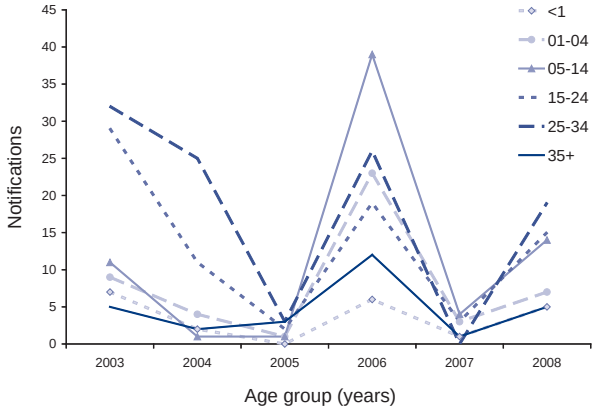


Figure 44: Trends in measles notifications, Australia, 2003 to 2008, by age group



and India. There were 2 outbreaks with more than 5 cases during 2008: one with 9 cases in Western Sydney associated with an emergency department and another in Queensland with 8 cases where the source of infection was not identified.

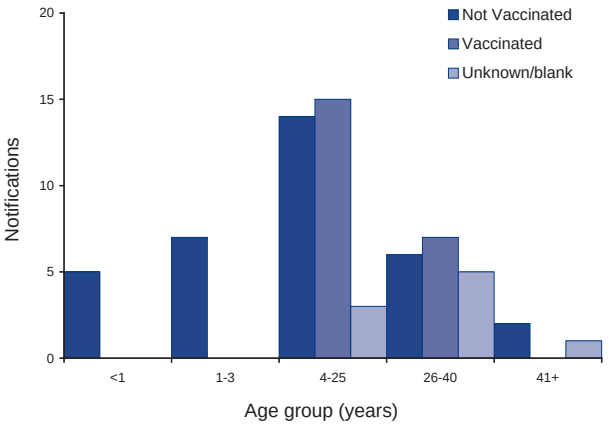
Two doses of MMR are funded for children and provided at 12 months and 4 years of age under the NIP. The MMR vaccine induces long-term measles immunity in 95% of recipients after a single dose and 99% of recipients after the second dose.[11]

Nationally, there was information on vaccination status for 86% (56/65) of notifications in 2008, of which 61% (34/56) were not vaccinated and 39% (22/56) had been vaccinated (7 with 2 doses, 10 with 1 dose of a measles-containing vaccine and the remaining 5 with no dose stated) (Figure 45). The 5 non-vaccinated infants aged less than 1 year of age at diagnosis were ineligible for routine vaccination. None of the 7 notifications for children aged 1–3 years and eligible for 1 dose of the measles-mumps-rubella vaccine (MMR) were vaccinated.

For the 29 notifications aged 4–25 years and eligible for 2 doses of MMR (with vaccine information available), 48% (14/29) were not vaccinated and 52% (15/29) had been vaccinated, seven of which had 2 doses and five of which had 1 dose of a measles-containing vaccine.

There were 13 notifications with information on vaccination status in those aged 26–40 years. These are considered to be a susceptible age cohort because many may have missed being vaccinated as infants when coverage was still low and the risk of natural immunity through exposure was declining. Of these, 46% (6/13) were not vaccinated and 54% (7/13) were vaccinated, five of these with 1 dose and two had no dose number stated.

Figure 45: Notifications for measles, Australia, 2008, by age group and vaccination status



The remaining 2 notifications with vaccine information provided were both 41 years or older and not vaccinated.

Mumps

In 2008, there were 286 notifications of mumps (1.3 per 100,000 population). This was approximately half of the 586 notifications of mumps (2.8 per 100,000 population) reported in 2007. In 2008, notifications were similar to the 5-year mean, with a ratio of 1.1.

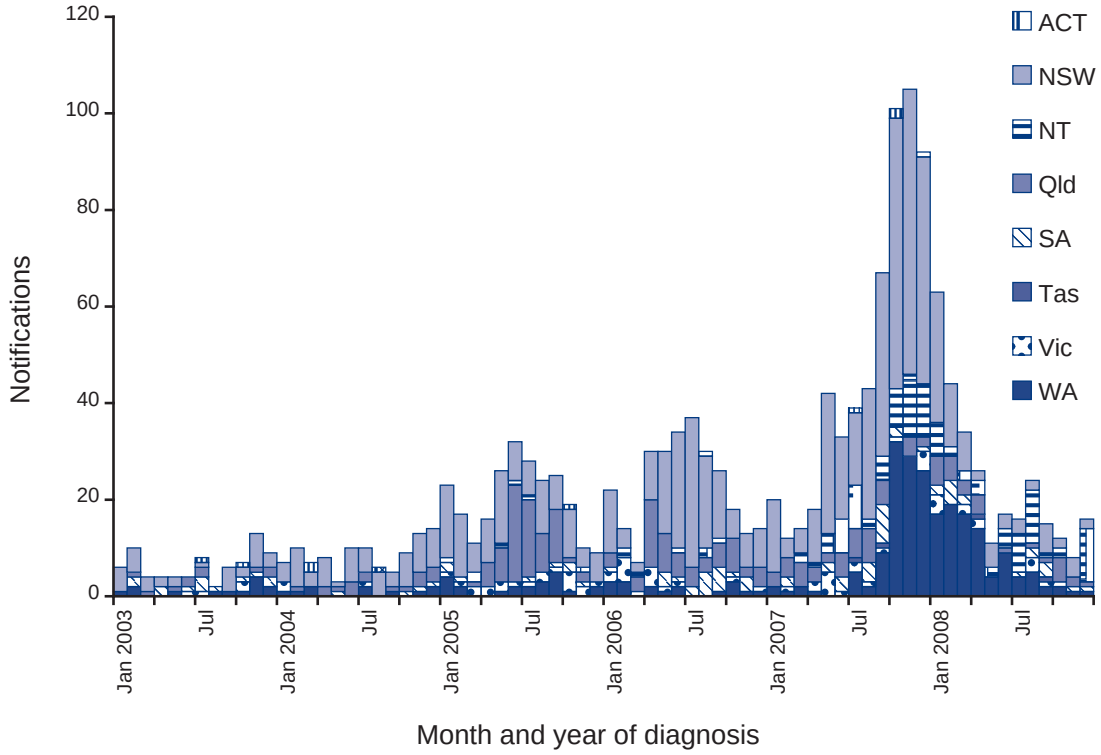
Notifications were reported from all jurisdictions except the Australian Capital Territory. The majority were reported from Western Australia with 33% (95/286), followed by 27% (77/286) from New South Wales and 18% (53/286) from the Northern Territory (Figure 46). The highest mumps notification rate was in the Northern Territory with 24 notifications per 100,000 population. Western Australia had the second highest notification rate in 2008 with 4.4 notifications per 100,000 population. New South Wales experienced the largest decrease in mumps notification rates from 2007 (4.7 per 100,000 population, 323 notifications) to 2008 (1.1 per 100,000 population, 77 notifications).

While the crude annual national mumps notification rate in Australia has been increasing since 2004, the rate in 2008 was the same as for 2006 (1.3 per 100,000 population) and close to that for 2005 (1.2 per 100,000 population), with rates in the less than 5 years and the 35 years or over age groups remaining relatively constant over the last 5 years (Figure 47).

In 2008, there were notifications of mumps in all age groups with the highest notification rates amongst adolescents and young adults. Rates in children aged less than 5 years (1.09 per 100,000 population, or 15 notifications) and adults greater than 40 years of age remained low (Figure 48). A decrease in the notification rates for both the 15–24 and 25–34 year age groups in 2008 compared with 2007 was apparent (Figure 47). In 2008, the highest notification rates for males were in the 10–14 and 15–19 year age groups (Figure 48), compared with 2007 where the highest rates occurred in the 25–29 year age group. The majority of notifications (55%, 156/286) were male, a similar proportion to the past 5 years.

Nationally, information on vaccination status was available for 85% (242/286) of the notifications of which 39% (94/242) were not vaccinated, 36% (89/242) were vaccinated, and the remaining 24% (59/242) were reported as not applicable or

Figure 46: Notifications of mumps, Australia, 2003 to 2008, by state or territory and month of diagnosis



unknown. Of the vaccinated notifications 2% (2/89) had 3 doses, the majority 68% (62/89) had 2 doses and 22% (20/89) had 1 dose of a mumps-containing vaccine, and the remaining five had missing or unknown dosage information.

Of the 69 Indigenous notifications with a known vaccination status, 96% (66/69), were vaccinated; of which 3% (2/66) had received 3 doses, 82% (54/66) had 2 doses and 15% (10/66) had 1 dose of a mumps-containing vaccine. Only 4% (3/69) of Indigenous notifications in 2008 were not vaccinated.

Indigenous status was reported for 77% (220/286) of mumps notifications, of which 50% (110/220) were reported as Indigenous and 50% as non-Indigenous. This represents a 15.5% increase in the proportion of Indigenous notifications in 2008 compared with the 23% (135/586) reported in 2007.

Of the cases notified from Western Australia and Northern Territory in 2008, 69% (66/95) and 75% (40/53) respectively were identified as Indigenous. In 2008, Western Australia experienced the end of a prolonged mumps outbreak in the Kimberly region that began in July 2007 and had peaked

Figure 47: Trends in notification rates for mumps, Australia, 2003 to 2008, by age group

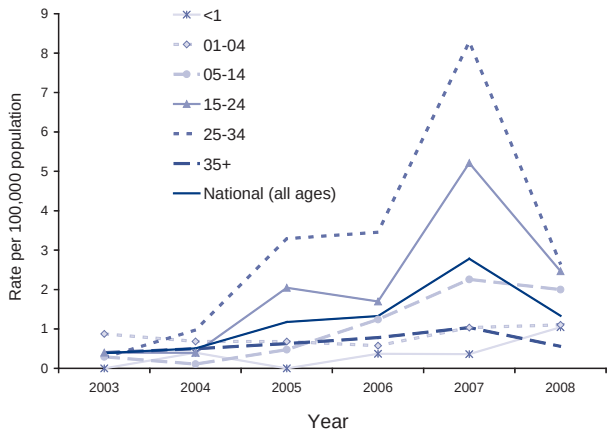
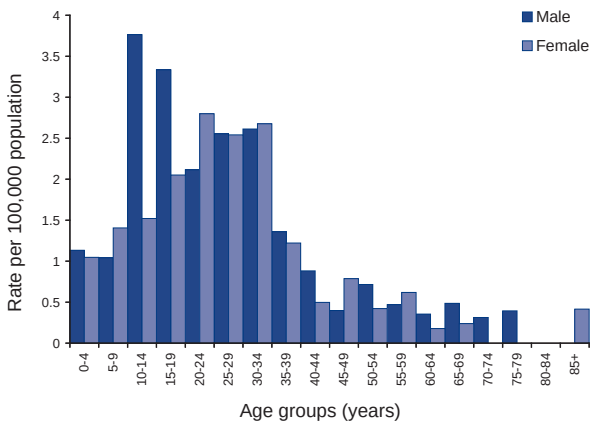


Figure 48: Notification rate for mumps, Australia, 2008, by age group



by the end of 2007.[28] The outbreak occurred predominantly amongst adolescent and young adult Aboriginal people (median age 18 years)[44] and had epidemiological links to an outbreak in Indigenous communities in the Northern Territory (personal communication, Gary Dowse, Communicable Disease Control, Directorate, Western Australian Department of Health). The affected population had a high rate of vaccination, with 52% (80/153) having received 2 doses and 14% (22/153) having received at least 1 dose of mumps containing-vaccine. Genotype J was identified in 20 mumps isolates and it remains unclear whether the outbreak was linked to the introduction of new genotypes from overseas outbreaks.[44]

The mumps component of the MMR vaccine is the least effective of the 3 components, providing 62%–85% and 85%–88% protection for the first and second dose respectively, compared with 95% for measles and 98% for rubella. Reduced effectiveness of the mumps vaccine component over time has been demonstrated to wane for 1 dose from 96% in 2-year-olds to 66% in 11–12-year-olds; and for 2 doses to wane from 99% in 5–6-year-olds to 86% in 11–12-year-olds.[45] This may at least partially account for the proportion of vaccinated mumps cases. Reduced efficacy has been suspected as a factor in recent mumps outbreaks in Israel and the United States of America in 2009 and 2010. Public health officials in New York are trialling a 3rd dose of vaccine in students in certain schools in Orange County as mumps transmission has continued despite a high rate of 2-dose vaccination coverage.[45,46]

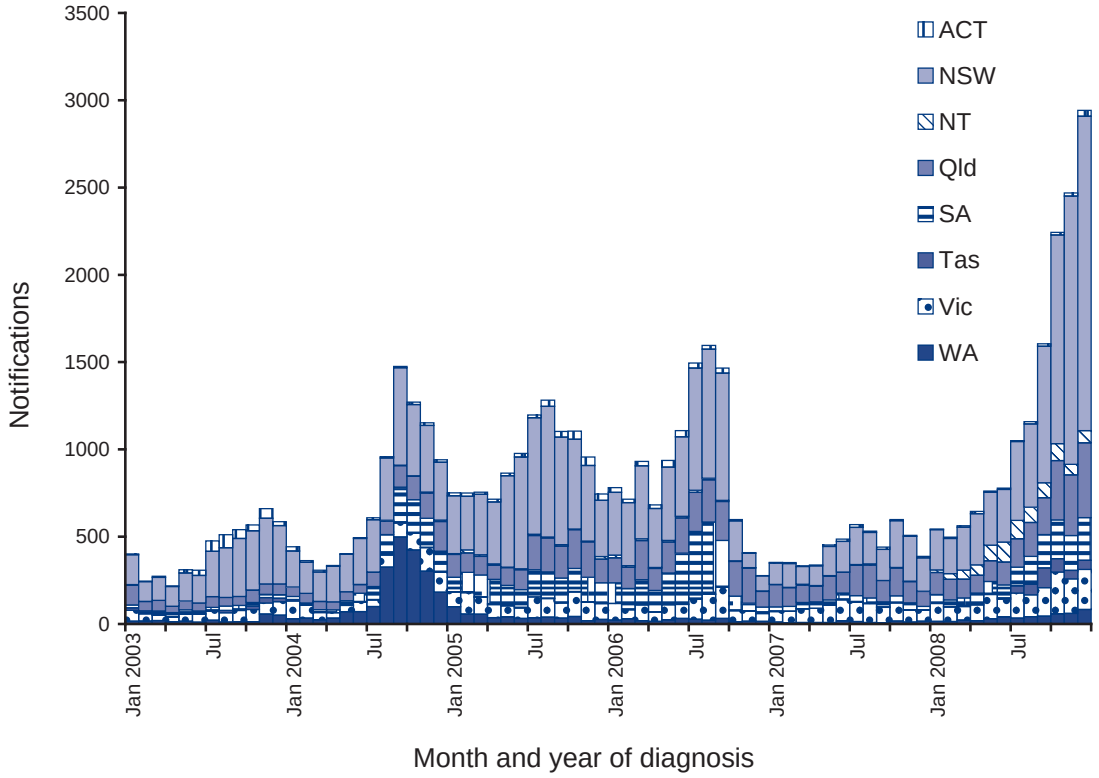
Pertussis

Pertussis is the most common vaccine preventable illness in Australia, with periodic epidemics occurring at intervals of three to 5 years on a background of endemic circulation. Notifications are normally higher in late winter and spring, however from 2004 to 2006, non-seasonal activity remained elevated compared with previous years (Figure 49). This may have been partially due to errors in diagnosis as discussed in the 2007 NNDSS annual report.[28]

In 2008, 14,516 notifications of pertussis were reported to NNDSS representing a notification rate of 67.7 per 100,000 population and was higher than in 2007 (5,345; 25.4 per 100,000 population). There was a large increase in the number of notifications from mid-2008, particularly in New South Wales, marking the beginning of an epidemic period which peaked in March 2009. In 2008, uptake of nucleic acid testing overtook serological methods for diagnosing new cases in New South Wales.

Notification rates in 2008 varied with age, with the highest notification rates in those aged less

Figure 49: Notifications of pertussis, Australia, 2003 to 2008, by month of diagnosis



than 15 years (114.2 per 100,000 population). This contrasted with 2006 where those aged 20–59 years and 60 years or over had the highest notification rate (Figure 50). Rates in these older age groups increased between 2003 and 2006, however by 2007 rates in these age groups had decreased. These older age groups were seen to have increasing rates since 2003, however by 2007 their notification rates had returned to a lower level. The notification rates of all groups less than 15 years increased more rapidly between 2007 and 2008 than those aged greater than 15 years.

There were more notifications amongst females (8,167; 56.3%) than males (6,333; 43.7%) in 2008, with 16 notifications for which sex was not specified (Figure 51). The highest notification rate amongst females was in the 0–4 year age group (126.9 per 100,000 population) with the highest rate in males being in the 10–14 year age group (122.5 per 100,000 population). While the greatest notification rates in 2008 were in those aged less than 15 years, the pattern of predominance of female notification rates compared with male notification rates for all age groups was similar to 2007 except for those aged 10 years, 70–74 years and those aged 85 years or over.

Nationally, information on vaccination was available for 71% (10,257/14,516) of notifications of which 65% (6,670/10,257) were not vaccinated and 35% (3,587/10,257) were vaccinated. No data were entered or vaccination status was unknown for 29% (4,259/14,516) of notifications. Information on the number of vaccine doses was less than 35% complete, thereby restricting further analysis of this field.

The newer nationally agreed vaccine type field used by Queensland and the Northern Territory was complete or status known for 91% (2,478/2,737) of notifications of which the majority (91%; 2,264/2,478) reported no vaccine given. Of those reporting no vaccine given, 89% (2,004/2,264) were aged 15 years or more, 8% (175/2,264) were between five and 15 years and 4% (85/2,264) were aged less than 5 years. All notifications aged more than 4 years would have been eligible for at least 4 doses of pertussis containing vaccine. Thirty-nine notifications would have been eligible for 1 dose at age of diagnosis and 15 notifications were less than 8 weeks of age and thus not eligible for their 1st dose of pertussis containing vaccine. Vaccine effectiveness is estimated to be 68% after receiving 1 dose of vaccine, increasing to 92% and greater after the 2nd dose[47] increasing to 99% following subsequent doses.[48] Immunity to disease decreases over time post vaccination with estimates of protection remaining for 4–12 years.[47] For this reason, current vaccine schedules for pertussis under the NIP are at 2, 4 and 6 months followed by a booster at aged 4 years and again at 15–17 years of age.

Notification rates of pertussis varied considerably by residential location. This was particularly noticeable in the 2nd half of 2008. By jurisdiction, the highest rates were in the Northern Territory (217.0 per 100,000 population) and New South Wales (111.9 per 100,000 population). When comparing rates by Statistical Divisions in Australia in the 1st half of 2008 with the 2nd half (Map 3), Northern Tasmania had the highest notification rate of 11.8 per 100,000 population. Loddon and the Central Highlands in Victoria had the next highest notification rates (10.0 and 8.1 respectively), and Central West New South Wales, South Eastern New South Wales and Murrumbidgee in New South Wales also demonstrated marked increases in the 2nd half compared with the 1st half of 2008 with rates of 9.3, 7.2 and 7.0 per 100,000 population respectively.

Figure 50: Trends in the notification rates of pertussis, Australia, 2003 to 2008, by age group

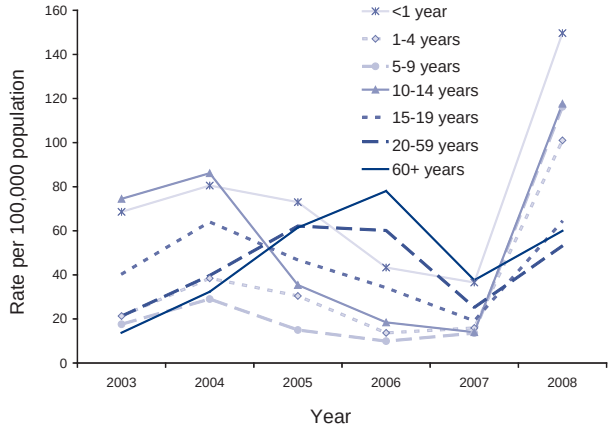
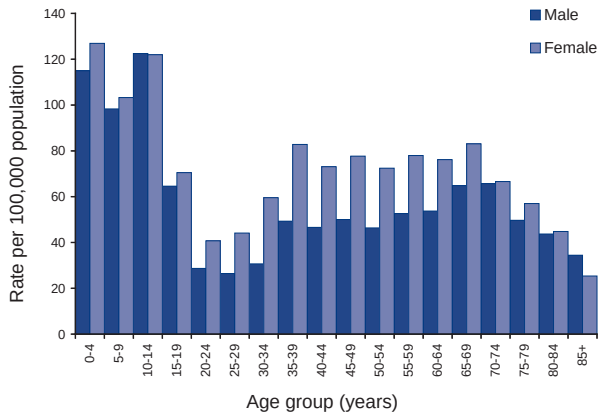
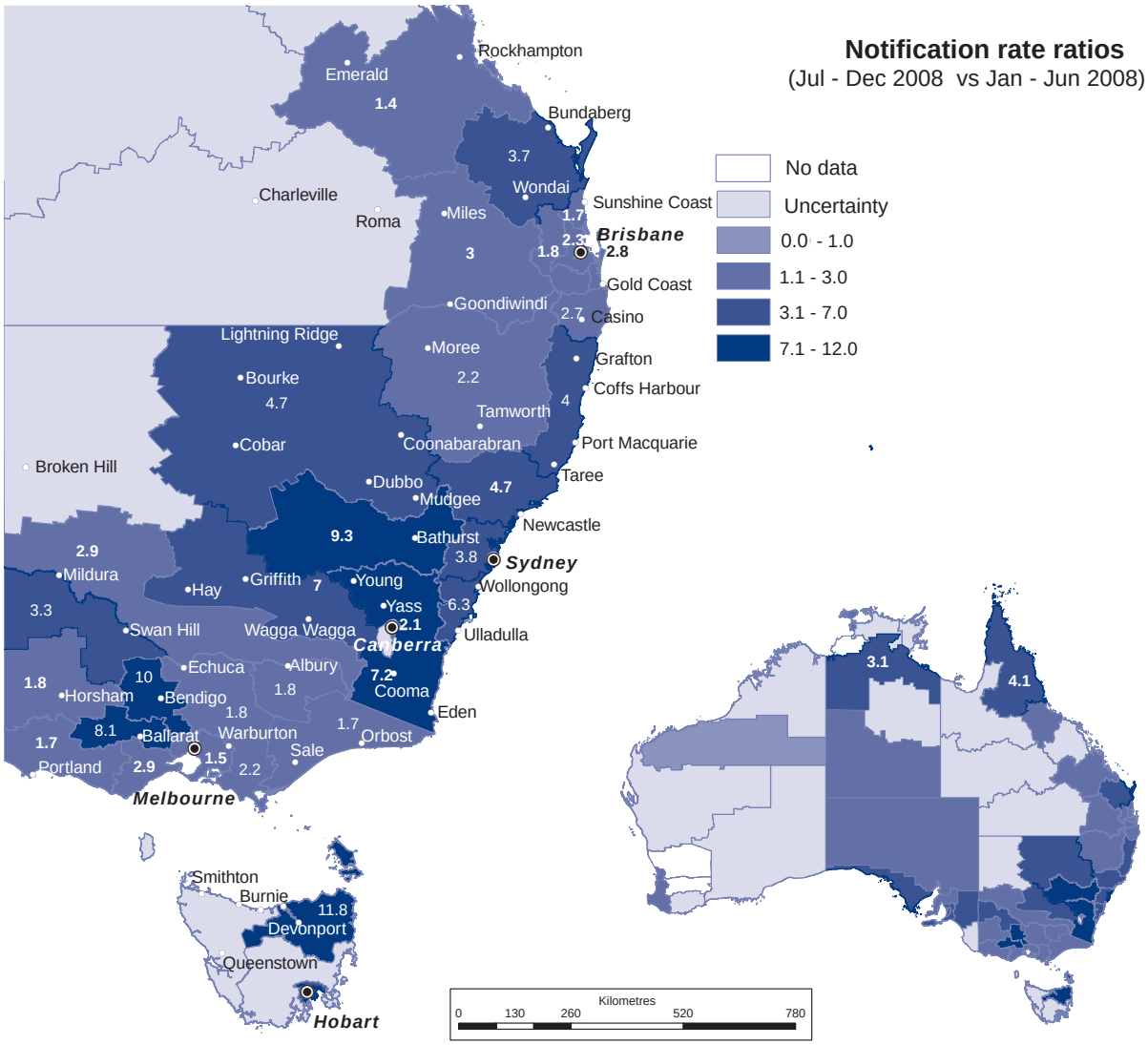


Figure 51: Notification rate for pertussis, Australia, 2008, by age and sex



Map 3: Notification rate ratio for pertussis comparing January to June with July to December 2008, by Statistical Division of residence



* Numbers shown in the Statistical Divisions represent the count of notifications.

Notification rates in geographic areas where estimated residential population and case numbers are small should be interpreted with caution.

Poliomyelitis

In 2008 there were no notifications of poliomyelitis in Australia, which along with the Western Pacific Region (WPR), remained poliomyelitis free. Poliomyelitis is a notifiable disease in Australia with clinical and laboratory investigation conducted for cases involving patients of any age with a clinical suspicion of poliomyelitis. Australia follows the WHO protocol for poliomyelitis surveillance and focuses on investigating cases of acute flaccid paralysis (AFP) in children under 15 years of age. Since 2000, the surveillance for AFP has been co-ordinated by the Victorian Infectious Diseases Reference Laboratory (VIDRL) in collaboration with the Australian Paediatric Surveillance Unit (APSU). 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. Between 1 January and 31 December 2008 there were 60 eligible AFP cases notified to the National Polio Reference Laboratory (NPRL) all of which were classified as non-poliomyelitis. The 2008 non-poliomyelitis AFP rate was 1.5 hence meeting the WHO AFP surveillance indicator for the fifth time since 1995. Details of the 2008 notifications are provided in the 2008 annual report of the Australian NPRL.[49]

During 2008, Australia finalised *An Acute Flaccid Paralysis and Poliomyelitis Response Plan for Australia*. The plan was endorsed by the Australian Health Protection Committee at their meeting on 4 December 2008 and is now available on the Australian Government's website at http://www.health.gov.au/internet/main/publishing.nsf/Content/polio-plan.htm

Rubella

In 2008, there were 37 notifications of rubella (0.2 per 100,000 population), a slight increase compared with the 34 notifications in 2007. Notifications were reported from New South Wales (17), Victoria (8), Western Australia (7), Queensland (4), and South Australia (1). There were small numbers of notifications reported across the age groups with no notifications for infants less than 1 year of age or for those adults between 50 and 80 years of age. The majority of notifications (29; 78%) were adults between 20 and 49 years of age (Figure 52). The median age was 32 years. The overall male to female ratio of notifications in 2008 was 1.1:1, with 19 males and 18 females. Of the 18 notifications that were female 15 (83%) were notified in women of child bearing age (17–47 years). Despite this, there were no notifications of congenital rubella reported in 2008.

Figure 52: Notifications of rubella, Australia, 2008, by age group and sex

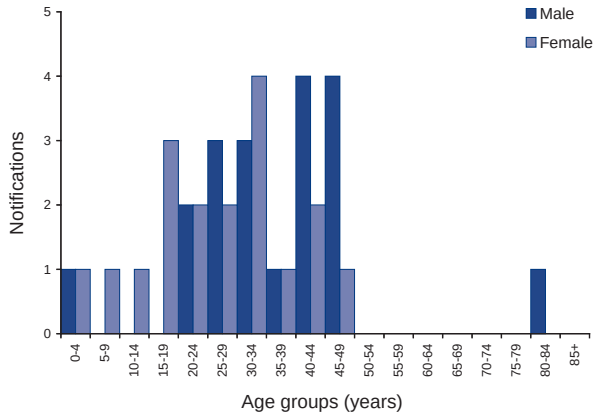
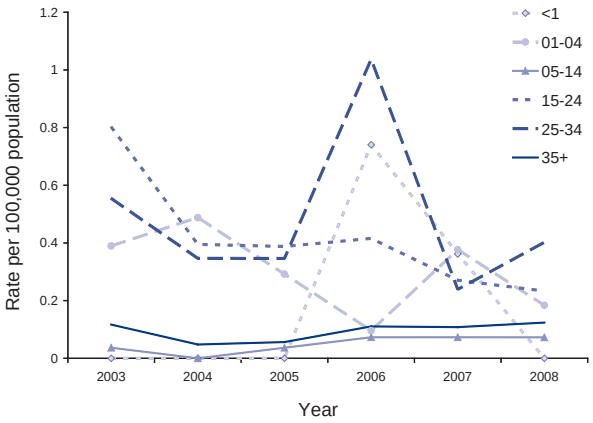


Figure 53 shows that trends in rubella notifications in different age groups have continued at low levels since 2003, except for a spike amongst those aged 25–34 in 2006. This spike was primarily due to an increase of notifications from South Eastern and Central Sydney, New South Wales. It was concentrated in those aged 15–44 years, however there was no single identifiable source for the increase in notifications.[50]

In Australia, populations at risk of rubella have previously been identified as including young men who did not receive the rubella immunisation in school based programs,[51] migrant women who did not receive rubella vaccines in their countries of birth,[52,53] and Indigenous women from rural and remote communities in the Top End of the Northern Territory.[54]

Figure 53: Trends in notification rates of rubella, Australia, 2003 to 2008, by age group



Nationally, information on vaccination status was available for 59% (22/37) of rubella notifications of which the majority, (82%; 18/22), were not vaccinated and 18% (4/22) were vaccinated. The remaining 41% (15/37) were stated as either unknown or blank. Of the 12 male notifications with information on vaccination reported, 83% (10/12) were not vaccinated, all of whom were adults ranging from 21 to 80 years of age and two had received 1 dose of a rubella-containing vaccine. Of the 10 female notifications in 2008 with vaccination information reported, 80% (8/10) were not vaccinated (all except one were women of child-bearing age between 19 and 43 years) and two had received 1 dose of a rubella containing vaccine (aged 9 years and 35 years).

Two doses of MMR are funded for children and provided at 12 months and 4 years of age under the NIP. A single dose of rubella vaccine produces an antibody response in more than 95% of recipients. Vaccine-induced antibodies have been shown to persist for at least 16 years in the absence of endemic disease, providing long-term protection against clinical rubella for those who seroconvert.[11]

None of the rubella notifications in 2008 were identified as Indigenous, although of the 37 notifications, 12 were reported as unknown indigenous status.

Tetanus

In 2008, there were 4 notifications of tetanus, one each reported from New South Wales, Victoria, Western Australia and Queensland and were all aged greater than 70 years. Of the 4 notifications, three were female and one was male.

Varicella-zoster infections

In November 2005, the varicella zoster 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 as a catch-up dose at 10–13 years of age. In 2006, CDNA agreed to make varicella infections notifiable in Australian jurisdictions. Three categories of varicella infection are notifiable: chickenpox, shingles and varicella infection (unspecified).

By the end of 2008, all jurisdictions except New South Wales were sending data to NNDSS, however because varicella only became notifiable in Victoria on 21 September 2008, the reported notifications for 2008 are incomplete and may underestimate actual disease incidence.

New South Wales decided in 2006 not to make varicella infections notifiable however varicella surveillance occurs in this state through monitoring of emergency department presentations available from http://www.health.nsw.gov.au/data/diseases/chickenpox.asp

In 2008, there were 8,526 varicella notifications from the 7 notifying jurisdictions, with 21% (1,790/8,526) reported as chickenpox, 27% (2,309/8,526) as shingles and 52% (4,427/8,526) as unspecified varicella infection.

Varicella zoster infection (chickenpox)

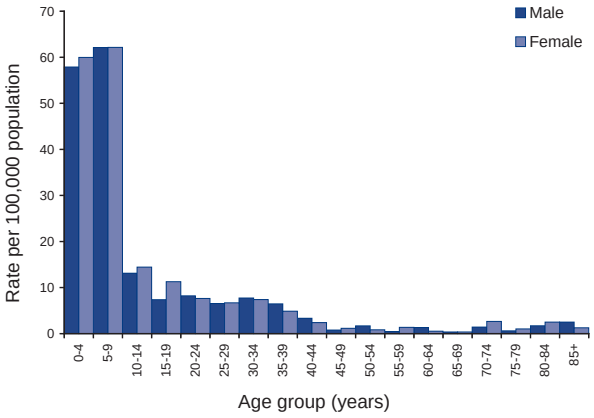
In 2008, there were a total of 1,790 notifications of chickenpox reported from all jurisdictions except New South Wales, corresponding to a rate of 12.4 notifications per 100,000 population. The highest rates were reported from the Northern Territory (52.3 per 100,000 population; 115 notifications) and South Australia (38.7 per 100,000 population; 620 notifications).

A total of 1,203 notifications (67.2 %) occurred in children aged less than 10 years. The highest rates were in the 5–9 year age group (62.2 per 100,000 population; 651 notifications) (Figure 54).

Indigenous status was recorded for 87% (1,554/1,790) of notifications, the majority (91%; 1,418/1,554) of which were non-Indigenous.

Of the 1,790 notifications for chickenpox, information on vaccination was available for 30% (543/1,790), 80% (432/543) of these were unvaccinated.

Figure 54: Notification rate for chickenpox, Australia,* 2008, by age group and sex



* Excluding New South Wales.

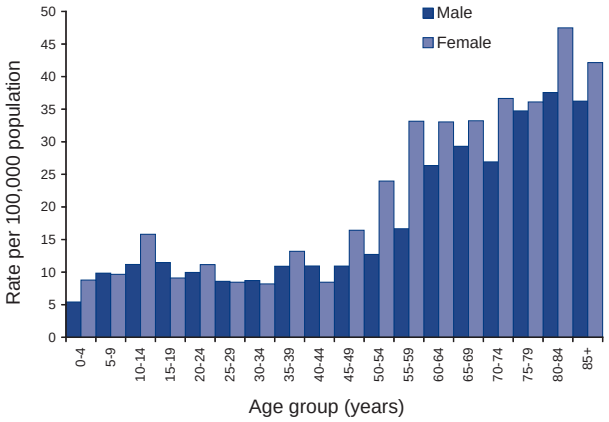
Varicella zoster infection (shingles)

There were 2,309 notifications of shingles reported to NNDSS in 2008 from 7 jurisdictions, corresponding to a rate of 16 notifications per 100,000 population. The highest rates were in South Australia (58.1 per 100,000 population, 931 notifications) and the Northern Territory (48.2 per 100,000 population, 106 notifications).

There were more female notifications (852; 55.1%) than males (695; 44.9%). The highest rates were in the 80–84 year age group (43.7 per 100,000 population; 121 notifications. (Figure 55).

Indigenous status was recorded for 81% (1,881/2,309) of notifications with the majority (96%; 1,803/1,881) reported as non-Indigenous.

Figure 55: Notification rate for shingles, Australia,* 2008, by age group and sex



* Excluding New South Wales.

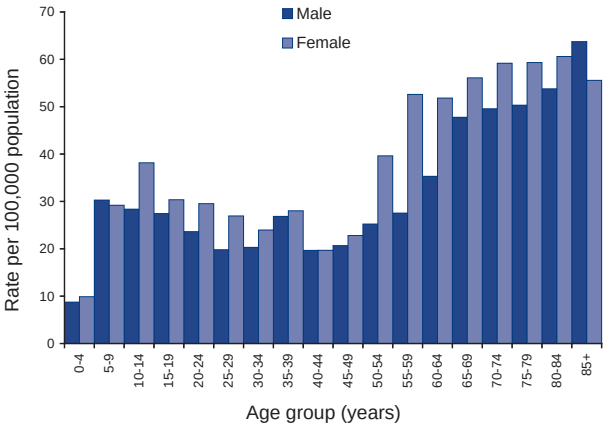
Varicella zoster infection (unspecified)

There were 4,427 notifications of varicella infections (unspecified) based on laboratory diagnoses from 7 jurisdictions in 2008, corresponding to a rate of 30.6 notifications per 100,000 population. The high proportion of unspecified varicella zoster virus infection compared with varicella zoster chickenpox or shingles is directly attributable to the varying capacity of jurisdictions to follow-up on laboratory notifications to determine the clinical presentation of each case. The highest rates were reported from Queensland (73.1 per 100,000 population; 3,138 notifications), Western Australia (34.7 per 100,000 population; 754 notifications) and the Australian Capital Territory (29.5 per 100,000 population; 102 notifications).

There were more notifications in females (2,477; 56%) than males (1,949; 46%). The age distribution of unspecified varicella infections is shown in Figure 56.

Indigenous status was recorded for 29% (1,295/4,427) of notifications, with the majority (94%; 1,219/1,295) reported as non-Indigenous.

Figure 56: Notification rate for varicella zoster infection (unspecified), Australia,* 2008, by age group and sex



* Excluding New South Wales and Victoria.

Vectorborne diseases

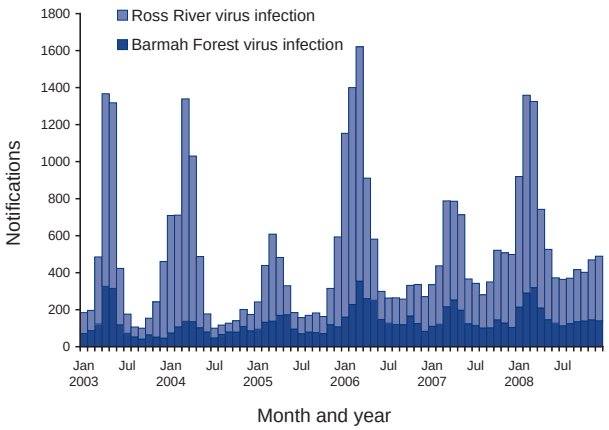
A disease that is transmitted to humans or other animals by an insect or other arthropod is called a vectorborne disease. Vectors of human disease of most concern in Australia are typically mosquitoes that are able to transmit viruses or parasites to humans.

During 2008, there were 8,876 notifications of mosquito-borne diseases reported to NNDSS (5.5% of total notifications). This was a 30% increase in the number of notifications compared with 2007 (6,828). The notifiable mosquito-borne diseases include those caused by the alphaviruses (Barmah Forest virus and Ross River virus), flaviviruses (the viruses causing dengue, Murray Valley encephalitis, Kunjin, Japanese encephalitis and yellow fever—which is reported under quarantinable diseases) and malaria. Geographical location rates for vectorborne disease notifications represent the place of residence rather than the place of acquisition of infection, although in many instances this may be the same. Further information about these vectorborne diseases can be found in the National Arbovirus and Malaria Advisory Committee annual (NAMAC) 07–08 annual report.[55]

Alphaviruses

Alphaviruses are single-stranded RNA viruses that cause disease epidemics characterised by fever, rash and polyarthritis. There is a variety of mosquito vectors for Barmah Forest virus (BFV) infection and Ross River virus (RRV) infection, which facilitates the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[56] In Australia, BFV and RRV are the alphaviruses of major public health significance, accounting for 87% (7,753 cases) of the total mosquito-borne disease notifications for 2008. Between 2003 and 2008 notifications ranged annually for BFV from 1,367 (2003) to 2,140 (2006), and for RRV from 2,540 (2005) to 5,651 (2008) (Figure 57).

Figure 57: Notifications of Barmah Forest and Ross River virus infections, Australia, 2003 to 2008, by month and year of diagnosis



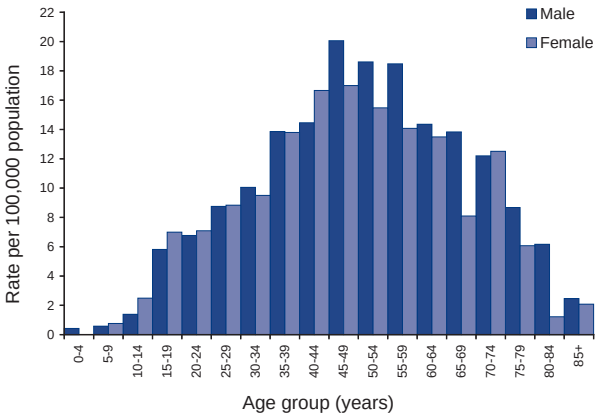
Barmah Forest virus infection

There were 2,102 notifications of BFV infections notified to NNDSS in 2008, which accounted for 24% of total mosquito-borne disease notifications for the reporting period. Fifty-nine per cent of BFV notifications were reported from Queensland (1,242 notifications) and 25% from New South Wales (533 notifications). BFV notifications during 2008 were 1.4 times the mean for the previous 5 years.

The highest rates of BFV notifications were reported by the Northern Territory (34.6 per 100,000 population compared with 42.3 per 100,000 population in 2007), Queensland (28.9 per 100,000 population compared with 19.8 per 100,000 population in 2007), and New South Wales (7.6 per 100,000 population compared with 8.3 per 100,000 population in 2007). Cases were reported in all jurisdictions. The national BFV notification rate in 2008 was 9.8 per 100,000 population, compared with 8.1 per 100,000 population in 2007. Notification rates for BFV varied by geographic location.

Figure 58 shows the age and sex distribution of BFV notifications. The BFV notification rate was highest amongst the 45–49 year age group (18.5 per 100,000 population). Overall, 52% of notifications reported to NNDSS were males.

Figure 58: Notification rate for Barmah Forest virus infections, Australia, 2008, by age group and sex



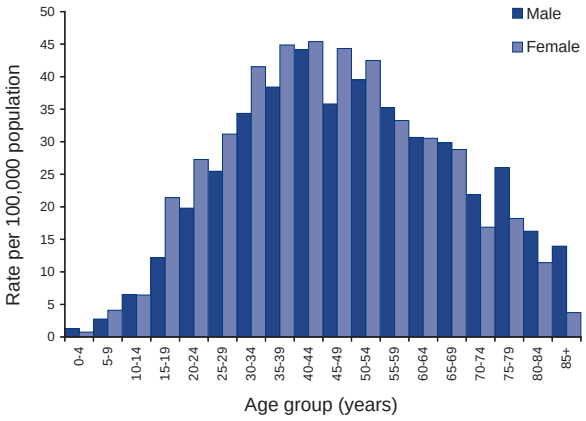
Ross River virus infection

There were 5,651 notifications of RRV infections reported to NNDSS in 2008, which accounted for 63% of the total mosquito-borne disease notifications received during this period.

Notification rates varied by geographic region, but the majority of notifications in 2008 were from Queensland (50%, 2,838 notifications) and New South Wales (20%, 1,152 notifications). The national RRV notification rate for 2008 was 26.4 per 100,000 population compared with 20.0 per 100,000 population in 2007.

The age and sex distribution of RRV notifications is shown in Figure 59. The RRV national notification rate was highest in the 40–44 year age group (44.9 per 100,000 population). Overall, 47% of notifications reported to NNDSS were males.

Figure 59: Notification rate for Ross River virus infections, Australia, 2008, 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 virus (MVEV), Kunjin virus (KUNV), Japanese encephalitis virus (JEV) and dengue viruses (DENV).

The Sentinel Chicken Program is a surveillance scheme involving New South Wales, the Northern Territory, Victoria and Western Australia. Chicken flocks are located in strategic locations and are regularly tested for antibodies to MVEV and KUNV. This program is designed to provide early warning of flavivirus activity (excluding dengue and JEV).[57] A sentinel chicken surveillance report was published as part of the NAMAC annual report 2007–08.55

Murray Valley encephalitis virus infection

There were 2 cases of MVEV reported to NNDSS in 2008 compared to no cases in 2007. One case

was a 60-year-old male from New South Wales who recovered and the other case was a 49-year-old male from Western Australia, who died from the infection.[58]

Kunjin virus infection

During 2008, 1 case of KUNV was reported to NNDSS from Queensland compared with 1 notification in 2007 from Victoria.

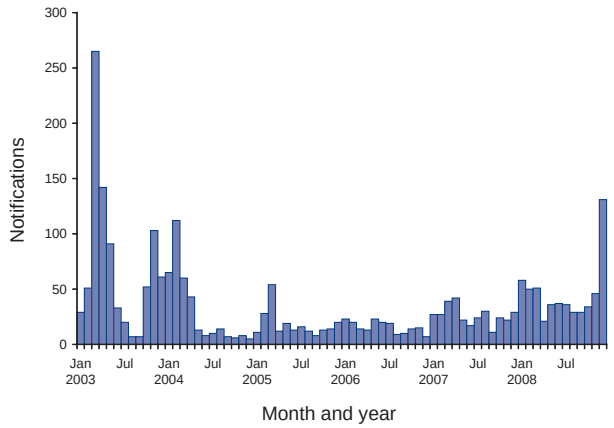
Dengue virus infection

There were 558 notifications of DENV reported to NNDSS in 2008 (Figure 60), of which 75% were acquired overseas. The number of cases reported in 2008 was a 78% increase in the number of cases reported in 2007 (314).

Local transmission in Australia is restricted to areas of northern Queensland where the key mosquito vector, *Aedes aegypti*, is present. Dengue is not endemic to Queensland, but outbreaks can occur when the virus is introduced via international travellers or residents returning home from overseas. Queensland reported 232 notifications of DENV in 2008 (41% of all DENV notifications). Locally-acquired cases represented 25% (137/558 cases) of the total number of dengue notifications for Queensland in 2008, which were mainly attributable to an outbreak of locally-acquired dengue serotype 3 in Cairns that occurred between November and December 2008.

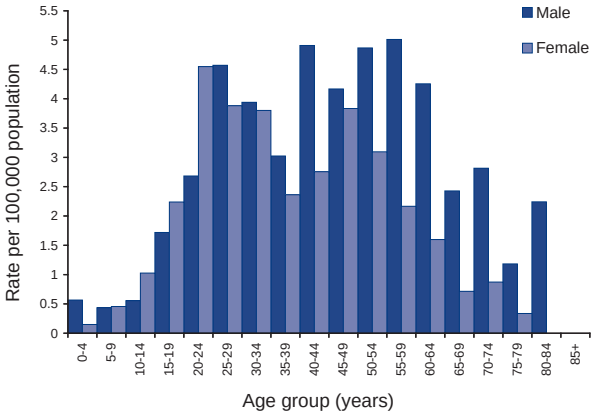
In early 2004, 2 deaths due to dengue fever were reported in Australia. These were the first deaths attributed to dengue in over 100 years and there have been no other deaths reported since.[59]

Figure 60: Notifications of dengue, Australia, 2003 to 2008, by month and year of diagnosis



In 2008, 57% of DENV notifications were male (318 notifications) and 90% of notifications were aged between 15 and 64 years (503 notifications). The highest notification rate for males was in the 55–59 year age group (4.2 per 100,000 population) and in females was in the 20–24 year age group (4.5 per 100,000 population) (Figure 61).

Figure 61. Notification rate for locally-acquired and imported cases of dengue, Australia, 2008, by age group and sex



Japanese encephalitis virus infections

There was 1 case of JEV notified in New South Wales in 2008 in a man who had recently travelled to Japan. This was the first JEV case notified in Australia since 2004.

Arbovirus infections (NEC)

In 2008, there were 28 notifications of arbovirus infection (not elsewhere classified or NEC). Twenty-one notifications in Queensland, 6 notifications in Victoria and 1 notification in New South Wales.

Of the Queensland notifications, 4 cases were further identified as Kokobera virus infection.

Malaria

There were 533 notifications of malaria in Australia in 2008, compared with 568 notifications in 2007 (Figure 62). There were no locally-acquired infections in 2008. Since Australia was declared malaria free in 1981 there have been two reported locally acquired outbreaks in 1986 and 2002 respectively, with a total of 15 cases. The majority of cases were reported by Queensland (31%; 167), New South Wales (22%; 116), Victoria (20%;

105), and Western Australia (16%; 85). Queensland reported that 79 (47%) of 167 notifications were acquired in Papua New Guinea, which was similar to 2007.

The largest number (70) of malaria notifications was in the 20–24 year age group and 69% of malaria notifications were for males (Figure 63).

The infecting *Plasmodium* species was reported for 98% of malaria notifications in 2008 (Table 16). Of these 533 notifications, *P. falciparum* (43%) and *P. vivax* (50%) were the predominant species.

Zoonoses

Zoonoses are 'those diseases and infections which are naturally transmitted between vertebrate animals and man'.[60] Approximately 60%–70% of emerging human infectious diseases are zoonoses[61,62] and more than 70% of emerging zoonoses originate from wildlife.[61] An emerging zoonosis is defined by WHO as 'a zoonosis that is newly recognised or newly evolved, or that has occurred previously but shows an increase in incidence or expansion in geographical, host or vector range'.[63]

The zoonoses notifiable to the NNDSS included in this chapter are anthrax, Australian bat lyssavirus (ABL) or lyssavirus (NEC) infection, brucellosis, leptospirosis, ornithosis, Q fever, and tularaemia. During 2008, the zoonotic diseases notified to NNDSS were for brucellosis, leptospirosis, ornithosis, and Q fever with a total of 633 notifications to NNDSS. Notifications were generally higher in males (72%, 453 notifications). There were only 20 notifications (3%) in cases aged less than 15 years and 27 notifications (4%) in cases over the age of 70 years.

Figure 62. Notifications of imported cases of malaria, Australia, 2003 to 2008, by month and year of diagnosis

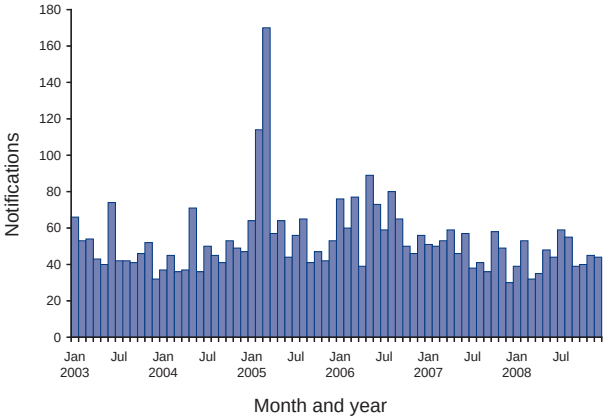


Figure 63: Notifications of malaria, Australia, 2008, by age group and sex

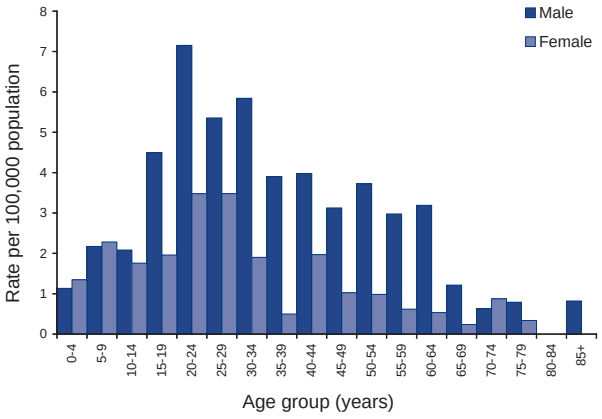


Table 16. Notifications of malaria, Australia, 2008, by parasite type and state or territory

Parasite type	State or territory									Type (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
Plasmodium falciparum	2	42	13	71	11	4	26	57	226	43
Plasmodium malariae	1	2	0	2	0	1	0	3	9	2
Plasmodium ovale	0	6	0	3	0	0	0	0	9	2
Plasmodium vivax	12	65	5	82	5	3	73	20	265	50
Plasmodium species	0	1	0	9	1	0	0	2	13	2
Mixed *P. falciparum* and other species*	0	0	1	0	0	0	5	1	7	1
Mixed other species*	0	0	0	0	0	0	0	2	2	1
Total	15	116	19	167	17	8	104	85	531	

* New South Wales, South Australia, Tasmania, Victoria, Western Australia and the Northern Territory report mixed species infections per notified case. Queensland and the Australian Capital Territory report 1 notification for each species in a mixed infection.

Several zoonoses notifiable to the NNDSS are included under other headings in this report. A zoonotic infection can be acquired directly from an animal or indirectly via an insect vector, the environment or contaminated food. For example, *Salmonella* and *Campylobacter* infections are typically acquired from contaminated food and are listed under the gastrointestinal diseases section.

Anthrax

Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts.[17] Anthrax has a low incidence in animals, and occurs only sporadically in Australia.[64] It can be an occupational hazard for veterinarians, and agriculture, wildlife and industry livestock workers who handle infected animals or by-products.

No cases of anthrax were reported to NNDSS in 2008. Over the previous 10 years, only 3 human cases of anthrax have been reported in Australia, all which were the cutaneous form, in 1998, 2006 and 2007.[65–67] Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

In 2008, 12 outbreaks of anthrax were reported in livestock. Ten outbreaks occurred in New South Wales, where cases have been known to occur in the past, and two in northern Victoria. In all instances, 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 food chains. During 2008, an 'animal side' immunochromatographic test was used as a rapid anthrax screening test to investigate sudden ruminant deaths. The results of this testing were consistent with confirmatory blood cultures and will continued to be used in Victoria.[64]

Australian bat lyssavirus and lyssavirus (NEC) infections

No cases of either ABL or lyssavirus (NEC) infections were notified during 2008. Only 2 known cases of ABL infection in humans have been reported in Australia, in 1996 and 1998. Both cases occurred after close contact with an infected bat and both cases were fatal.[21]

Surveillance indicates ABL is and may have been present in Australian bats for at least 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.[68] However, bat testing conducted by the Australian Wildlife Health Network between January and December 2008 yielded no ABL detections compared with 8 detections in bats during 2007.[69]

Brucellosis

Brucellosis is mainly an occupational disease for farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[70] However, the most common source of human infection in Australia is from infected feral pigs and inadequate measures by feral pig hunters to prevent brucellosis infection.[71]

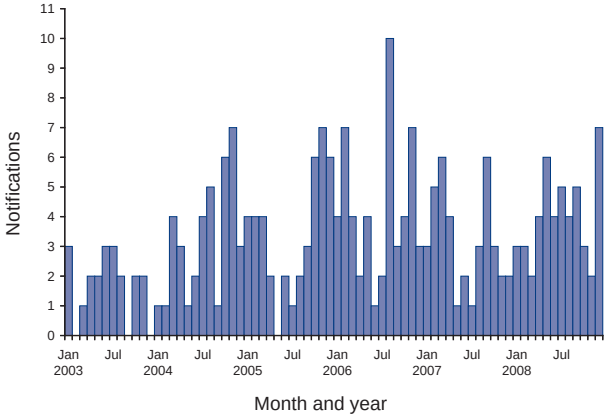
Several *Brucella* species can infect both animals and humans. Infections that can cause illness in humans include *Brucella melitensis* from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle.

In 2008, 48 cases of brucellosis were reported to the NNDSS; a national notification rate of 0.2 per 100,000 population. Queensland reported 46 cases, with New South Wales reporting the remaining 2 cases. There has been little change in the number of notifications of brucellosis over the last 6 years (Figure 64). The national notification rate for brucellosis was the same in 2008 as in 2007. The majority of cases were male (38) and aged between 15 and 49 years (40).

Species data were available for 14% of notifications (7) and all of these were *B. suis* (all from Queensland).

Bovine brucellosis (*B. abortus*) was eradicated from the Australian cattle herd in 1989 and is presently considered an exotic animal disease in Australia.[64]

Figure 64: Notifications of brucellosis, Australia, 2003 to 2008, by month and year of diagnosis



Caprine and ovine brucellosis (caused by *B. melitensis*) have never been reported in Australian sheep or goats. [64] Swine brucellosis (caused by *B. suis*) is confined to small areas of Queensland, where it occurs in feral pigs, with human cases predominantly seen in recreational feral pig hunters.[64,71] Swine brucellosis was not detected in any of Queensland's domestic piggeries during 2008.[64]

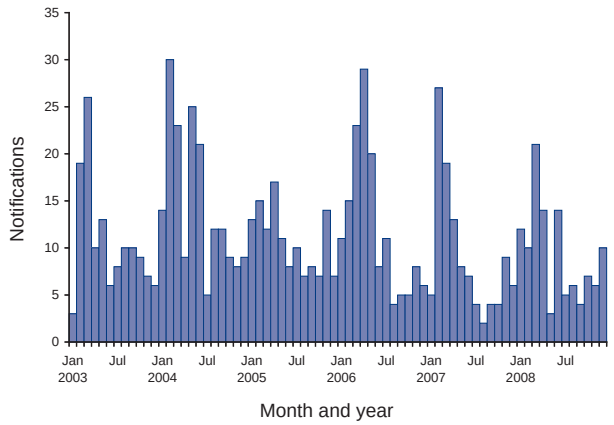
Leptospirosis

Leptospirosis is caused by spirochaetes of the genus, *Leptospira,* which are 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 (such as swimming or wading in contaminated water).[17]

Between 2003 and 2008 leptospirosis notifications ranged annually from 108 (2007) to 177 (2004), with 112 notifications in 2008 (0.5 per 100,000 population). Cases were reported in all jurisdictions except for the Australian Capital Territory, South Australia and Tasmania (Figure 65). In 2008, the majority of notifications were from Queensland (89 notifications, 2.1 per 100,000 population). Ninety-two per cent of leptospirosis cases were male (103 notifications) and 58% of all cases were aged between 20 and 39 years (65 notifications).

The WHO/FAO/OIE Collaborating centre for reference and research on leptospirosis provides an annual surveillance report of leptospirosis cases in 2008. The most frequently identified leptospirosis serovars in 2008 were Arborea, Zanoni and Hardjo. Serovar Arborea was the most frequently reported during 2008, accounting for 24 (21%) of all notifications and was an increase from 8 (8%) notifications in 2007.[72]

Figure 65: Notifications of leptospirosis, Australia, 2003 to 2008, by month and year of onset

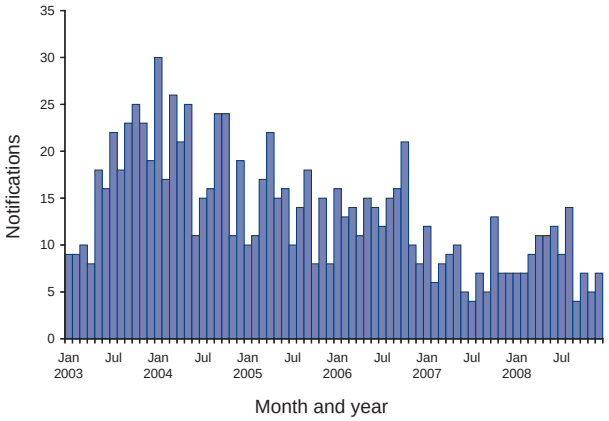


Ornithosis

Ornithosis is caused by infection with the bacteria *Chlamydia 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.[17] Person-to-person transmission is rare.

In 2008, there were 103 ornithosis infections notified to NNDSS, corresponding to a national rate of 0.5 per 100,000 population. This was similar to the 2007 rate of 0.4 per 100.000 population. Between 2003 and 2008, the annual number of ornithosis notifications ranged from 239 (2004) to 93 (2007) (Figure 66).

Figure 66: Notifications of ornithosis, Australia, 2003 to 2008, by month and year of diagnosis



Victoria had the highest number of notifications (53 notifications, 1.0 per 100,000 population). Notifications were also received from New South Wales (41 cases), Western Australia (6 cases) and Queensland (3 cases). Forty-seven per cent of the notifications in 2008 were male (48 notifications) compared to 2007 where the majority of cases were male (64%). All cases were aged 10 years or over and 83% of cases were aged 40 years or over (Figure 67).

People at risk of 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.[73]

Figure 67: Notifications of ornithosis, Australia 2008, by age group and sex

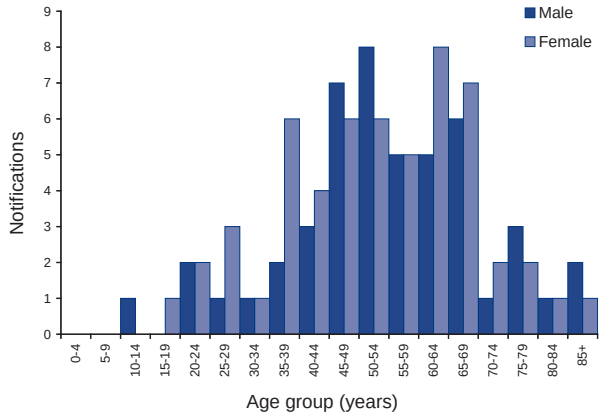
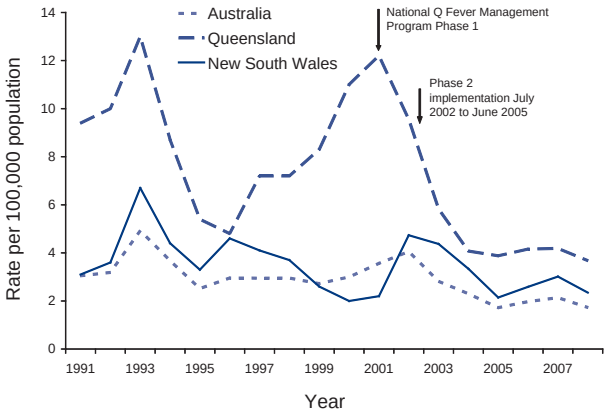


Figure 68: Notification rate for Q fever, Australia, New South Wales and Queensland, 1991 to 2008



Q fever

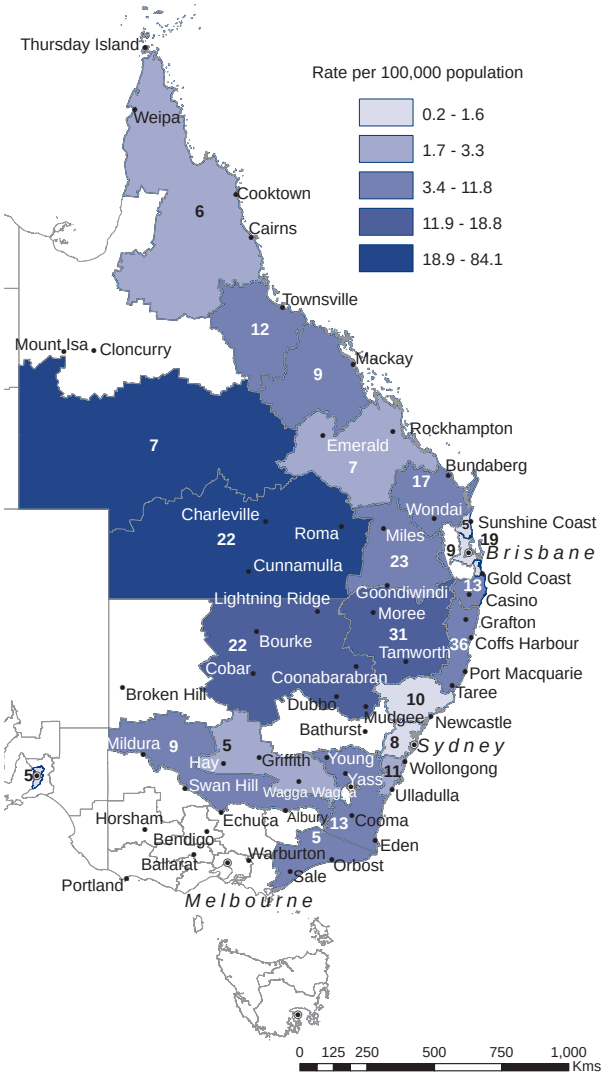
Q fever is caused by infection with the bacteria, *Coxiella burnetii*. Primary reservoirs of these bacteria are cattle, sheep and goats. These organisms are resistant to heat, drying and many common disinfectants, which enables the bacteria to survive for long periods in the environment. The mode of transmission to humans is most commonly by the airborne route through inhalation of contaminated 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.[17]

In 2008, 370 cases of Q fever were notified to the NNDSS, corresponding to a national rate of 1.7 per 100,000 population (Figure 68). Between 1991 and 2001, and prior to the introduction of the National Q Fever Management Program, Q fever notification rates ranged between 2.5 per 100,000 population and 4.9 per 100,000 population. The national notification rate for Q fever was lower in 2008 than in 2007 (1.7 and 2.1, respectively). Between 2003 and 2008, the annual number of Q fever notifications ranged from 560 (2003) to 351 (2005).

The highest notification rates were from Queensland (158 notifications, 3.7 per 100,000 population) and New South Wales (164 notifications, 2.3 per 100,000 population). On a regional basis, the South West Statistical Division of Queensland had the highest notification rate of 84 per 100,000 population (Map 4).

The highest age specific rates of Q fever for males was in the 55–59 year age group (32 notifications, 5.0 per 100,000 population), and for females in the 60–64 year age group (2.1 per 100,000

Map 4: Notification rates for Q fever in Queensland, New South Wales and Victoria, by Statistical Division of residence



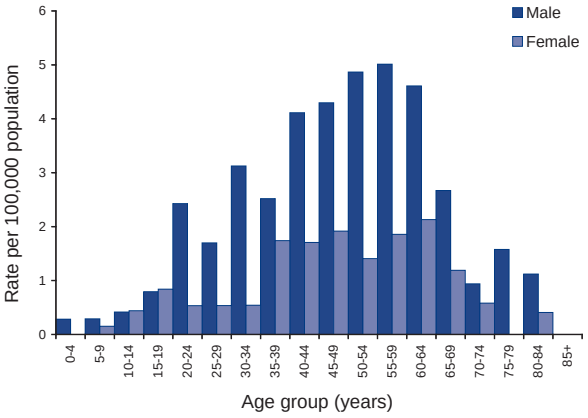
* Numbers shown in the Statistical Divisions represent the count of notifications.

Notification rates in geographic areas where estimated residential population and case numbers are small should be interpreted with caution.

population) (Figure 69). There were 11 notifications aged less than 15 years and 71% of notifications were male (264 cases).

Adults at risk of Q fever infection, including abattoir workers, farmers, veterinarians, stockyard workers, shearers and animal transporters should be considered for vaccination. The administration of the Q fever vaccine requires a pre-vaccination screening test to exclude those recipients with a previous (unrecognised) exposure to the organism. A Q fever vaccine may cause an adverse reaction in a person who has already been exposed to the bacterium. Vaccine is not recommended for children under 15 years of age.[11]

Figure 69: Notification rate for Q fever, Australia, 2008, by age group and sex



Tularaemia

Tularaemia is caused by infection with the bacteria *Francisella tularaensis.* The most common modes of transmission are through arthropod bites, handling infected animals, inhalation of infectious aerosols or exposure to contaminated food or water. Small mammals such as rodents, rabbits and hares are often the reservoir host.[74]

There were no notifications of tularaemia in 2008, and there has never been a case notified in Australia.

Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2008 and classified as 'other bacterial infections' in the NNDSS. A total of 1,795 notifications were included in this group in 2008, which accounted for 1.1% of all the notifications to NNDSS, a similar total and proportion as in 2007 (1,799 notifications and 1.2% of total).

Legionellosis

Legionellosis includes notifications of infections caused by all *Legionella* species that meet the national surveillance case definition. There were 271 notifications of legionellosis reported in 2008, corresponding to a national rate of 1.3 notifications per 100,000 population. This was an 11% decrease from the 306 notifications reported in 2007 (1.5 per 100,000 population). State and territory notification rates ranged from 0.2 notifications per 100,000 population in Tasmania to 3.2 notifications per 100,000 population in Western Australia.

In 2008, the largest number of legionellosis notifications were diagnosed in May (31 notifications, 11%) and December (27 notifications, 10%) (Figure 70). As observed in previous years, the largest number of notifications of *L. longbeachae* in 2008 occurred in the spring months (Figure 71). In previous years *L. pneumophila* notifications have peaked in autumn and spring, however, in 2007 and 2008 these peaks have occurred slightly later, in late autumn and summer.[75, 76]

In 2008, males accounted for 184 (68%) of the 271 notifications of legionellosis resulting in a male to female ratio of 2.1:1. There were no notifications in people under the age of 19 years. Overall, the age group with the highest notification rate was the 75–79 year age group (4.9 per 100,000 population, 27 notifications). The highest age and sex specific rates were observed in men aged 70–74 years (7.5 per 100,000 population, 24 notifications) and women aged 75–79 years (3.4 per 100,000 population, 10 notifications) (Figure 72). An infecting species analysis by age group shows that 92% of *L. longbeachae* notifications were reported in persons 45 years or older and is most predominant in the 75–79 year age group with 19 notifications (3.5 per 100,000 population). The proportion of *L. pneumophila* infections in persons aged 45 years or older was 82% and is most predominant in the 70–74 year age group with 12 notifications (1.8 per 100,000 population).

Data on the causative species were available for 260 (96%) of the legionellosis notifications: 158 (58%) were *L. longbeachae,* 97 (36%) were identified as *L. pneumophila* and 4 (1.5%) were *L. micdade*i or *L. bozemanii*. One notification was a co-infection of *L. longbeachae* and *L. bozemanii* (Table 17).

Figure 70: Notifications of legionellosis, Australia, 2004 to 2008, by month of diagnosis

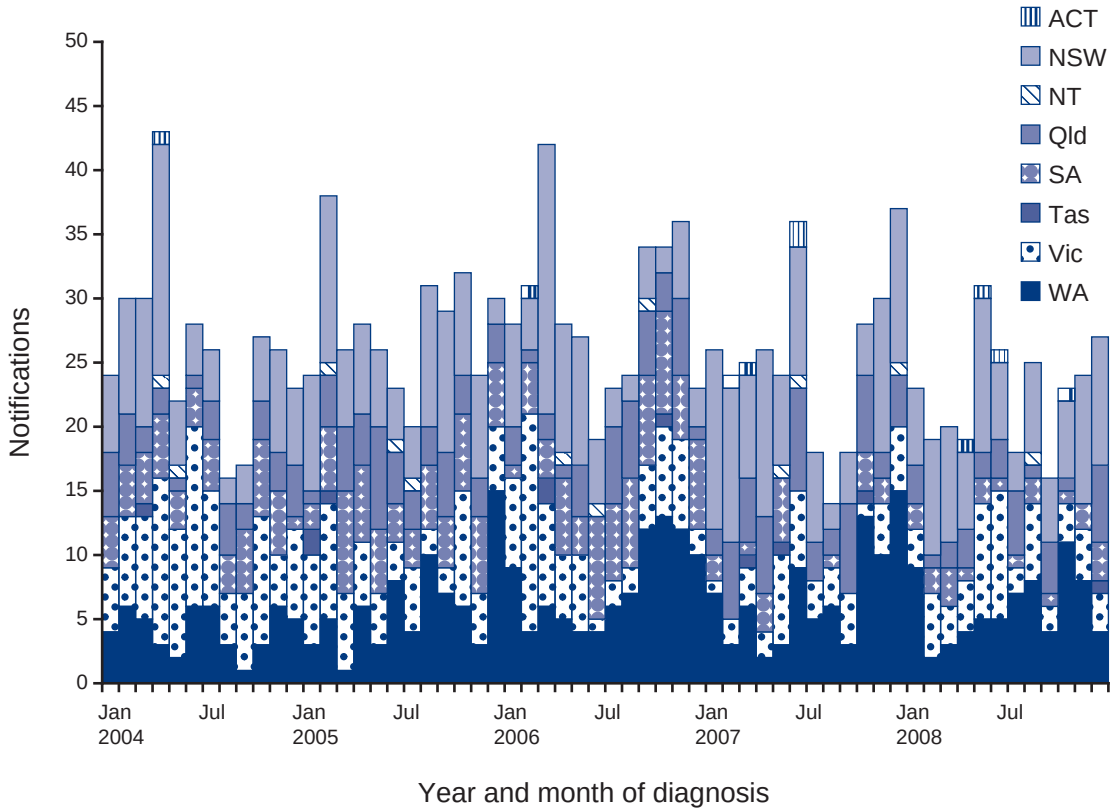


Figure 71: Notifications of legionellosis, Australia, 2004 to 2008, by month of diagnosis and species

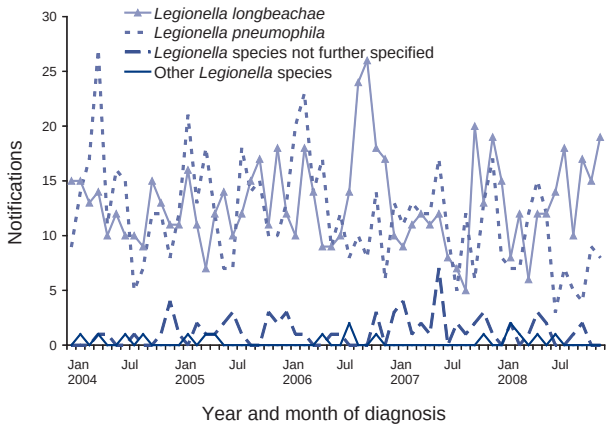
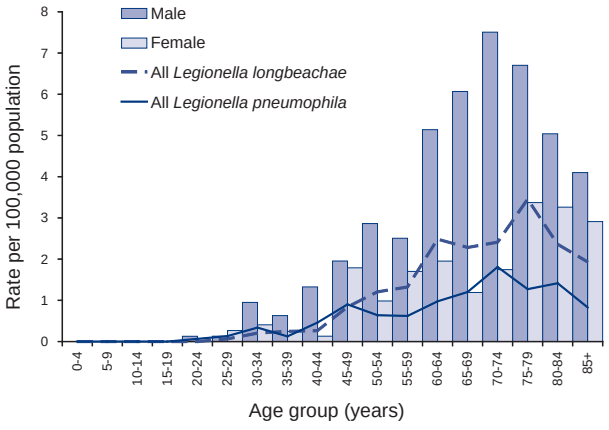


Figure 72: Notification rates of legionellosis, Australia, 2008, by age group and sex



Of the 97 *L. pneumophila* notifications, 56 (58%) were serogroup 1, 2 (2%) were serogroup 2 and 34 (35%) were reported without serogroup data.

Historically, there have been differences in the geographic distribution of *L. longbeachae* and *L. pneumophila*, with *L. longbeachae* making up the majority of species in notifications from South Australia and Western Australia, while *L. pneumophila* has been the most common infecting species in the eastern states (Queensland, New South Wales and Victoria). However, in 2008 *L. longbeachae* notifications were more common in the eastern states of Queensland and New South Wales than notifications of *L. pneumophila*.

Seven notifications of *L. pneumophila* serogroup 1 infection with disease onset dates between 11 April and 10 May 2008 were associated with an outbreak at a suburban car wash in Victoria. A molecular analysis indicated a microbiological link between isolates recovered from 2 patient

Table 17: Notifications of legionellosis, 2008, by species and state or territory

Species	State or territory								Total (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
*Legionella longbeachae**	0	51	0	17	18	1	8	63	158	58.3
Legionella pneumophila	0	37	1	12	2	0	40	5	97	35.8
Legionella micdadei†	0	0	0	0	0	0	2	1	3	1.1
Legionella bozemanii	0	0	0	0	1	0	0	0	1	0.4
Legionella longbeachae and *bozemanii*	0	0	0	0	0	0	1	0	1	0.4
Unknown species	4	1	0	2	0	0	3	1	11	4.1
Total	4	89	1	31	21	1	54	70	271	100

* Four deaths.

† One death.

specimens and water samples from the car wash. A further 4 cases of *L. pneumophila* serogroup 1 notified during the period were residents of adjacent local government areas in Melbourne's southern suburbs although no definitive source for, or links between, these or any other cases notified in the 2nd quarter of 2008 were identified.[77]

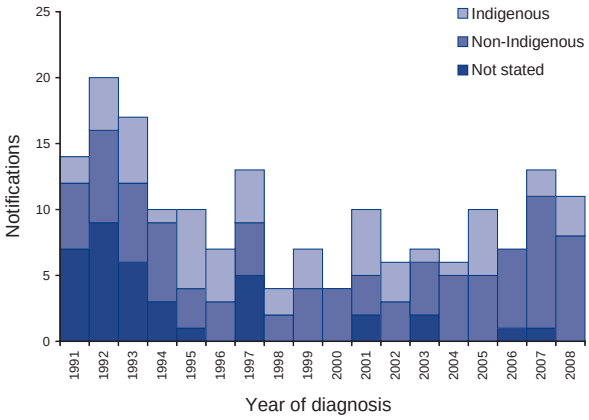
Mortality data were available for 134 (49%) notifications. There were 5 reported deaths due to legionellosis in Australia in 2008, which was similar to 2007. The age range for the deaths was between 59 and 92 years (median age being 79 years); all deaths were in males. There were 4 deaths associated with *L. longbeachae* infection and 1 death was associated with *L. micdadei* (Table 17). Mortality data should be interpreted with caution given the large proportion of cases without details of death outcomes, and the variability across jurisdictions to report legionellosis to the NNDSS as the primary and secondary cause of death.

Leprosy

Leprosy is a chronic infection of the skin and peripheral nerves caused by the bacterium *Mycobacterium leprae*. Leprosy is a rare disease in Australia, with the majority of cases occurring amongst migrants to Australia from leprosy-endemic countries and occasional locally-acquired cases from Indigenous communities. Trends in the numbers of leprosy notifications in Indigenous and non-Indigenous Australians are shown in Figure 73.

In 2008, 11 leprosy notifications were received compared with 13 in 2007. There were 4 notifications in New South Wales, 2 notifications each in Queensland, Victoria and Western Australia and 1 notification in the Northern Territory.

Figure 73: Notifications of leprosy in Indigenous and non-Indigenous Australians, 1991 to 2008



Eight notifications occurred in men and three in women. Three notifications were identified as Indigenous Australians. The age range of notified cases was 25–79 years (median 41 years).

Invasive meningococcal disease

Historically, in Australia, *Neisseria meningitidis* serogroups B and C have been the major cause of invasive meningococcal disease (IMD). There has been a marked decrease in rates for IMD due to *N. meningitidis* serogroup C infections following the introduction of the National Meningococcal C Vaccination Program by the Australian Government in 2003. In 2008, coverage of children aged 12 months immunised with meningococcal serogroup C vaccine reached 92.6% (data provided by the National Centre for Immunisation Research and Surveillance).

In 2008, there were 286 notifications of IMD, a 7% decrease from the 306 notifications in 2007,

and the lowest number of notifications since 1996. Since 2003, the notification rates have decreased from 2.8 notifications per 100,000 populations to 1.3 notifications per 100,000 population in 2008.

In 2008, males accounted for 53% of IMD notifications (153 notifications), giving a male to female ratio of 1.1:1. The largest number of notifications was diagnosed in July (Figure 74). The majority of notifications (275 notifications, 96%) were laboratory confirmed, through the isolation of *Neisseria meningitidis or* detection of specific meningococcal DNA sequences through nucleic acid amplification. There were an additional 11 notifications (4%) reported as probable diagnosis, based on clinical symptoms only.

Of the 286 IMD notifications in 2008, 221 (77%) were caused by serogroup B organisms, 21 (7%) were serogroup C, 8 (3%) were serogroup W135, 7 (2%) were serogroup Y, and 29 (10%) were reported with an unknown serogroup (Table 18). Serogroup C infections were confined to the eastern seaboard states; New South Wales, Queensland and Victoria. In comparison, in 2007 of 306 notifications, 212 (69%) were serogroup B, 20 (7%) were serogroup C and 43 (14%) were reported with an unknown serogroup.

The highest age specific IMD notification rate in 2008 was in children aged 0–4 years (7.2 notifications per 100,000 population). Of the notifications reported in this age group, 85% were serogroup B, this was also the age group with the highest age specific rate for serogroup B infection (6.1 notifications per 100,000 population).

Although there is no vaccine available to protect against serogroup B infections in Australia, the notification rates for IMD due to serogroup B infections has declined in most age groups over the period 2003 to 2008 (Figure 75). The highest notification rate for serogroup B infections was 6.1 notifications per 100,000 population in the 0–4 year age group (84 notifications) in 2008. This represents a 34% decline from the rate in 2003 (9.5 per 100,000 population, 121 notifications). The serogroup B notification rate in the 5–9 year age group saw a 54% decline in the notification

Figure 74: Trends in notification rates of invasive meningococcal disease, Australia, 2003 to 2008, by month of diagnosis and serogroups B and C

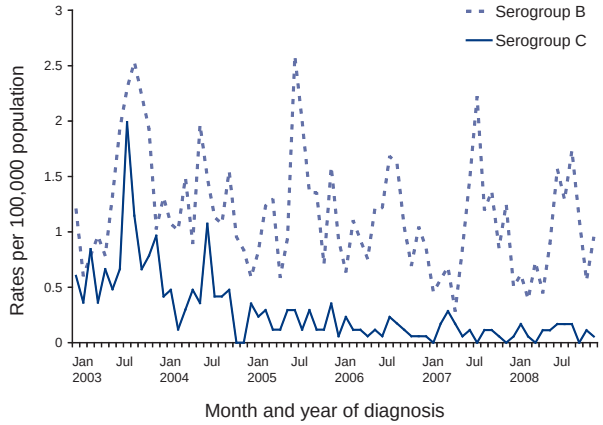


Figure 75: Notification rate for serogroup B invasive meningococcal disease, Australia, 2003 – 2008, by select age group

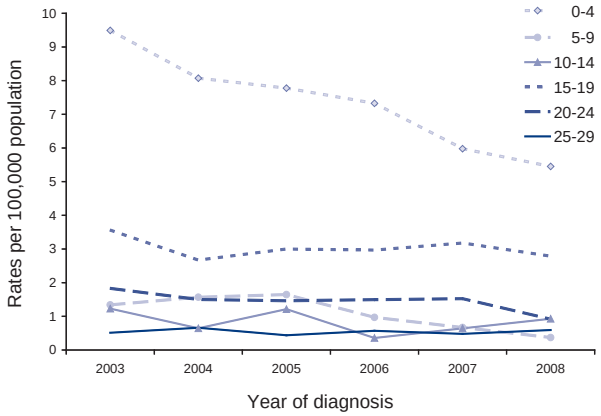


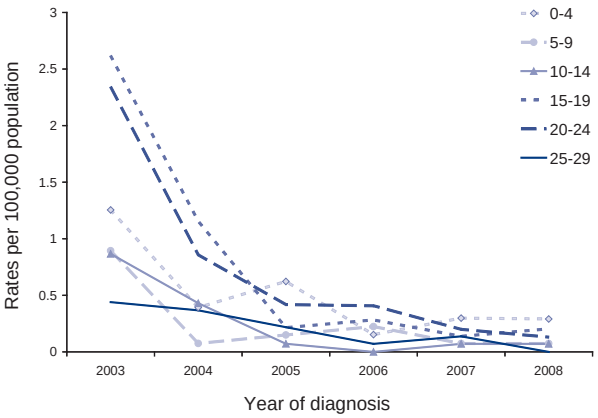
Table 18: Notifications of invasive meningococcal disease, Australia, 2008, by serogroup and state or territory

Serogroup	State or territory								Total	
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	(%)
Serogroup B	2	50	4	72	19	1	50	23	221	77.3
Serogroup C	1	9	4	5	0	0	2	0	21	7.3
Serogroup W135	0	5	0	2	1	0	0	0	8	2.8
Serogroup Y	0	3	0	1	0	0	3	0	7	2.4
Unknown serogroup	0	14	0	5	0	0	9	1	29	10.1
Total	3	81	8	85	20	1	64	24	286	100.0

rate from 1.3 per 100,000 population (18 notifications) in 2003 to 0.6 per 100,000 population (8 notifications) in 2008.

Notification rates for IMD due to serogroup C infections remained low in all age groups in 2008 (Figure 76). Since 2003, the largest decline has been in the 20–24 year age group with 0.1 notifications per 100,000 population (2 notifications) in 2008 compared with 2.3 notifications per 100,000 population (32 notifications) in 2003; an overall decline of 94.4%. The notification rate in the 15–19 year age group fell from 2.6 notifications per 100,000 population (36 notifications) to 0.2 notifications per 100,000 populations (3 notifications) over the same period; a 92.2% decline. Rates in the 0–4 year age group fell from 1.3 notifications per 100,000 population in 2003 (16 notifications) to 0.3 notifications per 100,000 population (4 notifications) in 2008.

Figure 76: Notification rate for serogroup C invasive meningococcal disease, Australia, 2003 to 2008, by select age group



Mortality data for IMD were available for 145 notifications (51%). Of these notifications, there were 7 deaths (6 serogroup B and 1 serogroup C) due to IMD in 2008. This was a decrease from 9 deaths in 2007 (mortality data were provided for 40% of notifications in 2007). Mortality data should be interpreted with caution given the large proportion of cases without details of death outcomes, and the variability across jurisdictions to report meningococcal to the NNDSS as the primary and secondary cause of death.

Laboratory based meningococcal disease surveillance

The Australian Meningococcal Surveillance Program (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *Neisseria meningitidis* from cases of IMD 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 2008 have recently been published.[78]

In 2008, there were 260 laboratory confirmed cases of IMD. Consistent with the NNDSS data, the AMSP reported that 85% were identified as serogroup B (223 notifications) and 6.5% (17 notifications) were serogroup C. No evidence of meningococcal capsular 'switching' was detected. About three-quarters of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). All isolates remained susceptible to ceftriaxone. One isolate had reduced susceptibility to rifampicin and two had reduced susceptibility to ciprofloxacin.

Tuberculosis

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 2008, 1,228 TB notifications were received by the NNDSS, corresponding to a rate of 5.7 notifications per 100,000 population. In 2007 there were 1,174 notifications (5.6 per 100,000 population). The notification rate of TB was higher than the national average in the Northern Territory (14.6 notifications per 100, 000 population), New South Wales (7.2 per 100,000 population), and Victoria (7.1 per 100,000 population. The lowest rate occurred in Tasmania (1.6 per 100,000 population).

Further details and analysis of TB notifications can be found in the tuberculosis annual report series to be published in *CDI.*

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Appendices

Appendix 1: Mid-year estimate of Australian population, 2008, by state or territory

	State or territory								Aus
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Male	171,348	3,460,125	113,997	2,145,760	791,597	245,374	2,631,983	1,099,406	10,660,917
Female	174,203	3,524,047	105,821	2,148,155	811,764	252,155	2,681,840	1,071,791	10,770,864
Total	345,551	6,984,172	219,818	4,293,915	1,603,361	497,529	5,313,823	2,171,197	21,431,781

Source: ABS 3201.0 Population by Age and Sex, Australian States and Territories. June 2008 population (http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3201.0Jun%202006)

Appendix 2: Mid-year estimate of Australian population, 2008, by state or territory and age group

Age group	State or territory								Aus
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
0–4	22,447	439,634	18,093	287,674	94,284	32,096	337,862	143,035	1,375,267
5–9	20,442	439,831	17,498	282,088	94,738	31,288	323,326	138,804	1,348,195
10–14	21,266	452,389	16,708	294,786	100,898	33,735	336,313	146,014	1,402,314
15–19	24,329	474,627	16,448	301,727	107,266	34,434	359,433	152,736	1,471,149
20–24	29,997	486,691	17,841	307,406	111,205	30,867	388,120	158,314	1,530,590
25–29	29,628	495,230	19,335	301,693	102,800	28,143	382,295	153,672	1,512,964
30–34	26,479	485,117	18,303	292,684	99,902	28,486	372,839	149,033	1,472,985
35–39	26,904	515,115	18,247	321,871	112,778	33,788	404,328	165,120	1,598,345
40–44	24,527	485,845	16,266	303,718	113,083	33,626	379,466	159,104	1,515,862
45–49	25,037	504,069	15,867	309,680	117,724	37,455	380,389	159,968	1,550,374
50–54	22,655	457,946	14,012	278,797	109,699	35,316	344,862	145,943	1,409,430
55–59	20,670	415,275	11,766	257,900	102,509	33,367	311,919	131,309	1,284,914
60–64	16,712	369,600	8,260	227,457	90,800	30,228	274,503	109,724	1,127,395
65–69	11,075	275,791	5,070	164,403	67,560	22,420	205,834	79,854	832,096
70–74	8,167	225,166	2,803	124,075	55,671	17,666	167,956	61,904	663,447
75–79	6,253	189,367	1,654	99,347	48,373	14,358	141,332	49,443	550,149
80–84	4,923	146,665	984	74,418	39,642	10,929	109,915	36,381	423,860
85+	4,040	125,814	663	64,191	34,429	9,327	93,131	30,839	362,445
Total	345,551	6,984,172	219,818	4,293,915	1,603,361	497,529	5,313,823	2,171,197	21,431,781

Source: ABS 3201.0 Population by Age and Sex, Australian States and Territories. June 2008 population (http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3201.0Jun%202006)

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2008, by notifiable disease*

Disease	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Donovanosis	2	0	0	0	0	0	2	100.0	2	0
Leprosy	3	0	0	8	0	0	11	100.0	11	0
Measles	0	0	0	65	0	0	65	100.0	65	0
Tetanus	0	0	0	4	0	0	4	100.0	4	0
Kunjin virus infection	0	0	0	1	0	0	1	100.0	1	0
Tuberculosis	32	2	1	1,170	23	0	1,228	98.1	1,205	23
Meningococcal infection	18	3	1	254	10	0	286	96.5	276	10
Haemophilus influenzae type b	3	0	0	21	1	0	25	96.0	24	1
Syphilis < than 2 years duration	180	0	3	1059	57	1	1,300	95.5	1,242	58
Typhoid	0	1	0	98	6	0	105	94.3	99	6
Hepatitis A	3	0	0	242	30	1	276	88.8	245	31
Hepatitis C (incident)	37	0	0	298	42	4	381	87.9	335	46
Haemolytic uraemic syndrome	1	1	0	25	4	0	31	87.1	27	4
Varicella zoster (chickenpox)	124	6	6	1,418	190	46	1,790	86.8	1,554	236
Legionellosis	3	0	0	231	37	0	271	86.3	234	37
Syphilis – congenital	2	0	0	4	1	0	7	85.7	6	1
Pneumococcal disease (invasive)	141	4	3	1,201	279	1	1,629	82.8	1,349	280
Shigellosis	310	2	2	361	149	4	828	81.5	675	153
Varicella zoster (shingles)	68	4	6	1,803	365	63	2,309	81.5	1,881	428
Hepatitis B (incident)	19	1	0	179	46	0	245	81.2	199	46
Malaria	3	6	1	421	102	0	533	80.9	431	102
Listeriosis	0	0	1	53	14	0	68	79.4	54	14
Mumps	110	0	0	110	66	0	286	76.9	220	66
Hepatitis E	0	0	0	33	11	0	44	75.0	33	11
STEC / VTEC	2	1	0	75	28	0	106	73.6	78	28
Gonococcal infection	3,281	133	56	2,057	2,193	3	7,723	71.6	5,527	2,196
Ornithosis	0	0	0	73	30	0	103	70.9	73	30
Rubella	0	0	0	25	12	0	37	67.6	25	12
Dengue	9	4	0	361	181	3	558	67.0	374	184

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2008, by notifiable disease,* *continued*

Disease	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Q fever	8	0	1	237	124	0	370	66.5	246	124
Leptospirosis	4	1	1	65	41	0	112	63.4	71	41
Syphilis > 2 years or unspecified duration	322	8	7	794	809	0	1,940	58.3	1,131	809
Hepatitis D	6	0	0	17	19	0	42	54.8	23	19
Pertussis	435	10	13	7,292	6,757	9	14,516	53.4	7,750	6,766
Cholera	0	0	0	2	1	1	4	50.0	2	2
Salmonellosis	377	13	12	3,713	4,069	126	8,310	49.5	4,115	4,195
Chlamydial infection	4,763	581	199	22,624	27,821	2496	58,484	48.2	28,167	30,317
Cryptosporidiosis	163	0	4	797	1,017	24	2,005	48.1	964	1,041
Campylobacteriosis	190	4	6	6,979	7,870	486	15,535	46.2	7,179	8,356
Hepatitis B (unspecified)	262	36	10	2,357	3,918	17	6,600	40.4	2,665	3,935
Hepatitis C (unspecified)	561	8	21	3,823	6,404	121	10,938	40.3	4,413	6,525
Brucellosis	2	0	0	17	29	0	48	39.6	19	29
Influenza (laboratory confirmed)	237	8	7	3,181	5,432	272	9,137	37.6	3,433	5,704
Arbovirus infection (NEC)	0	0	0	10	18	0	28	35.7	10	18
Ross River virus infection	86	9	5	1,776	3,518	257	5,651	33.2	1,876	3,775
Varicella zoster (unspecified)	64	4	8	1,219	2,967	165	4,427	29.3	1,295	3,132
Barmah Forest virus infection	35	2	2	449	1,545	69	2,102	23.2	488	1,614

* Indigenous status is usually obtained from medical notification and completeness varies by disease and by state and territory. This reflects differences in notification requirements (i.e. depending on the jurisdiction, some diseases are primarily or completely notified by pathology laboratories rather than clinicians) and the fact that it is not possible to follow-up all cases for diseases with a large volume of notifications and/or not requiring specific case-based public health action.

TSI Torres Strait Islander

Abbreviations

7vPCV	7 valent pneumococcal conjugate vaccine
23vPPV	23 valent pneumococcal polysaccharide vaccine
ABL	Australian bat lyssavirus
ABS	Australian Bureau of Statistics
AFP	acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AIDS	acquired immunodeficiency syndrome
AMSP	Australian Meningococcal Surveillance Programme
ANCJDR	Australian National Creutzfeldt-Jakob Disease Registry
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
DENV	dengue virus
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HUS	haemolytic uraemic syndrome
IMD	invasive meningococcal disease
IPD	invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
MMR	measles-mumps-rubella
MVEV	Murray Valley encephalitis virus
NAMAC	National Arbovirus and Malaria Advisory Committee
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NEC	not elsewhere classified
NIP	National Immunisation Program
NN	not notifiable
NNDSS	National Notifiable Diseases System
NPRL	National Polio Reference Laboratory
NSC	National Surveillance Committee
PCR	polymerase chain reaction
RRV	Ross River virus
SARS	severe acute respiratory syndrome
SD	Statistical Division
SSD	Statistical Subdivision
STEC	Shiga toxin-producing *Escherichia coli*
STI(s)	sexually transmissible infections(s)
TB	tuberculosis
VPD(s)	vaccine preventable disease(s)
VTEC	verotoxigenic *Escherichia coli*
WHO	World Health Organization
WPR	Western Pacific Region
WPV	wild-type polio virus

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National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
National Enteric Pathogens Surveillance Scheme
OzFoodNet Working Group
World Health Organization Collaborating Centre for Reference and Research on Influenza

State and territory health departments

Communicable Diseases Control, ACT Health, Australian Capital Territory
Communicable Diseases Surveillance and Control Unit, NSW Department of Health, New South Wales
Centre for Disease Control, Northern Territory Department of Health and Community Services, Northern Territory
Communicable Diseases Branch, Queensland Health, Queensland
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References

1. National Health Security Act, No 174. 2007. Available from: http://www.comlaw.gov.au/ComLaw/Legislation/Act1.nsf/0/A005BA0145A00248CA25736A00126AA5?OpenDocument Accessed November 2009.

2. National Notifiable Diseases List. 2008. Available from: http://www.comlaw.gov.au/ComLaw/legislation/LegislativeInstrument1.nsf/0/7162D634C6DD1BAACA25740B0079D6B8?OpenDocument Accessed November 2009.

3. National Health Security Agreement. 2008. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-nhs-agreement.htm Accessed November 2009.

4. National Centre in HIV Epidemiology and Clinical Research. *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report, 2009:* National Centre in HIV Epidemiology and Clinical Research, The University of New South Wales, Sydney; 2009.

5. Klug GM, Boyd A, Lewis V, McGlade AR, Roberts H, Douglass SL, et al. Surveillance of Creutzfeldt-Jakob Disease in Australia: 2008. *Commun Dis Intell* 2008;32(2):232–236.

6. Communicable Diseases Network Australia. National Notifiable Diseases Surveillance System. Available from: www.health.gov.au/nndssdata

7. Australian Bureau of Statistics. Population by age and sex, Australian States and Territories, Estimated Resident Population By Single Year of Age, Australia. Canberra: Australian Bureau of Statistics; 2008. Catalogue no: 3201.0.

8. Australian Bureau of Statistics. Australian Standard Geographical Classification (ASGC) Concordance, June 2008 Canberra: Australian Bureau of Statistics.

9. Medicare Australia. Australian Childhood Immunisation Register – Quarterly Coverage Report 30 June 2009. Canberra: Medicare Australia; 2009.

10. Australian Institute of Health and Welfare. National Health Data Dictionary 13.3; 2008.

11. National Health and Medical Research Council. *The Australian Immunisation Handbook* 9th edn. Canberra, Australia: Australian Government Department of Health and Ageing; 2008.

12. Medicare Australia. Australian Childhood Immunisation Register – Quarterly Coverage Report 30 September 2009. Canberra: Medicare Australia; 2009.

13. Medicare Australia. Australian Childhood Immunisation Register – Quarterly Coverage Report 31 December 2009. In. Canberra: Medicare Australia; 2009.

14. Medicare Australia. Australian Childhood Immunisation Register – Quarterly Coverage Report 31 March 2010. Canberra: Medicare Australia; 2010.

15. Razali K, Thein HH, Bell J, Cooper-Stanbury M, Dolan K, Dore G, et al. Modelling the hepatitis C virus epidemic in Australia. *Drug and Alcohol Dependence* 2007;91(2–3):228–235.

16. Butler T, Papanastasiou C. National Prison Entrants' Bloodborne Virus and Risk Behaviour Survey Report 2004 and 2007; 2008.

17. Heymann DL. *Control of Communicable Diseases Manual*. 19 edn. Washington: American Public Health Association, USA; 2008.

18. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2008. *Commun Dis Intell* 2009;33(4):389–413.

19. Hanna JH, SL. Humpreys, JL. Impact of hepatitis A vaccination on Indigenous children on notifications of hepatitis A in north Queensland. *Med J Aust* 2004;181(9):482–485.

20. Combs B, Raupach J, Kirk M. Surveillance of Shiga toxigenic *Escherichia coli* in Australia. *Commun Dis Intell* 2005;29(4):366–369.

21. Begg K, Roche P, Owen R, Liu C, Kaczmarek M, Hii A, et al. Australia's notifiable diseases status, 2006: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2008;32(2):139–207.

22. Cumpston JHL. *Health and disease in Australia*. Canberra: Australian Government Publishing Service; 1989.

23. Grattan-Smith PJ, O'Regan WJ, Ellis PS, O'Flaherty SJ, McIntyre PB, Barnes CJ. Rabies. A second Australian case with a long incubation period. *Med J Aust* 1992;156(9):651–654.

24. World Health Organization. The Global Eradication of Smallpox: Final Report of the Global Commission for the Certification of Smallpox Eradications, Geneva, December 1979. Geneva: World Health Organization; 1980.

25. Miller M, Roche P, Yohannes K, Spencer J, Bartlett M, Brotherton J, et al. Australia's notifiable diseases status, 2003: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2005;29(1):1–61.

26. World Health Organization. Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to the World Health Organization. Geneva: World Health Organization; 2009. Accessed on 9 February 2009. Available from: http://www.who.int/csr/disease/avian_influenza/country/cases_table_2009_02_05/en/index.html

27. Australian Government Department of Health and Ageing. Factsheets: Viral haemorrhagic fever. 2009. Accessed on 9 February 2009. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-communic-factsheets-vhf.htm

28. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2007: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2009;33(2):89–154.

29. Chen MY, Fairley CK, Donovan B. Nowhere near the point of diminishing returns: correlations between chlamydia testing and notification rates in New South Wales. *Aust N Z J Public Health* 2005;29(3):249–253.

30. Hocking J, Fairley C, Counahan M, Crofts N. The pattern of notification and testing for genital *Chlamydia trachomatis* infection in Victoria, 1998–2000: an ecological analysis. *Aust N Z J Public Health* 2003;27(4):405–408.

31. Australian Institute of Health and Welfare. Age-standardised rate – Identifying and definitional attributes. 2005. Accessed on 17 March 2010. Available from: http://meteor.aihw.gov.au/content/index.phtml/itemId/327276

32. Bowden F, Fairly C. Endemic STDs in Northern Territory: estimations of effective rates of partner change. In: Northern Territory RACP meeting; November 1996: Unpublished; 1996.

33. Queensland Health. Queensland HIV, Hepatitis C and Sexually Transmissible Infections Strategy: 2005–2011: Queensland Health; 2005.

34. Chen M, Donovan B. Genital *Chlamydia trachomatis* infection in Australia: epidemiology and clinical implications. *Sex Health* 2004;1(4):189–196.

35. Stephens N, O'Sullivan M, Coleman D, Shaw K. *Chlamydia trachomatis* in Tasmania 2001–2007: rising notification trends. *Aust N Z J Public Health* 2010;34(2):120–125.

36. Bowden FJ. Donovanosis in Australia: going, going. *Sex Transm Infect* 2005;81(5):365–366.

37. Australian Gonococcal Surveillance Programme. Annual report of the Australian Gonococcal Surveillance Programme, 2008. *Commun Dis Intell* 2009;33(3):268–274.

38. Tapsall JW, Limnios EA, Murphy D. Analysis of trends in antimicrobial resistance in *Neisseria gonorrhoeae* isolated in Australia, 1997–2006. *J Antimicrob Chemother* 2008;61(1):150–155.

39. Jin F, Prestage GP, Zablotska I, Rawstorne P, Kippax SC, Donovan B, et al. High rates of sexually transmitted infections in HIV positive homosexual men: data from two community based cohorts. *Sex Transm Infect* 2007;83(5):397–399.

40. Fairley CK, Hocking JS, Medland N. Syphilis: back on the rise, but not unstoppable. *Med J Aust* 2005;183(4):172–173.

41. Brotherton J, Wang H, Schaffer A, Quinn H, Menzies R, Hull B, et al. Vaccine preventable diseases and vaccination coverage in Australia, 2003 to 2005. *Commun Dis Intell* 2007;31 (Suppl):S1–S152.

42. Wang H, Deeks S, Glasswell A, McIntyre P. Trends in invasive *Haemophilus influenzae* type B disease in Australia, 1995–2005. *Commun Dis Intell* 2008;32(3):316–325.

43. Lambert SB, Faux CE, Grant KA, Williams SH, Bletchly C, Catton MG, et al. Influenza surveillance in Australia: we need to do more than count. *Med J Aust* 2010;193(1):43–45.

44. Bangor-Jones RD DG, Giele CM, van Buynder PG, Hodge MM, Whitty MM. A prolonged mumps outbreak among highly vaccinated Aboriginal people in the Kimberley region of Western Australia. *Med J Aust* 2009;191(7):4.

45. Stein-Zamir C, Shoob H, Abramson N, Tallen-Gozani E, Sokolov I, Zentner G. Mumps outbreak in Jerusalem affecting mainly male adolescents. *Euro Surveill* 2009;14(50).

46. High P, Handschur E, Eze O, Montana B, Robertson C, Tan C, et al. Update: mumps outbreak – New York and New Jersey, June 2009 – January 2010. *MMWR Morb Mortal Wkly Rep* 2010;59(5):4.

47. Wendelboe AM, van Rie A, Salmaso S, Englund JA. Duration of immunity against pertussis after natural infection or vaccination. *Pediatr Infect Dis J* 2005;24(5 Suppl):S58–S61.

48. Juretzko P, von Kries R, Hermann M, Wirsing von Konig CH, Weil J, Giani G. Effectiveness of acellular pertussis vaccine assessed by hospital-based active surveillance in Germany. *Clin Infect Dis* 2002;35:162–167.

49. Roberts J, Grant K, Ibrahim A, Thorley B. Annual report of the Australian National Poliovirus Reference Laboratory. *Commun Dis Intell* 2007;32(3):308–315.

50. Department of Health and Ageing. Communicable diseases surveillance – Highlights for 4th quarter, 2006. *Commun Dis Intell* 2007;31(1):135–138.

51. Kelly H, Worth L, Karapanagiotidis T, Riddell M. Interruption of rubella virus transmission in Australia may require vaccination of adult males: evidence from a Victorian sero-survey. *Commun Dis Intell* 2004;28(1):69–73.

52. Francis BH, Thomas AK, McCarty CA. The impact of rubella immunisation on the serological status of women of child-bearing age: a retrospective longitudinal study in Melbourne, Australia. *Am J Public Health* 2003;93(8):1274–1276.

53. Santhanandan D, Gupta L, Liu BH, Rutherford A, Lane J. Factors associated with low immunity to rubella infection on antenatal screening. *Aust N Z J Obstet Gynaecol* 2005;45(5):435–438.

54. Hunt JM, Lumley J. Top end rural and remote Indigenous women: an Australian population group vulnerable to rubella. *Commun Dis Intell* 2004;28(4):499–503.

55. Fitzsimmons G, Wright P, Johansen C, Whelan P. National Arbovirus and Malaria Advisory Committee annual report. *Commun Dis Intell* 2009; 33(2):155–170.

56. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.

57. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW, et al. Australian encephalitis: Sentinel Chicken Surveillance Programme. *Commun Dis Intell* 2001;25(3):157–160.

58. Western Australia Department of Health. Media release. Upgraded warning on mosquito-borne disease in the Kimberly and Pilbara. 29 April 2008.

59. McBride WJH. Deaths associated with dengue haemorrhagic fever: the first in Australia in over a century. *Med J Aust* 2005;183(1):35–37.

60. World Health Organization. Zoonoses. Technical report series no. 169. Geneva: World Health Organization; 1959.

61. Jones KE, Patel NG, Levy MA. Global trends in emerging infectious diseases. *Nature* 2008(451):990–994.

62. Woolhouse MEJ, Gowtage-Sequeria S. Host range and emerging and reemerging pathogens. *Emerg Infec Dis* 2005;11(12):1842–1847.

63. World Health Organization. Report of the WHO/FAO/OIE joint consultation on emerging zoonotic diseases. Geneva: World Health Organization; 2004.

64. Animal Health Australia. Animal Health in Australia 2008. Canberra; 2009.

65. McCall B, Looke D, Crome M, Nimmo G, O'Kane G, Harper J, et al. Sporadic human anthrax in urban Brisbane. *Commun Dis Intell* 1998;22(9):189–190.

66. Kolbe A, Yuen M, Doyle B. A case of human cutaneous anthrax. *Med J Aust* 2006;185(5):281–282.

67. Fielding J. Zoonoses: Anthrax. Victorian Infectious Diseases Bulletin 2007;10(2):47.

68. Field H. The ecology of Hendra virus and Australian bat lyssavirus. 2004. Accessed on 1 August 2009. Available from: http://espace.library.uq.edu.au/eserv.php?pid=UQ:13859&dsID=field_thesis_05.pdf

69. Australian Bat Lyssavirus Focus Group. Australian Bat Lyssavirus Report, December 2008: Australian Wildlife Health Network; 2008.

70. Hellard ME, Sinclair MI, Harris AH, Kirk M, Fairley CK. Cost of community gastroenteritis. *J Gastroenterol Hepatol* 2003;18:322–328.

71. Queensland Health:, Sweeny AL, Beard FH. Queensland Health Notifiable Diseases Report 2002–2006. Brisbane: Communicable Diseases Branch, Brisbane: Queensland Health.; 2009

72. Queensland Health Forensic and Scientific Services. National leptospirosis surveillance report number 17, January – December 2008. Accessed on 12 February 2009. Available from: http://www.health.qld.gov.au/qhcss/documents/lepto/08_annual.pdf

73. Victorian Department of Human Services. Blue Book. Revised Edition 2005. Accessed on 15 August 2009. Available from: http://www.health.vic.gov.au/ideas/bluebook

74. Whipp MJ, Davis JM, Lum GD, de Boer J, Zhou Y, Bearden SW, et al. Characterization of a *novicida* – like *Francisella tularensis* isolated in Australia. *J Med Microbiol* 2003;52:839–842.

75. Li J, O'Brien ED, Guest C. A review of national legionellosis surveillance in Australia, 1991 to 2000. *Commun Dis Intell* 2002;26:462–470.

76. O'Connor BA, Carman J, Eckert K, Tucker G, Givney R, Cameron S. Does potting mix make you sick? Results from a *Legionella longbeachae* case control study in South Australia. *Epidemiol Infect* 2007;135:34–39.

77. Stylianopoulos, Easton T, Veitch M. Reports of bloodstream infections and meningitis to the Victorian Hospital Pathogen Surveillance Scheme, January–June 2008. *Victorian Infectious Diseases Bulletin* 2008;11(23).

78. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2008. *Commun Dis Intell* 2009;33(3):259–267.

Arboviral diseases and malaria in Australia, 2008–09: Annual report of the National Arbovirus and Malaria Advisory Committee

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Abstract

The National Notifiable Diseases Surveillance System received 8,677 notifications of diseases transmitted by mosquitoes in Australia from 1 July 2008 to 30 June 2009. The alphaviruses, Barmah Forest and Ross River, accounted for 6,574 (78%) of these notifications during 2008–09. There were 1,009 notifications of dengue virus infection locally-acquired in North Queensland and 484 notified cases resulted from overseas travel. Notification rates of dengue virus infection for 2008–09, regardless of where infection was acquired, exceeded the five-year mean rate and may be attributed to increased disease activity in the Asia–Pacific region. North Queensland was the site of several outbreaks of locally-acquired dengue virus infection involving all 4 serotypes. These dengue outbreaks affected several locations with over 1,000 notifications. Detection of flavivirus seroconversions in sentinel chicken flocks across Australia provides an early warning of increased levels of Murray Valley encephalitis virus and Kunjin virus activity. Increased levels of flavivirus activity were detected in western and northern Australia, which prompted public health action. This action preceded 4 notifications of Murray Valley encephalitis infections, 2 (fatal) cases acquired in the Northern Territory and two in Western Australia. There were no notifications of locally-acquired malaria in Australia and 567 notifications of overseas-acquired malaria during 2008–09. This annual report presents information of diseases transmitted by mosquitoes in Australia and notified to the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2010;34(3):225–240.

Keywords: arbovirus; Barmah Forest virus, chikungunya, dengue, disease surveillance; epidemiology, flavivirus, Japanese encephalitis, Kunjin, malaria, mosquito-borne disease, mosquitoes, Murray Valley encephalitis virus, Ross River virus, yellow fever

Introduction

This report describes the surveillance of mosquito-borne diseases of public health importance in Australia from 1 July 2008 to 30 June 2009. It includes those diseases caused by the alphaviruses (Barmah Forest, chikungunya and Ross River), flaviviruses (dengue, Murray Valley encephalitis, Kunjin, Japanese encephalitis and yellow fever) and malaria.

The Australian Government Department of Health and Ageing established the National Arbovirus Advisory Committee (NAAC) in 2001 as a technical advisory group. In March 2003, the NAAC became the National Arbovirus and Malaria Advisory Committee (NAMAC) when malaria was included in its terms of reference. NAMAC monitors arbovirus and malaria surveillance, strategic arbovirus and malaria disease management, and vector control, and has a key role in making recommendations on the management of mosquito-borne diseases. NAMAC provides expert technical advice on arboviruses and malaria to the Australian Health Protection Committee through the Communicable Diseases Network Australia. It also assists in the detection, management and control of actual or potential outbreaks of arboviral and malarial disease. Members of the Committee have expertise in disease surveillance, virology, vector surveillance, vector control and quarantine, and represent agencies with a substantial interest in this area.

Methods

Human cases of arbovirus infection and malaria are monitored using the National Notifiable Diseases Surveillance System (NNDSS). All Australian states and territories require doctors and/or pathology laboratories to notify cases of infectious diseases that are important to public health including several arboviruses and malaria. The *National Health Security Act 2007* provides the legislative basis for communicable disease notifications in Australia and authorises the exchange

of health information between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be provided. State and territory health departments transfer these notifications regularly to the NNDSS, as described in the *National Health Security Agreement 2008*. The primary responsibility for public health action resulting from a notification resides with state and territory health departments. This report presents data extracted from NNDSS during February 2010 and analysed by date of diagnosis. This is a derived field and represents the earliest of the reported fields of notification date and notification received date. The dataset represents a 'snap shot', and numbers in this report may vary slightly from those reported from other NNDSS sources. Detailed notes on the interpretation of NNDSS are available in the 2008 NNDSS annual report.[1] Case definitions for the diseases included in this report are available from: http://www.health.gov.au/casedefinitions The report includes information on the following pathogens transmitted by mosquitoes:

- alphaviruses (Barmah Forest, Ross River, and chikungunya);
- flaviviruses (dengue, Japanese encephalitis, Kunjin, Murray Valley encephalitis, yellow fever and arbovirus not elsewhere classified); and
- malaria.

To compare notifications in 2008–09 to historical totals, crude numbers and rates of notification were compared either with the mean of the previous 5 years or with data from the previous year. The Australian Bureau of Statistics estimated resident populations for Australia and each state or territory at June 2008, was used to calculate rates of notification.

Additional information was available from a survey conducted with state and territory public health surveillance managers. The survey sought to confirm cases reported to NNDSS and determine the place of acquisition for locally-acquired cases of dengue virus infections. States and territories may conduct follow-up of arbovirus and malaria cases to determine the likely place of acquisition of infection. To date, the Northern Territory, Queensland, Victoria, and Tasmania are able to transfer place of acquisition details to NNDSS.

Overseas and locally-acquired dengue notifications from the Cairns and Townsville areas were mapped, based on the residential postcode of each case, to illustrate the spatial distribution of reported cases during the 2008–09 season. Each dot on the map is randomly assigned to an urban area within the postcode boundary (Map 1).

Results

During the 2008–09 season, there were 8,677 notifications of diseases transmitted by mosquitoes. This represented a 27% increase from the mean of 6,848 notifications for the previous 5 years and can be largely attributed to increased numbers of dengue notifications from the outbreak in Queensland. A summary of the number and rates of these mosquito-borne diseases is shown in Table 1. There were no reported cases of yellow fever during 2008–09.

Alphavirus

The main alphaviruses occurring in Australia, Ross River virus (RRV) and Barmah Forest virus (BFV), can cause illnesses characterised by fever, rash and polyarthritis. These viruses are transmitted by numerous species of mosquitoes that breed in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[2] No specific treatment or vaccine is available for these diseases. During 2008–09, there were 6,574 notifications of alphaviruses (BFV and RRV) of which RRV infections accounted for 74% (4,858).

Barmah Forest virus infections

There were 1,716 notifications of BFV infections during 2008–09, representing a rate of eight per 100,000 population. This was a 4% increase over the mean of the previous 5 years (Table 1). Queensland reported the largest number of notifications of BFV (940) while the highest rate was reported in the Northern Territory (56 per 100,000 population).

The highest age specific rate for males of 16 per 100,000 population was reported in the 50–54 year age group and for females 14 per 100,000 population in the 45–49 year age group. Approximately half of all notifications were male (53%). Cases were reported in all jurisdictions.

As in previous years, there was a marked seasonal trend with the highest number of notifications being diagnosed in the months of January (162) and February (214). The number of BFV notifications per month did not exceed the 5-year rolling mean during the 2008–09 season.

Table 1: Number of notified cases, rate and 5-year mean rate per 100,000 population of mosquito-borne diseases, Australia, 2003–04 to 2008–09, by disease and state or territory

Disease		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Arbovirus infection (NEC*)	Notified cases 2008–09	0	0	0	29	0	0	6	0	35
	Rate, 08–09	0.0	0.0	0.0	0.7	0.0	0.0	0.1	0.0	0.2
	Mean rate, 2003–04 to 07–08	0.0	0.0	0.0	0.7	0.0	0.0	0.1	0.0	0.2
Barmah Forest virus infection	Notified cases 2008–09	2	393	123	940	39	2	15	202	1,716
	Rate, 08–09	0.6	5.6	56.0	21.9	2.4	0.4	0.3	9.3	8.0
	Mean rate, 2003–04 to 07–08	1.3	7.5	32.3	20.1	4.0	0.0	0.5	5.7	7.7
Dengue virus infection– infection acquired from north Queensland	Notified cases 2008–09	0	5	0	1,001	0	0	2	1	1,009
	Rate, 08–09	0.0	0.1	0.0	23.3	0.0	0.0	0.0	0.0	4.7
	Mean rate, 2003–04 to 07–08	0.0	0.0	0.1	3.0	0.0	0.0	0.0	0.0	0.6
Dengue virus infection – infection acquired from overseas	Notified cases 2008–09	13	147	24	131	25	6	18	120	484
	Rate, 08–09	3.8	2.1	10.9	3.1	1.6	1.2	0.3	5.5	2.3
	Mean rate, 2003–04 to 07–08	1.3	0.9	9.2	1.3	0.8	0.2	0.2	1.6	0.9
Japanese encephalitis virus infection – infection acquired from overseas	Notified cases 2008–09	0	1†	0	0	0	0	0	0	1
	Rate, 08–09	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Mean rate, 2003–04 to 07–08	0.0	0.0	0.0	0.01	0.0	0.0	0.0	0.0	0.0
Kunjin virus Infection	Notified cases 2008–09	0	0	1	2	0	0	0	0	3
	Rate, 08–09	0.0	0.0	0.5	0.0	0.0	0.0	0.00	0.0	0.01
	Mean rate, 2003–04 to 07–08	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	Notified cases 2008–09	9	111	20	200	24	9	113	81	567
	Rate, 08–09	2.6	1.6	9.1	4.7	1.5	1.8	2.1	3.7	2.6
	Mean rate, 2003–04 to 07–08	3.9	1.9	19.5	5.9	1.9	4.1	1.9	3.9	3.1
Murray Valley encephalitis virus infection	Notified cases 2008–09	0	0	1	1‡	0	0	0	2	4
	Rate, 08–09	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.1	0.0
	Mean rate, 2003–04 to 07–08	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	Notified cases 2008–09	8	937	413	2,116	234	25	104	1,021	4,858
	Rate, 08–09	2.3	13.4	187.9	49.3	14.6	5.0	2.0	47.0	22.7
	Mean rate, 2003–04 to 07–08	2.9	12.5	113.7	51.4	10.9	5.1	2.7	39.0	20.9

Does not include 21 chikungunya virus infections reported to the National Notifiable Diseases Surveillance System during the 2008–09 season.

NEC Not elsewhere classified

* Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008.

† New South Wales resident acquired Japanese encephalitis virus infection while visiting Japan.

‡ Queensland resident acquired Murray Valley encephalitis infection while visiting the Northern Territory.

Authorities are considering the possibility of the first evidence of local transmission of BFV in Tasmania based on the travel histories of 2 cases reported during early 2009. All previous notified cases of BFV in Tasmania have reported a travel history to other parts of Australia affected by BFV. These cases justify the provision of advice to general practitioners in Tasmania to assist with the diagnosis of future cases. Further investigation will also be undertaken in an attempt to isolate the virus from known vector mosquito species and local wildlife in Tasmania (personal communication: Department of Health and Human Services, Tasmania).

Ross River virus infections

There were 4,858 notifications of RRV infection during 2008–09 representing a rate of 23 per 100,000 population (Table 1). This was a 9% increase over the mean of the previous five years. Queensland reported the largest number of notifications of RRV (2,116) while the highest rate was reported in the Northern Territory (188 per 100,000 population).

The highest age specific rate for males of 33 per 100,000 population was reported in the 45–64 year age groups and for females 40 per 100,000 population in the 40–45 year age group. Nearly half of all notifications were male (48%).

As in previous years, there was a marked seasonal trend with the highest number of notifications being diagnosed in March (680) and April (597). The number of notifications per month was either similar or less than the 5-year rolling mean during the 2008–09 season.

Both Western Australia and the Northern Territory reported a large increase in RRV notifications when compared with the mean of the previous 5-year period. In 2008–09, the number of notifications reported was the largest for a season since NNDSS began in 1991. This increase was probably due to variations in the amount and timing of rainfall in the various regions compared with previous years, but was possibly complicated to some extent by movement of people between the regions, and differences in pathology test requests or methods.[3]

Chikungunya virus infection

Chikungunya virus (CHIKV) is a member of the Alphavirus genus in the family Togaviridae and is closely related to RRV and BFV. Illness is characterised by an abrupt onset of fever, rash and severe joint pain. The acute disease lasts one to 10 days, but convalescence may include prolonged joint swelling and pain lasting months. It has clinical similarities to dengue, including occasional cases with haemorrhagic manifestations.[4] CHIKV is of concern given that humans are amplification hosts rather than incidental hosts, and other vertebrates are not required for high levels of transmission to occur. In Australia, the known competent mosquito vectors for CHIKV include *Aedes aegypti*, which occurs in northern Queensland and *Aedes albopictus* (Asian Tiger mosquito), which is found on the Cocos, Christmas and the Torres Strait islands.[5] Other Australian mosquitoes have been shown to be competent laboratory vectors of CHIKV and in particular *Aedes* spp., which have been implicated previously as endemic RRV and BFV vectors.[6]

CHIKV infection is a notifiable disease in all jurisdictions other than the Australian Capital Territory. There were 21 notifications of overseas-acquired CHIKV infection reported to NNDSS during 2008–09 compared with 3 cases notified during 2007–08. Ten of the cases were reported to have acquired their infection during travel to Malaysia.

Flaviviruses

This section provides information on several flaviviruses notified to NNDSS including dengue virus, Murray Valley encephalitis virus (MVEV) infection, Kunjin virus (KUNV) infection and Japanese encephalitis virus (JEV) infection. Other flaviviruses may be notified under the Arbovirus (NEC) category. Dengue is characterised by flu like symptoms (fever, headache, muscle or joint pain) and has 4 distinct serotypes. MVEV, KUNV and JEV can, in a small percentage of cases, result in illness involving the central nervous system including encephalitis of variable severity. *Ae. aegypti* is the major vector of dengue in Australia and *Culex annulirostris* is the major vector of MVEV, JEV and KUNV. No specific treatment is available for these diseases and care is largely supportive. A vaccine is not available for dengue, MVEV or KUNV infection but a vaccination to prevent JEV infection is available.[7] Dengue is the most commonly notified flavivirus infection in Australia and accounted for 99% (1,493) of the 1,501 flavivirus notifications reported during 2008–09 (Table 1). The remaining flavivirus notifications included 4 notifications of MVEV infection, 3 notifications of KUNV infection and a single notification of overseas-acquired JEV infection.

Dengue virus infection

There were 1,493 notifications of dengue infection notified during the season of 2008–09. Of

these, 1,009 notified cases were locally-acquired from north Queensland and 484 notified cases acquired their dengue infection while overseas (Table 2). The highest age specific rate for males of 12 cases per 100,000 population was reported in the 40–44 year age group and for females 11 cases per 100,000 population in the 25–29 year age group. Approximately half of all notifications were male (54%). A case of dengue notified from jurisdictions other than Queensland and who did not acquire their infection in Queensland were reported as overseas-acquired cases of infection.

Locally-acquired dengue virus infection

Local transmission of dengue is restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.[8] Dengue is not endemic in North Queensland, however local transmission can occur upon introduction of the virus to the key mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.[9] These cases of dengue acquired from overseas are of particular public health importance as they resulted in outbreaks in Cairns and Innisfail, Townsville, Yarrabah, Injinoo, Mareeba and Port Douglas. (Table 3). There were 1,009 notifications of locally-acquired dengue infection during 2008–09 representing a rate of 4.7 per 100,000 population (Table 1). The number of notifications reported was the largest for a season since NNDSS began in 1991. All cases of infection were acquired in North Queensland, including a single dengue associated death in March 2009.

Table 2: Place of acquisition of notified cases of dengue virus infection, Australia, 1 July 2004 to 30 June 2009, by state or territory

Place of acquisition	Season	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Locally-acquired	2004–05	0	0	0	72	0	0	0	2*	74
(North Queensland)	2005–06	0	0	0	42	0	0	1*	0	43
	2006–07	0	0	1*	46	0	0	0	0	47
	2007–08	0	2*	0	26	2*	0	0	0	30
	2008–09	0	5*	0	1,001	0	0	2*	1*	1,009
Sub-total		0	7	1	1,187	2	0	3	3	1,203
Overseas-acquired	2004–05	1	33	16	41	3	0	8	11	113
	2005–06	7	54	16	33	10	0	12	21	153
	2006–07	2	71	14	67	12	0	9	27	202
	2007–08	4	102	26	84	33	4	15	94	362
	2008–09	13	147	24	131	25	6	18	120	484
Sub-total		27	407	96	356	83	10	62	273	1,314
Total		27	414	97	1,543	85	10	65	276	2,517

* Cases acquired their infection while visiting North Queensland.

Table 3: Number of notified cases of dengue virus infection, Australia, 1 July 2008 to 30 June 2009, by location of outbreak

Outbreak location	Reported cases	Type/s	Past outbreak		Comments
			Year	Serotype[10]	
Cairns (Port Douglas, Yarrabah Injinoo, Mareeba)	915	3	2006	2	Index case imported from Indonesia
Townsville	16	3	2007	3	Linked to Cairns
Cairns	2	2	2006	2	Index case likely imported from Papua New Guinea
Townsville	57	1	2007	3	Index case not identified
Innisfail	35	4	Not reported		Index case imported from Vanuatu

Source: Queensland Health

Published case numbers differ from the National Notifiable Diseases Surveillance System data due to different notification criteria.

The first and largest of these outbreaks started in Cairns with subsequent spread to Townsville and other towns. The initial outbreak in Cairns was declared on 1 December 2008. An investigation identified the earliest case as a Queensland resident who had become unwell after returning from a trip to Kalimantan in Indonesia. Recognition of this outbreak was delayed as the case did not seek prompt medical attention despite being unwell for four to 5 days early in November. Local weather conditions were ideal for mosquito breeding and a relatively short virus incubation period led to a rapid expansion of cases.[10]

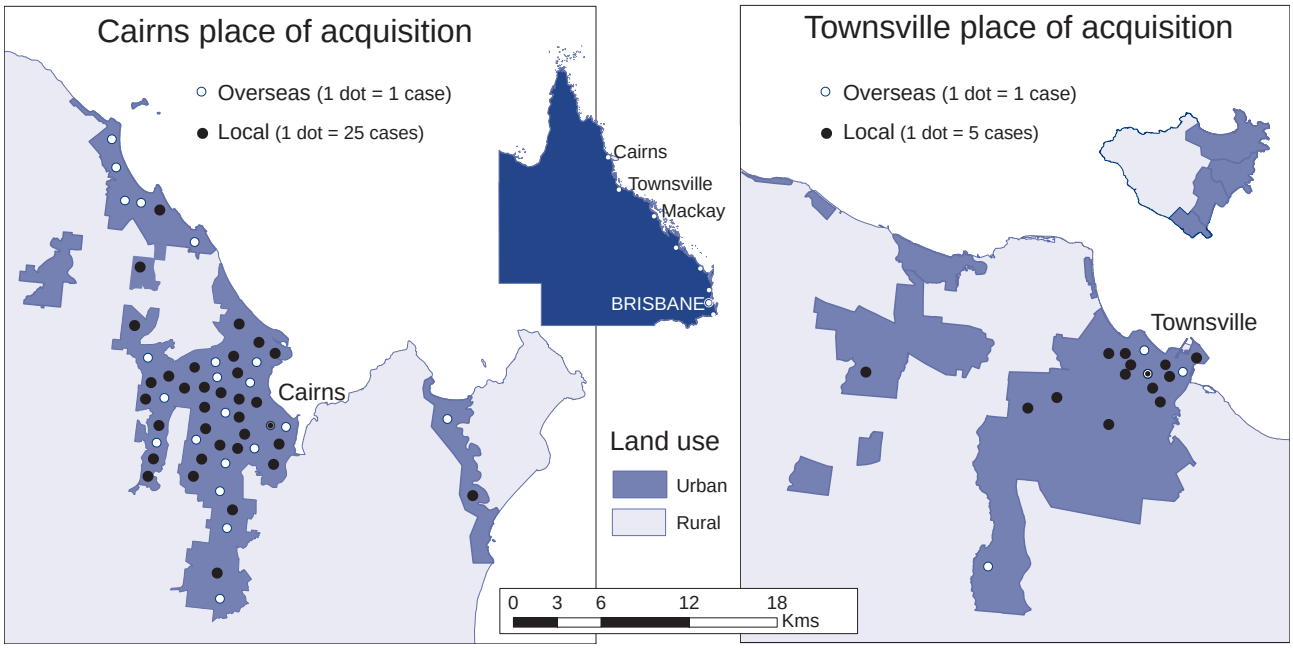
Map 1 shows the extent and general location of cases in the 2008–09 outbreaks in North Queensland, including the notification of 21 overseas-acquired cases of dengue returning to Cairns and 3 cases in Townsville during the season. Of these, just a few of the overseas-acquired cases were responsible for the outbreaks of locally-acquired dengue infection in North Queensland during the 2008–09 season. While authorities attempt to identify the index case (imported case that leads to a local outbreak), it is not always possible, particularly if the index case does not seek or delays seeking medical attention. These overseas-acquired cases present a challenge for local authorities, as any impediment in the identification of a case will delay other public health actions, including mosquito control activities.

As in previous years, there was a marked seasonal trend with locally-acquired cases predominantly being diagnosed between October and May (Figure 1). All 4 serotypes of dengue have circulated in North Queensland at some time in the last 7 years. However, this is the first time that all 4 serotypes had been in circulation at the same time.[10] Having more than 1 strain of the virus circulating in an area may increase the risk of a case of dengue haemorrhagic fever (DHF) or dengue shock syndrome (DSS). DHF/DSS may occur when a person, infected at some time in the past, becomes infected with a different dengue serotype. DHF/DSS occurs most frequently in infants and young children.

Overseas-acquired dengue virus infection

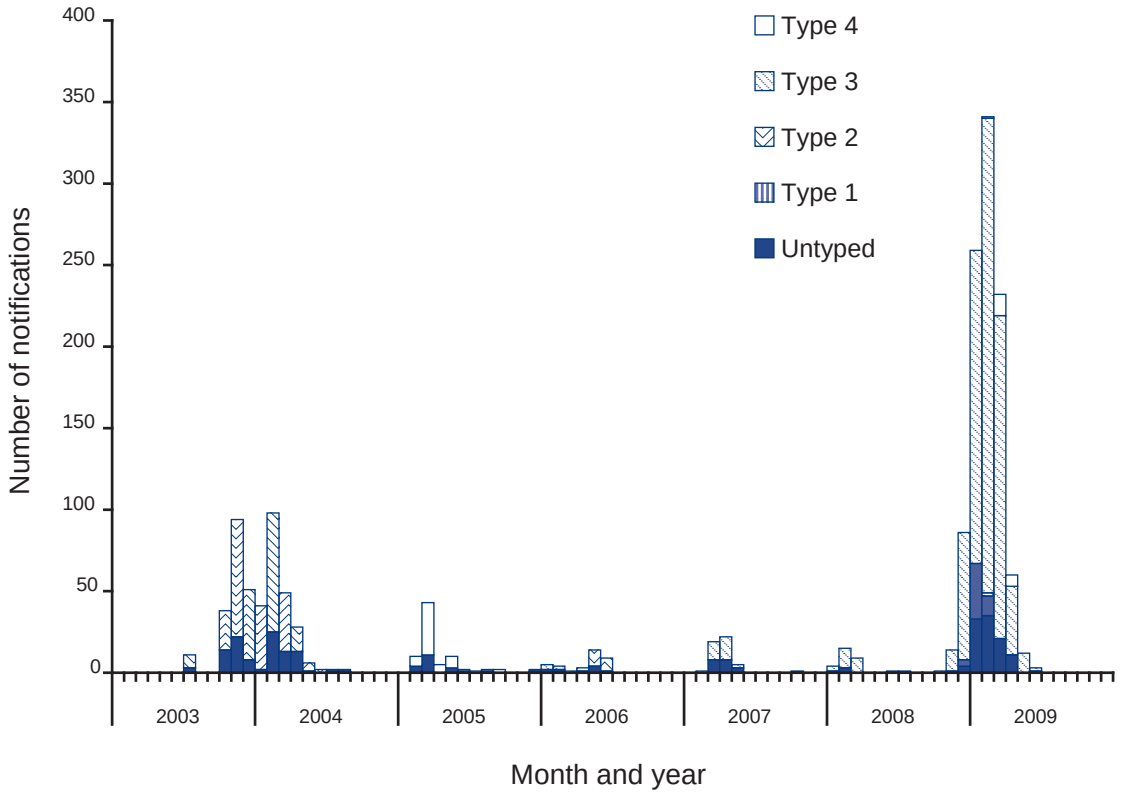
There were 484 notifications of dengue virus infection acquired overseas during the 2008–09 season (Table 1). This was an increase when compared with the mean rate of the previous 5 years of 193 overseas-acquired dengue cases and began at the beginning of 2007 (Figure 2). Case numbers per month were similar throughout the season other than for a peak in January (81). All jurisdictions reported increased numbers of notifications of overseas-acquired dengue virus infection.

Map 1: Geographic distribution of notified cases of overseas and locally-acquired dengue virus infection, Cairns and Townsville, Queensland, Australia, 2008–09



Each dot is randomly assigned within the urban area of the postcode boundary.

Figure 1: Number of notified locally-acquired cases of dengue virus infection, Australia, 1 July 2003 to 30 June 2009, by serotype



Country of acquisition was available for 132 (27%) cases of overseas-acquired dengue reported to NNDSS (Table 4). Indonesia (including Bali) was reported for 32 (7%) cases and involved 3 dengue serotypes. Twenty-four other destinations were identified by cases, which reflect the worldwide distribution of dengue virus infection. The infecting DENV serotype was determined for 105 (22%) of the 484 overseas-acquired dengue cases of which DENV serotype 4 (35) was the most frequently reported.

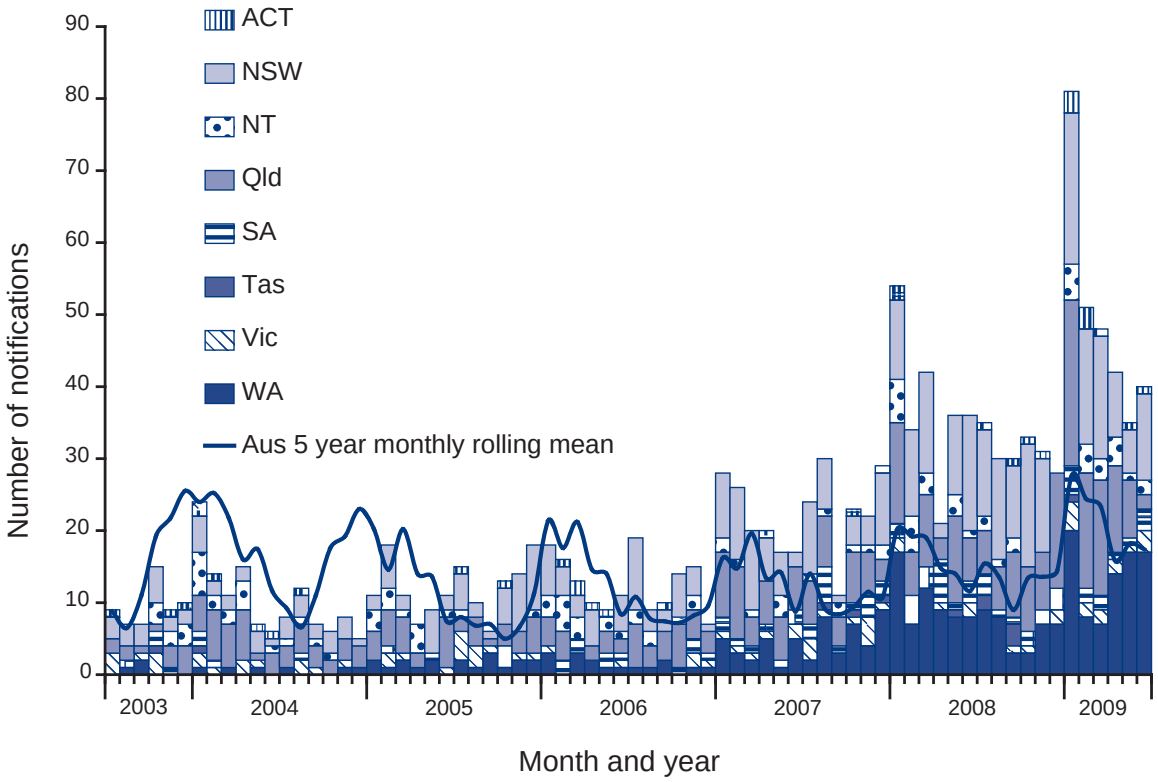
Japanese encephalitis virus infections

There was 1 case of JEV infection notified by New South Wales in Australia in 2008–09. The case was a male in his 20s who reported recent travel to Japan (personal communication: NSW Health). Prior to this notification, the last JEV infection notification was reported by Queensland in February 2004 when a 66-year-old male acquired JEV in Papua New Guinea.[11] There were no cases of locally-acquired JEV infection notified

Table 4: Serotype and country of acquisition of overseas-acquired dengue notifications, Australia, 1 July 2008 to 30 July 2009

Country of acquisition	Total	Dengue serotype				
		Untyped	Virus 1	Virus 2	Virus 3	Virus 4
Indonesia	32	20	0	5	5	2
Thailand	19	12	1	0	6	0
East Timor	12	8	2	0	2	0
Fiji	11	3	0	1	0	7
Tonga	11	7	0	0	1	3
Vanuatu	7	4	0	0	0	3
Samoa	7	1	1	0	0	5
Papua New Guinea	6	1	2	0	3	0
Other country	27	12	4	2	5	4
Country not listed	352	311	15	6	9	11
Total	484	379	25	14	31	35

Figure 2: Number of notified overseas-acquired cases of dengue virus infection, Australia, 1 July 2003 to 30 June 2009, by state or territory



to NNDSS in Australia during 2008–09. The last case of locally-acquired JEV infection was reported in 1998.[12]

Kunjin virus infection

There were 3 locally-acquired human cases of KUNV infection reported in Australia during 2008–09. Two cases were from Queensland and a single case was from the Northern Territory.

Murray Valley encephalitis virus infection

There were 4 notifications of locally-acquired MVEV in Australia resulting in 2 deaths during 2008–09. Two MVEV cases were notified from Western Australia, with 1 case in Broome in March 2009 and 1 case in Port Hedland in May 2009.

The 2 fatal cases of MVEV infection were reported from the Northern Territory. The first case was a long term resident from the Batchelor area in March 2009 and the other was a Queensland resident holidaying at Channel Point in May 2009. Health warnings were given both before and after the cases, with warnings based on vector numbers, rainfall, historical risk periods and/or detections of seroconversions in sentinel chicken flocks.

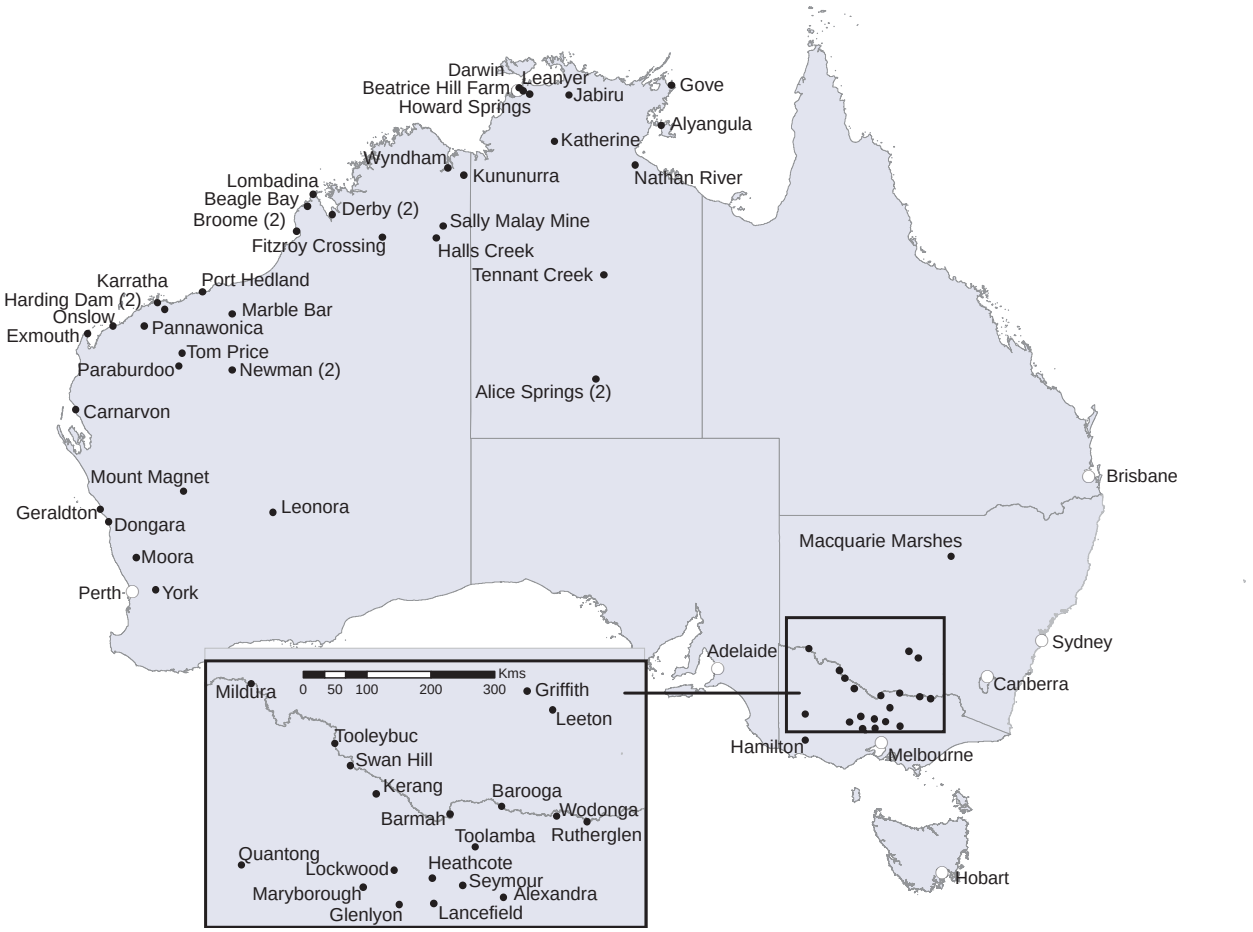
Sentinel chicken flavivirus surveillance programs

The sentinel chicken program is designed to detect flavivirus activity in Western Australia, New South Wales, Victoria and the Northern Territory. The program aims to provide early warning of the endemic arboviruses MVEV and KUNV, as well as exotic arboviruses such as JEV.[13] A public health response or warning can be implemented when chickens from a flock develop new antibodies to a flavivirus of interest. These warnings advise residents of the need to take added precautions to avoid mosquito bites and may be used to direct mosquito management programs. Chickens are replaced at least annually and more frequently if birds die or large proportions seroconvert. The flocks are well positioned to detect flavivirus activity and provide a timely and accurate indication of risk to people.[14] The location of sentinel chicken sites during 2008–09 is shown in Map 2.

Northern Territory

Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for flavivirus antibodies in a combined program between the Northern Territory Department of Health and Families, the Northern Territory Department of Primary Industry, Fisheries and Mines (DPIFM), and volunteers.

Map 2: Sentinel chicken testing sites, Australia, 2008–09



Sentinel chicken flocks are presently located at Leanyer, Howard Springs, Coastal Plains Research Station, Katherine, Nhulunbuy, Tennant Creek, Jabiru, Alice Springs (2), Nathan River, and Alyangula (Map 2). DPIFM officers or volunteers usually bleed flocks once a month and the samples are tested for antibodies to MVEV and KUNV.

In the 2008–09 season, MVEV activity was detected in the flocks at Howard Springs in May, Leanyer in May, Adelaide River in March and May, Nhulunbuy in January and June, Katherine in December, February, March and April, Tennant Creek in February and March, Jabiru in March and Nathan River in January, March and April. It was notable that during the 2008–09 MVEV season, unusually large numbers of chickens seroconverted to MVEV in a number of flocks including Coastal Plains, Katherine and Tennant Creek, indicating widespread and relatively high virus activity in the Top End and as far south as Tennant Creek. However, there was no seroconversion to either MVEV or KUNV in the Alice Springs flocks. This reflected relatively dry weather conditions and the present lack of the Ilparpa wetland during summer after drainage provisions were installed in 2001–02, whereby excess effluent is being pumped to a site for injection into an aquifer rather than routinely released into the wetland. The Jabiru, Nathan River and Katherine flock were not bled in May or June, and the Leanyer, Coastal Plains and Tennant Creek flocks were not bled in June, generally because appreciable numbers of chickens had already seroconverted. The Howard Springs chickens were not bled between August 2008 and March 2009 due to operational problems, and the Robinson River chickens were only bled once in July 2008. The Robinson River flock will no longer be part of the flavivirus surveillance program.

KUNV activity occurred in all Northern Territory regions except in the Alice Springs region, where chickens seroconverted to a flavivirus that could not be further identified. Seroconversions to KUNV occurred between January and May 2009. Seroconversions to both MVEV and KUNV were the highest this year since the current sentinel chicken program started in 1992–93. Most of the MVEV and KUNV seroconversions occurred in March, notably around the same time as the first human case of MVEV in the Northern Territory.

Northern Territory sentinel chicken surveillance indicated that during the 2008–09 season, MVEV was wide-spread in the Northern Territory as far south as Tennant Creek, with large numbers of chickens seroconverting to MVEV, and all flocks except Alyangula and the Alice Springs flocks showing seroconversions. The absence of MVEV seroconversions around Alyangula over the last 3 years indicate that the Alyangula locality is not conducive to MVEV transmission, probably as a result of relatively low numbers of the principle vector and the lack of a nearby wetland and associated birds. There have been no seroconversions to MVEV in the Alice Springs flocks since 2001–02, when the Ilparpa swamp was drained. Health warnings were issued throughout the main MVEV risk period between January and June 2009.

Western Australia

The Arbovirus Surveillance and Research Laboratory (ASRL) at The University of Western Australia, on behalf of the Western Australian Department of Health, undertakes the flavivirus sentinel chicken program in Western Australia. Many state and local government authorities and community volunteers also take part in the program. Thirty sentinel chicken flocks (of up to 12 chickens) are located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Central Coastal regions of Western Australia (Map 2). Blood samples are collected from the chickens by environmental health officers or trained volunteers at fortnightly intervals during the peak MVEV risk season (December to June). At other times of the year, monthly blood samples are collected, unless prolonged flavivirus activity warrants continued fortnightly sampling. Samples are transported to the ASRL where they are tested for antibodies to flaviviruses using an epitope blocking ELISA.[15]

The passage of Tropical Cyclone Billy in the northern Kimberley region in December caused heavy rain and flooding. Above average rainfall was recorded across most of the Kimberley, Pilbara, Interior and southern Goldfields. Successive tropical cyclones and low pressure systems caused further rainfall and flooding in northern Western Australia in January, February and March. The west Pilbara was particularly affected in February, and the eastern Pilbara received heavy rainfall in March. Conditions were generally dry in northern Western Australia between April and June 2009.

A total of 4,067 serum samples from the 30 sentinel chicken flocks were tested for antibodies to flaviviruses during 2008–09.[16] Seroconversions were detected in 247 (6.1%) of the samples. Two MVEV seroconversions detected at Onslow in the Pilbara region in July and August 2008 were associated with activity extending from the 2007–08 wet season. The first activity associated with the 2008–09 wet season occurred in February 2009, when MVEV was detected at Kununurra in the north-east Kimberley region, Halls Creek and Sally Malay mine in the south-east Kimberley region, Fitzroy Crossing in the West Kimberley region, and a couple of weeks later at Harding Dam in the Pilbara region. KUNV activity was also detected in February, when 2 seroconversions to KUNV were detected at Marble Bar, in the Pilbara region. Widespread MVEV activity continued, and was ultimately detected at all locations where sentinel chickens were in place in the Kimberley, Pilbara and Gascoyne regions. The level of MVEV activity was very high in 2008–09, with 108 MVEV seroconversions in the Kimberley, 99 in the Pilbara and four in the Gascoyne region. Overall, the level of MVEV activity was substantially higher than the previous season, and was almost as high as 2000, when there was widespread activity of MVEV and 11 clinical cases, including 9 cases of encephalitis.[17] Despite the high level of MVEV activity, no seroconversions to MVEV were detected south of the Gascoyne region. In contrast, the level of activity of KUNV was relatively low, and activity of KUNV was not detected south of Newman. A small proportion of unidentified flavivirus infections were detected at several locations in the Kimberley and Pilbara regions, possibly due to activity of other flaviviruses that have previously been isolated from mosquitoes collected in northern Western Australia.

New South Wales

The NSW Arbovirus Surveillance and Mosquito Monitoring program at the Institute of Clinical Pathology and Medical Research undertakes the New South Wales sentinel chicken program. The 2008–09 season began on 2 October 2008 and ended on 12 April 2009. A total of 1,509 samples were received from 6 sentinel chicken flocks in New South Wales over a 7-month period in 2008–09. The sentinel chicken flocks were located at Bourke, Deniliquin, Griffith, Leeton, Macquarie Marshes and Menindee (Map 2). There were no seroconversions to MVEV or KUNV.[18] A description of the bleeding method of the chickens and the testing regime is outlined in the 2003–2004 New South Wales Arbovirus Surveillance Program annual report.[19]

Victoria

The Victorian Department of Primary Industry on behalf of the Victorian Department of Health undertakes the Victorian sentinel chicken program.

The program received 6,160 samples from the 10 sentinel chicken flocks in Victoria during the season (Map 2). There were no seroconversions to MVEV or KUNV. Detection of MVEV activity in Victoria during March and April 2008 prompted the addition of 9 sites to test weekly from 1 January 2009 to 30 March 2009 (total tests 1,620). These sites were across an east/west direction and further south than the established 10 sites in the Murray Valley region. Similarly, no seroconversions were detected (personal communication: Victorian Government Department of Health).

Malaria

Malaria is a serious acute febrile illness that is normally transmitted from person to person through the bite of an infected mosquito. It is caused by a protozoan parasite called *Plasmodium* that includes 4 species – *vivax*, *falciparum*, *malariae* and *ovale*.[20] A 5th species, *Plasmodium knowlesi* has been recently identified as a cause of human malaria occurring predominantly throughout South East Asia. Infection with this primate malaria has the potential of being fatal if treatment is not given early in the course of an infection.[21]

There were 567 notifications of overseas-acquired malaria during the season 2008–09, representing a rate of 2.6 per 100,000 population (Table 1). This was a decrease when compared with the mean rate of the previous 5 years of 3.1 per 100,000 population. There were no reports of locally-acquired malaria. The last outbreak of locally-acquired malaria occurred in North Queensland during 2002.[22] Notification rates ranged from 1.5 per 100,000 population in South Australia to 9.1 per 100,000 population in the Northern Territory. All jurisdictions reported a decrease in notifications when compared with the previous 5 years, other than in Victoria (1.9 to 2.1 per 100,000 population). Seventy-one per cent of notifications were male, which was consistent with the past 5 years. The highest age specific rate for males of 8.2 per 100,000 and 4.1 per 100,000 population for females was reported in the 20–24 year age group. No deaths from malaria were reported during the 2008–09 season.

The infecting *Plasmodium* species was reported for 96% of malaria notifications during 2008–09 (Table 5). Of these 567 notifications, *P. falciparum* and *P. vivax* were the predominant species. There were no cases of *P. knowlesi* notified to NNDSS during 2008–09.

The country of acquisition of infection was available for 227 (40%) cases of malaria reported to NNDSS (Table 6). Papua New Guinea was reported as the country of acquisition by 113 (20%) cases and included both *P. falciparum* and *P. vivax* species. Twenty-four other destinations were identified by cases. They included India (34), Indonesia (9) and Mozambique (7).

Arbovirus infection (NEC)

The category Arbovirus infection (NEC) includes notifications of vectorborne infections not elsewhere classified. There were 35 notifications in this category during 2008–09, which was similar when compared with the previous 5 years. Queensland (29) and Victoria (6) accounted for all notified cases. Single notifications were identified as Kokobera and Stratford virus infection.

Other surveillance and research activities

National Arbovirus Monitoring Program

The National Arbovirus Monitoring Program (NAMP) monitors the distribution of economically important arboviruses of livestock and their vectors

Table 5: Overseas-acquired malaria cases, Australia 1 July 2008 to 30 June 2009, by species and state or territory

Plasmodium species	State or territory									Type (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
Plasmodium falciparum	2	41	10	80	11	6	32	49	231	41
Plasmodium vivax	6	63	8	94	11	2	73	21	278	49
Other *Plasmodium* species	1	7	1	6	0	1	2	4	22	4
Mixed *Plasmodium* species	0	0	1	0	2	0	5	5	13	2
Plasmodium species	0	0	0	20	0	0	1	2	23	4
Total	9	111	20	200	24	9	113	81	567	

New South Wales, Victoria, South Australia, Western Australia, Tasmania and the Northern Territory report mixed species infections per notified case. Queensland and the Australian Capital Territory report 1 notification for each species in a mixed infection.

Table 6: Overseas-acquired malaria cases, Australia, 1 July 2008 to 30 June 2009, by country of acquisition and species

Country of acquisition	Total	*Plasmodium* species				
		Not specified	*Falciparum*	*Vivax*	Other *Plasmodium* species	Mixed *Plasmodium* species
Papua New Guinea	113	6	31	73	2	1
India	34	1	0	33	0	0
Indonesia	9	0	3	4	0	2
Mozambique	7	0	7	0	0	0
Other country	64	0	46	15	1	2
Country not listed	340	16	144	153	19	8
Total	567	23	231	278	22	13

in Australia. Important arboviruses include bluetongue, Akabane and bovine ephemeral fever and are further described in the NAMP 2008–2009 annual report. NAMP is jointly funded by its primary beneficiaries, including the cattle, sheep and goat industries and the state, territory and Australian governments.[23]

Northern Australia Quarantine Strategy

The Australian Quarantine and Inspection Service Northern Australia Quarantine Strategy continues to undertake limited surveillance for transmission of JEV in the Torres Strait and mainland Australia. A sentinel pig herd at Injinoo airport near Bamaga in Cape York, Queensland has not shown any serological evidence of mainland transmission since early 2004.[24]

Torres Strait Mosquito Elimination Program

The mosquito *Ae. albopictus*, which is exotic to Australia, was found on the outer islands of Torres Strait in April 2005.[25] If this mosquito establishes in Australia it will increase the number and spread of mosquitoes capable of transmitting dengue and chikungunya as well as becoming a new serious pest mosquito. Since 2005, the Australian Government has provided funding to Queensland Health towards a mosquito elimination program in the Torres Strait. The initial aim of the program was to eliminate *Ae. albopictus* from the Torres Strait islands. The development and implementation of a program based on the 'cordon sanitaire' approach (a barrier designed to prevent a disease or other undesirable condition from spreading) around Thursday and Horn islands was initiated in May 2008 in an attempt to prevent the spread of *Ae. albopictus* further south, following unsuccessful attempts to eliminate *Ae. albopictus* from the outer islands of the Torres Strait.[26] Multiple incursions of *Ae. albopictus* into the Torres Strait had likely occurred and resulted from human activity or traffic moving these mosquitoes around the Torres Strait. In May 2009, the Australian Government agreed to provide further funding to Queensland Health over 4 years, to continue support towards the Torres Strait Health Protection Strategy mosquito program.[27] The focus of the program is surveillance and control of *Ae. albopictus* in the Torres Strait and prevention of the spread of *Ae. albopictus* from the Torres Strait to mainland Australia.

Enhancing emerging zoonotic disease surveillance data from animals

In light of worldwide animal and human health crises such as severe acute respiratory syndrome, the pandemic influenza (H1N1) 2009 and Nipah virus, the need for an interdisciplinary approach to emerging and re-emerging zoonoses is gaining significant international recognition. Samples currently collected in Australia from wildlife are only analysed for a limited number of diseases such as avian influenza or Newcastle disease.

The Australian Government Department of Agriculture, Fisheries and Forestry funded a pilot project in the 2008–2009 financial year to determine how surveillance of zoonotic infections in animals can add to the understanding of the epidemiology of emerging zoonoses in humans, using MVEV as a model. Project partners, which reflect the 'One Health' philosophy, include the Australian Government Department of Health and Ageing, the Australian Wildlife Health Network and Animal Health Australia.

Initial findings of this report (which is to be published in *Vector-Borne and Zoonotic Diseases*) support the use of chickens for surveillance, and also recommend the use of young cattle and horses for general MVEV surveillance. Eastern grey kangaroos also showed a high prevalence of antibody to MVEV, making them a potential source for

monitoring outbreaks and retrospectively determining the extent of an outbreak (personal communication: Australian Government Department of Agriculture, Fisheries and Forestry).

Discussion

This report summarises the surveillance of nationally notifiable mosquito-borne disease in Australia for 1 July 2008 to 30 June 2009. Of particular concern were the outbreaks of locally-acquired dengue infection in North Queensland and the occurrence of fatal human cases of MVEV infection in the Northern Territory.

Australia experienced several outbreaks of locally-acquired dengue virus infections involving all 4 serotypes in Queensland from 1 July 2008 to 30 June 2009. It is important to rapidly diagnose the disease in returning residents and tourists to prevent local spread in North Queensland. Queensland health authorities are experienced in responding to outbreaks of the disease and implemented the Dengue Fever Management Plan. A major focus of the response was raising public awareness of the need to take responsibility for reducing mosquito breeding opportunities around homes, and for those people living in areas where dengue fever was known to occur to seek medical advice if feeling unwell. Control measures also included spraying known mosquito breeding sites with insecticide. People were encouraged to avoid dengue infection by taking measures to prevent mosquito bites. This included using insect repellent, wearing long sleeve clothing and reducing mosquito breeding sites by ensuring that pools and other receptacles of water are not available in and around the home.

Outbreaks of dengue in North Queensland are not unprecedented; in 2003–04 there were over 800 cases of locally-acquired dengue reported in Queensland and in 1998 nearly 500 cases of dengue serotype 2 were recorded.[10] By comparison, the 2008–09 outbreaks of all 4 serotypes affected several locations with over 1,000 dengue cases in a short period and represented the largest reported annual number of cases in recent times.[10,28] Much of the increase in overseas-acquired dengue virus infections over the past few years can be attributed to an increase in disease activity in the Asia–Pacific region. The World Health Organization has warned of a spreading threat of dengue outbreaks in the Asia–Pacific region. This threat has been recognised in the publication of a regional strategic plan.[29]

Authorities are concerned that the more severe forms of the disease, DHF/DSS may eventuate as outbreaks of multiple serotypes of dengue continue to occur in North Queensland. DHF/DSS may occur when a person, infected at some time in the past, becomes infected with a different dengue serotype. As a result, NAMAC is considering the feasibility of the eradication of the *Ae. aegypti* mosquito from North Queensland.

The main way of preventing and controlling the further spread of dengue fever is to control the vector *Ae. aegypti* through environmental management (e.g. eliminating larval habitats) and/or insecticide application. Mosquito control strategies will continue to evolve but must now take account of the recent drought conditions that has led to water storage vessels increasingly being used across Australia. If vessels such as water tanks are not mosquito 'proof' then there is the potential for increased mosquito breeding and mosquito-borne diseases such as dengue.[30] Water tanks in Australia built to Australian standards and maintained to that standard will not allow mosquito breeding because of the mosquito proofing at access points. However, poorly maintained water tanks may quickly become a suitable site for mosquito breeding.

Malaria and dengue, although almost completely preventable, remain a significant risk to travellers overseas despite warnings and other travel advice. Travellers continue to acquire malaria and dengue infections. The main way to minimise the risk of infection is to avoid being bitten by mosquitoes through the application of personal prevention measures. Travellers are encouraged to consider the information available on the Smartraveller travel health web site and to seek a doctor's advice prior to travel.

MVEV activity in the sentinel chicken flocks in northern Western Australia and the Northern Territory both led to public health actions in the form of media releases to warn the public of potential infection and other prevention strategies. These warnings started prior to the human cases of MVEV infection during the season and demonstrate that sentinel chicken surveillance provided public health authorities forewarning of virus activity.

The limitations of surveillance data used in this report are referred to in detailed notes on the interpretation of NNDSS, which are available in the 2008 NNDSS annual report.[1] A specific limitation of the data used in this report relates to the virological testing, which is required to distinguish alphavirus disease from other causes of arthritis. The alphavirus infections notified to NNDSS each season are based on laboratory definitive evidence only and assumes a clinically compatible arthritic infection. A case can still be

notified when clinical illness may not be consistent with the diagnosis of alphavirus infection. Furthermore, false positive reactions are an issue in the serological diagnosis of some arboviral infections and cross-reacting IgM can occur, particularly with flavivirus infections. Following some infections, particularly alphaviruses and flaviviruses, IgM antibodies can persist for long periods and should be interpreted as presumptive evidence of recent infection.[31] The Case Definitions Working Group of the Communicable Diseases Network Australia is reviewing this issue. Human surveillance for alphavirus infection enables local authorities to implement public health action and manage local disease outbreaks, but does not necessarily provide a reliable indication of the true incidence of a disease.

Another limitation on the findings of this report relates to place or country of acquisition of infection. This information is currently not available for all notifications due to system limitations. The Northern Territory, Queensland, Victoria, and Tasmania are the jurisdictions able to provide place of acquisition details to NNDSS.

Surveillance and reporting systems for arbovirus disease and malaria encompassing humans and animals provides information to assist in the detection, management and control of real or potential outbreaks in Australia. The surveillance of these diseases contributes to the preparation for and prevention of outbreaks, implementation of response measures to control outbreaks, and enables NAMAC to provide advice on the strategic approaches for the management of arbovirus disease and malaria.

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References

1. NNDSS Writing Group. Australia's notifiable disease status, 2008: annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2010;34(3):157–224.

2. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.

3. Medical Entomology, Centre for Disease Control, Department of Health and Families, Northern Territory. *Medical Entomology Annual Report 2008/09*. Darwin: Department of Health and Families, Northern Territory; 2009.

4. Parida MM, Santhosh SR, Dash PK, Lakshmana Rao PV. Rapid and real-time assays for detection and quantification of chikungunya virus. *Future Virol* 2008;3(2):179–192.

5. Harrington S, Lindsay MD, Douglas A. *Christmas Island and Cocos (Keeling) Islands, Indian Ocean: Mosquito fauna and mosquito-borne disease risk assessment and management recommendations*. FINAL REPORT of investigations undertaken in 2007–08: Public Health Division, Western Australian Department of Health; 2009.

6. van den Hurk AF, Hall-Mendelin S, Pyke AT, Smith GA, Mackenzie JS. Vector competence of Australian mosquitoes for chikungunya virus. *Vector Borne Zoonotic Dis* 2010;10(5):489–495.

7. National Health and Medical Research Council. *The Australian Immunisation Handbook* 9th edn. Canberra, Australia: Australian Government Department of Health and Ageing; 2008.

8. Hanna JN, Ritchie SA, Richards AR, Humphreys JL, Montgomery BL, Ehlers GJ, et al. Dengue in north Queensland, 2005–2008. *Commun Dis Intell* 2009;33(2):198–203.

9. Queensland Health. *Dengue fever management plan for North Queensland 2005–2010*. Cairns, Queensland: Tropical Public Health Unit Network, Queensland Health; 2005.

10. Hanna JN, Ritchie SA. Outbreaks of dengue in north Queensland, 1990–2008. *Commun Dis Intell* 2009;33(1):32–33.

11. Hanson JP, Taylor CT, Richards AR, Smith IL, Boutlis CS. Japanese encephalitis acquired near Port Moresby: implications for residents and travellers to Papua New Guinea. *Med J Aust* 2004;181(5):282–283.

12. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF, et al. Japanese encephalitis in north Queensland, Australia, 1998. *Med J Aust* 1999;170(11):533–536.

13. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW, et al. Australian encephalitis: Sentinel Chicken Surveillance Programme. *Commun Dis Intell* 2001;25(3):157–160.

14. Broom AK. Sentinel Chicken Surveillance Program in Australia, July 2002 to June 2003. *Commun Dis Intell* 2003;27(3):367–369.

15. Hall RA, Broom AK, Harnett AC, Howard MJ, Mackenzie JS. Immunodominant epitopes on the NS1 protein of MVE and KUN viruses serve as targets for a blocking ELISA to detect virus-specific antibodies in sentinel animal serum. *J Virol Methods* 1995;51:201–210.

16. Johansen C, Avery V, Cashen C, Power C, McFall S, Masters M, et al. *The University of Western Australia Arbovirus Surveillance and Research Laboratory Annual Report: 2008–2009*. Discipline of Microbiology and Immunology, The University of Western Australia; 2009.

17. Cordova SP, Smith DW, Broom AK, Lindsay MD, Dowse GK, Beers MY. Murray Valley encephalitis in Western Australia in 2000, with evidence of southerly spread. *Commun Dis Intell* 2000;24(12):368–372.

18. Doggett S, Clancy J, Haniotis J, Webb C, Russell RC, Hueston L, et al. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program 2008–2009 Annual Report*. Medical Entomology Department, Institute of Clinical Pathology and Medical Research, University of Sydney and Westmead Hospital; 2009.

19. Doggett S, Clancy J, Haniotis J, Russell R, Hueston L, Marchetti M, et al. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program 2003–2004 Annual Report*. Medical Entomology Department, Institute of Clinical Pathology and Medical Research, University of Sydney and Westmead Hospital; 2004.

20. Heymann D, ed. *Control of Communicable Diseases Manual*. 18th edn. Washington: American Public Health Association; 2004.

21. Cox-Singh J, Davis TM, Lee KS, Shamsul SS, Matusop A, Ratnam S, et al. *Plasmodium knowlesi* malaria in humans is widely distributed and potentially life threatening. *Clin Infect Dis* 2008;46(2):165–171.

22. Hanna JN, Ritchie SA, Eisen DP, Cooper RD, Brookes DL, Montgomery BL. An outbreak of *Plasmodium vivax* malaria in Far North Queensland, 2002. *Med J Aust* 2004;180(1):24–28.

23. Animal Health Australia. *National Arbovirus Monitoring Program Annual Report 2008–2009*. Canberra: Animal Health Australia; 2010.

24. Animal Health Australia. *Animal Health in Australia 2008*. Canberra, Animal Health Australia; 2009.

25. Ritchie SA, Moore P, Carruthers M, Williams C, Montgomery B, Foley P, et al. Discovery of a widespread infestation of *Aedes albopictus* in the Torres Strait, Australia. *J Amer Mosquito Control Assoc* 2006;22(3):358–365.

26. Queensland Health. *Aedes albopictus* elimination program in the Torres Strait: Report for the period 1 July 2007–30 June 2008. Queensland, Australia: Queensland Health; 2009.

27. Australian Government Department of Health and Ageing. Portfolio budget statements 2009–10: Budget related paper No. 1.10; 2009.

28. Hanna JN, Ritchie SA, Hills SL, Pyke AT, Montgomery BL, Richards AR, et al. Dengue in north Queensland, 2002. *Commun Dis Intell* 2003;27(3):384–389.

29. Regional Office for South-East Asia and the Western Pacific World Health Organization. *The Dengue Strategic Plan for the Asia Pacific Region, 2008–2015*. Geneva: World Health Organization; 2008.

30. Beebe NW, Cooper RD, Mottram P, Sweeney AW. Australia's dengue risk driven by human adaptation to climate change. *PLoS Negl Trop Dis* 2009;3(5):e429.

31. Public Health Laboratory Network. *Alphavirus and flavivirus laboratory case definitions. 2001.* Accessed on 11 June 2009. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-phlncd-flavivirus.htm

Immunisation coverage annual report, 2008

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Abstract

This, the 2nd annual immunisation coverage report, documents trends during 2008 for a range of standard measures derived from Australian Childhood Immunisation Register data, including overall coverage at standard age milestones and for individual vaccines included on the National Immunisation Program (NIP). Coverage by indigenous status and mapping by smaller geographic areas as well as trends in timeliness are also summarised according to standard templates. With respect to overall coverage, Immunise Australia Program targets have been reached for children at 12 and 24 months of age but not for children at 5 years of age. Coverage at 24 months of age exceeds that at 12 months of age, but as receipt of varicella vaccine at 18 months is excluded from calculations of 'fully immunised' this probably represents delayed immunisation, with some contribution from immunisation incentives. Similarly, the decrease in coverage estimates for immunisations due at 4 years of age from March 2008, is primarily due to changing the assessment age from 6 years to 5 years of age from December 2007. A number of individual vaccines on the NIP are not currently assessed for 'fully immunised' status or for eligibility for incentive payments. These include pneumococcal conjugate and meningococcal C conjugate vaccines for which coverage is comparable to vaccines which are assessed for 'fully immunised' status, and rotavirus and varicella vaccines for which coverage is lower. Coverage is also suboptimal for vaccines recommended for Indigenous children only (i.e. hepatitis A and pneumococcal polysaccharide vaccine) as previously reported for other vaccines for both children and adults. Delayed receipt of vaccines is an important issue for vaccines recommended for Indigenous children and has not improved among non-Indigenous children despite improvements in coverage at the 24-month milestone. Although Indigenous children in Australia have coverage levels that are similar to non-Indigenous children at 24 months of age, the disparity in delayed vaccination between Indigenous and non-Indigenous children, which is up to 18% for the 3rd dose of DTP, remains a challenge. *Commun Dis Intell* 2010;34(3):241–258.

Keywords: immunisation coverage, immunisation delay, small area coverage reporting

Introduction

This is the 2nd annual Australian Childhood Immunisation Register (ACIR) coverage report. This series of annual reports was established to consolidate the various forms of regular coverage reports and ad-hoc publications produced by the National Centre for Immunisation Research and Surveillance using ACIR data, highlighting important trends and significant issues over the preceding 12 months.[1–13] It follows the format of the 1st report, providing a detailed summary for 2008 that includes vaccination coverage at standard milestone ages, coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children, and data for small geographic areas on vaccination coverage and prevalence of conscientious objectors. Readers are referred to the 1st report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.[14]

This report uses the long-standing international practice of reporting coverage at key milestone ages, to measure coverage against national targets and to track trends over time. The first 2 milestones are unchanged since the previous report (12 months for vaccines due at 6 months and 24 months for vaccines due at 12 months). However, from the beginning of 2008, assessment of the oldest milestone (for vaccines due at 4 years) was changed from 6 years to 5 years.[15] No new vaccines were introduced to the National Immunisation Program (NIP) during 2008, however, this report does include the 1st full year of coverage data for rotavirus vaccine, which was introduced in 2007.

Incentives for vaccination and reporting to the Australian Childhood Immunisation Register

There were important recent changes to the payment of incentives to providers and carers, which had potential impacts on reported coverage. The Australian Government, through the Department of Health and Ageing, advises the ACIR on whether calculation of coverage of the new vaccines/antigens should be included in the completed schedule assessment for eligibility for payments to parents or immunisation providers. In 2008, the ACIR made information payments (up

to $6) to all immunisation providers and general practitioners (GPs), under the General Practice Immunisation Incentive (GPII) Scheme. In the 2008–09 Budget, the Australian Government announced that one of the components of the GPII Scheme, the GPII Service Incentive Payment (SIP), would stop from 1 October 2008. Service Incentive Payments (SIP) ($18.50) were made for reporting a vaccination that completed a schedule point on the NIP.[16] However, the GPII Outcomes Payments, which paid practices that achieve 90% or greater proportions of full immunisation, was maintained. The vaccines/antigens included in assessment for the Outcomes Payment in 2008 were the same as in recent years, i.e. diphtheria, *Haemophilus influenzae* type b (Hib), hepatitis B, measles, mumps, pertussis, polio, rubella and tetanus. Vaccines included in the NIP in 2008 but not part of the completed schedule assessment for provider payments were: meningococcal C vaccine (Men C); 7-valent pneumococcal conjugate vaccine (7vPCV); and rotavirus vaccine. Varicella vaccine was also not included for coverage assessment but eligible providers received an information and SIP payment (up to October 2008) for reporting completion of the current 18-month schedule point. While the ACIR records sub-population vaccines such as hepatitis A and pneumococcal polysaccharide vaccines (23vPPV) and non-National Immunisation Program vaccines, such as bacille Calmette-Guérin, they do not attract a GPII payment. Table 1 shows the Australian National Immunisation Program Schedule in 2008.

In 2004–05, the means test to qualify for the Maternity Immunisation Allowance (MIA) was removed. This payment, of $233 per child in 2008, is likely substantial enough to provide motivation both to complete immunisation and for parents to prompt their provider to notify any outstanding reports to the ACIR before the child reaches 24 months of age. In the 2008–09 budget, in addition to the changes mentioned above, it was announced that the MIA payment would be paid in 2 equal amounts of $167, with eligibility for the 2nd payment assessed at 4–5 years of age. However, this did not come into effect during the period of this report.

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January, 1996, by incorporating demographic data from Medicare on all enrolled children under the age of 7 years.[2] Participation in the ACIR is opt-out so it constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age.[2] Children not enrolled in Medicare can also be added to the ACIR via a supplementary number. Since 2001, immunisations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible immunisation either through the Internet using the Medicare Australia web site or by submitting paper encounter forms, which are scanned at a central location. The existence of medical contraindications and conscientious objection to immunisation are also recorded on the ACIR. All vaccination records for a child remain on the register indefinitely, but no new immunisation encounter records are added after the 7th birthday.

Immunisations recorded on the Register must be rendered in accordance with the guidelines issued by the National Health and Medical Research

Table 1: Australian National Immunisation Program Schedule for children in 2008

Age	Vaccine									
Birth	Hep B									
2 months	Hep B	DTPa	Hib	Polio				7vPCV		Rotavirus
4 months	Hep B	DTPa	Hib	Polio				7vPCV		Rotavirus
6 months	Hep B	DTPa	Hib*	Polio				7vPCV		Rotavirus†
12 months			Hib		MMR		Hep A‡		Men C	
18 months						VZV	Hep A§‡	23vPPV‡		
24 months							Hep A§	23vPPV§		
4 years		DTPa		Polio	MMR					

* The 3rd dose of Hib vaccine at 6 months is dependent on the vaccine brand used in each state or territory.

† The 3rd dose of rotavirus vaccine at 6 months is dependent on the vaccine brand used in each state or territory.

‡ Aboriginal and Torres Strait Islander children in Western Australia and the Northern Territory.

§ Aboriginal and Torres Strait Islander children in Queensland and South Australia.

Council as stated in *The Australian Immunisation Handbook*.[17] Notifications falling outside these guidelines or duplicate notifications prompt an enquiry with the provider, and if their validity cannot be established they are rejected.

Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state or territory)[18] since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 months), and 5 years of age (for vaccines due at 4 years). A minimum 3-month lag period is allowed for late notification of immunisations to the Register, but only immunisations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.[18] If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.[4,5]

Three-month birth cohorts are used for time trend analyses, and 12-month cohorts used for other analyses in this report. These cohorts are children born between 1 January and 31 December 2007 for the 12-month milestone age; children born between 1 January and 31 December 2006 for the 24-month milestone age; and children born between 1 January and 31 December 2003 for the 5-year (60-month) milestone age.

The proportion of children designated as 'fully immunised' is calculated using the number of Medicare-registered children completely immunised with the vaccines of interest by the designated age as the numerator, and the total number of Medicare-registered children in the age cohort as the denominator. 'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of a 3rd dose of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine; a 3rd dose of polio vaccine; 2 or 3 doses of PRP-OMP containing Hib vaccine or 3 doses of any other Hib vaccine; and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of any other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of a 3rd dose of a DTP-containing vaccine; a 3rd dose of polio vaccine; 3 or 4 doses of PRP-OMP containing Hib vaccine or 4 doses of any other Hib vaccine; 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of any other hepatitis B vaccines; and the 1st dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 5 years of age is defined as a child having a record on the ACIR of a 4th or 5th dose of a DTP-containing vaccine; a 4th dose of polio vaccine; and a 2nd dose of an MMR-containing vaccine.

Immunisation coverage estimates were also calculated for individual NIP vaccines, including the 6 NIP vaccines not routinely reported in *Communicable Diseases Intelligence (CDI)*. They were: a 3rd dose of 7vPCV and 2nd or 3rd dose of rotavirus vaccine by 12 months of age; the 1st dose of varicella vaccine and the 1st dose of meningococcal C vaccine by 24 months of age; a 2nd dose of hepatitis A vaccine in Indigenous children by 30 months of age; and the 1st dose of 23-valent pneumococcal polysaccharide vaccine in Indigenous children by 36 months of age.

Timeliness

Age-appropriate immunisation was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTP (due at 60 days of age) when he or she was more than 90 days of age, was classified as not age-appropriately immunised (i.e. late for the dose). For descriptive purposes, we categorised the outcome measure for each dose as either vaccines received 'too early' (greater than 30 days prior to when it was due), vaccine received 'acceptably early' (within 30 days prior to when it was due), 'no delay' (age-appropriately immunised), 'delay of between 1 to 6 months', 'delay greater than 6 months', or vaccine dose 'not recorded'. However, we have only reported on the 2 'delay' categories within this report. All children included in the analysis were at least 36 months of age when the data were extracted and, therefore, old enough to potentially experience delays in immunisation greater than 6 months for immunisation due by 24 months of age or earlier. The interval between doses was not evaluated. Timeliness of different vaccines and doses was also compared by plotting the cumulative percentage receiving each vaccine dose by age, with the proportion ever immunised set as 100%.

Remoteness status

The area of residence of children was defined as accessible or remote using the Accessibility/Remoteness Index of Australia (ARIA), which was developed by the Department of Health and Aged Care, and proposed as the national standard measure of remoteness for inclusion in the Australian Bureau of Statistics (ABS) 2001 census.[19] We define the 2 ARIA categories with most restricted

access to services as 'remote' (approximately 2.6% of the Australian population) and all other areas as 'accessible'.

Indigenous status

Indigenous status on the ACIR is recorded as 'Indigenous', 'non-Indigenous' or 'unknown', as reported by the child's carer to Medicare, or by the immunisation provider to the ACIR. For this report we considered 2 categories of children: 'Indigenous' and 'non-Indigenous', children with unknown indigenous status were presumed to be 'non-Indigenous'. Coverage estimate time trends are presented from 2004 only, due to poor rates of reporting indigenous status prior to then.[20]

Small area coverage

Coverage was calculated for ABS-defined Statistical Subdivisions (SSD).[21] We chose ABS-defined SSD as areas to be mapped because each is small enough to show differences within jurisdictions but not too small to render maps unreadable. Maps were created using version 10 of the MapInfo mapping software[22] and the ABS Census Boundary Information. As postcode is the only geographical indicator on the ACIR, the ABS Postal Area to Statistical Local Area Concordance 2006 was used to match ACIR postcodes to SSDs, in order to create a SSD field for each child in the relevant study cohorts.[23]

Conscientious objection/no vaccine recorded

A child must be registered with Medicare before its parent(s) can lodge a conscientious objection to immunisation. Parents can also object to immunisation but refuse to lodge any official objection to the ACIR. We used the percentage of children with no vaccines recorded on the ACIR as a proxy measure of the number of these children. Proportions of conscientious objectors and children with no vaccines recorded by region were calculated from the cohort of children registered with Medicare, and born between 1 January 2001 and 31 December 2007. At the time of data extraction on 31 March 2009, they were between 12 and 72 months of age. We chose this cohort when calculating proportions so that children under the age of 12 months were not included, to allow sufficient time for registration of objection.

Results

Coverage estimates

Overall

The 2008 coverage estimates, calculated for full-year birth cohorts, for the 3 milestone ages of 12 months, 24 months and 5 years are provided in Tables 2, 3 and 4. Nationally, 'fully immunised' coverage and coverage for all individual vaccines for the 12-month and 24-month age groups are greater than the Immunise Australia Program's target of 90%. Recorded coverage for the 5-year age group is well below the target, sitting at just above 80% for all vaccines and even lower in particular jurisdictions. Figure 1 shows time trends in 'fully immunised' childhood vaccination coverage in Australia, assessed at 12 months, 24 months, and at 60 months of age, for 3-month cohorts born from 1 January 1996 to 31 December 2007. The proportion 'fully immunised at 1 year of age increased steadily from 75% for the 1st cohort to 91.7% by the 46th cohort, assessed on 31 December 2008. At the 24 month milestone, coverage estimates also increased steadily from 64% for the 1st cohort to 92.5% by December 2008. Coverage estimates at

Table 2: Percentage of children in 2008 immunised at 12 months of age, by vaccine and state or territory*

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	4,697	96,867	3,802	61,274	19,370	6,432	70,616	30,005	293,063
Diphtheria, tetanus, pertussis (%)	94.2	91.9	90.9	91.5	92.1	91.9	92.8	90.3	91.9
Poliomyelitis (%)	94.2	91.9	91.1	91.9	91.7	92.5	92.8	89.7	91.9
Haemophilus influenzae type b (%)	96.1	94.8	94.1	93.9	94.5	94.6	95.0	93.9	94.5
Hepatitis B (%)	95.9	94.8	94.6	93.8	94.3	94.6	94.8	93.8	94.5
Fully immunised (%)	93.9	91.6	90.4	90.9	91.4	91.7	91.9	89.8	91.4

* For the birth cohort born in 2007.

Table 3: Percentage of children in 2008 immunised at 24 months of age, by vaccine and state or territory*

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,665	93,818	3,630	58,675	18,654	6,338	68,992	29,293	284,065
Diphtheria, tetanus, pertussis (%)	96.5	94.8	95.9	94.6	94.7	95.4	95.7	93.6	94.9
Poliomyelitis (%)	96.5	94.7	95.9	94.5	94.7	95.4	95.7	93.5	94.9
Haemophilus influenzae type b (%)	96.4	95.2	94.5	93.6	93.6	95.4	94.6	93.4	94.4
Hepatitis B (%)	96.9	95.6	97.1	95.3	95.4	96.3	96.3	94.4	95.6
Measles, mumps, rubella (%)	95.5	93.7	95.5	93.6	93.9	94.8	94.8	92.7	94.0
Fully immunised (%)	94.5	92.5	93.8	92.2	92.6	93.8	93.6	90.8	92.6

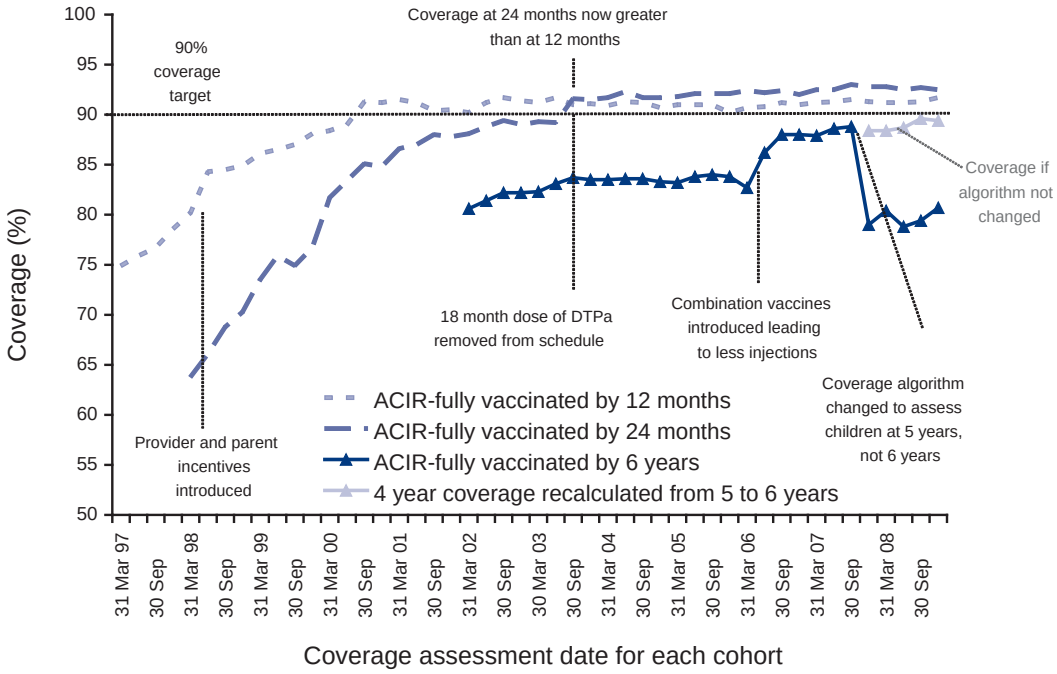
* For the birth cohort born in 2006.

Table 4: Percentage of children in 2008 immunised at 5 years of age, by vaccine and state or territory*

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,276	88,761	3,511	55,480	18,108	5,815	64,769	36,996	267,519
Diphtheria, tetanus, pertussis (%)	86.9	78.1	82.6	82.2	75.3	82.1	84.5	79.2	80.7
Poliomyelitis (%)	86.6	77.9	82.6	82.0	75.2	82.1	84.4	79.0	80.5
Measles, mumps, rubella (%)	86.3	79.4	82.4	81.8	75.1	81.7	84.1	78.9	80.3
Fully immunised (%)	85.9	77.2	81.9	81.3	74.7	81.2	83.8	78.1	79.8

* For the birth cohort born in 2003.

Figure 1: Trends in 'fully immunised' vaccination coverage, Australia, 1997 to 2008, by age cohort



6 years of age, for vaccines due at 4 years, were first reported in *CDI* in 2002, and increased steadily from 80.6% in early 2002 to 87.3% in late 2007, including a noticeable increase in June 2006, corresponding with the introduction of combination vaccines. However, from the beginning of 2008, the assessment age was changed from 6 years to 5 years and this resulted in a dramatic decrease in coverage for this age group, to 80.7% by December 2008. Figure 1 shows that coverage calculated at 6 years was unchanged.

Coverage estimates for the 24-month age group increased substantially and suddenly in September 2003 to 91.6% following the removal from the immunisation schedule of the 4th dose of DTPa (due at 18 months of age) from this quarter onwards. Coverage estimates for the 12-month age group have, however, remained steady over the past 5 years, fluctuating around the 91% level.

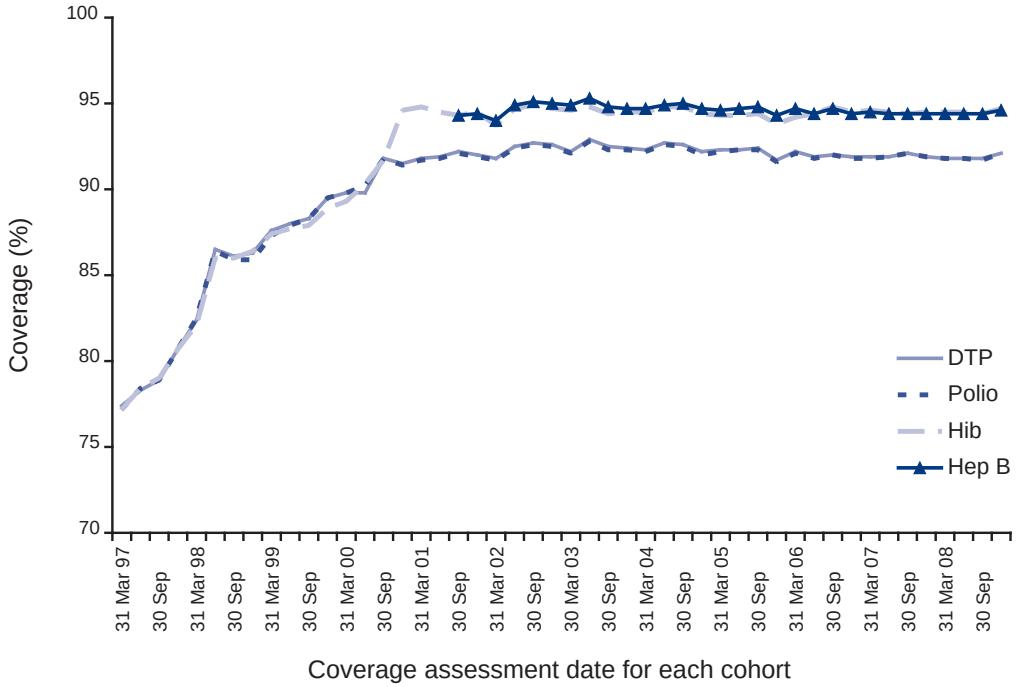
There is a clear trend of increasing vaccination coverage over time for children of all age groups assessed, with the 2 youngest age cohorts having the highest coverage. Coverage at 24 months of age exceeded that at 12 months of age for the first time at the end of 2003 and has remained higher since that time.

Individual vaccines

The trends in childhood vaccination coverage in Australia for individual vaccines at 12 months of age (DTP, polio, Hib and hepatitis B) are shown in Figure 2, for 3-month cohorts born from 1 January 1996 to 31 December 2007. Coverage estimates for all vaccines remained relatively stable throughout the latter part of 2001 to 2008. Coverage for the Hib and hepatitis B vaccines are greater than DTP and polio coverage. This is likely to be largely due to the change in the immunisation schedule in mid-2000, altering the algorithm used to calculate coverage at 12 months of age such that a record of 2 doses of Hib and hepatitis B on the ACIR renders a child 'fully immunised' for these vaccines.

The trends in childhood vaccination coverage in Australia for individual vaccines at 24 months of age (DTP, polio, Hib, hepatitis B and MMR) are shown in Figure 3, for 3-month cohorts born from 1 January 1996 to 31 December 2006. The significant increase in coverage for DTP during 2003 has been previously mentioned. For most of the study period, hepatitis B coverage was higher than for all other vaccines, just below 96%, due to the coverage algorithm changes described above. Coverage was lowest for MMR and Hib, which are the only

Figure 2: Trends in vaccination coverage estimates for individual vaccines at 12 months of age (DTP, polio, hepatitis B and Hib)*

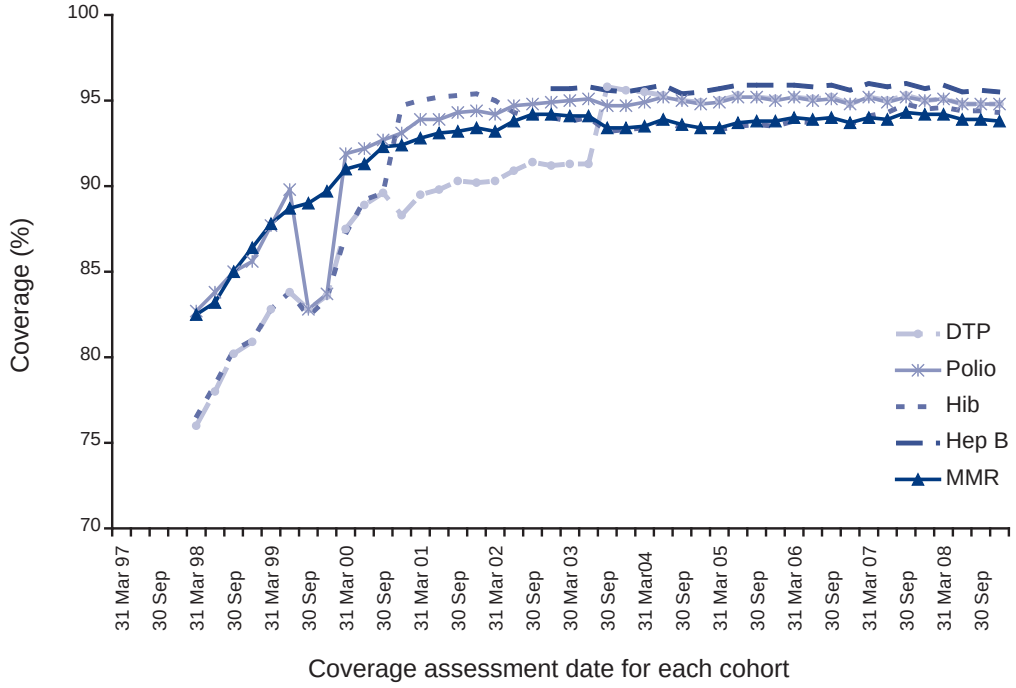


Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2007. Coverage assessment date was 12 months after the last birth date of each cohort.

* 3rd dose of DTP and polio, 2nd or 3rd dose of Hib and Hep B.

Figure 3: Trends in vaccination coverage estimates for individual vaccines at 24 months of age (DTP, polio, hepatitis B, Hib and MMR)*



Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2006. Coverage assessment date was 24 months after the last birth date of each cohort.

* 3rd or 4th dose of DTP, 3rd dose of polio, 3rd or 4th dose of Hib, 2nd or 3rd dose of Hep B, and 1 dose of MMR.

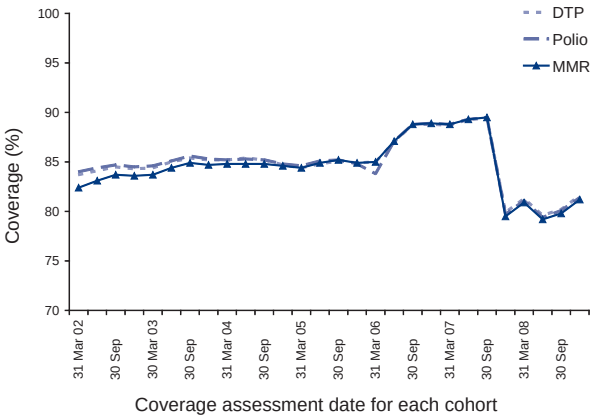
vaccines that have a 12 month dose included in the algorithm for calculation of coverage. However, coverage for all vaccines currently assessed at this age has been stable over recent years.

The trends in childhood vaccination coverage in Australia for individual vaccines (DTP, polio and MMR) at 6 years of age (5 years of age from December 2007) are shown in Figure 4 for 3-month cohorts born from 1 January 1996 to 31 December 2003. Coverage for all 3 vaccines was almost identical and remained steady across the whole period, at approximately 85%, until mid-2006 when a sharp increase of almost 5% was recorded. This increase may have been related to either or both of the campaigns to promote parental awareness of the 4-year milestone and school entry provisions in many jurisdictions becoming simpler to administer due to uniform ACIR certificates. The large decrease in coverage from December 2007, due to the change in assessment age discussed previously, is also evident for all vaccines due by 5 years of age.

Coverage estimates for Indigenous children

Vaccination coverage estimates in 2007 for the 3 milestone ages for individual vaccines by

Figure 4: Trends in vaccination coverage estimates for individual vaccines (DTP, polio, and MMR)* at 6 years of age (5 years from December 2007)



Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2003. Coverage assessment date was 72 months after the last birth date of each cohort up to December 2007 and then 60 months after the last birth date of each cohort.

* 4th dose of DTP and polio, 2nd dose of MMR.

Indigenous status are shown in Table 5. These show that coverage is lower for Indigenous children than non-Indigenous at the 12-month and 5-year age milestones, with the difference being greatest at 12 months of age. The difference in coverage at 12 months of age has been relatively consistent for the past 6 years. However, the coverage differential between Indigenous and non-Indigenous children for individual vaccines varies, with coverage at 24 months of age for most vaccines being almost identical for both groups and greater among Indigenous children for hepatitis B vaccine.

The trends in 'fully immunised' childhood vaccination coverage in Australia at 12 months, 24 months, and 6 years of age (5 years of age from December 2007) for Indigenous children since 2004 are shown in Figure 5, for 3-month cohorts assessed from 1 March 2004 to 31 December 2008. Coverage for all vaccines due by 24 months of age has consistently remained higher than at 12 months and 6 years of age. Since the beginning of 2006, coverage for Indigenous children at 6 years of age eclipsed coverage at 12 months of age until it plummeted below 80% in December 2007 due to the change in assessment age.

Table 6 shows 'fully immunised' vaccination coverage estimates in 2008 for Indigenous children at the 3 milestone ages by state or territory. At age 12 months, the proportion of Indigenous children fully vaccinated was 84.6%, compared with 91.4% for all Australian children (i.e. includes both Indigenous and non-Indigenous children, Table 2) and was lower among Indigenous children in all jurisdictions. The extent of the difference varied among jurisdictions, reaching more than 13 percentage points in some. However, by age 24 months, coverage disparities between Indigenous and all Australian children had almost disappeared nationally and in most jurisdictions, with the proportion fully vaccinated at 91.1% for Indigenous and 92.6% for all Australian children (Tables 3 and 6).

Table 5: Vaccination coverage estimates, 2008, by age, vaccine and indigenous status

Vaccine	Milestone age	Indigenous	Non-Indigenous
DTP	12 months*	85.1	92.3
	24 months†	94.9	94.9
	5 years‡	77.9	80.8
Polio	12 months*	85.1	92.2
	24 months†	94.3	94.4
	5 years‡	77.9	80.7
Hib	12 months*	92.7	94.7
	24 months†	93.0	94.5
	5 years‡	n/a§	n/a§
Hep B	12 months*	93.0	94.5
	24 months†	96.9	95.6
	5 years‡	n/a§	n/a§
MMR	12 months*	n/a§	n/a§
	24 months†	93.6	94.0
	5 years‡	77.9	80.3

* Birth cohort born 1 January 2007 to 31 December 2007.
† Birth cohort born 1 January 2006 to 31 December 2006.
‡ Birth cohort born 1 January 2003 to 31 December 2003.
§ Not included in coverage estimates for that group.

Figure 5: Trends in 'fully immunised' vaccination coverage for Indigenous children in Australia, 2004 to 2008, by age cohorts

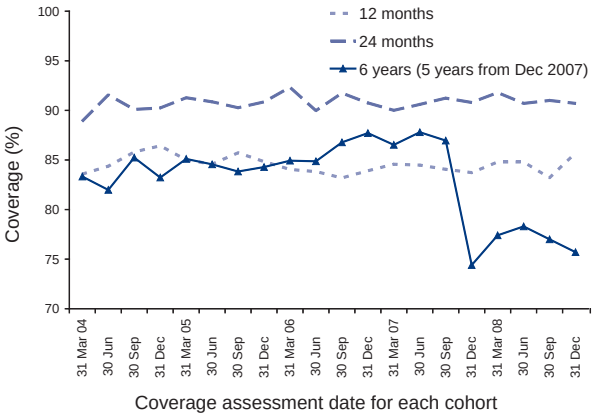


Table 6: Percentage of Indigenous children fully immunised at 12 months, 24 months and 5 years of age, 2008, by state or territory

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
12 months – fully immunised (%)	89.8	85.5	87.2	86.2	78.6	91.0	86.0	77.4	84.6
24 months – fully immunised (%)	87.5	91.2	94.4	92.5	89.8	93.1	90.0	85.8	91.1
5 years – fully immunised (%)	81.4	72.4	88.5	78.8	65.3	77.9	80.4	73.6	77.1

At 5 years of age, the proportion recorded as being 'fully vaccinated' was lower than that at earlier age milestones. There was little difference between Indigenous and all Australian children at the national level (77.1% and 79.8%, respectively) while, for individual jurisdictions, coverage in Indigenous children ranged from 9% lower (in South Australia) to 7% higher (in the Northern Territory) than in all Australian children (Tables 3 and 6).

Coverage for National Immunisation Program vaccines not routinely reported elsewhere

7vPCV and rotavirus

The 7vPCV vaccine was first added to the NIP in January 2005. Since coverage was first calculated for this vaccine in early 2006, it has remained at high levels, with a slight increase from 89% to 91%. Coverage is similar in all jurisdictions at greater than or approaching 90% (Table 7).

Rotavirus vaccine was added to the NIP in July 2007 so coverage for 2 or 3 doses (depending on vaccine) at 12 months of age could be calculated only from the December 2008 quarter. Rotavirus coverage was lower nationally, and had greater variation between jurisdictions compared with other vaccines given at 2, 4 and 6 months, which is expected from the vaccine most recently introduced onto the NIP. Reported coverage for 2 or 3 doses (depending on vaccine) of rotavirus at 12 months of age varied from 78.7% in Western Australia to 84.9% and 88.0% in New South Wales and the Australian Capital Territory, respectively (Table 7).

Meningococcal C and varicella

Meningococcal C vaccine was added to the NIP in January 2003. Since coverage was first calculated for this vaccine in early 2006, it has remained at high levels, with an increase over 2 years from 88% to around 93% (Figure 6), and there was little variation by jurisdiction, with all jurisdictions at greater than 92% (Table 7).

Varicella vaccine was added to the NIP in November 2005. Figure 6 shows coverage for this vaccine has consistently been 10–15 percentage points lower than that for meningococcal C vaccine, with coverage just above 80% for the latest assessment. This is probably partly due to the shorter time varicella has been on the NIP and the recommendation to give the vaccine at 18 months of age, which was historically associated with lower coverage and is not as well established as a milestone, especially following removal of the 18-month pertussis booster in 2003. However, varicella vaccine coverage varies by jurisdiction from 77.8% in Western Australia to greater than 83% in Queensland, the Northern Territory and the Australian Capital Territory (Table 7). Data

Table 7: Vaccination coverage for 7vPCV, rotavirus, meningococcal C, varicella, hepatitis A (Indigenous only) and 23vPPV (Indigenous only) for the last 3-month cohort assessable in 2008, by state or territory

State or territory	Vaccine type					
	7vPCV*	Rotavirus†	Men C‡	Varicella§	Hep A‖	23vPPV¶
ACT	94.1	88.0	95.1	85.4	na	na
NSW	91.7	84.9	93.1	78.5	na	na
NT	89.5	81.1	94.5	83.7	82.1 (90.3)	78.9
Qld	90.5	80.7	93.3	83.8	43.6 (60.8)	52.7
SA	91.3	82.1	92.9	78.1	25.4 (45.3)	36.0
Tas	91.7	82.6	94.4	82.0	na	na
Vic	91.9	81.3	94.5	81.7	na	na
WA	88.4	78.7	92.0	77.8	55.2 (73.0)	64.0
Australia	91.1	82.3	93.4	80.5	52.3 (68.1)**	59.1**

Na Not applicable.

* 3 doses at 12 months of age.

† 2 or 3 doses at 12 months of age.

‡ 1 dose at 24 months of age.

§ 1 dose at 24 months of age.

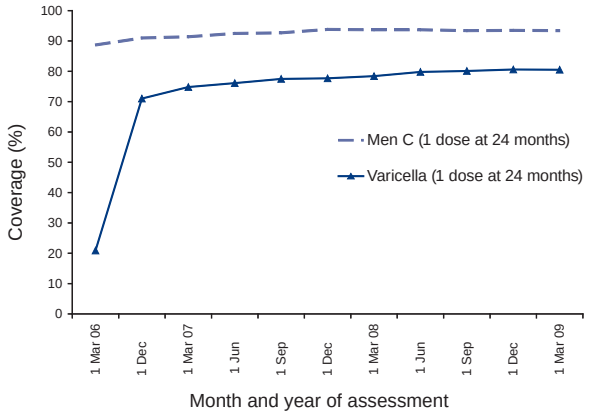
‖ Indigenous only: 2 doses at 30 months of age (1 dose at 24 months of age).

¶ Indigenous only: 1 dose at 36 months of age.

** Northern Territory, Queensland, South Australia and Western Australia only.

are also available from the ACIR on the number of reports from GPs stating that children born since May 2004, have natural immunity to varicella and do not require varicella vaccination. Reports of natural immunity to varicella during 2008 were around 1,000 reports per quarter (not shown), corresponding to approximately 1.7% of the cohort. However, it is likely that these are underestimates due to possible under-reporting.

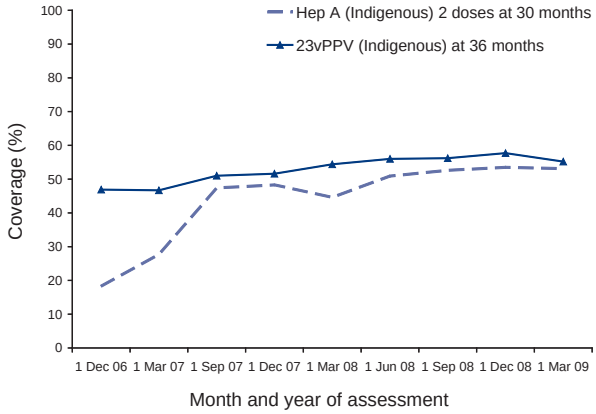
Figure 6: Trends in coverage for meningococcal C (Men C) and varicella vaccines



Hepatitis A and 23vPPV

Hepatitis A vaccine was available in Australia prior to the development of the ACIR in 1996 and has been included on the NIP for Indigenous children in the Northern Territory, South Australia, Western Australia, and in Queensland since November 2005. Since December 2006, coverage of 2 doses of hepatitis A vaccine by 30 months of age for Indigenous children has increased from below 20% to just over 50% (Figure 7). An additional 16% had received 1 dose by 24 months (Table 7). The 23vPPV has been available in Australia since 1983 and recommended for Indigenous children in the same 4 jurisdictions as a booster at 18–24 months of age since 2001; coverage has gradually increased from 47% in December 2006 to a high of 59% in December 2008 (Figure 7). There is a large variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 25.4% in South Australia to a high of 82.1% in the Northern Territory (Table 7). Similarly, there is variation in 23vPPV coverage by jurisdiction from a low of 36% in South Australia to a high of 78.9% in the Northern Territory (Table 7).

Figure 7: Trends in coverage for hepatitis A and pneumococcal polysaccharide (23vPPV) vaccines for Indigenous children



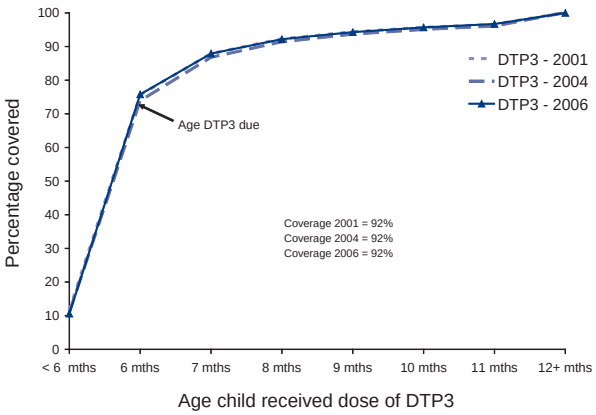
Timeliness of immunisation

Timeliness has been examined for vaccines requiring both multiple doses (DTP, 7vPCV and MMR) and a single dose (Men C) at 12 and 24 months of age.

Since 2001, the proportion with timely receipt of the 3rd dose of DTP vaccine has increased very slightly from 88% to 89% (Figure 8). Across the 6-year period, 2001–2006, timely receipt of 1 dose of MMR vaccine initially decreased by 3 percentage points but then rose 1.5 percentage points, although estimated coverage by 24 months of age remained stable at almost 94% (Figure 9).

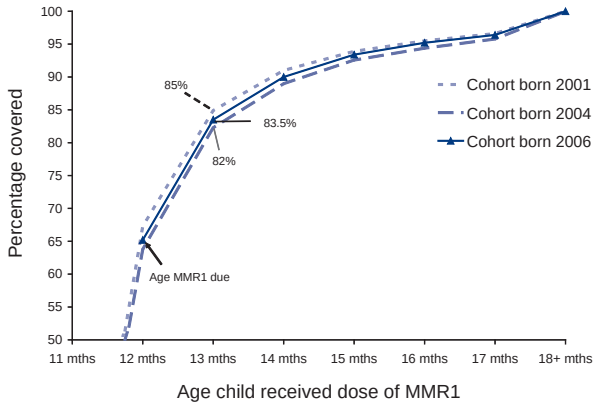
A comparison of vaccination delay for the 3rd dose of DTP, due at 6 months of age, and the 1st doses of MMR and meningococcal C, due at 12 months of age, for the 2004 cohort is shown in Figure 10. As demonstrated in previous studies, the proportion with vaccination delay increased with vaccine

Figure 8: Trends in timeliness of the 3rd dose of DTP vaccine (DTP3) – cohorts born in 2001, 2004 and 2007*



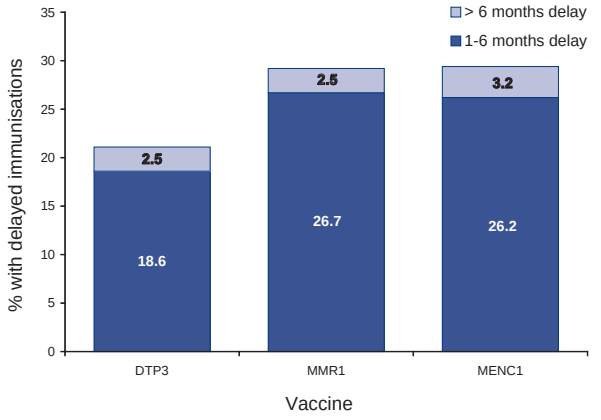
* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.

Figure 9: Trends in timeliness of the 1st dose of MMR vaccine (MMR1) – cohorts born in 2001, 2004 and 2006*



* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.

Figure 10: Vaccination delay for the 3rd dose of DTP vaccine (DTP3), and the 1st doses of MMR (MMR1) and Men C (MENC1) vaccines for Australia – cohort born in 2006



doses given at an older age. The greatest proportion with any delay was seen with meningococcal C vaccine with just under 30% of doses given late and over 3.2% given more than 6 months late.

Timeliness of the 3rd dose of DTP and the 1st dose of MMR vaccine by indigenous status and remoteness is shown in Table 8. Vaccination was delayed by more than 1 month for 40%–45% of Indigenous children and 20%–30% of non-Indigenous children. The proportion with long delays (i.e. greater than 6 months) was 3–4 times higher in Indigenous children than in non-Indigenous children, with no real differences between accessible and remote areas or vaccines. Delays of 1–6 months were also more frequent for Indigenous children, although less marked. The proportion with short delays was greater among Indigenous children residing in remote areas than in accessible areas for the 3rd dose of DTP vaccine (35% versus 31%), but not for the 1st dose of MMR.

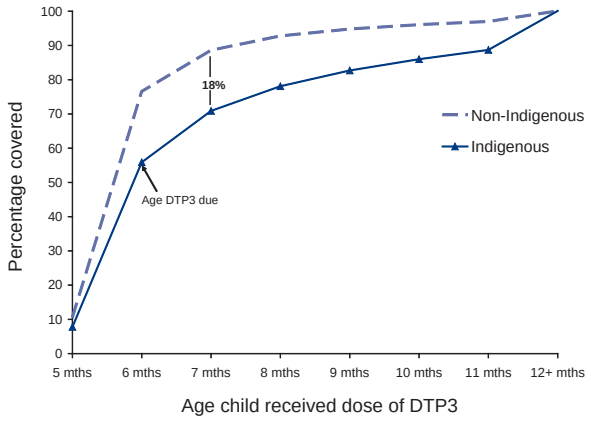
Figures 11 and 12 provide a comparison of timeliness of immunisation between Indigenous and non-Indigenous children in Australia for the 3rd dose of DTP vaccine, and the 1st dose of MMR vaccine, respectively. For the 3rd dose of DTP, there was significantly greater delay for Indigenous children than non-Indigenous children, with an 18% differential at 7 months of age. The same pattern was found for timeliness of the 1st dose of MMR, but with a smaller differential of 11%. Although Indigenous children had similar coverage levels to non-Indigenous children by 24 months of age, they were more likely to have delayed vaccination.

Vaccination delay for Indigenous children by jurisdiction was measured for 7vPCV, with greater delays in Western Australia and South Australia (Figure 13). The degree of long delay in vaccination for the 3rd dose of 7vPCV vaccine in South Australian Indigenous children was twice that which occurred in Queensland Indigenous

Table 8: Vaccination delay for the cohort of children born in 2006, Australia, by indigenous and remoteness status

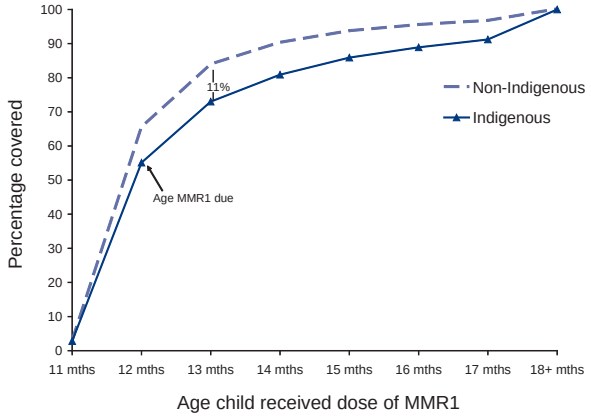
Vaccine dose	Indigenous status	Remoteness	1–6 months delay %	> 6 months delay %
DTP3	Indigenous	Accessible	31	9
		Remote	35	9
	Non-Indigenous	Accessible	18	2
		Remote	19	2
MMR1	Indigenous	Accessible	34	7
		Remote	33	6
	Non-Indigenous	Accessible	26	2
		Remote	28	2

Figure 11: Timeliness of the 3rd dose of DTP vaccine (DTP3) by Indigenous status – cohort born in 2006*



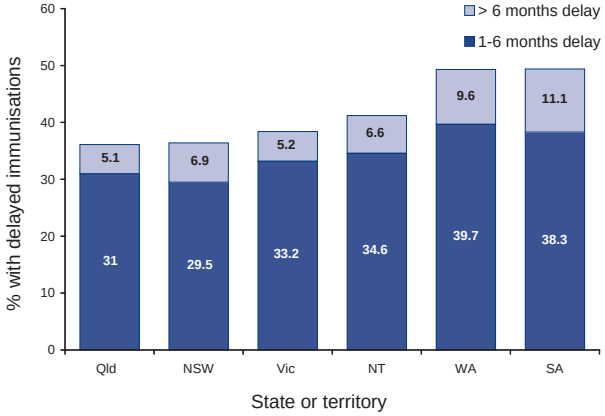
* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.

Figure 12: Timeliness of the 1st dose of MMR vaccine (MMR1) by Indigenous status – cohort born in 2006*



* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.

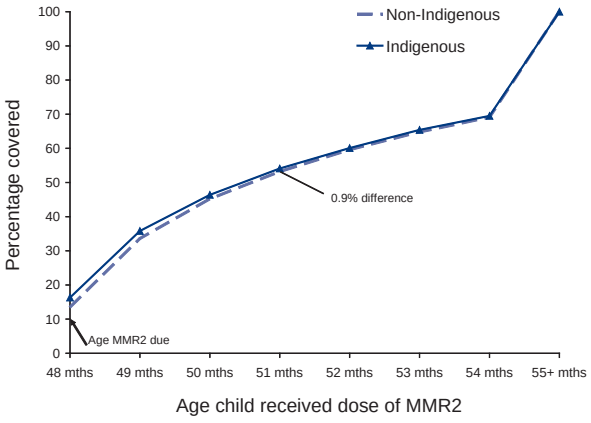
Figure 13: Vaccination delay for Indigenous children for the 3rd dose of 7vPCV in selected jurisdictions – cohort born in 2006



children. There were no important differences in vaccination delay for non-Indigenous children by jurisdiction (not shown).

In contrast to earlier ages, analysis of timeliness of immunisation for a vaccine due at 4 years of age, the 2nd dose of MMR, showed similar delay in receiving this vaccine for non-Indigenous children and Indigenous children, with only a 0.9% differential at 4 years and 3 months of age (Figure 14).

Figure 14: Timeliness of the 2nd dose of MMR vaccine (MMR2) by Indigenous status – cohort born in 2002*



* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.

Small area coverage

'Fully immunised' coverage for Australia by SSD for the 12-month, 24-month and 5-year milestone age groups, respectively, is shown in Figures 15–17. All 3 maps demonstrate that immunisation coverage in Australia in 2007 varies substantially within jurisdictions, with some having recorded coverage below the level required to prevent outbreaks of some highly contagious diseases such as measles. In particular, there are very few small areas in Australia with 'fully immunised' coverage for vaccines due at 4 years of age at levels required to prevent disease.

The proportions of children recorded as conscientious objectors and with no vaccines recorded are presented by SSD in Figures 18 and 19, respectively. No vaccines recorded may represent either non-immunisation (parents refusing any vaccines) or, and probably much less commonly, non-reporting by a provider. The percentage of children with no vaccines recorded nationally (3.5%) is greater than those recorded as conscientious objectors (1.4%).

Figure 15: 'Fully immunised' coverage at 12 months of age, by Statistical Subdivision, Australia, 2008

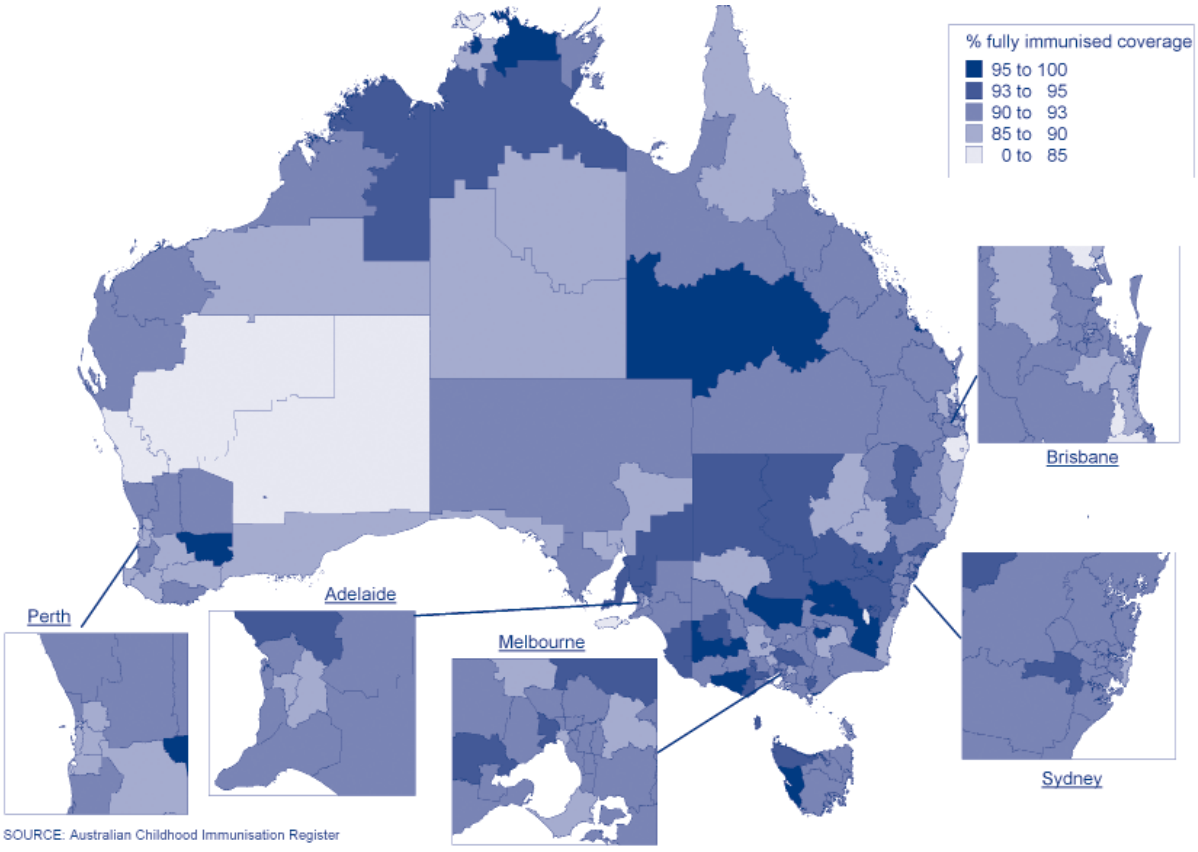


Figure 16: 'Fully immunised' coverage at 24 months of age, by Statistical Subdivision, Australia, 2008

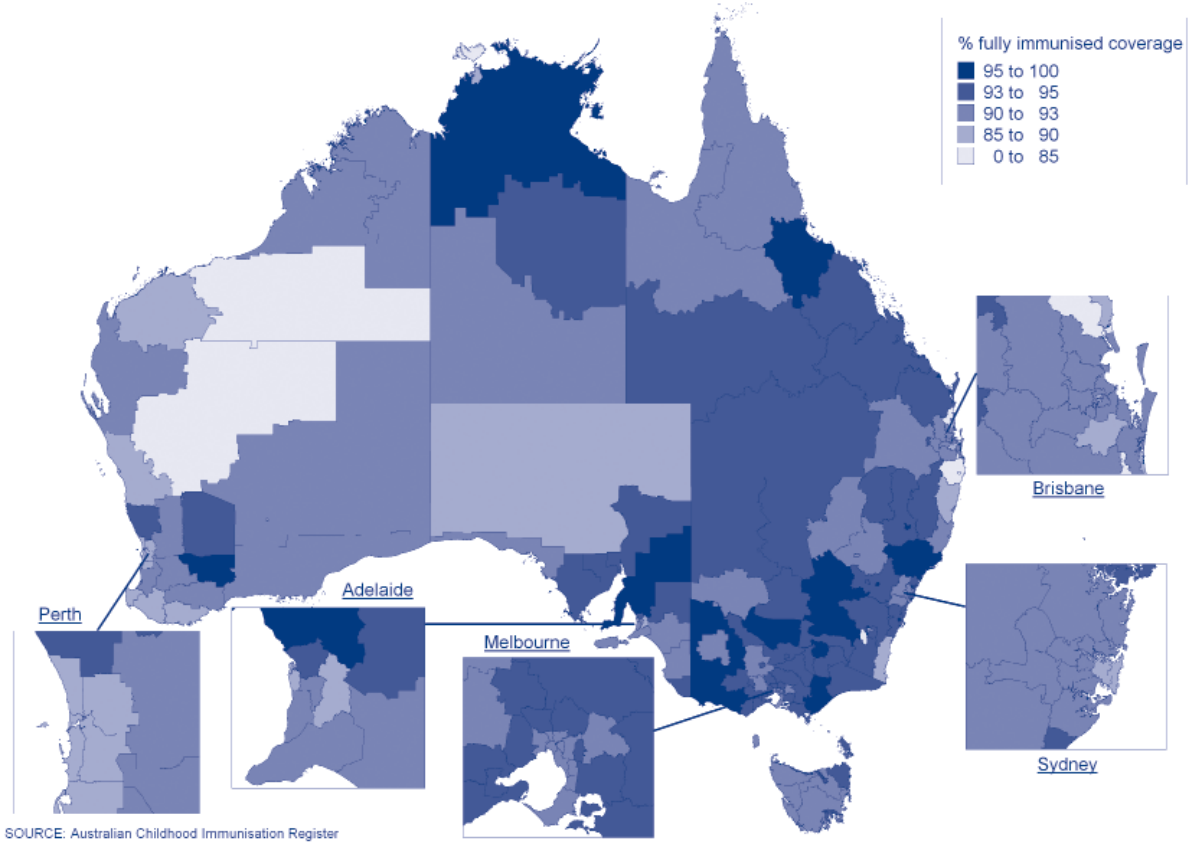
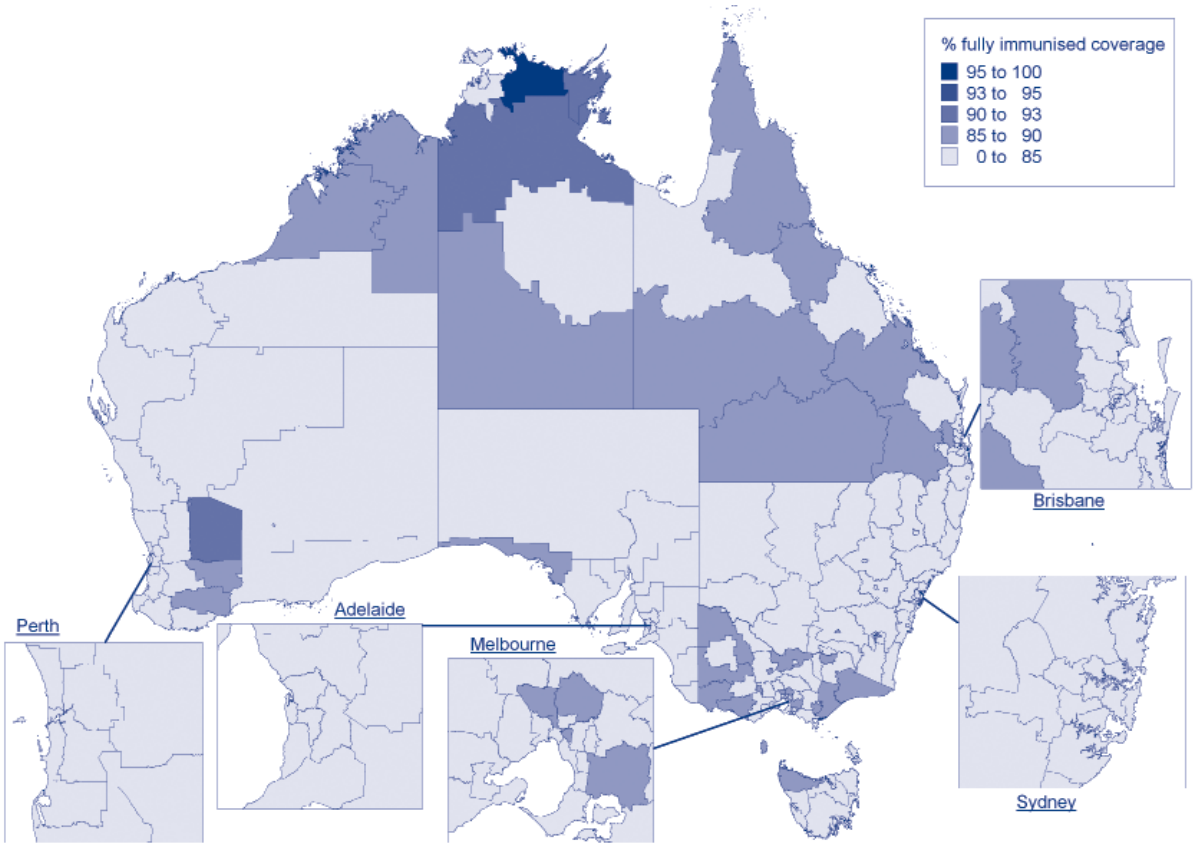


Figure 17: 'Fully immunised' coverage at 5 years of age, by Statistical Subdivision, Australia, 2008



The map of the proportion of conscientious objectors to immunisation (Figure 18) shows pockets of high levels of objection within jurisdictions in 2008, particularly in coastal areas of South East Queensland, northern New South Wales, Adelaide and south west Western Australia, which would be hidden if these data were reported at broader geographical levels.

The map of the proportion of children with no vaccines recorded (Figure 19) shows some additional areas not evident from maps of official conscientious objection, such as the eastern suburbs of Sydney and regional Victoria.

Provider type

The proportion of immunisations recorded on the ACIR as given by GPs, municipal councils and other providers in Australia by jurisdiction is shown in Figure 20. GPs administer the large majority of immunisations in Australia; the proportion given by GPs has increased over the past 10 years by almost 5% (not shown). Local government clinics also administer a substantial proportion of immunisations, especially in some jurisdictions. The only other category of provider administering major numbers of immunisations nationally is community health centres. Regional differences are marked, with immunisations almost entirely administered by GPs in some jurisdictions, while in others a majority are given by local government and community health clinics.

Discussion

These data reveal that Immunise Australia Program coverage targets have been reached for children both 12 and 24 months of age. However, this is not the case for children 5 years of age where coverage is poor in all jurisdictions.

Coverage at 24 months of age exceeds that at 12 months of age, and this is likely related to the exclusion of varicella vaccine at 18 months from calculation of 'fully vaccinated', the absence of any other vaccines administered between those ages, and the impact of immunisation incentives. The change in December 2007 in assessment age from 6 to 5 years for vaccines due at 4 years, resulted in a dramatic drop in coverage estimates for vaccines due at this age and has revealed that many children are not fully protected in a timely way for the diseases these vaccines guard against. This has been of particular concern during the pertussis epidemic of 2008 and 2009, when children aged 5 to 9 years were seriously affected.[24]

Figure 18: Proportion of official conscientious objectors to immunisation, Australia, 2008 (cohort born January 2001 – December 2007)

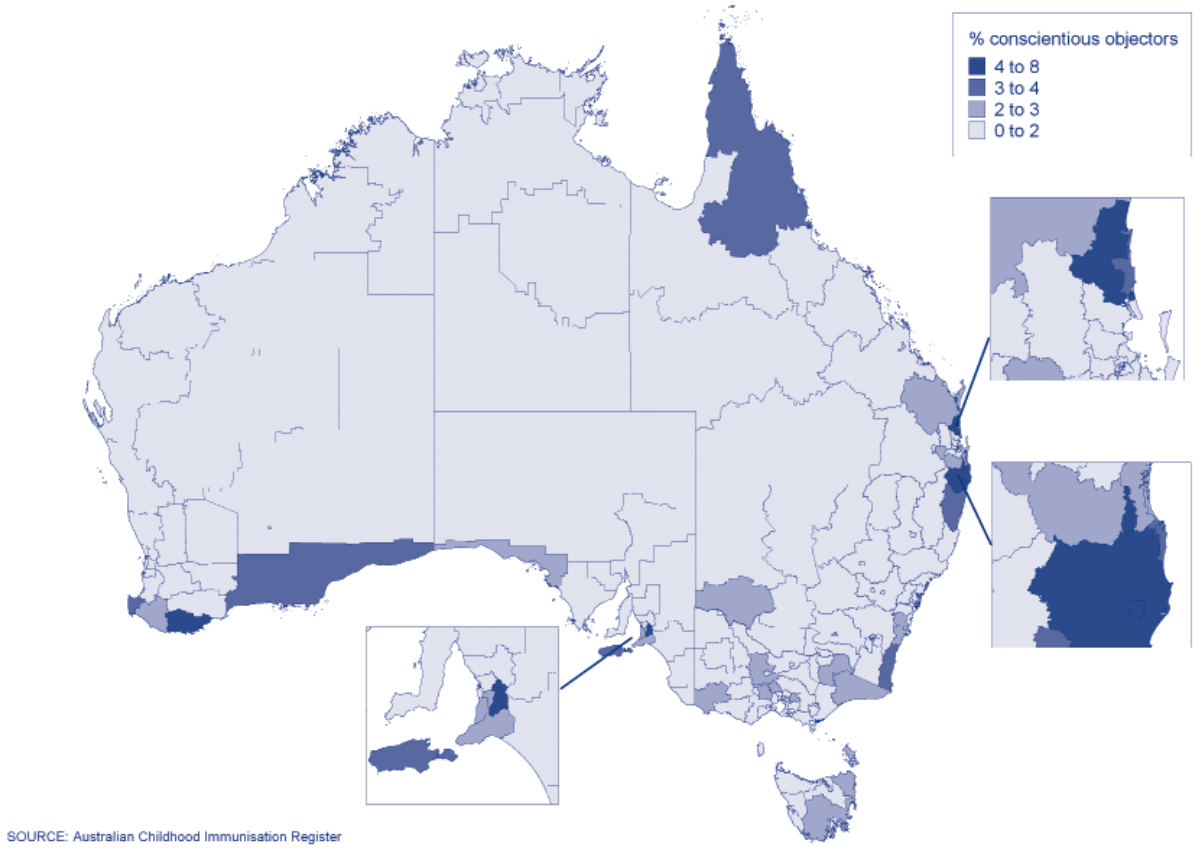


Figure 19: Proportion of children with no vaccines recorded on the ACIR, Australia, 2008 (cohort born January 2001 – December 2007)

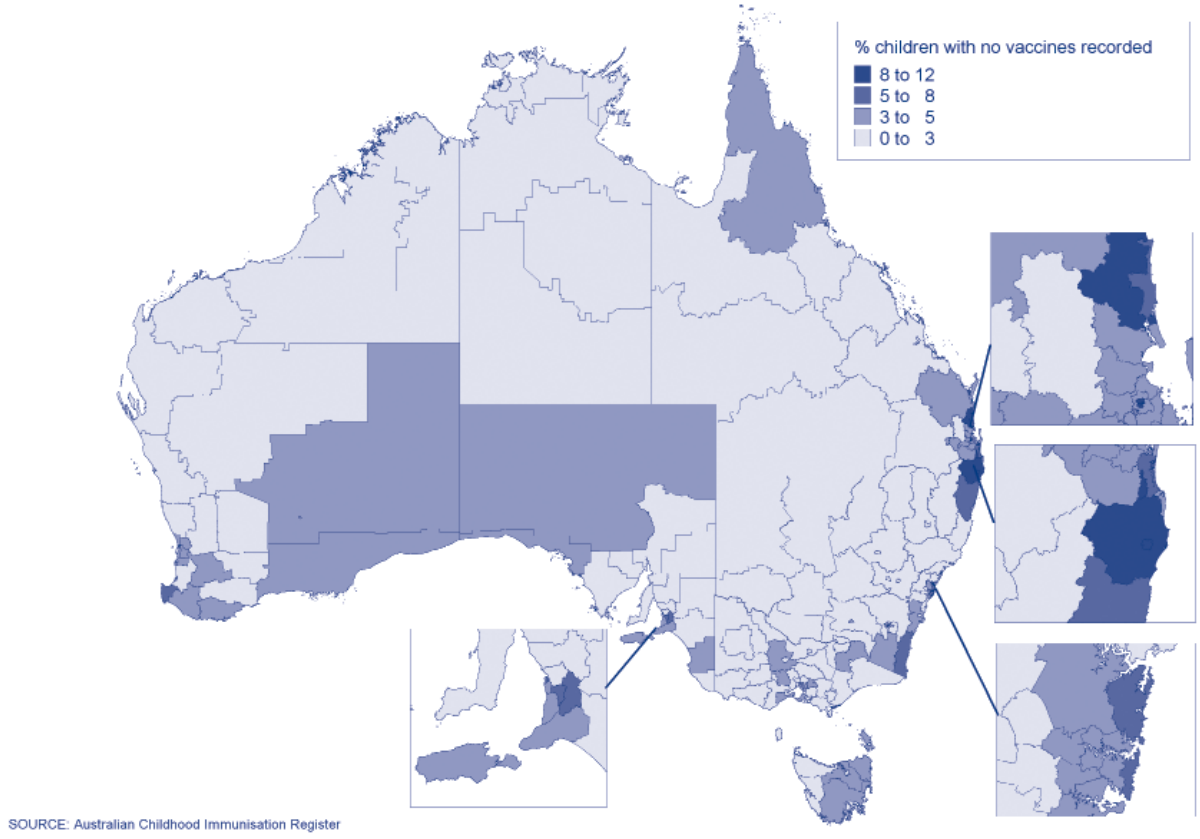
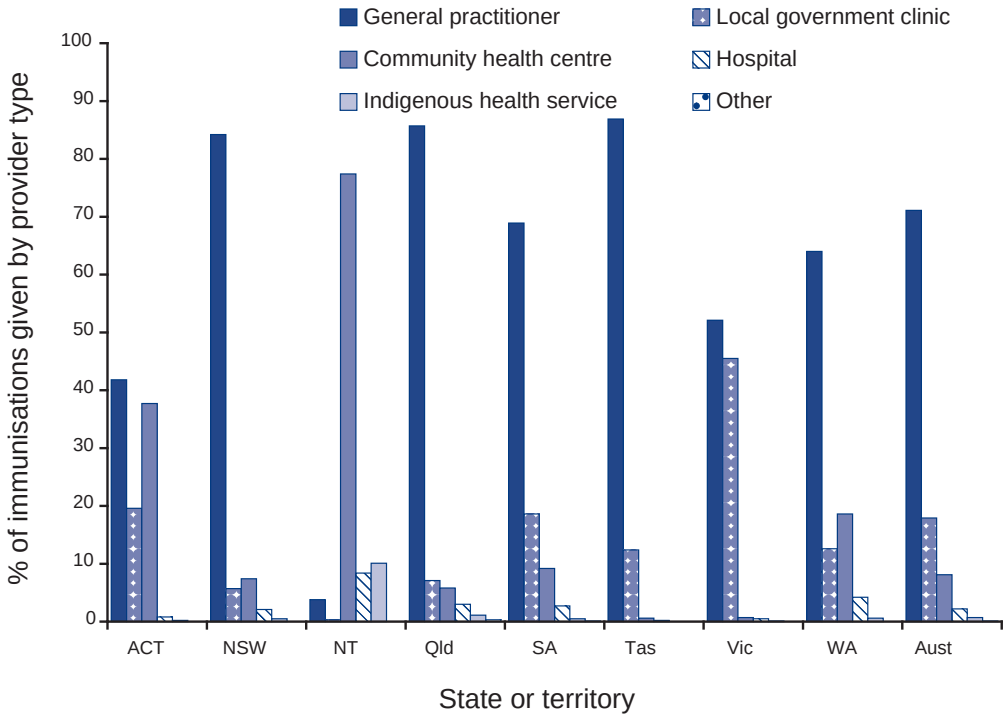


Figure 20: Proportion of immunisations on the ACIR given by various provider types, by state or territory, 2008



Immunisation incentives have positively impacted coverage estimates over time[12] and significant changes were made to these during 2008 for both providers and parents, as outlined in the Introduction. While it is too early to assess any impact on reported coverage in this analysis, it will be important to monitor the specific impacts of the 2008 changes to the incentives in the near future.

A number of vaccines that are included in the NIP are not included when calculating 'fully immunised' status or in eligibility for incentive payments. While these annual reports provide coverage data on these vaccines for the first time, data for the more long-standing and established vaccines are also available in quarterly *CDI* reports and in data provided to GP Divisions and immunisation providers. Coverage estimates for 7vPCV and meningococcal C vaccines are comparable with estimates for vaccines that are included in 'fully vaccinated' calculations, but estimates for varicella and rotavirus are lower. Strict upper age limits applying with rotavirus vaccination probably explains lower rotavirus coverage whilst varicella is the only vaccine due at 18 months, which makes this milestone a weak one. As these vaccines have been routinely incorporated into the childhood immunisation schedule for some time, their inclusion in the official coverage assessments for 'fully immunised', and wider dissemination, should be considered to facilitate monitoring of program delivery, although this will inevitably decrease official 'fully immunised' coverage estimates.

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and pneumococcal polysaccharide vaccine) remain sub-optimal. The extent of under-reporting to the ACIR for these vaccines is unknown but likely to be more than for 'universal' vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children[11] and adults.[25] A lack of provider knowledge about the recommendations, and poor identification of Indigenous children by immunisation providers, are also likely to be important contributing factors. Differences in schedules between jurisdictions may also contribute. For hepatitis A, the 1st dose is given at 12 months of age in the Northern Territory and Western Australia, whereas in Queensland and South Australia it is given at 18 months of age. Coverage in jurisdictions where it is given at 12 months of age is higher. Failure to receive a 2nd dose by 16% of children also contributed to the low coverage for hepatitis A vaccine. However, protective antibody responses after 1 dose is expected from a majority of children.[26] Differences in the scheduling of pneumococcal polysaccharide vaccine by jurisdiction may also partially explain the variation in coverage seen for that vaccine, with the Northern Territory and Western Australia giving the 1st dose of this vaccine at 18 months of age, while Queensland and South Australia give it at 24 months of age.

Although coverage data reveal that most children eventually complete the scheduled vaccination series by the 24-month milestone, many still do not do so in a timely manner. While there have been significant improvements in coverage in Australia over the past 4–5 years, vaccination delay as measured in this report has improved only marginally. This is a concern, especially for diseases where multiple vaccine doses are required for protection and the disease risk among young infants is significant (e.g. pertussis). Immunisation at the earliest appropriate age should be a public health goal for countries such as Australia where high levels of vaccine coverage at milestone ages have been achieved.

In comparison with other countries, reported coverage at 12 months of age is higher in many other countries.[27] However, with more than 3% of children not vaccinated due to ideological reasons, the greater than 91% of Australian infants fully immunised at 12 months of age is above the national target and would be difficult to improve upon. Rather, the ACIR has shown the rapid uptake of new vaccines and consistently high coverage for all vaccines, unlike some other developed countries.[28,29] The reporting of national small area coverage data has not been noted elsewhere and vaccination timeliness has been reported elsewhere but not routinely.[7]

In conclusion, data provided by the ACIR in this report reflect the successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for varicella and rotavirus vaccines are below that for other vaccines, coverage is low in some small geographic areas, timeliness of vaccination could be improved, particularly for Indigenous infants, and coverage for vaccines recommended only for Indigenous infants is lower than for other vaccines. The ACIR continues to be a very useful tool for administering the NIP and monitoring its implementation.

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References

1. Hull B, Lawrence G, MacIntyre CR, McIntyre P. *Immunisation coverage: Australia 2001*. Canberra: Australian Government Department of Health and Ageing, 2002.

2. Hull BP, McIntyre PB, Heath TC, Sayer GP. Measuring immunisation coverage in Australia. A review of the Australian Childhood Immunisation Register. *Aust Fam Physician* 1999;28(1):55–60.

3. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Immunisation coverage in Australia corrected for under-reporting to the Australian Childhood Immunisation Register. *Aust N Z J Public Health* 2003;27(5):533–538.

4. Hull BP, McIntyre PB. Immunisation coverage reporting through the Australian Childhood Immunisation Register – an evaluation of the third-dose assumption. *Aust N Z J Public Health* 2000;24(1):17–21.

5. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Estimating immunisation coverage: is the 'third dose assumption' still valid? *Commun Dis Intell* 2003;27(3):357–361.

6. Hull BP, McIntyre PB. Timeliness of childhood immunisation in Australia. *Vaccine* 2006;24(20):4403–4408.

7. Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? *Commun Dis Intell* 2004;28(2):238–243.

8. Hull BP, McIntyre PB, Couzos S. Evaluation of immunisation coverage for Aboriginal and Torres Strait Islander children using the Australian Childhood Immunisation Register. *Aust N Z J Public Health* 2004;28(1):47–52.

9. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Is low immunisation coverage in inner urban areas of Australia due to low uptake or poor notification? *Aust Fam Physician* 2003;32(12):1041–1043.

10. Hull BP, McIntyre PB, Sayer GP. Factors associated with low uptake of measles and pertussis vaccines – an ecologic study based on the Australian Childhood Immunisation Register. *Aust N Z J Public Health* 2001;25(5):405–410.

11. Hull BP, Deeks S, Menzies R, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait islander children? A 2007 update. *Commun Dis Intell* 2008;32(2):257–260.

12. Lawrence GL, MacIntyre CR, Hull BP, McIntyre PB. Effectiveness of the linkage of child care and maternity payments to childhood immunisation. *Vaccine* 2004;22(17–18):2345–2350.

13. Lawrence GL, Hull BP, MacIntyre CR, McIntyre PB. Reasons for incomplete immunisation among Australian children. A national survey of parents. *Aust Fam Physician* 2004;33(7):568–571.

14. Hull B, Deeks S, Menzies R, McIntyre P. Immunisation coverage annual report, 2007. *Commun Dis Intell* 2009;33(2):170–187.

15. Australian Government Department of Health and Ageing. Communicable Diseases Surveillance – additional reports: Australian childhood immunisation coverage. *Commun Dis Intell* 2008;32(3):357–359.

16. Australian Government Department of Health and Ageing, Medicare Australia. *General Practice Immunisation Incentives Scheme, 2007.* Accessed on 18 December 2007. Available from: http://www.medicare.gov.au/provider/incentives/gpii/index.jsp#N100D3

17. National Health and Medical Research Council. *The Australian Immunisation Handbook* 9th edn. Canberra, Australia: Australian Government Department of Health and Ageing; 2008.

18. O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. *Commun Dis Intell* 1998;22(3):36–37.

19. Australian Government Department of Health and Aged Care. *Measuring Remoteness: Accessibility/Remoteness Index of Australia*. Occasional Papers, New Series No. 14. Canberra: Department of Health and Aged Care, 2001.

20. Rank C, Menzies RI. How reliable are Australian Childhood Immunisation Register coverage estimates for indigenous children? An assessment of data quality and coverage. *Commun Dis Intell* 2007;31(3):283–287.

21. Australian Bureau of Statistics. Australian Standard Geographical Classification, 2001. Cat. no. 1216.0. Canberra: Australian Bureau of Statistics, 2001.

22. MapInfo version 10.0 [computer program]. Version 7. New York: MapInfo Corporation, 2009.

23. Australian Bureau of Statistics. Statistical Subdivision from Postal Area 2006 Concordance. Canberra: Australian Bureau of Statistics, 2007. Accessed on 6 December 2008. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/39433889d406eeb9ca2570610019e9a5/5942283858e38743ca25730c00009f2e!OpenDocument

24. Australian Government Department of Health and Ageing. Communicable Diseases Surveillance – Tables: National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2009;33(1):63–69.

25. Menzies R, Turnour C, Chiu C, McIntyre P. Vaccine Preventable Diseases and Vaccination Coverage in Aboriginal and Torres Strait Islander People, Australia 2003 to 2006. *Commun Dis Intell* 2008;32(Suppl):S2–S67.

26. Plotkin S, Orenstein W, Offit P. *Vaccines*. 5th edn. Elsevier, 2008.

27. World Health Organization. *Reported estimates of DTP3 coverage. 2009.* Accessed on 6 November 2009. Available from: http://www.who.int/immunization_monitoring/en/globalsummary/timeseries/tscoveragedtp3.htm

28. Centers for Disease Control and Prevention. National, state, and local area vaccination coverage among children aged 19–35 months—United States, 2008. *MMWR Morb Mortal Wkly Rep* 2009;58(33):921–926.

29. Health Protection Agency. *NHS Immunisation Statistics, England 2008–09 Report*. The Health and Social Care Information Centre, 2009. Accessed on 6 November 2009.

Annual report: surveillance of adverse events following immunisation in Australia, 2009

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Abstract

This report summarises Australian passive surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration (TGA) for 2009, and describes reporting trends over the 10-year period 2000 to 2009. There were 2,396 AEFI records for vaccines administered in 2009, the highest number reported, a 46% increase over the 1,638 in 2008. The increase was almost entirely due to reports related to the introduction of pandemic H1N1 (pH1N1) 2009 influenza vaccine from September 2009 (n=1,312) largely from the members of the public. The pH1N1 AEFI reporting rate for people aged ≥18 years was 34.2 per 100,000 administered doses compared with 2.8 for seasonal influenza vaccine. The rates in ≥65 year-olds were 28.0, 1.6 and 13.3 for pH1N1, seasonal influenza and polysaccharide pneumococcal, respectively. The high reporting rate for pH1N1 vaccine is likely to be at least partly due to enhanced reporting seen for all new vaccines and greater levels of reporting from members of the public in response to the implementation of strategies to encourage reporting, as part of the pH1N1 program. For children <7 years, AEFI reporting rates in 2009 (14.1 per 100,000 administered doses) were similar to previous years. There were 193 (8%) AEFI reports classified as serious; 6 deaths temporally associated with immunisation were reported but none were judged to have a causal association. As in previous years, the most commonly reported reactions were allergic reaction, injection site reaction, fever, headache, malaise, nausea and myalgia. The most commonly reported reactions following pH1N1 influenza vaccine were allergic reaction (n=381), headache (n=289), fever (n=235), pain (n=186), nausea (n=180) and injection site reaction (n=178). The data within the limitation of passive surveillance provide a reference point for ongoing reporting of trends in AEFI by age group, severity and vaccine type and illustrate the value of the national TGA database as a surveillance tool for monitoring AEFI nationally. *Commun Dis Intell* 2010;34(3):259–276.

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

Introduction

The aim of passive post-licensure surveillance of adverse events following immunisation (AEFI) is to monitor vaccine and immunisation program safety. An 'adverse event following immunisation' is generally regarded as any serious or unexpected adverse event that occurs *after* a vaccine has been given, which may be related to the vaccine itself or to its handling or administration. An AEFI can be *coincidentally* associated with the *timing* of immunisation without necessarily being caused by the vaccine or the immunisation process. Analysing trends in passive reports can identify signals that are unexpected adverse events that have not been detected in pre-licensure vaccine trials.[1,2] Passive surveillance is unable to determine the causal relationship between an event and vaccination. Hence, a signal may require the generation of a hypothesis and appropriate epidemiological studies to investigate causality.

Several important changes to vaccine funding and availability occurred in 2008 and 2009 that impact on the AEFI surveillance data presented in this report.

- The most significant change during 2009 was the introduction of pandemic H1N1 2009 influenza (pH1N1) vaccine (Panvax®), which was rolled out across Australia on 30 September 2009 for people aged ≥10 years. In December 2009, the pandemic vaccine was made available to children aged 6 months to 10 years.

- The Northern Territory commenced using a new 10-valent pneumococcal vaccine (Synflorix®) from October 2009 at 2, 4, 6 and 12 months of age instead of the 3-dose 7-valent pneumococcal schedule (Prevenar®). At the same time they also ceased using the 23-valent pneumococcal polysaccharide booster for Indigenous children at 18 months of age.

- By late 2009, all states and territories were using the single hexavalent DTPa-IPV-HepB-Hib (Infanrix hexa® vaccine for all children at 2, 4 and 6 months of age,[3–5] due to an

international shortage of *Haemophilus influenzae* type b (Hib) (PedvaxHib® (monovalent) and Comvax® (Hib-HepB)) vaccines.[6] In March 2008, Queensland, South Australia and Victoria changed from using 2 combination vaccines (quadrivalent DTPa-IPV and Hib-HepB) to the single hexavalent DTPa-IPV-HepB-Hib vaccine. In February 2009, Western Australia stopped using PedvaxHib® for Indigenous children so that all children received the single hexavalent DTPa-IPV-HepB-Hib vaccine. The Northern Territory continued using Comvax®until October 2009, when it also changed to the hexavalent vaccine. All other jurisdictions had already been using the hexavalent vaccine since November 2005.

- In 2008, Western Australia commenced a seasonal influenza vaccination program for all children aged 6 months – 5 years (born after 1 April 2003). Children should receive 2 doses of vaccine given at least 1 month apart followed by 1 dose annually.

Previous changes to the National Immunisation Program (NIP) schedule[7–9] also impact on the interpretation of trend data, and have been described in detail in previous reports published regularly since 2003.[10–22] These are:

(i) in 2003, the commencement of the meningococcal C conjugate vaccine (MenCCV) immunisation program and the removal of the 18-month dose of DTPa vaccine;[8]

(ii) from 2004, the progressive introduction of a dose of dTpa for adolescents;[8]

(iii) in January 2005, the commencement of the 7-valent pneumococcal conjugate vaccine (7vPCV) program for infants and the 23-valent polysaccharide vaccine (23vPPV) for adults aged ≥65 years;[7]

(iv) in November 2005, varicella for infants and at 12–13 years of age for those with no evidence of previous vaccination or varicella infection, and the replacement of oral poliovirus vaccine with inactivated poliovirus vaccine (IPV) for children. All IPV-containing vaccines include diphtheria-tetanus-acellular pertussis (DTPa) antigens (i.e. quadrivalent vaccines) and some also include hepatitis B (HepB) and/or *Haemophilus influenzae* type b (Hib) antigens (i.e. pentavalent and hexavalent vaccines). The specific combination vaccines administered at 2, 4, and 6 months of age at times varied between states and territories during the period covered by this report, but all jurisdictions provide DTPa-IPV quadrivalent vaccine at 4 years of age;[9]

(v) in April 2007, the national human papillomavirus (HPV) immunisation program commenced for all girls aged 12–18 years, and was extended to the 19–26 year age group in July 2007;[7] and

(vi) in July 2007, rotavirus vaccines were added to the NIP for all infants in Australia,[7] following the earlier introduction in the Northern Territory in October 2006.

Methods

AEFI are notified to the Therapeutic Goods Administration (TGA) by state and territory health departments, health professionals, vaccine manufacturers and members of the public.[8,9] All reports are assessed using internationally consistent criteria[23] and entered into the Australian Adverse Drug Reactions System (ADRS) database. All reports for vaccines and complementary medicines, plus all serious reports for drugs, are forwarded to the Adverse Drug Reactions Advisory Committee (ADRAC) for review at regular meetings. This is an expert committee of the TGA composed of independent medical experts who have expertise in areas of importance to the evaluation of medicine safety.

Adverse events following immunisation data

De-identified information on all AEFI reported to the TGA from 1 January 2000 to 28 February 2010 and stored in the ADRS database were released to the National Centre for Immunisation Research and Surveillance. Readers are referred to previous AEFI surveillance reports for a description of the surveillance system and methods used to evaluate reports to the TGA.[13,14]

AEFI records* contained in the ADRS database were eligible for inclusion in the analysis if a vaccine was recorded as 'suspected'† of involvement in the reported adverse event and *either;*

(a) the vaccination occurred between 1 January 2000 and 31 December 2009 *or;*

* The term 'AEFI record' is used throughout this report because a single AEFI notification/report to the Medicine Safety Monitoring Unit can generate more than 1 record in the ADRS database. This may occur if there is a time sequence of separate adverse reactions in a single patient, such as systemic and local reactions.

† Records are classified as 'suspected' if the report contains sufficient information to be valid and the relationship between reported reactions and drugs are deemed as biologically plausible.

(b) for records where the vaccination date was not recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2009.

Study definitions of adverse events following immunisation outcomes and reactions

AEFI were defined as 'serious' or 'non-serious' based on information recorded in the ADRS database and criteria similar to those used by the World Health Organization[23] and the US Vaccine Adverse Events Reporting System.[24] In this report, an AEFI is defined as 'serious' if the record indicated that the person had recovered with sequelae, been admitted to a hospital or hospitalisation was prolonged, experienced a life-threatening event, or died.

The causality ratings of 'certain', 'probable' and 'possible' are assigned to individual AEFI records by the TGA. They describe the likelihood that a suspected vaccine or vaccines was/were associated with the reported reaction at the level of the individual vaccine recipient. Factors that are considered in assigning causality ratings include the timing (minutes, hours etc) and the spatial correlation (for injection site reactions) of symptoms and signs in relation to vaccination, and whether one or more vaccines were administered, and are outlined in more detail elsewhere.[13] However, in many instances a causal association between vaccines administered to an individual and events that subsequently occurred cannot be clearly ruled in or out. In addition, children in particular often receive several vaccines at the same time. Therefore, all administered vaccines are usually listed as 'suspected' of involvement in a systemic adverse event as it is usually not possible to attribute the AEFI to a single vaccine.

Typically, each AEFI record lists several symptoms, signs and/or diagnoses that have been re-coded by TGA staff from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).[25] AEFI reports of suspected anaphylaxis and hypotonic-hyporesponsive episodes (HHE) were reviewed by ADRAC and classified using the Brighton Collaboration case definitions.[26,27]

To analyse reported AEFI, MedDRA® coding terms were grouped to create a set of reaction categories. Firstly, reaction categories were created that were analogous to the AEFI listed and defined in *The Australian Immunisation Handbook* (9th edition).[9] Where MedDRA® coding terms could not be categorised into *Handbook* categories, additional categories were created for those that were listed in more than 1% of AEFI records (e.g. headache, dizziness, change in heart or respiratory rate or rhythm). Reaction terms listed in less than 1% of records were grouped into broader categories based on the organ system where the reaction was manifested (e.g. gastrointestinal, neurological).

Data analysis

All data analyses were performed using SAS software version 9.1.3.[28] The distribution of AEFI records was analysed by age, gender and jurisdiction. Average annual population-based reporting rates were calculated for each state and territory and by age group using population estimates obtained from the Australian Bureau of Statistics.

AEFI reporting rates per 100,000 administered doses were estimated where reliable information was available on the number of doses administered – for influenza and pH1N1 vaccines in adults aged ≥18 years, for 23vPPV in ≥65 year-olds and for 10 vaccines funded through the NIP for children aged <7 years.

Denominator data to estimate influenza and 23vPPV AEFI reporting rates were obtained from a national adult coverage survey conducted in 2006 (unpublished), and for pH1N1 using the Pandemic Vaccination Survey.[29] For 23vPPV the number of people vaccinated per year was derived from the number of people fully vaccinated in 2006 divided by 5. The number of administered doses of each of the 10 childhood vaccines was calculated from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99% of children aged <7 years.[30]

Notes on interpretation

Caution is required when interpreting the AEFI data presented in this report. Due to reporting delays and late onset of some AEFI, the data are considered preliminary, particularly for the 4th quarter of 2009. Data published in previous reports for 2000–2009[10–22] differ from that presented in this report for the same period because the data in this report have been updated to include delayed notifications of AEFI to the TGA prior publication.

The information collated in the ADRS database is intended primarily for signal detection and hypothesis generation. While AEFI reporting rates can be estimated using appropriate denominators, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected AEFI, and the variable quality and completeness of information provided in individual AEFI notifications.[10–22,31]

It is important to note that this report is based on vaccine and reaction term information collated in the ADRS database and not on comprehensive clinical notes or case reviews. Individual database records list symptoms, signs and diagnoses that were used to define a set of reaction categories based on the case definitions provided in the 9th edition of *The Australian Immunisation Handbook*.[9] These reaction categories are similar, but not identical, to the AEFI case definitions.

The reported symptoms, signs and diagnoses in each AEFI record in the ADRS database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines.

Results

The ADRS database included a total of 2,396 AEFI records where the date of vaccination (or onset of adverse event, if vaccination date was not reported) occurred between 1 January and 31 December 2009. Of these, 1,312 records (55%) related to pH1N1 influenza vaccine, accounting for the increase of 46% over the total records for 2008.

In 2009, 43% of AEFI (n=1,025) were reported to the TGA via states and territories, with others reported directly. Of those directly reported to TGA, 28% (n=664) were reported by members of the public, 23% (n=552) by doctors or health professionals, 5% (n=110) by hospitals, and 2% (n=45) by drug companies (Table 1). The proportion reported by members of the public was much greater in 2009 than in 2008 (n=51, 3%), with 94% of the reports by members of the public following pH1N1 influenza vaccine.

Reporting trends

The overall AEFI reporting rate for 2009 was 11.0 per 100,000 population, compared with 7.2 per 100,000 population in 2008, and the highest in the decade 2000 to 2009.

Table 1: Reporter types for adverse events following immunisation (AEFI), ADRS database, 2008 and 2009

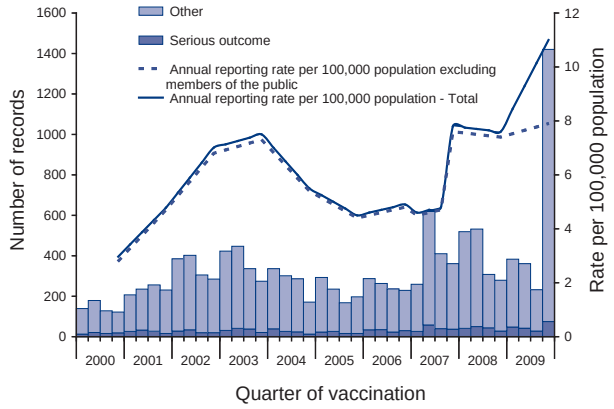
	State or territory									
Reporter type	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Other*	Total
2008										
Hospital	0	9	0	17	2	0	424†	8	0	460
Drug company	0	0	0	0	0	0	0	0	34	34
Doctors/medical	5	83	2	63	19	29	53	32	5	291
Public	0	7	0	18	0	2	1	3	20	51
State/territory	58	241	41	134	232	0	33	63	0	802
Total	63	340	43	232	253	31	511	106	59	1,638
2009										
Hospital	8	19	0	15	6	0	49	12	1	110
Drug company	0	0	0	0	0	0	0	0	45	45
Doctors/medical	10	190	0	106	36	33	87	80	10	552
Public	21	147	0	138	78	16	157	80	27	664
State/territory	45	94	40	164	198	0	440	35	9	1,025
Total	84	450	40	423	318	49	733	207	92	2,396
2009 (without Panvax®)										
Hospital	8	10	0	12	3	0	41	6	0	80
Drug company	0	0	0	0	0	0	0	0	33	33
Doctors/medical	4	51	0	42	9	16	27	43	7	199
Public	0	3	0	18	0	0	3	8	5	37
State or territory	35	81	33	19	135	0	398	34	0	735
Total	47	145	33	91	147	16	469	91	45	1,084

* Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. AEFI records in this category were notified mainly by pharmaceutical companies, members of the public, and general practitioners.

† 2008 SAFEVIC (Victoria) reports were counted as hospital but 2009 reports are state or territory.

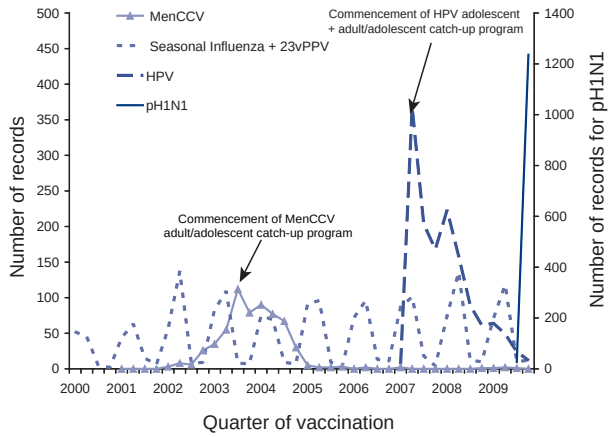
Figure 1 shows the sharp rise in AEFI in the last quarter of 2009 and Figure 2a shows that this rise was due to reports following receipt of pH1N1 influenza vaccine, introduced on 30 September. The impact of previous changes to the NIP on reported AEFI in adolescents and adults are also evident in Figure 2a, such as the commencement of the MenCCV program in 2003 and HPV program in 2007. Figures 2b and 2c show the impact on AEFI reports of other changes to the vaccination programs for children, including the removal of the 18-month DTPa dose in 2003, and commencement of 7vPCV in 2005 and rotavirus vaccine in 2007. Reporting rates usually increased with the commencement of a new vaccination program and then stabilised at lower rates.

Figure 1: Adverse events following immunisation, ADRS database, 2000 to 2009, by quarter of vaccination



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

Figure 2a: Frequently suspected vaccines, adverse events following immunisation for individuals aged >7 years, ADRS database, 2000 to 2009, by quarter of vaccination



The usual seasonal pattern of AEFI reporting, with peaks in the first half of the year, was also apparent in 2009 (Figure 2a). The seasonal peaks generally correspond to the months when more vaccinations are administered in Australia, particularly among 4– and 5-year-old children receiving measles-mumps-rubella (MMR) and DTPa-containing vaccines prior to commencing school in February, and older Australians receiving 23vPPV and influenza vaccine during the autumn months (March to June) (Figures 2a and 2b).

Figure 2b: Frequently suspected vaccines, adverse events following immunisation for children aged 1 to <7 years, ADRS database, 2000 to 2009, by quarter of vaccination

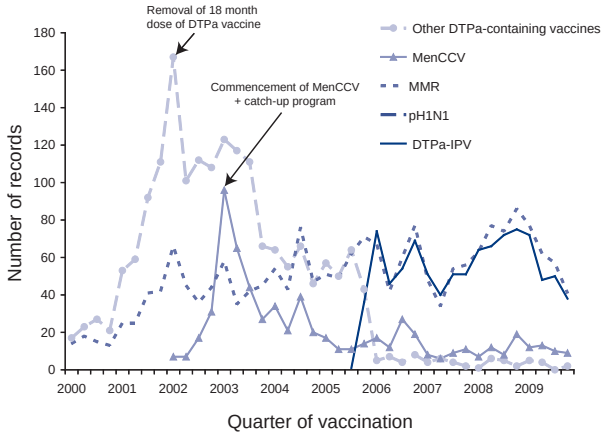
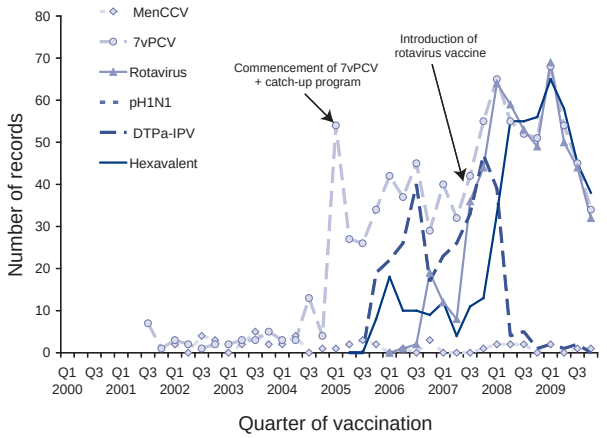


Figure 2c: Frequently suspected vaccines, adverse events following immunisation for children aged <1 years, ADRS database, 2000 to 2009, by quarter of vaccination



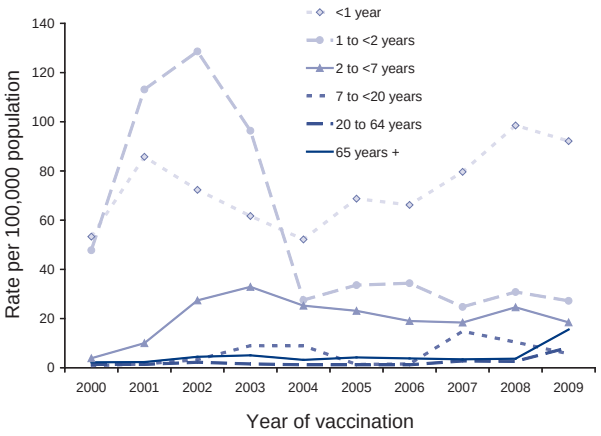
* Meningococcal C conjugate vaccine (MenCCV) was introduced into the NIP schedule on 1 January 2003; 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005; DTPa-IPV and DTPa-IPV-HepB-Hib (hexavalent) vaccines in November 2005; rotavirus (RotaTeq® and Rotarix®) vaccines on 1 July 2007; and pH1N1 influenza vaccine on 30 September 2009.

Age distribution

In 2009, the highest AEFI reporting rate per 100,000 population was in infants <1 year of age, the age group that received the highest number of vaccines (Figure 3). Compared with 2008, AEFI reporting rates decreased slightly among the < 1 year age group (6% decrease from 98.5 to 92.1 per 100,000 population), the 1 to < 2 year age group (12% decrease, from 30.8 to 27.2 per 100,000 population), the 2 to < 7 year age group (25% decrease, from 24.6 to 18.5 per 100,000 population) and for older children and adolescents (46% decrease, from 10.4 to 5.6 per 100,000 population). The decline in AEFI reporting rates for older children and adolescents were mainly attributable to a reduction in the numbers of reports related to HPV vaccine following cessation of the catch-up component of the HPV program.

However, AEFI reporting rates increased for the 20–64 year age group (2.6 to 8.2 per 100,000 population) and the >65 year age group (3.7 to 15.5 per 100,000 population), mainly associated with the introduction of the pH1N1 influenza vaccine.

Figure 3: Reporting rates of adverse events following immunisation per 100,000 population, ADRS database, 2000 to 2009, by age group and year of vaccination



Geographical distribution

AEFI reporting patterns varied between states and territories for vaccines received during 2009 (Table 2) as reported previously.[11,13,14,17,19–21] The Australian Capital Territory, South Australia and the Northern Territory had the highest reporting rates (23.9, 19.6 and 17.8 per 100,000 population, respectively) while New South Wales had the lowest rate (6.3 per 100,000 population). With the exception of the Northern Territory, AEFI reporting rates increased in all jurisdictions in 2009, largely related to the commencement of pH1N1 vaccination in September 2009. After excluding pH1N1, there was a decrease in reporting rates in all jurisdictions and in all age groups.

Table 2: Adverse events following immunisation (AEFI), ADRS database, January to December 2009, by jurisdiction

State or territory	AEFI records		Annual reporting rate per 100,000 population*			
	n	%	Overall	'Certain'/ 'probable' causality rating†	'Serious' outcome‡	Aged <7 years
Australian Capital Territory	84	4	23.9	2.8	1.7	101.7
New South Wales	450	19	6.3	0.5	0.4	9.9
Northern Territory	40	2	17.8	4.4	4.4	55.0
Queensland	423	18	9.6	0.8	0.6	9.4
South Australia	318	13	19.6	1.1	1.4	50.2
Tasmania	49	2	9.7	2.6	0.6	22.1
Victoria	733	31	13.5	1.5	1.1	67.1
Western Australia	207	9	9.3	1.3	0.8	26.3
Other§	92	4	na	na	na	na
Total	2,396	100	11.0	1.1	0.9	31.1

* Average annual rates per 100,000 population calculated using mid-2009 population estimates (Australian Bureau of Statistics).

† See previous report[13] for criteria used to assign causality ratings.

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

§ Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. AEFI records in this category were notified mainly by pharmaceutical companies (n=45), members of the public (n=27), and general practitioners (n=8).

Outcomes

Thirty-five per cent of reported AEFI in 2009 were defined as 'non-serious' while 8% were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event or death) (Table 3), and is similar to the proportions of serious AEFI observed in previous years.[11,19] A further 19% were recorded as not fully recovered at the time of reporting and 55% of these were following receipt of pH1N1 influenza vaccine. A total of 244 (10%) AEFI records were assigned causality ratings of either 'certain' (n=216, 9%) or 'probable' (n=28, 1%). Fewer 'serious' AEFI were assigned certain or probable causality ratings compared with 'non-serious' AEFI (7% versus 14%) (Table 3). The number of reported AEFI, severity of outcome and causality, for each vaccine, are shown in Table 4.

There was a relatively high number (918, 38%) of AEFI records in 2009 for which severity could not be definitively determined due to insufficient data, usually the absence of follow-up data on whether a full recovery had occurred. Eighty-two per cent of these were following the receipt of pH1N1 influenza vaccine and 50% were reported by members of the public. The most commonly reported adverse reactions were allergic reactions (25%), headache (20%), fever and injection site reaction (19% each), pain (14%), malaise (13%), myalgia and nausea (12% each), abdominal pain (6%), dizziness (8% each) and weakness (2%).

Six deaths were recorded as temporally associated with receipt of vaccines; five in adults following receipt of pH1N1 influenza vaccine and one in a child following seasonal influenza vaccination. The adults ranged in age from 47 to 90 years. Three of the adults had co-morbidities including cardiac, pulmonary and renal disease. The child had an intercurrent respiratory illness. All deaths were investigated and classified as not related to vaccination.

Vaccines

Thirty-three different vaccines were included in the 2,396 AEFI records received in 2009 (Table 4). The percentage of records where only 1 vaccine was reported differed by vaccine, typically varying according to whether multiple vaccines are routinely co-administered for the patient's age. The percentage of AEFI records assigned causality ratings of 'certain' or 'probable' also varied, in accordance with the frequency of injection site reactions, for which the attribution of causality is more straightforward. There were also variations in the proportions with outcomes defined as 'serious'.

Table 3: Outcomes of adverse events following immunisation (AEFI), ADRS database, 2009

Outcome	AEFI records		'Certain'/ 'probable' causality rating†		Age group‡			
					<7 years		≥7 years	
	n	%	n	%§	n	%§	n	%§
Non-serious	841	35	118	14	314	37	519	62
Not recovered at time of report	444	19	62	14	95	21	341	77
Not known (missing data) – total	918	38	50	5	120	13	785	86
Not known (missing data)‖	463	19	39	8	110	24	351	76
Serious:	193	8	14	7	80	41	110	57
recovered with sequelae	3		0		0		3	
hospital treatment – admission	172		13		75		95	
life-threatening event	12		1		4		7	
death	6		0		1		5	
Total	2,396	100	244	10	609	25	1,755	73

* Percentages relate to the total number of AEFI records (n=2,396).

† Causality ratings were assigned to AEFI records using criteria described previously.[13]

‡ AEFI records where both age and date of birth were not recorded are not shown (32 missing).

§ Percentages relate to the number of AEFI records with the specific outcome, e.g. of 841 AEFI records with a 'non-serious' outcome, 14% had causality ratings of 'certain' or 'probable' and 37% were for children aged <7 years.

‖ AEFI records with missing data reported by health professionals only (excluding reports from members of the public)

Table 4: Vaccine types listed as 'suspected' in records of adverse events following immunisation (AEFI), ADRS database, 2009

Suspected vaccine type*	AEFI records	One suspected vaccine or drug only†		'Certain'/ 'probable' causality rating‡		'Serious' outcome§		Age group‖ <7 years		Age group‖ ≥7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶	n	%¶
pH1N1	1,312	1,287	98	46	4	56	4	23	2	1,265	96
DTPa-IPV	218	80	37	64	29	12	6	213	98	5	2
MMR	213	22	10	9	4	18	8	197	92	16	8
7vPCV	212	2	1	1	1	37	17	210	99	2	1
DTPa-IPV-HepB-Hib	206	10	5	4	2	32	16	204	99	2	1
Rotavirus**	202	30	1	4	2	36	18	199	99	2	1
Influenza	162	134	83	27	17	0	19	17	10	144	89
HPV	153	110	72	13	9	13	9	1	1	149	97
23vPPV	82	67	82	35	43	4	5	2	2	80	98
dTpa	79	60	76	18	23	5	6	0	–	78	99
Hepatitis B	71	22	31	10	14	4	6	10	14	61	86
MenCCV	52	4	8	1	2	5	10	48	92	4	8
Hib	46	1	2	0	–	7	15	45	98	1	2
Varicella	41	23	56	1	2	6	15	23	56	18	44
DTPa	12	4	33	2	17	4	33	11	92	0	–
Hib-Hepatitis B	10	0	–	0	–	1	10	9	90	1	10
dT	9	6	67	4	44	1	11	0	–	9	100
Hepatitis A	9	2	22	1	11	0	–	4	44	5	56
BCG	7	7	100	4	57	2	29	6	86	1	14
Hepatitis A + B	7	5	71	1	14	2	29	0	–	7	100
Typhoid	7	1	14	0	–	2	29	1	14	5	71
Yellow fever	6	5	83	0	–	2	33	0	–	6	100
Hepatitis A-Typhoid	5	1	20	2	40	2	40	1	20	4	80
IPV	4	1	25	1	25	0	–	4	100	0	–
Japanese encephalitis	4	3	75	0	–	1	25	1	25	3	75
Men4PV	4	0	–	0	–	0	–	4	100	0	–
DTPa-IPV-HepB	3	0	–	0	–	2	67	3	100	0	–
dTpa-IPV	2	0	–	0	–	2	100	0	–	2	100
Rabies	2	2	100	1	50	0	–	1	50	1	50
10vPCV	2	0	–	0	–	0	–	2	100	0	–
Cholera	1	1	100	0	–	1	100	0	–	1	100
Tetanus	1	1	100	0	–	0	–	0	–	0	–
Q fever	1	1	100	0	–	0	–	0	–	1	100
Total‡‡	2,396	1,893	79	244	10	193	8	609	25	1,755	73

* See appendix for abbreviations of vaccine names.

† AEFI records where only 1 vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[13]

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

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

¶ Percentages are calculated for the number of AEFI records where the vaccine was suspected of involvement in the AEFI, e.g. HPV was 'suspected' in 153 AEFI records; this was the only suspected vaccine in 72% of the 153 AEFI records, 9% had 'certain' or 'probable' causality ratings, 9% were defined as 'serious' and 97% were for those aged ≥7 years.

** Rotavirus vaccine was added to the National Immunisation Program schedule on 1 July 2007.[7]

‡‡ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 vaccine.

The most frequently reported individual vaccine was pH1N1 with 1,312 records (55%) (Table 4). Vaccines containing diphtheria, tetanus and acellular pertussis antigens (including combination DTPa-containing vaccines and dTpa (adult/adolescent formulation)) were the next most commonly reported (520; 22% of records) (Table 4), with DTPa-IPV (218 records; 9%) and hexavalent DTPa-IPV-HepB-Hib (206 records; 9%) the most frequently reported vaccines in this group. In the <1 year age group, reports that included DTPa-IPV decreased and reports of DTPa-IPV-HepB-Hib increased, in line with the changes in usage of those vaccines as outlined in the Introduction (Figure 2c). The other frequently reported vaccines were MMR (213 records; 9%), 7vPCV (212 records; 9%), and rotavirus (202 records; 8%). The pH1N1 influenza vaccine, seasonal influenza vaccine and 23vPPV were among the more common vaccines listed as suspected of involvement in reported AEFI, particularly where only 1 vaccine was listed as suspected (Table 4).

In comparison to the number reported in 2008, AEFI reports were substantially reduced for the HPV vaccine (153 in 2009 vs 497 in 2008) following the peak in the catch-up program in 2008–2009, and for Hib-HepB (10 in 2009 vs 63 in 2008) following its reduced availability. Reports following 23vPPV were also lower in 2009 (82 vs 137), but data on vaccine use in 2009 comparison with 2008 are not available. Reports increased for Hib (46 vs 33) and DTPa-IPV-HepB-Hib (206 vs 169, Figures 2b and 2c) in line with increased usage, while dTpa reports also increased in 2009 (79 vs 44).

Reactions

The distribution and frequency of reactions listed in AEFI records for vaccines received in 2009 are shown in Tables 5 and 6. In Table 5, only the reaction categories analogous to those listed in *The Australian Immunisation Handbook*[9] are shown. In Table 6, other reaction categories are listed in descending order of frequency.

The most frequently reported adverse events were allergic reaction (26%) followed by injection site reaction (ISR) (25% of 2,396 AEFI records), fever (18%), headache (15%), malaise (11%), nausea (10%), myalgia (10%) and pain (9%) (Tables 5 and 6). ISR was the most commonly reported individual adverse event following receipt of DTPa-IPV (86%; 188/218), 23vPPV (80%; 66/82), MMR (60%; 128/213), and influenza vaccine (35%; 56/162), administered alone or in combination with other vaccines. Fourteen per cent of both pH1N1 (178/1312) and HPV (22/153) vaccine-related AEFI records listed ISR.

More severe AEFI included reports of convulsion (n=46), HHE (n=34), anaphylactic reaction (n=18), Guillain-Barré syndrome (GBS; n=12), thrombocytopenia (n=7), death (n=6; described previously in this report) and encephalitis (n=1).

The 46 reports of convulsion included 9 febrile convulsions. Nineteen were for children aged <7 years and 35% were from Victoria. The most commonly suspected vaccines in reposts of convulsion were HPV (n=18), 7vPCV (n=10), DTPa-IPV-HepB-Hib (n=9), rotavirus (n=7) and pH1N1 (n=5). The majority of HHE (22/34) were notified by Victoria. DTPa-containing vaccines were suspected for 29 reports, with hexavalent DTPa-IPV-HepB-Hib in 24 reports and DTPa-IPV in three. Other vaccines given concomitantly with hexavalent vaccine (7vPCV (n=25) and rotavirus (n=21)) were also frequently included in reports of HHE. Seven of the 18 reports of anaphylaxis in 2009 occurred following receipt of only pH1N1 influenza vaccine, while others occurred following receipt of DTPa-IPV (n=3), MMR (n=3), HPV (n=2), seasonal influenza vaccine (n=2), 23vPPV (n=2), HepB (n=1), rotavirus (n=1), DTPa-IPV-HepB-Hib (n=1) and adult dTpa (n=1). The 12 records coded as GBS included 10 reports following receipt of pH1N1 influenza vaccine and two following seasonal influenza vaccine.

Reactions shown in Table 6 include headache, malaise, myalgia, nausea, pain, dizziness and gastrointestinal reactions. Many of the reaction terms shown in this table were reported for pH1N1, HPV and rotavirus vaccines. Reactions mentioned in less than 1% of AEFI records in 2009 are shown in the lower portion of Table 6, grouped by organ system categories.

The number of reports in each reaction category has changed over time (Figure 4). Reports of headache and allergic reactions peaked in 2003, 2007 and again in 2009, coinciding with the national school-based MenCCV immunisation program in 2003, the HPV school program in 2007 and the commencement of pH1N1 vaccination from September 2009. Much of the variation in reporting of ISR related to specific changes in the immunisation schedules for vaccines that are known to have higher rates of ISR, including DTPa-containing vaccines, MenCCV, 23vPCV and HPV vaccine.[10–22,32,33] Increases in reports of fever are associated with the new vaccines added to the NIP in the reporting period, including rotavirus and HPV in 2007.

Table 5: Reaction categories of interest* mentioned in records of adverse events following immunisation (AEFI), ADRS database, 2009

Reaction category*	AEFI records	Only reaction reported†		'Certain'/'probable' causality rating‡		Age group§			
						<7 years		≥7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Allergic reaction¶	634	94	15	24	4	131	21	497	78
Injection site reaction	600	121	20	202	34	238	40	358	60
Fever	430	10	2	6	1	131	30	295	69
Rash**	130	49	38	3	2	61	47	67	52
Arthralgia	83	5	6	0	–	1	1	80	96
Syncope	73	18	25	4	5	5	7	68	93
Lymphadenopathy/itis††	51	7	14	5	10	7	14	44	86
Convulsions	46	19	41	3	7	19	41	27	59
Abnormal crying	44	2	5	0	–	44	100	–	–
Hypotonic-hyporesponsive episode	34	20	59	3	9	34	100	–	–
Arthritis	26	4	15	1	4	3	12	22	85
Anaphylactic reaction	18	9	50	4	22	4	22	14	78
Guillain-Barré syndrome	12	11	92	0	–	0	–	12	100
Abscess	11	4	36	7	64	5	45	6	55
Intussusception	8	5	63	0	–	7	88	0	–
Thrombocytopenia	7	4	57	0	–	3	43	4	57
Death	6	4	67	0	–	1	17	5	83
Brachial neuritis	4	2	50	0	–	0	–	3	75
Parotitis	2	0	–	0	–	0	–	2	100
Orchitis	2	0	–	0	–	1	50	1	50
Encephalitis	1	1	100	0	–	0	–	1	100
Osteitis	1	0	–	0	–	0	–	1	100
Encephalopathy	1	0	–	0	–	0	–	1	100
Total‡‡	2,396	1,893	79	244	10	609	25	1,755	73

* Reaction categories were created for the AEFI of interest listed and defined in *The Australian Immunisation Handbook*, (9th edition, p 58–65 and 360–3)[9] as described in the Methods section.

† AEFI records where only 1 reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[13]

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

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 600 AEFI records listing injection site reaction, 20% listed only 1 type of reaction while 34% had a causality rating of 'certain' or 'probable' and 40% were for children aged <7 years.

¶ Allergic reaction includes skin reactions including pruritus, urticaria, periorbital oedema, facial oedema, erythema multiforme etc. (excludes skin reactions presented elsewhere in this table); and/or gastrointestinal (e.g. diarrhoea, vomiting) symptoms and signs but does not include other abdominal symptoms like abdominal pain, nausea, flatulence, abnormal faeces, haematochesia etc. Does not include anaphylaxis.

** Includes general terms of rash but does not include pruritic rash.

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

‡‡ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 reaction term.

Table 6: 'Other'* reaction terms listed in records of adverse events following immunisation (AEFI), ADRS database, 2009

Reaction term*	AEFI records	Only reaction reported†		'Certain'/'probable' causality rating‡		Age group§			
						<7 years		≥7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Headache	362	13	4	9	2	7	2	345	95
Malaise	256	6	2	6	2	44	17	207	81
Nausea	237	1	0.4	6	3	7	3	229	97
Myalgia	233	14	6	2	1	8	3	222	95
Pain	217	9	4	7	3	4	2	208	96
Respiratory	212	23	11	6	3	66	31	146	69
Neurological/psychological	176	8	5	4	2	66	38	110	62
Dizziness	169	6	4	6	4	2	1	167	99
Circulatory	102	6	6	2	2	18	18	81	79
Reduced sensation	102	19	19	9	9	–		100	98
Abdominal pain	98	1	1	3	3	16	16	80	82
ENT	91	10	11	3	3	4	4	85	93
Gastrointestinal – RVV¶	87	9	10	3	3	87	100	–	–
Somnolence	53	3	6	2	4	23	43	30	57
Increased sweating	69	2	3	3	4	2	3	67	97
Erythema	49	8	16	1	2	15	31	33	67
Pallor	41	1	2	4	10	15	37	26	63
Flushing	39	2	5	3	8	3	8	36	92
Weakness	37	–	–	1	3	–	–	37	100
Vision impaired	34	–	–	1	3	1	3	33	97
Oedema	31	2	6	2	6	7	3	23	74
Tremor	29	3	10	2	7	3	10	26	90
Spinal chord/peripheral nerve	28	13	46	–	–	1	4	27	96
Haematological/metabolic	25	5	20	3	3	3	12	22	88
Aphasia	15	1	7	–	–	–	–	15	100
Other	301	27	9	9	3	51	17	244	81
eye or ear	43	1	2	1	2	6	14	37	86
cardiovascular	32	3	9	2	6	8	25	24	75
infection	27	9	33	1	4	6	22	20	74
general non-specific	27	4	15	–	–	5	19	21	78
renal/urogenital	20	–	–	1	5	3	15	17	85
gastrointestinal**	16	1	6	–	–	2	13	13	81
respiratory	14	–	–	–	–	–	–	14	100
skin††	14	3	21	–	–	5	36	9	64
musculoskeletal	11	1	9	1	9	–	–	10	91
metabolic/endocrine	9	–	–	–	–	5	56	4	44
haematological	8	2	25	1	13	–	–	8	100
psychological	7	1	14	–	–	1	14	6	86
miscellaneous	6	–	–	–	–	–	–	6	100
neurological	6	2	33	–	–	2	33	4	67
pregnancy/congenital	6	–	–	–	–	1	17	5	83

* Reaction terms not listed in *The Australian Immunisation Handbook*[9] but included in AEFI records in the ADRAC database. The top part of the table shows reaction terms included in 1% or more of AEFI records; the bottom part of the table shows reaction terms, grouped by organ system, that were included in less than 1% of AEFI records.

† AEFI records where only 1 vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[13]

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

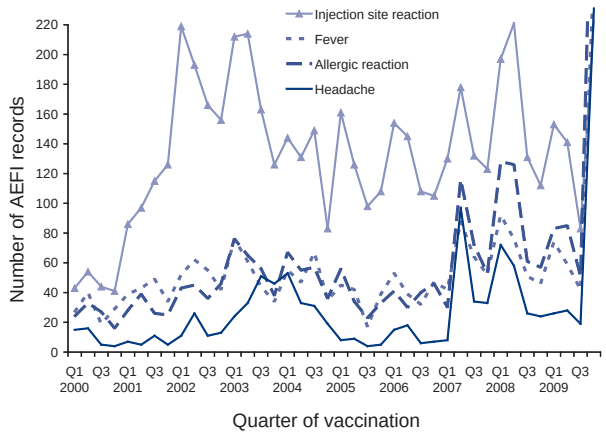
‖ AEFI records are not shown if both age and date of birth were not reported.

¶ Gastrointestinal – RVV includes all the GI reactions following rotavirus vaccination.

** Other, gastrointestinal does not include GI reactions and Gastrointestinal – RVV signs and symptoms.

†† Other, skin includes purpura, petechiae, blister, burning, dermatitis, dry skin etc. but does not include skin reactions.

Figure 4: Selected frequently reported adverse events following immunisation, ADRS database, 2000 to 2009, by quarter of vaccination



Dose-based adverse events following immunisation reporting rates

Seasonal influenza vaccine and adults aged ≥18 years

In 2009, there were 135 adverse events following influenza vaccination of people aged ≥ 18 years. The AEFI reporting rate was 2.5 per 100,000 administered doses, similar to the rate in 2008 (Table 7). As seen in previous years, the overall AEFI reporting rates were higher for vaccinees aged 18–64 years than among older people. However, there was an increase in the reporting rate of serious AEFI in all age groups and particularly among older people (aged ≥65 years). The most frequently reported adverse events were ISR, allergic reaction, fever, myalgia, malaise, dizziness, nausea and headache (reporting rate 0.9, 0.8, 0.5, 0.4 and 0.3 (malaise, dizziness, nausea and headache each) per 100,000 doses, respectively). The reporting rate for each of these reactions was higher in the 18–64 year age group. There were 2 reports of GBS following seasonal influenza vaccination in 2009 giving a reporting rate of 0.04 per 100,000 doses, well within the expected reporting rates.

Pneumococcal vaccine and adults aged ≥65 years

There were 57 AEFI reports for older adults that included 23vPPV, with 2 reports coded as serious and 47 reports of ISR. The AEFI reporting rate was 13.3 per 100,000 doses, with 0.5 per 100,000 doses serious and 10.9 per 100,000 doses for ISR reports. This was lower than the rate reported for 2008 (18.9 per 100,000 doses with 1.2 serious).[11]

Scheduled vaccines for children aged <7 years

There were a total of 609 AEFI records for children aged <7 years for vaccines administered in 2009, which was a 13% decrease compared with 2008 (n=699).

Of the 609 AEFI records in 2009, 552 records included at least one of the 10 vaccines for which ACIR data could be used to estimate AEFI reporting rates per 100,000 administered doses (Table 8). Vaccines for which reliable denominator data were not available included pH1N1 (n=23), seasonal

Table 7: Reporting rate of adverse events following immunisation (AEFI) per 100,000 doses of seasonal influenza and pH1N1 influenza vaccine,* 18 years and over, ADRS database, 2009

AEFI category†	Age group	AEFI records‡		Vaccine doses*	Rate per 100,000 doses§					
					2009		2008		2007	
		All	Serious	n	All	Serious	All	Serious	All	Serious
Seasonal Influenza	≥18 years	135	27	4,746,900	2.8	0.6	2.7	0.2	2.3	0.3
	18–64 years	101	15	2,626,400	3.8	0.6	3.4	0.2	3.0	0.4
	≥65 years	34	12	2,120,500	1.6	0.6	1.7	0.2	1.4	0.1
pH1N1 influenza vaccine	≥18 years	1,209	49	3,533,800	34.2	1.4	na		na	
	18–64 years	846	26	2,238,100	37.8	1.2	na		na	
	≥65 years	363	23	1,295,700	28.0	1.8	na		na	

* Number of administered doses of seasonal influenza vaccine estimated from the 2006 Australian Institute of Health and Welfare national survey (unpublished) and Number of administered doses of pH1N1 influenza vaccine estimated from the 2010 AIHW Pandemic Vaccination survey (published in August 2010 – Cat. No. PHE 128).

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

‡ Number of AEFI records in which vaccine was 'suspected' and the vaccination was administered in 2009.

§ The estimated reporting rate of adverse events per 100,000 administered doses of respective vaccines.

influenza (n=17), hepatitis B (n=10), BCG (n=6), hepatitis A (n=4), and 23vPPV (n=2) (Table 4). The overall reporting rate for the 10 NIP vaccines was 14.1 per 100,000 administered doses, while the reporting rate for serious AEFI was 1.8 per 100,000 doses (Table 8).

AEFI reporting rates across jurisdictions were consistently similar to, or lower than, those for the same period in 2008 for most age groups, reaction categories and vaccines (Table 8). The largest declines were for varicella (43%; reporting rates 8.3 per 100,000 doses in 2009 compared with 14.9 in 2008) and DTPa-IPV (34%; 72.1 vs 92.1). Reporting rates also declined for rotavirus (12%; 38.2 vs 43.1) and MMR (8%; 34.0 vs 38.5).

The AEFI reporting rates for pentavalent DTPa-IPV-HepB and Hib-HepB vaccines are less reliable due to the small number of reports.

New pandemic pH1N1 2009 influenza vaccine

There were a total of 1,312 AEFI reports received for 2009 where pH1N1 influenza vaccine was listed as a suspected vaccine (Table 4). It was the only suspected vaccine in 1,287 (98%) reports, 46 (4%) had causality ratings of 'certain' or 'probable' and 56 (4%) were defined as 'serious' (Table 4). Five deaths were recorded as temporally associated with receipt of pH1N1 influenza vaccine (described earlier in this report). Twenty-five per cent of reports (n=332) came from Queensland, 23% (n=305) from New South Wales, 20% (n=264) from Victoria, 13% (n=171) from South

Table 8: Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, ADRS database, 2009

	AEFI records†	Vaccine doses*	Reporting rate per 100,000 doses‡		
	n	n	2009	2008	2007
Vaccine					
DTPa-containing vaccines	420	1,122,430	37.4	46.3	33.0
DTPa-IPV	213	295,237	72.1	92.1	45.4
Pentavalent (DTPa-IPV-HepB)	3	10,566	28.4	22.5	43.7
Hexavalent (DTPa-IPV-HepB-Hib)	204	816,627	25.0	25.0	10.7
Haemophilus influenzae type b	45	276,878	16.3	19.4	18.3
Haemophilus influenzae type b-hepatitis B	9	5,500	163.6	39.6	30.8
Measles-mumps-rubella	197	579,066	34.0	38.5	23.3
Meningococcal C conjugate	48	292,754	16.4	17.5	12.2
Pneumococcal conjugate	210	826,947	25.4	27.0	20.6
Rotavirus vaccine	199	521,181	38.2	43.1	40.2
Varicella	23	277,496	8.3	14.9	10.9
Age group					
<1 year	249	2,217,680	11.2	13.0	9.7
1 to <2 years	71	1,035,641	6.9	8.2	6.5
2 to <7 years	232	648,931	35.8	52.9	38.5
AEFI category§					
Total	552	3,902,252	14.1	17.8	13.3
'Certain' or 'probable' causality rating	80	3,902,252	2.1	4.9	4.2
'Serious' outcome	69	3,902,252	1.8	2.3	1.6

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 31 December 2009.

† Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 31 December 2009. More than 1 vaccine may be coded as 'suspected' if several were administered at the same time.

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

§ Records where at least one of the vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[13] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.[13]

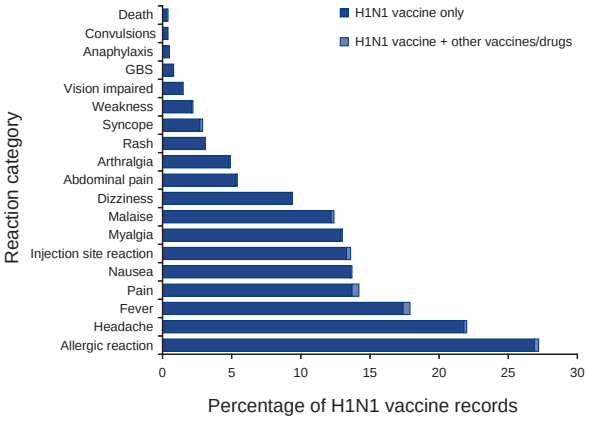
Australia, 8% (n=116) from Western Australia, 3% each from the Australian Capital Territory (n=37) and Tasmania (n=33) and 0.5% (n=7) from the Northern Territory.

The AEFI reporting rate for people aged ≥18 years was 34.2 per 100,000 administered doses (Table 7). The overall AEFI reporting rates were higher for vaccinees aged 18–64 years than among older people. However, the reporting rate of serious AEFI was highest (1.8 per 100,000 doses) among older people (aged ≥65 years).

The majority of the AEFI (48%; n=627) were reported by members of the public, 22% (n=290) by states and territories, 15% (n=191) by GPs, 9% (n=118) by nurses, 2% each by hospitals (n=30) and pharmacists (n=27), 1% (n=12) by drug companies, and 1.2% (n=17) by specialists.

The most frequently reported categories of reactions associated with administration of pH1N1 influenza vaccine are shown in Figure 5. They included non-anaphylactic allergic reactions (27%; n=357); headache (22%); fever (18%); ISR, pain and nausea (14% each); myalgia (13%); malaise (12%); and dizziness (9%). There were a total of 7 reports of anaphylactic reaction and 5 reports of convulsion (including 2 febrile convulsions; aged 1 and 4 years). Both the febrile convulsion cases were following only pH1N1 influenza vaccine and symptoms appeared within 12 hours post vaccination. All the anaphylactic reactions occurred immediately after pH1N1 administration. Among the 7 records of anaphylaxis, two were reported to have a history of asthma and one had known allergies to eggs. There were 10 cases reported as GBS following pH1N1 influenza vaccination.

Figure 5: Most frequently reported adverse events following pH1N1 immunisation,* ADRS database, 2009



* Percentage of 1,312 AEFI records where pH1N1 vaccine was listed as suspected of involvement in the reported AEFI.

Discussion

The majority of AEFI reported to the TGA in 2009 were mild, transient and well recognised vaccine side-effects. There was, however, a large increase (55%) in the number of AEFI reports received for 2009 compared with 2008, mainly related to the commencement of the pH1N1 immunisation program in September 2009, which contributed 54% of the total AEFI reports for 2009. Of particular note was the large increase in reports from members of the public direct to the TGA, from 3% of the total in 2008 to 28% in 2009, 94% of which were for pH1N1 influenza vaccine. The reporting rate for pH1N1 was 34.2 per 100,000 doses administered in persons aged ≥18 years, higher than that for seasonal influenza vaccine (2.8). Rates for those aged ≥65 years were 28.0, 1.6 and 13.3 for pH1N1, seasonal influenza and polysaccharide pneumococcal vaccines respectively. The high AEFI reporting rate for pH1N1, including high rates from members of the public, are likely due at least in part to the fact that the H1N1 influenza vaccination program used strategies to encourage consumers and health professionals to report adverse events to allow TGA to closely monitor the safety of the vaccine,[34] as well as the known effect of enhanced reporting for new vaccines.

The safety of the pH1N1 influenza vaccine has been examined closely both internationally and in Australia. The World Health Organization reported that approximately 30 different pH1N1 influenza vaccines have been developed using a range of methods.[35] All progressed successfully through vaccine trials to licensure, showing satisfactory safety profiles. However, these clinical trials were not powered to detect rare adverse vaccine reactions that occur with a frequency of less than one in 1,000, emphasising the need for post-licensure surveillance. In general, the safety profile, including that for the Australian vaccine, has been similar to those of seasonal influenza vaccines, with predominantly mild transient events and a small number of serious reactions reported.[36]

The data presented here for pH1N1 influenza vaccine in 2009 include very few AEFI in children, as the pH1N1 vaccine was licensed for children only in December 2009. The majority of the 1,132 reports were mild vaccine side-effects similar to that identified in pre-licensure clinical trials.[36] These included mainly non-anaphylactic allergic reactions, fever and injection site reactions. A range of mild non-specific symptoms including headache, nausea, dizziness, malaise and weakness were also commonly reported (Tables 5 and 6; Figure 5). This constellation of symptoms

is known to be associated with any new event of vaccination rather than any specific vaccine; data presented here are consistent with this experience. While GBS was associated with a previous swine influenza vaccine in 1976,[37] international assessment of the current pH1N1 vaccines has found either no association,[34] or a slightly higher rate of GBS in vaccinees up to one per million vaccine doses, which is consistent with estimates for seasonal influenza vaccine.[38] Initial national analysis by the TGA has shown no indication of an increased rate of GBS, or anaphylaxis, another serious reaction of concern, associated with pH1N1 influenza vaccine in Australia.[39] None of the 5 deaths reported following receipt of pH1N1 influenza vaccine were regarded as likely to be causally associated with the vaccine.

After excluding reports for pH1N1, there was a 30% reduction in the number of AEFI reported to the TGA in 2009 compared with 2008 (Table 1). The majority of these (68%) were reported by states and territories and only 3% were reported by members of the public. Decreases were seen in all jurisdictions and in all age groups. The decreases were greater in adolescents, associated with the tapering off of the HPV catch-up campaign and possibly reduced reporting associated with greater familiarity with that vaccine. Decreases among children aged <7 years are likely to be a combination of a stable vaccination schedule during 2008 and 2009, and reporting delay, which usually results in an underestimation of reports in the latest year of approximately 5%.

Conclusion

There was a substantial increase in AEFI reported in 2009 associated with the introduction of the new pH1N1 influenza vaccine in September. A large number of reports were received from members of the public. However, the majority of AEFI reports were of mild, transient and well-recognised vaccine side-effects.

The regular analysis and publication of national AEFI surveillance data collated in the ADRAC database remains an important aspect of Australia's immunisation program.

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References

1. Chen RT, DeStefano F, Pless R, Mootrey G, Kramarz P, Hibbs B. Challenges and controversies in immunization safety. *Infect Dis Clin North Am* 2001;15(1):21–39.

2. Duclos P. A global perspective on vaccine safety. *Vaccine* 2004;2215–16):2059–2063.

3. Immunisation Section, Communicable Disease Control Branch, South Australian Department of Health. New vaccine schedule from 1st March 2008. *Sharp and to the point, Quarterly newsletter* 2008;23:1. Accessed on 30 August 2010. Available from: http://www.dh.sa.gov.au/pehs/Immunisation/0803-sharp-point-news.pdf

4. Queensland Health. National Immunisation Program Queensland schedule commencing 1 March 2008. Accessed on 1 September 2010. Available from: http://www.health.sa.gov.au/immunisationcalculator/QldMar2008.pdf

5. Immunisation Program, Victorian Department of Human Services. Changed immunisation schedule from 1 March 2008. *Immunisation newsletter* Issue 32, January 2008. 2008;23:2. Accessed on 1 September 2010. Available from: http://www.health.vic.gov.au/__data/assets/pdf_file/0015/130083/Immunisation-newsletter-issue-32.pdf

6. Centers for Disease Control and Prevention. Continued shortage of *Haemophilus influenzae* type b (Hib) conjugate vaccines and potential implications for Hib surveillance—United States, 2008. *MMWR Morb Mortal Wkly Rep* 2008;57(46):1252–1255.

7. Australian Government Department of Health and Ageing. Timeline of mass immunisation programs and initiatives. 2009. Accessed on 30 August 2010. Available from: http://immunise.health.gov.au/internet/immunise/publishing.nsf/Content/past-immu-prog

8. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 8th edn. Canberra: Australian Government Department of Health and Ageing, 2003.

9. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 9th edn. Canberra: Australian Government Department of Health and Ageing, 2008.

10. Mahajan D, Menzies R, Roomiani I, Lawrence GL. Supplementary report: surveillance of adverse events following immunisation among children aged < 7 years in Australia, 1 January to 30 June 2009. *Commun Dis Intell* 2010;34(1):49–53.

11. Menzies R, Mahajan D, Gold MS, Roomiani I, McIntyre P, Lawrence G. Annual report: surveillance of adverse events following immunisation in Australia, 2008. *Commun Dis Intell* 2009;33(4):365–381.

12. Lawrence GL, Mahajan D, Roomiani I. Supplementary report: surveillance of adverse events following immunisation among children aged less than 7 years in Australia, 1 January to 30 June 2008. *Commun Dis Intell* 2009;33(1):27–31.

13. Lawrence G, Menzies R, Burgess M, McIntyre P, Wood N, Boyd I, et al. Surveillance of adverse events following immunisation: Australia, 2000–2002. *Commun Dis Intell* 2003;27(3):307–323.

14. Lawrence G, Boyd I, McIntyre P, Isaacs D. Surveillance of adverse events following immunisation: Australia 2002 to 2003. *Commun Dis Intell* 2004;28(3):324–338.

15. Lawrence G, Boyd I. Surveillance of adverse events following immunisation for children aged less than 7 years, 1 January to 30 June 2004. *Commun Dis Intell* 2004;28(4):490–492.

16. Lawrence G, Boyd I. Supplementary report: surveillance of adverse events following immunisation among children aged less than 7 years in Australia, 1 January to 30 June 2005. *Commun Dis Intell* 2005;29(4):413–416.

17. Lawrence G, Boyd I, McIntyre P, Isaacs D. Annual report: surveillance of adverse events following immunisation in Australia, 2005. *Commun Dis Intell* 2006;30(3):319–333.

18. Lawrence G, Boyd I. Supplementary report: surveillance of adverse events following immunisation among children aged <7 years in Australia, 1 January to 30 June 2006. *Commun Dis Intell* 2006;30(4):438–442.

19. Lawrence G, Gold MS, Hill R, Deeks S, Glasswell A, McIntyre PB. Annual report: surveillance of adverse events following immunisation in Australia, 2007. *Commun Dis Intell* 2008;32(4):371–387.

20. Lawrence GL, Boyd I, McIntyre PB, Isaacs D. Annual report: surveillance of adverse events following immunisation in Australia, 2004. Erratum in *Commun Dis Intell* 2005;29(4):416. *Commun Dis Intell* 2005;29(3):248–262.

21. Lawrence GL, Aratchige PE, Boyd I, McIntyre PB, Gold MS. Annual report on surveillance of adverse events following immunisation in Australia, 2006. *Commun Dis Intell* 2007;31(3):269–282.

22. Lawrence GL, Aratchige PE, Hill R. Supplementary report: surveillance of adverse events following immunisation among children aged less than 7 years in Australia, 1 January to 30 June 2007. *Commun Dis Intell* 2007;31(4):379–382.

23. Uppsala Monitoring Centre. WHO Collaborating Centre for International Drug Monitoring. Accessed on 30 August 2010. Available from: http://www.who-umc.org/

24. Zhou W, Pool V, Iskander JK, English-Bullard R, Ball R, Wise RP, et al. Surveillance for safety after immunization: Vaccine Adverse Event Reporting System (VAERS)—United States, 1991–2001. Erratum in *MMWR Morb Mortal Wkly Rep* 2003;52(06):113. *MMWR Surveill Summ* 2003;52(1):1–24.

25. Brown EG, Wood L, Wood S. The medical dictionary for regulatory activities (MedDRA). *Drug Saf* 1999;20(2):109–117.

26. Bonhoeffer J, Gold MS, Heijbel H, Vermeer P, Blumberg D, Braun M, et al. Hypotonic-hyporesponsive episode (HHE) as an adverse event following immunization: case definition and guidelines for data collection, analysis, and presentation. *Vaccine* 2004;22(5–6):563–568.

27. Ruggeberg JU, Gold MS, Bayas JM, Blum MD, Bonhoeffer J, Friedlander S, et al. Anaphylaxis: case definition and guidelines for data collection, analysis, and presentation of immunization safety data. *Vaccine* 2007;25(31):5675–5684.

28. The SAS system for Windows (computer program). Version 9.1.3. Cary, N.C.: SAS Institute Inc, 2005.

29. Australian Institute of Health and Welfare 2010. 2010 Pandemic Vaccination Survey: summary results. Cat no PHE 128 Canberra:AIHW 2010;

30. Australian Government Department of Health and Ageing. Communicable diseases surveillance: Additional reports – Childhood immunisation coverage. *Commun Dis Intell* 2008;32(2):288–289.

31. Varricchio F, Iskander J, DeStefano F, Ball R, Pless R, Braun MM, et al. Understanding vaccine safety information from the Vaccine Adverse Event Reporting System. *Pediatr Infect Dis J* 2004;23(4):287–294.

32. Joura EA, Leodolter S, Hernandez-Avila M, Wheeler CM, Perez G, Koutsky LA, et al. Efficacy of a quadrivalent prophylactic human papillomavirus (types 6, 11, 16, and 18) L1 virus-like-particle vaccine against high-grade vulval and vaginal lesions: a combined analysis of three randomised clinical trials. *Lancet* 2007;369(9574):1693–1702.

33. Reisinger KS, Block SL, Lazcano-Ponce E, Samakoses R, Esser MT, Erick J, et al. Safety and persistent immunogenicity of a quadrivalent human papillomavirus types 6, 11, 16, 18 L1 virus-like particle vaccine in preadolescents and adolescents: a randomized controlled trial. *Pediatr Infect Dis J* 2007;2(3)6:201–209.

34. Australian Government Department of Health and Ageing, Therapeutic Goods Administration, Cook J. Medicines Safety Update No.4, 2010. Australian experience with non-adjuvant H1N1 vaccine (Panvax and Panvax Junior). Accessed on 30 August 2010. Available from: http://www.tga.gov.au/adr/msu/msu1008.htm

35. World Health Organization. Safety of pandemic A (H1N1) influenza vaccines. *Wkly Epidemiol Rec* 2010;85(5):29–36.

36. Nolan T, McVernon J, Skeljo M, Richmond P, Wadia U, Lambert S. Immunogenicity of a monovalent 2009 influenza A(H1N1) vaccine in infants and children: a randomized trial. *JAMA* 2010;303(1):37–46.

37. Schonberger LB, Bregman DJ, Sullivan-Bolyai JZ, Keenlyside RA, Ziegler DW, Retailliau HF, et al. Guillain-Barré syndrome following vaccination in the National Influenza Immunization Program, United States, 1976–1977. *Am J Epidemiol* 1979;110(2):105–123.

38. Centers for Disease Control and Prevention. Preliminary results: surveillance for Guillain-Barré syndrome after receipt of influenza A (H1N1) 2009 monovalent vaccine – United States, 2009–2010. *MMWR Morb Mortal Wkly Rep* 2010;59(21):657–661.

39. Australian Government Department of Health and Ageing, Therapeutic Goods Administration. Suspected adverse reactions to Panvax® reported to the TGA 30 September 2009 – 30 April 2010. Accessed on 30 August 2010. Available from: http://www.tga.gov.au/alerts/medicines/h1n1vaccine1.htm

Abbreviations of vaccine types

7vPCV	7-valent pneumococcal conjugate vaccine
10vPCV	10-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
BCG	Bacille Calmette-Guérin (i.e. tuberculosis)
dT	diphtheria-tetanus – adolescent and adult formulation
DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
dTpa	diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation
dTpa-IPV	combined dTpa and inactivated poliovirus
DTPa-HepB	combined diphtheria-tetanus-pertussis (acellular) and hepatitis B
DTPa-IPV	combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus (quadrivalent)
DTPa-IPV-HepB	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus and hepatitis B (pentavalent)
DTPa-IPV-HepB-Hib	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus, hepatitis B and *Haemophilus influenzae* type b vaccine (hexavalent)
HepB	hepatitis B
Hib	*Haemophilus influenzae* type b
Hib-HepB	combined *Haemophilus influenzae* type b and hepatitis B
HPV	human papillomavirus
IPV	inactivated poliovirus vaccine
Men4PV	meningococcal polysaccharide tetravalent vaccine
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
pH1N1	pandemic H1N1 influenza 2009

Annual report of the Australian National Poliovirus Reference Laboratory, 2009

Jason A Roberts, Linda Hobday, Sophie Polychronopoulos, Aishah Ibrahim, Bruce R Thorley

Abstract

The Australian National Poliovirus Reference Laboratory (NPRL) is accredited by the World Health Organization (WHO) for the testing of faecal specimens from acute flaccid paralysis (AFP) cases and operates as a regional poliovirus reference laboratory for the Western Pacific Region. The NPRL, in collaboration with the Australian Paediatric Surveillance Unit, co-ordinates surveillance for cases of AFP in children in Australia, according to criteria recommended by the WHO. Specimens are referred from AFP cases in children and suspected cases of poliomyelitis in persons of any age. The WHO AFP surveillance performance indicator is 1 non-polio AFP case per 100,000 children less than 15 years of age. In 2009, the Polio Expert Committee classified 48 cases as non-polio AFP, a rate of 1.17 cases per 100,000 children less than 15 years of age. An additional WHO AFP surveillance performance indicator is that more than 80% of notified AFP cases have 2 faecal samples collected 24 hours apart and within 14 days of onset of paralysis. Adequate faecal samples were received from 16 (33.3%) of the 48 classified cases. A poliovirus was referred via the Enterovirus Reference Laboratory Network of Australia from a non-AFP case and was determined to be Sabin-like. This case most likely represents an importation event, the source of which was not identified, as Australia ceased using Sabin oral polio vaccine in 2005. The last report of a wild poliovirus importation in Australia was from Pakistan in 2007. In 2009, 1,604 wild poliovirus cases were reported in 23 countries with Afghanistan, India, Nigeria and Pakistan remaining endemic for poliomyelitis. *Commun Dis Intell* 2010;34(3):277–284.

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus, poliomyelitis, eradication, vaccination

Introduction

The National Polio Reference Laboratory (NPRL) is responsible for the virological testing of faecal specimens from cases with a clinical suspicion of poliomyelitis. This includes cases of acute flaccid paralysis (AFP)—a major clinical presentation of poliomyelitis—in children less than 15 years of age and cases of suspected poliomyelitis in patients of any age. The World Health Organization (WHO) recommends that 2 faecal specimens be collected from cases of AFP for virological investigation at least 24 hours apart and within 14 days of the onset of paralysis. It is a requirement of the WHO polio eradication program that the specimens are tested in a WHO accredited laboratory, which for Australia is the NPRL at the Victorian Infectious Diseases Reference Laboratory (VIDRL). Laboratory testing may exclude poliovirus as the causative agent of AFP. Enteroviruses other than poliovirus have been associated with AFP.

From November 2005, inactivated poliomyelitis vaccine (IPV) replaced oral poliomyelitis vaccine (OPV) in the National Immunisation Program.[1] IPV is administered to children at 2, 4 and 6 months of age, with a booster dose at 4 years of age. With the removal of OPV, containing 'live' attenuated virus, from the immunisation schedule, any poliovirus identified by Australian virology laboratories requires further investigation to determine its origin, as it represents an importation event.

It is important that Australia maintains high levels of polio vaccine coverage to avoid a resurgence of poliomyelitis in the event of a wild poliovirus importation. Reinforcement of this recommendation is evidenced by the large type 1 wild poliovirus outbreak in Tajikistan in 2010, a country with reportedly similar polio vaccination coverage (87% in 2008) and AFP surveillance performance as Australia (non-polio AFP rate of 1.4 in 2009).[2] As of 12 July 2010 there have been 413 confirmed cases of poliomyelitis with 19 deaths in Tajikistan.[3] People travelling to polio endemic countries and countries with recent wild poliovirus importations should receive a booster polio vaccine prior to departure, or a full course of vaccination if they are unsure of their vaccination history. Individuals who are at continuing risk of infection,[4] such as health care workers, are recommended to have a booster polio vaccine every 10 years.[5] The WHO provides a searchable database of global case counts and surveillance data at http://apps.who.int/immunization_monitoring/en/diseases/poliomyelitis/case_count.cfm

The Australian NPRL is also the National Poliovirus Reference Laboratory for Brunei Darussalam, Papua New Guinea and the Pacific Island countries, and is a regional reference laboratory for the

WHO Western Pacific Region. Specimens and isolates are referred to the laboratory from national laboratories throughout the region in accordance with requirements determined by the WHO.

Methods

AFP surveillance was initiated by the Australian Government in 1995 in collaboration with the Australian Paediatric Surveillance Unit (APSU) as part of Australia's commitment to the WHO poliomyelitis eradication program. Since 2000, AFP surveillance has been co-ordinated by VIDRL in collaboration with the APSU.

The strategy adopted for AFP surveillance is as follows:

- Paediatricians reviewing a patient less than 15 years of age and presenting with AFP, or a clinician reviewing a patient of any age suspected of poliomyelitis, are requested to notify the NPRL (telephone 03-9342 2607, email polio@mh.org.au). Notification of the case is also included on the paediatrician's monthly report card to the APSU (http://www.apsu.org.au/).
- Two faecal specimens should be collected 24 to 48 hours apart and within 14 days of onset of paralysis. Collection of specimens within these time frames will enable them to be classified as adequate by WHO.
- The faecal specimens are referred free of charge for testing by the NPRL, which is accredited by WHO for this purpose.
- Upon notification of an AFP case, clinicians are forwarded a clinical questionnaire for completion.
- The Polio Expert Committee (PEC), convened by the Department of Health and Ageing (DoHA), reviews the clinical and laboratory data for all notified cases of AFP, irrespective of whether they are an eligible or ineligible case. An eligible case is: an Australian child under 15 years of age with AFP (including Guillain-Barré syndrome) or an Australian of any age with paralytic illness if polio is suspected. Examples of ineligible cases are if the patient is aged 15 years or older, an overseas resident and cases notified in error or later determined to be non-AFP.
- The PEC classifies cases of AFP as:
  - poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated paralytic poliomyelitis (VAPP);
  - non-polio AFP or;
  - non-AFP.
- A follow-up questionnaire is sent to notifying clinicians if the PEC requires more information regarding the AFP case before a final classification can be made.
- After each PEC meeting the Australian AFP data are forwarded to WHO for inclusion in the global AFP surveillance data published in the *Weekly Epidemiological Record* (available from http://www.who.int/wer/en/). Ineligible cases are not reported to WHO.
- The WHO AFP surveillance performance indicator for a polio non-endemic country is 1 non-polio AFP case per 100,000 children aged less than 15 years. For Australia in 2009, this equated to 41 cases per year, based on the Australian Bureau of Statistics data released in December 2008. An AFP surveillance scheme that satisfies the surveillance performance indicator is deemed sufficiently sensitive to detect a wild poliovirus importation in children of that country.
- The WHO surveillance performance indicator for laboratory testing is that at least 80% of notified AFP cases have adequate faecal specimens collected and tested in a WHO accredited laboratory.
- At the end of each calendar year, a number of AFP notifications remained unclassified as insufficient clinical and laboratory data were available to enable the PEC to review the cases. In 2008, after consulting with WHO, the PEC resolved to classify pending cases as 'polio compatible–zero evidence'.

Upon receipt at the NPRL, faecal specimens are treated with Minimum Essential Medium containing Hank's salts, chloroform (9.1% v/v) and foetal bovine serum (2%). The suspension is clarified and the supernatant inoculated onto a series of mammalian cell lines. Two WHO recommended cell lines are used for the isolation of poliovirus; L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155) and RD-A (human rhabdomyosarcoma).[6,7] The NPRL utilises 2 additional cell lines for the isolation of poliovirus and non-polio enteroviruses: BGMK (buffalo green monkey kidney) and HEL (human embryonic lung). Diagnostic laboratories in Australia are encouraged to refer enteroviruses of unknown serotype to the NPRL for further characterisation as poliovirus infection can lead to clinical presentations without paralysis such as aseptic meningitis.

A series of tests known as intratypic differentiation (ITD) are performed on poliovirus isolates to determine whether the virus is a wild poliovirus strain, OPV strain (Sabin-like) or a VDPV. In 2009, the WHO introduced diagnostic poliovirus

real time reverse transcriptase polymerase chain reaction (rRT-PCR), developed by the US Centers for Disease Control and Prevention (CDC), Atlanta, as the primary ITD method.[8] The Australian NPRL sequences the complete poliovirus VP1 genomic region, which contains a major neutralising antibody binding site. The VP1 genomic sequence provides valuable biological information, including the number of mutations within a significant region of the OPV virus strain and it enables phylogenetic analysis of wild poliovirus to rapidly determine the likely source of the virus, as utilised in the 2007 importation.[9]

Results

Notification of acute flaccid paralysis cases

A total of 61 notifications of AFP cases were received in 2009 (Table 1). Two AFP cases were already notified by other clinicians and so were regarded as duplicate notifications.

(i) Eligible AFP cases

Sufficient information was available for the PEC to review 48 cases of AFP involving children less than 15 years of age with onset of paralysis in 2009. All cases were classified as non-polio AFP by the PEC. The 48 cases equates to a non-polio AFP rate of 1.2 cases per 100,000 children less than 15 years of age. This result meets the WHO AFP surveillance performance criterion for a polio-free country of 1 case of non-polio AFP per 100,000 children less than 15 years of age (Figure).

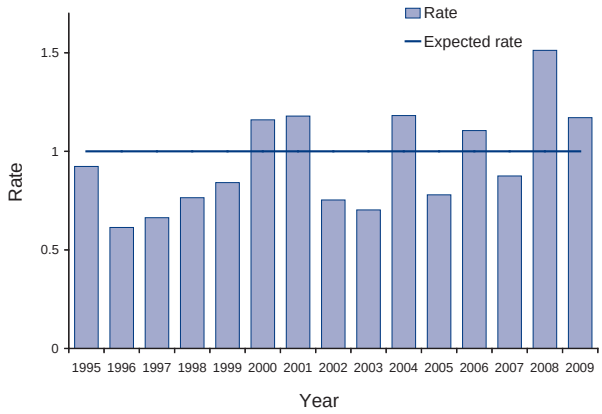
(ii) Ineligible cases

Eight cases did not meet the criteria for an eligible case. Four were later reported as non-AFP and the other four involved patients aged over 14 years; one with onset of paralysis in 2008. The cases involving patients over 14 years of age were all classified by the PEC as non-polio AFP but were not reported to the WHO since the organisation focuses on the onset of AFP in children less than 15 years of age.

Notification of acute flaccid paralysis cases by state and territory

In 2009, AFP cases were notified from all jurisdictions in Australia except for the Northern Territory (Table 2). After excluding duplicate notifications and ineligible cases, the non-polio AFP rates per

Figure: Non-polio acute flaccid paralysis rate after final classification of cases by the Polio Expert Committee



WHO AFP Surveillance Performance Indicator = 1 non-polio AFP case per 100,000 population <15 years

Table 1: Surveillance for acute flaccid paralysis (AFP) cases in children less than 15 years of age, Australia, 2009, compared with the World Health Organization (WHO) AFP surveillance performance indicators

WHO surveillance performance indicator for AFP cases in children less than 15 years*	Australia's surveillance for AFP cases in children with onset of paralysis in 2009	Australia's AFP surveillance performance in 2009
Non-polio AFP case rate of 1.0 per 100,000 children (41 cases for Australia in 2009).	60 unique cases of AFP notified 48 cases classified by the Polio Expert Committee as non-polio AFP	AFP notification rate: 1.46 per 100,000 children. Non-polio AFP case rate: 1.17 per 100,000 children.
More than 80% of classified AFP cases with 2 adequate faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis.	16 AFP cases with 2 or more adequate specimens	Referral of adequate specimens from AFP cases: 33.3% (16 of 48) of the eligible cases.

* Based on data supplied by the Australian Bureau of Statistics, estimated population, preliminary – 30 June 2008. ABS publication 3201.0.

jurisdiction exceeded the AFP surveillance performance indicator of 1 case per 100,000 children in New South Wales, Victoria, Western Australia and Tasmania. The surveillance performance indicator was not achieved in Queensland, South Australia, the Australian Capital Territory or the Northern Territory (Table 2).

Faecal collection from acute flaccid paralysis cases

WHO defines adequate specimens for poliovirus culture as 2 faecal specimens collected at least 24 hours apart and within 14 days of the onset of paralysis. A further surveillance criterion set by WHO is for adequate faecal collection from 80% of the eligible AFP cases.

In 2009, a total of 47 faecal specimens from 29 of the 48 eligible cases were tested at the NPRL (Table 1):

- 16 (33%) of the eligible cases had adequate specimens with 2 specimens collected within 14 days of symptom onset;
- 11 (23%) cases had 1 specimen collected within 14 days of onset; two of the cases had a 2nd specimen collected after 14 days;
- 2 (4%) cases had 1 faecal specimen collected more than 14 days after onset;
- no faecal specimens were received from the remaining 19 (40%) eligible cases.

The 33% (16 of 48 cases) proportion of eligible cases with adequate faecal specimen collection compares with the WHO criterion of 80%. Queensland was the only jurisdiction to reach the WHO performance indicator within the reporting period (Table 2).

Laboratory testing of specimens

Acute flaccid paralysis cases

Between 1 January and 31 December 2009, a total of 54 specimens were referred from 31 cases of AFP involving patients aged less than 15 years (Table 3). No poliovirus was isolated from any of these specimens.

A non-polio enterovirus (NPEV) was isolated from a faecal specimen of 1 patient with onset of paralysis in Victoria in February 2009. Ribonucleic acid (RNA) was extracted from the virus isolate and a fragment of the VP1 genomic region

Table 2: Notification of acute flaccid paralysis (AFP) cases, Australia, 2009, by state or territory

State or territory	Estimated population aged <15 years*	Expected number of AFP cases in 2009	Total number of notifications	Ineligible notifications	Duplicate notifications	Cases with insufficient information to classify	Eligible cases with final classification by PEC	Notification rate per 100,000 children†	Non-polio AFP rate per 100,000 children
ACT	63,874	1	1	0	0	1	0	1.0	0.0
NSW	1,332,066	13	20	2	1	0	17	1.5	1.3
NT	52,253	1	0	0	0	0	0	0.0	0.0
Qld	861,002	8	8	1	3	1	3	1.0	0.4
SA	289,309	3	2	1	0	0	1	0.7	0.3
Tas	97,012	1	1	0	0	0	1	1.0	1.0
Vic	995,096	10	25	3	0	0	22	2.5	2.2
WA	426,476	4	4	0	0	0	4	1.0	1.0
Aust	4,117,612	41	61	7	4	2	48	1.5	1.2

* Australian Bureau of Statistics, estimated population, preliminary – 30 June 2008. Australian Bureau of Statistics publication 3201.0.

† Excludes duplicate notifications and ineligible cases.

PEC Polio Expert Committee

Table 3: Test results of specimens referred to the Australian National Polio Reference Laboratory, from within Australia, 2009

Result	Specimens from AFP cases involving children < 15 years of age	Specimens from AFP cases involving patients ≥15 years of age	Specimens from sources other than AFP	Total
Sabin poliovirus type 1	0	0	1	1
Non-polio enterovirus	2	0	61	63
Rhinovirus	0	0	5	5
No enterovirus identified	52	4	17	73
Total	54	4	84	142

AFP Acute flaccid paralysis.

amplified by RT-PCR. The virus was identified by nucleotide sequence alignment and phylogenetic analysis as Coxsackievirus B3. A second NPEV was identified from a faecal specimen of an AFP case from a patient with onset of paralysis in New South Wales in June 2009. The NPEV was identified as Coxsackievirus A4. Both cases had only 1 specimen referred for virus culture.

No enterovirus was isolated from the remaining 52 specimens.

Fifty of the total specimens received were from 29 cases with onset of AFP in 2009. Sufficient clinical information was available for all the AFP cases with specimens referred to be classified by the PEC as non-polio AFP. Four specimens were received in January 2009 from 2 AFP cases with onset of symptoms in December 2008; 1 specimen from 1 case and 3 specimens from the other.

Two specimens each were received from 2 cases involving patients aged 15 years or over, which was outside of the WHO AFP surveillance criterion. No enterovirus was isolated from the 4 specimens.

Sources other than acute flaccid paralysis

Echovirus 30 was identified from a non-AFP case aged over 14 years. An additional 4 specimens were referred from non-AFP cases, all of which were found to be enterovirus negative.

The Enterovirus Reference Laboratory Network (ERLNA) was established through seed funding from the Department of Health and Ageing for extended polio surveillance. A total of 89 samples (specimen extracts and nucleic acid extracts) were referred through the network (Table 4). The serotypes of 60 enteroviruses were identified by sequencing a fragment of the VP1 genomic region using 'CODEHOP' methodology.[10]

In October 2009, an uncharacterised poliovirus was referred through the ERLNA. A diagnostic virology laboratory in Victoria isolated the poliovirus from a faecal specimen of an unimmunised 1-month-old infant admitted for a respiratory infection with no indication of AFP. The virus was initially identified as an enterovirus by cytopathic effect in culture and subsequently as a poliovirus by immunofluorescence. The virus was identified as Sabin-like by the WHO diagnostic rRT-PCR ITD tests and the nucleotide sequence for the VP1 genomic region showed 905/906 (99.9%) nucleotide sequence identity to Sabin 1 prototype. A second specimen from the infant was requested to determine if virus shedding was prolonged but was not received. The source of the Sabin virus remains unknown.

Polio serology

Poliovirus serology is only performed for cases with a clinical suspicion of acute poliovirus infection. Nineteen requests for polio serology were received by the laboratory in the reporting period. All tests were cancelled after discussion with the referring doctor, as the requests were made to determine the patient's immune status for work or travel purposes.

Regional reference laboratory activities

In addition to the Australian samples, 206 specimens and virus isolates were received from various countries of the Western Pacific Region in 2009.

- Twenty-six faecal specimens from 14 AFP cases were referred from Pacific Island countries. Seven NPEVs were isolated from the specimens.

Table 4: Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, referred from within Australia, 1995 to 2009

Year	Poliovirus		Non-polio enterovirus	No enterovirus detected	Total samples tested
	Sabin-like	Non-Sabin-like			
1995	190	0	200	13	403
1996	224	0	198	9	431
1997	124	0	76	0	200
1998	52	0	15	4	71
1999*	60	1	9	9	79
2000	45	0	44	47	136
2001*	46	5	33	75	159
2002	36	0	21	49	106
2003	9	0	15	47	71
2004	6	0	26	61	93
2005	18	0	10	39	67
2006	2	0	6	71	79
2007†	0	2	32	115	149
2008	0	0	20	92	112
2009‡	1	0	63	78	142

* Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory. The six isolates tested as non-Sabin-like and were subsequently identified as wild type poliovirus prototype strains and were destroyed.

† Wild poliovirus type 1 was imported from Pakistan.

‡ Includes samples received for testing via the Enterovirus Reference Laboratory Network of Australia.

- Fifty-four faecal specimens from 33 cases of AFP were referred from Papua New Guinea. Eleven NPEVs were isolated from the specimens.
- Thirty-eight specimens and isolates were received from Malaysia including 18 polioviruses referred for intratypic differentiation.
- Eighty-eight specimens and isolates were received from the Philippines including 16 polioviruses referred for intratypic differentiation.

During 2009, The NPRL was invited to participate in the field evaluation of the poliovirus ITD rRT-PCR test kits by the CDC during the development and evaluation phases over an 18-month period. Subsequently the WHO requested the NPRL to host a regional training workshop on rRT-PCR techniques, in August 2009. The NPRL hosted facilitators from the CDC, WHO Headquarters, WHO Western Pacific Regional Office and an observer from the DoHA. The workshop participants were from Australia, China, Japan, Malaysia and Singapore.

Quality assurance program

The NPRL completed the WHO poliovirus ITD rRT-PCR proficiency panel in November 2009 and was the first polio reference laboratory to be fully accredited in the technique in the Western Pacific Region.

The NPRL also successfully completed the WHO poliovirus isolation and identification proficiency testing panel, which uses a revised testing algorithm introduced in endemic regions in 2006. The new algorithm is designed to shorten the time for issuing virus isolation reports from 28 days to 14 days and poliovirus ITD reports from 14 days to 7 days.

Discussion

Australia reported a non-polio AFP rate of 1.7 cases per 100,000 children less than 15 years of age in 2009. This exceeds the WHO AFP surveillance performance indicator of 1 non-polio AFP case per 100,000 children less than 15 years of age, which is an international standard to assess the sensitivity of a national AFP surveillance program. Australia has reached the WHO AFP surveillance performance indicator in 5 other

years since the program was established in 1995: 2000, 2001, 2004, 2006 and 2008. It should be noted, however, that while the national AFP surveillance scheme targets an age group at high risk of poliovirus infection, persons of any age are a potential source of a wild poliovirus importation, as evidenced by the 2007 importation involving a 22-year-old student from Pakistan.[9]

Four states (New South Wales, Tasmania, Victoria and Western Australia) achieved the AFP surveillance performance indicator rate in 2009. Victoria reported 2.2 non-polio AFP cases per 100,000 children less than 15 years of age, one of the highest rates ever reported for any jurisdiction in Australia and a vast turnaround from the consistent under-reporting for many years by that state. The introduction of the Paediatric Active Enhanced Disease Surveillance (PAEDS) scheme at a sentinel hospital in Victoria was a decisive factor in this result, with 21 of the 22 cases notified via the PAEDS system.[11]

Queensland and the 2 territories, the Australian Capital Territory and the Northern Territory, did not reach the non-polio AFP indicator rate in 2009. Queensland is the only jurisdiction in Australia where AFP in children is a notifiable condition.

Despite the introduction of the PAEDS scheme in 2007, with hospital-based nurses to ascertain AFP cases and arrange for the referral of specimens to the NPRL, Australia has still never reached the WHO AFP surveillance performance indicator for the collection of adequate faecal specimens from 80% of AFP cases. One of the difficulties in achieving this target is the strict definition of adequate specimens: 2 faecal specimens collected more than 24 hours apart and within 14 days of the onset of paralysis. In 2009, 16 cases (33%) had adequate faecal collections, while a further 11 cases (23%) had only 1 adequate specimen collected, making a total of 56% of cases with at least 1 specimen collected within 14 days of the onset of symptoms.

No poliovirus was isolated from the specimens referred to the NPRL from AFP cases. A NPEV was isolated from each of the faecal specimens referred from 2 AFP cases: a coxsackievirus B3 was isolated from 1 case and a coxsackievirus A4 from the other. The establishment of the ERLNA by the NPRL provides another means of surveillance for poliovirus: virological surveillance rather than clinical surveillance for cases of AFP. The referral of an uncharacterised poliovirus from an unimmunised infant admitted to hospital with a respiratory infection through the ERLNA in October 2009, was a significant result underscoring the usefulness of the reference network. Laboratories interested in collaborating with the ERLNA are encouraged to contact the NPRL for details.

Australia, along with the other countries of the Western Pacific Region, was declared free of indigenous wild poliovirus in 2000 and ceased usage of the Sabin 'live', attenuated oral polio vaccine in November 2005. The virus from the infant was typed as a Sabin type 1 poliovirus with a single mutation in the VP1 genomic region. The 99.9% sequence identity of the isolated virus to Sabin 1 prototype sequence indicates the virus was likely to have originated from a recent immunisation event, since poliovirus accumulates ~1% nucleotide mutations per year of replication.[12] The source of the Sabin poliovirus was never established but is likely to have been an importation from a country using OPV. The result demonstrates the ongoing risk faced by Australia of poliovirus importation, both wild and vaccine strains, since more than 90% of poliovirus infections are asymptomatic.

The introduction of the WHO poliovirus diagnostic real time RT-PCR at the NPRL will enable faster characterisation of untyped polioviruses compared with the previous end-point RT-PCR methodology. The real time RT-PCR assay includes an additional protocol to screen for vaccine derived polioviruses; Sabin strains with greater than 1% mutations compared with prototype sequence that is indicative of long-term viral replication and potentially person-to-person transmission. The NPRL will continue to sequence the full VP1 genomic region of all polioviruses referred to the laboratory, no matter what their source, as an additional precaution to further characterise all polioviruses. Laboratories identifying poliovirus from any patient of any age within Australia are requested to immediately contact the NPRL to arrange further identification of the virus.

Globally in 2009, a total of 1,604 cases of AFP were reported with 1,256 cases occurring within endemic countries. A total of 348 cases of AFP were reported in 23 non-endemic countries with Chad, Sudan and Guinea reporting 64, 45 and 42 cases respectively. These cases were related to the importation of wild poliovirus from Nigeria, which reported 388 cases in 2009.[13] Significant progress has been made in Nigeria in 2010 with only 6 cases of AFP reported as of 18 June compared with 346 cases reported during the same period in 2009. The Tajikistan outbreak in 2010, with 413 reported cases of AFP and 19 deaths

due to wild poliovirus,[3] is a salient reminder of the need for continued surveillance and vaccine coverage.

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References

1. National Health and Medical Research Council. *The Australian Immunisation Handbook* 9th edn. Canberra, Australia: Australian Government Department of Health and Ageing; 2008; pp. 251–256.

2. United Nations Children's Fund. *Immunization Summary, a Statistical Reference Containing Data through 2008*. Geneva: United Nations Children's Fund and the World Health Organization, January 2010.

3. Martin, R. *Tajikistan polio outbreak and regional response*. WHO Epidemiological Brief; 19 July 2010. Available from: http://www.euro.who.int/__data/assets/pdf_file/0019/118342/EPI_TJK_Issue3.pdf

4. World Health Organization. International Travel and Health; Situation as at 1 January 2010. Geneva: World Health Organization; 2010.

5. Wood DJ, Hull B. L20B cells simplify culture of polioviruses from clinical samples. *J Med Virol* 1999,58(2):188–192.

6. World Health Organization. *Polio Laboratory Manual*. Department of Immunization, Vaccines and Biologicals 2004. WHO/IVB/04.10.

7. Kilpatrick DR, Yang CF, Ching K, Vincent A, Iber J, Campagnoli R, et al. Rapid group–, serotype–, and vaccine strain-specific identification of poliovirus isolates by real-time reverse transcription PCR using degenerate primers and probes containing deoxyinosine residues. *J Clin Microbiol* 2009;47(6):1939–1941.

8. Carnie JA, Lester R, Moran R, Brown L, Meagher J, Roberts JA, et al. Public health response to imported case of poliomyelitis, Australia. *Emerg Infect Dis* 2009 15(11): 1733–1737.

9. Nix WA, Oberste MS, Pallansch MA. Sensitive, seminested PCR amplification of VP1 sequences for direct identification of all enterovirus serotypes from original clinical specimens. *J Clin Microbiol* 2006;44(8):2698–2704.

10. Pym M, Adams J, Booy R, Buttery J, Elia S, Elliott E, et al. The development and trial of paediatric active enhanced disease surveillance (PAEDS): a new surveillance mechanism for Australia. *J Paed Child Health* 2008;44:A16.

11. Kew O, Sutter RW, de Gourville EM, Dowdle WR, Pallansch MA. Vaccine-derived polioviruses and the endgame strategy for global polio eradication. *Annu Rev Microbiol* 2005;59:587–635.

12. World Health Organization. Global Polio Eradication Initiative. Wild poliovirus weekly update. wild poliovirus list 2000–2010. Available from: http://www.polioeradication.org/casecount.asp

Australian Paediatric Surveillance Unit annual report, 2008 and 2009

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Background

National active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions is conducted by the Australian Paediatric Surveillance Unit (APSU). The study of communicable and vaccine-preventable diseases is supported in part by the Department of Health and Ageing through its communicable diseases program. In 2008 and 2009, APSU conducted national surveillance for the following infectious diseases or vaccine preventable conditions:

- acute flaccid paralysis (AFP): a major clinical presentation of poliomyelitis;[1]
- acute rheumatic fever (ARF): occurs due to group A streptococcal (GAS) infection and repeat GAS infections, if not treated, may lead to heart valve damage and rheumatic heart disease;[2]
- congenital cytomegalovirus infection: a leading cause of congenital abnormality in Australia;[3]
- congenital rubella: an extremely rare condition leading to birth defects;[4]
- perinatal exposure to HIV: the most frequently reported source of HIV infection in Australian children;[5]
- neonatal herpes simplex virus (HSV) infection: a very rare, but serious infection that may cause chorioretinitis, intracerebral calcification and birth defects;
- neonatal group B streptococcus (GBS) infection: the most common cause of life threatening infections in neonates;
- intussusception: the most common cause of bowel obstruction in infants and young children that has been associated with rotavirus infection and previous rotavirus vaccines;[6,7]
- congenital and neonatal varicella: a rare infection that may result in birth defects.[8]
- severe complications of varicella: a range of rare but serious complications; genotyping of samples will inform future vaccine and policy development;[8] and
- severe complications of influenza infection: complications such as pneumonia, encephalitis, myocarditis, rhabdomyolysis, disseminated coagulopathy, transverse myelitis, polyneuritis and Guillain-Barré syndrome have a significant burden among children aged less than 15 years.[9]

Methods

The APSU study protocols are developed with collaborating investigators and/or institutions that have expertise in each of the conditions studied. Detailed protocols including case definitions for each condition under surveillance and contact details of the expert investigators for each condition are available at www.apsu.org.au The APSU sends monthly report cards listing the conditions under surveillance to approximately 1,300 paediatricians and child health clinicians around Australia. Report cards are returned whether the clinician has a case to report or not, and the rate of returned report cards provides a measure of participation. In 2009 approximately 80% of clinicians chose to receive and respond to the APSU report card via e-mail. All reported cases are followed-up by a questionnaire requesting de-identified data on the child's clinical presentation, treatment and short-term outcome. Clinicians were asked to return all questionnaires by fax as soon as children who met criteria for severe complications of influenza were identified during 2008 surveillance for seasonal influenza and during 2009 surveillance for seasonal and pandemic influenza.

The APSU aims to provide epidemiological information that is representative of the Australian child population and maximal case ascertainment is a high priority. Despite a representative mailing list (92% of all paediatricians in active clinical practice in Australia participate in monthly surveillance) and high response rates (average 96% per annum since 2000), complete case ascertainment is unlikely.[10] This is particularly relevant in remote communities where children have limited access to paediatricians or when hospital admission is brief.

However, for most conditions studied by the APSU no alternative national data are available to allow an estimate of completeness of ascertainment. The APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximise case identification.[10,11] Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate of the incidence of these conditions in the relevant Australian child populations.

To further enhance surveillance for childhood conditions where hospital stays are minimal; where biological samples are required; and where a detailed history might be needed from parents or caregivers, the APSU, in collaboration with the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, initiated and coordinates the Paediatric Active Enhanced Disease Surveillance (PAEDS) system.[12] This is a hospital-based surveillance system reliant on active case ascertainment by specialist surveillance nurses. Since August 2007, PAEDS has operated in 4 tertiary paediatric hospitals in 4 states: New South Wales, Victoria, South Australia, and Western Australia (www.apsu.org.au)

All data are provided after review by the expert investigators responsible for each surveillance study and are accurate as at May 2010. However, it is possible that some notifications may be reclassified at a later date as additional clinical data for existing notifications, or additional notifications, are received.

Results

In 2008 and 2009, 1,318 and 1,340 clinicians respectively participated in the monthly surveillance of the 11 communicable or vaccine preventable diseases under surveillance. Consistent with previously reported high rates of participation by paediatricians,[10] the report card return rate was 94% in 2008 and 91% in 2009. Enhanced data on diagnosis, clinical management and short-term outcome were available for more than 85% of all cases notified. The Table shows the number of confirmed cases ascertained in 2008 and 2009 and for the whole study period, and the reported rate per 100,000 of child population for each condition.

Acute flaccid paralysis

Australia reported a non-polio AFP rate of 1.5 per 100,000 children aged less than 15 years in 2008 and 1.2 per 100,000 in 2009, exceeding the World Health Organization (WHO) AFP surveillance target of 1 case per 100,000. This is due to additional cases reported via the PAEDS system developed jointly by the APSU and the National Centre for Immunisation Research and Surveillance. The most common diagnosis of non-polio AFP was Guillain-Barré syndrome (39% in 2008 and 34% in 2009). Adequate faecal specimens (2 within 14 days of onset of paralysis) were obtained for 31% of cases, which was well below the 80% WHO target. The importation of a type 1 wild poliovirus in an adult into Australia in 2007[14] and the continued detection of cases of wild polio internationally, highlight the need for continued national surveillance.

Acute rheumatic fever

Between October 2007 and December 2009 cases of ARF were reported in all states and territories of Australia, except for Tasmania, suggesting the need for a national approach to the control of ARF and rheumatic heart disease. Almost all children were born in Australia (98%); 1 child was born in New Zealand and one in Papua New Guinea. The majority of children with ARF were Aboriginal or Torres Strait Islander, however, a small number of Caucasian children were reported from 5 states (New South Wales, Victoria, South Australia, Western Australia, and Queensland). These include the southern states where ARF is not recognised as a priority. Approximately 70% of all children reported resided in small rural towns or remote areas, with approximately 30% residing in urban or suburban areas.

Congenital cytomegalovirus infection

Congenital cytomegalovirus (cCMV) is the most common infectious cause of congenital malformations in Australia. APSU data show that cCMV infection is not associated with maternal illness in approximately one-third of cases, and should be considered regardless of maternal history.[15] cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture, use of polymerase chain reaction for urinary screening for CMV may increase diagnostic yield. Universal neonatal hearing screening programs may also help identify new cases. The total of 159 cases confirmed by the end of 2009 includes 6 cases that were notified between 2004 and 2007, but only confirmed recently.

Congenital rubella (with defects)

In 2008 there were three notifications, of which one was confirmed as a case. This was a child born to an immigrant woman from India whose

Table: Confirmed cases identified for 2008 and 2009 and for the total study period to December 2009 including reported rates per 100,000 of the relevant child population

Condition	Date study commenced	Questionnaire response (%) for total study period	Number of confirmed cases		Reported rate (per 10^5)		Number of confirmed cases for total study period	Reported rate for total study period (per 10^5 per annum)*
			2008	2009	2008	2009		
Acute flaccid paralysis (AFP)	March 1995	92	63†	50†	1.5‡	1.2‡	560†	0.9‡
Congenital cytomegalovirus	Jan 1999	74	34	32	11.5§	10.6§	159	5.5§
Acute rheumatic fever	October 2007	88	48	40	1.2‡	1.0‡	104	1.0‡
Congenital rubella (with defects)	May 1993	95	1	0	0.02	0.0	51	0.1‖
Perinatal exposure to HIV	May 1993	87	36	33	12.1§	11.0§	424	9.8§
Neonatal herpes simplex virus infection	Jan 1997	96	9	10	3.0§	3.3§	117	3.4§
Neonatal B group streptococcus Infection¶	July 2005	82	18	–	12.1§	–	150	18.0§
Congenital varicella	May 2006	100	0	0	0.0	0.0	2	0.2§
Neonatal varicella	May 2006	77	0	1	0.0	0.3§	15	1.5§
Severe complications of varicella	May 2006	69	7	4	0.2‡	0.1‡	30	0.2‡
Intussusception	May 2007	80	69	40	12.2**	6.8**	154	10.8**
Severe complications of influenza	July to Sep 2008	100	59	–	5.7‡	–	59	N/A
	May to Sep 2009	97	–	100	–	9.5‡	100	N/A

* All reported rates based on child population estimates published by the Australian Bureau of Statistics.[13]

† All cases of AFP reported via the Australian Paediatric Surveillance Unit or the Paediatric Active Enhanced Disease Surveillance system that have been classified by the Polio Expert Committee were 'non-polio AFP' according to World Health Organization criteria.

‡ Based on population of children aged less than 15 years.

§ Based on number of births.

‖ Based on population of children aged less than 16 years.

¶ Group B streptococcus sepsis study finished in June 2008.

** Based on number of children aged 24 months or younger.

N/A Due to the limited surveillance period a reported rate was not calculated.

vaccination history could not be confirmed. Serological testing was not performed. In 2009 there were no notifications. The risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs, justifying continued surveillance.[4] Such women should have serological testing for rubella after arrival in Australia, and vaccination when appropriate. For women with no prior rubella immunity, travel to rubella-endemic countries in the first trimester poses a risk of congenital rubella to the foetus. Perinatal exposure to HIV and HIV infection

In 2008 there were 36 perinatal exposures to HIV in Australia, and 33 in 2009. Antenatal diagnosis of the mother's HIV infection and use of interventions including antiretroviral treatment during pregnancy, caesarean delivery and avoidance of breastfeeding, continues to minimise the risk of mother-to-child HIV transmission.[5]

Neonatal herpes simplex virus infection

A significant number of cases of neonatal herpes simplex virus (HSV) infection continue to be confirmed, with a preponderance in females. Presentation with skin, eye and mouth disease occurred in half of confirmed cases, whereas disseminated HSV infection occurred in a quarter of confirmed cases. Among cases with disseminated infection, more than half were diagnosed with encephalitis and a third with pneumonitis. More than 20% of confirmed cases had died before notification, with almost half of these diagnosed at post-mortem examination.

Intussusception

The small number of cases ascertained suggests under-reporting and the APSU data will be greatly supplemented by cases identified by PAEDS. There was a small number of cases of intussusception observed in infants who received a rotavirus vaccine but a temporal association between either Rotarix® or RotaTeq® vaccines and intussusception could not be confirmed using APSU data alone. Accepting the limitation of under-reporting of intussusception and limited vaccination data on confirmed cases, ongoing intussusception surveillance is better justified through the PAEDS system rather than the APSU, to further explore in detail any possible relationship between the number of observed intussusception cases, the age at vaccination, dose and vaccine given.

Neonatal and infant *Streptococcus agalactiae* (group B streptococcus) sepsis

The number of notifications received over the total study period are consistent with other available data. Over half (59%) of the confirmed cases of *Streptococcus agalactiae* group B (GBS) sepsis had early onset disease (EOD: at younger than 8 days of age). Pre-term birth was more common in mothers of infants with late onset disease (LOD: at 8 days of age or older) than in mothers of infants with EOD (58% versus 28%). Infant death was more common in those with LOD than in those with EOD (8% versus 4%). A detailed final report is in preparation for peer review publication by the investigators for this study.

Severe complications of varicella infection

In 2008, 7 children hospitalised with severe complications of varicella were reported, compared with 4 cases in 2009. The complications in 2008 included septic arthritis, focal purulent collection, osteomyelitis, and ataxia, while in 2009 there were 3 cases of ataxia and one of bacteraemia. Median stay in hospital was 12.5 days in 2008 compared with 3.5 days in 2009. All of the reported children were unvaccinated and family members were the infecting contacts.

Congenital and neonatal varicella

There were no cases of neonatal varicella reported in 2008 and only 1 case in 2009. This was an infant born to a woman who experienced symptoms of varicella infection 1 day after delivery. The infecting contact was identified as the woman's husband who had been told that the illness he was experiencing was not chicken pox. No cases of congenital varicella were reported in 2008 and 2009.

Severe complications of influenza

In 2008, influenza B was the dominant influenza type among the 59 children hospitalised with severe complications of influenza and reported to APSU. In 2009, the dominant strain was pandemic influenza H1N1 2009 among 100 children reported to APSU. A range of complications were reported with x-ray-confirmed pneumonia the most common during both years. However, in 2009 serious complications such as encephalitis and rhabdomyolysis were more common than in 2008. Admission to paediatric intensive care was more common in 2009 (38%) compared with 2008 (29%) and 7 (7%) of the reported children died in 2009 compared with only 1 child (2%)

in 2008. Vaccination for seasonal influenza was uncommon during both 2008 and 2009, even among children with pre-existing chronic disorders who were eligible for vaccination according to current recommendations.

Conclusions and future directions

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policymakers and the community.[10,11,16] The APSU is often the only source of national data that includes clinical and/or laboratory details, and data on both inpatients and outpatients.[10,11]

After demonstrating the feasibility of the APSU to respond rapidly to an outbreak of influenza in 2007, it has conducted surveillance for seasonal influenza in 2008 and surveillance for both seasonal and pandemic influenza in 2009. The APSU will again conduct surveillance for the severe complications of influenza from June to September in 2010.

A surveillance study of juvenile respiratory papillomatosis is planned for late 2010. Respiratory papillomatosis is a rare but devastating condition in children aged less than 12 years, and is thought to be perinatally transmitted.[17] Juvenile respiratory papillomatosis is difficult to treat, recurrences are common and may lead to airway obstruction. The human papillomavirus (HPV) vaccine, which protects against HPV6 and HPV11, is currently nationally recommended and it is hoped that the rates of juvenile papillomatosis among young children will reduce with increased vaccination rates.

The APSU continues to provide useful data and clinical and public health insights relating to infectious diseases in Australian children. Ongoing surveillance through the PAEDS system will continue to complement the work of the APSU, and both APSU and PAEDS provide a platform for the rapid response to potential emerging infectious diseases threatening Australian children.

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References

1. Roberts JA, Grant KA, Yoon YK, Polychronopoulos S, Ibrahim A, Thorley BR. Annual report of the Australian National Poliovirus Reference Laboratory, 2008. *Commun Dis Intell* 2009;33(3):291–297.

2. Carapetis JR, Steer AC, Mulholland EK, Weber M. The global burden of group A streptococcal diseases. *Lancet Infect Dis* 2005;5(11):685–694.

3. Rawlinson WD, Hall B, Jones CA, Jeffery HE, Arbuckle SM, Graf N, et al. Viruses and other infections in stillbirth: what is the evidence and what should we be doing? *Pathology* 2008;40(2):149–160.

4. Gidding HF, Young M, Pugh R, Burgess M. Rubella in Australia: can we explain two recent cases of congenital rubella syndrome? *Commun Dis Intell* 2003;27(4):537–540.

5. McDonald AM, Zurynski YA, Wand HC, Giles ML, Elliott EJ, Ziegler JB, et al. Perinatal exposure to HIV among children born in Australia, 1982–2006. *Med J Aust* 2009;190(8):416–420.

6. Bines JE, Patel M, Parashar U. Assessment of postlicensure safety of rotavirus vaccines, with emphasis on intussusception. *J Infect Dis* 2009;200(Suppl 1):S282–S290.

7. Haber P, Patel M, Izurieta HS, Baggs J, Gargiullo P, Weintraub E, et al. Postlicensure monitoring of intussusception after RotaTeq vaccination in the United States, 1 February 2006, to 25 September 2007. *Pediatrics* 2008;121(6):1206–1212.

8. Peadon E, Burgner D, Nissen M, Buttery J, Zurynski Y, Elliott E, et al. Case for varicella surveillance in Australia. *J Paediatr Child Health* 2006;42(11):663–664.

9. Zurynski YA, Lester-Smith D, Festa MS, Kesson AM, Booy R, Elliott EJ. Enhanced surveillance for serious complications of influenza in children: role of the Australian Paediatric Surveillance Unit. *Commun Dis Intell* 2008;32(1):71–76.

10. He S, Zurynski YA, Elliott EJ. Evaluation of a national resource to identify and study rare diseases: The Australian Paediatric Surveillance Unit. *J Paediatr Child Health* 2009;45(9):498–504.

11. Zurynski YA, Peadon E, Bower C, Elliott E. Impacts of national surveillance for uncommon conditions in childhood. *J Paediatr Child Health* 2007;43(11):724–731.

12. Pym M, Adams J, Booy R, Buttery J, Elia S, Elliott E, et al. The development and trial of paediatric active enhanced disease surveillance (PAEDS): A new surveillance mechanism for Australia. *J Paediatr Child Health* 2008;44:A16.

13. Australian Bureau of Statistics. Australian Demographic Statistics. September quarter 2009. Canberra: Australian Bureau of Statistics; 2010. Catalogue no: 3101.0

14. Carnie JA, Lester R, Moran R, Brown L, Meagher J, Roberts JA, et al. Public health response to imported case of poliomyelitis, Australia, 2007. *Emerg Infect Dis* 2009;15(11):1733–1737.

15. Howard J, Hall B, Brennan LE, Arbuckle S, Craig ME, Graf N, et al. Utility of newborn screening cards for detecting CMV infection in cases of stillbirth. *J Clin Virol* 2009;44(3):215–218.

16. Grenier D, Elliott EJ, Zurynski YA, Pereira RR, Preece M, Lynn R, et al. Beyond counting numbers: Public health impacts of national paediatric surveillance units. *Arch Dis Child* 2007;92(6):527–533.

17. Schaffer A, Brotherton J, Booy R. Do human papillomavirus vaccines have any role in newborns and the prevention of recurrent respiratory papillomatosis in children? *J Paediatr Child Health* 2007;43(9):579–580.

Annual report of the Australian Meningococcal Surveillance Programme, 2009

The Australian Meningococcal Surveillance Programme

Abstract

In 2009 there were 233 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, Australia, a nationwide network of reference laboratories. One hundred and thirty–five isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were available for which the phenotypes (serogroup, serotype and serosubtype) and/or genotype and antibiotic susceptibility were determined. An additional 98 cases were confirmed by non-culture-based methods (92 by nucleic acid amplification testing (NAAT) and six by serology) , and where possible serotyping was determined. Nationally, 194 (83%) laboratory-confirmed cases where a serogroup was determined were infected with serogroup B and 13 (5.6%) serogroup C meningococci. The national total of confirmed cases has remained relatively stable since 2006, but the number of cases may vary between jurisdictions each year. New South Wales had the highest number of recorded cases in 2009. Typical primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents and young adults respectively. Serogroup B cases predominated in all age groups and jurisdictions. The common phenotypes circulating in Australia continue to be B:15:P1.7 and B:4:P1.4. Although serogroup C cases were low, phenotype C:2a:P1.5 again predominated in this group. No evidence of meningococcal capsular 'switching' was detected. Approximately two-thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). All isolates remained susceptible to ceftriaxone. Four isolates had reduced susceptibility to ciprofloxacin, and none to rifampicin. *Commun Dis Intell* 2010;34(3):291–302.

Keywords: disease surveillance; meningococcal disease; *Neisseria meningitidis*

Introduction

The National Neisseria Network (NNN) is a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria species, *Neisseria meningitidis* and *N. gonorrhoeae*. Since 1994 the NNN has operated through a network of reference laboratories in each state and territory to provide a national laboratory-based program for the examination of *N. meningitidis* from cases of invasive meningococcal disease (IMD).[1] The NNN supplies data on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility, supplementing clinical notification data from the National Notifiable Diseases Surveillance System (NNDSS). The NNN receives samples for analysis from about 90% (range 85%–92% 2004–2009) of IMD cases notified to NNDSS. The NNN annual reports are published in *Communicable Diseases Intelligence*.[2]

The characteristics of the meningococci responsible for IMD are important both for individual patient management, and to tailor the public health response for outbreaks or case clusters locally and nationally. The introduction of publicly funded conjugate serogroup C meningococcal vaccine onto the National Immunisation Program in 2003 (with a catch up program for 1–19-year-olds that ran until May 2007) saw a significant and sustained reduction in the number of cases of IMD evident after 2004. However, IMD remains an issue of public health concern in Australia. The success of any further vaccine initiatives in Australia is dependent upon detailed analysis of the *N. meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in Australia in 2009.

Methods

Isolate based invasive meningococcal disease cases

Case confirmation

Case confirmation was based upon isolation of, or positive nucleic acid amplification testing for *N. meningitidis* from a normally sterile site or by

positive serology and defined as IMD according to Public Health Laboratory Network criteria.[3] Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was grown from both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case was classified as one of meningitis. It is recognised that total number of cases and particularly the number of cases of meningitis, e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture sterile, is underestimated. However the above approach has been used since the beginning of this program[1] and is continued for comparative purposes.

Phenotyping and genotyping

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein (porin) antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health, The Netherlands. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* and *FetA* has been used to supplement and supplant serotyping analyses based on the use of monoclonal antibodies.

Antibiotic susceptibility

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique.[4]

sensitive	MIC ≤ 0.03 mg/L
less sensitive	MIC 0.06 – 0.5 mg/L
relatively resistant	MIC ≥ 1 mg/L

Strains with MIC values which place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses. However precise MIC outcome correlations are difficult to obtain because of the nature of IMD.

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD was obtained by means of non-culture-based methods primarily by nucleic acid amplification testing (NAAT) and occasionally by serological techniques. NAAT testing is essentially by polymerase chain reaction (PCR) techniques[5] that demonstrate the presence of meningococcal-specific nucleic acid in appropriate samples and has been progressively introduced and updated in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report. The serological results are based on results of tests performed using the methods and test criteria of the Manchester Public Health Laboratory Service reference laboratory, United Kingdom, as assessed for Australian conditions.[6–9] Where age, sex and outcome data for patients with non-culture-based diagnoses are available, these were also recorded. The site of a sample of a positive NAAT is also used to define the clinical syndrome.

Results

Aggregated data on cases confirmed by culture-based and non-culture-based methods

Number of laboratory-confirmed cases

There were 233 laboratory-confirmed cases of IMD in 2009 (Table 1) compared with 260 in 2008; 281 in 2007; 271 in 2006; 345 in 2005 and 361 in 2004. In 135 (58%) cases, a positive culture was obtained with or without a positive non-culture-based test and 98 (42%) cases were confirmed by a non-culture-based method alone. The highest number of laboratory-confirmed cases was from New South Wales (82 cases), which has

Table 1: Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2009, by serogroup and state or territory

State or territory	Serogroup					Total
	B	C	Y	W135	NG	
ACT	3	0	0	0	0	3
NSW	60	7	4	4	7	82
NT	2	1	0	0	0	3
Qld	54	2	1	0	3	60
SA	21	0	2	0	0	23
Tas	2	0	0	0	0	2
Vic	34	1	1	0	3	39
WA	18	2	1	0	0	21
Australia	194	13	9	4	13	233

NG Non groupable

increased from 62 in 2008 but decreased from 101 in 2007. The total number of all laboratory-confirmed cases decreased in Queensland from 83 in 2008 (and 75 in 2007) to 60 in 2009. There was also a decrease in cases from Victoria (39 cases in 2009), which was lower than the 61 cases in 2008 and 59 cases in 2007. Small or no numerical differences were noted in other jurisdictions.

Seasonality

Forty-five (19%) cases occurred between 1 January and 31 March, 56 (24%) between 1 April and 30 June, 83 (36%) between 1 July and 30 September and 49 (21%) between 1 October and 31 December. A winter peak of meningococcal disease is usual and the above pattern was also present in 2007 and 2008.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those aged 4 years and under (Table 2). Those aged less than 1 year or in the 1–4 year age group together accounted for 78 (33%) cases of the total) in 2009. There were 94 (36%) cases confirmed in these age groups in 2008 and 100 (36%) in 2007. A secondary disease

Table 2: All laboratory-confirmed cases of invasive meningococcal disease, Australia, 2009, by age, state or territory and serogroups B and C

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	2	1	0	0	0	0	0	0	0	0	3
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	2	1	0	0	0	0	0	0	0	0	3
NSW	B	10	12	2	2	13	2	6	3	2	8	60
	C	0	1	0	1	1	0	1	1	0	2	7
	Total	10	13	2	3	14	2	7	4	2	10	67
NT	B	1	0	1	0	0	0	0	0	0	0	2
	C	0	0	0	0	0	0	1	0	0	0	1
	Total	1	0	1	0	0	0	1	0	0	0	3
Qld	B	7	13	6	2	7	5	10	4	0	0	54
	C	0	0	1	1	0	0	0	0	0	0	2
	Total	7	13	7	3	7	5	10	4	0	0	56
SA	B	2	3	0	1	9	2	3	1	0	0	21
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	2	3	0	1	9	2	3	1	0	0	21
Tas	B	0	2	0	0	0	0	0	0	0	0	2
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	2	0	0	0	0	0	0	0	0	0
Vic	B	4	7	1	2	7	5	5	1	2	0	34
	C	1	0	0	0	0	0	0	0	0	0	1
	Total	5	7	1	2	7	5	5	1	2	0	35
WA	B	4	4	1	3	2	0	2	1	1	0	18
	C	0	0	0	0	0	1	1	0	0	0	2
	Total	4	4	1	3	2	1	3	1	1	0	20
Australia	B	30	42	11	10	38	14	26	10	5	8	194
	C	1	1	1	2	1	1	3	1	0	2	13
	Total B+C	31	43	12	12	39	15	29	11	5	10	207
	Other	3	1	2	2	7	1	4	3	3	0	26
	Total	34	44	14	14	46	16	33	14	8	10	233
	% of all	14.6	18.5	6.0	6.0	19.7	6.9	14.2	6.0	3.4	4.3	

NS Not stated.

Totals include cases due to other serogroups (13) and cases where the serogroup was not determined (13).

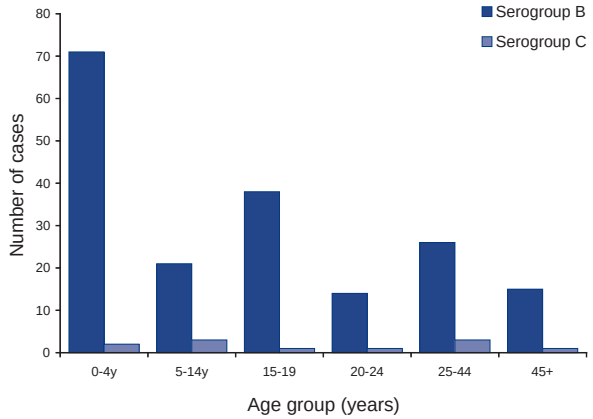
peak is also usual in the adolescent/young adult age group (15–24 years). The total of 46 (20%) of all confirmed cases in those aged 15–9 years in 2009 was a little less than the 50 cases (19%) in 2008 and 56 (20%) cases in this age group in 2007. Those aged 15–24 years accounted for 62 (27%) cases compared with 71 (27%) cases in 2008 and 87 (31%) cases in 2007.

Serogroup data

The serogroup of the meningococci causing disease was determined in 220 of the 233 laboratory-confirmed cases of IMD. Of these 220 cases where a serogroup was determined, 194 (88%) were serogroup B and 13 (5.9%) were serogroup C. This distribution was unchanged from 2008 where 219 (88%) were serogroup B and 15 (6%) were serogroup C and was much the same in 2007, where 223 (85%) were serogroup B and 17 (6.5%) were serogroup C. In 2009, an additional 9 (3.9%) cases were of serogroup Y and 4 (1.7%) of serogroup W135. With the continuing low numbers of serogroup C infections, serogroup B meningococci predominated in all age groups (Figure) and jurisdictional differences in serogroup distribution were not evident. The 13 serogroup C cases of IMD were distributed in 5 jurisdictions: New South Wales (7), Queensland (2), Western Australia (2) Victoria and the Northern Territory (1 each). Four of the 13 cases of serogroup C disease in 2009 were aged 25 years or more; 3 cases were in the 5–14 age group; 2 cases were reported in those aged 4 years or less, a single case in the 15–19 year age group and one in the 20–24 year age group. The age of the patient was not specified for 2 cases.

Table 3 shows a national comparison of the number and proportion of serogroup B and C cases by age from 2004 to 2009. In those aged 14 years or less, there was a decrease in total case numbers and in serogroup B cases in 2009, from 2008. Serogroup C case numbers were low in these age groups across this period. In the 15–19 year age group, the number of serogroup B cases has again decreased, but in the 20-24 years group the proportion of serogroup B cases increased as serogroup C cases declined. In older (25 years or more) age groups in 2009, there was a decrease in the number and proportion of both serogroup B and serogroup C cases when compared with 2008. This reduction follows an increase in the number and proportion of serogroup B cases from 2007 to 2008 when the number and proportion of serogroup C cases was unchanged.

Figure: Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2009, by age



Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2009, the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype were analysed for New South Wales, the Australian Capital Territory, Western Australia and the Northern Territory (Darwin) isolates. The serogroup B and C serotypes/serosubtypes are shown in Table 4. Serogroup B meningococci are in general more difficult to characterise by serological methods and a number could not be phenotyped. A total of 53 isolates were serotyped, 44 were serogroup B and 9 were serogroup C. Of those that were serogroup B, 14 were serogroup B serotype 15, and 11 of the 14 were serosubtype P1.7, which has been circulating in Australia for many years. Nine were serotype 4 and five of these were serosubtype P1.4, which has been circulating in New Zealand at high rates for many years. Nine were non-typeable.

Nine serogroup C strains were phenotyped (including 2 from Queensland) and all were serotype 2a. This phenotype has predominated in serogroup C meningococci in Australia for many years. Of these nine, eight were phenotype C: 2a:P1.5 and 1 strain was non-subtypeable. There is continuing interest in the presence of any serogroup B or serogroup C meningococci of serotypes that indicate the possibility of genetic recombination events. Among serogroup C strains, phenotype C: 2a:P1.4 has been of particular interest. This phenotype has figured prominently in Victorian data in former years. For example, in 2003 there were 29, 21 in 2004, and in 2005 8 serogroup C isolates of this serotype/serosubtype were detected nationally. No isolates with this phenotype were seen in 2009.

Table 3: Comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2009, by known age

Year	Serogroup	Age (years)									
		< 4		5–14		15–19		20–24		25+	
		n	%	n	%	n	%	n	%	n	%
2009	B	72	92.2	21	75.0	38	82.6	14	87.5	41	74.5
	C	2	2.6	3	10.7	1	2.2	1	6.3	4	7.3
	All*	77		28		46		16		55	
2008	B	82	89.1	23	95.8	42	91.3	15	83.3	57	85.1
	C	4	4.4	0	0.0	1	2.2	2	11.1	8	11.4
	All*	92		24		46		18		67	
2007	B	83	90.0	19	83.0	48	91.0	24	80.0	49	75.0
	C	4	4.0	0	0.0	2	4.0	3	10.0	8	12.0
	All	92		23		53		30		65	
2006	B	93	93.0	21	84.0	40	82.0	21	70.0	38	61.3
	C	2	2.0	3	12.0	4	8.2	7	23.0	10	16.1
	All	100		25		49		30		62	
2005	B	99	90.0	38	75.0	39	81.0	22	67.0	51	50.0
	C	6	5.5	5	10.0	4	8.0	8	24.0	27	27.0
	All	110		51		48		33		101	
2004	B	97	88.0	27	77.0	40	65.0	20	57.0	59	50.0
	C	6	5.5	5	14.0	17	28.0	11	31.0	32	27.0
	All	110		35		61		35		117	

* All cases where a serogroup was determined and patient's age was supplied.

Genotyping data of invasive meningococcal samples (culture or NAAT products)

Sequencing products derived from amplification of the variable region *porA* and *porB* and *FetA* genes has been used in an increasing number of jurisdictions in place of serotyping, based on the use of monoclonal antibodies. In 2009, some jurisdictions have moved to the use of genotyping (Victoria, Queensland, South Australia (including Alice Springs) and Tasmania). There was a heterogeneity of typing data across jurisdictions with predominance of a few phenotypes or genotypes as shown in Table 4.

In New South Wales there was a cluster of 3 cases (1 culture-positive and non-phenotypeable and 2 NAAT positive) where genotyping was performed with the results indistinguishable for all 3 cases (Table 4).

Outcome data for invasive meningococcal disease for laboratory-confirmed cases

Outcome data (survived or died) were available for 69 (30%) of the 233 laboratory-confirmed cases (Table 5). Three deaths were recorded in this group (1.3%), all of which were attributable to septicaemia and with serogroup B infection. Outcome data were available for 58 of 194 cases with serogroup B infection. No deaths were recorded for the remainder of infections caused by other serogroups.

Anatomical source of samples for laboratory-confirmed cases

Table 6 shows the source of clinical samples by which laboratory confirmation of IMD was obtained. Those diagnoses shown as culture positive may have had positive PCR and/or serology; those shown as PCR positive were culture negative with or without positive serology. There were 77 diagnoses of meningitis based on cultures or PCR examination of CSF either alone or with a positive blood sample; and 144 from blood samples (cultures or PCR) alone. There were 3 other isolates from synovial fluid and in 2 cases the source of the clinical sample was not disclosed. There were 6 cases that were serologically positive where culture and PCR were negative.

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease infection, 2009, by state or territory

State or territory	Serogroup	Phenotype				Genotype					
		Sero-type	n	Sero-subtype	n	*porB*	n	*porA*	n	*FetA*	n
ACT	B	15	1	P1.7	1						
		NT	2	P1.14	1						
				P1.9	1						
NSW	B	15	12	P1.7	9						
				P1.15	1						
				NST	2						
		4	8	P1.4	5						
				P1.15	1						
				P1.14	1						
				NST	1						
		1	3	P1.14	1						
				P1.6,	1						
				NST	1						
		NT	4	NST	4						
	*Cluster	NT	1	NST	1	B,C,7,14b	3	P1.19-1;15-11	3	F5-1	3
		14	1	P1.6.3	1						
	C	2a	5	P1.5	4						
				NST	1						
NT	B	4	1	P1.14	1			P1.22-1,14	1		
	C							P1.5-1,10-8	1		
Qld	B							P1.22,14	6	F1-7	1
										F1-68	1
										F5-5	1
										F1-31	1
										ND	2
								P1.19,13-1	3	F5-1	3
								P1.18-1,34	2	F1-5	2
								P1.22-21,14	2	F5-5	2
								P1.1,16-26	1		

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease infection, 2009, by state or territory, *continued*

State or territory	Serogroup	Phenotype				Genotype					
		Sero-type	n	Sero-subtype	n	*porB*	n	*porA*	n	*FetA*	n
Qld, cont'd								P1.19-1,10-8	1		
								P1.19-1,15	1	F5-1	
								P1.22,14-6	1	F1-5	1
								P1.4,16-26	1	ND	
								P1.5-1,10-4	1	F1-5	
								P1.7,16-106	1	F3-3	
								P1.7-2,4		F3-6	1
	C	2a	2	P1.5	2			P1.5-1,10-8	2		
SA	B							P1.7-2,4	9	F1-5	7
								P1.18-1,3	2	F5-1	1
								P1.18-1,34	1	F1-5	1
								P1.19-1, 15-11	1	F5-1	1
								P1.17, 16-93	1	F5-5	1
								P1.7, 16	1	F3-3	1
								P1.22, 14	1	F5-9	1
								P1.5-1,10-8	1		
Tas	B	ND		ND							
Vic	B					A,A,A,Ba	4	P1.7,16-26	4		
						4,D,7,14a	3	P1.7-2,4	3		
						4,C,7,14a	2	P1.18-1,3	2		
						19,Ac,7a,1	2	P1.18-1,34	2		
						19,Ab,7var,Aa	1	P1.17,16-3	1		
						19,Ac,7a,1	2	P1.22,14	2		
						19,Db,7c,14	1	P1.22,14-6	1		
						19,Dvar,7b,14	1	P1.22,14	1		
						4,C,7,14a	1	P1.7-2,13-2	1		
						B,C,7,14b	1	P1.5-1,2-2	1		
						ND	2	P1.19-1,15-11	2		
						ND	2	P1.22,14	2		

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease infection, 2009, by state or territory, *continued*

State or territory	Serogroup	Phenotype				Genotype					
		Sero-type	n	Sero-subtype	n	*porB*	n	*porA*	n	*FetA*	n
Vic, cont'd						ND	2	P1.7,16-26	2		
						ND	1	P1.7-2,4	1		
						new,D(var),7b,B(var)	1	P1.22,9	1		
						new,D(var),7c,B(var)	1	P1.18-1,34	1		
	C							P1.5-1,10-8			
WA	B	14	2	P1.7	1						
				NST	1						
		15	1	P1.7	1						
		NT	8	NST	4						
				P1.14	1						
				P1.9	2						
				P1.5	1						
	C	2a	2	P1.5	2						

* Cluster of 3 cases (1 culture positive and non phenotypeable; and 2 NAAT positive) where genotyping was performed.

NT Not typeable

NST Not subtypable

ND Not determined.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

All 135 isolates from culture confirmed cases of IMD in 2009 were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 91 (67%) isolates were less sensitive to penicillin in the MIC range 0.06 to 0.5 mg/L and the remainder (23%) fully sensitive (MIC 0.03 mg/L or less). The proportion of less sensitive strains is lower than that reported in 2008 (72%) and 2007 (79%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone and by extrapolation to other 3rd generation cephalosporins. Four isolates had altered susceptibility (MIC, 0.06–0.5 mg/L) to ciprofloxacin (MIC, 0.25 mg/L), three from Victoria and one from Western Australia. There were no isolates with altered susceptibility to rifampicin.

Discussion

In 2009, there were 233 cases analysed by the NNN representing a decrease in numbers from previous years. The total number of laboratory-confirmed cases of IMD nationally was relatively stable from 2006 to 2008 (range 260–281) after recording 345 cases in 2005. However; there have been fluctuations in the frequency of detection of cases between jurisdictions over this period with New South Wales recording the highest number of cases in 2009 (82), whereas Queensland recorded the highest number of cases in 2008 (83). There was also a decrease in the number of cases in Victoria from 61 in 2008 to 39 in 2009. These changes in case distribution were essentially attributable to altered numbers of serogroup B cases in 2009 and once again little change was detected in serogroup C numbers. Cultures were obtained from sterile sites in 135 (58%) cases, the lowest number of isolates detected over the duration of the program since it commenced in 1994; however this is proportionally similar to the number of isolates received in recent years: 2008 (149: 57%) 2007 (154: 55%) and 2006

Table 5: Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, 2009, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG*	
Meningitis	Survived	17	0	1	0	1	19
	Died	0	0	0	0	0	0
	Total	17	0	1	0	1	19
Septicaemia	Survived	38	2	3	2	2	47
	Died	3	0	0	0	0	3
	Total	41	2	3	2	2	50
All cases	Survived	55	2	4	2	3	66
	Died	3	0	0	0	0	3
	Total	58	2	4	2	3	69

NG Not groupable

Table 6: Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2009

Specimen type	Isolate of meningococci	PCR positive*	Serology alone	Total
Blood	100	44	–	144
CSF +/– blood	32	45	–	77
Other†	3	3	–	6
Serum/serology	–	–	6	6
Total	135	92	6	233

* Polymerase chain reaction (PCR) positive in the absence of a positive culture.

† Other samples: 3 isolates from joints, 1 PCR from joint and 2 PCR diagnoses from an unknown source.

CSF Cerebrospinal fluid.

(166: 61%). Non-culture-based diagnoses were used to confirm a further 91 (39%) cases of IMD in 2009, compared with 111 (43%) in 2008 and 127 (45%) in 2007. Attention is specifically drawn to earlier Australian Meningococcal Surveillance Programme (AMSP) reports that explain differences between the numbers of clinically notified cases and laboratory-confirmed cases.[10] It should also be noted that surveillance systems rarely capture all cases in any given period so that small differences in numbers of cases should be expected.

Only 13 serogroup C infections were identified nationally in 2009 so that serogroup B disease accounted for 88% of all infections where a serogroup was determined. No serogroup C cases were identified in South Australia, the Australian Capital Territory or Tasmania while there were 7 cases in New South Wales and small numbers present in the other jurisdictions. Only low numbers of infections due to serogroups Y and W135 were encountered, which is usual for Australia. A primary peak in IMD infection rates was again evident in younger age groups, with a secondary peak in adolescents and young adults. The distribution of serogroup C disease was low across all age groups in 2009. As in previous years, there was a small number of serogroup C cases in those aged 25 years or more (Table 3), which may reflect the secondary benefit of herd immunity accruing to the wider community following vaccination of those age groups where disease was formerly highly concentrated.[11]

Phenotypic and genotypic data again found no evidence of substantial numbers of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination, although sporadic instances of this occurrence have been detected in Australia. There were some concerns expressed that the documented capacity for genetic reconfiguration within meningococci may lead to the emergence of new and invasive subtypes following extensive vaccine use.[11] Analysis of meningococcal subtypes and any evidence of the expansion of 'new' subtypes will continue as part of the NNN Programme. Mortality data were assessable in only a low proportion of cases and must be interpreted with caution. All of the small number of fatal cases of IMD were associated with serogroup B infections. The NNN does not attempt collection of morbidity data associated with IMD.

The distribution of penicillin MIC values from invasive isolates in 2009 showed that the proportion with decreased susceptibility to penicillins was 67%, a little less than that observed in 2008 (72%) and 2007 (79%). It is emphasised that this decreased susceptibility does not affect clinical outcomes and penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the 3rd generation cephalosporins and to the 'clearance' antibiotics rifampicin and ciprofloxacin with the exception of 4 isolates with decreased susceptibility ciprofloxacin: three from Queensland and one from Western Australia. The group of strains with decreased susceptibility to quinolone antibiotics is the subject of on-going international interest following their first description from the AMSP group in 2000.[12–15] There were 2 isolates in AMSP data with decreased susceptibility to quinolone antibiotics detected in 2008, and one in 2007.

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References

1. National Neisseria Network. Meningococcal isolate surveillance Australia, 1994. *Commun Dis Intell* 1995:19:286–289.

2. The Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2006. *Commun Dis Intell* 2007;31(2):185–194.

3. Public Health Laboratory Network. Meningococcal long case definition. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-phlncd-mening.htm

4. Tapsall J and members of the National Neisseria Network of Australia. Antimicrobial testing and applications in the pathogenic *Neisseria*. In: Merlino J, ed. *Antimicrobial susceptibility testing: methods and practices with an Australian perspective*. Australian Society for Microbiology, Sydney, 2004. pp 175–188.

5. Porritt RJ, Mercer JL, Munro R. Detection and serogroup determination of *Neisseria meningitidis* in CSF by polymerase chain reaction (PCR). *Pathology* 2000;32(1):42–45.

6. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1999. *Commun Dis Intell* 2000;24(7):181–189.

7. Gray SJ, Borrow R, Kaczmarski EB. Meningococcal serology. In: Pollard AJ, Martin MCJ, eds. *Meningococcal disease methods and protocols*. Humana Press, Totawa, New Jersey, 2001 pp. 61–87.

8. Robertson PW, Reinbott P, Duffy Y, Binotto E, Tapsall JW. Confirmation of invasive meningococcal disease by single point estimation of IgM antibody to outer membrane protein of *Neisseria meningitidis*. *Pathology* 2001:33(3):375–378.

9. Lahra MM, Robertson PW, Whybin R, Tapsall JW. Enhanced serological diagnosis of invasive meningococcal disease by determining anti-group C capsule IgM antibody by EIA. *Pathology* 2005;37(3):239–241.

10. The Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme 2002. *Commun Dis Intell* 2003;27(2):196–208.

11. Maiden MCJ, Ibarrz-Pavon AB, Urwin R, Gray SJ, Andrews NJ, Clarke SC, et al. Impact of meningococcal serogroup C cojugate vaccines on carriage and herd immunity. *J Infect Dis* 2008;197(5):737–743.

12. Shultz TR, Tapsall JW, White PA, Newton PJ. An invasive isolate of *Neisseria meningitidis* showing decreased susceptibility to quinolones. *Antimicrob Agents Chemother* 2000;44:1116.

13. Singhal S, Purnapatre KP, Kalia V, Dube S, Nair D, Deb M, et al. Ciprofloxacin-resistant *Neisseria meningitidis,* Delhi, India. *Emerg Infect Dis* 2007;13(10):1614–1616.

14. Centers fro Disease Control and Prevention. Emergence of fluoroquinolone-resistant *Neisseria meningitidis*—Minnesota and North Dakota, 2007–2008. *MMWR Morb Mortal Wkly Rep* 2008;57(7):173–175.

15. Shultz TR, White PA, Tapsall JW. An *in-vitro* assessment of the further potential for development of quinolone resistance in *Neisseria meningiditis*. *Antimicrob Agent Chemother* 2005;49(5):1753–1760.

Peer-reviewed articles

Antiviral distribution data — a potential syndromic surveillance system to assist pandemic health service operational planning

Andrew SC Way, David N Durrheim, Tony Merritt, Hassan Vally

Abstract

A pilot study was conducted in rural northern New South Wales from 15 July to 28 August 2009, during Australia's Protect Phase response to the Influenza A H1N1 California 7/09 pandemic. This study explored the feasibility of using administrative data, generated from the distribution of stockpiled antivirals, as a syndromic surveillance system. The purpose was to identify recently affected towns or those with increasing influenza-like illness activity to assist in rural health service operational planning. Analysis of antiviral distribution data was restricted to 113 general practices in rural parts of the Hunter New England Area Health Service. By 2 September 2009 a total of 6,670 courses of antivirals for adults, of which 455 courses were replacement orders, had been distributed to these general practices. Distribution of replacement antivirals were mapped to local government areas on a weekly basis. The syndromic surveillance system delivered timely data on antiviral distribution; used readily available software to generate visual activity maps in less than 30 minutes; proved adaptable; was of low cost; and was well received by health service planners. Full evaluation of the system's utility was limited by the relatively large initial distribution of antivirals and the brief nature of Australia's first pandemic wave. The pilot study demonstrated that a syndromic surveillance system based on distribution of supplies, such as treatment or vaccines, can support local health service operational planning during health emergencies. *Commun Dis Intell* 2010;34(3):303–309.

Keywords: pandemic influenza, antiviral, syndromic surveillance, health service planning

Introduction

The World Health Organization global pandemic alert level was raised to Phase 6 on 11 June 2009,[1] indicating a global pandemic was underway and that it was considered no longer possible to contain the novel pandemic Influenza A H1N1 California 7/09 (pH1N1) virus within a particular geographical area. On 17 June 2009, the Australian Government announced a change in its pandemic response from the Contain Phase to the Protect Phase.[2] The newly developed Protect Phase focused on treating and caring for individuals who were more vulnerable to a severe outcome from pH1N1.

Antiviral medication was distributed from State and Commonwealth medical stockpiles during the Contain and Protect phases. Antiviral usage in the Contain Phase targeted the treatment of suspected pH1N1 cases and prophylaxis of individuals in close contact with suspected cases. In New South Wales, antivirals were mostly dispensed from hospital emergency departments with authorisation from public health units. In contrast, antiviral usage in the Protect Phase aimed to reduce disease impact through treatment of individuals with influenza-like illness (ILI) who were classified as being in defined vulnerable groups. During this phase in New South Wales, antivirals were dispensed through both hospitals and primary health care providers (PHCPs), including general practices and Aboriginal Medical Services.[3]

A pilot study was conducted during the Protect Phase in the Hunter New England (HNE) region of northern New South Wales to explore the feasibility of using data generated from the distribution of stockpiled antivirals for syndromic surveillance. Replacement orders of antivirals were only distributed to PHCPs once a line-list of patients who had received antivirals was provided. Thus replacement orders could be used to measure antiviral usage and serve as a proxy for local ILI activity. It was assumed that PHCPs adhered to the vulnerable group criteria, as defined in the Protect Phase plan, for dispensing antivirals and that they were experienced in ILI diagnosis.

This system aimed to assist rural HNE health services operationally plan their response to pH1N1 through early identification of towns recently affected via rapidly mapping increases in antiviral distribution. If successful, it could permit public health investigation and surging of area health service (AHS) and divisions of general practice resources in a timely manner.

Methods

Distribution of stockpiled antivirals in New South Wales during the pandemic response in 2009

The distribution of antiviral medication by the NSW Department of Health (NSW Health) occurred through the State Vaccine Centre (SVC), a well established system used for routine vaccine distribution throughout New South Wales. The antiviral distribution data were stratified by each quantity distributed to a PHCP and circulated daily as a Microsoft Excel spreadsheet from the SVC to NSW Health and from NSW Health to each AHS. All New South Wales PHCPs which ordered antivirals received an initial antiviral pack of 50 adult courses of oseltamivir (Tamiflu®) and 5 adult courses of zanamivir (Relenza®), regardless of the initial amount of antivirals ordered. Thus each initial antiviral pack contained 55 adult courses.

Data analysis

In HNE, a total of 261 PHCPs received an initial antiviral pack, including 249 general practices, 8 HNE Aboriginal Medical Services, 3 HNE Student Health Services and 1 HNE Nursing Home. Analysis of antiviral distribution data was restricted to 113 general practices in rural areas of HNE. Patients in urban areas of Newcastle and Lake Macquarie had ready access to 4 large public hospitals, community pharmacies and PHCPs, to obtain antivirals. This level of access was not available in rural HNE and therefore the distribution of antivirals to PHCPs was more likely to be representative of pH1N1 activity.

The number of full-time equivalent (FTE) General Practitioners (GPs) working in each general practice was obtained from the 4 divisions of general practice serving rural HNE. Due to privacy concerns certain divisions provided FTE data by geographical area (e.g. at the town level), rather than for a specific practice. Where this occurred the FTEs were allocated on a proportional basis to each practice in that geographic area. This allowed comparison of replacement antiviral distribution by general practice, town or Local Government Areas (LGA).

LGAs are a commonly used geospatial area in Australia. It was assumed that the demographic characteristics within an LGA were similar and that most residents would seek medical services from within that LGA. This allowed identification of differences in replacement antiviral rates between LGAs and between individual towns within a single LGA, thus alerting health planners to recent changes in ILI activity at a local level.

Using the statistical software SAS,[4] a program was developed that managed data for rural HNE general practices, removed duplicate entries, and assigned the number of FTE GPs to each general practice. Two outputs were generated from the program: a) the cumulative total of replacement antivirals distributed to each general practice, until 2 weeks prior to analysis; and b) the total replacement antivirals distributed to each general practice during the 2 weeks preceding analysis.

MapInfo[5] was used to display the surveillance system results. Geospatial data for HNE LGAs were combined with the SAS outputs to produce the following two data displays:

- The town replacement antiviral rate was displayed as town indicator using relative size and shaded classifications (Map 1). This reflected the average number of replacement antivirals per FTE GP distributed to that town during the 2 weeks preceding analysis. The same rate categories used for the LGA classifications were applied to allow comparison.

- The LGA replacement antiviral rate was displayed by shaded classification of LGAs (Map 2). This reflected the total number of replacement antivirals per FTE GP distributed to general practices in that LGA, until 2 weeks prior to analysis. MapInfo used 1 standard deviation increments bounded by the minimum and maximum values of the sample to determine the rate categories.

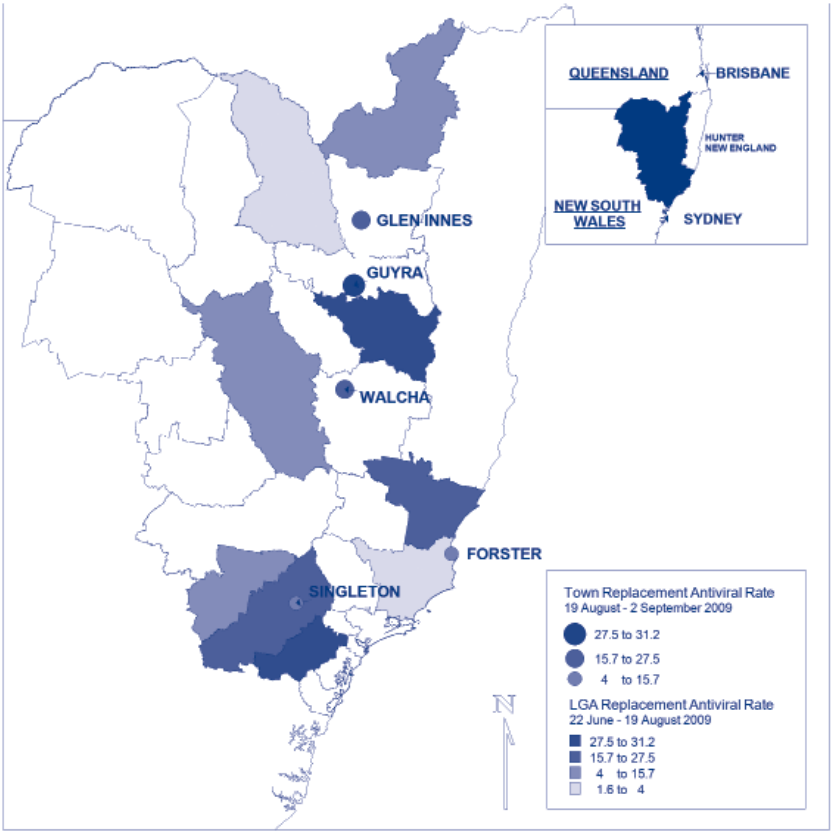
The mapping output was presented to the Area Health Service Pandemic Incident Controller and Divisions of general practice at regular operational planning meetings.

Results

The distribution of antivirals to rural HNE general practices (Figure 1) commenced on 22 June 2009 and by 2 July 2009, 9 working days later,

Map 1: Antiviral distribution in rural Hunter New England during the Protect Phase, 31 July 2009

Map 2: Antiviral distribution in rural Hunter New England during the Protect Phase, 2 September 2009



distribution of an initial antiviral pack had occurred to 82% (93/113) of rural HNE general practices; a total of 5,060 adult courses of antivirals. Following this, further initial antiviral packs were distributed, totalling 1,155 adult courses of antivirals.

The first distribution of replacement antivirals to rural HNE general practices occurred on 20 July 2009 and by 2 September 2009, 27 rural HNE general practices had been distributed replacement antivirals, representing a total of 455 adult courses. By 2 September 2009 a total of 6,670 adult courses of antivirals had been distributed to rural HNE general practices; approximately 5% of the antivirals distributed by the SVC in New South Wales.

Development and implementation of this pilot surveillance system occurred from 15 July to 2 September 2009. It delivered timely data on antiviral distribution, once replacement orders had been processed by the SVC. The system proved to be efficient with weekly data processing, statistical analysis and map generation taking less than 30 minutes.

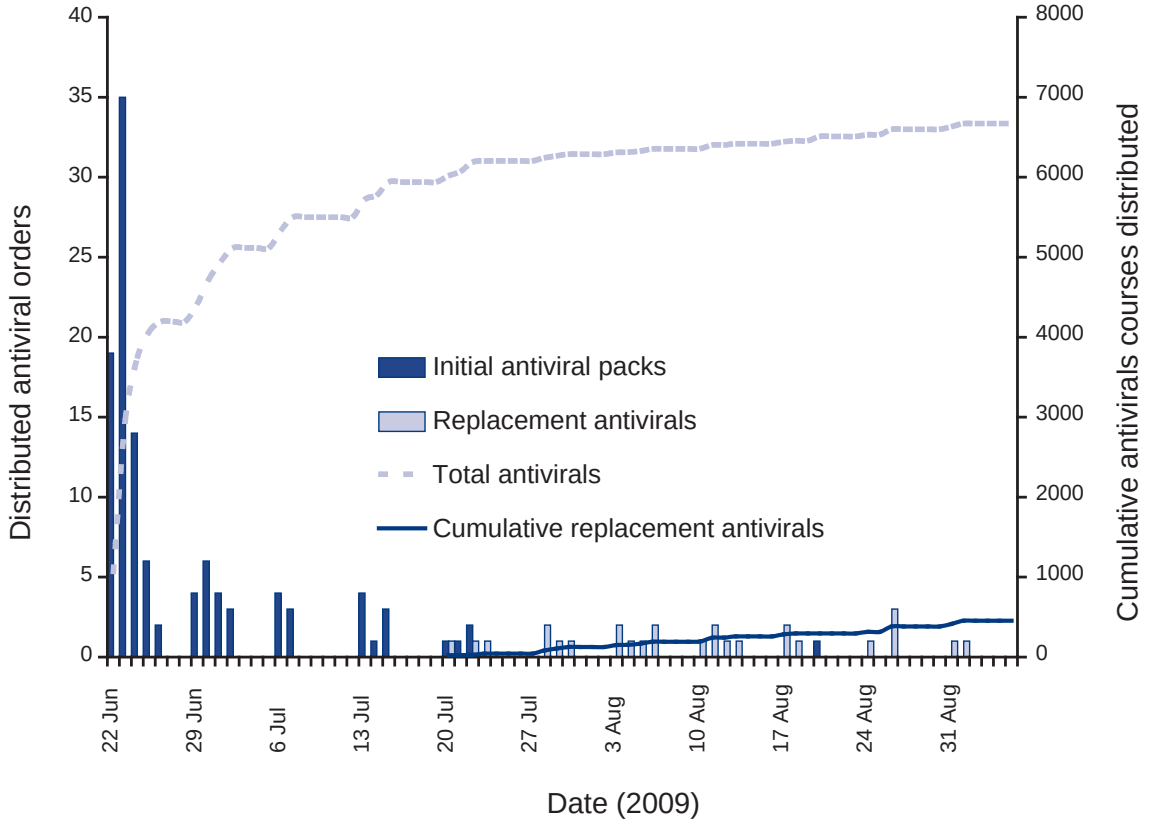
Recent change in ILI activity was noted on 31 July in Armidale, Cessnock, Taree, Muswellbrook and Singleton (Map 1). This result represented the first distribution of replacement antivirals to these towns. On 2 September, a recent change in ILI activity in Guyra was noted, with activity also prominent in Glen Innes, Walcha and Forster (Map 2). Singleton demonstrated a decrease in the replacement antiviral rate compared with the LGA replacement antiviral rate.

Discussion

This pilot surveillance system used existing administrative antiviral distribution data, which were available almost immediately. It was able to identify the spread and burden of ILI in vulnerable groups judged by GPs as requiring antiviral treatment within rural HNE. The system had similarities to a pilot syndromic surveillance system developed in Japan using over–the-counter influenza medication sales to describe influenza activity.[6] Both systems used pre-existing rapidly available data, which placed only a small burden on public health resources.

The described system was low cost. It used readily available software to generate visual activity maps, was easily customised to local needs, and was well received by health service planners. It

Figure 1: Antivirals distributed to rural Hunter New England general practices during the Protect Phase, 2 September 2009



could potentially be used for surveillance of any prolonged health emergency that requires distribution of a product.

Management of surveillance information during a pandemic is challenging because of the need to use multiple sources of imperfect data, with certain data items changed in response to the public health needs of an evolving pandemic.[7] Comprehensive influenza surveillance requires a number of complementary surveillance methods.[8] During pandemics, this may include 'conventional' methods used for seasonal influenza surveillance and 'novel' surveillance methods to more rapidly understand evolving situations and build more accurate 'surveillance pyramids'.[9] In addition to laboratory data and systems that capture ILI activity in health services, pandemic responses produce administrative data that can be used for surveillance. Intelligent use of all available data is essential to allow appropriate deployment of resources to best respond to demands on the health system.

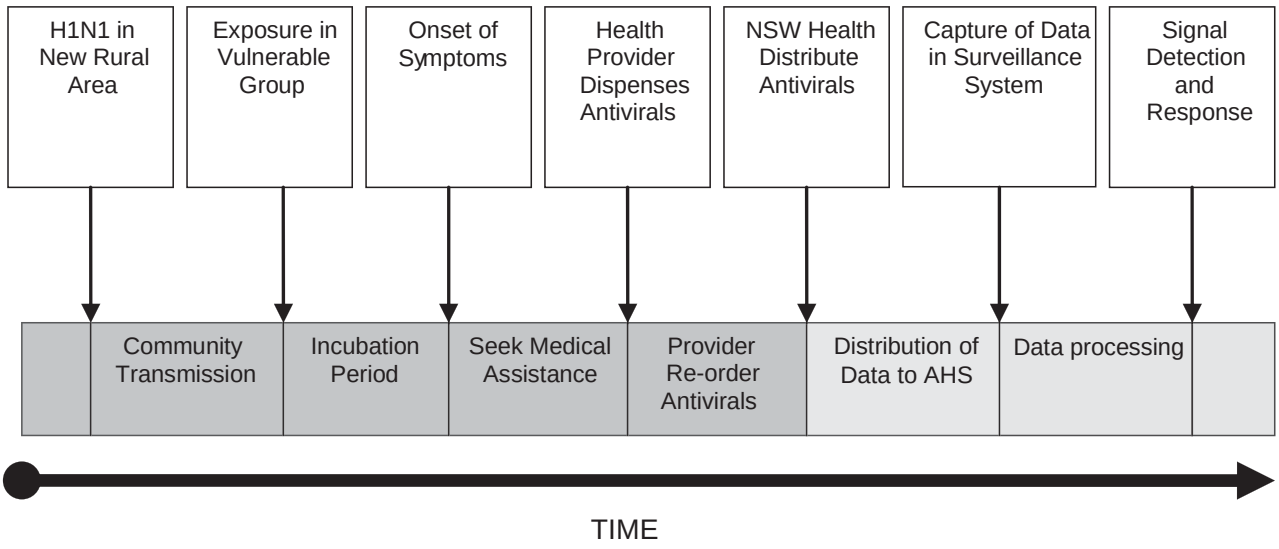
Syndromic surveillance (as defined by Henning)[10] has been used to detect outbreaks early; to monitor the size, geographic distribution, and evolution of outbreaks; to monitor disease trends; or provide reassurance that an outbreak has not occurred in high risk settings such as a mass event or following a natural disaster.[10–13] The piloted syndromic surveillance system, shows promise as a contributor to health system planning during appropriate emergency events.

The relatively large initial distribution of antivirals during the response to pH1N1 limited the sensitivity and evaluation of this piloted system. It is worth noting that the method of distribution chosen by NSW Health, through the SVC, appeared extremely efficient, with 82% of the initial antiviral medication distributed to HNE rural general practices occurring within 9 working days. Additionally, the relatively brief nature of Australia's first pandemic wave, with return to baseline influenza activity within 18 weeks,[14] meant that most general practices had dispensed little of the initial supply of antivirals and thus had not needed replacement antivirals. With a low number of general practices ordering replacement antivirals and no requirement to report progressive usage, the true antiviral usage remains unknown. The first replacement antivirals were distributed on 20 July, which was after the New South Wales peak of pH1N1 positive laboratory tests on 10 July 2009.[15]

Ideally the distribution of medical supplies should correspond closely to their demand, as this would ensure that adequate supplies of essential medicines are provided, prevent wastage of valuable resources and provide useful data for surveillance. If this is not possible, due to logistics or other reasons, then strict requirements for reporting of usage would ensure accountability and allow assessment of supply and demand. During this response the line-lists of patients who had received antivirals, which were required for ordering replacement antivirals, were unfortunately not collected or stored in a usable manner. Availability of these data would have allowed for a more sensitive and informative surveillance system as well as allowing for evaluation.

The contributors to completeness, representativeness and timeliness of the system are depicted in Figure 2. It is clear that personal knowledge of existing high risk medical conditions, community

Figure 2: Flow chart of contributors to completeness, representativeness and timeliness for antiviral distribution surveillance system



awareness and perceptions of disease severity, access to general practice, individual practitioners ability to assess patient risk and willingness to dispense antivirals, and availability of antivirals would all affect the representativeness and timeliness of the system. These factors would impact on any other PHCP surveillance system. The fact that the majority of rural HNE general practices were participating in the distribution of stockpiled antivirals to vulnerable groups provided reassurance of system coverage.

There is potential for further refining alert levels for individual towns and for establishing the ideal historical comparison period for each locality, factors that could not be adequately evaluated due to the study limitations. In addition, if used in a protracted emergency, the comparison LGA rates would need to be adjusted to reflect activity over a recent time period, rather than the entire emergency, to permit recognition of recent changes. The system was unable to be fully validated due to the absence of an appropriate dataset. Flutracking[16] data were unable to be used due to the relatively small number of participants within each LGA. Furthermore, due to laboratory capacity constraints, testing was scaled down in primary care (GPs) towards the end of the Delay Phase and restricted to individuals requiring hospitalisation. Thus laboratory test results did not allow validation either. We are not aware of any other dataset that would serve as a gold standard.

This syndromic surveillance system, as implemented during the Protect Phase, was biased towards ILI presentations of greater severity in higher risk groups and thus not fully representative of the introduction and spread of pandemic influenza into new populations and areas. Although it is theoretically possible to stratify by the estimated population prevalence of underlying risk factors, the primary purpose of the system was to monitor the burden on PHCPs thereby allowing rapid detection of increased activity that might require surging of emergency department or general practice resources. Thus this bias towards more severe presentations was a useful feature of the system.

Conclusions

The pilot study has demonstrated the concept used in this system has the potential to support rural health service planning during protracted health emergencies that require distribution of medical supplies. However, there is a need to gain further experience in other settings and during future events. Ideally, the distribution of medical supplies should correspond closely to their demand. This would ensure that adequate supplies of essential medicines are provided, prevent wastage of valuable resources and provide useful data for surveillance. This is certainly possible when an efficient logistic supplier, such as the SVC in New South Wales, is utilised. This type of system may complement other surveillance systems in tracking the epidemiology of a particular infectious disease threat and support planning at a local level.

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References

1. New influenza A (H1N1) virus: global epidemiological situation, June 2009. *Wkly Epidemiol Rec* 200919;84(25):249–257.

2. Australian Government, Department of Health and Ageing. Media Release from the Minister of Health and Ageing, Nicola Roxon, 17 June 2009. Available from: http://www.health.gov.au/internet/ministers/publishing.nsf/Content/mr-yr09-nr-nr082.htm?OpenDocument&yr=2009&mth=6

3. Australian Government, Department of Health and Ageing. Annex to the Australian Health Management Plan of Pandemic Influenza: Protect Phase. 2009.

4. SAS 9.1, SAS Institute Inc., SAS Campus Drive, Cary, North Carolina 27513, USA.

5. MapInfo 10.0, Pitney Bowes Software Inc, One Global View, Troy, New York,12180, USA.

6. Ohkusa Y, Shigematsu M, Taniguchi K, Okabe N. Experimental surveillance using data on sales of over-the-counter medications—Japan, November 2003–April 2004. *MMWR Morb Mortal Wkly Rep* 2005;54(Suppl):47–52.

7. Muscatello DJ, Cretikos MA, Bartlett MJ, Churches T, Carter IW, Eastwood K, et al. Planning for pandemic influenza surveillance in NSW. *N S W Public Health Bull* 2006;17(9–10):146–149.

8. Parrella A, Dalton CB, Pearce R, Litt JC, Stocks N. ASPREN surveillance system for influenza-like illness – A comparison with FluTracking and the National Notifiable Diseases Surveillance System. *Aust Fam Physician* 2009;38(11):932–936.

9. Armstrong P, Chant K. Preparing for the next influenza pandemic: a New South Wales perspective. *N S W Public Health Bull* 2006;17(7–8):114–117.

10. Henning KJ. What is syndromic surveillance? *MMWR Morb Mortal Wkly Rep* 2004;53(Suppl):5–11.

11. Hope K, Merritt T, Eastwood K, Main K, Durrheim DN, Muscatello D, et al. The public health value of emergency department syndromic surveillance following a natural disaster. *Commun Dis Intell* 2008;32(1):92–94.

12. Hope K, Durrheim DN, Muscatello D, Merritt T, Zheng W, Massey P, et al. Identifying pneumonia outbreaks of public health importance: can emergency department data assist in earlier identification? *Aust N Z J Public Health* 2008;32(4):361–363.

13. Hope K, Durrheim DN, d'Espaignet ET, Dalton C. Syndromic Surveillance: is it a useful tool for local outbreak detection? *J Epidemiol Community Health* 2006;60(5):374–375.

14. Bishop JF, Murnane MP, Owen R. Australia's winter with the 2009 pandemic influenza A (H1N1) virus. *N Engl J Med* 2009;361(27):2591–2594.

15. Progression and impact of the first winter wave of the 2009 pandemic H1N1 influenza in New South Wales, Australia. *Euro Surveill* 2009;14(42). Pii 19365.

16. Dalton C, Durrheim D, Fejsa J, Francis L, Carlson S, d'Espaignet ET, et al. 'Flutracking: a weekly Australian community online survey of influenza-like illness in 2006, 2007 and 2008.' *Commun Dis Intell* 2009;33(3):316–322.

Evaluating the utility of Emergency Department syndromic surveillance for a regional public health service

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Abstract

Communicable disease monitoring and response activities must be based upon local public health surveillance systems, even during infectious disease emergence, natural disasters, and during bioterrorism events. The NSW Department of Health has developed an emergency department surveillance system intended to monitor important public health conditions during mass gatherings and to identify outbreaks of importance. An evaluation of this system conducted in the Hunter New England region of New South Wales emphasised its usefulness when it was focused on a limited number of syndromes of public health importance and during mass gatherings and public health disaster responses. *Commun Dis Intell* 2010;34(3):310–318.

Keywords: syndromic surveillance, disease surveillance, public health evaluation

Introduction

During the past 2 decades, biological and other terrorism incidents, have resulted in health authorities globally investing considerable resources into implementing syndromic surveillance that identifies outbreaks of public health importance earlier than traditional surveillance systems.[1–3] However there is currently limited evidence that syndromic surveillance systems add value to local public health disease surveillance.[4–7]

The real-time evaluation of an emergency department (ED) syndromic surveillance system in Taiwan identified peaks in seasonal community-wide illness, such as influenza and gastroenteritis.[3] In addition, a syndromic surveillance system developed for the military in French Guiana identified a dengue fever outbreak 3 weeks earlier than traditional surveillance, permitting early implementation of control measures.[8] However, the ability to detect localised disease clusters remains an elusive goal for many syndromic surveillance systems.[4,5–9]

Syndromic surveillance

Syndromic surveillance generally relies on recognising clinical features that are discernible before a diagnosis is confirmed. Data sources used include ED primary diagnoses, ambulance dispatches and sales of over-the-counter medication.[10] Unlike traditional surveillance systems, which may rely on voluntary passive reports from health practitioners, syndromic surveillance systems rely on continual data acquisition generated by routine activities that are generally electronically transmitted.[2] While many syndromic surveillance systems are focused on detecting bioterrorism events, it is important that these are integrated into routine health surveillance to ensure staff become familiar with them and know how to interpret signals.

New South Wales real-time public health emergency department surveillance system

In 2003, the Centre for Epidemiology and Research developed a real-time public health emergency department surveillance system in response to biosecurity threats associated with the Rugby World Cup.[11] Currently, over 50 EDs provide data to the system; eight are situated within the Hunter New England (HNE) area in northern New South Wales. HNE currently has 38 EDs, ranging from 1 large tertiary referral hospital to small general practitioner-serviced multi-purpose centres. The 8 hospitals include the tertiary referral hospital and one of the 2 rural referral hospitals in the region.[12]

The NSW Department of Health (NSW Health) receives 4 hourly electronic file transmission from participating EDs (using HL7 messaging) containing data items including the primary ED diagnoses (ICD-10 codes), which are mapped to one of 39 syndrome categories.[11]

Statistical control charts are used to automatically detect increases in syndrome activity (signals) based on the previous 51 weeks' data. The measures used to detect increased activity include:

1. when a specific day's ED presentations exceed the average number of presentations for that day for the previous 51 weeks, by more than 5 standard deviations;

2. when a specific week's ED presentations exceed the average number of presentations for the previous 51 weeks, by more than 5 standard deviations and

3. when a 15-fold increase occurs in the modified cumulative sum. This is called the Index of increase.

The cumulative sum is calculated by cumulatively summing the difference between the previous day's count and the count 7 days prior. The accumulated cumulative sum is divided by the mean syndrome count for the available baseline up to a maximum 365 days. The mean-standardised cumulative sum is then divided by the standard deviation of its differenced values, again using whatever baseline is available up to a maximum of 365 previous days.[11,13]

Regional public health services

New South Wales public health units are legally mandated to monitor and respond to infectious disease and other public health threats.[14] This is facilitated by notification of specific diseases by laboratories, doctors and institutions.[15] To enhance regional surveillance, the ED syndromic surveillance system would need to identify these specific diseases earlier or more completely than the traditional notification systems, or identify other disease threats not covered by existing surveillance.

The regional objectives identified by Hunter New England Population Health (HNEPH) prior to the evaluation were:

a. routine monitoring to identify cases or clusters of public health importance requiring a prompt response, without duplicating existing surveillance systems; and

b. during emergency situations or mass gathering events, to actively monitor ED presentations for situational awareness.

While the system has met state level surveillance objectives,[11,13,16] the current evaluation aimed to determine the utility of the New South Wales ED syndromic surveillance in a regional public health service.

Methods

The US Centers for Disease Control and Prevention 'framework for evaluating public health surveillance system for early detection of outbreaks' was used to guide the evaluation process.[17] Some system attributes, including algorithms, information technology platform and syndrome groupings were not assessed during this evaluation. Outbreak detection was assessed prospectively for all syndromes and retrospectively for a subset of syndromes classified as syndromes of public health importance.

Prospective signals produced by the ED surveillance system were investigated to determine if they represented an outbreak; while retrospectively, signals were compared against known outbreaks reported through existing surveillance systems. System experience including the usefulness, flexibility and acceptability of the system was assessed through a stakeholder survey during the prospective evaluation component.

Prospective evaluation

Each weekday (excluding public holidays) between 1 January and 31 December 2008 the ED surveillance reports for participating HNE hospitals were accessed and any syndrome signal noted. The date of signal; syndrome; type of signal (daily, weekly, index of increase); hospital; threshold level; standard deviation of the signal (i.e. signal strength) and whether it was the first signal for that hospital and syndrome in that week, were systematically documented. In addition, the investigation description, whether an outbreak was confirmed and the resulting outbreak response, were also recorded.

An investigation hierarchy was used with each progression involving a more in depth public health response. The initial investigation was labelled a 'subgroup' investigation which involved reviewing age, sex, admission and triage categories of patients that had elicited the signal. If a subgroup investigation showed clustering by age, sex, location or admission status, an 'internal' investigation was conducted. An internal investigation involved a public health unit (PHU) staff member reviewing the ED patient database or pathology database to ascertain further information on demographic characteristics, clinical assessment and pathology results of patients. If warranted an 'external' investigation was then conducted, involving PHU staff contacting ED directors or treating physicians to ascertain further patient-specific clinical or epidemiological information. The time taken was also recorded.

Syndromes of public health interest

A reference group consisting of surveillance, public health and epidemiology staff reviewed the list of ED syndromes and selected a syndrome subset, classified as syndromes of public health interest, based on: their severity or public health consequence; potential for large outbreaks; limitations of traditional surveillance; and local disease epidemiology. The syndromes selected were: 'meningitis', 'pneumonia', 'influenza', 'gastrointestinal' and 'poisoning'. Signals and investigations were however, recorded for all syndromes.

Retrospective evaluation

Standard ED surveillance reports for 2005–2007 were generated for the syndromes; 'gastrointestinal', 'influenza', 'pneumonia' and 'meningitis', and all signals identified. Reports for selected syndromes were also generated by a restricted age grouping (5–65 years) and 'admission to hospital' status and all signals identified. The signals were compared to known outbreaks recorded in 3 existing surveillance systems; notifiable diseases, OzFoodNet foodborne outbreaks and the institutional gastrointestinal and respiratory outbreak databases.

Stakeholder survey

A questionnaire was sent to local stakeholders including ED directors, PHU and laboratory staff, and senior managers who received NSW Health emails reporting syndrome signals. Participants were asked how often they read these emails, whether they had taken any action in response to an email, the outcome of any action, and their preference for future email alerts. This self-administered questionnaire was sent and returned electronically, with prompting by a senior PHU staff member after a month if a response had not been forthcoming.

Results

Prospective evaluation

All syndromes

During 2008, a total of 958 signals occurred across the 8 EDs; 237 daily signals, 467 weekly signals and 254 index of increase signals (Table 1). Elevated counts in 1 syndrome could result in signals over multiple days and across multiple categories (daily, weekly, index of increase). When repeat signals were ignored, there were 382 initial signals by syndromes.

Overall, 366 (38%) signals were investigated to the sub-group level with an internal investigation necessary for 25 (3%) and an external investigation on 6 occasions (0.6%). The remaining signals were not investigated. No HNE outbreaks were detected by the ED surveillance system during 2008, while existing surveillance systems detected 17 gastroenteritis outbreaks and 9 meningococcal cases were notified during this period.

The 6 external investigations included four for meningitis and one each for gastrointestinal and pneumonia syndromes. Of the 4 meningitis syndrome signals that led to an external investigation, 1 viral meningitis cluster was identified by the ED surveillance system with additional information concurrently received through existing surveillance systems. One suspected bacterial meningitis case was identified, which had not been notified through traditional surveillance but was subsequently proven not to be meningococcal disease. None of the 9 meningococcal disease notifications were identified by the ED surveillance system but four had a purely septicaemic presentation and would likely not be identified as a meningitis syndrome.

The single gastrointestinal and pneumonia signals were investigated externally with no outbreak detected. However, the pneumonia signal external investigation resulted in a better understanding of ED coding practices with only two of the 6 presentations subsequently confirmed as pneumonia cases.

Of the 3 poisoning signals investigated internally, two were chemical exposures requiring attendance by the Fire Department's Hazardous Materials Response Team but both had already been notified through regular emergency communication channels to the PHU. The signals however demonstrate the ED surveillance system's ability to identify acute events requiring ED presentation due to chemical exposures.

The initial signal review took a median time of 15 minutes (range 2–20 minutes). Where further investigation was required, the time required ranged from a 10 minute telephone consultation with a physician to 4 hours checking clinical details and laboratory results.

Fourteen per cent (143 of 958) of signals related to a syndrome of public health interest. All 2008 external investigations related to one of these 5 syndromes.

Table 1: Summary of signals produced by the New South Wales emergency department syndromic surveillance system for Hunter New England Area Health Service participating emergency departments, 1 January to 31 December 2008

Syndromes	Alerts in standard reports					Investigation		
	Total	Initial	Daily	Weekly	Index	Sub-group†	Internal‡	External§
All syndromes	958	412	237	467	254	366	25	6
Syndromes public health interest	143	76	45	85	11	100	22	6
Abdominal pain	2	2	0	2	0	1	0	0
Convulsions (not clearly epilepsy)	7	7	6	1	0	7	0	0
Collapse/ syncope/coma/ delirium/ dizziness	0	0	0	0	0	0	0	0
Neuromuscular/ vision problems	19	16	14	5	0	16	2	0
Cough	24	17	13	7	4	13	0	0
Headache/ migraine	2	2	2	0	0	2	0	0
Malaise/fatigue	30	19	7	21	2	18	0	0
Meningitis/ encephalitis*	18	15	6	5	5	17	10	4
Dehydration*	11	11	10	1	0	11	0	0
Gastrointestinal	6	5	3	3	0	6	3	1
All cardiovascular/ chest pain	1	1	1	0	0	1	0	0
Chest pain	0	0	0	0	0	0	0	0
Cardiac arrest	9	7	6	3	0	8	0	0
Cardiac dysrhythmias	3	3	2	1	0	3	0	0
All respiratory diagnoses	175	29	2	53	120	27	0	0
Asthma	16	6	3	7	6	6	0	0
Influenza	78	29	14	58	6	42	2	0
Pneumonia	23	13	10	13	0	20	4	1
Other/unspecified respiratory infections	106	31	11	58	37	29	1	0
Respiratory failure/distress	3	3	2	1	0	0	0	0
Bronchiolitis	51	23	11	30	10	13	0	0
All injury diagnoses	7	6	1	4	2	0	0	0
Joint injury	17	6	2	9	6			
Head injury	5	3	2	3	0	2	0	0
Burns	6	6	3	2	1	6	0	0
Bite or sting (insect/spider/ snake)	158	53	47	92	19	15	0	0
Open wounds	9	1	0	7	2	0	0	0
Hypothermia*	9	3	1	7	1	5	0	0
Heat stroke*	12	6	0	7	5	5	0	0
Illicit drugs diagnoses	8	8	7	0	1	4	0	0
Alcohol acute effects	15	13	11	4	0	8	0	0
Unspecified infection	26	15	6	14	6	9	0	0
Skin problems	24	9	3	17	4	17	0	0
Poisoning (not illicit drug or alcohol)	18	13	12	6	0	15	3	0
Mental health diagnosis	3	2	2	1	0	2	0	0
Admitted to critical care unit	0	0	0	0	0	0	0	0
Death in emergency department	10	10	9	0	1	5	0	0
Triage one	11	8	4	7	0	6	0	0
Ambulance arrival	0	0	0	0	0	0	0	0
All unplanned visits	38	2	4	18	16	2	0	0

* Syndromes added or altered in August 2008.

† When a signal occurred the emergency department syndromic surveillance system, then produces a breakdown page by subgroup, using the same algorithms.

‡ Entailed a public health unit staff member accessing the emergency department patient database, the pathology database or NetEpi.

§ When a public health unit staff member contacted a person external to the public health unit such as an emergency department director or treating physician

Retrospective evaluation

The retrospective analysis identified 1 pneumonia signal, which was associated with a confirmed outbreak, and an increase in influenza and gastroenteritis signals during the winter and spring months respectively, but no other outbreaks were identified.

The pneumonia outbreak was first reported on 10 August 2006 by a hospital paediatrician who noted an unusual pneumonia cluster in young, previously healthy males.[18] The ED surveillance system signalled in the 'pneumonia' syndrome 3 days later. However, by stratifying the pneumonia syndrome by age group, a signal appeared in the 5–16 year age group 4 days earlier than the paediatrician's notification and this signal was maintained for 14 days. This signal would not have been identified by current ED standard reports.[13] Thirty-five of the 69 pneumonia signals during 2008 occurred in this age group.

Influenza signals were increased (158) during June to September in 2007 compared with 2006 (78) and 2008 (78). The increases were predominantly in 2 non-metropolitan EDs but no specific outbreak was identified and existing surveillance systems indicated 2007 was generally a more severe influenza season than 2006 and 2008.[19] The influenza syndrome ICD-10 codes were used infrequently by all HNE EDs. In HNE during 2008, counts at individual EDs ranged from nine to 57 with a median of 13.

Most gastroenteritis syndrome signals between 2005 and 2007 occurred in 2 hospitals, both being referral centres for children. The majority of alerts occurred during September and October each year, but this trend was absent in 2008 (Table 2). There was no clear association between known outbreaks and ED surveillance signals (Table 2). However, information captured in the OzFoodNet outbreak investigation database indicated that very few known outbreak cases presented to hospital.

Stakeholder survey

Six of the 7 ED directors returned completed questionnaires. Only one of the 6 ED directors surveyed always read the alert emails, others responded that they only did so intermittently depending on workload. None of the ED directors reported taking any action from the alerts unless the PHU contacted them.

Six of the 10 public health staff surveyed returned completed questionnaires. All six reported utilising the surveillance data during mass events and disaster responses, 67% indicated they had used the surveillance data to inform public health action, while all indicated they still wanted to receive the alert emails, with five only wanting to know about a restricted number of syndromes.

Table 2: Comparison of gastrointestinal outbreak notifications in institutions, OzFoodNet notifications and emergency department syndromic surveillance signals, 2006 to 2008

	2008			2007			2006		
	Institutions	OzFoodNet	ED signal	Institutions	OzFoodNet	ED signal	Institutions	OzFoodNet	ED signal
Jan	4	3	1	5	1	0	2	1	3
Feb	3	1	0	4	2	0	3	1	0
Mar	5	1	0	6	0	0	6	1	0
Apr	4	1	0	6	1	0	7	1	0
May	10	0	0	6	0	0	11	0	1
Jun	5	0	0	5	0	0	12	4	4
July	7	0	0	9	2	0	19	0	0
Aug	17	1	3	18	0	2	13	0	18
Sept	12	0	0	9	1	1	4	2	56
Oct	5	0	0	20	1	69	10	2	26
Nov	7	2	0	14	0	12	6	0	0
Dec	4	1	3	10	1	2	6	2	0

ED Emergency department.

Additional applications

Acute events surveillance

Severe storms and extensive flooding occurred in the Hunter Valley during the June 2007 long weekend (Friday 8 to Monday 11 June) resulting in the region being declared a natural disaster area.[20] No gastroenteritis outbreaks were identified by the ED surveillance system during the recovery phase. The system detected increased presentations of respiratory syndromes, which were within seasonally expected levels when compared with data for the previous 5 years. The ED data informed response planning, while providing reassurance that there were no large infectious disease outbreaks threatening the health of the disaster affected population.[21] The surveillance system acquired data on all presentations to EDs, allowing consideration of additional syndromes or conditions not included in the standard surveillance reports. This occurred during the 2007 Hunter storms when information was provided on hypothermia presentations to local EDs. Hypothermia was subsequently added to the standard reports in 2008.

Mass gatherings surveillance

The Tamworth country music festival is a mass gathering occurring for 2 weeks each January, ending on the Australia Day long weekend, with a doubling of the Tamworth population from 40,000 to over 80,000 people. Temporary camping facilities accommodate the influx of people, and many transitory food vendors cater for the crowd. Enhanced ED surveillance through the New South Wales ED syndromic surveillance system has been used during the festival since 2007 with data reported daily to the local disaster emergency management team. Signals have prompted public health investigations, for example clusters of otitis externa and respiratory illness in 2007, informed workforce planning and assisted in prioritising public health activity and media messages.

Discussion

The New South Wales real-time emergency department surveillance system was evaluated from the perspective of a regional PHU for its capacity to identify cases or clusters of public health importance requiring a prompt response, without duplicating existing surveillance systems; and for enhanced surveillance during emergency situations or mass gathering events for situational awareness.

The ED surveillance system is potentially a useful tool to assist with situational awareness, particularly during natural disasters and mass gathering events. Recent experience in HNE has demonstrated the value of ED syndromic surveillance in both these circumstances. The recent H1N1 pandemic also established the value of the ED syndromic surveillance system in monitoring statewide demand on ED services during a prolonged public health emergency.[22] The system is flexible, allowing for adding or adapting syndromes in response to changing situations, and accommodates tight reporting time frames. ED surveillance data have informed health messages for the media and guided response planning. Previous reviews of the use of syndromic surveillance systems during acute events support their efficacy.[23–25]

This study identified potential utility for this surveillance system to detect public health threats requiring prompt intervention for a few specific syndromes. During 2008, only six of 958 signals across 8 HNE EDs required further public health intervention. The prospective evaluation of the 39 syndromes provided empirical support for focusing on only 5 syndromes (gastrointestinal, meningitis, pneumonia, influenza and poisoning). If daily monitoring had been restricted to these syndromes then there would have been substantially fewer signals (143 in table versus 958) to investigate. System acceptability and representativeness could be improved by including the second rural referral hospital in HNE and by investigating the specific surveillance needs of ED staff.

Outbreaks are relatively rare events and their severity determines whether there are ED presentations. There is no gold standard of outbreak identification to which to compare syndromic surveillance, as existing surveillance systems themselves do not detect all outbreaks even with a delay. This complicates the determination of the sensitivity and specificity of the ED syndromic surveillance system. During the prospective evaluation, 1 viral meningitis cluster was detected by the ED surveillance system, concurrently with existing surveillance systems. In addition, the system did not identify any outbreaks during the Hunter 2007 storms or during mass gathering events in the region, consistent with results from existing surveillance.

The single pneumonia outbreak indicated that although the syndrome may not signal earlier than reporting by an astute clinician, a narrower age-band excluding the 'noise' generated by the very young and old, may be a more sensitive measure of unusual respiratory outbreak activity. For example, during the second wave of an

influenza pandemic, when different age groups may be hospitalised compared with those usually affected by seasonal influenza.

Past evaluations of emergency department syndromic surveillance systems have found that they can detect community wide outbreaks, such as seasonal influenza and gastroenteritis, however their effectiveness in identifying smaller clusters of interest to local PHUs has not been established.[4–7] When known gastrointestinal outbreaks investigated by OzFoodNet and institutional outbreaks reported to the HNE PHU were compared to the gastrointestinal syndrome signals, no known outbreaks were identified by the ED surveillance system. The OzFoodNet database did indicate that a restricted number of people had presented to EDs, however the high level of 'background noise' and fixed threshold level reduced the likelihood of a signal being generated. Similarly, none of the notified cases of meningococcal disease in 2008 resulted in a signal.

While detection of the influenza and gastroenteritis season commencement has value at a state level for providing information on severity and spread and to inform media releases, such seasonal trends are of less value at the local level where outbreak detection and investigation are the priority.

While local objectives were developed for this evaluation, it is important that specific operational objectives are established for the ED surveillance system to guide reporting and investigation of signals. As each syndrome represents a separate disease or condition it may be necessary for each syndrome to have its own surveillance objectives describing local public health relevance (Box). Clear regional objectives may also assist in engaging EDs in the reporting process, as the benefit of investing time may become more evident. ED surveillance objectives should complement existing surveillance systems rather than duplicate efforts.

Limitations

During the evaluation, the ED standard reports were generally only monitored on weekdays; reports from Fridays and Saturdays were not reviewed until Monday, except during the declared disaster and mass event monitoring. This could have lead to delays in event detection. The retrospective reproduction of the ED standard reports may differ slightly to reports that were produced in real time. This is due to higher data completeness in the retrospective data compared to the real-time data.

Box: Examples of surveillance objectives

Objectives for pneumonia syndrome

- Identify unusual clusters of pneumonia that require a rapid response, without duplicating existing surveillance systems (e.g. *Legionella*)
- Identify bioterrorism event

Objectives for meningitis/encephalitis syndrome

- Identify unusual clusters of viral meningitis or encephalitis, to ensure appropriate testing

Objectives for monitoring poisoning syndrome

- Identify chemical exposures requiring response
- Identify foodborne poisoning events requiring response

Objectives for gastrointestinal syndrome

- Identify unusual clusters of severe gastrointestinal disease that require an acute and timely response, without duplicating existing surveillance systems

Objectives during acute events/disaster response

- Identify clusters or increasing trends in presentations of public health importance that require a rapid response

During this evaluation it was not possible to adequately measure the 'cost-effectiveness' of the ED syndromic surveillance system. While PHU staff time required to follow-up on signals was recorded, information on the cost of setting up and maintaining the system was not available to the researchers. Therefore it is not possible to determine whether the costs of providing situational awareness and community reassurance are justified, nor can it prospectively be determined if the system will repay its running costs by averting a major disaster.

The stakeholder survey conducted as part of this evaluation only consisted of a small number of participants; which limits the ability to generalise the findings. When applying these results to other regions it is important to consider that ED patient management systems and coding practices may vary across EDs and regions. Therefore the performance of specific syndromes may differ between hospitals and regions.

Conclusion

ED syndromic surveillance may inform local public health action or serve as a surveillance safety net for traditional surveillance when focused on pneumonia, meningitis/encephalitis, poisoning and possibly gastrointestinal syndromes. It appears to have specific local utility during mass gathering or disaster response surveillance. Clear objectives for each syndrome are needed, emphasising the difference between local and state surveillance objectives and variability between syndromes. A handbook of response options may prove valuable in guiding the response to specific syndromic surveillance signals.

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References

1. Reis B, Mandl K. Syndromic surveillance: the effects of syndrome grouping on model accuracy and outbreak detection. *Ann Emerg Med* 2004;44(3):235–241.

2. Mandl K, Overhage J, Wagner M, Lober W, Sebastiani P, Mastashari F, et al. Implementing syndromic surveillance; a practical guide informed by the early experience. *J Am Med Inform Assoc* 2004;11(2):141–150.

3. Wu T, Shih F, Yen M, Wu J, Lu S, Chang K, et al. Establishing a nationwide emergency department-based syndromic surveillance system for better public health responses in Taiwan. *BMC Public Health* 2008;8:18.

4. Hope K, Durrheim D, D'Espaignet ET, Dalton C. Syndromic surveillance: is it a useful tool for local outbreak detection? *J Epidemiol Community Health* 2006;60(5):374–375.

5. Bravata DM, McDonald KM, Smith WM, Rydzak C, Szeto H, Buckeridge DL, et al. Systematic review: surveillance systems for early detection of bioterrorism-related diseases. *Ann Intern Med* 2004;140(11):910–922.

6. McLeod M, Mason K, White P, Read D. The 2005 Wellington influenza outbreak: syndromic surveillance of Wellington Hospital Emergency Department activity may have provided early warning. *Aust N Z J Public Health* 2009;33(3):289–294.

7. Wallace DJ, Arquilla B, Heffernan R, Kramer M, Anderson T, Bernstein D, et al. A test of syndromic surveillance using a severe acute respiratory syndrome model. *Am J Emerg Med* 2009;27(4):419–423.

8. Meyard J, Chaudet H, Texier G, Ardillon V, Ravachol F, Deparis X, et al. Value of syndromic surveillance within the armed forces for early warning during a dengue fever outbreak in French Guiana in 2006. *BMC Med Informatics Decision Making* 2008;8:29–38.

9. Buckeridge D. Outbreak detection through automated surveillance: a review of the determinants of detection. *J Biomedical Informatics* 2007;40:370–379.

10. Centers for Disease Control and Prevention. Syndromic Surveillance: reports from a national conference, 2004. *MMWR Morb Mortal Wkly Rep* 2005;54(Suppl):1–180.

11. Muscatello D, Churches T, Kaldor J, Zheng W, Chiu C, Correll P, et al. An automated, broad-based, near real-time public health surveillance system using presentations to hospital Emergency Departments in New South Wales, Australia. *BMC Public Health* 2005;5:141–152.

13. Hunter New England Area Health Service. *HNE services and facilities*. Accessed on 10 February 2010. Available from: http://www.hnehealth.nsw.gov.au/services_and_facilities

14. Hope K, Durrheim D, Muscatello D, Merritt T, Zheng W, Massey P, et al. Identifying pneumonia outbreaks of public health importance: can emergency department data assist in earlier identification? *Aust N Z J Public Health* 2008;32(4):361–363.

15. New South Wales Government. *NSW Public Health Act 1991*. Accessed on 10 February 2010. Available from: http://www.austlii.edu.au/au/legis/nsw/consol_act/pha1991126/

16. NSW Department of Health. *Notification of Infectious Diseases under the Public Health Act 1991*. 2006. Policy directive PD2006_014. Accessed on 10 February 2010. Available from: http://www.health.nsw.gov.au/policies/pd/2006/pdf/PD2006_014.pdf

17. Zheng W, Aitken R, Muscatello DJ, Churches T. Potential for early warning of viral influenza activity in the community by monitoring clinical diagnoses of influenza in hospital emergency departments. *BMC Public Health* 2007;7:250.

18. Buehler J, Hopkins R, Overhage J, Sosin D, Tong V, CDC Working Group. Framework for evaluating public health surveillance systems for early detection of outbreaks: recommendations from the CDC working group. *MMWR Recomm Rep* 2004;53(RR–5):1–11.

19. Cashman P, Massey P, Durrheim D, Islam F, Merritt T, Eastwood K. Pneumonia cluster in a boarding school – implications for influenza control. *Commun Dis Intell* 2007;31(3):296–298.

20. Dalton C, Durrheim D, Fejsa J, Francis L, Carlson S, d'Espaignet ET, et al. Flutracking: A weekly Australian community online survey of influenza-like illness in 2006, 2007 and 2008, *Commun Dis Intell* 2009;33(3):316–322.

21. Cretikos MA, Merritt TD, Main K, Eastwood K, Winn L, Moran L, et al. Mitigating the health impacts of a natural disaster— the June 2007 long-weekend storm in the Hunter region of New South Wales. *Med J Aust* 2007;187(11–12):670–673.

22. Hope K, Merritt T, Eastwood K, Main K, Durrheim D, Muscatello D, et al. The public health value of emergency department syndromic surveillance following a natural disaster, *Commun Dis Intell* 2008;32(1):92–94.

23. Churches T, Conaty SJ, Gilmour RE, Muscatello DJ. Reflections on public health surveillance of pandemic (H1N1) 2009 influenza in NSW. *N S W Public Health Bull* 2010;21(1–2):19–25.

24. Toprani A, Ratard R, Straif-Bourgeois S, Sokol T, Averhoff F, Brady J, et al. Surveillance in hurricane evacuation centers—Louisiana, September–October 2005. *MMWR Morb Mortal Wkly Rep* 2006;55(2):32–35.

25. Steiner-Sichel L, Greenko J, Heffernan R, Layton M, Weiss D. Field investigations of emergency department syndromic surveillance signals—New York City. *MMWR Morb Mortal Wkly Rep* 2004; 3(Suppl):184–189.

26. Carrico R, Goss L. Syndromic surveillance: hospital emergency department participation during the Kentucky Derby Festival. *Disaster Manag Response* 2005;3(3):73–79.

A new national chlamydia sentinel surveillance system in Australia: Evaluation of the first stage of implementation

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Abstract

The Australian Collaboration for Chlamydia Enhanced Sentinel Surveillance (ACCESS) was established with funding from the Department of Health and Ageing to trial the monitoring of the uptake and outcome of chlamydia testing in Australia. ACCESS involved 6 separate networks; 5 clinical networks involving sexual health services, family planning clinics, general practices, antenatal clinics, Aboriginal community controlled health services, and 1 laboratory network. The program ran from May 2007 to September 2010. An evaluation of ACCESS was undertaken in early 2010, 2 years after the program was funded. At the time of the evaluation, 76 of the 91 participating sites were contributing data. The jurisdictional distribution of the 76 sites generally matched the jurisdictional distribution of the Australian population. In 2008, the chlamydia testing rates in persons aged 16–29 years attending the 26 general practices was 4.2% in males and 7.0% in females. At the 25 sexual health services, the chlamydia testing rates in heterosexuals aged less than 25 years in 2008 was 77% in males and 74% in females. Between 2004 and 2008, the chlamydia positivity rate increased significantly in heterosexual females aged less than 25 years attending the sexual health services, from 11.5% to 14.1% ($P < 0.01$). Data completeness was above 85% for all core variables except Aboriginal and/or Torres Strait Islander status and country of birth, which ranged from 68%–100%, and 74%–100%, respectively, per network. There were delays in establishment of the system due to recruitment of 91 sites, multiple ethics applications and establishment of automated extraction programs in 10 different database systems, to transform clinic records into a common, pre-defined surveillance format. ACCESS has considerable potential as a mechanism toward supporting a better understanding of long-term trends in chlamydia notifications and to support policy and program delivery. *Commun Dis Intell* 2010;34(3):319–328.

Keywords: chlamydia, sentinel surveillance, Australia

Introduction

The primary role of public health surveillance is to guide the planning and evaluation of policy and programs, through the collection, analysis and interpretation of statistical information. In Australia, the main form of chlamydia surveillance is passive reporting of cases to health departments by doctors or laboratories.[1] Passive surveillance has shown *Chlamydia trachomatis* to be the most commonly notified infection in Australia with rates having risen nearly 4-fold in the past decade.[1]

Passive surveillance has a natural appeal, in that it can be established on an ongoing basis, provides full geographic coverage and does not involve substantial programmatic expense. On the other hand, passive surveillance may be biased by testing patterns, as indicated by the strong correlation between the number of diagnoses and number of tests.[2–4] Also, notification data do not routinely include information on characteristics such as gender of sex partner and in several jurisdictions are far from complete with regard to indigenous status.[5]

A supplementary approach to surveillance is the use of selected clinical sites to collect systematic data on uptake and the outcome of chlamydia testing. Such data can be used to evaluate clinic-based initiatives, broader prevention programs and help interpret trends in passive surveillance.[6,7]

In May 2007 the Australian Government Department of Health and Ageing (DoHA) funded the trialling of a new national sentinel surveillance system, entitled the Australian Collaboration for Chlamydia Enhanced Sentinel Surveillance (ACCESS) (www.access-study.org), based on 5 networks of clinical sites and a laboratory network. This paper reports on an evaluation of this new system.

Methods

This evaluation of ACCESS was conducted from 1 January to 31 March 2010 by project staff, using the framework promulgated by the US Centers for Disease Control and Prevention.[8] Specific goals were to:

1. assess the simplicity, flexibility, acceptability, timeliness, stability, validity, usefulness and representativeness of ACCESS;

2. assess the data quality of the system by examination of the first 12 months of data collection; and

3. make recommendations to improve the system.

For each ACCESS variable, completeness was the proportion of entries that were not missing or unknown. To determine representativeness, the proportion of ACCESS sites per jurisdiction was compared with the proportion of the population in the jurisdiction. The ratio of metropolitan and non-metropolitan ACCESS sites was also compared with this ratio for the Australian population. Population data were accessed from the 2006 Australian Bureau of Statistics data.[9] Other surveillance attributes were assessed qualitatively through feedback from other ACCESS members and select partners, ACCESS meeting Minutes and quarterly progress reports submitted to DoHA.

Results

Description of ACCESS

ACCESS was established as a collaboration between the National Centre in HIV Epidemiology and Clinical Research (NCHECR) and the Perinatal and Reproductive Epidemiology and Research Unit at the University of New South Wales (UNSW), the Burnet Institute's Centre for Population Health, and the National Serology Reference Laboratory (NRL). UNSW (via NCHECR) and the Burnet Institute are jointly responsible for ACCESS. Other partners are the participating sentinel sites (Appendix), the National Aboriginal Community Controlled Health Organisation (NACCHO) and its state and territory based affiliates; and DoHA as funding agency.

The overall aim of ACCESS is to establish a sentinel surveillance system to evaluate the impact of interventions to control genital chlamydia infection. Specific objectives were to:

1. establish 6 separate surveillance networks, each providing unique information on chlamydia testing;

2. enhance the data management systems of sentinel sites with a view to routinely sending chlamydia surveillance data to a central location;

3. monitor the extent of chlamydia testing at these sites;

4. determine the chlamydia positivity in priority populations; including young heterosexuals (< 25 years), men who have sex with men (MSM), Aboriginal and/or Torres Strait Islander people, pregnant women and sex workers; and

5. interpret trends determined by other chlamydia surveillance mechanisms.

ACCESS involves 5 clinical networks made up of sexual health services, family planning clinics, general practices, antenatal clinics and Aboriginal community controlled health services, and a laboratory network. Each network involves multiple sites, chosen under the following criteria (which varied by network):

1. a specified minimum number of chlamydia tests per year;

2. geographic representation; and

3. a minimum number of tests in priority populations specified by the *National Sexually Transmissible Infections Strategy 2005–2008,*[10] as defined in Table 1.

On a quarterly or 6-monthly basis, a core set of data in de-identified line-record format were extracted from sites (apart from the antenatal clinic network) and include patient demographic and chlamydia testing information. Additional information was collected from some specific networks; 'gender of sexual partners', 'current sex work', 'sex overseas in the last 12 months', 'traveller or migrant status' (sexual health service network), 'parity' (antenatal clinic network) and specimen type (laboratory network). Extraction programs were developed to transform these records into a common, pre-defined format.

Analyses were conducted of the proportion of patients tested for chlamydia and the proportion of those tested found to have infection (chlamydia positivity). Both were restricted to new or unique patients (those attending the clinic or tested for the first time in the surveillance period). Analyses

Table 1: Priority populations seen at ACCESS network sites

Network	Priority populations
Sexual health service network	Young men and women (<25 years), men who have sex with men, Aboriginal and/or Torres Strait Islander people, and sex workers
Family planning clinic network	Young women and men aged 16–29 years
Antenatal clinic network	Young pregnant women aged 16–24 years including Aboriginal and/or Torres Strait Islander people
Aboriginal community controlled health service network	Aboriginal and/or Torres Strait Islander people aged 16–39 years
General practice network	Young women and men aged 16–29 years
Laboratory network	All individuals tested for chlamydia

for the sexual health service network were further broken down into heterosexuals aged less than 25 years, MSM, Aboriginal and/or Torres Strait Islander people, and sex workers. The sexual health service network was able to compile retrospective data at the time of this evaluation. An analysis of time trends could be undertaken in the annual proportion of patients undergoing a chlamydia test on their first visit and of chlamydia positivity over time. Significance of the trend was assessed with a chi-squared test.

Feasibility

Feasibility was demonstrated by success in recruiting and establishing sites. By the end of February 2010, 91 sentinel sites across 6 networks had agreed to participate in ACCESS, representing all jurisdictions in Australia (Table 2).

Of the 91 sites, 76 (84%) provided data at the time of the evaluation (Table 3). The 5 clinical networks compiled information on about 90,000 episodes of care in new patients and the laboratory network compiled information on about 40,000 chlamydia tests, (which may have overlapped with the clinical networks).

Feasibility was also demonstrated in the ability to estimate the proportion of patients tested for chlamydia and chlamydia positivity in a range of priority populations. In young men and women, the general practice network found that 4.2% of males and 7.0% of females aged 16–29 years who attended the 26 clinics, were tested and chlamydia positivity was 9.9% and 7.0% respectively. In the sexual health service network, 77% of males and 74% of females aged less than 25 years were tested in the same period and positivity was 9.5% and 9.1%, respectively. The overall chlamydia positivity rate was 7.0% among pregnant women aged 16–24 years in the antenatal network.

In the laboratory network, the chlamydia positivity estimate was 6.2% in rectal swabs collected

Table 2: ACCESS participating sentinel sites, 1 March 2010, by network and state or territory

Location of sentinel sites	Population[9]		Network							
			GP	FPC	SHS	ANC	ACCHS	Lab	All	
	n	%	n	n	n	n	n	n	n	%
ACT	339,865	1.6	0	1	1	1	0	0	3	3.3
NSW	6,889,072	32.8	5	1	15	1	2	3	27	29.7
NT	214,975	1.0	0	1	2	1	1	0	5	5.5
Qld	4,182,062	19.9	6	1	4	1	2	4	18	19.8
SA	1,584,513	7.5	4	1	0	0	0	2	7	7.7
Tas	493,341	2.3	2	1	1	0	0	1	5	5.5
Vic	5,205,216	24.8	8	1	1	4	1	5	20	22.0
WA	2,105,783	10.0	2	1	1	1	1	0	6	6.6
Aust	21,017,222	100	27	8	25	9	7	15	91	100.0

GP=General practice, FPC=Family planning clinic, SHS=Sexual health service, ANC=Antenatal clinic, ACCHS=Aboriginal community controlled health service, Lab=Laboratory

Table 3: ACCESS operational sentinel sites, 1 March 2010, by network and state or territory

Location of sentinel sites	Population[9]		Network							
			GP	FPC	SHS	ANC	ACCHS	Lab	All	
	n	%	n	n	n	n	n	n	n	%
ACT	339,865	1.6	0	1	1	1	0	0	3	3.9
NSW	6,889,072	32.8	4	1	15	1	0	0	21	27.6
NT	214,975	1.0	0	1	2	1	0	0	4	5.3
Qld	4,182,062	19.9	6	0	4	1	2	3	16	21.1
SA	1,584,513	7.5	4	1	0	0	0	0	5	6.6
Tas	493,341	2.3	2	1	1	0	0	0	4	5.3
Vic	5,205,216	24.8	8	1	1	4	1	3	18	23.7
WA	2,105,783	10.0	2	0	1	1	1	0	5	6.6
Aust	21,017,222	100	26	6	25	9	4	6	76	100.0

GP=General practice, FPC=Family planning clinic, SHS=Sexual health service, ANC=Antenatal clinic, ACCHS=Aboriginal community controlled health service, Lab=Laboratory

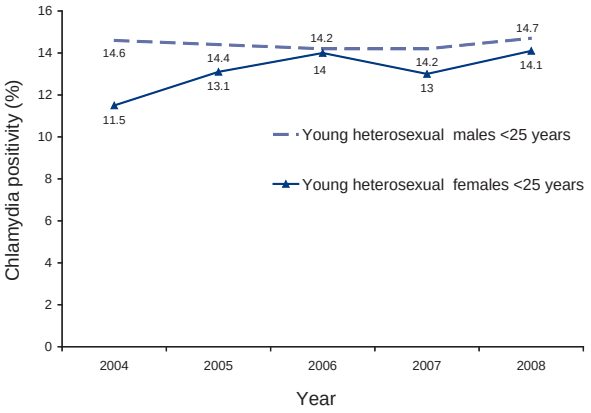
in men who were most likely to be MSM. In the sexual health service network, chlamydia positivity was 7.2% in MSM.

The feasibility of reporting time trends was demonstrated through the sexual health network. Between 2004 and 2008 at 19 sexual health services the annual proportion of patients tested for chlamydia on their first visit increased significantly for all priority populations, $P<0.001$ (Table 4) and chlamydia positivity increased significantly in heterosexual females from 11.5% to 14.1%, $P<0.001$ (Figure), but not other populations.

Simplicity

The automatic collation of routine clinical data from the network sites, apart from antenatal clinics, reflects simplicity. The 2 key outcomes are relatively straightforward to calculate from the variables compiled. Also, although not formally assessed, the ACCESS system is likely to be very much cheaper than repeated community surveys of this magnitude.

Figure: Chlamydia positivity among new heterosexual patients (<25 years) at the 19 sexual health services in ACCESS, 2004 to 2008, by sex



On the other hand, initiation of 91 sentinel sites was complex because of the multiple ethics applications required, and the range of patient management systems used at these sites. The antenatal clinic network used a model that differs from other networks, because underlying testing rates

Table 4: Chlamydia testing rates among new patients at the 19 sexual health services in ACCESS, 2004 to 2008, by priority population

Priority population	2004 (%)	2005 (%)	2006 (%)	2007 (%)	2008 (%)
Young heterosexuals <25 years	60.9	64.9	67.5	70	71.1
Men who have sex with men	74.8	77.3	78.9	80.9	78.0
Aboriginal and/or Torres Strait Islanders	48.9	49.5	54.6	55.8	53.0
Sex workers	60.9	64.9	67.5	70.0	71.1

The population breakdowns are not exclusive and individuals may be present in more than 1 priority population

were low in this setting. Accordingly, the network introduced chlamydia testing for consecutively recruited women aged 16–24 years, based on written consent, which substantially increased the human resources required to carry out the study. The recent increase in birth rates put further pressure on antenatal clinics participating in ACCESS.

Representativeness

Across networks, the jurisdictional distribution of the 76 operational sites generally matched the distribution of the Australian population (Table 2). For example, the proportion of sites in New South Wales, Victoria and Queensland were 28%, 24% and 21%, compared with the proportion of the resident population in these jurisdictions of 33%, 25%, 20% respectively (Table 2).

The correspondence was poorer within networks, with only 2 jurisdictions represented in the laboratory network, four in the ACCHS network and five in the family planning and general practice networks (Table 2).

Overall, 70% of the operational sites were located in metropolitan areas, 28% in regional areas and 3% in remote areas (Table 5), which is similar to the distribution of the resident population in Australia of 66%, 31% and 3%, respectively.[13]

Flexibility

Health surveillance system can adapt to changing information needs or operation conditions with little additional time, personnel, or allocated funds.[11] Apart from the antenatal clinic network, ACCESS was based on collection of routine clinical data, so as long as any additional information is systematically recorded in the patient management system by sites, ACCESS has the capacity to be modified relatively easily including collection of data on additional infections such as syphilis, gonorrhea and HIV.

Timeliness

The ACCESS system has not been operational long enough to demonstrate whether or not its outputs are sufficiently timely to support public health programs and policy. However, all of the networks (except the antenatal clinic network) are now in a position to undertake regular data collection automatically, and generate reports 3–6 months after the end of each calendar year. Given that sexually transmitted infections (STIs) do not generally require an acute public health response, this time frame should respond well to the needs of program planning and evaluation.

Sustainability

The key feature of ACCESS is that it utilises routinely collected data from clinical and laboratory settings. Therefore as long as staffing is available for analysis and reporting, the system will be sustainable.

Acceptability

Acceptability is reflected by the willingness of persons and organisations to participate in the surveillance system.[11] The entire premise of ACCESS was to establish data collection systems that operate automatically and have no impact on routine clinical operations. Each network was overseen by a steering committee that includes representation from sites to ensure that operations are acceptable to the clinicians and data managers at the sites. No participating sites withdrew from participating once they became operational. One general practice site recently became ineligible due to changing to a patient management system not

Table 5: Location of operational sentinel sites in the 5 clinical networks, 1 March 2010

Clinical network	Location of sentinel site							
	Metropolitan		Regional		Rural		Total	
	n	%	n	%	n	%	n	%
GP	19	73.1	7	26.9	0	0.0	26	100
FPC	5	100.0	0	0.0	0	0.0	5	100
SHS	16	64.0	8	32.0	1	4.0	25	100
ANC	7	77.8	1	11.1	1	11.1	9	100
ACCHS	1	25.0	3	75.0	0	0.0	4	100
All	48	69.6	19	27.5	2	2.9	69	100

The laboratory network was not included as most laboratories service all areas.

GP=General practice, FPC=Family planning clinic, SHS=Sexual health service, ANC=Antenatal clinic, ACCHS=Aboriginal community controlled health service

compatible with the extraction program, but was interested in participating in the future if ACCESS was able to develop a compatible interface.

Data quality

Overall, the completeness of the data from ACCESS sites was excellent, and for most variables, exceeded the recommendation of 85% in the CDC surveillance standards.[12] The exceptions were Aboriginal and/or Torres Strait Islander status and country of birth, which ranged from 68%–100%, and 76%–100%, respectively, per network. Aboriginal and Torres Strait Islander status was 95% complete in the 25 sexual health services who provided data at the time of the evaluation, 86% complete in the 4 Aboriginal community controlled health services and 68% in the general practice and family planning clinic networks. In the family planning clinic network the 'country of birth' variable had a 76% completion rate.

Validity

There are several ways in which validity might be assessed. At a basic level of reporting accuracy, the performance of the software used to identify chlamydia tests in the patient management systems of 3 clinics in the general practice network can be compared with testing data from the same clinics, collated directly from laboratory services participating in a separate Victorian surveillance project.[13] When linked to version 2 of Medical Director as used by 1 clinic, the ACCESS reporting software detected 84% of the chlamydia tests reported by the laboratories, and the sensitivity increased to 97% at the other 2 clinics, which used version 3 of Medical Director. Conversely all of the tests detected via the ACCESS software were identified in the Victorian surveillance dataset (specificity).

The validity of the system was also supported by the stability of the reported profile of patients attending the participating clinics. At the 19 sexual health services, annual numbers of new patients remained quite steady (between 21,929 and 23,267). The median age was 28 years from 2004 to 2005 then 27 years from 2007 to 2008.

Sensitivity

Sensitivity is generally quantified as the proportion of cases of a disease or health event that are detected by a surveillance system.[12] ACCESS does not aim to capture all chlamydia diagnoses in Australia but instead focuses on priority populations attending clinical sites, and monitors testing uptake and chlamydia positivity in these groups. In this context, the main factor that could have an impact on sensitivity is under-reporting. As noted above under validity, ACCESS data extractions rely on the test and result being recognisable and extractable in the patient management system, and appear to have high sensitivity when compared with an alternative data source in the general practice network.

Usefulness

The sexual health service network has provided some important data on time trends, as described under feasibility. These findings suggest that the steadily increasing chlamydia diagnoses observed through passive surveillance in recent years in Australia may reflect a true increase in chlamydia incidence in Australia. In the long term, outcomes from other networks will be important to interpret alongside those observed in the sexual health service network. Another important finding from the sexual health service network and laboratory network was the chlamydia positivity estimates in MSM, based on testing of rectal swabs in men.

Discussion

The first 2 years of ACCESS demonstrated that it is possible to establish a national network of diverse clinical and laboratory sites for the purpose of collecting, analysing and reporting standardised data on the uptake and outcome of testing for chlamydia. ACCESS has also demonstrated that clinical services can routinely compile information on chlamydia positivity in large numbers of patients. Although alternative models were not costed, it is likely that ACCESS costs a fraction of what would be required to conduct repeated surveys among the priority populations.

The evaluation led to 6 main recommendations about how the operation of ACCESS could be improved (Box).

As shown in other countries, systems similar to ACCESS can help to interpret trends in chlamydia passive surveillance.[7,14–17] Data from the sexual health network indicated a 23% increase in chlamydia positivity in young heterosexual women between 2004 and 2008, in contrast to the much sharper rise in case counts reported from passive surveillance in the same time period, suggesting some of the increase in case counts is likely to be related to increased testing.

For populations such as MSM who undergo frequent testing for Chlamydia, the sexual health service network will be able to provide national incidence estimates, that have previously only been available from single study cohorts.[18] Incidence is

Box: Key evaluation recommendations for the ACCESS system

1. Each network should undertake validity studies along the lines of those conducted by the general practice network;
2. ACCESS findings should be disseminated widely, to ensure that all relevant stakeholders can use them to plan and evaluate interventions related to chlamydia testing;
3. The general practice network should be enhanced by the addition of more sites in certain jurisdictions and expanded to a much larger number of clinics over the long-term;
4. Subject to community consultation, the Aboriginal community controlled service network should be expanded to include more sites over the long term, particularly in New South Wales and Queensland.
5. A less resource-intensive surveillance system should be used for antenatal services such as the model used in other networks of ACCESS;
6. The collection of information on other sexually transmissible infections such as syphilis and gonorrhoea should be considered.

the most sensitive indicator of changes in disease transmission, but is very expensive to assess through prospective cohorts. Line-listed records, linked by unique personal identifiers and information related to serial consultations can be used to provide incidence data.[19]

Although the system has been developed for monitoring chlamydia, its design is such that it could easily be adapted to the monitoring of other treatable bacterial STIs, such as syphilis and gonorrhea, or viral STIs such as HIV. The marginal cost of expanding the surveillance system to other infection would be far less than the cost of starting new systems for each of these infections.

ACCESS provided information on some variables not available through national chlamydia passive surveillance, in particular the sex of sexual partner, which allows trends to be analysed separately for MSM and heterosexual populations. As with passive surveillance, completeness in ACCESS was poorest for Aboriginal and Torres Strait Islander status, but ACCESS did achieve somewhat higher completeness rates for this variable than passive surveillance in New South Wales, Queensland, the Australian Capital Territory and Tasmania.[5]

The evaluation found that ACCESS sites were represented in all jurisdictions. The general practice network could be further expanded to increase the capacity of jurisdictions to evaluate local testing initiatives. In the longer term, data collected through ACCESS, particularly the general practice network, would also be able to provide pre– and post-descriptions of clinical populations, as a basis for evaluating new programs. Another application of ACCESS data would be the assessment of compliance with clinical testing guidelines.[20] The laboratory network also provides a very large sample size to ensure very robust chlamydia positivity rate estimates and in the long term will provide important population level testing data, covering both the public and private laboratory sectors.

ACCESS would also be able to provide valuable information about testing and chlamydia positivity rates in Aboriginal and/or Torres Strait Islander people attending a variety of clinical services in urban, regional and remote areas in Australia. Currently, the limited available data about STI testing undertaken by health services in Aboriginal and Torres Strait Island people are biased toward remote and regional settings.[21–23] Also because health departments in New South Wales and Queensland, with substantial Aboriginal populations, rely on laboratory notifications, the results of passive STI surveillance cannot be used to describe the STI epidemiology in this population.[5] A clearer picture may emerge if there was expansion of the Aboriginal community controlled health service network over the long term, particularly in New South Wales and Queensland. Any future directions of this network are subject to community consultation.

The antenatal clinic network methodology proved much more expensive and complex to implement because chlamydia testing is not routine in that setting. Nevertheless, this network has netted invaluable data on the prevalence of chlamydia in pregnant women in Australia. It is anticipated

that antenatal chlamydia testing will increase in the future, so the model used in other ACCESS networks could provide a less resource-intensive surveillance system for antenatal services.

There is no perfect surveillance mechanism for monitoring the prevalence of infections such as chlamydia in populations. Surveys of the whole population are inevitably subject to bias because of incomplete participation, and surveys that aim to recruit particular population groups inevitably rely on sampling frames that can not truly replicate the membership of these groups. The approach adopted by ACCESS is to monitor chlamydia positivity rates, as a surrogate for prevalence, in patients attending specialised clinical services. This approach has similar limitations to the population surveys, in that it is unknown how representative these patients are of any wider group from which they are drawn. Chlamydia positivity rates may be influenced by the proportion and nature of the patients being tested. Some groups may undergo testing at particular times in response to campaigns, or clinics may change testing policies resulting in more asymptomatic patients being tested. The restriction of the analysis to new patients (or those testing for the first time in the surveillance period) was intended to reduce the impact of this potential bias and provide accurate chlamydia positivity for surveillance purposes.

Passive notification data demonstrated that chlamydia diagnoses have increased sharply over the past decade in Australia. ACCESS has the potential to complement this observation by providing a systematic means of measuring any changes in testing levels by specific priority populations and monitoring trends in chlamydia positivity in these groups, thereby enhancing our capacity to respond to and control this infection.

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References

1. Australian Government Department of Health and Ageing. National Notifiable Diseases Surveillance System data. Number of notifications of chlamydial infections, Australia, 2007 by age group and sex. Accessed on 29 February 2008. Available from: http://www1.health.gov.au/cda/Source/Rpt_5.cfm

2. Chen MY, Fairley CK, Donovan B. Nowhere near the point of diminishing returns: correlations between chlamydia testing and notification rates in New South Wales. *Aust N Z J Public Health* 2005;29(3):249–253.

3. Chen MY, Karvelas M, Sundararajan V, Hocking JS, Fairley CK. Evidence for the effectiveness of a chlamydia awareness campaign: increased population rates of chlamydia testing and detection. *Int J STD AIDS* 2007;18(4):239–243.

4. Hocking J, Fairley C, Counahan M, Crofts N. The pattern of notification and testing for genital *Chlamydia trachomatis* infection in Victoria, 1998–2000: an ecological analysis. *Aust N Z J Public Health* 2003;27(4):405–408.

5. National Centre in HIV Epidemiology and Clinical Research. *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander People. Surveillance Report 2007.* Sydney, NSW: National Centre in HIV Epidemiology and Clinical Research, The University of New South Wales; 2007.

6. Slater W, Sadler K, Cassell JA, Horner P, Low N. What can be gained from comprehensive disaggregate surveillance? The Avon Surveillance System for Sexually Transmitted Infections. *Sex Transm Infect* 2007;83(5):411–415.

7. Fine D, Dicker L, Mosure D, Berman S. Increasing chlamydia positivity in women screened in family planning clinics: do we know why? *Sex Transm Dis* 2008;35(1):47–52.

8. Centers for Disease Control and Prevention. Updated guidelines for evaluating public health surveillance systems: recommendations from the Guidelines Working Group. *MMWR Recomm Rep* 2001;50(RR13):1–35.

9. Australian Bureau of Statistics. Population by Age and Sex, Australian States and Territories. Canberra: Australian Bureau of Statistics. Catalogue No: 3201.0 – A. June 2008.

10. Australian Government Department of Health and Ageing. *National Sexually Transmissible Infections Strategy 2005–2008.* Canberra; 2005. Accessed 6 June 2006. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/phd-sti-strategy-cnt.htm/$FILE/sti_strategy.pdf

11. UNAIDS. *Guidelines for Second Generation HIV Surveillance.* Geneva: UNAIDS; 2000. Report No: WHO/CDS/CSR/EDC/2000.5.

12. Centers for Disease Control and Prevention. Guidelines for national human immunodeficiency virus case surveillance, including monitoring for human immunodeficiency virus infection and acquired immunodeficiency syndrome *MMWR Recomm Rep* 1999;48(RR–13):1–27, 9–31.

13. Goller J, Guy R, Gold J, et al. Establishing a linked sentinel surveillance system for BBVs and STIs, methods, system attributes and early findings. *Sex Health* In press.

14. Hiltunen-Back E, Haikala O, Kautiainen H, Paavonen J, Reunala T. A nationwide sentinel clinic survey of *Chlamydia trachomatis* infection in Finland. *Sex Transm Dis* 2001;28(5):252–258.

15. Giuliani M, Suligoi B, The STD Surveillance Working Group. Sentinel surveillance of sexually transmitted diseases in Italy. *Euro Surveill* 1998;3(6):55–58.

16. Defraye A, Sasse A. STI surveillance by a sentinel network of physicians in Belgium. *Acta Clin Belg* 2005;60(2):70–74.

17. Bachmann LH, Macaluso M, Hook EW 3rd. Demonstration of declining community prevalence of *Chlamydia trachomatis* infection using sentinel surveillance. *Sexy Transm Dis* 2003;30(1):20–24.

18. Jin F, Prestage GP, Zablotska I, Rawstorne P, Kippax SC, Donovan B, et al. High rates of sexually transmitted infections in HIV positive homosexual men: data from two community based cohorts. *Sex Transm Infect* 2007;83(5):397–399.

19. Lee DM, Binger A, Hocking J, Fairley CK. The incidence of sexually transmitted infections among frequently screened sex workers in a decriminalised and regulated system in Melbourne. *Sex Transm Infect* 2005;81(5):434–436.

20. Guy R, Goller J, Spelman T, El-Hayek C, Gold J, Lim M, et al. Does the frequency of HIV and STI testing among MSM in primary care adhere with Australian guidelines? *Sex Transm Infect* 2010. PMID: 20460263 [Epub ahead of print].

21. Huang RL, Torzillo PJ, Hammond VA, Coulter ST, Kirby AC. Epidemiology of sexually transmitted infections on the Anangu Pitjantjatjara Yankunytjatjara Lands: results of a comprehensive control program. *Med J Aust* 2008;189(8):442–445.

22. Latif AS, Smith KS. Sexually transmitted infections in Central Australia – time for concerted action. *Public Health Bulletin South Australia.* 2006;4:32–34.

23. Su JY, Skov S. An assessment of the effectiveness of the Tiwi Sexual Health Program 2002–2005. *Aust N Z J Public Health* 2008;32(6):554–558.

Appendix: Participating ACCESS sites

Note: some participating sites preferred not to be named in this paper

Sexual health service network

Hunter New England Sexual Health Service; NSW

Sydney West Area Health Service – Clinical Sexual Health Services; NSW

Illawarra Sexual Health, Wollongong; NSW

Royal Prince Alfred Hospital Sexual Health, Camperdown; NSW

Holden Street Clinic, Gosford; NSW

Lismore/ Tweed Heads Sexual Health & AIDS Services, Lismore; NSW

Northern Sydney Sexual Health Service, St Leonards; NSW

Greater Southern Area Health Service; NSW

Orange Sexual Health Service, Orange; NSW

Kirketon Road Centre, Darlinghurst; NSW

Sydney Sexual Health Centre, Sydney; NSW

Short Street Sexual Health Clinic, NSW

St George Hospital, St George; NSW

Coffs Harbour Sexual Health Service, Coffs Harbour; NSW

Grafton Sexual Health Clinic, Grafton; NSW

Gold Coast Sexual Health Clinic, Miami; Qld

Cairns Sexual Health Services, Cairns Base Hospital, Cairns; Qld

Princess Alexandra Sexual Health, Princess Alexandra Hospital, Woolloongabba, Qld

Townsville Sexual Health Service, Townsville, Qld

Melbourne Sexual Health Centre, Carlton, Vic

Hobart, Devonport and Launceston Sexual Health Service, Tas

Fremantle Hospital, Fremantle, WA

NT Sexual Health and BBV Unit, NT

Family planning clinic network

Sexual Health and Family Planning, ACT,

Newcastle FPNSW Centre, Cooks Hill, NSW

Family Planning NT, Coconut Grove, NT

Family Planning Queensland, Toowoomba, Qld

Shine SA (Sexual Health information networking and education Inc), SA

Family Planning Tasmania, Hobart, Tas

Family Planning Victoria, (Action Centre), Melbourne, Vic

Quarry Health Centre for under 25s, Fremantle, WA

General practice clinic network

Charlestown Family Medical Services, Charlestown, NSW

Midway Family Medical Centre, Denistone East, NSW

Glendale Medical Centre, Glendale, NSW

Young District Medical Centre, Young, NSW

Brindabella Family Practice, Queanbeyan, NSW

Angaston Medical Centre, Angaston, SA

Genesis Medical Centre, Brighton, Vic

Footscray Medical Centre, Footscray , Vic

Goulburn River Group Practice, Seymour, Vic

Wellness Centre Medical Clinic, Malvern East, Vic

Brighton Medical Clinic, Brighton, Vic

Mooroopna Medical Centre, Mooroopna, Vic

Duncraig Medical Centre, Duncraig , WA

AK medical/dental Clinic, Kelmscott, WA

Chancellor Park Family Medical Practice, Sippy Downs, Qld

Nambour Medical Centre, Nambour, Qld

Eli Waters Medical Centre, Eli Waters, Qld

Yeppoon Family Practice, Yeppoon , Qld

Kewarra Family Practice, Kewarra Beach, Qld

Turton St Medical Centre, Sunnybank, Qld

O'Brien Street Practice, Adelaide, SA

Davey Street Medical Centre , Hobart, Tas

Newstead Medical, Launceston, Tas

Duncraig Medical Centre, Duncraig, WA

Brighton Medical Clinic, Brighton, Vic

North Sydney Medical Practice, North Sydney, NSW

Centre Clinic, St Kilda, Vic

Aboriginal community controlled health service network

Aboriginal Medical Service Western Sydney, Mount Druitt Village, NSW

Durri Aboriginal Corporation Medical Service, Kempsey, NSW

Victorian Aboriginal Health Service, Fitzroy, Vic

Geraldton Regional Aboriginal Medical Service, Geraldton, WA

Danila Dilba Health Service, Darwin, NT

Carbal Medical Service, Toowoomba, Qld

Goondir Health Service, Dalby, Qld

An outbreak of gastroenteritis due to *Salmonella* Typhimurium phage type 170 associated with consumption of a dessert containing raw egg

Anna Reynolds, Cameron RM Moffatt, Amalie Dyda, Rebecca L Hundy, Andrew L Kaye, Radomir Krsteski, Simon Rockliff, Riemke Kampen, Paul M Kelly, Eddie D O'Brien

Abstract

Eggs are frequently implicated as a source of foodborne salmonellosis. In February 2009 an investigation was commenced following reports of gastrointestinal illness among diners at a Canberra restaurant. The investigation sought to confirm the existence of an outbreak, identify a source and implement public health measures to prevent more cases. Menus and booking lists were obtained from the restaurant and a case-control study was commenced. A suspected case was defined as a person who ate at the restaurant on 13 or 14 February 2009 and subsequently developed diarrhoea and/or vomiting. Twenty cases and 31 controls were enrolled in the study. Eating a tiramisu dessert containing raw egg had a highly statistically significant association with illness (crude odds ratio 130.50, 95% confidence interval 13.54–1605.28). Among the 20 cases, nine of 12 stool samples were positive for *Salmonella* Typhimurium phage type 170 (STm 170). No microbiological evidence of STm 170 was obtained from the restaurant or during the egg trace-back investigation. This report highlights the risk associated with consumption of foods containing raw or undercooked shell egg. *Commun Dis Intell* 2010;34(3):329–333.

Keywords: *Salmonella* Typhimurium; disease outbreak; foodborne disease; Australia; salmonellosis

Introduction

On 20 February 2009, a general practitioner in the Australian Capital Territory notified the Health Protection Service (HPS) at ACT Health of a patient with gastroenteritis. The case reported that they had eaten at a local restaurant and that a number of others at the same table were also ill. A laboratory-confirmed case of salmonellosis was also notified to the Communicable Disease Control Section later that same day. This case implicated the same restaurant as a potential source of their illness, also reporting a number of fellow diners as being unwell. In total, 7 cases of gastroenteritis from 2 separate tables were linked to the restaurant on 13 February. The venue was inspected by environmental health officers (EHOs) on 20 February to assess kitchen hygiene standards and identify any potential sources of infection. An Acute Response Team meeting was then held and an outbreak investigation launched to confirm that there was an outbreak associated with the restaurant, to identify the source of the illness and implement public health measures to prevent further illness.

Methods

Case-control study

A case-control study was conducted to test the hypothesis that gastrointestinal illness was associated with consumption of a particular food item at the restaurant. During this study, cases were identified using the following case definition.

Confirmed – a person who has a laboratory-confirmed case of *Salmonella* and ate at the restaurant on 13 or 14 February 2009.

Suspected – a person who ate at the restaurant on 13 or 14 February 2009 and developed diarrhoea (defined as three or more loose stools in a 24-hour period) and/or vomiting with onset of these symptoms on or after those dates.

A structured questionnaire was developed from the menu provided by the restaurant and was used during telephone interviews with cases and controls. The aim of the questionnaire was to obtain information on illness symptoms, onset date and time, and consumption of specific food and beverage items at the restaurant. Restaurant reservation lists for 13 and 14 February 2009 were also obtained and used for case ascertainment and to recruit controls for the study.

Cases were identified via the reservation lists, in conjunction with an examination of *Salmonella* notifications received following the suspected days of exposure. Some cases were identified through an examination of the notifications for people who lived in suburbs close to the restaurant. Controls were recruited using the reservation lists and via a convenience sampling process in which cases were asked who they dined with at the restaurant. Controls were defined as persons who ate at the restaurant and did not develop gastrointestinal illness. An unmatched analysis was conducted as there were often whole tables affected by illness and cases varied by age and gender.

Data analysis

Data from the interviews were entered into Epi Info™ version 3.3.2 and analysed using STATA™ version 9.0, with both suspected and laboratory confirmed cases included in the analysis. Case and control demographic details such as sex were compared using Fisher's exact test, and age using Student's *t*-test. Univariate analysis was conducted to calculate crude odds ratios (OR) with 95% confidence intervals (CI) for all exposures. A multivariate logistic regression model was constructed to adjust for confounding using food items that had a *P* value of <0.01 in the univariate analysis. As around 50 food and beverage items were being examined, a conservative cut off for the *P* value was chosen to reduce the probability of a chance association between a food item and gastrointestinal illness.

Environmental investigation

Environmental health officers conducted an inspection of the restaurant kitchen facilities and food preparation procedures. Advice regarding safe food handling practices and the preparation of foods containing egg was provided to restaurant staff. Samples of mascarpone cheese, raw shell eggs and tiramisu were taken, however there were no leftover foods from the suspected days of exposure. In this case, the specific batch of eggs used to make the tiramisu could not be determined. A trace-back investigation identified a specific supplier of fresh eggs to the restaurant. Because the Australian Capital Territory does not have a Department of Primary Industries, the NSW Food Authority was contacted as they have extensive experience in trace-back investigations. They provided advice on egg farm investigations, including a sampling protocol. The supplier/producer of the fresh eggs was inspected with a number of samples taken, including ready for sale and fresh laid eggs, wash and rinse waters, swabs from cages, egg conveyor belts, and drag swabs from laying sheds.

Laboratory investigation

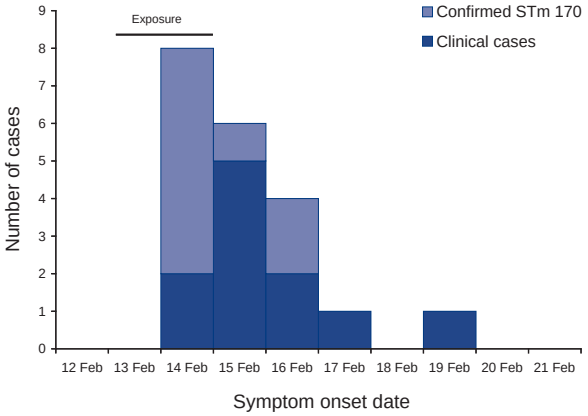
Stool samples were obtained from 12 cases and were tested for enteric pathogens using standard laboratory methods. Food samples taken from the restaurant and environmental samples from the local egg supplier were tested for the presence of *Salmonella*, by the Australian Capital Territory Government Analytical Laboratory using standard food and environmental laboratory methods. *Salmonella* isolates were serotyped, phage typed and identified by multiple locus variable-number tandem repeat analysis (MLVA) at the Microbiological Diagnostic Unit, Melbourne, Victoria.

Results

Epidemiological findings

A total of 20 cases that met the case definition were identified using *Salmonella* notification data and active case ascertainment. Eight cases reported that they ate at the restaurant on 13 February 2009 while 12 cases reported eating at the restaurant on 14 February 2009 (Figure). The median incubation period was 24 hours with an interquartile range of 15.5 hours. Symptom prevalence for the 20 cases was: watery diarrhoea 100%, lethargy 96%, abdominal pain 90%, fever 85%, headache 75%, nausea 70%, and vomiting 40%. None of the cases reported bloody diarrhoea. Fourteen cases (70%) consulted a doctor about their illness and 5 cases (25%) reported visiting a hospital

Figure: Cases of gastrointestinal illness associated with a restaurant, Australian Capital Territory, 12 to 21 February 2009, by symptom onset date



emergency department for treatment, although none of these was admitted. The duration of illness ranged from between 3 and 14 days.

All 20 cases identified during the investigation were included in the case-control study. A total of 31 controls were recruited and enrolled in the study. Controls were either nominated by cases or recruited through the restaurant booking list for the suspected dates of exposure. The mean age of the control group was 42 years (median: 43 years; range 9–71 years) and 56% were females. The sex distribution did not significantly differ between cases and controls (Fisher's exact 2-tailed test, $P = 0.39$) and there was no significant difference in the mean ages between the groups ($t = 0.80$ $P = 0.43$).

The exposure of cases and controls to approximately 50 foods and beverages served at the restaurant during the potential exposure period was determined using a questionnaire based on the restaurant menu. The restaurant served blackboard specials, which varied from day to day, but it was not possible to determine which specials were served on 13 and 14 February. However, cases and controls were asked if they ate a special and what that special was. Bread and coffee had elevated odds ratios but the association of illness with these items was not statistically significant. Tiramisu consumption was significantly associated with illness having a crude OR of 130.50 (95% CI 13.54–1605.28) (Table). Eighteen cases and 2 controls reported eating tiramisu, with some sharing a single serving and others reportedly eating very small amounts. The crude OR for carbonara sauce (16.15 95% CI 1.70–751.40) was also significantly elevated suggesting it could be associated with illness. However, this food item appeared to be confounded by tiramisu as the adjusted OR was no longer significant as the 95% confidence interval included one (Table).

Environmental findings

Investigation of the restaurant kitchen by EHOs determined that there was a good standard of general food hygiene and cleanliness. There were no sick food handlers working at the restaurant during the suspected exposure period. The tiramisu dessert was made on site in a large batch and did not undergo any cooking; it was cold set in the fridge and no temperature abuse was identified. However, raw fresh shell eggs were being used to prepare the tiramisu and the broken shells used to separate the egg yolk. The restaurant owners were advised that this practice increases the risk of *Salmonella* contamination and that in future they should either use an egg separator or pasteurised eggs during the dessert preparation. Investigation of the egg supplier/producer revealed production of eggs from both free range and caged chickens. The facility was semi-modern and fully automated, with eggs undergoing a two-stage washing procedure, followed by candling and ultrasonic detection of cracks.

Laboratory findings

Twelve stools samples were obtained from cases and nine were positive for *Salmonella* Typhimurium phage type 170 (STm 170). All 9 samples were also MLVA typed and found to have the same MLVA pattern: 03-09-08-13-526/523 (Australian nomenclature). Environmental samples taken

Table: Odds ratios for a selection of foods and beverages consumed, by cases and controls

	Foods eaten				Crude OR	95% CI	Multivariate analysis			
	Cases		Controls				*P* value*	Adjusted OR	95% CI	*P* value*
	n	%	n	%						
Bread	17	85	20	65	3.12	0.66–19.83	0.20			
Amatriciana sauce	2	10	1	3	3.33	0.16–203.06	0.55			
Original pizza	4	20	1	3	7.50	0.64–381.29	0.07			
Fettuccine	9	45	4	13	5.52	1.18–28.91	0.02			
Carbonara sauce	7	35	1	3	16.15	1.70–751.40	<0.001†	4.70	0.17 – 127.97	0.36
Tiramisu	18	90	2	6	130.50	13.54–1605.28	<0.001†	100.90	12.72 – 800.16	<0.001*
Coffee	9	45	8	26	2.35	0.61–9.14	0.23			

Cases = 20 and Controls = 31

OR odds ratio

* *P* value calculated using 2-tailed Fisher's exact Test.

† Statistically significant ($P<0.01$)

from the restaurant kitchen were all negative for *Salmonella*, including the raw shell egg samples. A drag swab collected on the supplier/producer's premises tested positive for both *Salmonella* Agona and *Salmonella* Infantis.

Discussion

The results of this investigation demonstrate that this was a point source outbreak, with the epidemiological evidence supporting the hypothesis that the source of illness was tiramisu containing raw shell egg. The attack rate of the tiramisu was very high (18/20 cases) and the crude odds ratio associating consumption of tiramisu with illness was also high (130.50). The crude odds ratios for carbonara sauce (also made using raw shell egg) was also significantly elevated. However, this was likely confounded by tiramisu as all of the people who ate fettuccine with carbonara sauce also ate tiramisu. In this outbreak, the tiramisu was made using raw shell eggs, with the shells used for separation of the yolks. It is therefore possible that one or more of the eggs used was contaminated with STm 170, although cross contamination from another unknown source cannot be excluded. Nevertheless, the authors consider this unlikely. Raw egg containing tiramisu has been implicated in a number of Australian outbreaks caused by various *Salmonella* Typhimurium phage types.[1–3] STm 170 has previously been isolated in outbreaks associated with foods containing raw or undercooked egg.[4,5] The initial public health action taken in response to this outbreak was to provide the restaurant with advice on methods that could reduce the risk of *Salmonella* transmission, such as the use of an egg separator or pasteurised eggs. In addition, the restaurant voluntarily removed the tiramisu from the menu for a short period of time.

From January 2009, the number of notifications of laboratory-confirmed STm 170 began to increase significantly in a number of states, including New South Wales, Queensland, Victoria and the Australian Capital Territory. This prompted a multi-jurisdictional outbreak investigation by OzFoodNet. While analyses of food frequencies identified several foods of interest, none of the hypotheses were tested through a case-control study, due to decreasing case numbers following the declaration of the outbreak (personal communication, Katrina Knope, OzFoodNet). An MLVA profile of the STm 170 cases in this outbreak was obtained and compared with other recent sporadic cases in the Australian Capital Territory and cases in other jurisdictions. All of the outbreak STm 170 positive cases in the Australian Capital Territory had the same MLVA pattern (03-09-08-13-526/523), suggesting a common source, and this profile was the predominant strain circulating in the Australian Capital Territory from January to March 2009.

During this investigation it was not possible to identify the specific batch of eggs used in the preparation of the tiramisu. However, the restaurant egg supplier/producer was traced and their facilities inspected for the presence of *Salmonella*. This outbreak highlighted an issue in the Australian Capital Territory regarding who has the authority to inspect food production facilities, as unlike other jurisdictions, the Australian Capital Territory does not have a Department of Primary Industries. Furthermore, inspections of any Australian Capital Territory egg supplier/producer as a direct response to a public health issue had not been previously conducted by the HPS. The inspection showed the egg supplier/producer had sophisticated processing facilities and no positive microbiological evidence of the presence of STm 170 was found. The investigators did have some difficulty in readily accessing the facility and obtaining a full quota of samples. This was in part due to the field investigators limited experience in egg farm investigation and also as a result of actions on behalf of the supplier/producer who initially refused access. A nationally agreed protocol for a program of auditing and investigation of primary production facilities would be useful for future outbreak investigations. Nevertheless, drag swabs taken from the floors of laying sheds were positive for both *S.* Agona and *S.* Infantis. This suggests that contamination of eggs could potentially occur at the facility and be passed on to consumers through unsafe food hygiene practices.

This outbreak investigation shows that despite advice from organisations such as the NSW Food Authority[6] and Food Science Australia[7] regarding the risks associated with eggs, the consumption of foods containing raw or undercooked shell egg continues to be associated with gastrointestinal illness. The risk of illness due to raw shell egg consumption must continue to be communicated to both the general public and hospitality industry. Improved communication between egg suppliers/producers and health departments, along with routine testing of egg supplier/producer facilities, could have public health benefits and may reduce the number of egg-related disease outbreaks.

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References

1. Hall R. Outbreak of gastroenteritis due to *Salmonella* Typhimurium phage type 135a following consumption of raw egg. *Commun Dis Intell* 2002;26(2):285–287.

2. OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, July to September 2004. *Commun Dis Intell* 2004;28(4):485–488.

3. Fullerton K. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: annual report of the OzFoodNet Network, 2007. *Commun Dis Intell* 2008;32(4):400–424.

4. OzFoodNet quarterly report, 1 October to 31 December 2006. *Commun Dis Intell* 2007;31(1):128–133.

5. OzFoodNet quarterly report, 1 January to 31 March 2008. *Commun Dis Intell* 2008;32(2):267–271.

6. NSW Food Authority. Enjoy eggs safely factsheet. Sydnay, New South Wales. Accessed 15 April 2009. Available from: http://www.foodauthority.nsw.gov.au/_Documents/consumer_pdf/enjoy_eggs_safely.pdf

7. Food Science Australia. *Salmonella* in food containing raw or undercooked eggs – information for the food service industry. August 2008. Accessed on 14 April 2009. Available from: http://www.foodscience.afisc.csiro.au/issues/salmonella-eggs.htm#6

Potential exposure to Australian bat lyssavirus in South East Queensland: what has changed in 12 years?

Megan K Young, Bradley J McCall

Abstract

Public health measures have been targeting potential exposure to Australian bat lyssavirus (ABLV) since the first recognised human cases, more than a decade ago. The effect of these measures on the epidemiology of notifications of potential exposure has not been investigated since 2003. Trends in notifications of potential exposure to ABLV reported to the Brisbane Southside Public Health Unit between November 1996 and October 2008 were examined. During the study period notification rates declined among all population groups and potential exposures were notified more promptly. The proportion of female notifications and the proportion of notifications from volunteer bat carers and their families and professional groups decreased over time. These changes over 12 years may indicate success of public health measures, under-reporting of potential exposure or both. Intentional handling of bats by untrained members of the public continues to be an important source of potential exposure to ABLV and requires a sustained public health response. *Commun Dis Intell* 2010;34(3):334–338.

Keywords: lyssavirus, bats, Chiroptera, disease notification, Queensland, Australia

Background

Australian bat lyssavirus (ABLV) is a member of the Rhabdoviridae family, which also includes European bat lyssavirus, and rabies virus. Classic rabies virus and ABLV possess marked similarity using both serotyping and molecular sequencing.[1] Like rabies, ABLV infection is lethal to humans. Two fatal cases of human ABLV infection have been reported in Australia, one in 1996 and the second in 1998.[2,3]

Bats are considered the natural host of ABLV. Natural infections have been recorded in both megachiropteran (flying fox) and microchiropteran (insectivorous bat) species.[4] The prevalence of ABLV infection in bats has been reported as <1%–7%.[5] Therefore, not all bat bites or scratches will result in human exposure to the virus.

Under current guidelines, all potential exposures require treatment (post exposure prophylaxis) with rabies vaccine and usually rabies immunoglobulin unless the bat involved is proven to be ABLV negative.[6] This requires that all available bats involved in potential human exposures are tested for ABLV. Detection of ABLV infection in bats requires examination of fresh brain impression smears.

Considering both human and animal welfare, it is desirable that potential exposure to ABLV is minimised. This is the objective of ongoing public health messages aimed at the general public.[7] Periodic examinations of notification data provide a measure of effect of these messages.

The epidemiology of potential exposure to ABLV in south east Queensland, Australia has been previously described.[4,8] Initial data showed that potential exposures were more likely to be the result of contact by people with some professional or volunteer interest in caring for bats than by members of the general public.[4] Between 1999 and 2003, the general public had a higher proportion of potential exposure than other groups, but absolute numbers of notifications had decreased.[8]

As no study had been conducted since 2003, population trends in potential exposure to ABLV reported to the Brisbane Southside Public Health Unit (BSPHU), in South East Queensland, between November 1996 and October 2008 were examined.

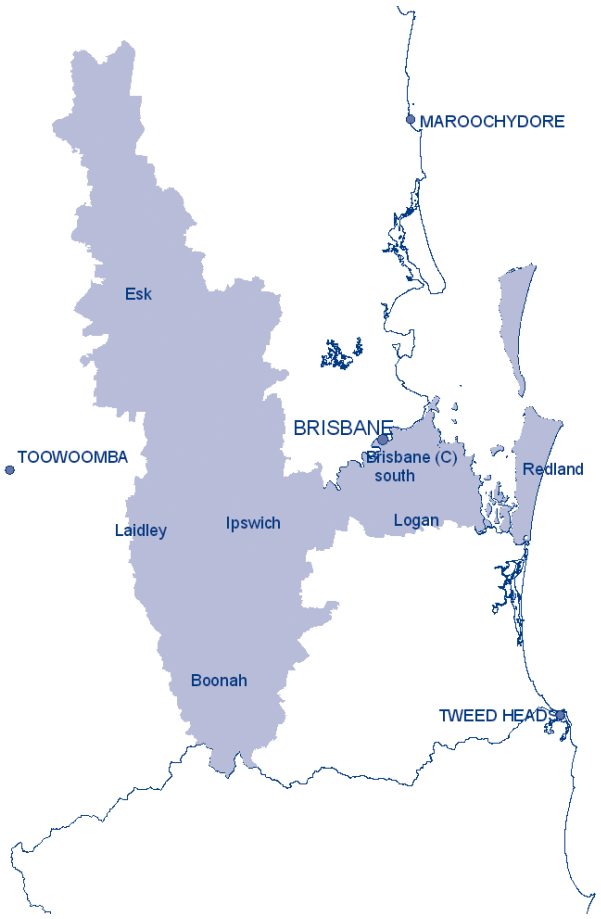
Methods

Potential exposure to ABLV is a clinical diagnosis notifiable condition in Queensland. Enhanced surveillance of potential exposure to ABLV commenced at the BSPHU in November 1996. Since then, BSPHU staff have collected details of all potential human exposures to ABLV through completion of a standard telephone administered questionnaire, and in accordance with national guidelines[6] and Queensland Health policy, all available bats involved in potential human exposure have been tested for ABLV infection at the local reference laboratory. Further details of

the questionnaire, methods of study and results until 31 January 1999 have been described.[4] Of particular relevance to this study were the questions about the circumstances surrounding the potential exposure. The resulting data were categorised for analysis. Mutually exclusive categories were termed: General public, bat initiated contact (including cases where bats swoop upon or otherwise engage in human contact without provocation); General public, intentional bat handling (including where members of the public have attempted to rescue bats caught in fruit tree nets or fences); Volunteer bat carers and their families (including people recognised as bat carers by the Queensland Department of Environment and Resource Management); and Professional duties (including veterinarians or other people who handle bats as part of their profession).

The geographic boundaries of the area served by the BSPHU changed after 1999, but were then consistent for the rest of the study. This area (Figure 1) includes several local government areas with an estimated resident population of 1.2 million as at 30 June 2006,[9] increased from 920,680 as at 30 June 2000.[10] To allow comparison of data across the entire study period, the original study data were restricted to those people who resided within the current BSPHU boundaries.

Figure 1: The geographical area covered by the Brisbane Southside Public Health Unit (shaded blue)



The trend in the number of notifications was examined using the curve estimation function in SPSS 16. Trend lines were modelled to determine which was the best fit for the data as indicated by the R^2 value. As a number of retrospective notifications occurred in the early years of the study, this analysis was repeated after restricting the data to those notifications where exposure occurred within the study period, and then to those notifications with an interval of 3 months or less between exposure and notification.

Because the number of notifications in each year of the study was small, the dataset was then examined in 3 periods of 4 years. Chi-squared tests were used to assess the statistical significance of changes in proportions. Where the assumptions of chi-squared testing were not met, Fisher's exact test was used. ANOVA was used to assess the statistical significance of changes in means. Analysis was conducted in SPSS 16.

Ethics committee approval was not sought because enhanced surveillance was conducted in accordance with Chapter 3 of the (Queensland) *Public Health Act 2005*.

Results

There were 385 notifications of potential exposure to ABLV over the 12 years of the study (November 1996 to October 2008), an average annual notification rate of 3.5 per 100,000 population. Notifications decreased over the first 4 years of the study and then seemed to plateau (Figure 2). The fitted line (Figure 2) accounted for 66% of the variability in the data (R^2 0.657; P=0.001). Restricting the data to notifications where exposure occurred within the study period (n = 332), and then to notifications with an interval of 3 months or less between exposure and notification (n = 343) did not appreciably alter this result (R^2 0.482; P = 0.012 and R^2 0.684; P = 0.001 respectively).

The mean age of those potentially exposed was 40 years, with equal proportions of males and females (Table 1). Of notifications where the circumstance of potential exposure was recorded, the majority (52%) occurred because members of the general public intentionally handled bats (Table 2). Volunteer bat carers and their families were the next most commonly notified group (27% of potential exposures). The majority of potential exposures were associated with bite injuries (55%;

n = 212). Two hundred and seventy-three notifications (71%) received post-exposure prophylaxis, although 17 of these notifications did not complete prophylaxis as the bat tested ABLV-negative.

There were 189 notifications of potential exposure to ABLV over the first 4 years of the study (November 1996 to October 2000 – period 1), 98 notifications over the second 4 years (November 2000 to October 2004 – period 2) and 98 notifications over the last 4 years (November 2004 to October 2008 – period 3). Accounting for population growth, the average annual notification rates were 5.1 per 100,000 (period 1), 2.7 per 100,000 (period 2), and 2.0 per 100,000 (period 3) ($P < 0.001$). The mean age of those notified was not different across the 3 periods of study ($P = 0.09$). There was significant difference in the ratio of males to females ($P = 0.018$) across the periods of study, with more females being notified in period 1 (58%) and more males being notified in periods 2 and 3 (58% and 55%) (Table 1).

The interval between potential exposure and notification was significantly different across the periods of study, decreasing from a mean of 228 days in period 1 to a mean of 3 days in period 2 and a mean of 22 days in period 3 ($P < 0.001$). Of those notifications where the circumstance of potential exposure was recorded (n = 376), these varied significantly across the periods of study ($P < 0.001$) (Table 2), although, in all periods, intentional bat handling by members of the general public was the most common circumstance. Both the number and the proportion of notifications from volunteer bat carers and their families showed the largest decline during the study (Table 2). Of female notifications, volunteer bat carers and their families accounted for 52% (n = 56) in period 1; 20% (n = 8) in period 2 and 30% (n = 13) in period 3. This was the largest decline in both numbers and proportion of female notifications across the study.

Figure 2: Notifications of potential exposure to Australian bat lyssavirus to the Brisbane Southside Public Health Unit, November 1996 to October 2008

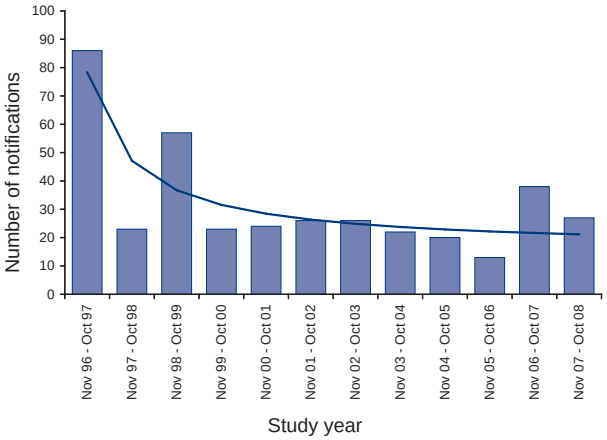


Table 1: Gender of notifications of potential exposure to Australian bat lyssavirus to the Brisbane Southside Public Health Unit

Gender	Nov 96–Oct 00		Nov 00–Oct 04		Nov 04–Oct 08		Entire study period	
	%	n	%	n	%	n	%	n
Male	42	80	58	57	55	54	50	191
Female	58	109	42	41	45	44	50	194
Total	100	189	100	98	100	98	100	385

Table 2: The circumstances of potential exposure to Australian bat lyssavirus of notifications to the Brisbane Southside Public Health Unit

Circumstance	Nov 96–Oct 00		Nov 00–Oct 04		Nov 04–Oct 08		Entire study period	
	%	n	%	n	%	n	%	n
General public, bat initiated contact	5	9	18	17	17	16	11	42
General public, intentional bat handling	43	79	64	61	59	57	52	197
Volunteer bat carers and families	41	75	11	10	19	18	27	103
Professional duties	12	22	7	7	5	5	9	34
All circumstances	101*	185	100	95	100	96	99*	376†

* Percentages do not add to 100 due to rounding error.

† Nine notifications did not have circumstance of exposure recorded and these have been excluded from the calculation of percentages.

There was a similar decline in the proportion of volunteer bat carers and their families among the male notifications, but the decrease in numbers was not as large (period 1: 24%, n = 19; period 2: 3.6%, n = 2; period 3: 9%, n = 5).

The proportion of notifications where bats were available for testing varied significantly across the study periods from 32% (n = 60) in period 1, to 51% (n = 50) in period 2, to 42% (n = 41) in period 3 ($P = 0.008$). Of notifications where bats were available for testing, the proportion with ABLV-positive bats decreased over time (20% (n = 12) in period 1; 6% (n = 3) in period 2; nil in period 3 ($P = 0.002$). A total of 6 bats tested positive over the 12 year study; four in period 1 and two in period 2. Fifteen people were exposed to ABLV-positive bats and provided with post exposure prophylaxis (in accordance with public health recommendations[6]). No new cases of human ABLV infection have been reported to date.

Discussion

Notification rates significantly decreased during the 12 years of enhanced surveillance, seeming to plateau in the latter part of the study. Changes in the distribution of notifications in various groups occurred. Notifications from the general public increased in proportion, but decreased in absolute numbers across the study period. The proportion of females notified decreased across the study period.

These results seem to support continuing effectiveness of public health messages about the importance of not handling bats. It is also possible that the observed reduction in notifications from the general public is due to a decline in community awareness about the risks associated with potential exposure.

The trend in notification numbers and rates was not linear. With only 12 data points, the fitted line gives a general picture of trend, showing that notifications decreased substantially over the first 4 years of the study and then seemed to plateau. The change in notification rates across the 3 periods of the study support the same conclusion. Retrospective notifications did not influence this general trend.

The change in gender of notifications during the study seems related to a reduction in reporting among volunteer bat carers as this group had the largest decline in numbers and proportion of female notifications. This conclusion is also supported by the fact that the majority of carers (62/84, 74%) in the largest volunteer bat care group in south east Queensland are female (personal communication R. Larkin, Department of Environment and Resource Management, 21 April 2010). Concerns remain about the potential for under-reporting of non-bite exposures among this group.

The notifications included some retrospective potential exposures associated with publicity about human cases during period 1. In recent years, with the exception of 1 delayed report in period 3, the interval between potential exposure and reporting has remained short. Reporting from medical practitioners has been consistently prompt. The overall improvement in reporting times suggests that those people who sought medical attention for a potential exposure were aware of the importance of prompt medical assessment for a bat related injury, if not the importance of avoiding intentional handling. Public health messages should continue to emphasise that members of the public can be of most help to orphaned or injured bats by contacting a trained, vaccinated bat handler.

The decline (from period 2 to period 3) in the proportion of notifications where the bat was available for testing is important because of the resultant increase in the need for post-exposure prophylaxis, especially rabies immunoglobulin, which is in short supply. However, public health messages should continue to reinforce that people should not risk potential exposure (or further potential exposure) in order to detain a bat for testing.

Reported potential and confirmed exposures to ABLV declined during the study. Further research is required to determine whether this is a genuine reduction in potential exposures, under-reporting, or a combination of the two. The plateau of notifications more recently and the lethality of the infection demand ongoing public health measures to improve and sustain public awareness of the potential for exposure to ABLV.

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We thank Bruce Harrower, Public Health Virology, Forensic and Scientific Services, Queensland Health, Australia for reviewing the aspects of the database relating to the tested bats.

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References

1. Gould AR, Hyatt AD, Lunt R, Kattenbelt JA, Hengstberger S, Blacksell SD. Characterisation of a novel lyssavirus isolated from Pteropid bats in Australia. *Virus Res* 1998;54(2):165–187.

2. Allworth A, Murray K, Morgan J, A human case of encephalitis due to a lyssavirus recently identified in fruit bats, *Commun Dis Intell* 1996;20(24):504.

3. Hanna JN, Carney IK, Smith GA, Tannenberg AE, Deverill JE, Botha JA, et al. Australian bat lyssavirus infection: a second human case, with a long incubation period. *Med J Aust* 2000;172(12):597–599.

4. McCall BJ, Epstein JH, Neill AS, Heel K, Field H, Barrett J, et al. Potential exposure to Australian bat lyssavirus, Queensland, 1996–1999. *Emerg Infect Dis* 2000;6(3):259–264.

5. Communicable Diseases Network Australia, Australian Government Department of Health and Ageing, Department of Agriculture, Fisheries and Forestry – Australia. *Australian Bat Lyssavirus, Hendra Virus and Menangle Virus Information for Veterinary Practitioners*. Australian Government Department of Health and Ageing; Canberra, August 2001.

6. National Health and Medical Research Council. *The Australian Immunisation Handbook* 9th edn. Canberra, Australia: Australian Government Department of Health and Ageing; 2008; pp. 110–119.

7. Scott JG. Australian bat lyssavirus: the public health response to an emerging infection. *Med J Aust* 2000;172(12):573–574.

8. Young, MK, McCall BJ, Trends in potential exposure to Australian bat lyssavirus in South East Queensland, 1996 to 2003. *Commun Dis Intell* 2004;28(2):258–260.

9. Australian Bureau of Statistics. Population by Age and Sex, Australia 2006. Catalogue no. 3235.0. Canberra, Australia: Australian Bureau of Statistics, 2007.

10. Australian Bureau of Statistics. 2000 Estimated Resident Population by Statistical Local Area. Cat. no. 3235.3. Canberra, Australia: Australian Bureau of Statistics, 2001.

Short reports

Zoonotic tuberculosis: on the decline

Paul R Ingram, Peter Bremner, Tim J Inglis, Ronan J Murray, Debbie V Cousins

Abstract

Mycobacterium bovis is a zoonotic member of the *Mycobacterium tuberculosis complex* responsible for a clinical syndrome indistinguishable from that due to *M. tuberculosis*. In Australia, infection with *M. bovis* has historically been associated with employment in the livestock industry or immigration from countries in which animal disease is endemic. It currently accounts for 0.2% of all human cases of tuberculosis within Australia. This paper describes a case of pulmonary *M. bovis* in a butcher and reviews factors responsible for the declining incidence of this disease in Australia. *Commun Dis Intell* 2010;34(3):339–344.

Keywords: *Mycobacterium bovis*, incidence, tuberculosis

Introduction

Mycobacterium bovis is a member of the *Mycobacterium tuberculosis* complex (MTBC) responsible for a clinical syndrome indistinguishable from that due to *M. tuberculosis*. It was first discovered in 1901 when Robert Koch observed that bacilli isolated from tuberculous lesions in humans differed from those in cattle.[1] The subsequent description of *M. bovis* disease in a butcher gave origin to the concept of 'zoonotic tuberculosis'.[1] Since then several other members of the MTBC have been shown to be zoonoses, including *M. pinnipedii* (seals), *M. microti* (rodents) and *M. caprae* (cattle).[2] *M. bovis* is acquired by gastrointestinal, percutaneous or respiratory routes. Human-to-human spread and laboratory acquired infection have been reported.[3] Currently, the incidence of *M. bovis* infection in Australia is low: only 1–2 cases have been notified per year since 2000.[4] This case highlights the potential occupational hazard created by this pathogen and is followed by a review of the factors responsible for the declining frequency of this disease in Australia.

Case report

A 52-year-old male presented in February 2009 with a 1 month history of a non-productive cough, vague abdominal discomfort, night sweats and unexplained weight loss. He was born in Italy and migrated to Australia in 1998. He had worked as a butcher for the past 35 years. He denied working with diseased animals in Australia, but recalled slaughtering animals suspected to have bovine tuberculosis several decades ago. This process was often accompanied by dissection of the diseased lungs. He also drank unpasteurised dairy products whilst in Italy. He had a 30 year history of cigarette smoking, had no co-morbidities and took no medications. He had received the bacillus Calmette-Guérin (BCG) vaccination during childhood. His father had previously been treated for tuberculosis.

Physical examination revealed no significant abnormalities. A chest X-ray showed a well defined density in the left upper zone. A CT scan demonstrated several left upper lobe nodular densities up to 4 cm in diameter with some areas of cavitation (Figure 1). Bronchoalveolar lavage showed no acid fast bacilli on microscopy, but cultured slow-growing, cream coloured, cauliflower like colonies confirmed to be *M. bovis* by genotypic methods. The patient was successfully treated with a 9 month course of isoniazid, rifampicin and ethambutol. Contact tracing among close family members and work colleagues revealed no evidence of secondary cases.

Declining incidence of *Mycobacterium bovis* infection in Australia

The inherent difficulties in culturing mycobacteria and failure to differentiate between the several species within the MTBC have limited our understanding of the incidence of *M. bovis* infection. Reports from England in the early 20th century attributed 5%–30% of all cases of tuberculosis to *M. bovis*.[5] In this setting, it classically caused abdominal lymphadenitis in children following consumption of unpasteurised milk. The first report of human *M. bovis* disease in Australia, published in 1932, described high proportions of *M. bovis* in cervical (56%) and mesenteric (69%) tissue from children.[6] Following the advent of pasteurisation in the 1950s, the incidence of *M. bovis* infection declined and the epidemiology

Figure 1: Thoracic CT scan demonstrating cavitating left upper lobe nodular densities up to 4 cm in diameter

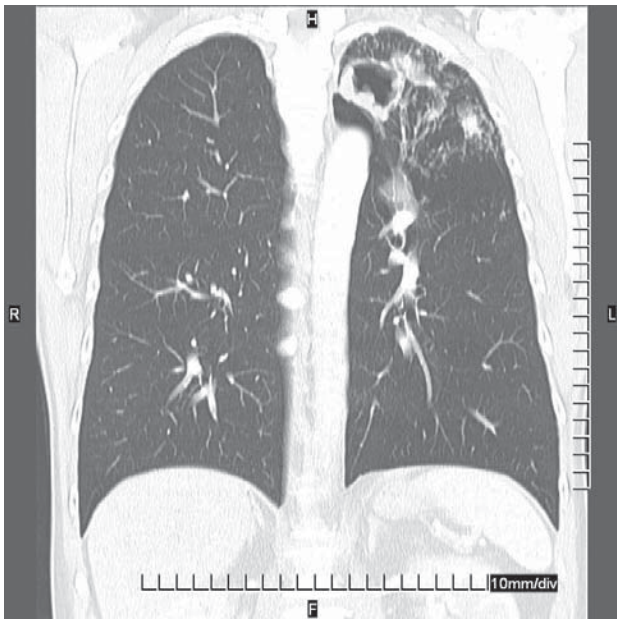
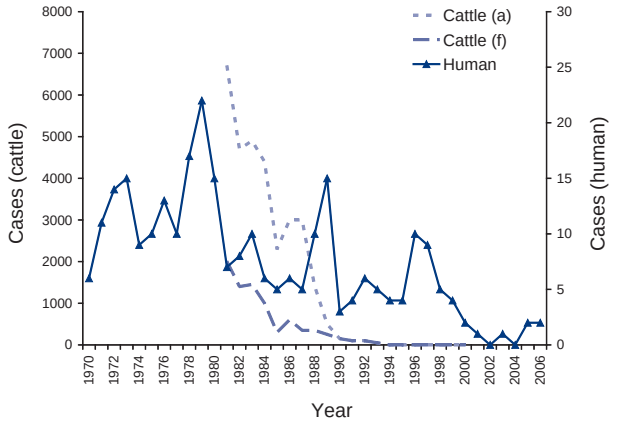


Figure 2: Annual number of human and cattle *Mycobacterium bovis* infections, Australia



(a) Detected during abattoir monitoring.

(f) Detected during field testing.

Data obtained from references 3, 4 and 12.

shifted towards pulmonary disease in adults with occupational exposure to cattle, or in immigrants. In a study of 236 cases in Australia between 1970 and 1994, 70% were male, the median age was 51–60 years and 52% reported working in the meat or livestock industry.[3]

The threat of trade restrictions, loss of productivity, and to a lesser extent, the risk of human disease led to the development of the Brucellosis and Tuberculosis Eradication Campaign (BTEC) in 1970. Targeting reservoirs of disease in cattle, this program focussed on herd testing (by tuberculin skin testing), abattoir inspections, contact tracing, culling of diseased animals and changes in animal husbandry—at the expense of over $1 billion.[7] Eradication was aided by the absence of feral maintenance host in Australia other than buffalo, which were included in the campaign from 1984. The impact of BTEC was dramatic, resulting in Australia being declared a free area with respect to bovine tuberculosis by 1997, using the criteria specified by the Office Internationale Epizooties.[8] Currently the livestock industry remains in a surveillance phase, focussing on meat inspection and granuloma detection in suspect carcasses. The last case of *M. bovis* infection detected in cattle was in 2000, while the last case detected in buffalo was in 2002 in the Northern Territory.

The incidence of *M. bovis* disease in humans has paralleled that seen in cattle since the 1970s (Figure 2). Currently, *M. bovis* is responsible for 0.2% of all cases of human tuberculosis within Australia.[4] Cases most frequently represent reactivation of latent disease, often acquired decades ago. Historically, employment in the livestock industry has been a risk factor.[3] Pre-employment screening and/or BCG vaccination for abattoir workers has previously been advocated.[9] Occasionally, iatrogenic disease due to live attenuated *M. bovis* (BCG strain) is seen following vaccination or intravesical therapy for bladder malignancy.[10,11]

Global trends in the incidence of *M. bovis* in humans and animals differ between the developed and the developing world. Countries with the capacity to implement the same control measures as Australia have experienced steep reductions in *M. bovis* human disease. In the United States of America the proportion of tuberculosis due to *M. bovis* is 1.4% and in the United Kingdom <1%.[13,14] Ongoing transmission between cattle and wild species (including the brushtail possum [*Trichosurus vulpecula*] in New Zealand)[5] is thought to explain the significant proportion (2.7%) of disease due to *M. bovis* in New Zealand.[15] By contrast, the proportion of human tuberculosis due to *M. bovis* in less developed countries such as Mexico (13.8%), Uganda (6.9%) and Nigeria (5%),[16] probably reflects consumption of unpasteurised dairy products from diseased animals as well as closer contact with infected bovines. In this setting, the potential impact on human health and food supply recently prompted the World Health Organization to classify bovine tuberculosis a 'neglected zoonosis'.[17]

This case reminds us that, despite success in eradicating this zoonoses from our livestock, reactivation of latent disease and migration of populations will result in ongoing, infrequent

M. bovis human disease in Australia. Vigilant surveillance and species level identification, applied to both sides of the animal-human interface, remain important control measures for this pathogen.

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References

1. Schmiedel A. Development and present state of bovine tuberculosis in man. *Bull Int Union Tuberc* 1968;40:5–32.

2. Animal Health Australia. *Bovine Tuberculosis Case Response Manual, 2007.* Primary Industries Ministerial Council, Canberra, ACT; 2007.

3. Cousins DV, Dawson DJ. Tuberculosis due to *Mycobacterium bovis* in the Australian population: cases recorded during 1970–1994. *Int J Tuberc Lung Dis* 1999;3(8):715–721.

4. Australian Government Department of Health and Ageing. Tuberculosis notifications in Australia annual reports. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-annlrpt-tbannrep.htm

5. Cousins DV. *Mycobacterium bovis* infection and control in domestic livestock. *Rev Sci Tech* 2001;20(1):71–85.

6. Webster R. Tuberculosis in childhood: the incidence of bovine infection in Victoria. *Med J Aust* 1932;1:315–320.

7. Radunz B. Surveillance and risk management during the latter stages of eradication: experiences from Australia. *Vet Microbiol* 2006;112(2–4):283–290.

8. McCormick B. *StockGuard Cattle: Strategic Assessment– Bovine Tuberculosis.* Western Australia Department of Agriculture; 2001 Available from: http://www.agric.wa.gov.au/objtwr/imported_assets/content/pw/ah/dis/cat/bovine_tuberculosis.pdf

9. Roberts JL, Cousins DV, Matthews WR, McCormack BS, Radunz BL, Tolson JW. Australia—a free area for bovine tuberculosis. Proceedings of the *XX World Association for Buiatrics Congress;* 21–25 July 1998; Sydney, Australia.

10. Robinson P, Morris D, Antic R. *Mycobacterium bovis* as an occupational hazard in abattoir workers. *Aust N Z J Med* 1988;18(5):701–703.

11. Nuttall JJ, Davies MA, Hussey GD, Eley BS. Bacillus Calmette-Guérin (BCG) vaccine-induced complications in children treated with highly active antiretroviral therapy. *Int J Infect Dis* 2008;12(6):e99–e105.

12. Coscas R, Arlet JB, Belhomme D, Fabiani JN, Pouchot J. Multiple mycotic aneurysms due to *Mycobacterium bovis* after intravesical bacillus Calmette-Guérin therapy. *J Vasc Surg* 2009;50(5):1185–1190.

13. Hlavsa MC, Moonan PK, Cowan LS, Navin T, Kammerer JS, Morlock GP, et al. Human tuberculosis due to *Mycobacterium bovis* in the United States, 1995–2005. *Clin Infect Dis* 2008;47(2):168–175.

14. Evans JT, Smith EG, Banerjee A, Smith RM, Dale J, Innes JA, et al. Cluster of human tuberculosis caused by *Mycobacterium bovis:* evidence for person-to-person transmission in the UK. *Lancet* 2007;369(9569):1270–1276.

15. Baker MG, Lopez LD, Cannon MC, De Lisle GW, Collins DM. Continuing *Mycobacterium bovis* transmission from animals to humans in New Zealand. *Epidemiol Infect* 2006;134(5):1068–1073.

16. Michel AL, Müller B, van Helden PD. *Mycobacterium bovis* at the animal-human interface: a problem or not? *Vet Microbiol* 2010;140(3–4):371–381.

17. World Health Organization. The Control of Neglected Zoonotic Diseases. WHO/SDE/FOS/2006.1. Report of a Joint WHO/DFID-AHP Meeting with the participation of FAO and OIE. Geneva, 20 and 21 September 2005. Available from: http://www.who.int/zoonoses/Report_Sept06.pdf

Penicillin-resistant *Neisseria meningitidis* bacteraemia, Kimberley region, March 2010

Shivanti D Abeysuriya, David J Speers, Jackie Gardiner, Ronan J Murray

Abstract

A 4-year-old fully immunised male presented to a regional hospital in the West Kimberley with fever and lethargy. Blood cultures yielded serogroup B *Neisseria meningitidis*, resistant to benzylpenicillin (minimum inhibitory concentration (MIC) 1.0 mg/L). The patient was treated with intravenous ceftriaxone and made a complete recovery. Although invasive *N. meningitidis* isolates with reduced penicillin susceptibility are not uncommon in Australia, this is the first report of a benzylpenicillin-resistant isolate (MIC > 0.5 mg/L) causing invasive disease. As benzylpenicillin is currently recommended as first line empiric and definitive therapy for invasive meningococcal disease, the emergence of penicillin-resistant *N. meningitidis* disease is of concern and emphasises the importance of ongoing surveillance for antimicrobial resistance. *Commun Dis Intell* 2010;34(3):324–326.

Keywords: *Neisseria meningitidis;* penicillin resistant; meningococcus; meningococcal disease

Case report

A 4-year-old fully immunised male presented to a regional hospital in the West Kimberley with fever and lethargy. On examination, he was febrile (T = 39.4°C), tachycardic (pulse rate 160 bpm) and tachypnoeic (respiratory rate 26 per minute), however there was no rash or signs of meningism. Blood cultures yielded serogroup B *Neisseria meningitidis*. The patient was treated with intravenous ceftriaxone 900 mg for 5 days and made a complete recovery. A lumbar puncture performed 72 hours after commencing ceftriaxone was negative for *N. meningitidis* on culture and by polymerase chain reaction.

Antimicrobial susceptibility testing was performed in the routine microbiology laboratory by Etest® (AB Biodisk, Solna, Sweden) and results interpreted according to Clinical Laboratory Standards Institute (CLSI) breakpoints.[1] Etest® minimum inhibitory concentration (MIC) results were as follows: benzylpenicillin, 0.5 mg/L (resistant); ceftriaxone, 0.004 mg/L (susceptible); ciprofloxacin, 0.006 mg/L (susceptible); rifampicin 0.012 mg/L (susceptible) and chloramphenicol, 1 mg/L (susceptible). The isolate was beta-lactamase negative by nitrocefin testing.

The isolate was referred to the National Neisseria Network Reference Laboratory, Prince of Wales Hospital, New South Wales for confirmatory susceptibility testing. The identification of the organism was confirmed and susceptibility testing for benzylpenicillin was performed using two alternative methods (Calibrated Dichotomous Susceptibility (CDS) disc testing and MIC determination using agar dilution and CLSI breakpoints). There was no zone to the Pen0.5u disc by the CDS method, indicating resistance, which was confirmed by the MIC method and demonstrated a benzylpenicillin MIC of 1.0 µg/mL (resistant).

Genosubtyping of the *N. meningitidis* isolate was performed by *por*A gene variable region (VR) 1 and 2 DNA sequencing as previously described.[2] When the deduced amino acid sequences of VR1 and VR2 were submitted to the *N. meningitidis por*A VR database (http://neisseria.org/nm/typing/pora), there were only partial matches to VR1 peptides 5–29 (56%) and 21–14 (60%) and VR2 peptides 2–39 (67%) and 16–107 (46%). When compared to a Western Australian database of 81 *N. meningitidis* isolates strains (including 7 from the Kimberley) collected from 2000–2006[3] this genosubtype had not previously been identified.

Discussion

Highly resistant (benzylpenicillin MIC > 256 mg/L), beta-lactamase producing *N. meningitidis* isolates have been sporadically reported from Canada, South Africa, and Spain.[4] However, beta-lactamase-negative *N. meningitidis* strains with increased benzylpenicillin MICs of > 0.06 mg/L have been isolated more commonly from the United Kingdom, Europe, Greece, South America, South Africa, Asia and the United States of America (USA). These relatively resistant *N. meningitidis* isolates have penicillin MICs ranging from 0.01 mg/L to 1 mg/L.[4] Reduced susceptibility in these isolates is due to decreased binding of benzylpencillin due to altered penicillin- binding proteins (PBP2 and PBP3).[4]

In 2008, 108 of 149 (72%) invasive *N. meningitidis* isolates submitted to the Australian National Neisseria Network demonstrated reduced susceptibility to benzylpenicillin (MICs 0.06–0.5 mg/L).[5] To date, this is the first report of an invasive *N. meningitidis* isolate with a benzylpenicillin MIC > 0.5 mg/L from Australia (personal communication, John Tapsall, National Neisseria Network Reference Laboratory).

The clinical significance of reduced penicillin susceptibility in *N. meningitidis* is unclear. Treatment failures and higher rates of complications have been observed, although administration of higher doses of penicillin has been reported as clinically effective.[4,6,7,8] Several reports indicate that there is no association between invasive meningococcal disease with decreased susceptibility to penicillin and mortality.[6,8] Current Australian guidelines recommend benzylpenicillin for the treatment of proven meningococcal meningitis, irrespective of penicillin susceptibility.[9] Current USA recommendations for the treatment of bacterial meningitis[10] recommend therapy with third-generation cephalosporins (ceftriaxone or cefotaxime) for meningococcal meningitis until susceptibilities are available, and recommends penicillin or ampicillin for *N. meningitidis* isolates with penicillin MICs of <0.1 mg/L and third-generation cephalosporins for isolates with MICs of 0.1–1.0 mg/L.[10,11]

Decreased susceptibility to benzylpenicillin in invasive *N. meningitidis* isolates is now common in Australia,[5] but fortunately benzylpencillin resistance appears to be rare. This report highlights the importance of culture and susceptibility testing in invasive meningococcal disease, and of ongoing national surveillance for antimicrobial resistance in *N. meningitidis*.

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References

1. Clinical Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. Twentieth Informational Supplement (CLSI) M100-S20. 2010;30(1):101.

2. Jelfs J, Munro R, Wedege E, Caugant DA. Sequence variation in the *por*A gene for a clone of *Neisseria meningitidis* during epidemic spread. *Clin Diagn Lab Immunol* 2000;7(3):390–395.

3. Pereira L, Harnett G, Chidlow G, Speers D. Prevalence of genosubtypes (*porA* types) of invasive meningococcal disease in Western Australia from 2000 to 2006. *Pathology* 2008;40(7):728–729.

4. Janda WM, Gaydos CA. *Neisseria*. In: Murray PR, Baron EJ, JH Hoergensen JH, Landry ML, Pfaller MA, eds. *Manual of Clinical Microbiology*. Washington: ASM press; 2007 pp. 601–620.

5. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2008. *Commun Dis Intell* 2009;33(3):259–267.

6. Plessis M, Gottberg A, Cohen C, Gouveia L, Klugman K. *Neisseia meningitidis* intermediately resistant to penicillin and causing invasive disease in South Africa in 2001–2005. *J Clin Microbiol* 2008;46(10):3208–3214.

7. Hedberg ST, Fredlund H, Nicholas P, Caugant DA, Olcen P, Unemo M. Antibiotic susceptibility and characteristics of *Neisseria meningitidis* isolates from the African meningitis belt, 2000 to 2006: phenotypic and genotypic perspectives. *Antimicrob Agents Chemother* 2009;53(4):1561–1566.

8. Brown EM, Fisman DN, Drews SJ, Dolman S, Rawte P, Brown S, et al. Epidemiology of invasive meningococcal disease with decreased susceptibility to penicillin in Ontario, Canada 2000 to 2006. *Antimicrob Agents Chemother* 2010;54(3):1016–1021.

9. Antibiotic Writing Group. Central nervous system infections. Therapeutic guidelines – Antibiotic. 2006; version 13: pp 55–68.

10. Tunkel AR, Hartman BJ, Kaplan SL, Kaufman BA, Roos KL, Sceld WM, et al. Practice guidelines for the management of bacterial meningitis. *Clin Infect Dis* 2004;39(9):1267–1284.

11. Apicella MA. *Neisseria meningitidis*. In: Mandell GL, Bennett JE, Dolin R eds. Mandell, Douglas and Bennett's. *Principles and Practice of Infectious Disease*. Elsevier Churchill Livingstone 2010; pp. 2737–2752.

Quarterly reports

OzFoodNet quarterly report, 1 April to 30 June 2010

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established OzFoodNet in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness, coordinates national investigations into outbreaks of foodborne disease, develops nationally standardised protocols and tools for surveillance, identifies foods or commodities that may cause human illness and trains people to investigate foodborne illness. This quarterly report documents investigation of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 April to 30 June 2010.

Data were received from OzFoodNet epidemiologists 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 2nd quarter of 2010, OzFoodNet sites reported 391 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric illness. In total, these outbreaks affected 7,275 people, of whom 154 were hospitalised. There were 24 deaths reported during these outbreaks. The majority of outbreaks (84%, n=327) were due to person-to-person transmission (Table 1).

Foodborne and suspected foodborne disease outbreaks

There were 35 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 771 people and resulted in 58 hospitalisations. There were 8 deaths reported during these outbreaks. This compares with 27 foodborne outbreaks for the 2nd quarter in 2009[1] and the 5-year average of 27 between 2005 and 2009, and 45 foodborne outbreaks during the 1st quarter of 2010.[2]

Salmonella was the aetiological agent for 10 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype (n=8). Of the remaining 25 outbreaks, four were due to norovirus, two each due to *Clostridium perfringens*, *Listeria monocytogenes* and *Campylobacter*, and one due to *Cyclospora*. For 14 outbreaks, the aetiological agent was unknown or not specified.

Sixteen outbreaks (46%) reported in this quarter were associated with food prepared in restaurants, six (17%) with aged care facilities, five (14%) with takeaway food outlets, and two (6%) from within the community. Single outbreaks (3%) were associated with a bakery, a camp, a cruise, a national franchised fast food outlet, a training facility and a commercial caterer.

To investigate these outbreaks, sites conducted 7 cohort studies, 2 case control studies and collected descriptive case series data for 23 investigations. Individual patient data were not collected for 3 outbreaks. As evidence for the implicated food vehicle, investigators obtained both microbiological and analytic evidence for 3 outbreaks, relied on microbiological evidence in 5 outbreaks and analytical evidence alone for 1 outbreak. Descriptive evidence alone was obtained in 26 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred in this quarter.

Table 1: Mode of transmission for outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 April to 30 June 2010

Transmission mode	Number of outbreaks	Percentage of total
Foodborne and suspected foodborne	35	9
Person-to-person	327	84
Unknown (*Salmonella* cluster)	8	2
Unknown	21	5
Total	391	100

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 April to 30 June 2010 (n=35)

State or territory	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
NSW	March	Restaurant	*S.* Typhimurium 170	6	3	M	Tartare sauce, prepared with raw egg
	April	Aged care facility	*S.* Infantis	26	5	D	Suspected fluid thickener contaminated by raw chicken mince
	April	Takeaway	*S.* Typhimurium 170	9	0	M	Mayonnaise made with raw egg
	April	National franchised fast food	*S.* Typhimurium 9	4	1	D	Unknown
	April	Restaurant	*S.* Typhimurium 170	19	0	D	Suspected peanut/cashew mix
	April	Takeaway	Norovirus	13	0	D	Suspected variety of ready to eat foods: sandwiches, salads, wraps
	May	Restaurant	Unknown	7	0	D	Unknown
	May	Takeaway	Unknown	2	0	D	Suspect Mongolian lamb/fried rice
	May	Commercial caterer	*S.* Saintpaul	7	3	D	Unknown
	May	Restaurant	*Campylobacter jejuni*	10	0	AM	Raw chicken
	May	Restaurant	Unknown	32	1	D	Unknown
	June	Restaurant	Unknown	4	0	D	Unknown
	June	National franchised fast food	Unknown	9	0	D	Unknown
	June	Restaurant	Unknown	3	0	D	Unknown
	June	Restaurant	Unknown	12	0	D	Unknown
	June	Restaurant	Unknown	7	0	D	Unknown
	June	Restaurant	*S.* Typhimurium 170	16	9	D	Suspected fried rice
	June	Restaurant	Unknown	11	0	D	Unknown
	June	Takeaway	*S.* Typhimurium 170	45	8	M	Chicken, hummus, tabouli
NT	June	Restaurant	Norovirus	19	0	D	Unknown
Qld	May	Bakery	*S.* Typhimurium	19	2	AM	Cheesecake, meat pies
	May	Restaurant	Norovirus	11	0	D	Unknown
	May	Restaurant	Norovirus	12	0	D	Unknown
	June	Restaurant	*S.* Typhimurium	34	1	AM	Citrus aioli
	June	Restaurant	*Clostridium perfringens*	4	0	M	Roti curry lamb
SA	April	Camp	Unknown	43	0	D	Unknown
	June	Other	Unknown	10	10	D	Unknown

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 April to 30 June 2010, *continued*

State or territory	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic	February	Community	*Listeria monocytogenes*	6	6	D	Still under investigation
	April	Aged care facility	Unknown	6	0	D	Unknown
	May	Aged care facility	Unknown	9	0	D	Unknown
	June	Aged care facility	Unknown	8	0	D	Unknown
	June	Aged care facility	*C. jejuni* and *C. coli*	15	1	D	Unknown
WA	May	Cruise/airline	Cyclospora	314	0	A	Cantaloupe, mint, lettuce
	June	Aged care facility	*C. perfringens*	10	0	D	Unknown
Multi-jurisdictional	February to current	Community	*L. monocytogenes*	9	8	M	Melons and/or melons contained within fruit salads

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

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

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

The month of outbreak represents the month of onset of outbreak.

* No foodborne outbreaks were reported by the Australian Capital Territory or Tasmania.

Australian Capital Territory

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter. However, a confirmed case of *S.* Typhimurium phage type 170 infection was linked to fried ice cream served at a Vietnamese restaurant in Sydney where five of 6 family members reported gastroenteritis following a lunch in April. Investigation and liaison with the NSW Department of Health and the New South Wales Food Authority were undertaken.

New South Wales

There were 19 reported outbreaks of foodborne or suspected foodborne illness during the quarter, with eight of these being due to *Salmonella*.

- Six cases of *S.* Typhimurium phage type 170, multi-locus variable number of tandem repeats analysis (MLVA) profile* 3-9-7-12-523 were identified through a cluster investigation. The same strain was also isolated from a tartare sauce prepared with raw egg and consumed by four of the 6 cases. Two of the cases were children of one of the food handlers at the café. They had not consumed any food from the café, suggesting some person-to-person transmission.

- A group of 7 work colleagues all developed abdominal cramps and diarrhoea after consuming chicken rolls that contained raw egg mayonnaise from a Vietnamese hot bread bakery. There were two other separate complaints about the same store around the same time. Three stool samples (one from the group of work colleagues and two from the separate complainants) were positive for *S.* Typhimurium phage type 170, MLVA type 3-9-7-13-523. A sample of the raw egg mayonnaise was positive for *S.* Typhimurium with the same MLVA profile.

- An outbreak of 31 confirmed cases of *S.* Typhimurium phage type 170, with one of 3 different MLVA profiles (3-9-7-13-523 (n=1), 3-9-7-14-523 (n=16), and 3-9-7-15-523 (n=14)) was identified through enhanced surveillance following 2 separate complaints. Cases had all consumed kebabs, mainly those filled with chicken, hummus, tabouli, lettuce, and tomato, or crepes from a food outlet in a shopping centre. A further 14 probable cases were also identified during the investigation. Samples of cooked chicken kebab, hummus and tabouli and several environmental samples were positive for *S.* Typhimurium MLVA profile 3-9-7-13-523. One environmental swab was positive for both *S.* Typhimurium phage type 170 and *S.* Typhimurium phage type 193. A sample of marinated raw chicken was positive for *S.* Infantis.

- A cluster of 9 cases of *S.* Typhimurium phage type 170, MLVA profile 3-9-7-12-523 was identified through follow-up of routine surveillance. Six cases had become unwell after dining at the same Thai restaurant. A further 10 cases reported being ill after dining with confirmed cases. A sample of a peanut/cashew mixture tested positive for *S.* Typhimurium MLVA profile 3-9-8-15-523. The MLVA profiles for the human isolates and the peanut/cashew mixture would be considered too different to be a match. However, both MLVA profiles are associated with phage type 170 and there is a clear epidemiological link to the restaurant and the peanut/cashew mixture, which is sprinkled on many of the dishes.

- Five people from a cluster of 16 cases of *S.* Typhimurium phage type 170, MLVA profile 3-9-7-13-523 had eaten fried rice at the same Chinese food outlet in a shopping mall. Two other cases had eaten at other establishments in the same shopping mall, and 2 cases had eaten food in another restaurant in the area. No link between these premises could be established. Food samples and environmental swabs were all negative for *Salmonella*.

- A cluster of salmonellosis among 3 members of one household, and a friend who was often at the home was investigated. Stool samples for all 4 cases were positive for *S.* Typhimurium phage type 9, MLVA profile 3-10-13-12-496. The only food shared by all was chicken pieces, purchased from a large fried chicken franchise outlet, and consumed at the home.

- An outbreak of salmonellosis in an aged care facility affected 26 people. Twenty-two residents and 1 staff member (not the index case) tested positive for *S.* Infantis and a further 3 residents had symptoms consistent with salmonellosis. Raw chicken mince sampled at the facility was also positive for *S.* Infantis. Epidemiological analysis found a strong association with the consumption of thickened fluids, which are drinking fluids to which a thickening agent is added to aid consumption by people with swallowing difficulties. However, a sample of the batch of powder used to thicken fluids at the time of the outbreak tested nega-

* Australian nomenclature used in New South Wales.

tive for any pathogens. Cross contamination from the chicken mince to the thickened fluid powder is suspected.

- Seven cases of *S*. Saintpaul were associated with consumption of salmon steak and pumpkin couscous salad served with a lemon aioli prepared with a commercially manufactured mayonnaise at a winery during a food and wine festival in the Hunter vineyards. Investigations were unable to identify how the contamination occurred or what ingredient was the cause of the outbreak. No environmental or food samples were taken.

The other foodborne investigations included an investigation of *Campylobacter jejuni* associated with the consumption of chicken affecting 10 people from a group of 16 who shared a buffet meal at a restaurant. The only 2 submitted stool specimens were both positive for *Campylobacter*, which was also detected in a sample of raw chicken. Epidemiological analysis showed a significant association between illness and consumption of the chicken curry (attack rate of 91%, relative risk undefined, $P = 0.004$). Further typing to establish a genetic similarity between the human and food isolates was not possible as the human specimens had been discarded.

A norovirus outbreak affecting 13 people in a workplace was found to be associated with commercially pre-prepared ready-to-eat foods. The symptoms profile was consistent with norovirus, with the pathogen detected in 1 stool specimen. The New South Wales Food Authority conducted an environmental investigation of the premises and identified at least 1 food handler who was symptomatic with gastroenteritis whilst working during the exposure period. The New South Wales Food Authority is considering further action.

There were a further 9 reports of suspected foodborne outbreaks during the quarter that were of unknown aetiology. One outbreak affected 26 of 60 people attending a wedding, and a further 6 secondary cases who became ill one incubation period (24–48 hours) later. It is suspected that the outbreak was caused by a viral pathogen, most likely norovirus, but it was not possible to ascertain the type of food that was the likely source, nor whether the outbreak was a result of the consumption of food contaminated by a food handler or by an environmental source. Another 6 outbreaks occurred in restaurant settings affecting 44 people, 1 outbreak was associated with a takeaway outlet affecting 2 people, and 1 outbreak was associated with a national fast food outlet affecting 9 people.

Northern Territory

There was 1 reported outbreak of foodborne or suspected foodborne illness during the quarter. This outbreak occurred amongst 2 different groups of attendees at a hotel restaurant who had eaten from a common menu on the same day. Food was prepared at the hotel. A cohort study was performed but did not identify a particular food vehicle. Of the 19 people affected, 1 faecal specimen was tested and was positive for norovirus. It is thought that wide-spread contamination of food or the environment at the functions could have occurred from a food handler, a staff member or an attendee of the function.

Queensland

There were 5 reported outbreaks of foodborne or suspected foodborne illness during the quarter. Four females aged between 27 and 41 years became ill with diarrhoea and abdominal cramps following the consumption of lamb curry at a restaurant in June. *C. perfringens* was cultured in a sample of lamb curry and in 2 faecal specimens. Cooking large volumes in conjunction with temperature abuse of food were identified as major contributing factors following the environmental health inspection.

An outbreak of 19 cases of *S.* Typhimurium was identified among residents in South East Queensland in May. Eighteen of the 19 cases of *S.* Typhimurium had the same MLVA profile† (1-1-8-2-9) and 1 case had a closely related MLVA profile (1-1-9-2-9). A large proportion of cases had reported consuming pies and/or cheesecake from the same bakery franchise within 5 days prior to illness. Extensive environmental sampling was conducted at both the individual franchise store level and a central manufacturing facility but the outbreak strain was not detected in any samples. However, another strain of *S.* Typhimurium (MLVA profile: 1-13-19-2-3) was detected from an egg wash sample. Egg wash, used for glazing pies, was supplied to franchises by the central manufacturing facility. It was concluded that multiple food vehicles were associated with the outbreak and that eggs were the likely source of infection.

A public health unit was alerted to a suspected foodborne outbreak among guests who had attended a wedding function in early June. A retrospective cohort study was conducted with 34 cases of gastroenteritis identified among 77 guests interviewed. Twelve of the 34 cases had faecal samples positive for *S.* Typhimurium

† Lindstedt nomenclature used in Queensland.

(MLVA profile 1-5-5-2-3). The epidemiological study found that guests who had consumed a barramundi meal served with a citrus aioli sauce were significantly more likely to develop illness compared with persons who had not eaten this meal (RR 4.1, 95% confidence interval (CI) 1.9 to 8.8). The same strain of *S.* Typhimurium was isolated from a sample of citrus aioli taken from the restaurant kitchen and whole egg samples were positive for *S.* Anatum, *S.* Mbandaka and *S.* Montevideo. The investigation identified that there was no heat treatment of the aioli sauce after the addition of raw egg yolk to the mixture. A traceback investigation sourced the eggs to a single egg producer, where several serotypes of *Salmonella* were detected from sheds and whole cage eggs, including the outbreak strain. A consumer level recall of cage eggs produced by the implicated farm was conducted based on these findings and the detection of multiple cartons of cage eggs at the farm and at retail level that contained eggs that appeared visibly contaminated with faecal matter. The function venue changed to using a commercially produced aioli. The egg producer, with the guidance of Safe Food Production Queensland, undertook reforms to their processes to enable the business to meet the Queensland Food Safety Scheme for Eggs and Egg Products.

Following the outbreak described above, community-acquired cases of the outbreak strain (*S.* Typhimurium MLVA profile 1-5-5-2-3) and cases of *S.* Montevideo, *S.* Anatum, *S.* Mbandaka and *S.* Tennessee notified prior to the consumer level recall date were investigated for possible exposure to eggs from the implicated farm. Eleven cases of *S.* Typhimurium MLVA profile 1-5-5-2-3 who did not attend the wedding, 2 cases of *S.* Montevideo and 1 case of *S.* Tennessee were notified from 1 June 2010. Of these, 4 cases of *S.* Typhimurium and 1 *S.* Montevideo case were epidemiologically linked to food businesses known to have been supplied eggs from the implicated farm.

Two outbreaks of norovirus genotype II were investigated. The 1st affected 11 people among 2 separate groups that consumed a meal at a café on different nights in the same week in May. Staff, including 2 waitresses and a chef, also fell ill but their onsets were reportedly on the same night as the patrons. The 2nd outbreak affected 8 patrons who attended a restaurant in May and 4 staff members. Both outbreaks were suspected viral foodborne outbreaks with person-to-food-to-person transmission, with 1 faecal specimen collected in each outbreak, both of which were positive for norovirus genotype II.

South Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

In the 1st outbreak, 43 of 90 attendees reported illness during a church camp held on the Anzac Day long weekend. A cohort study was conducted using an on-line study tool to investigate the cause of the illness. The investigation identified rice as the likely food vehicle due to biological plausibility and high attack rate (68.2%). However, an odds ratio could not be calculated as all attendees consumed this food. No left over food was available for testing.

In the 2nd outbreak, 10 of 40 trainers experienced vomiting illness within a very short time frame (20 minutes) after consuming food at a training facility. In addition to 40 trainers, there were 100 trainees at the training facility, however no trainees reported illness. Trainers and trainees did not consume the same foods. A case series was conducted to investigate the illness. The epidemiological and laboratory investigations did not identify an infectious cause of the illness.

Tasmania

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Victoria

There were 5 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Four outbreaks occurred in aged care facilities, three of which had no food source identified:

- Six residents all had an onset of diarrhoea on the same day. Two faecal specimens were collected and one had *C. perfringens* enterotoxin isolated.
- Eight residents all had an onset of diarrhoea or abdominal pain on the same day, and 1 staff member had an onset the following day. Duration of illness and symptoms were consistent with *C. perfringens* infection but all 3 faecal specimens collected were negative for bacterial and viral pathogens.
- Five residents and 3 staff members all had onsets of diarrhoea on the same day. Two faecal specimens were collected and were negative for

bacterial and viral pathogens, but clustered onsets, symptoms and duration were consistent with *C. perfringens*.

- Thirteen residents and 2 staff members had onsets of diarrhoea occurring over a 5-day period. Ten residents submitted faecal specimens and 3 residents were confirmed with *C. jejuni* and three were confirmed with *C. coli*. The cause of this outbreak was unable to be determined however, it was suspected that the outbreak was either caused by under-cooking of roast meats or through cross contamination of ready-to-eat foods during preparation.

One outbreak of listeriosis was reported with 6 cases with ages ranging from 55 to 86 years. All case isolates shared the same molecular serogroup, binary gene type (BT) and pulsed field gel electrophoresis (PFGE) pattern (molecular serogroup: 1a, BT: 155 and University of Melbourne Microbiological Diagnostic Unit (MDU) designated PFGE: 6:6:6A). Five of the cases spent part of their incubation period as inpatients or outpatients at the same hospital. Potential sources for this cluster are still under investigation at the time of writing this report.

Western Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

An outbreak caused by *Cyclospora* affected passengers and crew on 2 successive cruises of the same ship that departed from and returned to Western Australia in May and June 2010, and visited South East Asian destinations. Follow-up of laboratory confirmed cases and passenger enquiries identified 34 ill passengers associated with the 1st cruise, with 26 of these cases laboratory confirmed. From the 2nd cruise, 232 passengers and 48 crew members were reported to be affected, with 46 passengers and 1 crew member laboratory confirmed cases. The duration of illness ranged from 1 to 33 days, with a median of 6.5 days. The most common symptom for confirmed cases was diarrhoea, which was reported by 45 of the 47 cases for whom symptom information was recorded.

A case-control study was conducted among crew members, with questions focusing on fresh produce and water consumed on board, and on shore visits. There were 31 cases and 97 controls recruited into the study. Of the 117 exposure variables included in univariate analysis, nine were significant at a P value of < 0.01, with lettuce having the strongest association with illness (OR = 5.49, 95% CI 1.73–14.1, $P = 0.0005$). Drinking water on board was not associated with illness. Variables with P values < 0.1 (25 variables) were included in a backward stepwise logistic regression analysis. Eating in a speciality dining area, eating cantaloupe, mint and lettuce were significant in the logistic regression model ($P < 0.05$). It was concluded that illness was most likely related to eating fresh produce items taken on board during the 1st cruise, but the case-control study did not provide enough evidence to definitively determine which fresh produce item was the likely cause of illness.

In June, nine of 135 residents and 1 staff member of an aged care facility became ill with diarrhoea, with onset of illness over a 4-day period. The duration of diarrhoea for most cases was 2 days or less. The staff member was also ill with vomiting. Of the 9 ill residents, six consumed vitamised food. Two of 5 stool specimens tested positive for *C. perfringens*, with indistinguishable PFGE profiles, suggesting that infection had come from a common source, suspected to be a common food. Food was prepared on site. There were no remaining food samples from the period prior to onset of illness, and more recent food samples were negative for common bacterial pathogens and *C. perfringens*. An environmental investigation found satisfactory food handling practices and hand hygiene standards.

Multi-jurisdictional outbreak investigation

Listeriosis

OzFoodNet commenced a multi-jurisdictional outbreak investigation of listeriosis in May 2010 after notifications exceeded expected levels in the 1st quarter of 2010, with 12 cases per month compared with a 3-year average of 5.8 notifications per month. Increases were most apparent in New South Wales and Victoria (Figure).

Jurisdictions requested characterisation of all human isolates from cases notified in 2010. Nine cases met the outbreak case definition for a confirmed case: four from Victoria, two from Queensland and three from New South Wales. Seven of these were infected with a particular subtype (molecular serogroup: 1/2b, BT: 158, MDU designated PFGE: 121:119:1 with indistinguishable ribotype and multilocus sequence typing (MLST)), and two with a 2nd outbreak strain (molecular serogroup: not established, BT: 158 and MDU designated PFGE: 122:4N:1). Dates of onset were between 2 February and 23 May 2010 (n = 7), while the onset dates for the other 2 cases were unknown, but specimen dates for these cases were in April and May. These strains have not previously been known to have been isolated from human cases in Australia.

Outbreak cases were aged between 53 and 95 years of age and all would be considered immunocompromised. Fifty-six per cent 56% (5/9) were female and 88% (8/9) were hospitalised.

Preliminary investigations to identify a possible food vehicle showed a possible link to fruits and prepared fruit salads, with these foods having been consumed by more cases than expected when compared with data from the general community, and from similar vulnerable/immunocompromised people.[3] Of the outbreak strain cases, 44% (4/9) had eaten rockmelon (expected frequency 37.7%) and 33% (3/9) prepared fruit salad (expected frequency 12.9%) in the 4 weeks prior to onset.[3] Food exposure history has been difficult to ascertain for some recent cases due to the seriousness of their illness.

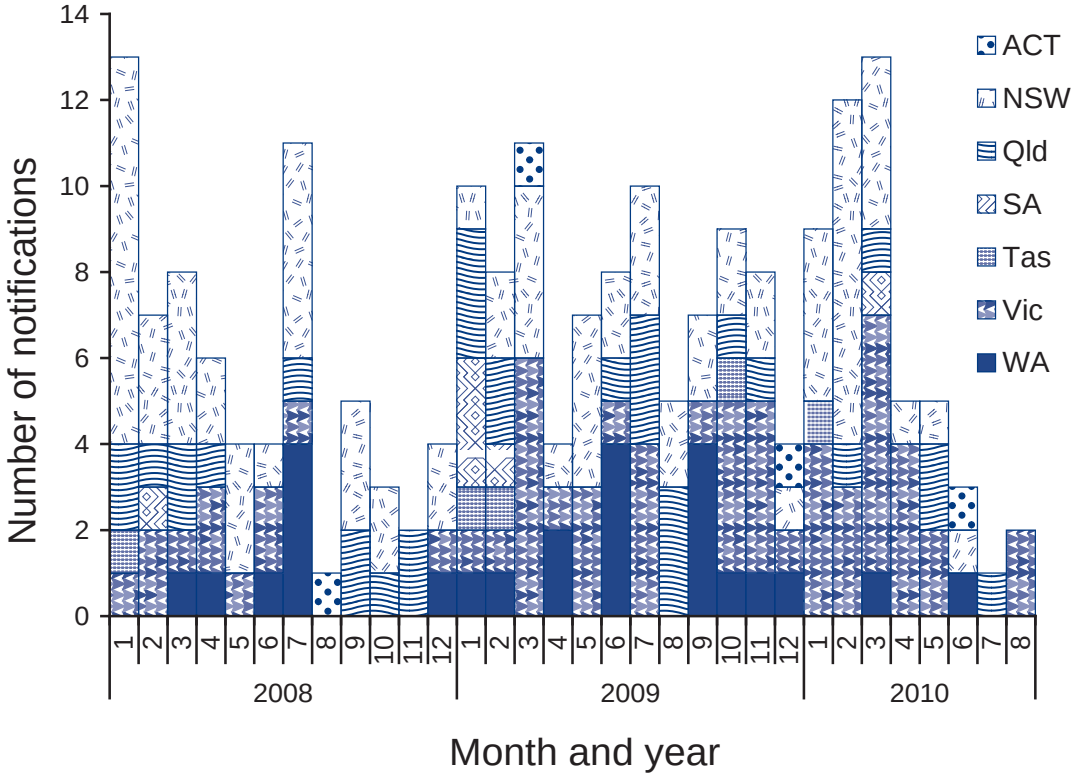
Hospital exposures were considered possible, with 33% (4/9) of outbreak cases hospitalised or with day visits for other underlying conditions during some of the period when they were likely to have been exposed.

There was a co-incidental finding of the outbreak strains of *Listeria* in samples obtained from a premises that manufactures fruit salad. These samples were taken as part of an investigation of a different cluster of *Listeria* cases in Victoria with a different serotype and BT. The outbreak strains of *Listeria monocytogenes* were isolated from by-products of manufacturing (waste juice from a stainless steel tub and fruit rinse water), and from a wash taken from the surface of a honeydew melon. There is no known interstate distribution from this manufacturer.

Separately, the outbreak strain (molecular serogroup: 1/2b, BT: 158, MDU designated PFGE: 121:119:1) was isolated from a sample of fruit salad taken by a local council at a delicatessen in Victoria, while a 2nd sample of fruit salad taken from a different delicatessen yielded the 2nd outbreak strain (molecular serogroup: not established, BT: 158 and MDU designated PFGE: 122:4N:1). These fruit salads were both prepared at the premises using whole fresh fruit. The Victorian Department of Health has also tested a range of other food samples, and none yielded the outbreak strain.

While the number of cases in this outbreak remains small, and there is no direct link between the positive environmental samples and the outbreak cases, there is a temporal association between outbreak cases and findings in a food reported as being consumed by many of the cases. The outbreak strain is rare, providing further evidence for the source being rockmelon and/or honeydew melon, eaten fresh or used in the preparation of fruit salads.

Figure: Notifications of listeriosis, Nationally Notifiable Diseases Surveillance System, Australia, 1 January 2008 to 6 September 2010, by month and year of diagnosis



Trace-back conducted in Victoria, New South Wales and Queensland indicated a common source for some of the melons, in south central New South Wales. Onset dates for cases were between February and May, and the supply of melons from growing districts is known to be seasonal, suggesting that the source of infection was likely to be a supplier from southern regions of Australia that ceased production after this time. If there are no further outbreak cases this year this would further support this theory.

This multi-jurisdictional outbreak investigation triggered the National Food Incident Response Protocol on 16 July 2010. The New South Wales Food Authority and New South Wales Department of Primary Industries have liaised with Horticulture Australia and are working to develop quality assurance education tools. Food Standards Australia New Zealand (FSANZ), in liaison with respective jurisdictions, is planning to meet with industry representatives to discuss issues at wholesale and retail levels, and will also consider including melons and prepared fruit salad in the Food Regulation Standing Committee's Implementation Sub-Committee's coordinated survey of *L. monocytogenes* in ready-to-eat foods. FSANZ will also coordinate the development of a discussion paper that identifies possible control measures and future preventative measures. This outbreak investigation has highlighted that detailed national level genotyping is critical for the detection of listeriosis clusters especially those involving cases across multiple jurisdictions.

Cluster investigations

During the 2nd quarter of 2010, OzFoodNet sites investigated several clusters. A cluster is defined as an increase in a specific infection in terms of time, place, or person where a source and mode of transmission remains unknown. The majority of these investigations involved *Salmonella* serotypes for which no common food vehicle or source of infection could be identified: *S.* Infantis, *S.* Poona, *S.* Virchow phage type 8 and *S.* Typhimurium (phage types 9, 135a and 170). However, in New South Wales, a cluster of *S.* Singapore cases was associated with the consumption of eggs but no common exposure or source of eggs could be identified.

Following a case series analysis in Tasmania, a large cluster of cryptosporidiosis was found to be associated with a public swimming pool and 2 smaller clusters associated with private swimming schools. After remedial intervention, *Cryptosporidium* infections in the area have returned to baseline levels.

Comments

The number of foodborne outbreaks reported during the quarter (n = 35) exceeded the average number during the same quarter over the past 5 years (n = 27). This increase in the number of foodborne outbreaks coincided with a general increase in the number of notifications of salmonellosis to the National Notifiable Diseases Surveillance System (NNDSS), with 2,893 notifications of salmonellosis during the quarter compared with a mean of 2,071 notifications for the same period over the past 5 years (National Notifiable Diseases Surveillance System, unpublished data).

In December 2009, the Public Health Microbiology Reference Laboratory in Queensland modified its screening procedures for detecting Shiga toxin-producing *Escherichia coli* (STEC) infections. All faecal samples that are submitted to the Public Health Microbiology Laboratory for STEC testing are now screened for the presence of Shiga toxin using an enzyme immunoassay (EIA – Premier EHEC, Meridian BioScience) method in conjunction with a polymerase chain reaction (PCR) technique for detecting Shiga toxin-producing genes. EIA only does not meet the national case definition for STEC.[4] Prior to December 2009, all stool specimens submitted for STEC testing were initially screened using the PCR method and EIA was performed on those specimens that were PCR positive. If the PCR was negative, there was no further testing conducted. Probable cases (EIA positive only; PCR and/or culture negative) are not being notified to the NNDSS. A study protocol is being developed in Queensland to evaluate the EIA test in terms of its specificity and level of agreement with the cytotoxicity assay and PCR.

Outbreaks of foodborne disease associated with eggs are of continuing concern in Australia. During the quarter, four of the 15 (27%) foodborne outbreaks for which sources could be determined, were associated with the consumption of egg-based sauces or egg wash used for glazing. Tartare sauce, aioli and mayonnaise continue to be a source of foodborne *Salmonella* infection.

During the quarter, OzFoodNet held an Advanced Disease Outbreak Investigation Workshop in Adelaide, South Australia, which was organised on behalf of the network by the OzFoodNet site and Communicable Disease Control Branch staff in South Australia. The workshop included

presentations by invited speakers from the Centers for Disease Control and Prevention, United States of America and Taranaki Public Health Service, New Zealand. The 2-day workshop covered the early stages of an outbreak investigation, descriptive analysis, analytical studies, risk assessment, environmental factors, novel vehicles of infection, laboratory issues including pathogen typing to improve outbreak detection and investigation, communication and media issues, and multi-jurisdictional outbreak investigations.

A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks reported should be interpreted with caution due to the small number each quarter.

Acknowledgements

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

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References

1. OzFoodNet Working Group. OzFoodNet quarterly report, 1 April to 30 June 2009. *Commun Dis Intell* 2009;33(3)341–347.

2. OzFoodNet Working Group. OzFoodNet quarterly report, 1 January to 31 March 2010. *Commun Dis Intell* 2010;34(2)127–136.

3. Dalton CB, Merritt TD, Unicomb LE, Kirk MD, Stafford RJ, Lalor K and the OzFoodNet Working Group. A national case-control study of risk factors for listeriosis in Australia. *Epidemiol Infect* 2010;30:1–9. [Epub ahead of print]

4. Communicable Diseases Network Australia. Shiga toxin-producing/vero toxin-producing ***Escherichia coli***—STEC/VTEC case definition Accessed on 18 August 2010. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd_stec.htm

Communicable diseases surveillance

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 44,904 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April and 30 June 2010 (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 (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	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
STEC, VTEC	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	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 <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia
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
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
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
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
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

* Notifiable in South Australia as of 1 May 2008.

† Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008.

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2010, by date of diagnosis*

Disease	State or territory								Total 2nd quarter 2010†	Total 1st quarter 2010	Total 2nd quarter 2009	Last 5 years mean 2nd quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Hepatitis B (newly acquired)	0	7	3	14	5	1	15	11	56	70	66	71.0	0.8	126	137.6
Hepatitis B (unspecified)	17	728	37	289	89	11	453	210	1,834	1,976	1,717	1,613.8	1.1	3,794	3,262.4
Hepatitis C (newly acquired)	4	8	0	NN	5	7	52	20	96	95	100	99.6	1.0	190	196.8
Hepatitis C (unspecified)	44	1,118	55	695	95	56	692	245	3,000	3,040	2,775	2,828.4	1.1	6,020	5,817.0
Hepatitis D	0	0	0	2	0	0	3	0	5	4	13	8.8	0.5	9	18.6
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.8
Campylobacteriosis§	102	NN	45	1,040	294	123	1,251	458	3,313	4,259	3,873	3,621.0	0.9	7,540	7,915.6
Cryptosporidiosis	5	77	34	70	14	31	134	31	396	533	1,154	803.4	0.5	920	2,305.6
Haemolytic uraemic syndrome	0	0	0	1	0	0	0	0	1	3	5	3.6	0.3	4	9.0
Hepatitis A	0	13	0	12	0	1	14	8	48	103	166	89.0	0.5	150	166.6
Hepatitis E	1	3	0	2	0	0	2	1	9	11	10	7.6	1.1	20	19.6
Listeriosis	1	3	0	2	0	0	6	1	13	34	19	12.4	1.0	47	34.6
STEC, VTEC‖	0	1	0	2	7	0	1	0	11	35	26	21.8	0.5	44	52.4
Salmonellosis	32	894	163	715	150	46	531	333	2,864	4,097	2,222	2,070.8	1.4	6,936	5,175.2
Shigellosis	2	22	25	20	10	3	15	27	124	165	168	160.8	0.8	287	364.2
Typhoid	1	7	1	10	0	1	6	4	30	31	19	19.2	1.6	60	49.6
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	1	0.6	0.0	0	1.6
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, 1 April to 30 June 2010, by date of diagnosis,* *continued*

Disease	State or territory								Total 2nd quarter 2010†	Total 1st quarter 2010	Total 2nd quarter 2009	Last 5 years mean 2nd quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	268	4,421	616	4,881	1,039	560	3,916	2,686	18,387	19,102	16,715	13,503.4	1.4	37,418	26,729.4
Donovanosis	0	0	0	0	0	0	0	0	0	0	1	1.6	0.0	0	3.2
Gonococcal infection	13	549	489	525	146	3	448	378	2,551	2,410	2,220	2,186.6	1.2	4,955	4,304.4
Syphilis < 2 years duration	2	60	17	48	9	2	41	26	205	297	338	294.2	0.7	503	563.4
Syphilis > 2 years or unspecified duration	3	49	26	50	NDP	2	161	10	301	310	357	334.0	0.9	611	659.8
Syphilis - congenital	0	0	0	1	0	0	0	0	1	1	0	3.4	0.3	2	6.0
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	2	0	2	1	0	1	0	6	3	8	5.2	1.2	9	10.0
Influenza (laboratory confirmed)	9	0	28	263	45	14	140	89	588	332	11,713	2,837.4	0.2	919	3,198.8
Measles	0	2	0	2	1	0	1	9	15	14	9	27.2	0.6	29	54.6
Mumps	1	4	1	5	0	0	1	4	16	19	44	71.8	0.2	35	139.4
Pertussis	61	850	72	1,375	1,149	48	1,113	179	4,847	5,600	7,597	3,136.4	1.5	10,406	6,193.0
Pneumococcal disease (invasive)	8	132	11	76	36	10	110	32	415	209	400	413.2	1.0	623	629.0
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	1	1	0	1	0	0	5	3	11	16	8	12.6	0.9	27	19.4
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.4
Tetanus	0	0	0	0	0	0	0	0	0	1	0	0.4	0.0	1	1.8
Varicella zoster (chickenpox)	1	NN	15	68	85	2	72	64	307	278	458	241.6	1.0	580	475.6
Varicella zoster (shingles)	5	NN	39	22	312	40	97	156	671	769	733	355.8	1.5	1,440	733.4
Varicella zoster (unspecified)	25	NN	2	938	78	19	385	225	1,672	1,805	1,697	893.2	1.5	3,457	1,825.4
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	1	0	0	0	0	1	3	5	6.8	0.2	4	17.8
Barmah Forest virus infection	2	70	18	257	9	0	8	17	381	456	354	500.2	0.8	834	1,088.2
Dengue virus infection	7	20	9	57	4	2	18	118	235	157	193	93.8	2.5	391	376.6
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	1	0	0	0	0	0	1	1	0	0.4	2.5	2	1.2
Malaria	1	27	3	27	2	1	19	14	94	102	147	160.6	0.6	195	354.6
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	2	0.8	0.0	0	1.8
Ross River virus infection	4	420	64	1,070	87	6	148	63	1,862	1,645	1,526	1,132.4	1.6	3,502	3,110.2

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2010, by date of diagnosis,* *continued*

Disease	State or territory								Total 2nd quarter 2010†	Total 1st quarter 2010	Total 2nd quarter 2009	Last 5 years mean 2nd quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	0.4
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	3	0	0	0	0	3	5	9	8.0	0.4	8	19.2
Leptospirosis	0	3	0	36	0	0	1	1	41	21	49	40.2	1.0	62	90.2
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	2	0	0	1	3	0	0	6	11	17	33.6	0.2	17	62.8
Q fever	1	14	0	43	2	0	5	3	68	74	80	94.4	0.7	142	194.4
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Other bacterial infections															
Legionellosis	0	23	0	15	5	1	26	6	76	62	99	82.6	0.9	138	160.8
Leprosy	0	0	0	1	0	0	1	0	2	1	0	2.2	0.9	3	5.2
Meningococcal infection**	0	13	0	20	5	1	14	3	56	44	63	65.4	0.9	100	121.8
Tuberculosis	2	75	5	61	13	2	106	21	285	305	272	262.6	1.1	587	537.4
Total	623	9,618	1,779	12,722	3,698	996	10,012	5,456	44,904	48,510	57,448			93,148	

* Date of diagnosis = true onset date, or where not available, the earliest of (i) specimen date, (ii) notification date, or (iii) notification receive date. Hepatitis B and C unspecified were analysed by the notification receive date.

† 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 varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 3 years of 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; the Northern Territory and Queensland, which exclude 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 April to 30 June 2010, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)	0.0	0.4	5.3	1.3	1.2	0.8	1.1	2.0	1.0
Hepatitis B (unspecified)	19.4	41.0	65.8	26.2	21.9	8.8	33.4	37.6	33.5
Hepatitis C (newly acquired)	4.6	0.5	0.0	NN	1.2	5.6	3.8	3.6	2.2
Hepatitis C (unspecified)	50.1	63.0	97.8	63.1	23.4	44.6	51.0	43.8	54.9
Hepatitis D	0.0	0.0	0.0	0.2	0.0	0.0	0.2	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	116.2	NN	80.1	94.4	72.5	97.9	92.2	81.9	89.7
Cryptosporidiosis	5.7	4.3	60.5	6.4	3.5	24.7	9.9	5.5	7.2
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.0	0.7	0.0	1.1	0.0	0.8	1.0	1.4	0.9
Hepatitis E	1.1	0.2	0.0	0.2	0.0	0.0	0.1	0.2	0.2
Listeriosis	1.1	0.2	0.0	0.2	0.0	0.0	0.4	0.2	0.2
STEC, VTEC‡	0.0	0.1	0.0	0.2	1.7	0.0	0.1	0.0	0.2
Salmonellosis	36.4	50.4	290.0	64.9	37.0	36.6	39.1	59.5	52.4
Shigellosis	2.3	1.2	44.5	1.8	2.5	2.4	1.1	4.8	2.3
Typhoid	1.1	0.4	1.8	0.9	0.0	0.8	0.4	0.7	0.5
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§	305.3	249.1	1,095.9	443.0	256.1	445.7	288.6	480.3	336.2
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	14.8	30.9	869.9	47.7	36.0	2.4	33.0	67.6	46.6
Syphilis <2 years duration	2.3	3.4	30.2	4.4	2.2	1.6	3.0	4.6	3.7
Syphilis >2 years or unspecified duration	3.4	2.8	46.3	4.5	0.0	1.6	11.9	1.8	5.5
Syphilis - congenital	0.0	0.0	0.0	0.1	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	0.0	0.2	0.2	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	10.3	0.0	49.8	23.9	11.1	11.1	10.3	15.9	10.8
Measles	0.0	0.1	0.0	0.2	0.2	0.0	0.1	1.6	0.3
Mumps	1.1	0.2	1.8	0.5	0.0	0.0	0.1	0.7	0.3
Pertussis	69.5	47.9	128.1	124.8	283.2	38.2	82.0	32.0	88.6
Pneumococcal disease (invasive)	9.1	7.4	19.6	6.9	8.9	8.0	8.1	5.7	7.6
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 April to 30 June 2010, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *continued*									
Rubella	1.1	0.1	0.0	0.1	0.0	0.0	0.4	0.5	0.2
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
Varicella zoster (chickenpox)	1.1	NN	26.7	6.2	21.0	1.6	5.3	11.4	8.3
Varicella zoster (shingles)	5.7	NN	69.4	2.0	76.9	31.8	7.1	27.9	18.2
Varicella zoster (unspecified)	28.5	NN	3.6	85.1	19.2	15.1	28.4	40.2	45.3
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Barmah Forest virus infection	2.3	3.9	32.0	23.3	2.2	0.0	0.6	3.0	7.0
Dengue virus infection	8.0	1.1	16.0	5.2	1.0	1.6	1.3	21.1	4.3
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	1.8	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.1	1.5	5.3	2.5	0.5	0.8	1.4	2.5	1.7
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	4.6	23.7	113.9	97.1	21.4	4.8	10.9	11.3	34.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	0.0	3.3	0.0	0.0	0.1	0.2	0.7
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.2	2.4	0.0	0.0	0.1
Q fever	1.1	0.8	0.0	3.9	0.5	0.0	0.4	0.5	1.2
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	1.3	0.0	1.4	1.2	0.8	1.9	1.1	1.4
Leprosy	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Meningococcal infection‖	0.0	0.7	0.0	1.8	1.2	0.8	1.0	0.5	1.0
Tuberculosis	2.3	4.2	8.9	5.5	3.2	1.6	7.8	3.8	5.2

* 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; the Northern Territory and Queensland, which exclude 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 10,640 reports received by the Laboratory Virology and Serology Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2010 (Tables 4 and 5).

Table 4: Laboratory Virology and Serology reports, 1 April to 30 June 2010 and total reports for the year,* by state or territory †

	State or territory								This period 2010	This period 2009	Year to date 2010	Year to date 2009
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	1	1	0	2	0	4	1	12	44
Mumps virus	0	0	0	1	2	0	0	0	3	4	9	30
Rubella virus	1	2	0	0	0	0	6	0	9	2	18	9
Hepatitis viruses												
Hepatitis A virus	0	0	0	11	3	0	0	4	18	6	34	28
Hepatitis D virus	0	0	0	1	1	0	0	0	2	4	6	14
Hepatitis E virus	0	1	0	1	0	0	0	0	2	–	3	3
Arboviruses												
Ross River virus	0	22	10	470	90	0	5	3	600	108	955	694
Barmah Forest virus	0	7	0	60	12	0	0	0	79	23	148	149
Flavivirus (unspecified)	0	15	0	38	0	0	8	0	61	13	120	171
Adenoviruses												
Adenovirus not typed/ pending	0	87	0	74	125	0	2	0	288	222	510	863
Herpesviruses												
Herpes virus type 6	0	0	0	0	0	0	1	0	1	1	2	1
Cytomegalovirus	1	22	0	120	154	2	7	0	306	90	700	636
Varicella-zoster virus	0	42	0	502	285	6	8	1	844	303	1,655	1,383
Epstein-Barr virus	0	6	26	309	247	2	16	82	688	191	1,554	1,160
Other DNA viruses												
Parvovirus	0	2	0	21	45	0	16	0	84	29	157	122
Picornavirus family												
Rhinovirus (all types)	0	50	1	0	0	0	0	0	51	19	101	65
Enterovirus not typed/ pending	0	1	0	3	13	1	0	0	18	5	37	52
Picornavirus not typed	0	0	0	0	0	6	0	0	6	–	8	5
Ortho/paramyxoviruses												
Influenza A virus	3	27	0	81	65	0	26	0	202	2,630	350	3,220
Influenza B virus	0	4	0	13	14	0	2	0	33	60	56	142
Newcastle Disease virus	0	1	0	0	0	0	0	0	1	–	2	–
Parainfluenza virus type 1	2	16	0	8	4	0	0	0	30	3	100	11
Parainfluenza virus type 2	0	7	0	9	5	0	0	0	21	24	39	69
Parainfluenza virus type 3	0	12	0	4	24	0	0	0	40	74	73	163
Respiratory syncytial virus	1	520	0	171	485	1	2	0	1,180	931	1,425	1,944

Table 4: Laboratory Virology and Serology reports, 1 April to 30 June 2010, and total reports for the year,*by state or territory † *continued*

	State or territory								This period 2010	This period 2009	Year to date 2010	Year to date 2009
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	0	0	0	1	24	0	0	0	25	13	42	142
Rotavirus	0	6	0	0	165	0	1	0	172	37	188	151
Norwalk agent	0	26	0	0	241	6	0	0	273	7	578	32
Other												
Chlamydia trachomatis not typed	2	85	1	2,067	782	19	10	0	2,966	918	6,033	4,559
Chlamydia pneumoniae	0	0	0	1	0	0	0	0	1	2	1	6
Chlamydia psittaci	0	0	0	2	0	1	2	0	5	5	13	39
Chlamydia spp typing pending	0	8	0	0	0	0	0	0	8	–	13	6
Chlamydia species	0	0	0	0	0	0	1	0	1	–	2	7
Mycoplasma pneumoniae	0	4	0	56	96	4	111	0	271	127	547	574
Coxiella burnetii (Q fever)	3	0	0	18	16	0	4	0	41	18	72	116
Rickettsia prowazeki	0	0	0	0	2	0	0	0	2	–	3	8
Rickettsia - spotted fever group	0	0	0	7	2	0	1	0	10	7	20	75
Streptococcus group A	0	9	0	176	0	0	41	0	226	84	402	315
Brucella species	0	0	0	1	0	0	0	0	1	1	4	9
Bordetella pertussis	0	15	0	451	910	1	185	1	1,563	529	3,136	2,934
Legionella pneumophila	0	0	0	1	4	1	2	0	8	8	12	20
Legionella longbeachae	0	0	0	0	2	0	1	0	3	3	8	10
Legionella species	0	3	0	8	0	0	0	0	11	4	21	13
Cryptococcus species	0	0	0	6	2	0	0	0	8	5	23	21
Leptospira species	0	0	0	8	1	0	0	0	9	2	24	25
Treponema pallidum	0	57	0	265	109	0	11	0	442	177	970	962
Entamoeba histolytica	0	0	0	2	0	0	1	0	3	1	6	1
Toxoplasma gondii	0	0	0	6	7	0	3	0	16	1	21	11
Echinococcus granulosus	0	0	0	0	2	0	2	0	4	3	5	14
Total	13	1,057	38	4,974	3,940	50	477	91	10,640	6,695	20,218	21,028

* Data presented are for reports with report dates in the current period.

† State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

– No data received this period.

Table 5: Laboratory Virology and Serology reports, 1 April to 30 June 2010,* by laboratory

State or territory	Laboratory	April 2010	May 2010	June 2010	Total
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	–	–	–	–
	New Children's Hospital, Westmead	122	112	189	423
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	83	114	159	356
Queensland	Queensland Medical Laboratory, West End	1,677	1,832	1,800	5,309
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	1,009	1,303	1,623	3,935
Tasmania	Northern Tasmanian Pathology Service, Launceston	15	13	20	48
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	–	–	–	–
	Monash Medical Centre, Melbourne	–	–	–	–
	Royal Children's Hospital, Melbourne	76	96	84	256
	Victorian Infectious Diseases Reference Laboratory	58	60	69	187
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	25	47	54	126
Total		3,065	3,577	3,998	10,640

* The complete list of laboratories reporting for the 12 months, January to December 2010, 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 childhood immunisation coverage

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 5 years of age, for 3-month birth cohorts of children at the stated ages between January and March 2010. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, or meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing Haemophilus influenzae *type b (Hib) vaccine or 3 doses of any other* Haemophilus influenzae *type b (Hib) vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP containing Haemophilus influenzae type b (Hib) vaccine or 4 doses of any other* Haemophilus influenzae *type b (Hib) vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 5 years of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.*

A full description of the basic methodology used can be found in CDI *1998;22:36-37.*

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1435, Email: brynleyh@chw.edu.au

The percentage of children 'fully immunised' at 12 months of age for Australia increased slightly by 0.1 of a percentage point to 91.5% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

The percentage of children 'fully immunised' at 24 months of age for Australia increased by 0.4 percentage points to 92.4 (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

The percentage of children 'fully immunised' at 5 years of age for Australia increased considerably, by 5.8 percentage points, to sit currently at 89.6% (Table 3). There were important changes in coverage for all individual vaccines assessed at 5 years of age and for all jurisdictions. The increases by jurisdiction ranged from 1.7 percentage points in the Australian Capital Territory to 8 percentage points in Queensland. These are the greatest quarter to quarter increases in coverage for any vaccine and at any age milestone since the inception of the ACIR. These increases are most likely due to a combination of recent developments designed to improve the timeliness of pre-school vaccines. They were:

1. the change to the overdue rules, where 4 year olds are now overdue at 4 years and 1 month instead of 5 years,

Table 1: Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2009; assessment date 30 June 2010

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,172	23,898	975	15,576	4,917	1,563	17,335	7,697	73,133
Diphtheria, tetanus, pertussis (%)	93.2	91.7	91.6	92.3	91.9	92.2	92.7	90.5	92.0
Poliomyelitis (%)	93.2	91.7	91.6	92.3	91.9	92.1	92.7	90.5	92.0
Haemophilus influenzae type b (%)	93.0	91.5	93.5	92.1	91.7	91.9	92.5	90.3	91.8
Hepatitis B (%)	92.3	91.4	91.5	92.0	91.4	91.9	92.2	90.3	91.6
Fully immunised (%)	92.2	91.3	90.3	91.9	91.3	91.7	92.1	90.1	91.5
Change in fully immunised since last quarter (%)	-0.6	-0.5	+0.9	+0.4	+0.7	-1.3	+0.1	+0.9	+0.0

Table 2: Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2008; assessment date 30 June 2010*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,188	24,333	972	15,823	4,940	1,619	17,966	8,019	74,860
Diphtheria, tetanus, pertussis (%)	95.8	94.9	96.7	94.5	94.7	94.7	95.4	93.9	94.9
Poliomyelitis (%)	95.7	94.9	96.6	94.5	94.6	94.6	95.4	93.8	94.8
Haemophilus influenzae type b (%)	95.4	95.1	94.6	94.3	94.4	94.6	95.1	93.2	94.7
Measles, mumps, rubella (%)	94.9	93.8	95.2	93.8	93.9	94.5	94.5	92.8	93.9
Hepatitis B (%)	95.1	94.5	96.1	94.0	94.3	94.2	94.7	93.2	94.3
Fully immunised (%)	93.8	92.5	93.4	92.2	92.5	92.8	93.0	90.5	92.4
Change in fully immunised since last quarter (%)	-0.1	+0.2	+1.4	+0.6	+1.0	-0.7	+0.4	+0.6	+0.4

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2009;34(3):360.

Table 3: Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2005; assessment date 30 June 2010

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,124	22,746	869	14,854	4,652	1,442	16,244	7,308	69,239
Diphtheria, tetanus, pertussis (%)	90.0	90.0	88.0	91.0	87.6	91.1	91.6	87.5	90.2
Poliomyelitis (%)	90.1	90.0	88.0	90.9	87.6	91.0	91.6	87.5	90.1
Measles, mumps, rubella (%)	89.2	89.8	87.7	90.7	87.5	91.0	91.4	87.1	89.9
Fully immunised (%)	89.0	89.5	87.3	90.2	87.2	90.6	91.2	86.6	89.6
Change in fully immunised since last quarter (%)	+2.7	+6.5	+4.1	+8.0	+6.1	+6.2	+3.9	+4.3	+5.8

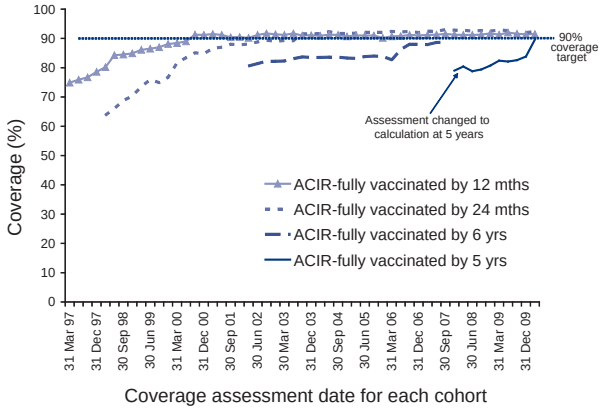
2. the accompanying reminder letter sent out by Medicare Australia informing parents of these changes, and

3. individual jurisdictional efforts to follow-up overdue children in the cohort.

Also, since October 2009 it has been recommended that the 4th dose of DTPa vaccine can be given from 3½ years of age, instead of the previously recommended 4 years (http://www.health.gov.au/internet/immunise/publishing.nsf/Content/atagi-meet41bulletin).

The Figure 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 (till December 2007). This trend continued when the age of coverage calculation was changed from 6 to 5 years in March 2008, and then increased further in the last quarter as outlined above.

Figure: Trends in vaccination coverage, Australia, 1997 to 31 March 2010 2010, by age cohorts



Australian Sentinel Practices Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Commonwealth's Department of Health and Ageing, 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. Electronic, web-based data collection was established in 2006.

In April 2010, the Northern Territory's Tropical Influenza Surveillance Scheme became affiliated with ASPREN, being the last jurisdiction to complete the national picture. June 2010 saw ASPREN's long awaited laboratory ILI testing implemented, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and H1N1(2009).

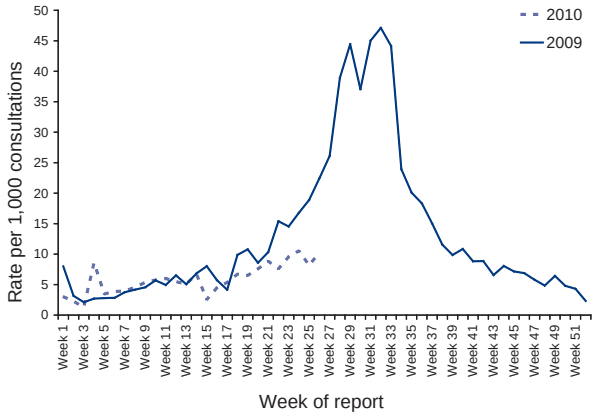
The list of conditions is reviewed annually by the ASPREN management committee. In 2010, 4 conditions are being monitored. They include influenza-like illness (ILI), gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2010;34(1):83–84.*

Reporting period 1 April to 30 June 2010

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 111 general practitioners contributed data to ASPREN in the 2nd quarter of 2010. Each week an average of 87 general practitioners provided information to ASPREN at an average of 8,372 (range 6,996–9,378) consultations per week and an average of 125 (range 77–159) notifications per week.

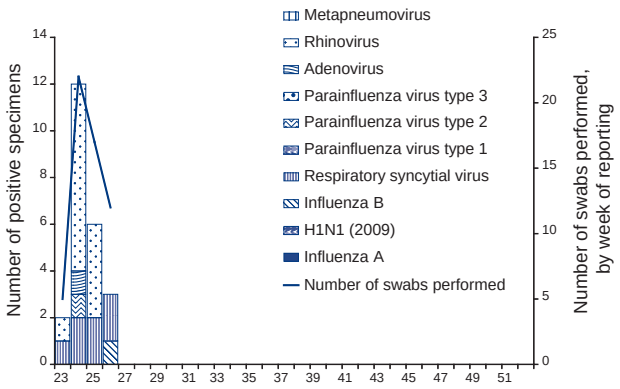
ILI rates reported from 1 April to 30 June 2010 averaged 7 cases per 1,000 consultations (range 1–9 cases per 1,000 consultations). The reported rates in April, May and June 2010 (3–7 cases per 1,000 consultations, 7–9 cases per 1,000 consultations and 8–11 cases per 1,000 consultations respectively) were slightly lower compared with rates in the same reporting period in 2009 (5–8 cases per 1,000 consultations, 9–15 cases per 1,000 consultations and 15–22 cases per 1,000 consultations respectively) (Figure 1).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2009 to 30 June 2010, by week of report



ILI swab testing commenced at the beginning of June 2010. The most commonly reported virus was rhinovirus (23% of all swabs performed), with respiratory syncytial virus the second most commonly reported (13% of all swabs performed) (Figure 2). To the end of week 26 2010, only 1 case of influenza has been detected, this being influenza B (untyped).

Figure 2: Influenza-like illness swab testing results, ASPREN, June 2010, by week of report



During this reporting period, consultation rates for gastroenteritis averaged 6.2 cases per 1,000 consultations (range 5–8 cases per 1,000, Figure 3). This was slightly lower compared with

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2009 to 30 June 2010, by week of report

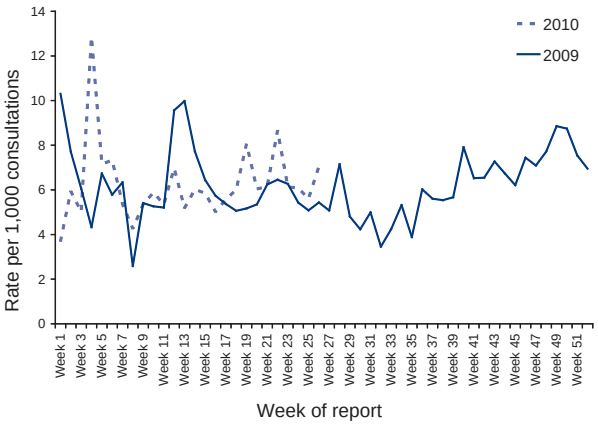
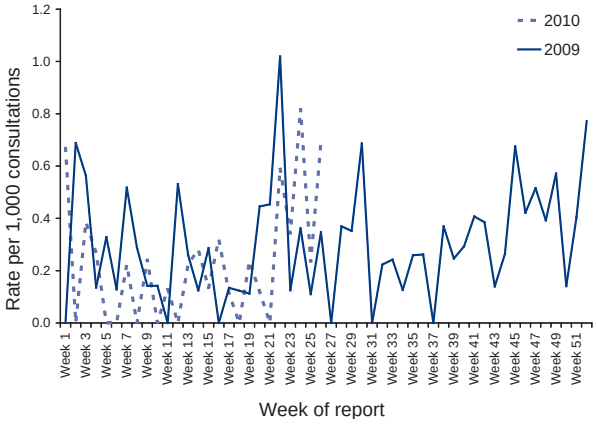


Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2009 to 30 June 2010, by week of report

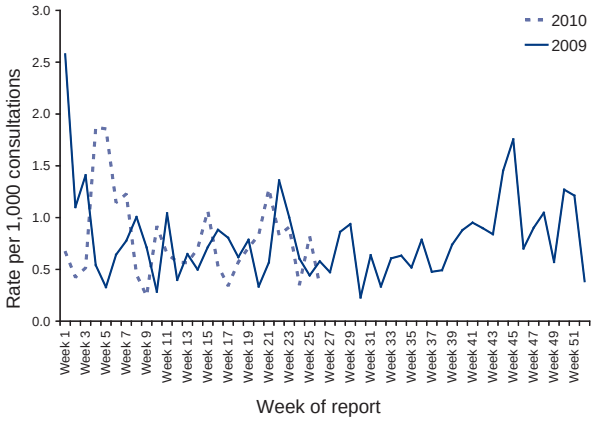


the same reporting period in 2009 when the average was 6.1 cases per 1,000 consultations (range 5–10 cases per 1,000 consultations).

Varicella infections were reported at a slightly higher rate for the 2nd quarter of 2010 compared with the same period in 2009. From 1 April to 30 June 2010, recorded rates for chickenpox averaged 0.3 cases per 1,000 consultations (range 0–0.8 cases per 1,000 consultations, Figure 4).

In the 2nd quarter of 2010, reported rates for shingles averaged 0.7 cases per 1,000 consultations (range 0.3–1.3 cases per 1,000 consultations, Figure 5), similar to the same reporting period in 2009 when the average shingles rate was also 0.7 cases per 1,000 consultations (0.3–1.4 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 1 January 2009 to 30 June 2010, by week of report



Gonococcal surveillance

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The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2010;34:82–83.*

Reporting period 1 January to 31 March 2010

The AGSP laboratories received a total of 1,056 isolates in this quarter of which 1,023 underwent susceptibility testing. This number is 181 more than the 875 isolates reported in this period in 2009. About 36% of this total was from New South Wales, 22% from Victoria, 17% from Queensland, 9% each from Western Australia and the Northern Territory and 6% from South Australia. A small number of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

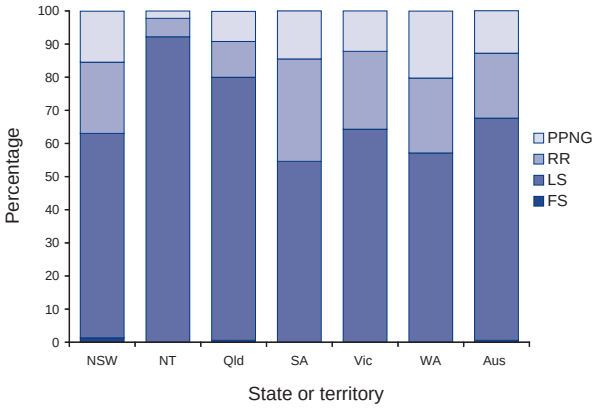
In this quarter 331 (32%) of all isolates examined were penicillin resistant by one or more mechanisms. One hundred and thirty-one (13%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 200 (20%) were penicillin resistant by chromosomal mechanisms, (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from 3.3% in locally-acquired disease in the Northern Territory to 46% in South Australia.

In the Northern Territory there were a further 4 cases of penicillin resistant gonococci (2 PPNG and 2 CMRP) that were acquired in South East Asia (Singapore, Korea, the Philippines, and Thailand).

In this quarter in 2009, 39% of isolates were penicillin resistant by any mechanism, part of a trend of a decrease in proportion of penicillin resistant by any mechanism from over the past few years (2008:45%; and 2007:39%). The decrease in penicillin resistant strains to below 2007 proportions was the result of decreased numbers of gonococci with chromosomally mediated resistance.

Figure 1 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), chromosomally mediated resistance (CMRP) (MIC ≥ 1 mg/L) and penicillinase producing aggregated for Australia and by state or territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 1: Categorisation of gonococci isolated in Australia, 1 January to 31 March 2010, by penicillin susceptibility and region



FS	Fully sensitive to penicillin, MIC ≤0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L.
RR	Relatively resistant to penicillin, MIC ≥1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae*.

The highest number of PPNG and CMRP were found in New South Wales where there were 81 CMRP (22%) and 58 PPNG (15%). Victoria had 54 (24%) CMRP and 28 (12%) PPNG. In Queensland there were 19 CMRP (11%) and 16 PPNG (9%). and in Western Australia there were 19 CMRP, (22.6%) and 17 PPNG (20%). In the same quarter in 2009 in both Queensland and

Western Australia, there were more CMRP isolates reported than PPNGs for this period. Five CMRP and 2 PPNG strains were found in the Northern Territory. There were 2 CMRP and 1 PPNG in the Australian Capital Territory and 3 CMRP and 1 PPNG reported from Tasmania. Of note was the increase in penicillin resistant strains in South Australia in this quarter, from 36.5% in 2009 to 46% in 2010 comprising 17 CMRP (31%) and 8 PPNG (15%). Corresponding proportions in 2008 were 70.7% CMRP and 5% PPNG.

Ceftriaxone

Sixty-two isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, 25 in New South Wales, 13 in Western Australia, 10 in South Australia, eight in Queensland, six in Victoria, and one in the Australian Capital Territory. This compares with 10 in the 1st quarter of 2009. This increase in the proportion of isolates with decreased susceptibility to ceftriaxone (MIC ≥ 0.06 mg/L) represents a microbiological warning regarding the raised MIC, which has yet to be reported to be associated with treatment failure in genital infection. It is possible that the increase is 'clonal' and parallels the increase in isolates for this quarter when compared with 2009.

Spectinomycin

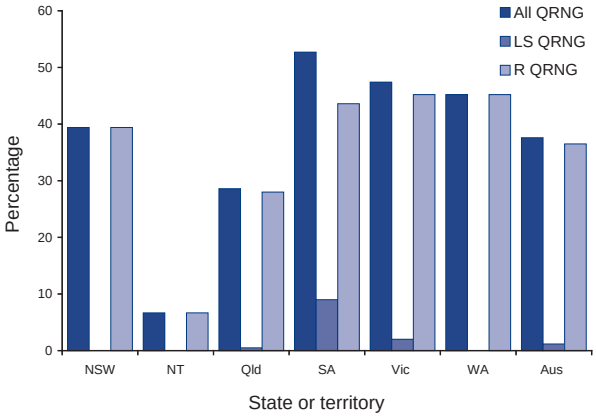
All isolates were susceptible to this injectable agent. This antibiotic is no longer available in Australia.

Quinolone antibiotics

The total number (385) and proportion (38%) of quinolone resistant *N. gonorrhoeae* (QRNG) was lower than data reported in recent quarters, which reported high levels of resistance to this group of antibiotics. In the equivalent period in 2009, there were 397 (46%) QRNG, lower than in 2008 (415 QRNG: 35%). All but 12 of the 385 QRNG detected in this quarter had ciprofloxacin MICs of 1 mg/L or more and 294 had ciprofloxacin MICs of 4 mg/L or more. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

QRNG were present in all jurisdictions (Figure 2). The highest number of QRNG was found in New South Wales (148), which represented 39% of all isolates. In Victoria, 109 QRNG also represented a high (47%) proportion of all isolates there. There were 50 (29%) QRNG in Queensland and in Western Australia 38 (45%) QRNG. The 29 (52%) QRNG in South Australia was a small increase in number compared with the 23 (37%) QRNG in the same quarter in 2009, and parallels the increase in penicillin resistance also noted in that jurisdiction in this quarter. Six QRNG were detected in the Northern Territory, five in the Australian Capital Territory and none in Tasmania.

Figure 2: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2010, by state or territory



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

Nationally, the number (203) and the proportion (20%) of high level tetracycline resistance (TRNG) detected increased when compared with the 2009 data (157 TRNG, 18%). TRNG were found in all states and territories except Tasmania, and elsewhere represented between 12% (Queensland) and 30% of isolates (Western Australia).

Reference

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

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, CFI Building, Cnr Boundary and West Streets, Darlinghurst NSW 2010. Internet: www.nchecr.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see Commun Dis Intell *2010;34:84.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 September 2009, are included in this issue of Communicable Diseases Intelligence (Tables 1, 2, 3 and 4).

Table 1: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2009, by sex and state or territory of diagnosis

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2009	This period 2008	YTD 2009	YTD 2008
HIV diagnoses	Female	1	11	2	9	2	1	6	3	35	49	76	78
	Male	5	81	3	56	10	7	66	14	242	219	454	456
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	6	93	5	65	12	8	72	17	278	268	531	534
AIDS diagnoses	Female	0	--	0	0	0	0	0	1	1	4	9	6
	Male	0	--	0	1	3	0	14	2	20	26	64	72
	Total*	0	--	0	1	3	0	14	3	21	30	73	78
AIDS deaths	Female	0	--	0	0	0	0	0	1	1	1	2	1
	Male	0	--	0	0	0	0	2	0	2	5	6	15
	Total*	0	--	0	0	0	0	2	1	3	6	8	16

* Totals include people whose sex was reported as transgender.

Dashes indicate that AIDS cases and deaths following AIDS diagnosed or occurring in NSW from January 2008 are not included.

Table 2: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing in 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 30 June 2009, by sex and state or territory

	Sex	State or territory								Aust
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	36	985	29	346	120	14	454	252	2,236
	Male	280	14,317	151	3,160	1,044	123	5,878	1,363	26,316
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	316	15,561	180	3,515	1,165	137	6,376	1,622	28,872
AIDS diagnoses†	Female	10	265	6	76	32	4	124	46	563
	Male	95	5,513	48	1,093	419	55	2,131	455	9,809
	Total*	105	5,796	54	1,171	452	59	2,268	503	10,408
AIDS deaths‡	Female	7	138	1	43	20	2	66	29	306
	Male	73	3,597	33	679	280	34	1,446	301	6,443
	Total*	80	3,746	34	724	300	36	1,521	331	6,772

* Totals include people whose sex was reported as transgender.

† AIDS cases diagnosed in New South Wales from January 2008 are not included.

‡ Deaths following AIDS occurring in NSW from January 2008 are not included.

Table 3: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 July to 30 September 2009, by sex and state or territory of diagnosis

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2009	This period 2008	YTD 2009	YTD 2008
HIV diagnoses	Female	0	14	1	8	0	1	5	4	33	30	109	108
	Male	3	81	4	51	12	4	66	22	243	200	697	656
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	3	96	5	59	12	5	71	26	277	230	808	764
AIDS diagnoses	Female	0	--	0	0	0	0	0	1	1	4	9	6
	Male	0	--	0	1	3	0	14	2	20	26	64	72
	Total*	0	--	0	1	3	0	14	3	21	30	73	78
AIDS deaths	Female	0	--	0	0	0	0	0	1	1	1	2	1
	Male	0	--	0	0	0	0	2	0	2	5	6	15
	Total*	0	--	0	0	0	0	2	1	3	6	8	16

* Totals include people whose sex was reported as transgender.

Dashes indicate that AIDS cases and deaths following AIDS diagnosed or occurring in NSW from January 2008 are not included.

Table 4: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing in 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 30 September 2009, by sex and state or territory

	Sex	State or territory								Aust
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	36	999	30	354	120	15	459	256	2,269
	Male	283	14,398	155	3,211	1,056	127	5,944	1,385	26,559
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	319	15,657	185	3,574	1,177	142	6,447	1,648	29,149
AIDS diagnoses†	Female	10	265	6	76	32	4	124	47	564
	Male	95	5,513	48	1,094	422	55	2,145	457	9,829
	Total*	105	5,796	54	1,172	455	59	2,282	506	10,429
AIDS deaths‡	Female	7	138	1	43	20	2	66	30	307
	Male	73	3,597	33	679	280	34	1,448	301	6,445
	Total*	80	3,746	34	724	300	36	1,523	332	6,775

* Totals include people whose sex was reported as transgender.

† AIDS cases diagnosed in New South Wales from January 2008 are not included.

‡ Deaths following AIDS occurring in NSW from January 2008 are not included.