Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 33 Number 2 Quarterly report June 2009

Annual reports

Peer-reviewed articles

Short reports

Quarterly reports

ISSN 0725-3141
ISSN 1445-4866 Online

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2009;33:87–89.

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

Website

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

Subscriptions and contacts

Communicable Diseases Intelligence is produced every quarter by:
Surveillance Branch
Office of Health Protection
Australian Government Department of Health and Ageing
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 2717
Facsimile: +61 2 6289 2600
Email: cdi.editor@health.gov.au

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

Disclaimer

Printed by Union Offset, Canberra

Annual reports

Australia's notifiable disease status, 2007: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

In 2007, 69 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 146,991 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, an increase of 5% on the number of notifications in 2006. In 2007, the most frequently notified diseases were sexually transmissible infections (62,474 notifications, 43% of total notifications), gastrointestinal diseases (30,325 notifications, 21% of total notifications) and vaccine preventable diseases (25,347 notifications, 17% of total notifications). There were 19,570 notifications of bloodborne diseases; 6,823 notifications of vectorborne diseases; 1,762 notifications of other bacterial infections; 687 notifications of zoonoses and 3 notifications of quarantinable diseases. *Commun Dis Intell* 2009;33:89–154.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2007, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, jurisdictional and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

- identifying national trends;
- guidance for policy development and resource allocation at a national level;
- monitoring the need for and impact of national disease control programs;
- coordination of response to national or multi-jurisdictional outbreaks;
- description of the epidemiology of rare diseases, that occur infrequently at state and territory levels;
- meeting various international reporting requirements, such as providing disease statistics to the World Health Organization (WHO); and
- support for quarantine activities, which are the responsibility of the national government.

Methods

Australia is a federation of 6 states (New South Wales, Queensland, South Australia, Tasmania, Victoria and Western Australia) and 2 territories (the Australian Capital Territory and the Northern Territory).

State and territory health departments collect notifications of communicable diseases under their public health legislation. In 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 about which personal information can be provided. The National Health Security Agreement,[3] which was drafted in 2007 and signed by Health Ministers in April 2008, establishes operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. In 2007, states and territories voluntarily forwarded de-identified data on 65 nationally agreed communicable diseases to the Department of Health and Ageing for the purposes of national communicable disease surveillance, although not all 65 were notifiable in each jurisdiction. Data were electronically renewed daily or several times a week from states and territories. The system was complemented by other surveillance systems, which provided information on various diseases, including four that are not reported to the National Notifiable Diseases Surveillance System (NNDSS) (HIV, AIDS and the classical and variant forms of Creutzfeldt-Jakob disease).

In 2007, 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), a jurisdictional committee comprised of surveillance and data managers. 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) was reported from states and territories to NNDSS but are 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 2007 and were forwarded 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]

The surveillance for the classical and variant forms of Creutzfeldt-Jakob disease (CJD) in Australia is 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. *CDI* is also available online from http://www.health.gov.au/cdi

Notification rates for each notifiable disease were calculated using the estimated 2007 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 2001 census data as the standard population.

The geographical distribution of selected diseases was mapped using ArcGIS (ESRI, Redlands, CA) software in conjunction with the Australian Standard Geographical Classification.[8] Maps were based on the postcode of residence of each notification aggregated to the appropriate Statistical Division[9] (SD) (Map 1, Table 1). The Northern Territory was represented by Statistical Subdivisions.[9] Some individual postcodes were used for a multitude of disparate localities. These postcodes were generally in close proximity to each other and contained within the same Statistical Division (95.5% of all postcodes). However a small number of postcodes (n=113) were scattered throughout neighbouring Statistical Divisions. ABS concordance files were used to proportionally allocate notifications into SDs according to the percentage of the population of that postcode unit living in the SD.[10] For instance, the postcode 2406 can be found in 2 distinct SDs, Northern (130) and South West (325). Almost 81% of the population live in Northern so this SD will get 81% of the notifications that have a postcode of 2406.

Rates for the different SDs were ordered into 5 groups using the Jenks Natural Breaks method which is the default multi-class numerical classification method used in ArcGIS. This classification method finds the largest breaks between natural clusters of ordered data by iteratively comparing the sum of the squared differences within the clusters and by adjusting class boundaries to minimise these differences. Another class '0' was added to account for areas with no notifications, for a total of 6 rate classes per map. Note that the classification is data dependent and changes from map to map. The 2 Statistical Divisions in the Australian Capital Territory were combined to calculate rates for the Territory as a whole.

There were 135 NNDSS postcodes which did not exist in the 2006 ABS concordance files (2006 being the latest available at time of publication) and consequently could not be mapped. These postcodes consisted of post office box numbers, special NNDSS postcode formats (3999/4999/6999/8888/9999 etc), fictitious postcodes (6444), missing postcodes and 2007 postcodes. These 135 notifications were omitted from the maps.

Notes on interpretation

The present report is based on 2007 'finalised' data from each state or territory agreed upon in September 2008 and represents a snap shot 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 of specimen date, health professional notification date or public health unit notification receive date was used for these conditions.

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

A survey of jurisdictional public health departments was conducted in 2005 to ascertain the source of each notification.[11] Notifications from Queensland were almost entirely supplied by laboratories (Table 2). In 3 other jurisdictions more than 90% of notifications originated from the laboratory. In 3 states almost half of the notifications were reported by both the doctor and laboratory. Only New South Wales, South Australia and Western Australia reported that greater than 15% of notifications in their jurisdictions originated from doctors only.

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, some diseases are not yet notifiable in all 8 jurisdictions (Table 3).

Changes in surveillance practices may have been introduced in some jurisdictions and not in others, and makes 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 2007 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, including those reported in 2007, by removing duplicates and preventing the loss of notification data in NNDSS.

Figure 1. Communicable diseases notifiable fraction

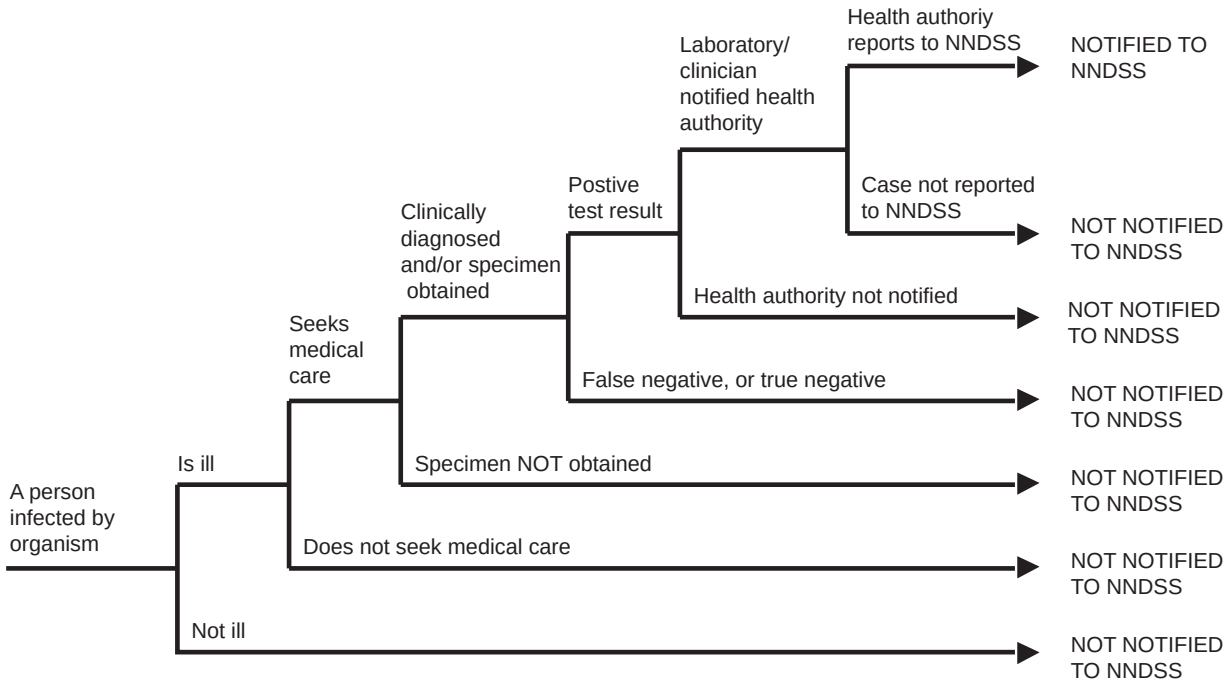


Table 1: Australian population by Statistical Division and Statistical Subdivision for the Northern Territory, 2007

SD code	Statistical Division	Population
Australian Capital Territory		
805	Canberra*	341,968
New South Wales		
105	Sydney	4,336,374
110	Hunter	624,296
115	Illawarra	417,901
120	Richmond-Tweed	232,948
125	Mid-North Coast	300,006
130	Northern	180,067
135	North Western	115,419
140	Central West	178,840
145	South Eastern	209,270
150	Murrumbidgee	154,663
155	Murray	116,471
160	Far West	22,817
Northern Territory (Subdivisions)		
70505	Darwin City	72,859
70510	Palmerston-East Arm	27,145
70520	Litchfield Shire	17,395
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
71035	Barkly	6,279
71040	Central NT	40,299
Queensland		
305	Brisbane	1,857,594
307	Gold Coast	535,528
309	Sunshine Coast	303,050
312	West Moreton	74,328
315	Wide Bay-Burnett	275,734
320	Darling Downs	229,254
325	South West	26,161
330	Fitzroy	204,537
335	Central West	11,397
340	Mackay	163,127
345	Northern	214,295
350	Far North	253,721
355	North West	33,336
South Australia		
405	Adelaide	158,259
410	Outer Adelaide	131,465
415	Yorke and Lower North	45,979
420	Murray Lands	69,763
425	South East	64,956
430	Eyre	34,893
435	Northern	79,198
Tasmania		
605	Greater Hobart	207,484
610	Southern	36,374
615	Northern	139,466
620	Mersey-Lyell	110,017
Victoria		
205	Melbourne	3,806,092
210	Barwon	273,619
215	Western District	103,307
220	Central Highlands	149,231
225	Wimmera	50,050
230	Mallee	92,707
235	Loddon	177,340
240	Goulburn	204,254
245	Ovens-Murray	97,069
250	East Gippsland	83,952
255	Gippsland	167,595
Western Australia		
505	Perth	1,554,769
510	South West	224,137
515	Lower Great Southern	55,946
520	Upper Great Southern	18,800
525	Midlands	53,593
530	South Eastern	56,858
535	Central	62,133
540	Pilbara	45,277
545	Kimberley	34,270
Other territories		–
Total		21,016,884

* Includes Statistical Division 810 ‘Australian Capital Territory – balance’.

Map 1: Australian Bureau of Statistics Statistical Division codes, Australia, and Statistical Subdivision codes, the Northern Territory, 2007

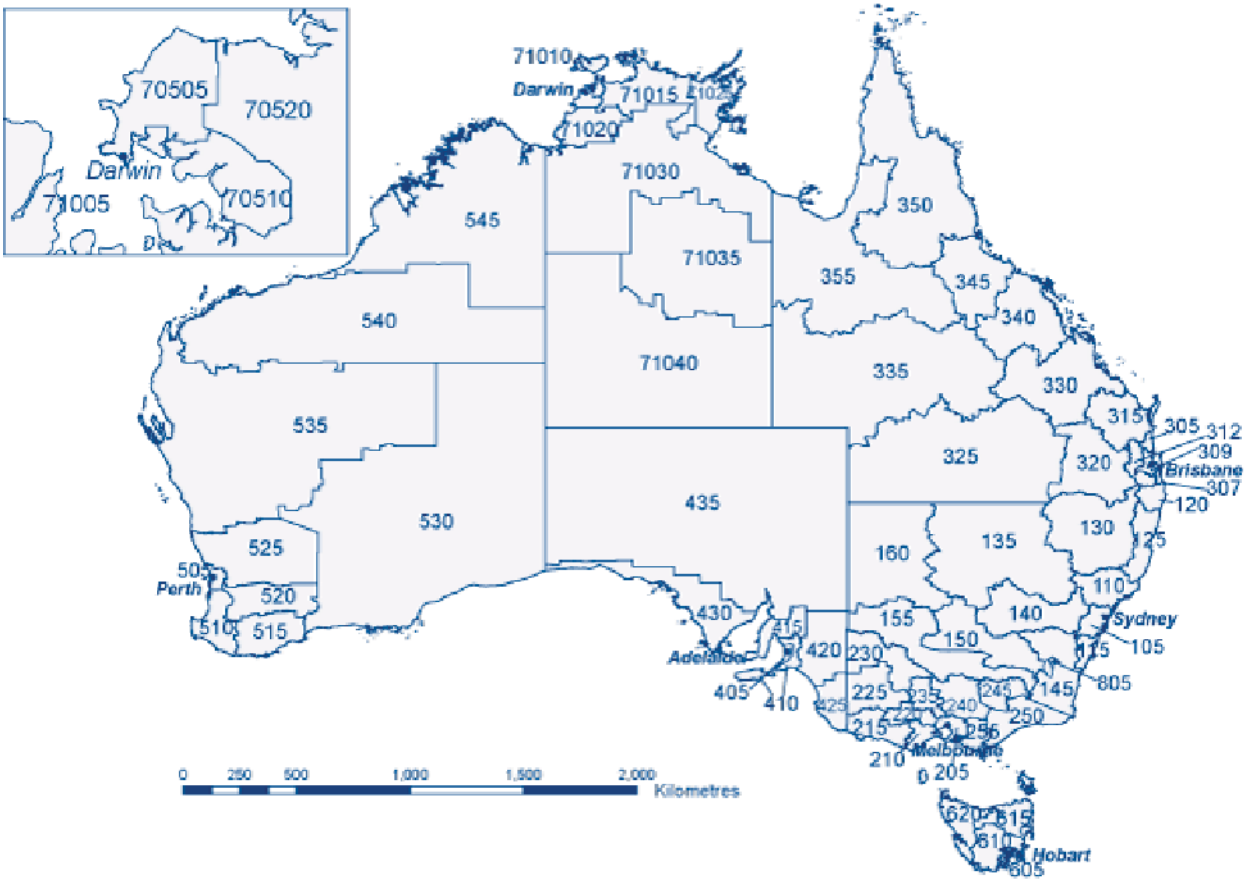


Table 2: Percentage of notifications from different sources, Australia, 2005, by state or territory

State or territory	Source of notifications		
	Laboratory only	Doctor only	Laboratory and doctor
ACT	98	1	1
NSW	70–80	20–30	<1
NT	95	5	<1
Qld	99	1	<1
SA	24	17	59
Tas	95	5	<1
Vic	50	7	43
WA	27	15	58

Source: Oxenford, Chapter 3 Current practices surrounding reporting of notifiable diseases by laboratories to state and territory health departments.[11]

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 per cent data completeness was defined as:

Per cent data completeness = (total notifications – missing or unknown) / total notifications x 100

The indigenous status was defined by the following nationally accepted values:[12]

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

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

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

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

9=Not stated, blank, unknown

Table 3: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2007

Disease	Data received from
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)*	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Queensland
Hepatitis C (unspecified)*,†	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis‡	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC,VTEC§	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial 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
Varicella zoster (chickenpox)**	All jurisdictions except New South Wales and Victoria
Varicella zoster (shingles)**	All jurisdictions except New South Wales and Victoria
Varicella zoster (unspecified)**	All jurisdictions except New South Wales and Victoria

Table 3: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2007, continued

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

* Unspecified hepatitis and syphilis includes cases in whom 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; Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Laboratory confirmed influenza was not a notifiable disease in South Australia but reports were forwarded to the National Notifiable Diseases Surveillance System.

** Nationally notifiable from 2006 and first full year of national reporting from 2007.

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

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

§§ Only invasive meningococcal disease is nationally notifiable. However, New South Wales and South Australia also report conjunctival cases.

NEC Not elsewhere classified.

Notes on cases definitions

Each notifiable disease is governed by a national surveillance case definition for reporting to NNDSS. 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 146,991 communicable disease notifications received by NNDSS in 2007 (Table 4).

In 2007, the most frequently notified diseases were sexually transmissible infections (62,474 notifications, 42.5% of total notifications), gastrointestinal diseases (30,325 notifications, 20.6% of total notifications) and vaccine preventable diseases (25,347 notifications, 17.2% of total notifications).

There were 19,570 notifications of bloodborne diseases; 6,823 notifications of vectorborne diseases; 1,762 notifications of other bacterial infections; 687 notifications of zoonoses and 3 quarantinable diseases (Table 4).

In 2007, the total number of notifications was the highest recorded in NNDSS since the surveillance system commenced data collection in 1991. There was an increase of 5% compared with the total number of notifications in 2006 (Figure 2). This was a small increase compared with increases observed in previous years and most likely related to the introduction of varicella as a new nationally notifiable disease.

Notifications and notification rates per 100,000 population for each disease by state or territory are shown in Table 5 and Table 6 respectively. Trends in notifications and rates per 100,000 population for the period 2002 to 2006 are shown in Table 7.

The major changes in communicable disease notifications in 2007 are shown in Figure 3 as the ratio of notifications in 2007 to the mean number of notifications for the previous 5 years, or in the case of infectious syphilis, 3 years. Notifications of mumps, laboratory-confirmed influenza, infectious syphilis < 2 years, leprosy, Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC), Barmah Forest virus infection, salmonellosis, and campylobacteriosis were above the historical mean. Notifications below the 5 year mean were *Haemophilus influenzae* type b, meningococcal infection, pertussis and measles. Notifications for the remaining diseases were within the historical range. The notification of a poliomyelitis case in 2007 was significant as it was the first case in 30 years, classified by WHO as an imported case as it was acquired in Pakistan.

Table 4: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2007, by disease category rank order

Disease category	Number	%
Sexually transmissible infections	62,474	42.5
Gastrointestinal diseases	30,325	20.6
Vaccine preventable diseases	25,347	17.2
Bloodborne diseases	19,570	13.3
Vectorborne diseases	6,823	4.6
Other bacterial infections	1,762	1.2
Zoonoses	687	0.5
Quarantinable diseases	3	<0.1
Total	146,991	100

Figure 2: Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2007

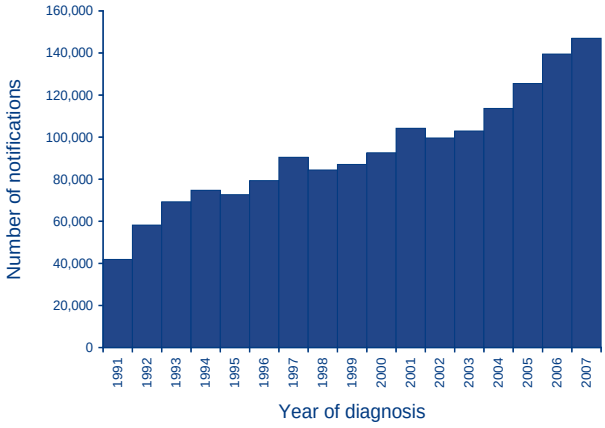
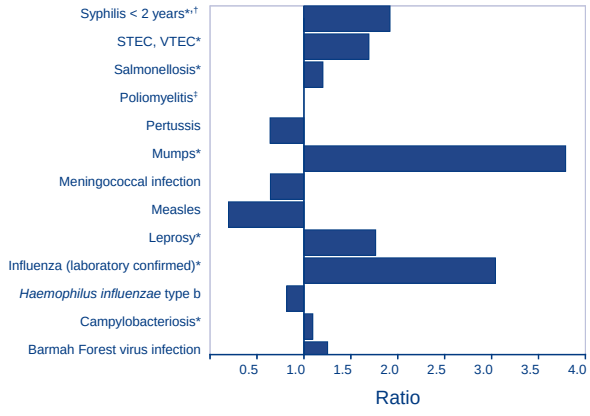


Figure 3: Comparison of total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2007, with the previous 5-year mean



* Exceeded 2 standard deviations above the 5-year mean.

† Syphilis < 2 years was based on a 3-year mean.

‡ Significant: 1st case in 30 years.

Table 5: Notifications of communicable diseases, Australia, 2007, by state or territory

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
Hepatitis B (incident)	13	56	9	63	11	9	84	42	287
Hepatitis B (unspecified)*	55	2,601	241	983	506	38	1,864	629	6,917
Hepatitis C (incident)	9	53	4	NN	48	20	145	76	355
Hepatitis C (unspecified)*,†	191	4,190	223	2,726	574	254	2,621	1,198	11,977
Hepatitis D	0	11	0	9	0	0	10	4	34
Gastrointestinal diseases									
Botulism	0	0	0	0	0	0	1	0	1
Campylobacteriosis‡	418	NN	289	4,438	2,675	712	6,352	2,100	16,984
Cryptosporidiosis	9	544	111	432	449	37	620	608	2,810
Haemolytic uraemic syndrome	1	13	0	1	1	0	3	0	19
Hepatitis A	2	65	5	28	5	3	36	21	165
Hepatitis E	1	8	0	3	0	0	6	0	18
Listeriosis	0	22	0	7	7	2	10	2	50
Salmonellosis	110	2,555	524	2,371	854	225	1,856	989	9,484
Shigellosis	0	71	173	88	62	3	96	104	597
STEC, VTEC§	1	23	3	24	41	0	13	2	107
Typhoid	0	34	3	6	5	3	30	9	90
Quarantinable diseases									
Cholera	0	2	0	1	0	0	0	0	3
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmitted infections									
Chlamydial infection‖	905	12,435	2,180	12,875	3,467	1,126	11,127	7,744	51,859
Donovanosis	0	0	1	2	0	0	0	0	3
Gonococcal infection	45	1,379	1,600	1,338	457	38	988	1,760	7,605
Syphilis (all)¶	28	1,106	281	440	51	36	843	214	2,999
Syphilis < 2 years duration*	9	434	119	232	51	8	427	101	1,381
Syphilis – > 2 years or unspecified duration*	19	672	162	208	NDP	28	416	113	1,618
Syphilis – congenital	0	6	2	0	0	0	0	0	8
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	7	2	3	1	0	2	2	17
Influenza (laboratory confirmed)**	390	1,918	183	4,590	280	415	1,589	1,038	10,403
Measles	0	4	0	4	1	0	2	1	12
Mumps	4	323	58	46	22	2	18	106	579
Pertussis	95	2,090	25	1,535	373	25	1,049	131	5,323
Pneumococcal disease (invasive)	34	522	66	323	91	30	278	131	1,475
Poliomyelitis	0	0	0	0	0	0	1	0	1
Rubella	2	8	0	14	1	0	7	4	36
Rubella – congenital	0	1	0	0	0	0	1	0	2

Table 5: Notifications of communicable diseases, Australia, 2007, by state or territory, *continued*

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Tetanus	0	2	0	0	0	1	0	0	3
Varicella zoster (chickenpox)††	9	NN	197	375	732	16	NN	322	1,651
Varicella zoster (shingles) ††	6	NN	89	387	587	92	NN	386	1,547
Varicella zoster (unspecified) ††	102	NN	3	3,072	437	25	NN	659	4,298
Vectorborne diseases									
Barmah Forest virus infection	6	572	91	826	58	0	26	137	1,716
Dengue virus infection	3	81	15	120	22	3	16	54	314
Flavivirus infection (NEC)‡‡	0	0	0	18	0	0	4	0	22
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0
Kunjin virus infection§§	0	0	0	0	0	0	1	0	1
Malaria	12	97	29	193	24	14	113	85	567
Murray Valley encephalitis virus infection§§	0	0	0	0	0	0	0	0	0
Ross River virus infection	12	840	300	2,137	211	7	95	601	4,203
Zoonoses									
Anthrax	0	0	0	0	0	0	1	0	1
Australia bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	4	0	30	1	1	1	1	38
Leptospirosis	0	8	1	75	1	0	16	5	106
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	34	0	2	2	1	50	3	92
Q fever	0	215	2	171	24	0	31	7	450
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial infections									
Legionellosis	4	105	3	52	17	3	42	81	307
Leprosy	0	4	0	1	2	1	2	2	12
Meningococcal infection‖‖	3	112	6	75	15	5	68	20	304
Tuberculosis	10	446	53	144	59	6	356	65	1,139
Total	2,480	32,567	6,772	40,028	12,174	3,153	30,474	19,343	146,991

* Unspecified hepatitis and syphilis includes cases in whom 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; Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Laboratory confirmed influenza was not a notifiable disease in South Australia but reports were forwarded to the National Notifiable Diseases Surveillance System.

†† Nationally notifiable from 2006 and first full year of national reporting from 2007.

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

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

‖‖ Only invasive meningococcal disease is nationally notifiable. However, New South Wales and South Australia also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Table 6: Notification rates for nationally notifiable communicable diseases, Australia, 2007, by state or territory

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 (incident)	3.8	0.8	4.2	1.5	0.7	1.8	1.6	2.0	1.4
Hepatitis B (unspecified)*	16.2	37.8	112.1	23.5	31.9	7.7	35.8	29.9	32.9
Hepatitis C (incident)	2.6	0.8	1.9	NN	3.0	4.1	2.8	3.6	2.1
Hepatitis C (unspecified)*,†	56.2	60.8	103.8	65.2	36.2	51.5	50.4	56.9	57.0
Hepatitis D	0.0	0.2	0.0	0.2	0.0	0.0	0.2	0.2	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	<0.1	0.0	<0.1
Campylobacteriosis‡	123.0	NN	134.5	106.1	168.9	144.3	122.0	99.7	120.2
Cryptosporidiosis	2.6	7.9	51.6	10.3	28.3	7.5	11.9	28.9	13.4
Haemolytic uraemic syndrome	0.3	0.2	0.0	<0.1	0.1	0.0	0.1	0.0	0.1
Hepatitis A	0.6	0.9	2.3	0.7	0.3	0.6	0.7	1.0	0.8
Hepatitis E	0.3	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.3	0.0	0.2	0.4	0.4	0.2	0.1	0.2
Salmonellosis	32.4	37.1	243.8	56.7	53.9	45.6	35.7	47.0	45.1
Shigellosis	0.0	1.0	80.5	2.1	3.9	0.6	1.8	4.9	2.8
STEC, VTEC§	0.3	0.3	1.4	0.6	2.6	0.0	0.2	0.1	0.5
Typhoid	0.0	0.5	1.4	0.1	0.3	0.6	0.6	0.4	0.4
Quarantinable diseases									
Cholera	0.0	<0.1	0.0	<0.1	0.0	0.0	0.0	0.0	<0.1
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‖	266.4	180.5	1014.3	307.9	218.8	228.2	213.8	367.7	246.8
Donovanosis	0.0	0.0	0.5	<0.1	0.0	0.0	0.0	0.0	<0.1
Gonococcal infection	13.2	20.0	744.4	32.0	28.8	7.7	19.0	83.6	36.2
Syphilis (all)¶	8.2	16.1	130.7	10.5	3.2	7.3	16.2	10.2	14.3
Syphilis < 2 years duration*	2.6	6.3	55.4	5.5	3.2	1.6	8.2	4.8	6.6
Syphilis – > 2 years or unspecified duration*	5.6	9.8	75.4	5.0	NDP	5.7	8.0	5.4	8.3
Syphilis – congenital	0.0	0.1	0.9	0.0	0.0	0.0	0.0	0.0	<0.1
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.9	0.1	0.1	0.0	<0.1	0.1	0.1
Influenza (laboratory confirmed)**	114.8	27.8	85.1	109.8	17.7	84.1	30.5	49.3	49.5
Measles	0.0	0.1	0.0	0.1	0.1	0.0	<0.1	<0.1	0.1
Mumps	1.2	4.7	27.0	1.1	1.4	0.4	0.3	5.0	2.8
Pertussis	28.0	30.3	11.6	36.7	23.5	5.1	20.2	6.2	25.3
Pneumococcal disease (invasive)	10.0	7.6	30.7	7.7	5.7	6.1	5.3	6.2	7.0
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	<0.1	0.0	<0.1
Rubella	0.6	0.1	0.0	0.3	0.1	0.0	0.1	0.2	0.2
Rubella – congenital	<0.1	<0.1	0.0	0.0	0.0	0.0	<0.1	0.0	<0.1

Table 6: Notification rates for nationally notifiable communicable diseases, Australia, 2007, by state or territory, *continued*

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Tetanus	0.0	<0.1	0.0	0.0	0.0	0.2	0.0	0.0	<0.1
Varicella zoster (chickenpox)††	2.6	NN	91.7	9.0	46.2	3.2	NN	15.3	18.5
Varicella zoster (shingles)††	1.8	NN	41.4	9.3	37.1	18.6	NN	18.3	17.3
Varicella zoster (unspecified)††	30.0	NN	1.4	73.5	27.6	5.1	NN	31.3	48.2
Vectorborne diseases									
Barmah Forest virus infection	1.8	8.3	42.3	19.8	3.7	0.0	0.5	6.5	8.2
Dengue virus infection	0.9	1.2	7.0	2.9	1.4	0.6	0.3	2.6	1.5
Flavivirus infection (NEC)‡‡	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.0	0.1
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	<0.1	0.0	<0.1
Malaria	3.5	1.4	13.5	4.6	1.5	2.8	2.2	4.0	2.7
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	3.5	12.2	139.6	51.1	13.3	1.4	1.8	28.5	20.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	<0.1	0.0	<0.1
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.7	0.1	0.2	<0.1	<0.1	0.2
Leptospirosis	0.0	0.1	0.5	1.8	0.1	0.0	0.3	0.2	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	0.5	0.0	<0.1	0.1	0.2	1.0	0.1	0.4
Q fever	0.0	3.1	0.9	4.1	1.5	0.0	0.6	0.3	2.1
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	1.2	1.5	1.4	1.2	1.1	0.6	0.8	3.8	1.5
Leprosy	0.0	0.1	0.0	<0.1	0.1	0.2	<0.1	0.1	0.1
Meningococcal infection‖‖	0.9	1.6	2.8	1.8	0.9	1.0	1.3	0.9	1.4
Tuberculosis	2.9	6.5	24.7	3.4	3.7	1.2	6.8	3.1	5.4

* Unspecified hepatitis and syphilis includes cases in whom 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; Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Laboratory confirmed influenza was not a notifiable disease in South Australia but reports were forwarded to the National Notifiable Diseases Surveillance System.

†† Nationally notifiable from 2006 and first full year of national reporting from 2007.

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

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

‖‖ Only invasive meningococcal disease is nationally notifiable. However, New South Wales and South Australia also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Table 7: Notifications and notification rate for communicable diseases, Australia, 2002 to 2007, (per 100,000 population)

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2002	2003	2004	2005	2006	2007			2002	2003	2004	2005	2006	2007
Bloodborne diseases														
Hepatitis (NEC)	0	0	0	0	1	0	0.2	0.0	0.0	0.0	0.0	0.0	<0.1	0.0
Hepatitis B (incident)	392	348	282	251	294	287	313.4	0.9	2.0	1.7	1.4	1.2	1.4	1.4
Hepatitis B (unspecified)*	6,673	5,814	5,789	6,327	6,276	6,917	6,175.8	1.1	34.0	29.2	28.8	31.0	30.3	32.9
Hepatitis C (incident)	452	518	453	376	450	355	449.8	0.8	2.8	3.2	2.8	2.3	2.7	2.1
Hepatitis C (unspecified)*,†	15,615	13,661	12,694	11,992	11,972	11,977	13,186.8	0.9	79.5	68.7	63.1	58.8	57.8	57.0
Hepatitis D	22	27	29	30	31	34	27.8	1.2	0.1	0.1	0.1	0.1	0.1	0.2
Gastrointestinal diseases														
Botulism	0	1	1	3	1	1	1.5	0.7	0.0	<0.1	<0.1	<0.1	<0.1	<0.1
Campylobacteriosis‡	14,744	15,361	15,579	16,493	15,407	16,984	15,516.8	1.1	113.2	116.2	116.1	120.9	111.0	120.2
Cryptosporidiosis	3,273	1,223	1,685	3,212	3,206	2,810	2,519.8	1.1	16.7	6.1	8.4	15.7	15.5	13.4
Haemolytic uraemic syndrome	13	15	16	20	14	19	15.6	1.2	0.1	0.1	0.1	0.1	0.1	0.1
Hepatitis A	392	430	319	326	281	165	349.6	0.5	2.0	2.2	1.6	1.6	1.4	0.8
Hepatitis E	12	12	28	30	24	18	21.2	0.8	0.1	0.1	0.1	0.1	0.1	0.1
Listeriosis	63	69	67	54	61	50	62.8	0.8	0.3	0.3	0.3	0.3	0.3	0.2
Salmonellosis	7,880	7,011	7,841	8,426	8,258	9,484	7,883.2	1.2	40.1	35.2	39.0	41.3	39.9	45.1
Shigellosis	507	442	520	729	545	597	548.6	1.1	2.6	2.2	2.6	3.6	2.6	2.8
STEC, VTEC§	59	52	49	86	70	107	63.2	1.7	0.3	0.3	0.2	0.4	0.3	0.5
Typhoid	69	51	76	52	77	90	65.0	1.4	0.4	0.3	0.4	0.3	0.4	0.4
Quarantinable diseases														
Cholera	5	1	5	3	3	3	3.4	0.9	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
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
Plague	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
Severe acute respiratory syndrome	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
Viral haemorrhagic fever	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

Table 7: Notifications and notification rate for communicable diseases, Australia, 2002 to 2007, (per 100,000 population), *continued*

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2002	2003	2004	2005	2006	2007			2002	2003	2004	2005	2006	2007
Sexually transmissible infections														
Chlamydial infection‖	24,459	30,415	36,186	41,353	47,449	51,859	3,5972.4	1.4	124.5	152.9	179.8	202.8	229.2	246.8
Donovanosis	17	16	10	13	6	3	12.4	0.2	0.1	0.1	<0.1	0.1	<0.1	<0.1
Gonococcal infection	6,439	6,771	7,145	8,039	8,573	7,605	7,393.4	1.0	32.8	34.0	35.5	39.4	41.4	36.2
Syphilis (all)¶	2,169	2,139	2,341	2,241	2,691	2,999	2,316.2	1.3	11.0	10.8	11.6	11.0	13.0	14.3
Syphilis < 2 years duration*	NN	NN	636	653	871	1,381	720.0**	1.9	NN	NN	3.2	3.2	4.2	6.6
Syphilis > 2 years or unspecified duration*	NN	NN	1,705	1,588	1,820	1,618	1,704.3**	0.9	NN	NN	9.2	8.4	9.5	8.3
Syphilis – congenital	18	13	13	15	13	8	14.4	0.6	0.1	0.1	0.1	0.1	0.1	<0.1
Vaccine preventable diseases														
Diphtheria	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	31	19	15	17	22	17	20.8	0.8	0.2	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)††	3,669	3,481	2,135	4,565	3,255	10,403	3,421.0	3.0	18.7	17.5	10.6	22.4	15.7	49.5
Measles	32	93	45	10	125	12	61.0	0.2	0.2	0.5	0.2	<0.1	0.6	0.1
Mumps	69	77	102	241	275	579	152.8	3.8	0.4	0.4	0.5	1.2	1.3	2.8
Pertussis	5,564	5,096	8,759	11,203	10,996	5,323	8,323.6	0.6	28.3	25.6	43.5	54.9	53.1	25.3
Pneumococcal disease (invasive)	2,441	2,233	2,369	1,745	1,455	1,475	2,048.6	0.7	12.4	11.2	11.8	8.6	7.0	7.0
Poliomyelitis	0	0	0	0	0	1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	253	54	31	31	59	36	85.6	0.4	1.3	0.3	0.2	0.2	0.3	0.2
Rubella – congenital	2	3	1	1	0	2	1.8	1.1	<0.1	<0.1	<0.1	<0.1	0.0	<0.1
Tetanus	4	4	5	2	3	3	3.6	0.8	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Varicella zoster (chickenpox)‡‡	NN	NN	NN	16	1,521	1,651	768.5§§	2.1	NN	NN	NN	0.2	17.4	18.5
Varicella zoster (shingles)‡‡	NN	NN	NN	7	1,079	1,547	543.0§§	2.8	NN	NN	NN	0.1	12.3	17.3
Varicella zoster (unspecified)‡‡	NN	NN	NN	141	3,664	4,298	1,902.5§§	2.3	NN	NN	NN	1.6	41.8	48.2
Vectorborne diseases														
Barmah Forest virus infection	910	1,367	1,105	1,324	2,142	1,716	1,369.6	1.3	4.6	6.9	5.5	6.5	10.3	8.2
Dengue virus infection	170	861	351	221	188	314	358.2	0.9	0.9	4.3	1.7	1.1	0.9	1.5
Flavivirus infection (NEC)‖‖	73	60	61	27	32	22	50.6	0.4	0.4	0.3	0.3	0.1	0.2	0.1
Japanese encephalitis virus infection	0	1	1	0	0	0	0.7	0.0	0.0	<0.1	<0.1	0.0	0.0	0.0
Kunjin virus infection¶¶	0	7	6	1	3	1	3.4	0.3	0.0	<0.1	<0.1	<0.1	<0.1	<0.1
Malaria	468	592	557	822	772	567	642.2	0.9	2.4	3.0	2.8	4.0	3.7	2.7
Murray Valley encephalitis virus infection¶¶	2	0	1	2	1	0	1.5	0.0	<0.1	0.0	<0.1	<0.1	<0.1	0.0
Ross River virus infection	1,459	3,850	4,209	2,545	5,547	4,203	3,522.0	1.2	7.4	19.4	20.9	12.5	26.8	20.0

Table 7: Notifications and notification rate for communicable diseases, Australia, 2002 to 2007, (per 100,000 population), *continued*

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2002	2003	2004	2005	2006	2007			2002	2003	2004	2005	2006	2007
Zoonoses														
Anthrax	0	0	0	0	1	1	0.2	5.0	0.0	0.0	0.0	0.0	<0.1	<0.1
Australian bat lyssavirus	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	40	20	38	41	50	38	37.8	1.0	0.2	0.1	0.2	0.2	0.2	0.2
Leptospirosis	160	126	177	129	147	106	147.8	0.7	0.8	0.6	0.9	0.6	0.7	0.5
Lyssavirus (NEC)	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	213	200	239	164	169	92	197.0	0.5	1.1	1.0	1.2	0.8	0.8	0.4
Q fever	795	560	464	353	407	450	515.8	0.9	4.0	2.8	2.3	1.7	2.0	2.1
Tularaemia	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections														
Legionellosis	315	333	312	331	350	307	328.2	0.9	1.6	1.7	1.6	1.6	1.7	1.5
Leprosy	6	5	7	10	6	12	6.8	1.8	<0.1	<0.1	<0.1	<0.1	<0.1	0.1
Meningococcal infection***	689	558	405	392	317	304	472.2	0.6	3.5	2.8	2.0	1.9	1.5	1.4
Tuberculosis	1,130	1,048	1,137	1,085	1,193	1,139	1,118.6	1.0	5.8	5.3	5.6	5.3	5.8	5.4
Total	99,599	102,910	113,666	125,497	139,482	146,991	11,6230.8	1.3						

* Unspecified hepatitis and syphilis includes cases in whom 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; Northern Territory, which excludes ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

** Ratios for syphilis <2 years; syphilis >2 years or unspecified duration based on 3 years data.

†† Laboratory confirmed influenza was not a notifiable disease in South Australia but reports were forwarded to the National Notifiable Diseases Surveillance System.

‡‡ Nationally notifiable from 2006 and first full year of national reporting from 2007.

§§ Ratios for varicella (chickenpox), varicella (shingles) and varicella (unspecified) based on 2 years data.

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

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

*** Only invasive meningococcal disease is nationally notifiable. However, New South Wales and South Australia also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

Data completeness

The case's sex was complete in 99.8% of notifications and age at onset in close to 100% of notifications (Table 8). In 2007, indigenous status was complete in 47.5% of notifications, and varied by jurisdiction. Indigenous status was complete for 88.8% of data reported in the Northern Territory, 79.7% in South Australia and 70.1% in Western Australia. In the remaining jurisdictions, less than 54% of data were complete for indigenous status.

Data completeness on indigenous status also varied by disease as summarised in Appendix 3. There were 6 diseases for which notifications were 100% complete for indigenous status.[12] A further 8 diseases equalled or exceeded 90% completeness for indigenous status. Of the 12 key diseases agreed to by CDNA and the NSC in 2007 for improving Indigenous identification, seven of these had an Indigenous completeness, which exceeded 90% (donovanosis, infectious syphilis, *Haemophilus influenzae* type b, tuberculosis, leprosy, meningococcal infection and measles). The diseases for which there was less than 90% Indigenous completeness included gonococcal infection, invasive pneumococcal disease, hepatitis A, dengue virus infection, and shigellosis. In 2008, CDNA set target thresholds of 95% completeness for key diseases and 85% 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 the National Centre in HIV Epidemiology and Clinical Research. Information on national HIV and AIDS surveillance can be obtained from the NCHECR website at http://www.nchecr.unsw.edu.au[4]

Hepatitis B

Hepatitis B notifications are classified as either newly acquired (incident) hepatitis B or hepatitis B with an unspecified period of infection. Classification of hepatitis B cases as newly acquired is based on serological evidence or evidence of a previously negative test within the last 24 months.

Incident hepatitis B notifications

In 2007, 287 cases of incident hepatitis B infection were reported to NNDSS, which was lower than in 2006 (n=294). Over the past 10 years, the notification rate for incident hepatitis B infection increased from 1.5 cases per 100,000 population in 1997 to 2.2 cases per 100,000 population in 2001, then declined to 1.2 cases per 100,000 population in 2005 and increased to 1.4 cases per 100,000 population in 2006 and 2007 (Figure 4).

The Northern Territory and the Australian Capital Territory recorded the highest notification rates in 2007 with 4.2 and 3.8 cases per 100,000 population respectively. At a regional level, incident hepatitis B rates were highest in the Barkly, Lower Top End and East Arnhem Statistical Subdivisions of the Northern Territory (range: 5.4–15.9 cases per 100,000 population, 5 cases total); and in the Far North and South West Statistical Divisions of Queensland, the Upper Great Southern Statistical Division in Western Australia, and in the East Gippsland, Central Highlands and Barwon Statistical Divisions of Victoria (range: 3.2–5.3 cases per 100,000 population) (Map 2).

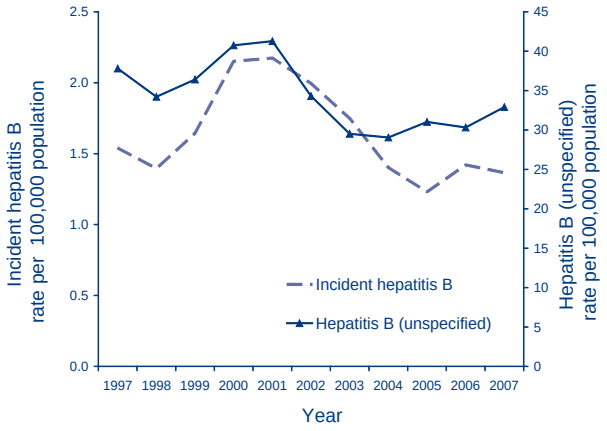
In 2007, the sex of cases was reported in 286 of the 287 cases. Figure 5 shows that the highest rate of incident hepatitis B infection was in the 25–29 years age group among both males and females (4.4 and 3.4 cases per 100,000 population, respectively). Notifications of incident hepatitis B infection in males exceeded those in females, with a male to female ratio of 1.8:1.

Table 8: Completeness of the National Notifiable Diseases Surveillance System data received, Australia, 2007, by state or territory

	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total notifications	2,480	32,575	6,772	41,219	12,179	3,153	30,478	20,047	148,903
Sex									
Unknown/missing	6	125	1	0	6	0	201	0	339
Per cent complete	99.8	99.6	100.0	100.0	100.0	100.0	99.3	100.0	99.8
Age at onset									
Unknown/missing	0	1	1	0	1	1	35	1	40
Per cent complete	100.0	100.0	100.0	100.0	100.0	99.9	99.9	100.0	99.9
Indigenous status									
Unknown/missing	2,099	25,797	757	24,728	2,470	1,195	15,106	6,004	78,156
Per cent complete	15.4	20.8	88.8	40.0	79.7	62.1	50.4	70.1	47.5

Trends in incident hepatitis B infection by year and age group are shown in Figure 6. Since the introduction of the adolescent hepatitis B vaccination program for children aged 10–13 years in 1997[13] there has been a general decline in hepatitis B among the 15–19 years and 20–29 years age groups. Between 2000 and 2007, the notification rate for incident hepatitis B fell by 75% among cases in the 15–19 years age group. In the 20–29 years age group, the notification rate fell by 55% between 2000 and 2005 and has remained stable at around 3.2 cases per 100,000 population from 2005 to 2007.

Figure 4: Notification rate for incident hepatitis B* and hepatitis B (unspecified),† Australia, 1997 to 2007, by year‡

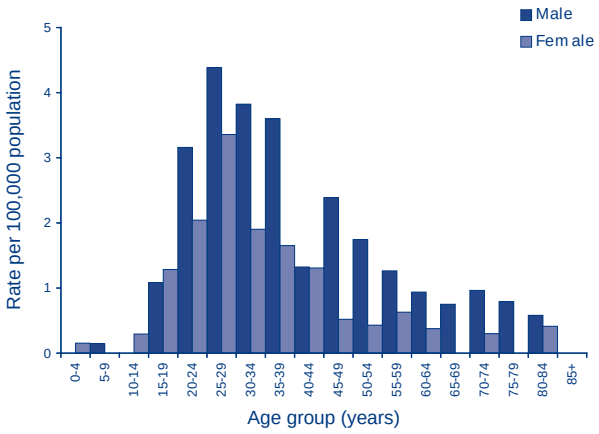


* Data for incident hepatitis B from all states except the Northern Territory between 1997 and 2004.

† Data provided from the Northern Territory (1997–2004) includes both incident and unspecified hepatitis B cases.

‡ Year of onset for incident hepatitis B and year of notification for hepatitis B (unspecified) notifications.

Figure 5: Notification rate for incident hepatitis B infections, Australia, 2007, by age group and sex*



* Excludes one case whose sex was not reported.

Map 2: Notification rates for incident hepatitis B, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory

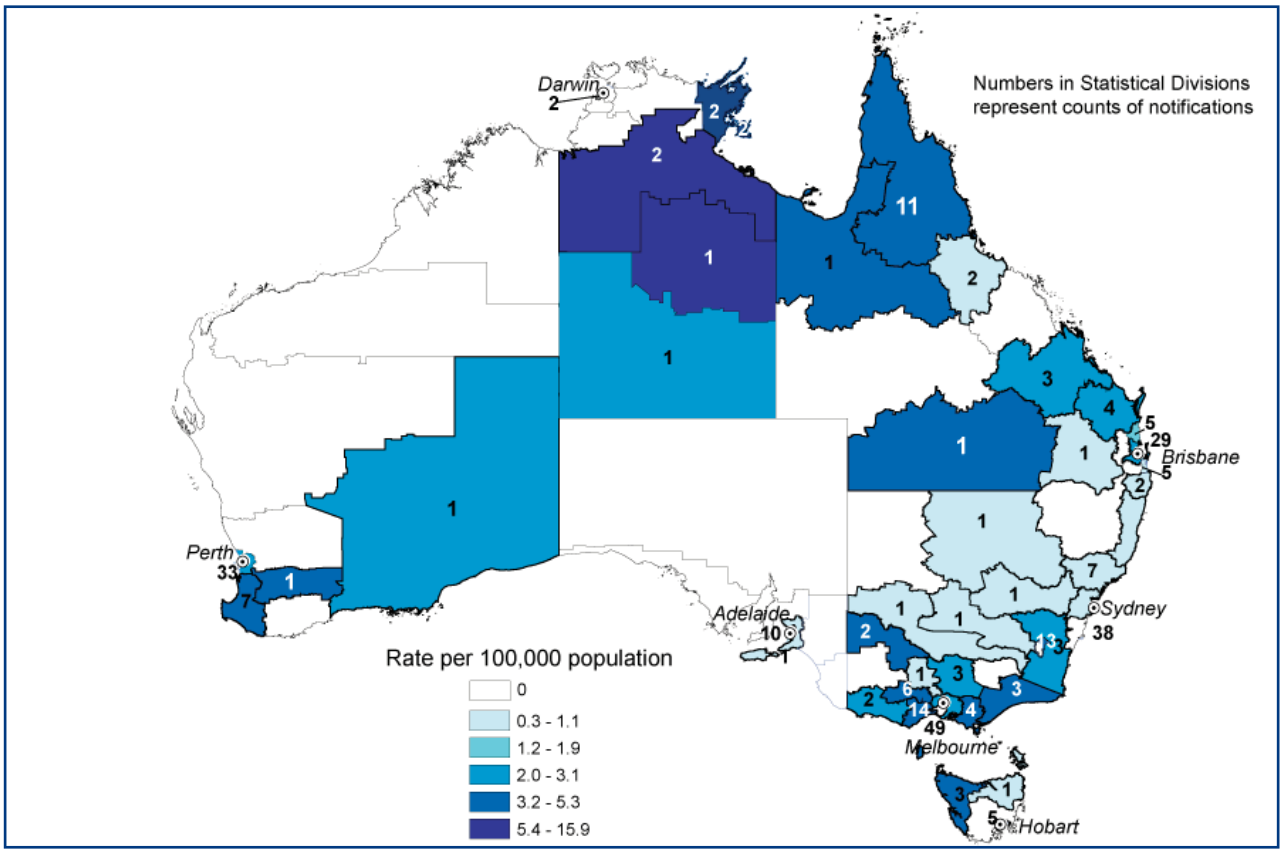
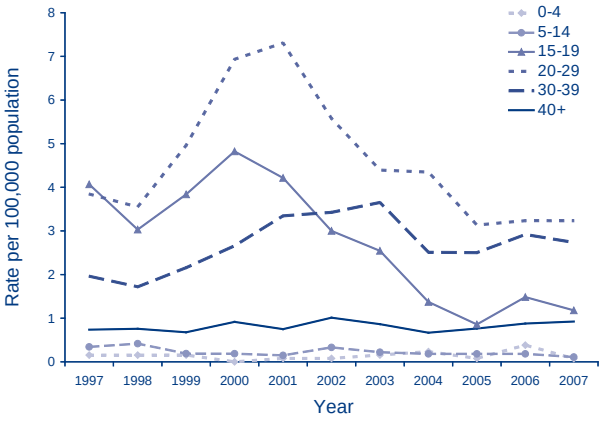


Figure 6: Notification rate for incident hepatitis B infections, Australia, 1997 to 2007, by year and age group



* Data provided from the Northern Territory (1997–2004) includes both incident and unspecified hepatitis B cases.

The source of exposure for cases of incident hepatitis B infection in 2007 was reported through health authorities in South Australia, Tasmania and Victoria (Table 9). From 2003 to 2007, the proportion of notifications of incident hepatitis B infection associated with injecting drug use remained relatively stable at around 49%. The proportion of diagnoses attributed to heterosexual contact decreased from 21% in 2003 to 16% in 2007. The source of exposure to hepatitis B was undetermined in around 21% of cases.[4]

Table 9: Incident hepatitis B infection,* 2007, by exposure category†

Exposure category	Number	Percentage (%)
Injecting drug use	49	47.6
Sexual contact	20	19.4
Male homosexual contact	*3*	*2.9*
Heterosexual contact	*17*	*16.5*
Not specified	*0*	*0*
Blood/tissue recipient	0	0
Skin penetration procedure	4	3.9
Healthcare exposure	1	0.9
Household contact	5	4.9
Other	20	19.4
Undetermined	4	3.9
Total exposures	103	100

* Includes diagnoses in South Australia, Tasmania and Victoria.

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

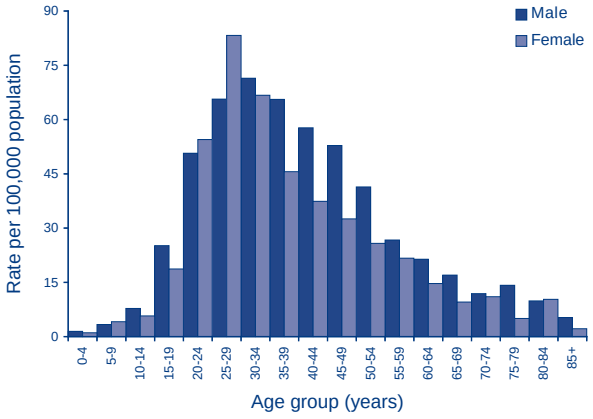
Source: National Centre in HIV Epidemiology and Clinical Research.[4]

Hepatitis B (unspecified) notifications

In 2007, a total of 6,917 cases of hepatitis B (unspecified) infection were notified to the NNDSS, compared with 6,276 in 2006. The Northern Territory recorded the highest notification rate (112.1 cases per 100,000 population), followed by New South Wales (37.8 cases per 100,000 population) and Victoria (35.8 cases per 100,000 population).

In 2007, the sex of cases was recorded in 6,848 of 6,917 cases (99%). Of these cases, the male to female ratio of notifications was 1.2:1. Among males, the highest notification rate was in the 30–34 years age group (71.4 cases per 100,000 population), followed by the 25–29 and 35–39 years age groups at 65.6 cases per 100,000 population in both age groups. Among females, the highest notification rate was in the 25–29 years age group (83.3 cases per 100,000 population), followed by 66.7 cases per 100,000 population in the 30–34 years age group (Figure 7).

Figure 7: Notification rate for hepatitis B (unspecified) infection, Australia, 2007, by age group and sex*

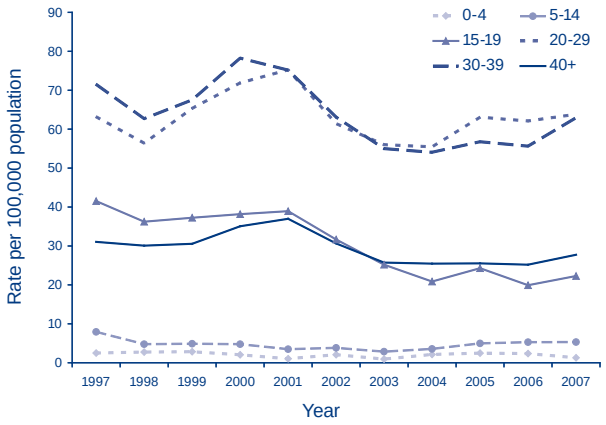


* Excludes 69 cases whose sex was not reported.

Notification rates for hepatitis B infection (unspecified) increased from 37.8 cases per 100,000 population in 1997 to 41.3 in 2001 and then declined to around 30.3 cases per 100,000 population in 2006 (Figure 4). In 2007, the rate of hepatitis B (unspecified) notifications (32.9 cases per 100,000 population) remained consistent with the range of rates seen between 2003 and 2006 (29.2–31.0 cases per 100,000 population). Trends in hepatitis B (unspecified) infection by age group, sex and year are shown in Figure 8. Rates in the 15–19 years age group increased in 2007 by 12.1% compared with 2006 (22.3 and 19.9 cases per 100,000 population respectively), however, the 2007 rate in this age group is consistent with rates between 2003 and 2005 (range 20.9–25.2 cases per 100,000 population).

In 2007, 1 case of incident hepatitis B and 17 cases of hepatitis B (unspecified) infection were notified in children in the 0–4 years age group and represented 0.4% and 0.3% of hepatitis cases notified in these specific categories respectively. Approximately 95% of infants born in Australia in 2007 received the full-course of the hepatitis B vaccine.[4]

Figure 8: Notification rate for hepatitis B (unspecified) infection, Australia,* 1997 to 2007, by year and age group



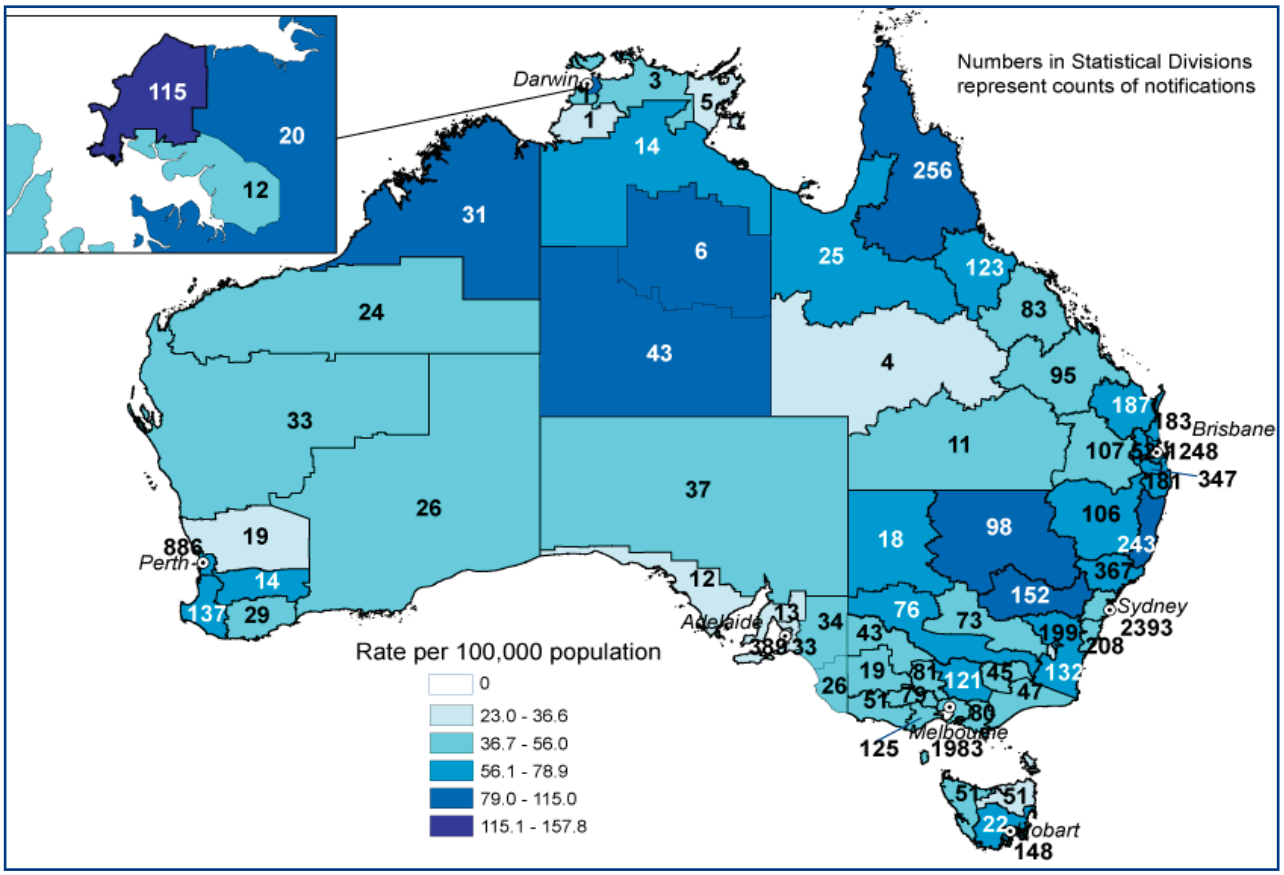
* Data for hepatitis B (unspecified) from all states except the Northern Territory between 1997 and 2004.

Hepatitis C

Hepatitis C notifications are classified as either newly acquired (incident) hepatitis C or hepatitis C with a period of infection greater than 2 years or unspecified. The categorising of hepatitis C cases is complex as current testing methods cannot distinguish between incident and chronic infections (greater than 2 years or unspecified). Cases are essentially categorised based on evidence of a previously negative test result within 2 years of their diagnosis. In most instances this requires active follow-up by public health units.

Since 2001, there has been a steady decline in cases of hepatitis C nationally (Figure 9). Map 3 shows the distribution of both incident hepatitis C and hepatitis C of greater than 2 years or unspecified duration diagnosed during 2007. The highest rates of hepatitis C were seen in the Darwin and Darwin City Statistical Subdivisions (125.2 and 157.8 cases per 100,000 population respectively). Notification rates were also substantially above the national notification rate in the Kimberley Statistical Division of Western Australia; the Central and Barkly Statistical Subdivisions of the Northern Territory; the Far North Statistical Division of Queensland; and the

Map 3: Notification rates for incident hepatitis C and hepatitis C (unspecified), Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory

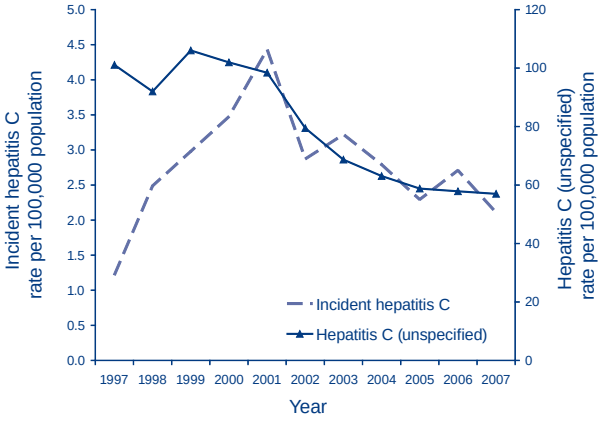


Mid-North Coast, North Western and Central West Statistical Divisions of New South Wales (79.0–115.0 cases per 100,000 population).

Incident hepatitis C notifications

Notifications of incident hepatitis C were received from all jurisdictions except Queensland, where all cases of hepatitis C are reported as hepatitis C (unspecified). A total of 355 cases of incident hepatitis C were notified in 2007 (450 cases in 2006), giving a notification rate of 2.1 cases per 100,000 population (Figure 9).

Figure 9: Notification rates for incident hepatitis C infection* and hepatitis C (unspecified),† Australia, 1997 to 2007



* Data from all states and territories except Queensland 1997–2007 and the Northern Territory 1997–2002.

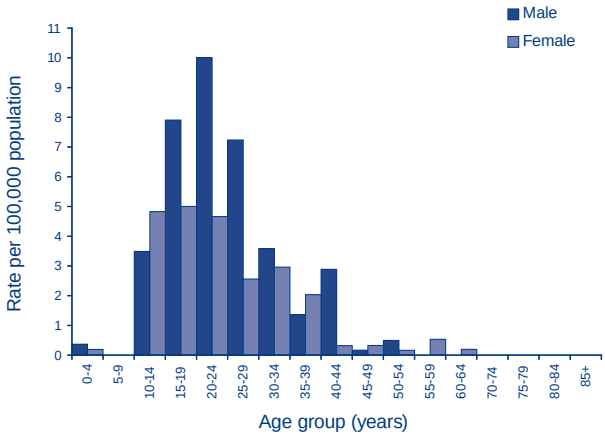
† Data provided from Queensland (1997–2007) and the Northern Territory (1997–2002) includes both incident and unspecified hepatitis C cases.

The proportion of all hepatitis C notifications in 2007, excluding Queensland, that were documented as incident cases was 3.6%, compared with 4.5% in 2006. The highest rates of incident hepatitis C infection were reported from Tasmania (4.1 cases per 100,000 population) and Western Australia (3.6 cases per 100,000 population). The number of incident hepatitis C notifications in 2007, both nationally and for each jurisdiction, is influenced by the level of case follow-up. One possible explanation for the highest rate observed in Tasmania is the opportunity to detect additional cases through follow-up and repeated surveys.

In 2007, the sex of cases was reported in 354 of the 355 cases notified. Figure 10 shows that in 2007 the highest incident hepatitis C notification rates were in the 25–29 years age group in males (9.7 cases per 100,000 population). In females, notification rates were highest in the 15–19 years age group (4.8 cases per 100,000 population) followed by the 10–14 and 20–24 years age groups (4.6 and 4.4 cases per 100,000 population respectively).

Trends in the age distribution of incident hepatitis C infection are shown in Figure 11. Notification rates from 2001 to 2007 declined by 62% in the 15–19 years age group; 59% in the 20–29 years age group; and by 44% in the 30–39 years age group. In 2006 to 2007, notification rates decreased by 26% in the 20–29 years age group and by 23% in the 30–39 years age group.

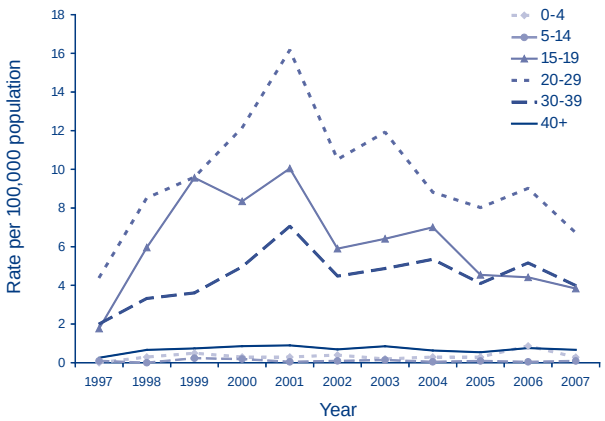
Figure 10: Notification rate for incident hepatitis C infection, Australia,* 2007, by age group and sex†



* Data from all states and territories except Queensland.

† Excludes 1 case whose sex was not reported.

Figure 11: Notification rate for incident hepatitis C infection, Australia,* 1997 to 2007, by age group and year



* Data from all states and territories except Queensland (1997–2007) and the Northern Territory (1997–2002).

The exposure history of cases of incident hepatitis C were collected in New South Wales, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia in 2007 (Table 10). Approximately 77% of these hepatitis cases were among people with a history of injecting drug use. In eight of the cases the only reported risk factor was having been born to a woman with hepatitis C infection.

Table 10: Incident hepatitis C infection, Australia,* 2007, by exposure category†

Exposure category	Number	Percentage
Injecting drug use	207	77.5
Sexual contact	7	2.6
Blood/tissue recipient	3	1.1
Skin penetration procedure	4	1.5
Healthcare exposure	2	0.8
Household contact	0	0
Other‡	14	5.2
Undetermined	30	11.3
Total exposures	267	100.0

Source: National Centre in HIV Epidemiology and Clinical Research.4

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

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

‡ Includes 8 cases for which the only reported risk factor was having been born to a woman with hepatitis C infection.

Hepatitis C (unspecified) notifications

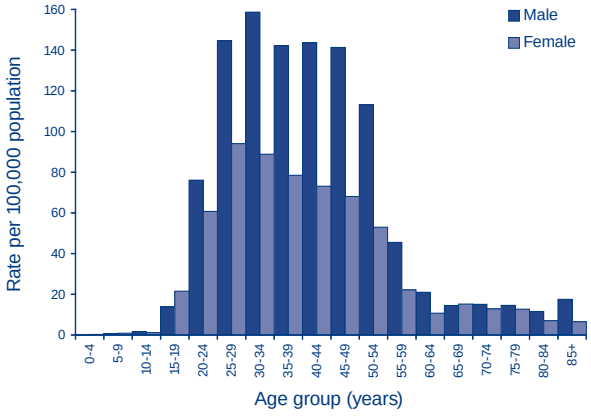
In 2007, 11,977 hepatitis C (unspecified) infections were notified to the NNDSS, representing 57.0 cases per 100,000 population (11,972 cases and 57.8 cases per 100,000 population in 2006).

The national notification rate for hepatitis C (unspecified) infection declined from 106.0 cases per 100,000 population in 1999 to 58.8 cases per 100,000 population in 2005 and has remained stable between 2005 and 2007 (range 57.0–58.8 cases per 100,000 population) (Figure 9). Improved surveillance practices, such as more complete follow-up and classification of incident cases; increased duplicate notification checks; and the Northern Territory separately reporting incident hepatitis C notifications from 2003, may account for some of the decrease in hepatitis C (unspecified) notifications since 2000.

In 2007, the Northern Territory continued to have the highest notification rate (103.8 cases per 100,000 population), followed by Queensland (65.2 cases per 100,000 population), which includes both incident and unspecified cases; and New South Wales (60.8 cases per 100,000 population).

The sex of cases was reported in 11,923 of the 11,977 cases in 2007. Of these cases nationally, the male to female ratio was 1.7:1. The highest notification rate occurred in the 30–34 years age group (158.7 cases per 100,000 population) among males and in the 25–29 and 30–34 years age groups (94.0 and 88.9 cases per 100,000 population respectively) among females (Figure 12).

Figure 12: Notification rate for hepatitis C (unspecified) infection,* Australia, 2007, by age group and sex†

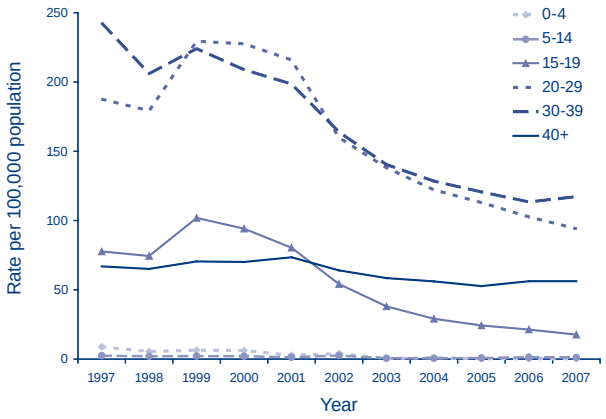


* Data provided from Queensland includes both incident and unspecified hepatitis C cases.

† Excludes 54 cases whose sex was not reported.

Trends in the age distribution of hepatitis C (unspecified) infection are shown in Figure 13. From 2003 to 2007, the notification rate for hepatitis C (unspecified) among the 15–19 years age group decreased by 67%. Between 2003 and 2007, notification rates fell on average by 9.1% per year among cases in the 20–29 years age group. In the 30–39 years age group, notification rates have also been declining, on average by 4.3% per year since 2003. The decline in the population rate of notifications of hepatitis C infection may be attributable to a reduction in the prevalence of risk behaviours related to injecting drug use, especially among young people, however, changes in the rates of testing and percentage classified as incident cases may also have contributed to the decline.

Figure 13: Notification rates for hepatitis C (unspecified) infection,* Australia, 1997 to 2007, by age group



* Data provided from Queensland (1997 to 2007) and the Northern Territory (1995 to 2002) includes both incident and unspecified hepatitis C cases.

Although initial infection with the hepatitis C virus may be asymptomatic (more than 90% of cases) or mildly symptomatic, a high percentage (50%–80%) of cases will develop a chronic infection. Of chronically infected persons, approximately 50% will eventually develop cirrhosis or cancer of the liver.[4] In 2007, it is estimated that 278,000 people living in Australia, had been exposed to the hepatitis C virus. Of these people approximately 160,000 had chronic hepatitis C infection and early liver disease (stage F0/1), and 42,000 had chronic hepatitis C infection and moderate liver disease (stage F2/3) associated with chronic hepatitis C infection; 5,600 were living with hepatitis C related cirrhosis; and 68,500 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.[4] People co-infected with hepatitis B and hepatitis D may have more severe acute disease and a higher risk of fulminant hepatitis compared to those with hepatitis B alone. The modes of hepatitis D transmission are similar to those for hepatitis B, and in countries with low hepatitis B prevalence, injecting drug users are the main group at risk for hepatitis D infection.

There were 34 notifications of hepatitis D to the NNDSS in 2007, compared with 31 notifications in 2006, giving a notification rate of 0.2 cases per 100,000 population. The male to female ratio was 2.4:1. Of the 34 notifications, 11 were reported from New South Wales, 10 from Victoria, 9 from Queensland and 4 from Western Australia.

Gastrointestinal diseases

In 2007, gastrointestinal diseases notified to NNDSS were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, STEC infections and typhoid.

Notifications of gastrointestinal diseases in 2007 increased 9% to 30,325 from 27,947 in 2006 (Table 7).

Campylobacteriosis, salmonellosis and STEC exceeded the 5-year mean by more than 2 standard deviations, while typhoid, HUS and cryptosporidiosis were increased but did not exceed 2 standard deviations (Figure 3).

OzFoodNet, Australia's enhanced foodborne disease surveillance network monitors the incidence of diseases caused by pathogens commonly transmitted by food through population-based passive and enhanced surveillance for notifiable gastrointestinal diseases and for outbreaks of gastroenteritis and enteric diseases. In 2007, OzFoodNet aggregated and analysed data from NNDSS and enhanced surveillance data from OzFoodNet sites on the following 8 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; and HUS. These data are reported in detail elsewhere.[14]

Botulism

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

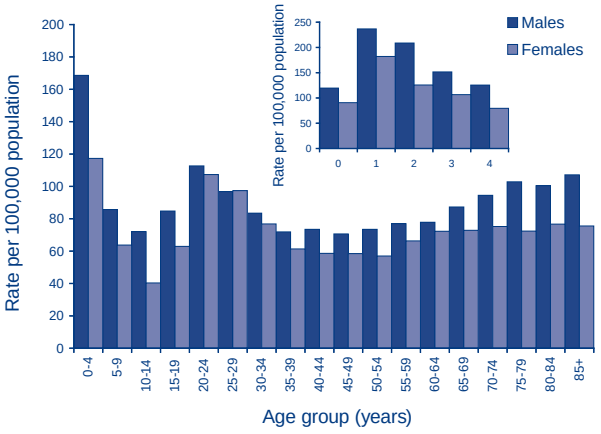
In 2007, there was 1 case of botulism, reported from Victoria. The Department of Human Services (DHS) was notified of a case of suspected botulism in a 25-year-old male. The notifying clinician gave a history of onset of dizziness, lethargy, blurred vision and respiratory distress followed by a rapid decline, which included respiratory failure requiring intubation and ventilation in an intensive care unit. A provisional diagnosis of stroke or multiple sclerosis was made but initial investigations were negative. The day following notification to DHS, the case became completely paralysed. A faecal enema specimen was forwarded to the University of Melbourne, Microbiological Diagnostic Unit for confirmation of the diagnosis. *Clostridium botulinum* toxin was detected in the faecal specimen, which was later identified as A2. An extensive investigation of a possible food source was conducted by DHS.[14]

Campylobacteriosis

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

In 2007, there were 16,984 notifications of campylobacteriosis, a 10.2% increase over the 15,407 notifications reported in 2006. The national rate of campylobacteriosis notifications in 2007 was 120.2 cases per 100,000 population, with the highest age and sex specific notification rates amongst males and females aged 0–4 years[14] (Figure 14). Amongst children aged under 5 years, the highest notification rates were in boys aged 1 year (236.8 notifications per 100,000 population) (Figure 14, inset). Prevention measures should be targeted towards more regular cleaning of hands and dummies of young children, particularly when contact with animals and outdoor environments has taken place, as a recent study conducted by OzFoodNet has shown that these are risk factor for *Campylobacter* infection in children aged 0–4 years.[15]

Figure 14: Notification rate for campylobacteriosis, Australia, 2007, by age group and sex, and inset: age and sex in children aged under 5 years



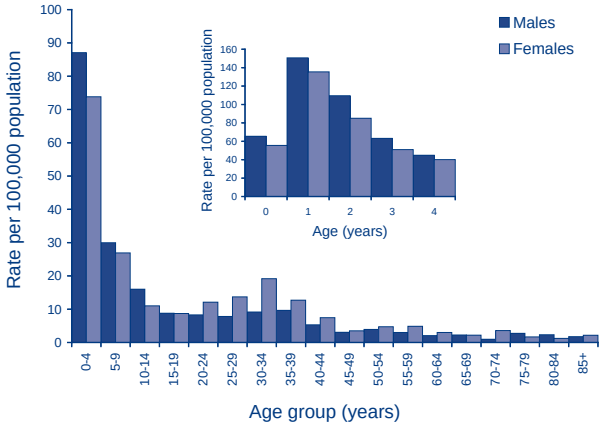
Cryptosporidiosis

In 2007, 2,810 notifications of cryptosporidiosis were reported to NNDSS representing a national rate of 13.4 cases per 100,000 population. This represents a 12% decrease over the number of notifications reported in 2006.

The highest rates of cryptosporidiosis were reported in the Northern Territory (51.6 cases per 100,000 population), Western Australia (28.9 cases per 100,000 population) and South Australia (28.3 cases per 100,000 population).

The majority of cryptosporidiosis cases in 2007 were in children aged under 10 years (52%). The highest age and sex specific notification rate was in boys aged 1 year, with 150.7 cases per 100,000 population (Figure 15).

Figure 15: Notification rate for cryptosporidiosis, Australia, 2007, by age group and sex, and inset: age and sex in children aged under 5 years



Haemolytic uraemic syndrome

During 2007, there were 19 cases of haemolytic uraemic syndrome; a rate of 0.1 cases per 100,000 population, the same as the mean of 0.1 cases per 100,000 population between 2002 and 2006. The majority of these were reported from New South Wales (n=13). The median age of notifications was 6 years, with a range of 1–44 years. Similar to previous years, the highest notification rate was in children aged 0–4 years, with eight of the 19 notifications in this age group (0.6 notifications per 100,000 population).[14]

Hepatitis A

The marked decline in notifications of hepatitis A in recent years is continuing (Figure 16).[13] In 2007, there were 165 cases of hepatitis A, compared with a mean of 349 cases per year between 2002 and 2006. This decline is likely to be due to increased uptake of vaccine amongst high risk groups such as travellers, and targeted vaccination programs for Indigenous children.[13] The proportion of cases who are known to be Indigenous is also decreasing. Between 2002 and 2006, an average of 11% of cases (39/349 cases per year) were Indigenous, while in 2007, no cases were known to have been Indigenous, with indigenous status known for 82% of cases (Table 11).

Figure 16: Trends in notifications of hepatitis A, Australia, 1991 to 2007, by month of diagnosis[13]

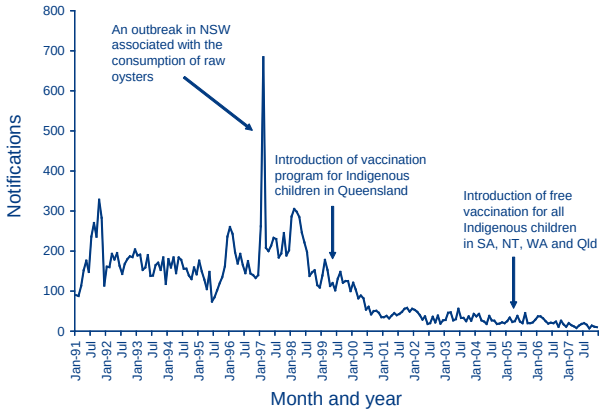


Table 11: Hepatitis A notifications, Australia, 2002 to 2007, by indigenous status

Year	Indigenous		Non-Indigenous		Unknown	
	n	%	n	%	n	%
2002	32	8	270	69	88	23
2003	52	12	322	75	56	13
2004	37	12	251	79	31	10
2005	48	15	232	71	46	14
2006	28	10	218	78	35	12
2007	0	0	136	82	29	18

In 2007, the majority of hepatitis A cases were acquired overseas (60%, 99/165), with Indonesia (16 cases) and India (14 cases) the most frequently reported place of acquisition for overseas acquired cases (Table 12).

Table 12: Notifications of hepatitis A, Australia, 2007, by state or territory

State	Number of cases	Number acquired overseas	Per cent overseas acquired
Australian Capital Territory	2	2	100
New South Wales	65	42	65
Northern Territory	5	4	80
Queensland	28	12	43
South Australia	5	4	80
Tasmania	3	0	0
Victoria	36	26	72
Western Australia	21	9	43
Total	165	99	60

Hepatitis E

In 2007, there 18 notifications of hepatitis E, compared with 24 notifications in 2006 and an average of 21 cases per year between 2002 and 2006. One case was reported from the Australian Capital Territory, eight from New South Wales, three from Queensland and six from Victoria.

In 2007, 89% (16/18) of cases were known to have been acquired overseas, of which 22% (4/18) were female. The median age of cases was 30 years (range 18–57 years), reflecting high rates of overseas travel in younger adults.

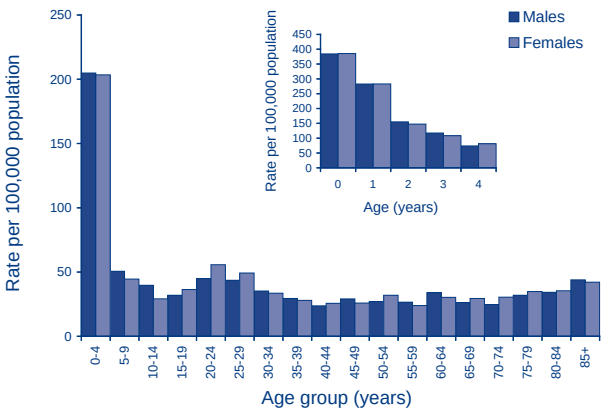
Listeriosis

In 2007, 50 cases of *Listeria monocytogenes* infection were reported to NNDSS, a crude rate of 0.2 per 100,000 population. The 2007 notification rate was similar to the 5-year historical mean (0.3 cases per 100,000 population). Seventy-six per cent (38/50) of notifications were in people aged 60 years or over. The highest age specific notification rate was in the 80–84 years age group, with a notification rate of 2.9 cases per 100,000 population. In 2007, 52% of cases were female. Four of the 50 cases were pregnancy-associated, occurring either in infants or pregnant women.[14]

Salmonellosis (non-typhoidal)

In 2007, there were 9,484 cases of *Salmonella* infection, a rate of 45 cases per 100,000 population, which is a 15% increase over the mean of the previous 5 years. Notification rates ranged from 32 cases per 100,000 population in the Australian Capital Territory to 244 cases per 100,000 population in the Northern Territory. The highest age specific rate of *Salmonella* infection was in children in the 0–4 years age group (202 cases per 100,000 population),[14] with 28% of all cases in this age group. Figure 17 shows

Figure 17: Notification rate for *Salmonella* infection, Australia, 2007, by age and sex



that in this age group, the highest rates were in those aged under 1 year (384 per 100,000 population for males and 385 per 100,000 population for females).

In 2007, the most commonly notified *Salmonella* serotype was *S.* Typhimurium. The most commonly notified phage type was *S.* Typhimurium 135, with 722 notifications in 2007. *S.* Typhimurium 9 was the second most common phage type notified in Australia in 2007. Western Australia ceased routine phage typing of *S.* Typhimurium, *S.* Enteritidis and *S.* Virchow in July 2007.[14]

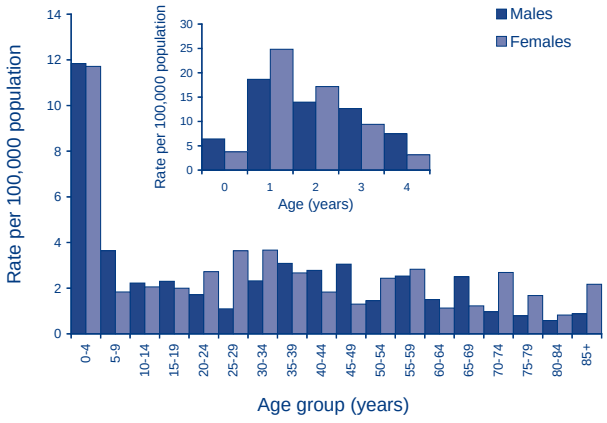
Risk factors for salmonellosis in children aged 0–4 years are currently under investigation through OzFoodNet. Most salmonellosis in Australia is transmitted through contaminated food.

Shigellosis

In 2007, there were 597 cases of shigellosis reported to NNDSS compared with 543 in 2006. The 2007 notification rate was 2.8 cases per 100,000 population compared with a mean of 2.7 cases per 100,000 population between 2002 and 2006. As in previous years, the highest notification rate was in the Northern Territory, with 80.5 cases per 100,000 population.[14]

The highest age specific notification rates were amongst males and females in the 0–4 years age group, with age specific rates of 11.8 and 11.7 notifications per 100,000 population (Figure 18). In 2007, 50% (301/597) of cases were female.

Figure 18: Notification rate for shigellosis, Australia, 2007, by age and sex



The highest burden of shigellosis continues to be in Indigenous populations. Indigenous people make up 2% of the Australian population,[16] however, 45% (269/596) of all shigellosis cases in 2007 were known to be Indigenous (indigenous status was known for 77% of cases). In the Northern Territory, 84% (146/173) of shigellosis cases were Indigenous (indigenous status was known for 97% of cases in the Northern Territory) and in South Australia 48% (30/62) were Indigenous (indigenous status was known for 79% of cases in South Australia).

The most common biotypes in 2007 were *Shigella sonnei* biotype a (21%) and *Shigella sonnei* biotype g (16%). In 2007, these 2 biotypes increased in number and proportion of notified cases compared with 2006.[14] In 2006, the most common biotype was *Shigella flexneri* 4a mannitol negative.[14]

Faecal-oral transmission is known to be a common source of infection for shigellosis.[17] Foodborne outbreaks of shigellosis are rare, and in 2007 there was only 1 foodborne outbreak of shigellosis, affecting 55 people. This outbreak was associated with imported fresh produce.[14, 18]

Shiga toxin-producing *Escherichia coli*

In 2007, there were 107 cases of STEC, a crude rate of 0.5 notifications per 100,000 population and an increase of 65% compared with an annual mean of 0.3 notifications per 100,000 population per year between 2002 and 2006.[14]

STEC rates were highest in South Australia (2.2 cases per 100,000 population) and the Northern Territory (1.4 cases per 100,000 population). South Australia reported 38% (41/107) of all STEC notifications, followed by Queensland (22%, 24/107), New South Wales (22%, 23/107), Victoria (12%, 13/107), the Northern Territory (2.8%, 3/107), Western Australia (2%, 2/107) and the Australian Capital Territory (1%, 1/107).

Jurisdictions use different methods in their screening of stools for STEC diagnosis, which can affect notification rates. As in previous years, in 2007 South Australia routinely tested all bloody stools by polymerase chain reaction (PCR) for genes coding for Shiga toxin. Queensland conducts routine culture on bloody stools. If there is no growth in culture, PCR is not performed, instead, ELISA for Shiga toxin is conducted on the specimen. Other jurisdictions do not routinely screen for STEC.

The highest age specific notification rate for STEC was amongst children in the 0–4 years age group (1.5 cases per 100,000 population), with peaks in older ages as well, with 1.0 cases per 100,000 population amongst the 60–65 years age group and 0.8 notifications per 100,000 population amongst the 70–74 years age group.[14]

Typhoid

There were 90 cases of *Salmonella* Typhi infection (typhoid) during 2007 compared with a mean of 65 cases per year between 2002 and 2006. Overseas travel is a significant risk factor for typhoid infection in Australia; in 2007, 92% (83/90) of cases reported overseas travel (Table 13).

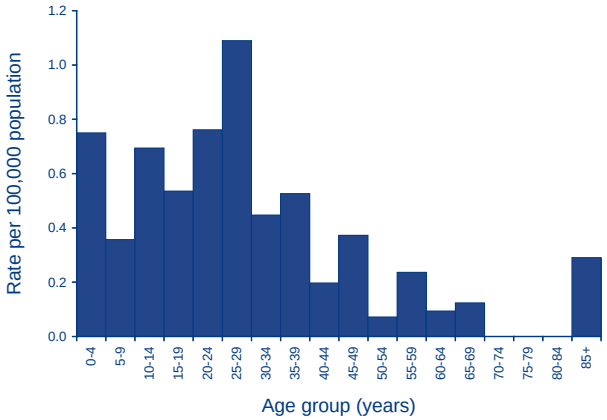
Table 13: Travel status for notified cases of typhoid, Australia, 2007

State or territory	History of overseas travel			Total
	Yes	No	Unknown	
Australian Capital Territory	0	0	0	0
New South Wales	32	1	1	34
Northern Territory	2	1	0	3
Queensland	4	1	1	6
South Australia	5	0	0	5
Tasmania	3	0	0	3
Victoria	30	0	0	30
Western Australia	7	2	0	9
Total	83	5	2	90

More than half of all overseas-acquired cases reporting overseas travel had travelled to India (51%, 42/83), with Bangladesh the second most frequently reported country or region with 13% (11/83) of cases. The predominant phage types isolated from cases returning from travel to India were E1 (19 cases) and E9 (9 cases). Similarly in cases returning from travel to Bangladesh, the most common infecting phage type was E9 (4 cases).

The highest typhoid notification rates were in the 20–24 years age group, with 0.8 cases per 100,000 population and in the 25–29 years age group with 1.1 cases per 100,000 population (Figure 19), compared with the overall notification rate of 0.4 cases per 100,000 population. This is likely to be due to high rates of overseas travel in these age groups.

Figure 19: Notifications of typhoid, Australia, 2007, by age group



Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and notifiable in Australia and to the WHO in 2007 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 at the Australian Government Department of Health and Ageing's web site: http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm, and the Australian Government's travel advisory and consular assistance service 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 2007. Table 14 provides information on the historical occurrence of these diseases in Australia.

Cholera

In 2007, there were 3 cases cholera notified in Australia, two from New South Wales and one from Queensland. All of them were acquired in India.

All cases of cholera reported since the commencement of the NNDSS in 1991 have been acquired outside Australia except 1 case of laboratory-acquired cholera in 1996 and 3 cases in 2006. There have been 20 cases of cholera notified between 2002 and 2007.[19]

Sexually transmissible infections

In 2007, the sexually transmissible infections (STIs) reported to NNDSS were chlamydial infection, donovanosis, gonococcal infection and syphilis.

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

Table 14: Australia's status for human quarantinable diseases, 2007

Disease	Status	Date of last record and notes
Cholera	Free	Small number of cases are reported annually[19]
Plague	Free	Last case recorded in Australia in 1923[20]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990[21]
Smallpox	Free	Last case recorded in Australia in 1938[22]
Yellow fever	Free	No cases recorded on shore in Australia – 5 occasions in which vessels arrived in Australian ports 1892–1915[20]
Severe acute respiratory syndrome	Free	Last case recorded in Australia in 2003[23]
Human pathogenic avian influenza in humans	Free	No cases recorded[24]
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded[25]
Marburg	Free	No cases recorded[25]
Lassa	Free	No cases recorded[25]
Crimean–Congo	Free	No cases recorded[25]

unknown duration. Reports were also received by NNDSS on 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.

Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance Programme (AGSP); a network of specialist laboratories monitoring the laboratory based indices of infections; and NCHECR.

The national trends in the number and rates of STI notifications reported to the NNDSS between 2002 and 2007 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,[26, 27] 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.

Indirect age standardised notification rates, using the method described by the Australian Institute of Health and Welfare,[28] were calculated for Indigenous and non-Indigenous populations for jurisdictions that had indigenous status data completeness in more than 50% of notifications. Incomplete notifications were counted as non-Indigenous cases when analysing these jurisdictions. These data however, need to be interpreted cautiously as STI screening occurs disproportionately among Indigenous populations and high rates in Indigenous populations may be attributed to poorer access to primary health care services and not necessarily associated with increased levels of sexual activity among Indigenous persons.[29,30] Similarly, rates between females and males need to be interpreted with caution as rates of testing for STIs differs between the sexes.

Cases were excluded for chlamydial, gonococcal and non-congenital syphilis infections in cases aged less than 15 years where mode of transmission was available 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 2007. A total of 51,859 notifications of chlamydial infection were received; a notification rate of 246.8 cases per 100,000 population. This represents an increase of 8% on the rate reported in 2006 (229.2 cases 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 2002 and 2007, chlamydial infection notification rates increased from 124.5 to 246.8 cases per 100,000 population, an increase of 97% (Table 7). This ongoing increase provided the impetus for the launch of Australia's first National STI Strategy in July 2005.[31] While the prevalence of chalmydial infection varies by age group and other demographic and behavioural factors, no major section of the population is spared.[32]

Chlamydial infection notification rates were higher than the national average (246.8 cases per 100,000 population) in the Northern Territory (1,014 cases per 100,000 population), Western Australia (367.7 cases per 100,000 population),

Queensland (307.9 cases per 100,000 population) and the Australian Capital Territory (266.4 cases per 100,000 population) (Table 3).

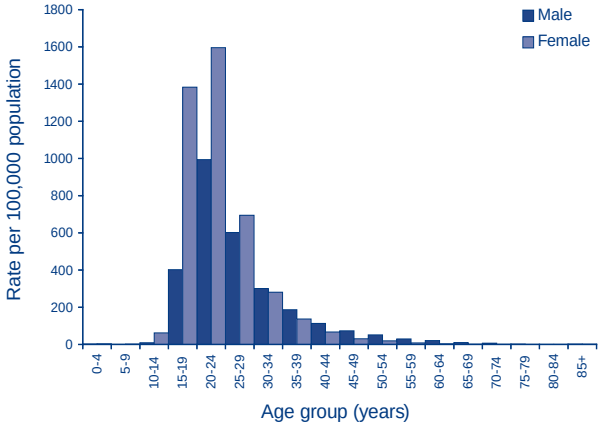
In 2007, sex was reported for 51,747 (99.8%) of the 51,859 cases of chlamydial infection. Of these cases, notification rates in males and females were 199.1 and 292.8 cases per 100,000 population respectively. In 2007, notification rates increased by 8% in both males and females when compared with 2006. The male to female ratio in 2007 was 0.7:1, which is similar to previous years. Rates in females exceeded those in males in the 0–29 years age range, but were higher in males in the 30 years or more age range (Figure 20).

Trends in age and sex notification rates between 2002 and 2007 show increases in all age ranges, especially between 15 and 29 years in both males and females (Figure 21). Between 2002 and 2007, the notification rate in males in the 20–24 years age group increased by 531.5 cases per 100,000 population (115%); and for female cases, in the 15–19 years and 20–24 years age groups, the notification rate increased by 704.8 and 802.7 cases per 100,000 population or 104% and 101%, respectively.

In 2007, data on indigenous status were complete in 43% of cases of chlamydia infection, which was the same as the preceding 5–year average of 43% (range: 40%–44%).

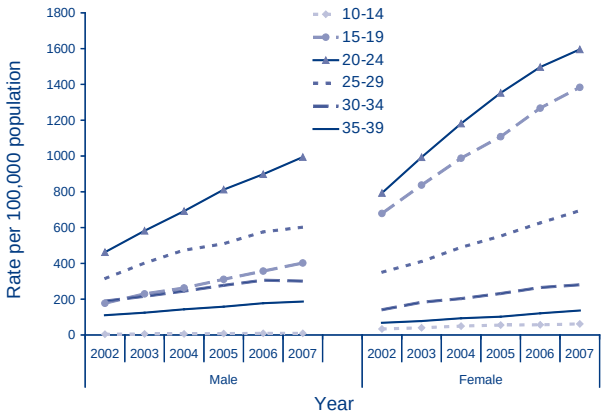
From 2002–2007 the rates of chlamydial infection diagnosis increased in both Indigenous and non-Indigenous populations as part of the overall increasing trend. In 2007, 5 jurisdictions had greater than 50% completeness of the indigenous status field: the Northern Territory, South Australia, Victoria, Tasmania and Western Australia. Among these jurisdictions, the age adjusted notification rate for the Indigenous population ranged from 64.5 to 1,782.2 cases per 100,000 population (Tasmania and the Northern Territory, respectively) with a median of 641.1 cases per 100,000 population. In comparison, for the non-Indigenous population, the age standardised notification rate ranged from 206.8 to 600.6 cases per 100,000 population (South Australian and the Northern Territory respectively) with a median of 237.9 cases per 100,000 population. During 2007, the age standardised ratio of Indigenous to non-Indigenous chlamydial infection notifications ranged between 0.27:1 and 3.5:1 (Tasmania and Western Australia respectively), median 3.0:1 (South Australia). This notification gap has improved substantially since 2000. It should be noted that indigenous status identification in the notification data is inconsistent and varies by jurisdiction. Research into high rates of STIs among the Indigenous population in the Northern Territory established that the disparity in notifications rates could be attributed

Figure 20: Notification rate for chlamydial infection, Australia, 2007, by age group and sex*



* Excludes 112 cases whose sex was not reported.

Figure 21: Trends in notification rates of chlamydial infection in persons aged 10–39 years, Australia, 2002 to 2007, by age group and sex



to more targeted screening programs and to poorer access to primary health care services, rather than increased levels of sexual activity among Indigenous people.[29,30]

Donovanosis

Donovanosis is a sexually transmissible infection characterised by a chronic ulcerative genital disease. Although uncommon, it is a disease of public health importance in Australia because it predominantly occurs in Indigenous communities and has been identified as a potential co-factor in HIV transmission. Donovanosis has been targeted for elimination from Australia through the National Donovanosis Elimination Project.[33] In 2007, 3 cases of donovanosis (2 male and 1 female) were reported to the NNDSS. Cases were reported from Queensland (2) and the Northern Territory (1) and

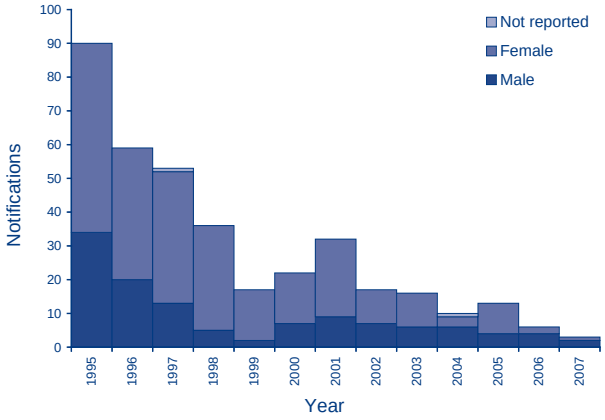
were aged 24, 41 and 58 years. Two of the cases were reported as Indigenous. In 2006, a total of 6 cases, 4 male and 2 female, with four of the cases reported as Indigenous, were notified (Figure 22).

Gonococcal infections

In 2007, 7,605 notifications of gonococcal infection were received by the NNDSS. This represents a notification rate of 36.2 cases per 100,000 population, a decrease of 12% from the rate reported in 2006 (41.4 cases per 100,000 population). The male to female ratio in 2007 was 1.9:1, similar to the previous 5 years (2002 to 2006).

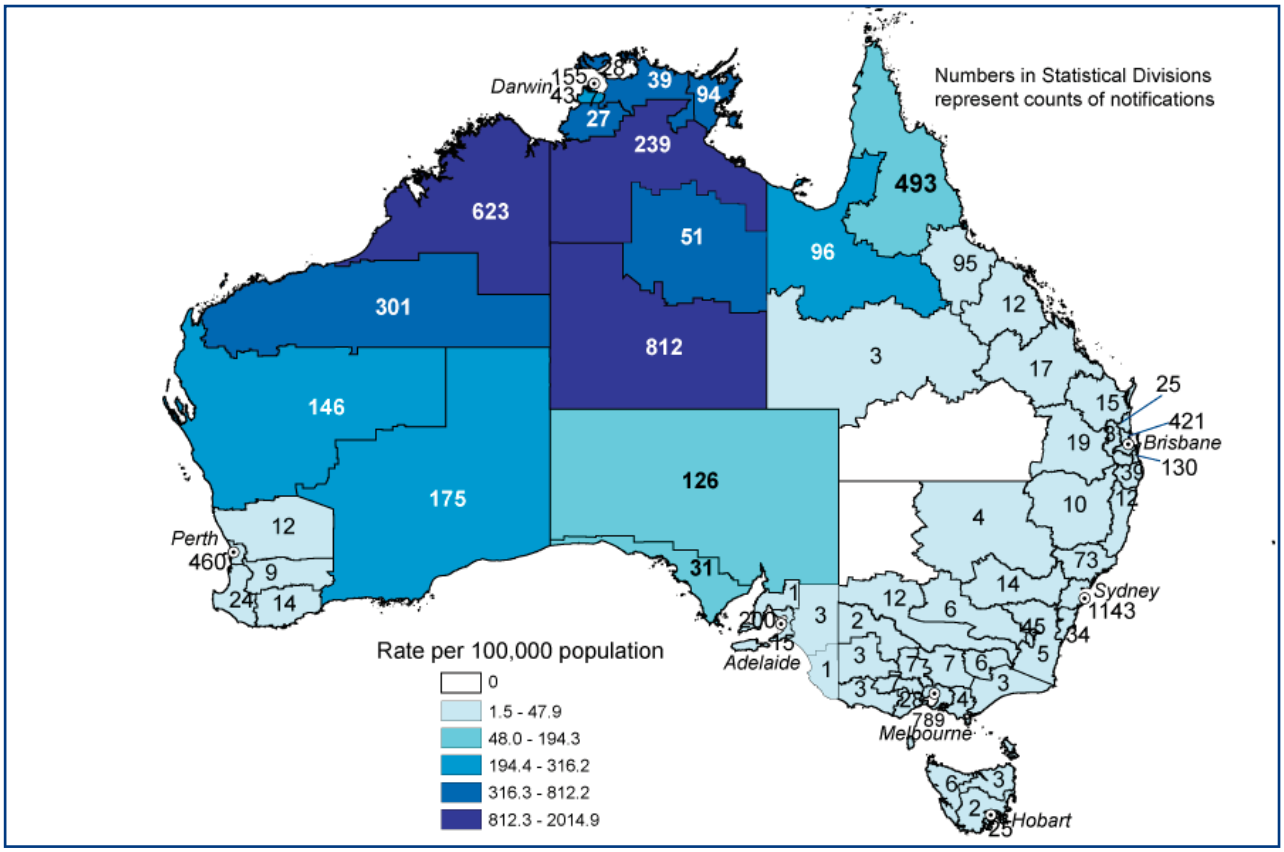
The highest notification rate in 2007 was in the Northern Territory at 744.4 cases per 100,000 population, compared with Western Australia, Queensland and South Australia (83.6, 32.0 and 28.8 cases per 100,000 population respectively) (Table 6). Considerable declines in the notification rate in 2007 compared with 2006, occurred in Victoria (24.9%), New South Wales (21.2%) and Queensland (15.2%). Notification rates in the Australian Capital Territory and Tasmania increased substantially compared with 2006 (34.1% and 109.6% respectively). At a regional level, gonococcal infection rates were highest in the Kimberley Statistical Division of Western Australia, and in the Central and Lower Top End

Figure 22: Notifications of donovanosis, Australia, 1995 to 2007, by sex



Statistical Subdivisions of the Northern Territory (range: 812.3–2,014.9 cases per 100,000 population). In the Pilbara Statistical Division of Western Australia and the Barkly, Daly, Alligator and East Arnhem Statistical Subdivisions of the Northern Territory rates were also substantially higher than the national rate (316.3–812.2 cases per 100,000 population) (Map 4).

Map 4: Notification rates for gonococcal infection, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



The sex of cases was reported in 7,599 of 7,605 cases in 2007. Nationally, gonococcal infection rates for males and females were 47.9 and 24.5 cases per 100,000 population, respectively. The exception to this national pattern was the Northern Territory, where females had an overall higher notification rate than males (804.9 versus 688.4 cases per 100,000 population). Nationally, notification rates for gonococcal infection in males exceeded those in females in all age groups except in the 10–14 and 15–19 years age groups (Figure 23).

Trends in sex specific notification rates show that in 2007 there has been a decrease in the rates in males in the 20–44 years age range compared with the general upward trend seen in previous years. In females, trends for all age groups appeared to remain relatively stable with a slight decrease occurring in the 15–19 and 20–24 years age groups (Figure 24).

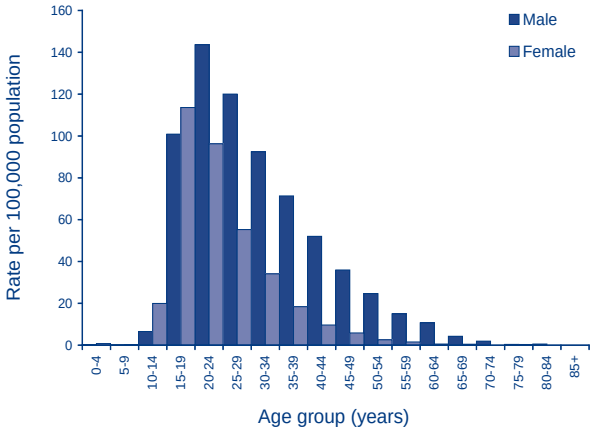
In 2007, the data completeness of indigenous status of gonococcal infection notifications was 70%, which is similar to previous years. In 2007, 6 jurisdictions had greater than 50% completeness of the indigenous status field: the Northern Territory, Queensland, South Australia, Tasmania, Victoria and Western Australia. Among these jurisdictions the age standardised notification rates for gonococcal infection in the Indigenous population ranged from 10.0 to 1,923.1 cases per 100,000 population (Victoria and the Northern Territory, respectively) with a median of 445.4 cases per 100,000 population. Whereas age standardised notification rates in the non-Indigenous population ranged from 7.4 to 131.9 cases per 100,000 population (Tasmania and the Northern Territory respectively) with a median of 19.5 cases per 100,000 population. During 2007, the age standardised ratio of Indigenous to non-Indigenous gonococcal infection notifications ranged between 0.5:1 and 63.4:1 (Victoria and Western Australia respectively), median 13.8:1.

Other surveillance of gonococcal infections

The Australian Gonococcal Surveillance Programme is the national surveillance system for monitoring antimicrobial resistance of *Neisseria gonorrhoeae* isolates. The monitoring is undertaken via a network of reference laboratories located in each jurisdiction to determine the susceptibility of gonococcal isolates, from both the public and private sectors, to a core group of antibiotics using a standard methodology. The core group of antibiotics are penicillin, ceftriaxone, spectinomycin, quinolone and tetracycline. The following is a summary of the AGSP 2007 report.[34]

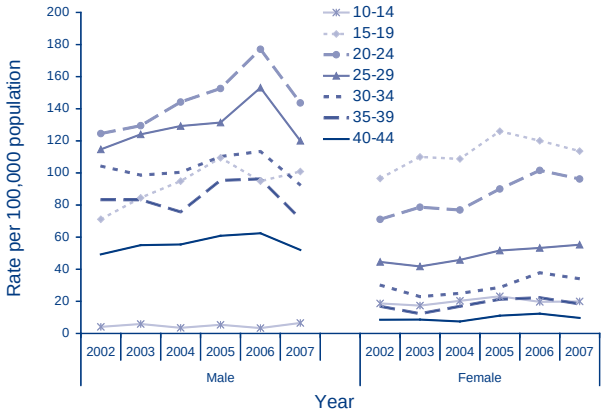
In 2007, a total of 3,103 gonococcal isolates were tested for antibiotic susceptibility, approximately 20% fewer than the 3,937 examined in 2006. The decline in the number of gonococcal isolates available for susceptibility testing is noted as a consequence of the increasing use of non-culture based diagnosis methods.

Figure 23: Notification rate for gonococcal infections, Australia, 2007, by age group and sex*



* Excludes 7 cases whose sex was not reported.

Figure 24: Trends in notification rates of gonococcal infection in persons aged 10–44 years , Australia, 2002 to 2007, by age group and sex



There were 2,560 isolates from males, 541 isolates from females (male to female ratio 4.7:1) and there were 2 isolates where the sex was not reported. In males, 75% of isolates were obtained from the urethra, 14% from the rectum and 9% from the pharynx. In females, the majority of isolates (90%) were obtained from the cervix.

Data on the place of acquisition were available for 23% (n=96) of isolates with 'penicillinase-producing' *N. gonorrhoeae* (PPNG) and for 33% (n=495) of isolates with quinolone resistance to *N. gonorrhoeae* (QRNG). This showed that half of the infections with PPNG (48/96, 50%) were acquired overseas,

principally from Western Pacific or South East Asian countries. Eighty-four per cent of QRNG (422/495) infections were locally acquired with the remainder from overseas sources similar to PPNG.

Trends in the proportion of isolates resistant to penicillin and quinolone were 40% and 50%, respectively, of all isolates and similar to previous years. There was also a historically high rate of gonococcal isolates with high-level tetracycline resistance. As in previous years, the pattern of gonococcal antibiotic susceptibility differed between states and territories, and rural and urban areas within each jurisdiction,[35] highlighting the need to continue the monitoring of treatment regime suitability on a regional basis.

Syphilis (all categories)

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

In 2007, a total of 2,999 cases of syphilis infection of all categories was reported, representing a notification rate of 14.3 cases per 100,000 population, an increase of 10.0% on the 13.0 cases per 100,000 population reported in 2006 (Figure 25). The Northern Territory continued to have the highest notification rate for syphilis (130.7 cases per 100,000 population), compared with Victoria and New South Wales (16.3 and 16.1 cases per 100,000 population respectively). The Australian Capital Territory reported an increase in the notification rate for syphilis of 129.5% compared with 2006 (28 cases 2007; 12 cases 2006). There were also increases in notification rates in Tasmania (62.5%), Victoria (39.8%) and Western Australia (13.7%). As in other developed countries, syphilis infection rates have continued to rise in Australia among men who have sex with men.[36, 37]

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

In 2007, a total of 1,381 cases of infectious syphilis (less than 2 years duration) were reported. This represents a notification rate of 6.6 cases per 100,000 population, an increase of 56.2% compared with 2006 (4.2 cases per 100,000 population) (Table 7). The Northern Territory had the highest notification rate at 55.4 cases per 100,000 population in 2007, a decrease of 22.3% compared with 2006. The Australian Capital Territory reported a substantial increase in their notification rate from 0.6 (in 2006, 2 cases) to 2.6 (in 2007, 9 cases) cases per 100,000 population. Increases in notification rates also occurred in Western Australia (101.5%), New South Wales (93.5%), Victoria (82.9%), Tasmania (58.9%) and Queensland (35.1%) (Table 7).

At a regional level, infectious syphilis rates were highest in the Central Statistical Subdivision of the Northern Territory (191.1 cases per 100,000 population, 77 cases). In the Kimberley and South Eastern Statistical Divisions of Western Australia; and in the Barkly, Lower Top End, Daly, Alligator and East Arnhem Statistical Subdivisions of the Northern Territory, notification rates of infectious syphilis (16.6–63.7 cases per 100,000 population) were also substantially above the national rate[8] (Map 5).

The notification rates for infectious syphilis for males and females were 11.8 and 1.4 cases per 100,000 population respectively (Table 15). Nationally,

Figure 25: Notification rate for syphilis infection (all categories), Australia, 2002 to 2007

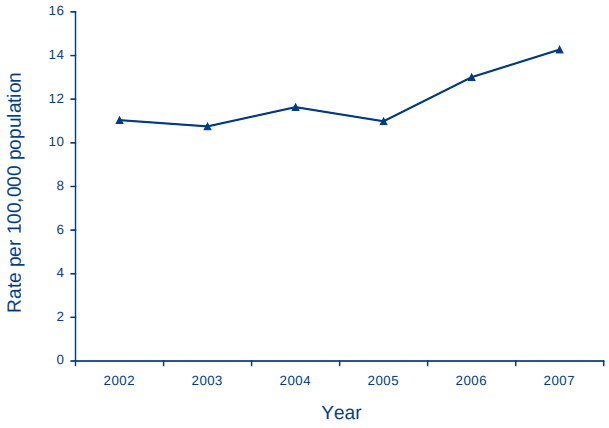
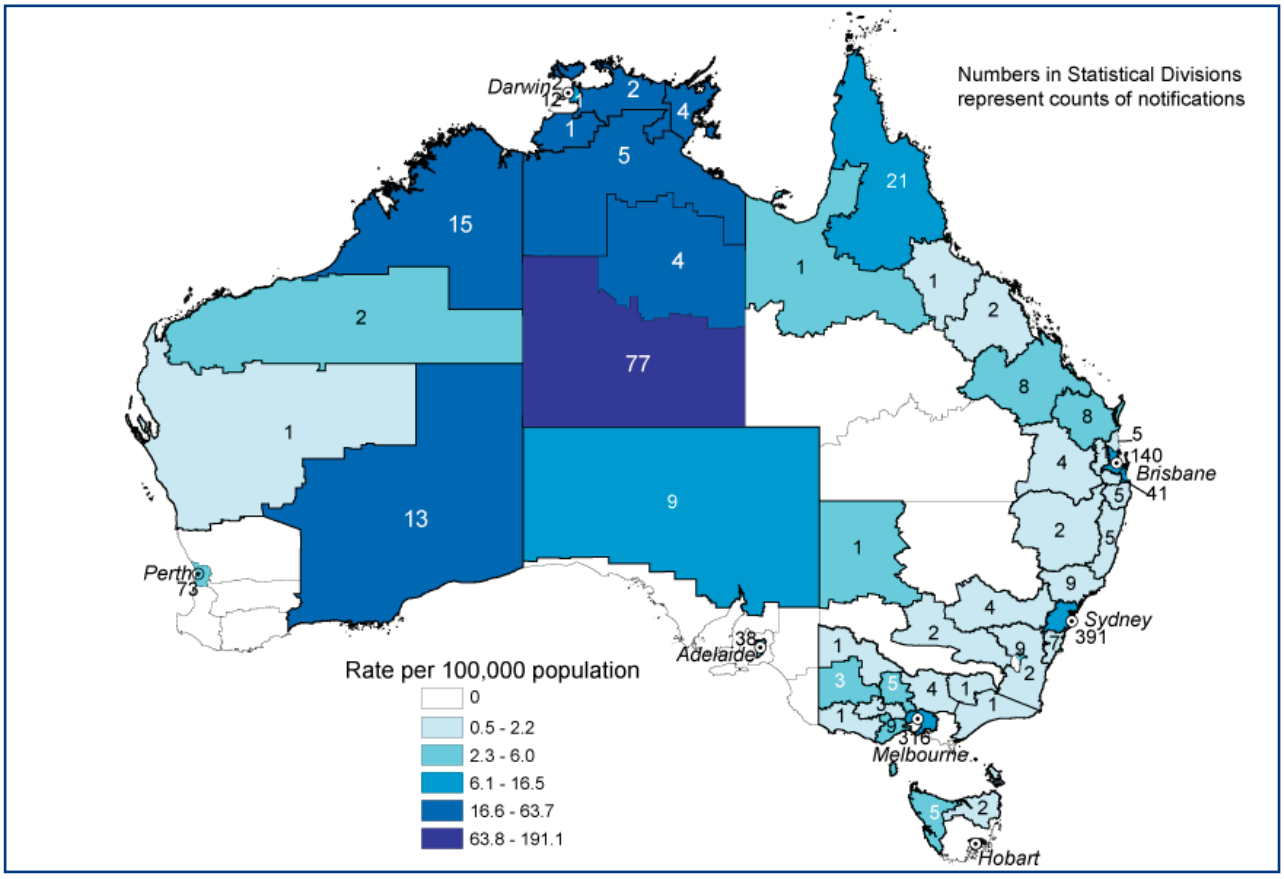


Table 15: Number and rate of notifications of infectious syphilis (less than 2 years duration), Australia, 2007, by sex and state or territory

State or territory	Male		Female		Total	
	n	Rate	n	Rate	n	Rate
ACT/ NSW	416	11.6	27	0.7	443	6.1
NT	72	64.5	47	45.5	119	55.4
Qld	204	9.8	28	1.3	232	5.5
SA	43	5.5	8	1.0	51	3.2
Tas	7	2.9	1	0.4	8	1.6
Vic	408	15.8	19	0.7	427	8.2
WA	81	7.6	20	1.9	101	1.9
Total	1,231	11.8	150	1.4	1,381	6.6

Map 5: Notification rates for infectious syphilis, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



the male to female ratio was 8.2:1. Notification rates in males peaked in the 35–39 years age group (29.6 cases per 100,000 population) and in females in the 15–19 years age group (4.1 cases per 100,000 population). Notification rates were higher in males than in females in all jurisdictions, and across all age groups, except the 10–14 years age group where the rate was slightly higher in females (0.6 cases per 100,000 population) than in males (0.4 cases per 100,000 population) (Figure 26).

Over the period 2004 to 2007 notification rates have increased substantially in most age groups for males. Increases ranged between 59%–126% in the 20–50 years or over age groups in males compared with 2006. In females, rates remained steady except in the 10–14 and 15–19 years age groups where they decreased by 66.6% and 53.2%, respectively, compared with 2006 (Figure 27). Increases in notifications of infectious syphilis occurred mainly in populations of men who have sex with men.[4]

Figure 26: Notification rate for infectious syphilis (less than 2 years duration), Australia, 2007, by age group and sex

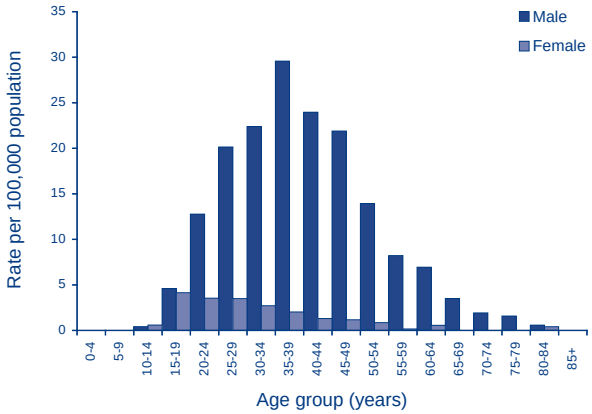
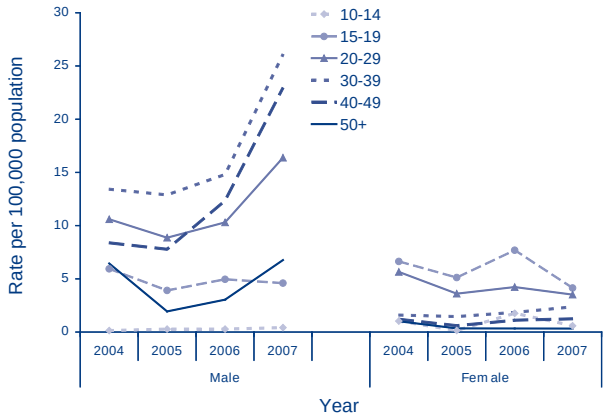


Figure 27: Trends in notification rates of infectious syphilis (less than 2 years duration) in persons aged 10 years or over, Australia, 2004 to 2007, by age group and sex



Data on indigenous status were complete in 94% of cases of infectious syphilis. In 2007, all jurisdictions except the Australian Capital Territory had greater than 50% completeness of the indigenous status field. Across these jurisdictions, the age standardised notification rate was 30.7 cases per 100,000 Indigenous population and 5.8 cases per 100,000 non-Indigenous population. These age adjusted notification rates ranged substantially across jurisdictions. For the Indigenous population the age standardised notification rate ranged from 0.0 to 146.0 cases per 100,000 population (Tasmania and the Northern Territory respectively). Whereas in the non-Indigenous population, the age standardised notification rate ranged from 1.7 to 9.9 cases per 100,000 population (Tasmania and the Northern Territory respectively). Across these jurisdictions the ratio of Indigenous to non-Indigenous age standardised rates were 5.3:1. Again this ratio varied between the individual jurisdictions from 0.0:1 to 14.7:1 (Tasmania and the Northern Territory respectively). This notification gap has decreased compared with previous years. Analysis of age specific notification rates show that compared with the non-Indigenous population, rates of infectious syphilis in the Indigenous population are higher and peak in a younger age group, 15–34 years age range compared with 34–49 years age range. Caution should be applied when interpreting these figures due to the wide variation in Indigenous and non-Indigenous population distributions for each jurisdiction, the completeness of the indigenous status field, and as noted in the methods section, where there are unknown indigenous status cases these have been counted as non-Indigenous.

Syphilis of more than 2 years or unknown duration

In 2007, a total of 1,618 cases of syphilis of more than 2 years or unknown duration were reported, a notification rate of 8.3 cases per 100,000 population. This rate represents a decrease by 12.6% compared with 2006 (9.5 cases per 100,000 population). The Northern Territory again had the highest notification rate at 75.4 cases per 100,000 population in 2007, which was an increase of 32.3% compared with 2006 (57.0 cases per 100,000 population).

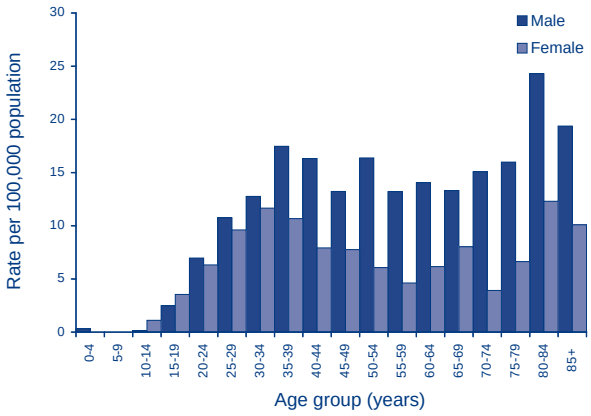
In 2007, the sex of cases was reported in 1,608 of the 1,618 cases. Notification rates for syphilis of more than 2 years or unknown duration in males and females were 10.3 and 6.2 cases per 100,000 population, respectively (Table 16). Notification rates were higher in males than in females in all jurisdictions except Queensland, where males had a slightly lower rate than females (4.9 and 5.0 cases per 100,000 population, respectively). Nationally, the male to female ratio was 1.6:1. Notification rates in males and females were similar in the younger age groups up to 34 years. In females the rate peaked in the 30–34 years age group while in males it remained high from 35 years, with a peak in the 80–84 years age group (Figure 28).

Table 16: Number and rates of notifications of syphilis of more than 2 years or unknown duration, Australia, 2007, by state or territory and sex

State or territory	Male		Female		Total*	
	n	Rate	n	Rate	n	Rate
ACT	12	7.1	7	4.1	19	5.6
NSW	451	13.2	218	6.3	672	9.8
NT	91	81.6	71	68.7	162	75.4
Qld	103	4.9	105	5.0	208	5.0
Tas	20	8.2	8	3.2	28	5.7
Vic	263	10.2	146	5.6	416	8.0
WA	58	5.5	55	5.3	113	2.2
Total	998	10.3	610	6.2	1,618	8.3

* Sex was not reported for 10 cases.

Figure 28: Notification rate for syphilis of more than 2 years or unspecified duration, Australia, 2007, by age group and sex*



* Excludes 10 cases where sex was not reported.

Over the period 2004 to 2007, notification rates increased substantially between 2005 and 2006 and then decreased to 2005 rates in 2007 for males aged over 30 years. In females, rates have remained relatively stable, except in females aged 10–14 years where the rates have increased between 2004 and 2007 from 0.5 to 1.1 cases per 100,000 population (Figure 29).

Figure 29: Notification rate for syphilis of more than 2 years or unspecified duration, Australia, 2004 to 2007, by age group and sex

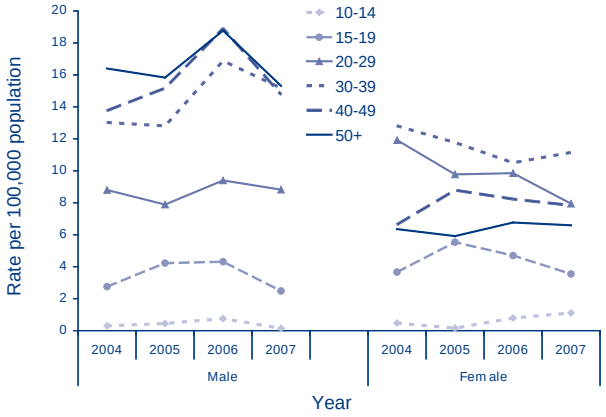
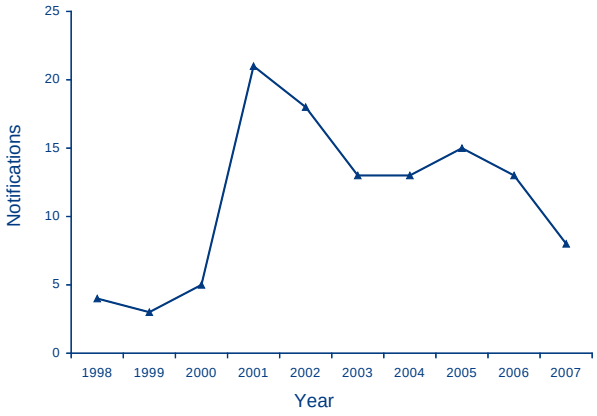


Figure 30: Trends in notifications of congenital syphilis, Australia, 1998 to 2007



Congenital syphilis

There were 8 cases of congenital syphilis notified in 2007, 5 males, 2 females and 1 case where the sex was not reported. Six of the cases were reported in New South Wales and three in the Northern Territory. Three were Indigenous, 2 non-indigenous and three were reported as unknown indigenous status. Notifications of congenital syphilis reached a plateau between 2003 and 2006 following a decline from a peak in 2001. In 2007 the number of cases decreased by 38% compared with 2006 (Figure 30).

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 2007. These include diphtheria, *Haemophilus influenzae* type b infection, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella, tetanus and varicella (chickenpox, shingles and unspecified). Data on hepatitis B and 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 under 'Gastrointestinal' and Q fever under 'Zoonoses'.

As of 1 July 2007, vaccines for human papilloma virus (HPV) and rotavirus were added to the funded NIP Schedule. In the lead up to this decision, the vaccines for HPV and rotavirus were registered by the Therapeutic Goods Administration and became available in the private market throughout Australia in 2006. In October 2006, the Northern Territory commenced a funded rotavirus immunisation program for infants born on or after 1 August 2006.

In 2007, the National HPV Vaccination Program was implemented for 12–13-year-old females, with a catch-up program for 13–26-year-old females. Currently there is no routine national surveillance system for monitoring HPV genotype infections in the general female population.

From 1 July 2007, all Australian children born on or after 1 May 2007 became eligible to receive a rotavirus vaccine. The rotavirus immunisation program aims to reduce the large social and economic burden of this disease in Australia where it is responsible for as many as 10,000 (50%) of all childhood hospital admissions for diarrhoea each year.[38] Two rotavirus vaccines are currently licensed for use in Australia: Rotarix® (GlaxoSmithKline), a monovalent vaccine containing 1 strain of live attenuated human rotavirus which protects against non-G1 serotypes; and Rotateq® (CSL Biotherapies/Merck & Co Inc), a pentavalent vaccine containing rotavirus reassortants of human serotypes G1, G2, G3, G4, and P1. Both vaccines have been demonstrated in large-scale phase 3 trials worldwide to be safe and highly effective in the prevention of severe diarrhoea and hospitalisation due to rotavirus infections. Immunisation is recommended in the routine schedule as 2 doses at 2 and 4 months of age using the Rotarix® vaccine or 3 doses at 2, 4 and 6 months using the Rotateq® vaccine.[13]

Rotavirus is currently not on the National Notifiable Disease List.[2] Although data were provided to the NNDSS by Western Australia and Queensland in 2007, it has not been analysed as part of this report. More details of rotavirus surveillance in Australia are provided in the Australian Rotavirus Surveillance Program annual report, 2007/2008.[38]

In 2007, there were 25,348 notifications of VPDs (17% of total notifications). This is 14% more than the 22,240 notifications of VPDs reported in 2006. Laboratory confirmed influenza was the most commonly notified VPD (10,403, 41% of all VPD notifications). The number of notifications and notification rates for VPDs in Australia are shown in Tables 5 and 6.

Diphtheria

There were no cases of diphtheria reported to NNDSS in 2007. The last case of diphtheria reported in Australia was a case of cutaneous diphtheria in 2001, which was the only case reported since 1992. Immunity to diphtheria measured in a national serosurvey in the late 1990s in Australia, showed high levels in people aged less than 30 years and declining immunity with increasing age.[39] As there is now little opportunity to acquire natural immunity or to boost declining immunity with subclinical infection, it is therefore important for Australians to retain high levels of immunity through high vaccination coverage. This is particularly important to protect Australians against diphtheria when travelling in the 21 countries where the disease is still prevalent.[40]

Haemophilus influenzae type b disease

There were 17 notifications of *Haemophilus influenzae* type b (Hib) disease in 2007, a rate of 0.1 cases per 100,000 population. This was 5 less cases than reported in 2006. Eleven cases (65% of total) were in children aged less than 5 years and seven were infants aged less than 1 year. There were 8 cases in males and 9 cases in females, (male to female ratio 0.9:1), unlike 2006 when the ratio was 0.5:1 (Figure 31).

Indigenous status was recorded for 16 of the 17 cases; seven were Indigenous and nine were non-Indigenous. The Hib notification rate was 1.4 cases per 100,000 in Indigenous people and 0.05 cases per 100,000 in non-Indigenous people—a ratio of 28:1. Between 2002 and 2006, Hib notification rates in Indigenous people have been between 6.2 and 18.8 times the rates in non-Indigenous people, except in 2002 when the Indigenous rate was 26 times that of the non-Indigenous rate (Figure 32).

Cases under the age of 15 years were eligible for Hib vaccination in infancy. Of the 12 cases aged less than 15 years in 2007, five were unvaccinated, one partially vaccinated for age and three were fully vaccinated for age. Vaccination status for 3 cases was unknown or not supplied. One of the fully vaccinated cases aged 3 years had received 3 validated doses of vaccine and met the case definition for vaccine failure.

Figure 31: Notifications of *Haemophilus influenzae* type b infection, Australia, 2007, by age group and sex

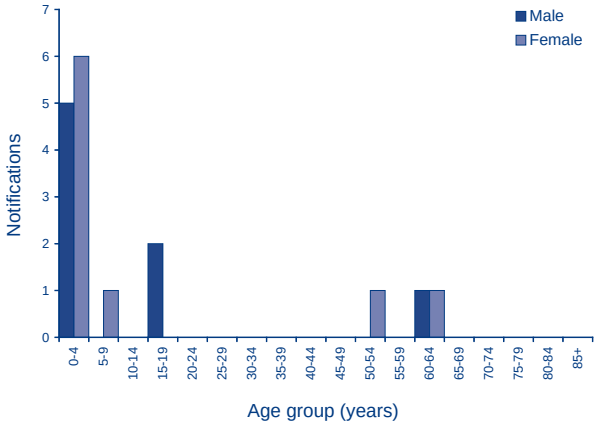
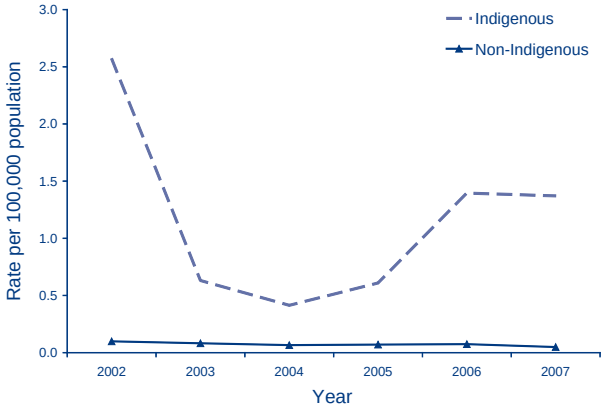


Figure 32: Notification rate for *Haemophilus influenzae* type b infection, Australia, 2002 to 2007 by indigenous status



After nearly 20 years 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 2007 influenza season was the highest season seen since influenza became nationally notifiable in 2001 (Figure 33). Notifications were 3.1 times the 5-year weekly rolling mean and peaked in August. As influenza only became nationally notifiable in 2001, a 5-year rolling mean cannot be calculated for years prior to 2006. There were 10,403 reports of laboratory-confirmed influenza in 2007, a rate of 49.5 cases per 100,000 population. Queensland notifications accounted for 44% of all influenza cases in Australia notified to NNDSS (Figure 34). Media coverage following the deaths of

Figure 33: Notifications of laboratory confirmed influenza, Australia, 2007, by week of diagnosis

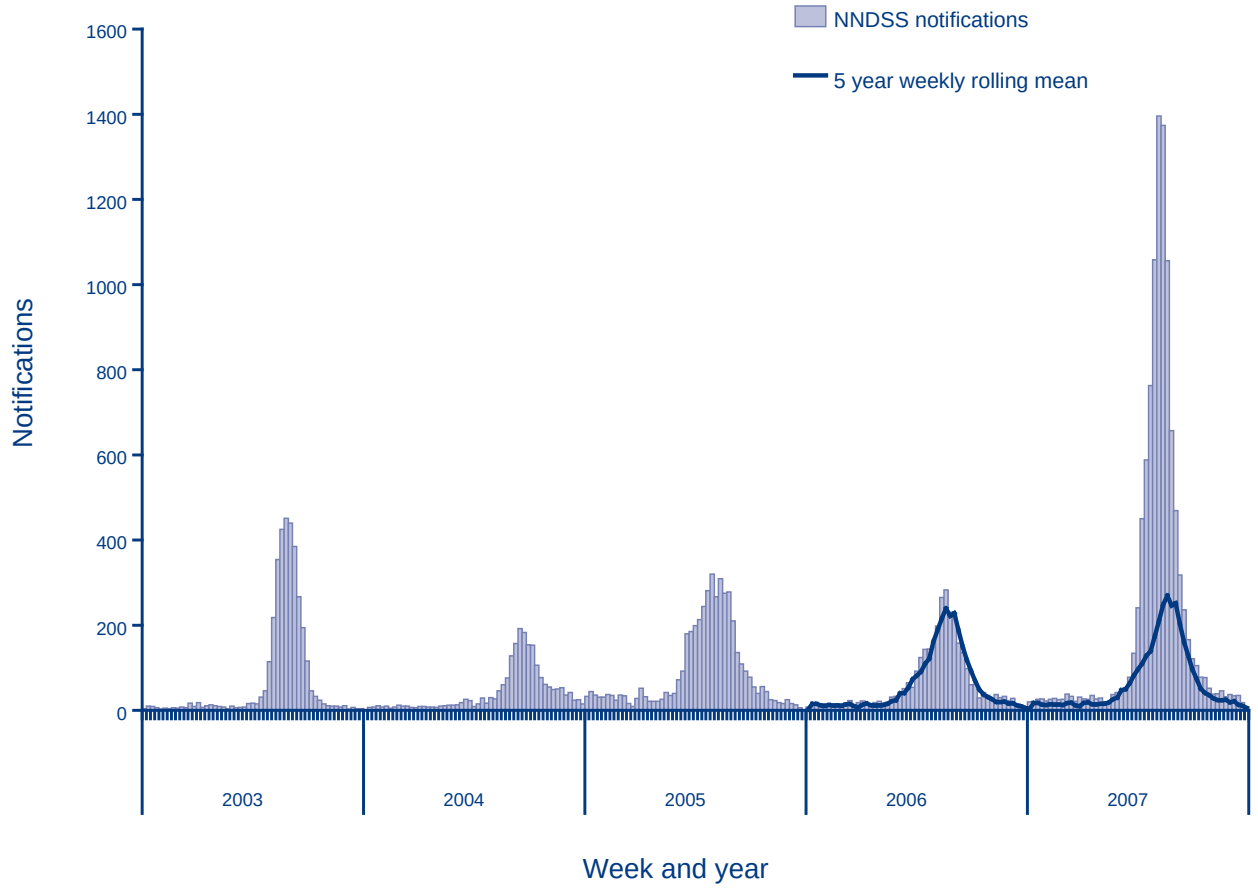
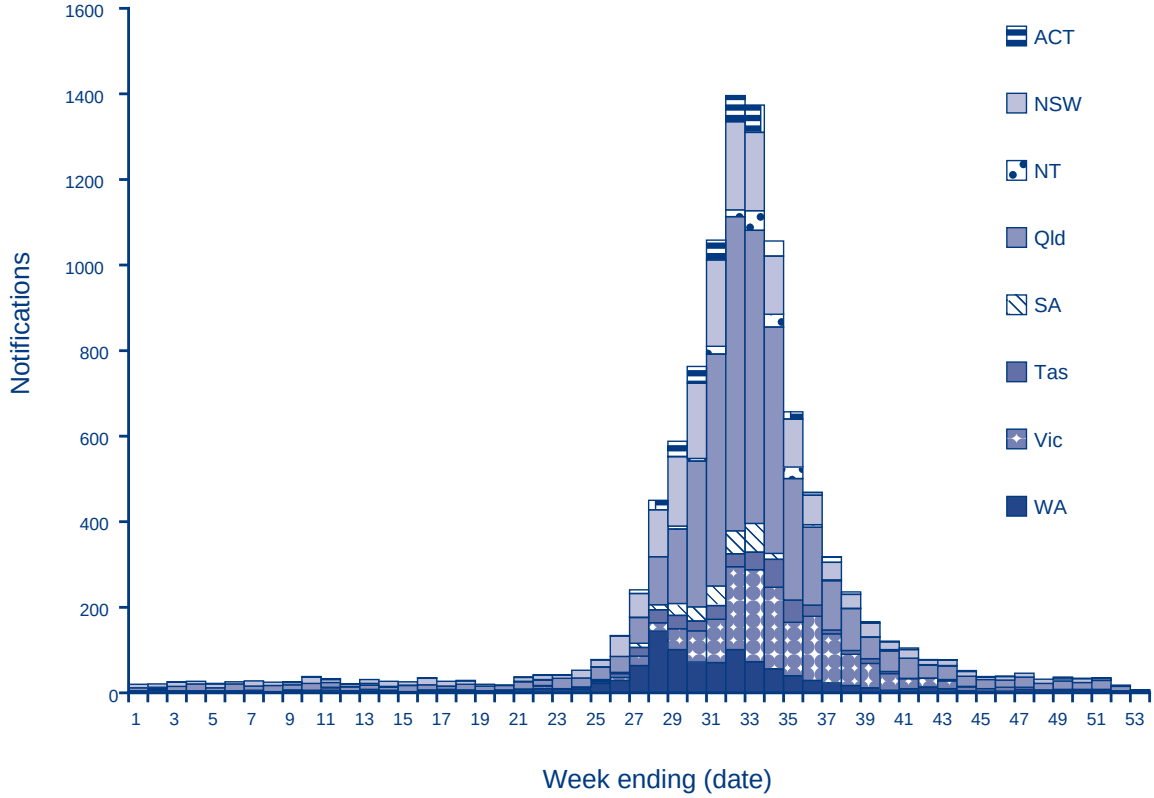


Figure 34: Notifications of laboratory confirmed influenza, Australia, 2007, by state or territory and week of diagnosis

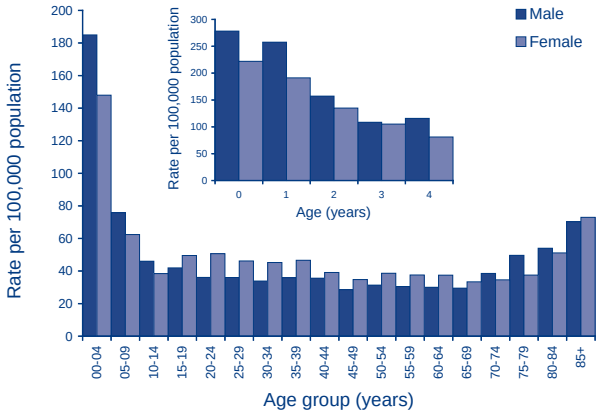


children due to influenza may have increased the rate of presentations for health care and testing for influenza in children and thus laboratory diagnosis and notification.[43]

The highest notification rates occurred in the Australian Capital Territory with 115 cases per 100,000 population, followed by Queensland (110 cases per 100,000 population), the Northern Territory (85 cases per 100,000 population) and Tasmania (84 cases per 100,000 population) (Table 6).

There were 2,240 notifications in children aged less than 5 years (22% of all notifications). As in previous years, influenza notification rates were markedly higher in children under 5 years (notification rate of 168 cases per 100,000 population) compared with older age groups (43 cases per 100,000 population) (Figure 35). The rate was highest in those under 1 year of age (271 cases per 100,000 population). The overall male to female ratio was 1:1.03.

Figure 35: Notification rate for laboratory-confirmed influenza, Australia, 2007, by age group and sex



In 2007, 9,901 (95%) influenza notifications in NNDSS had viral type data. Of cases including type data, 90% (8,942) were influenza A, 9% (914) were influenza B and 0.5% (45) were mixed infections. A breakdown of influenza notification by virus type and jurisdiction is shown in Table 17.

Of 1,406 influenza virus isolates analysed at the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) in 2007, 826 (58.7%) were A(H3N2) strains, 483 (34.4%) were A(H1N1) strains and 97 (6.9%) were influenza B. The WHOCC reported that early testing showed a difference in the proportion of H1 and H3 strains across jurisdictions. Western Australian and Victorian isolates were mainly type A(H3) while Queensland and New South Wales isolates were a mixture of type A(H1) and A(H3).[43]

Antigenic analysis of the Australian 2007 strains showed a genetic drift away from the 2007 vaccine strains for both A(H1) and A(H3).

Circulating strains were:

A(H1): A/New Caledonia/20/99-like and drift strain A/Solomon Islands/3/2006-like;

A(H3): A/Wisconsin/67/2005-like and newly emergent variant A/Brisbane/10/2007-like; and

B: B/Malaysia/2506/2004-like (21% – Victoria lineage) and B/Florida/7/2004-like (79% – Yamagata lineage).

The 2007 vaccine included:

A//New Caledonia/20/99(H1N1) – like strain.

A/Wisconsin/67/2005(H3N2) – like strain.

B/Malaysia/2506/2004 – like strain.

Table 17: Notification of laboratory confirmed influenza, Australia, 2007, by state or territory and type

Influenza type	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Influenza A	346	1,487	179	4,384	262	388	1,266	630	8,942
Influenza B	44	180	3	200	18	27	309	133	914
Influenza A&B	0	43	0	0	0	0	2	0	45
Influenza type unknown	0	208	1	6	0	0	12	275	502
Total	390	1,918	183	4,590	280	415	1,589	1,038	10,403

Invasive pneumococcal disease

There were 1,474 notifications of invasive pneumococcal disease (IPD) in Australia in 2007, a rate of 7.0 cases per 100,000 population. This was a small increase from the 1,445 cases reported in 2006 (7.0 cases per 100,000 population). An increase in notification rate between 2006 and 2007 was seen in the Australian Capital Territory (34 cases, 10.0 cases per 100,000 population), the Northern Territory (66 cases, 30.7 cases per 100,000 population) and Queensland (322 cases, 7.7 cases per 100,000 population). The lowest notification rate in 2007 was seen in Victoria (278 cases, 5.3 cases per 100,000 population).

In 2007, males accounted for 827 of the 1,474 notified cases of IPD. In all age groups there were more male than female cases, resulting in a male to female ratio of 1.3:1. Figure 36 shows that the highest rates of IPD in 2007 were among the elderly aged 85 years or over (34.3 cases per 100,000 population) and in children aged 1 year (33.4 cases per 100,000 population).

Additional data were collected on cases of invasive pneumococcal disease in all Australian jurisdictions during 2007. Details can be found in the invasive pneumococcal disease annual report published in the next edition of *CDI*.

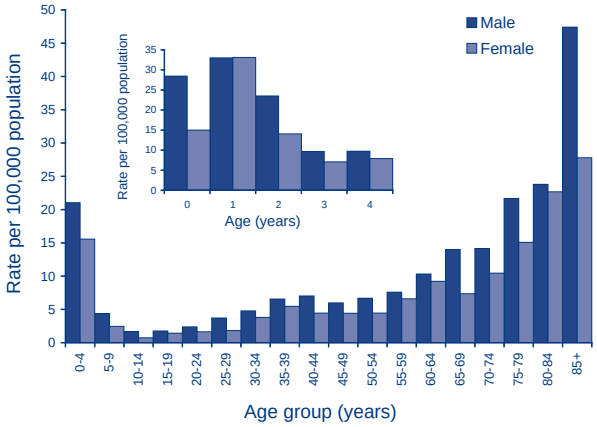
Measles

There were 12 cases of measles notified to NNDSS in 2007 representing a rate of 0.1 cases per 100,000 population. This was a significant reduction compared with the 125 cases notified in 2006 (0.6 cases per 100,000 population) associated with a large multi-state outbreak (Figure 37). Figure 38 shows that since national surveillance began in 1991, the measles annual rate for Australia has only been lower in 2005 (0.05 cases per 100,000 population).

In 2007, notifications were reported from New South Wales (4), Queensland (4), Victoria (2), South Australia (1), and Western Australia (1).

In 2007, there was a substantial decrease in the notification rate in all age groups compared with 2006 (Figure 38). There was 1 case in an 11 month-old infant, three in children aged between one and 4 years, one in the 5–14 years age group, four in the 15–24 years age group and three in the 25–34 years age group. Of the 12 cases, five (42%) were not vaccinated, one (8%) was partially vaccinated for age and the vaccination status for the remaining 6 cases (50%) was either unknown, missing or coded as not applicable. Overseas acquired measles infection accounted for seven (58%) of the 12 cases in 2007, four of which were not vaccinated, 1 case was partially vaccinated and in 2 cases the vaccination status was either unknown or not stated. The NIP recommends

Figure 36: Notification rate for invasive pneumococcal disease, Australia, 2007, by age group and sex



that children are vaccinated for measles with the combined measles, mumps, rubella vaccine (MMR), at 12 months and 4 years of age.[13] Data on serogroup type was available for 2 cases and was identified as D4 and D5 respectively. The majority of the measles cases in 2007 (8; 67%) were male.

Figure 37: Measles notifications, Australia, 2002 to 2007, by month of diagnosis

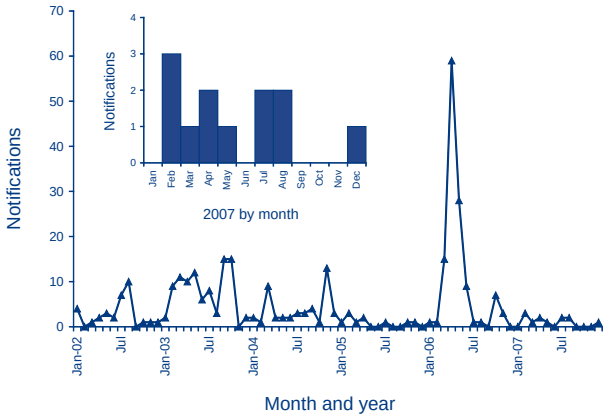
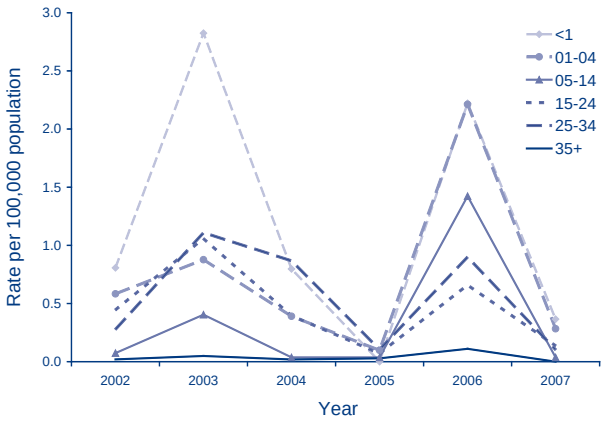


Figure 38: Trends in measles notification rates, Australia, 2002 to 2007, by age group



Mumps

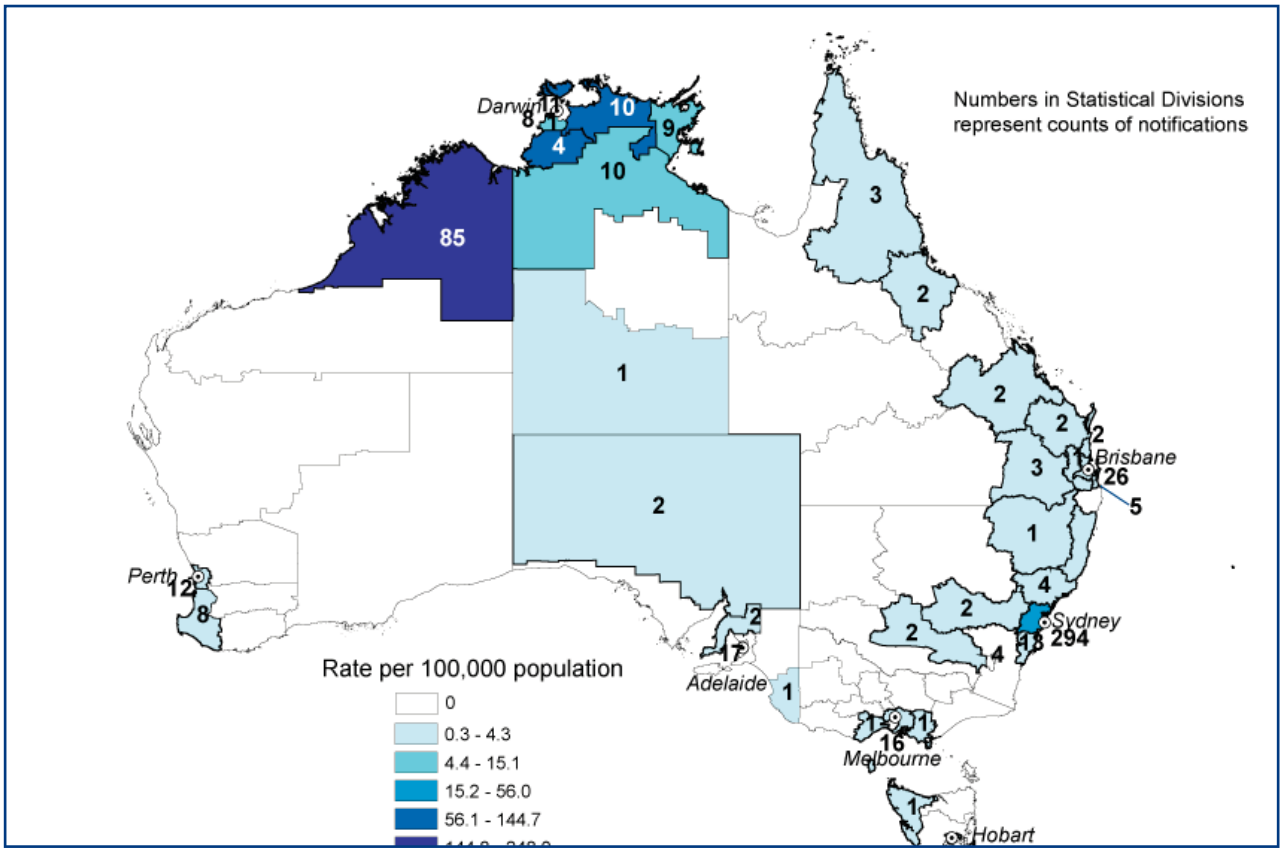
In 2007, there were 579 notifications of mumps (2.8 cases per 100,000 population), a twofold increase on the 275 notifications of mumps (1.2 cases per 100,000 population), reported in 2006 and a ratio of 3.8 compared with the 5-year mean. Cases were reported from all jurisdictions, with the majority (323) from New South Wales but also including large numbers from Western Australia (106 cases), the Northern Territory (58 cases) and Queensland (46 cases). However, the highest mumps notification rate was in the Northern Territory with 27 cases per 100,000 population, followed by Western Australia and New South Wales, each with 5 cases per 100,000 population.

Indigenous status was recorded for 396 of the 579 cases and of these, 126 (32%) were reported as Indigenous and 270 as non-Indigenous. The relatively large proportion of the total number of mumps cases in 2007 identified as Indigenous was a significant increase from the absence of Indigenous cases in 2006 and the 5-year mean of 1.2 Indigenous cases.

Of the Western Australian and Northern Territory cases in 2007, 75% (80 cases) and 78% (45 cases) respectively were identified as Indigenous and were associated with outbreaks in the Kimberley region of Western Australia and Indigenous communities in the Northern Territory (Map 6).

A clinical audit of immunisation status in a remote Indigenous community in the Northern Territory affected by the mumps outbreak identified low vaccination coverage rates in those over 20 years of age. More than 50% of mumps cases in the 10–49 year age range showed no record of vaccination (although this is based on case notes and may not have been complete for individual patients).[44] Ten of the cases reported in the Northern Territory occurred in students at a boarding school. Public health investigation at the time noted that these cases likely received an early dose of the MMR vaccine at 9–10 months of age, which was consistent with historical recommendations in the Northern Territory and now no longer apply.[45] The current NIP Schedule recommends 2 doses of MMR given at 12 months and 4 years of age, unless there is a contraindication. The efficacy following immunisation at less than 12 months of age may be reduced compared with those immunised at 12 months of age due to circulating maternal antibodies. *The Australian Immunisation Handbook* recommends that when MMR is given under 12 months of age, this dose should be repeated at 12 months of age or 4 weeks after the first dose, whichever is later.[13]

Map 6: Notification rates for mumps, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



The Kimberley outbreak, in which genotype J was identified, began in early July and peaked by the end of 2007. This outbreak had epidemiological links to cases in the Northern Territory (personal communication, Gary Dowse, Communicable Disease Control, Directorate, Western Australia Department of Health).

The number of mumps notifications in Australia has been increasing since 2004 (Figure 39). This increase in mumps notifications has meant that the rates in Australia in 2005, 2006 and 2007 (Figure 40) have exceeded the 1 case per 100,000 population threshold for disease elimination and are indicative of endemic mumps transmission in Australia.[41]

Figure 39: Notifications of mumps, Australia, 2002 to 2007, by month of diagnosis

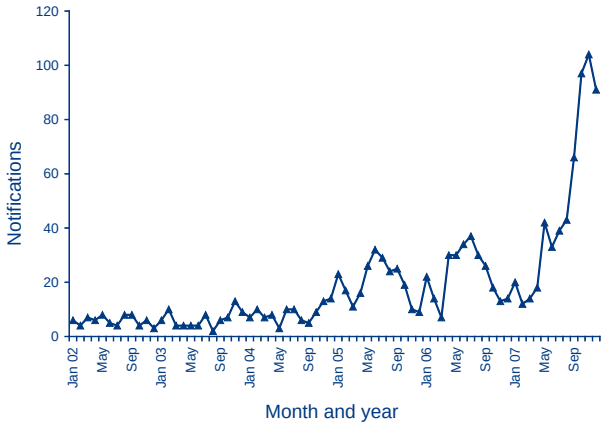
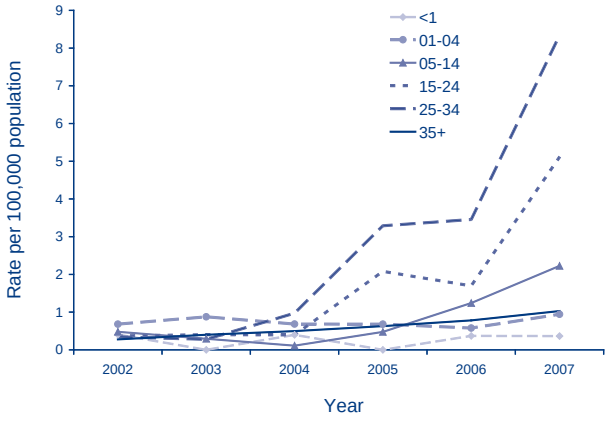


Figure 40: Notification rate for mumps, Australia, 2007, by age group

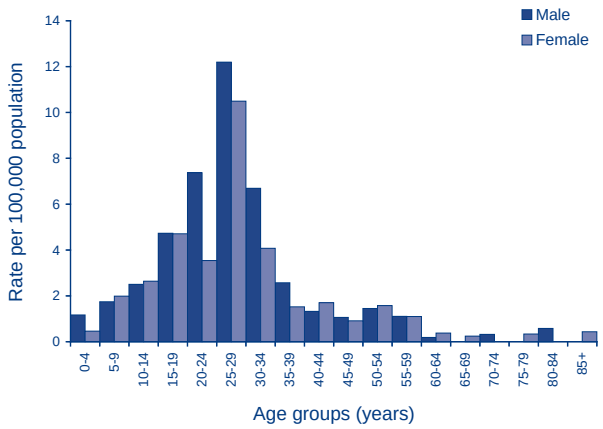


In 2007, there were cases of mumps in all age groups with the highest notification rates in the 25–29 years age group (11.4 cases per 100,000 population) while rates in young children aged less than 5 years remained low (0.8 per 100,000 population, or 11 cases) (Figure 41). In 2007, the majority of cases (325; 56%) were male.

Trends in notification rates by age group for mumps (Figure 40) show a sharp increase in the rates for the 25–34 and 15–24 years age groups, and a small decline in the less than 1 year age group compared with 2006.

Information on vaccination status was available for 330 cases (57%) of which the majority (60% or 197 cases) were not vaccinated, 7% (24 cases) were partially vaccinated for age and 33% (109 cases) were fully vaccinated for age. The high rate of mumps in the 25–34 years age group represents a susceptible cohort of individuals who may not have been immunised. In fact, 57% of those who were not vaccinated were in this age group. Of those with known vaccination status, males were 1.5 times less likely to be vaccinated than females. Mumps vaccine was made available in Australia in 1980 for use at 12–15 months of age and was combined with measles vaccine in 1982. Therefore, no childhood doses of mumps vaccine were available to most individuals in the 25–34 years age group (birth years 1973–1982). This cohort was also not targeted in the Measles Control Campaign in 1998 where the 2nd dose of MMR was offered to primary school aged children (5–12 years).

Figure 41: Trends in notification rates for mumps, Australia, 2002 to 2007, by age group



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. Rates are normally higher in late winter and spring, however from 2004 onward, non-seasonal activity remained elevated compared with previous years (Figure 42).

In 2007, 5,323 cases of pertussis were notified to NNDSS representing a rate of 25.3 cases per 100,000 population. This was lower compared with that reported in 2006 (10,996 cases; 53.1 cases per 100,000 population). The decrease in rate of pertussis notifications in Australia from 2006 to 2007 may be in part due to errors in diagnosis using serology identified in 2006. In October 2006, PanBio Ltd announced a major revision in the cut-off level for their pertussis serology tests. The original kits were withdrawn from the market towards the end of 2006 and a revised version released in October 2006. A decrease in notifications was observed in the last months of 2006.

Notification rates in 2007 increased with age, with the highest notification rate in the 65–69 years age group (45.9 cases per 100,000 population; Figure 43). There were more cases among females (3,079; 57.8%) than males (2,232; 42.0%), with 12 cases not having sex specified. The highest rates among females were in the 60–64 years age group (48.4 per 100,000 population) and the highest rates in males were in the 65–69 years age group (45.8 per 100,000 population). There were no recorded deaths for pertussis in 2007.

Trends in the pertussis notification rate in different age groups are shown in Figure 44. In 2007, pertussis notification rates declined in all age groups compared with 2006 with the exception of the 1–4 and the 5–9 years age groups, both of which experienced a small increase. In particular, the decline seen in

Figure 43: Notification rate for pertussis, Australia, 2007, by age and sex

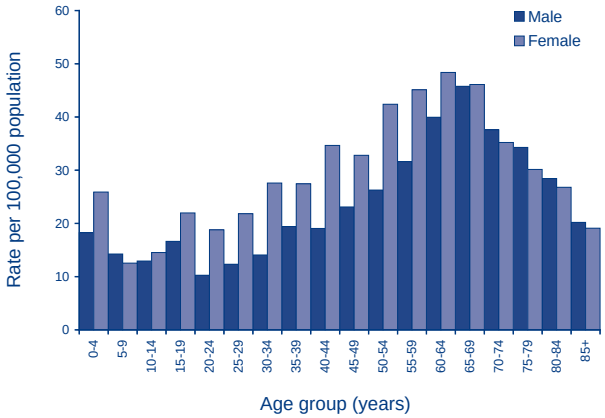
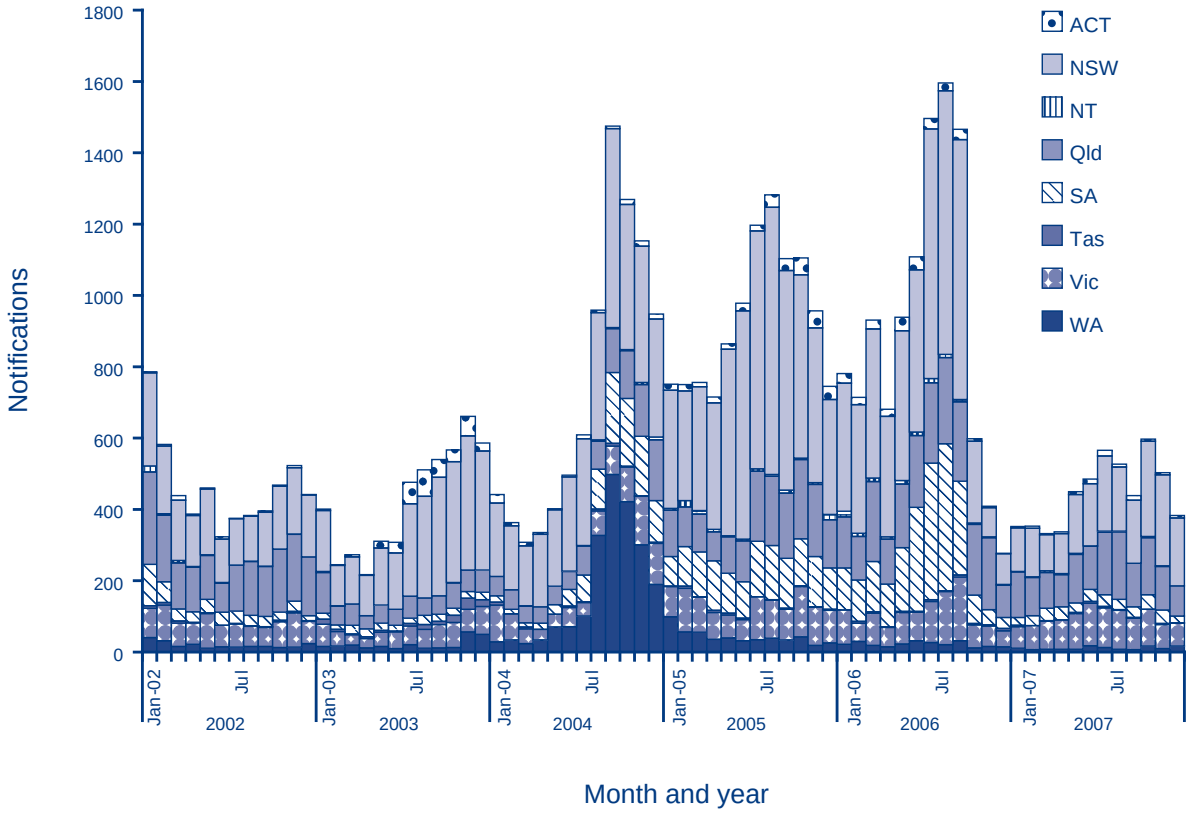


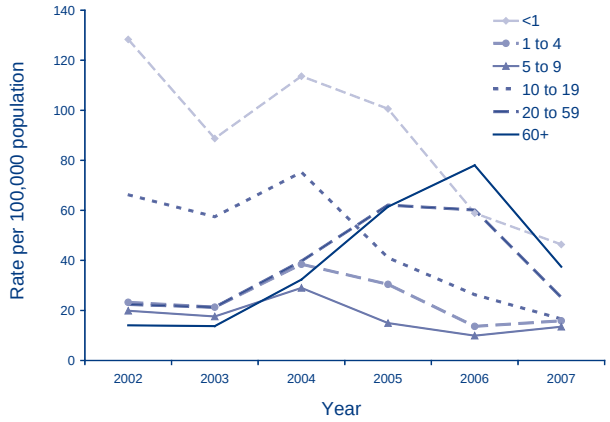
Figure 42: Notifications of pertussis, Australia, 2002 to 2007 by month of diagnosis



the 10–19 years age group following the introduction, of adolescent (15–17 years olds) vaccination to the NIP in January 2004, continued in 2007. In 2007, 82% of pertussis cases were aged 20 years or over, compared with 50% in 2000.

Notification rates for pertussis varied considerably by residential location (Map 7).

Figure 44: Trends in the notification rates of pertussis, Australia, 2002 to 2007, by age group

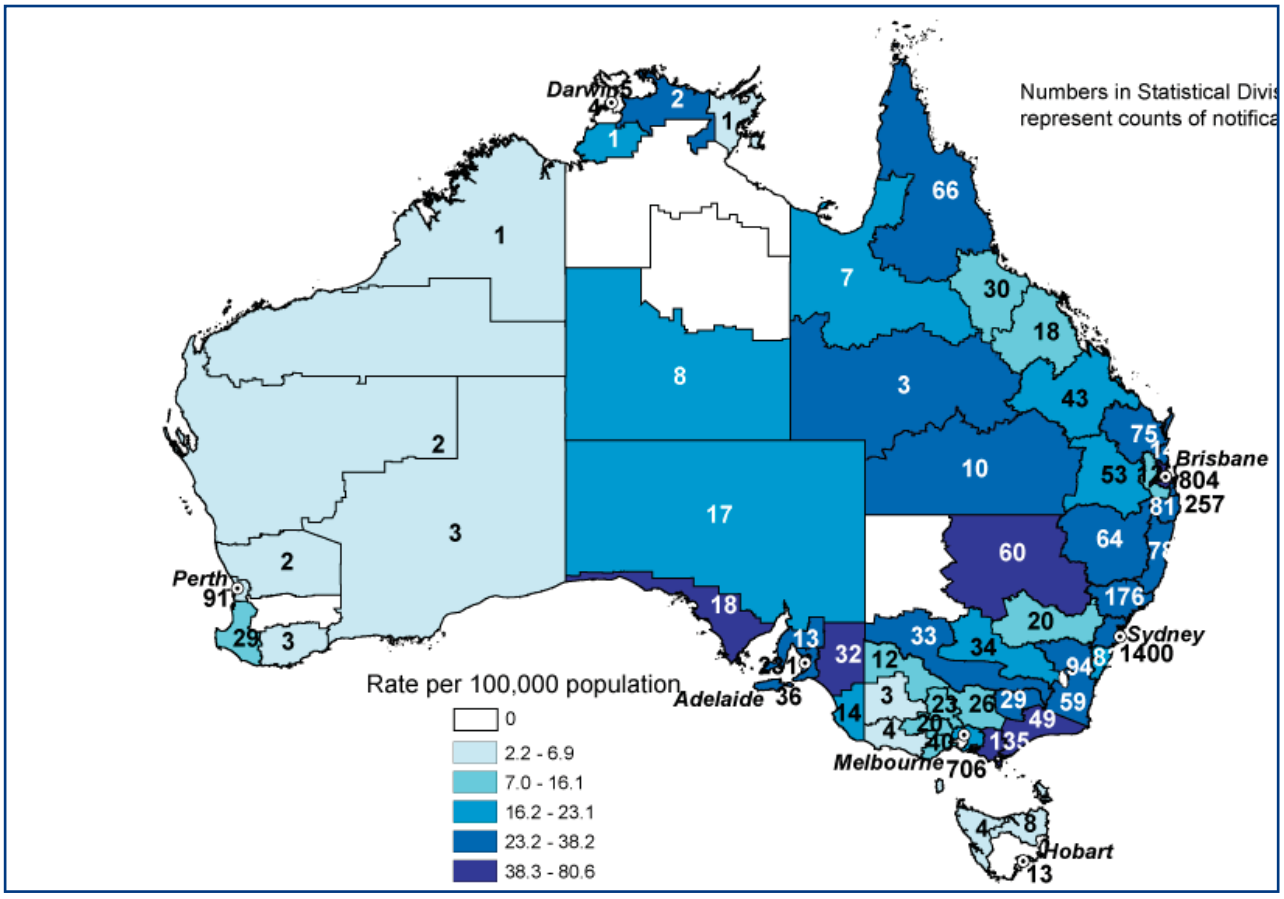


The highest rates were reported from the Darwin region of the Northern Territory, central northern New South Wales, parts of Victoria and the southern areas of South Australia, with rates in these locations being higher than the national rate. By jurisdiction, the highest rates were in Queensland (36.7 cases per 100,000 population) and New South Wales (30.0 cases per 100,000 population).

Poliomyelitis

In 2007, Australia had its first case of acute flaccid paralysis (AFP) due to wild poliovirus in 30 years. Although a major clinical presentation of the poliovirus infection, AFP occurs in less than 1% of poliovirus infections. The imported case of polio occurred in a 22-year-old male student arriving from Pakistan in July 2007. The poliovirus was detected by the National Poliovirus Reference Laboratory (NPRL) for Australia, which along with the Australian Paediatric Surveillance Unit (APSU), co-ordinates surveillance for AFP. This imported case highlights the importance of continued high quality AFP surveillance in Australia and the maintenance of high levels of polio vaccine coverage despite our current polio-free status.

Map 7: Notification rates for pertussis, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



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. In Australia in 2007, a total of 27 eligible AFP cases were notified to the NPRL via the APSU between 1 January and 31 December, of which 26 had sufficient information for classification. The 2007 non-polio AFP rate, based on the 26 cases as classified by the Polio Expert Committee (PEC), was 0.65 per 100,000 children aged less than 15 years and hence below the performance indicator set by the WHO. Details of the 2007 notifications, including the imported case, are provided in the 2007 annual report of the Australian NPRL.[46]

Since the removal of oral polio vaccine from the immunisation schedule and its replacement with inactivated polio vaccine in November 2005, poliovirus should no longer be isolated from clinical specimens in Australia without overseas travel.

The imported polio case in 2007 highlighted the need for a comprehensive, coordinated and consistent response to such events with any poliovirus isolated in Australia fully investigated to determine the source of the virus and to prevent any local transmission. As a certified polio-free country, Australia is required by the WHO to have an action plan for responding rapidly to importations of wild poliovirus. The *Acute Flaccid Paralysis and Poliomyelitis Outbreak Response Plan for Australia* (Polio Response Plan) was initiated in late 2006 by the Department of Health and Ageing in consultation with CDNA and PEC, and refined following the experience gained during the control of the imported case in July 2007. This plan, based on a risk management approach to biological emergencies, is designed as a high level national response outlining potential scenarios for occurrence of a case of poliovirus infection in Australia, the importance of surveillance and notification procedures and to guide key stakeholders involved in detection, investigation and containment of a potential poliovirus infection in Australia. The Polio Response Plan was endorsed by Australian Health Protection Committee at their meeting in December 2008 and satisfies the WHO requirement that all member states have an action plan for rapid response to outbreaks of poliovirus.

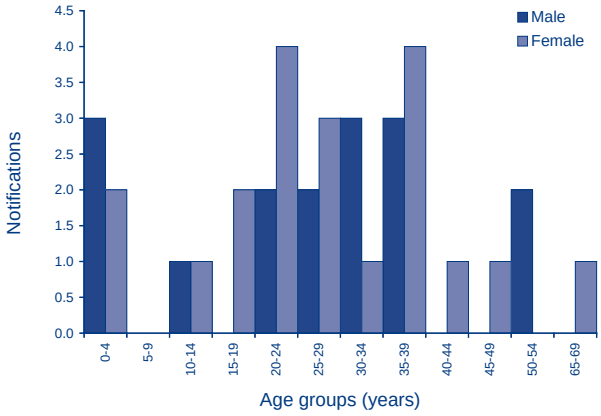
Renewed efforts in 2007 to eradicate polio worldwide saw an overall global decrease of 35% in case numbers between 2006 and 2007, including a significant 81% decrease in the number of wild poliovirus type 1 (WPV1) cases during this time. However, the risk of importation of WPV remains, with 1,304 confirmed cases reported globally to the WHO, the majority (92%) of which occurred in the 4 polio endemic countries of Nigeria, India, Pakistan and Afghanistan where the transmission of wild poliovirus continues.[47]

Rubella

In 2007, there were 36 notifications of rubella (0.2 cases per 100,000 population), a decrease compared with the 59 notifications in 2006. Cases were reported from Queensland (14), New South Wales (8), Victoria (7), Western Australia (4), the Australian Capital Territory (2) and South Australia (1).

Small case numbers were reported across the age groups between 0 and 69, except for the 5–9 years age group where no cases were reported. The majority (61%) occurred in cases aged between 20 and 39 years, with the next highest (14%) occurring in cases aged between 0 and 4.9 years (Figure 45). The mean age was 27.7 years.

Figure 45: Notification rate for rubella, Australia, 2007, by age group and sex



The overall male to female ratio of notified cases in 2007 was 0.8:1, with 16 males and 20 females. This contrasts with 2006 and some previous years (1999, 2002 and 2003) when there was an overall predominance of males notified.

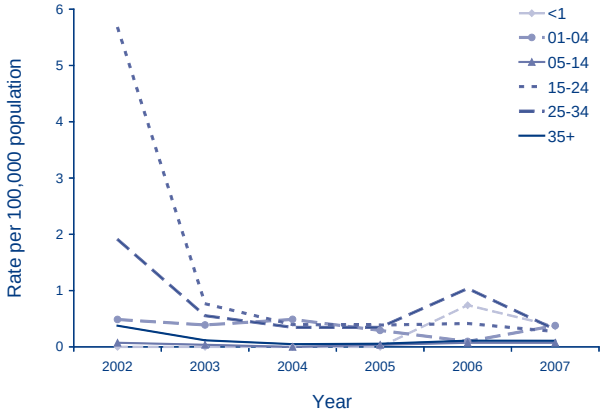
Information on vaccination status was available for 16 of the 36 cases of which six were fully vaccinated for age, three were partially vaccinated and the majority (7) were not vaccinated. Vaccination status in the remaining 20 cases was either unknown or missing.

In Australia, populations at risk of rubella include young males who did not receive the rubella immunisation in school based programs,[48] migrant women who did not receive rubella vaccines in their countries of birth,[49,50] and Indigenous women with inadequate immunity.[51] In 2007, of the 7 male cases where information on vaccination status was reported, five were not vaccinated (3 of which were between 20 and 30 years of age and 2 between 0 and 4 years of age) and two were partially vaccinated. Of the 9 female cases in 2007 with vaccination status

reported, seven were fully or partially vaccinated and two were not vaccinated (both of which were between 25 and 44 years of age). None of the rubella cases in 2007 was identified as Indigenous.

Figure 46 shows trends in rubella notification rates in different age groups, with a slight increase in rates in the 1–4 years age group in 2007 compared with 2006, but otherwise continuing at the low levels seen since 2003.

Figure 46: Trends in notification rates of rubella, Australia, 2002 to 2007, by age group



There were 2 cases of congenital rubella reported in 2007, one of which was fatal. The cases were reported from New South Wales and Victoria. While this is an increase compared with 2006 when there were no cases reported, and compared with the 5-year mean of 1.4 cases, it is consistent with notifications in earlier years including 2002 (2 cases), 2003 (3 cases), 2004 and 2005 (1 case each year). Altogether there were 16 cases of rubella notified in women of child bearing age (15–49 years) representing 80% of the total number of female cases in 2007.

Brotherton et al (2007)[41] suggest that the achievement and confirmation of the elimination of locally acquired rubella circulation may require targeted immunisation of migrants from countries with low levels of rubella vaccination and the establishment of rubella genotyping in Australia.

Tetanus

In 2007, there were 3 notifications of tetanus. One case occurred in an unimmunised 93-year-old male from Tasmania and resulted in his death. The other 2 cases were a male aged 76 years of unknown vaccination status and an unimmunised female aged 79 years.

Varicella infections

In November 2005, the varicella vaccine was added to the NIP Schedule as a single dose due at 18 months (for children born on or after 1 May 2004), or 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 2007, 6 jurisdictions were sending data to NNDSS, with NSW having decided in 2006 not to make varicella infections notifiable. The legal processes to make varicella notifiable in Victoria were still underway.

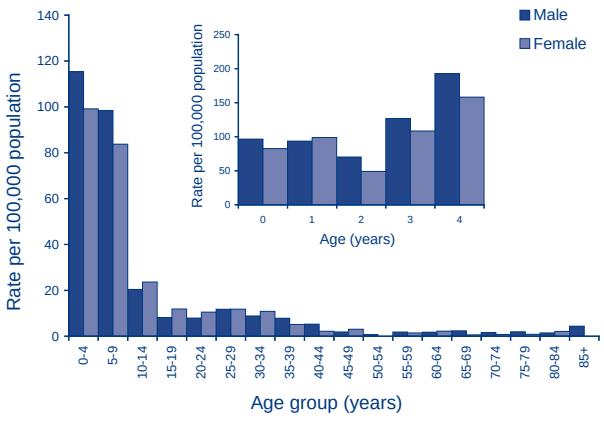
In 2007, there were 7,496 varicella notifications from the 6 notifying jurisdictions, with 1,651 (22%) reported as chickenpox, 1,547 (21%) as shingles and 4,298 (57%) as unspecified varicella infection.

Varicella zoster infection (chickenpox)

In 2007, there were 1,651 notifications of chickenpox reported from 6 jurisdictions, a rate of 18.5 cases per 100,000 population. The highest rates were reported from the Northern Territory (91.7 cases per 100,000 population; 197 cases) and South Australia (46.2 cases per 100,000 population; 732 cases).

A total of 1,145 cases (69.4 %) occurred in children aged less than 10 years. The highest rates were in the 0–4 years age group (107.5 cases per 100,000 population; 613 cases) and within this age group children aged 4 years had the highest notification rate (175.9 cases per 100,000 population; 196 cases; Figure 47).

Figure 47: Notification rate for chickenpox, Australia,* 2007, by age group and sex



* Excluding New South Wales and Victoria.

Of all notifications, there were slightly more male than female cases notified, with 881 males (53.4%) compared with 768 females (46.5%). Two cases did not have the sex specified.

Indigenous status was recorded for 85% of notifications, with the majority (1,224; 74.1%) being reported as non-Indigenous. A total of 172 notifications (10.4%) were Indigenous, with 255 (15.5%) being reported as not stated or blank.

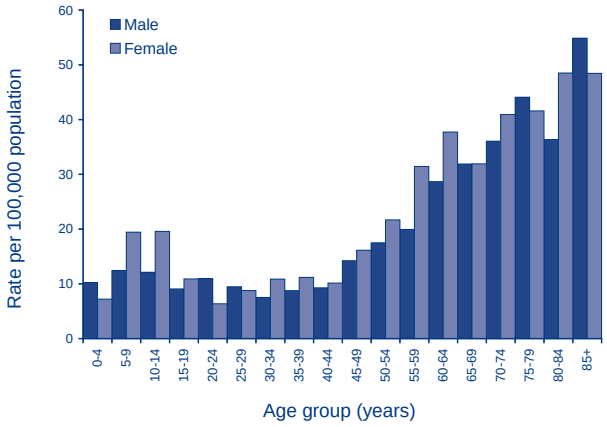
Ninety-two cases (5.6%) were recorded as fully vaccinated for age; three partially; and 458 unvaccinated. There was no vaccination status information on the remainder of the notified cases (1,098), and no recorded deaths from chickenpox in 2007.

Varicella zoster infection (shingles)

There were 1,547 notifications of shingles reported to NNDSS in 2007 from 6 jurisdictions, a rate of 17.3 cases per 100,000 population. The highest rates were in the Northern Territory (41.4 cases per 100,000 population, 89 cases) and South Australia (37.1 cases per 100,000 population, 587 cases).

There were more female cases (852; 55.1%) than males (695; 44.9%). The highest rates were in the over 85 years age group (50.6 cases per 100,000 population; 69 cases; Figure 48). There was 1 recorded death for cases of shingles.

Figure 48: Notification rate for shingles, Australia,* 2007, by age group and sex



* Excluding New South Wales and Victoria.

Indigenous status was recorded for 80.8% of notifications, with the majority (1,186; 76.7%) being reported as non-Indigenous. A total of 64 (4.1%) notifications were Indigenous, with 297 (19.2%) being reported as not stated or blank.

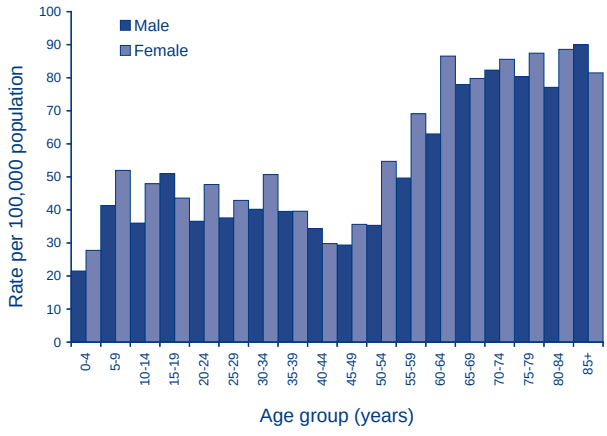
Varicella zoster infection (unspecified)

There were 4,298 cases of varicella infections (unspecified) based on laboratory diagnosis from 6 jurisdictions in 2007, a rate of 48.2 cases per 100,000 population. The highest rates were reported from Queensland (73.5 cases per 100,000 population; 3,072 cases), Western Australia (31.3 cases per 100,000 population; 659 cases) and the Australian Capital Territory (30.3 cases per 100,000 population; 102 cases).

There were more notifications in females (2,333; 54.3%) than males (1,957; 45.5%), with 2 deaths occurring due to unspecified varicella zoster infection. The age distribution of unspecified varicella infections is shown in Figure 49.

Indigenous status was recorded for 27.0% of notifications, with the majority (1,099; 25.6%) being reported as non-Indigenous. A total of 65 notifications (1.5%) were Indigenous, with 3,134 (73.0%) being reported as not stated or blank.

Figure 49: Notification rate for varicella zoster infection (unspecified), Australia,* 2007, by age group and sex



* Excluding New South Wales and Victoria.

Vectorborne diseases

Notifications

During 2007, there were 6,823 notifications of mosquito-borne diseases reported to NNDSS (4.6% of total notifications). This was a 20% decrease in the number of notifications for 2006 (8,606). 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 discussed under quarantinable diseases) and malaria.

Alphaviruses

Alphaviruses are single-stranded RNA viruses that cause disease epidemics characterised by fever, rash and polyarthritis. There are a variety of mosquito vectors for Barmah Forest virus and Ross River virus, which facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[52] In Australia, Barmah Forest virus (BFV) infection and Ross River virus (RRV) infection are the alphaviruses of major public health significance, accounting for 87% (5,919 cases) of the total mosquito-borne disease notifications for 2007. Between 2002 and 2006 (Figure 50), notifications ranged annually for BFV from 910 (2002) to 2,142 (2006), and for RRV from 1,459 (2002) and 5,547 (2006). In 2007, there were 1,716 notifications of BFV and 4,203 of RRV.

Chikungunya

Chikungunya virus is a member of the alphavirus genus in the family Togaviridae. It belongs to the Semliki Forest virus complex and is therefore closely related to Ross River and Barmah Forest. It is found epidemically in many parts of South East Asia and in Africa. Chikungunya causes illness characterised by an abrupt onset of fever, rash and severe joint pain (chikungunya is Bantu of the Makonde people of south-east Tanzania for 'that which bends up', reflecting the bent over appearance of those with severe joint pain). The acute disease lasts 3–10 days, but convalescence may include prolonged joint swelling and pain lasting weeks or months. It has clinical similarities to dengue, including occasional cases with haemorrhagic manifestations. Deaths are rare.[53]

In Australia, the known competent vectors for chikungunya virus *Aedes aegypti* occur in northern Queensland and *Aedes albopictus* are found on Cocos, Christmas and the Torres Strait Islands. Other Australian mosquitoes could be possible vectors, but there are no data on the competence of these at present.

There have been known imported cases of chikungunya virus into Australia from viraemic travellers during the recent epidemic in the Indian Ocean. Outbreaks in near neighbours such as Indonesia and Papua New Guinea, where we have more travel origins could feasibly increase the numbers of viraemic travellers and hence introduce the disease. Continued Australian military presence in South East Asia also provides a likely entry route.

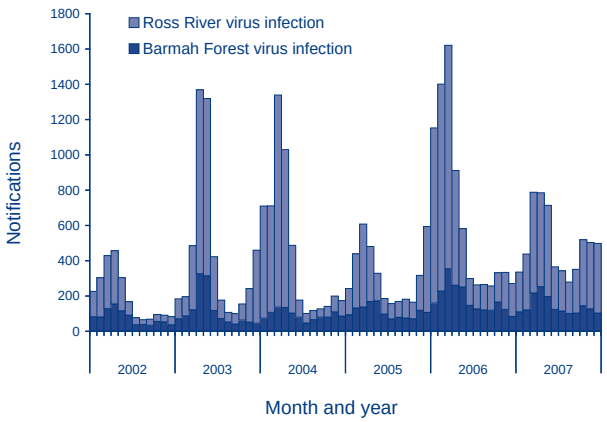
Northern Australia has suitable climate and environment parameters for the introduction of chikungunya. The National Arbovirus and Malaria Advisory Committee (NAMAC) considered and advised CDNA on 23 January 2008 that with the increased number of infected cases to Australia, and the possibility of local transmission, the number of cases of chikungunya in Australia will increase. NAMAC has initiated action, through the CDNA, to make chikungunya a nationally notifiable disease.

Barmah Forest virus infection

There were 1,716 notifications of BFV infections notified to NNDSS in 2007, which accounted for 25% of total mosquito-borne disease notifications for the reporting period. Forty-eight per cent of BFV notifications were reported from Queensland (n=826) and 33% from New South Wales (n=572). BFV notifications during 2007 were 1.3 times the mean for the previous 5 years.

The highest rates of BFV notifications were reported by the Northern Territory (42.3 cases per 100,000 population compared with 62.9 cases per 100,000 population in 2006), Queensland (19.8 cases per 100,000 population compared with 23.6 cases per 100,000 population in 2006), and New South Wales (8.3 cases per 100,000 population compared with 9.4 cases per 100,000 population in 2006). Cases were reported in all jurisdictions

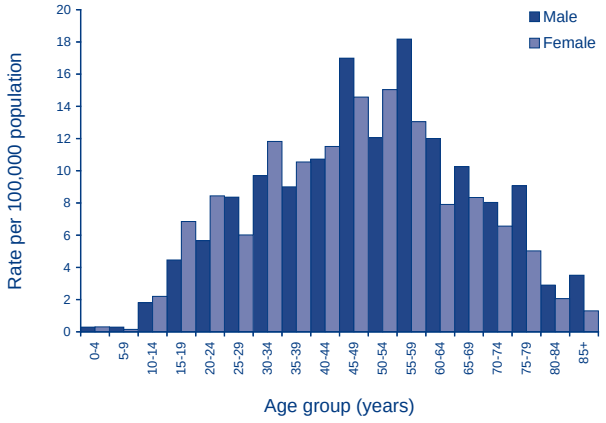
Figure 50. Notifications of Barmah Forest and Ross River virus infections, Australia, 2002 to 2007, by month and year of onset



except for Tasmania. The national BFV notification rate in 2007 was 8.2 cases per 100,000 population, compared with 10.3 cases per 100,000 population in 2006. Notification rates for BFV varied by geographic location (Map 8). These locations represent the place of residence of a notified case and not the place of acquisition of infection. For 2007, the highest regional BFV notification rate was reported in the Litchfield Shire of the Northern Territory (74.7 cases per 100,000 population).

Figure 51 shows the age and sex distribution of BFV notifications. The BFV notification rate was highest amongst the 45–49 year age group (15.8 cases per 100,000 population). A similar number of males and females were notified to NNDSS with BFV.

Figure 51: Notification rate for Barmah Forest virus infections, Australia, 2007, by age group and sex

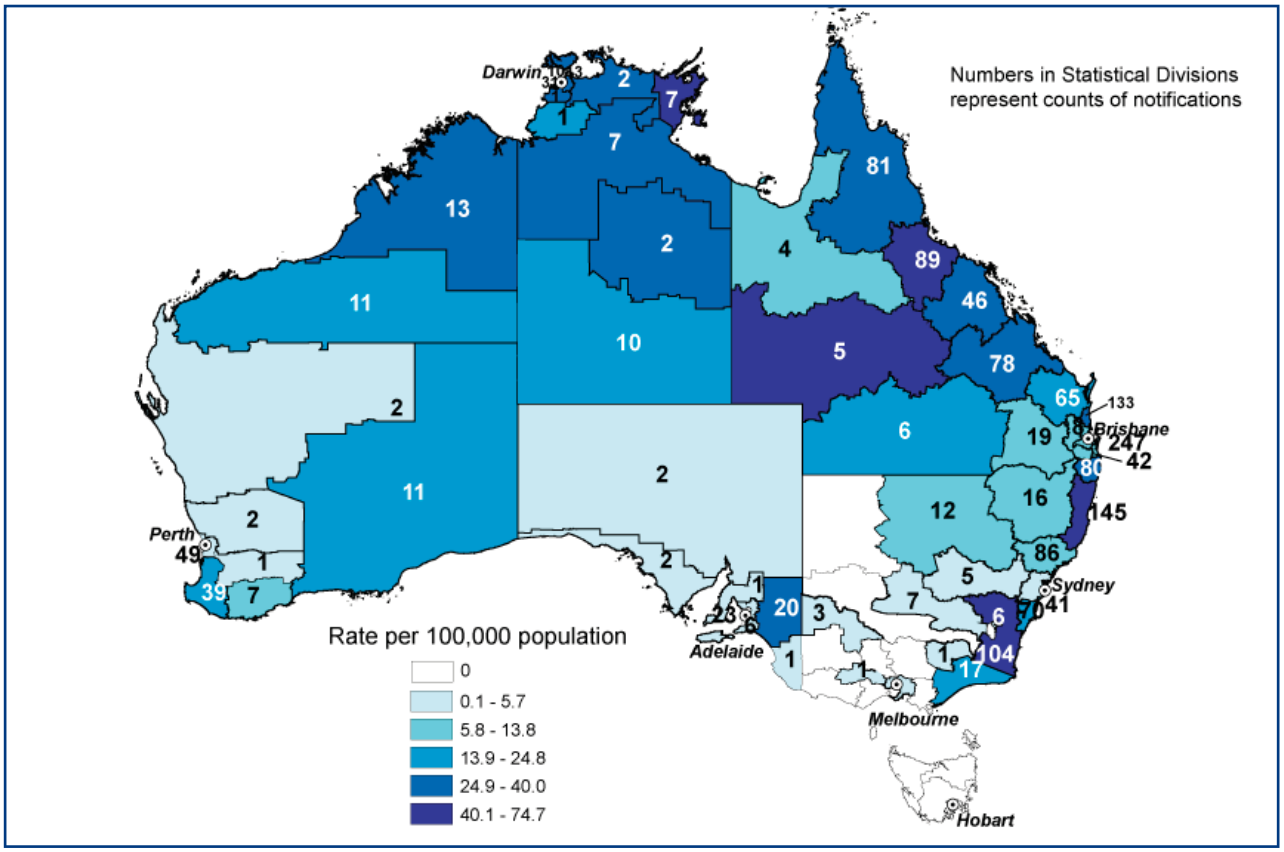


Ross River virus infection

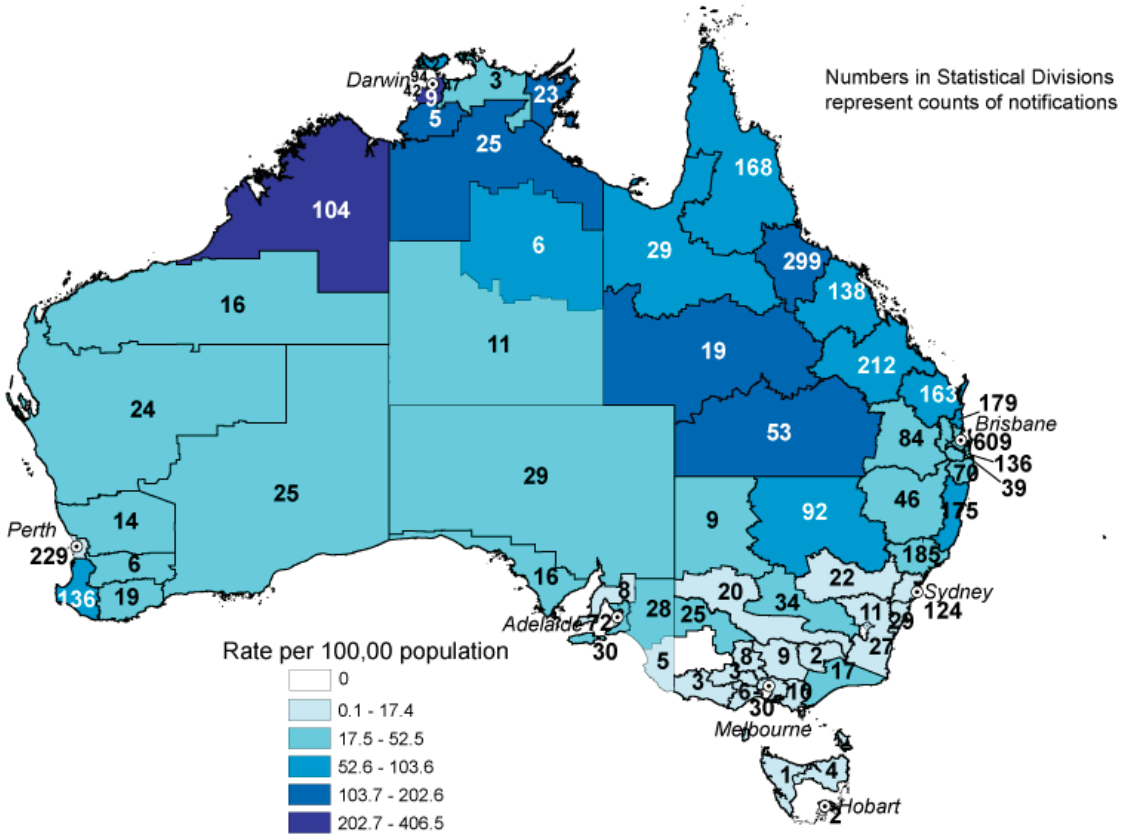
There were 4,203 notifications of RRV infections reported to NNDSS in 2007, which accounted for 62% of the total of mosquito-borne disease notifications received during this reporting period.

Cases of RRV infection reported to NNDSS varied by geographic region but the majority of notifications in 2007 were from Queensland (51%, n=2,137) and New South Wales (20%, n=841). These locations represent the place of residence of a notified case and not necessarily the place of acquisition of infection. Map 9 shows that the highest rates of notifications were reported in the Finniss area of the Northern Territory (406 cases per 100,000 population) and the Kimberley region of Western Australia (303 cases per 100,000 population). Five of the top 10 rates of RRV notification by region in Australia

Map 8: Notification rates for Barmah Forest virus infection, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for Northern Territory



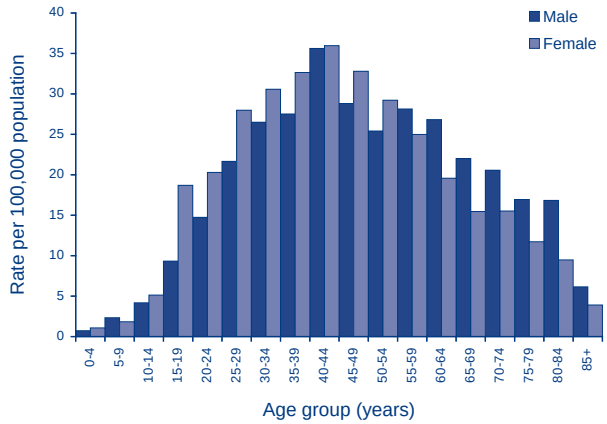
Map 9: Notification rates for Ross River virus infection, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



occurred in the Northern Territory in 2007. The national RRV notification rate for 2007 was 20 cases per 100,000 population compared with 26.6 cases per 100,000 population in 2006.

The age and sex distribution of RRV notifications is shown in Figure 52. The RRV national notification rate was highest in the 40–44 years age group (35.8 cases per 100,000 population). Overall, 48% of notifications reported to NNDSS were males.

Figure 52: Notification rate for Ross River virus infections, Australia, 2007, 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 network involving New South Wales, the Northern Territory, Victoria and Western Australia. The flocks are located in strategic locations and are regularly tested for antibodies to MVEV and KUNV. This program is designed to provide early warning of flavivirus activity (excluding dengue and JEV).[54] A sentinel chicken surveillance report was published as part of the National Arbovirus and Malaria Advisory Committee Annual Report 2006–07.[55]

Murray Valley encephalitis virus infection

There were no cases of MVEV infection reported to NNDSS in 2007, compared with 1 case reported in 2006 in Western Australia.

Kunjin virus infection

In October 2007, 1 case of KUNV was reported to NNDSS in Victoria compared with 3 notifications of KUNV in 2006. Further investigations resulted in the reclassification of the diagnosis as West Nile virus. This is the first report of a laboratory confirmed West Nile virus (New York 99) infection imported into Australia.

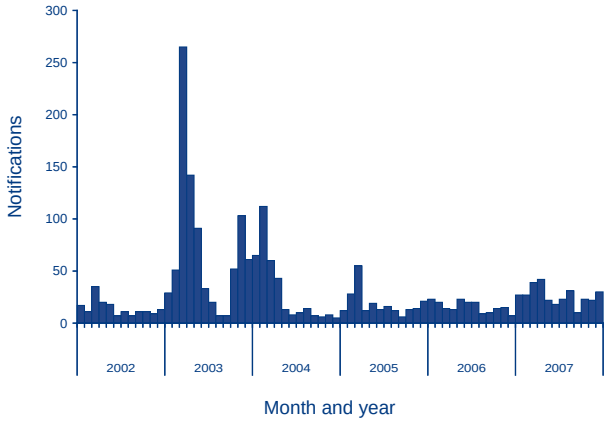
Dengue virus infection

There were 314 notifications of DENV infection reported to NNDSS in 2007 (Figure 53) including 268 notifications of overseas acquired dengue virus infection. In Australia, imported cases of DENV are reported each year with occasional local transmission. Local transmission is restricted to areas of northern Queensland where the key mosquito vector, *Aedes aegypti*, is present. The number of cases reported in 2007 was a 68% increase in the number of cases reported in 2006 (n=187).

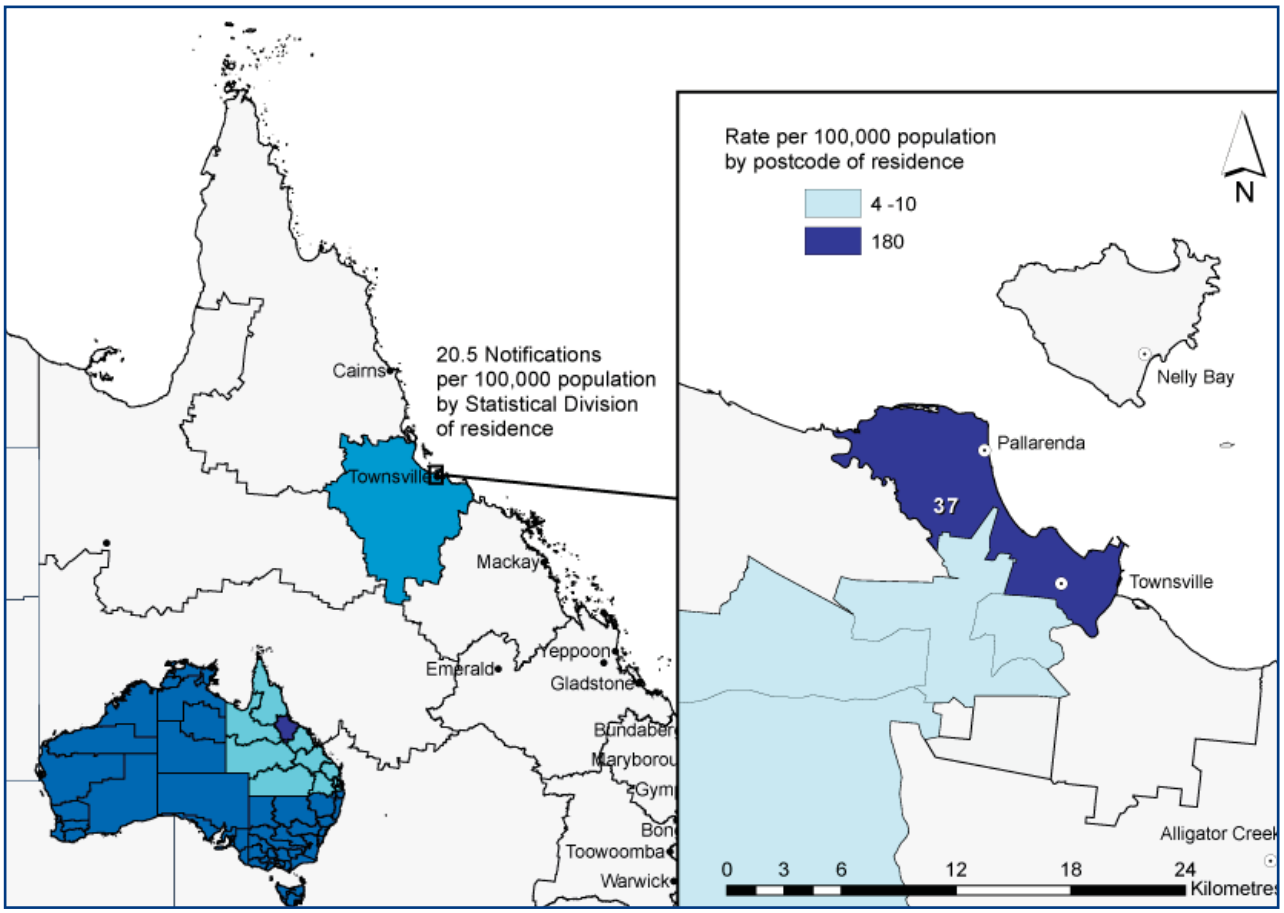
Queensland reported 120 (38%) notifications of DENV in 2007. An outbreak of locally-acquired dengue serotype 3 occurred in Townsville between February and April 2007. Locally-acquired cases represented 15% (46/314) of the total number of dengue notifications for 2007. Map 10 presents 44 of 46 notifications that were acquired locally and able to be represented geographically (2 cases, a resident of Brisbane and Darwin acquired their infection in Townsville).

In early 2004, 2 deaths were reported in Australia due to dengue fever. These were the first deaths attributed to dengue in over 100 years.[56]

Figure 53: Notifications of dengue (locally-acquired and imported cases), Australia, 2002 to 2007, by month and year of diagnosis

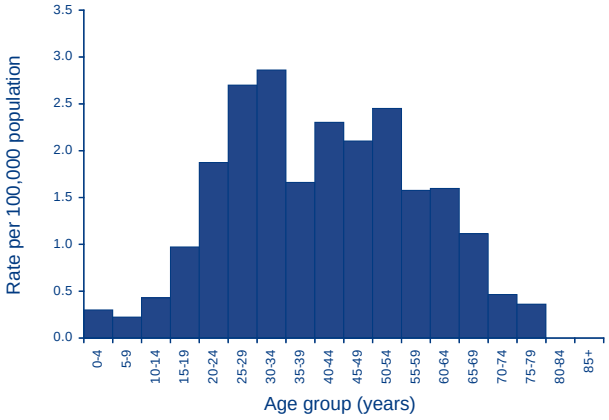


Map 10: Notification number and rate of locally-acquired dengue virus infection, Australia, 2007



In 2007, the highest notification rate for DENV occurred in the 25–34 years age range and the 50–54 years age group (Figure 54). The highest rate for males was in the 45–49 years age group (22 cases) and in females in the 25–29 years age group (22 cases). Fifty-two per cent of DENV cases were male (n=163) and 91% of cases were aged between 15 and 64 years (n=287).

Figure 54: Notification rate for dengue (locally-acquired and imported cases), Australia, 2007, by age group and sex



Japanese encephalitis virus infections

There were no human cases of JEV notified in Australia in 2007. The last JEV notification was reported by Queensland in February 2004 when a 66-year-old male acquired JEV in Papua New Guinea.[57] There have been 9 other cases of JEV reported to NNDSS since 1995, although JEV was not nationally notifiable until 2001.[57] Four of these 9 notifications were reported in Torres Strait Islanders from the Badu Island community.[57] The other locally acquired JEV case was reported in a resident from the Cape York Peninsula, Queensland.[57] The remaining 4 cases were reported as acquired from overseas countries.[57]

Flavivirus infections (NEC)

There were 22 flavivirus infection (not elsewhere classified) notifications during 2007; notified by Queensland (n=18) and Victoria (n=4).

There were 3 Kokobera virus notifications from Queensland in this category.

Malaria

There were 567 notifications of overseas acquired malaria in Australia in 2007, compared with 772 notifications in 2006 (Figure 55). There were no reports of locally acquired malaria in 2007. The majority of cases were reported by Queensland (34%), Victoria (20%), New South Wales (17%) and Western Australia (15%). Queensland reported that 87 of 193 notifications were acquired in Papua New Guinea.

The largest number (n=70) of malaria notifications was in the 20–24 years age group (Figure 56). Sixty-five per cent of malaria notifications were for males.

The infecting *Plasmodium* species was reported for 97% of malaria notifications in 2007 (Table 18). Of these 567 notifications, *P. falciparum* and *P. vivax* were the predominant species.

Figure 55: Notifications of malaria, Australia, 2002 to 2007, by month and year of onset

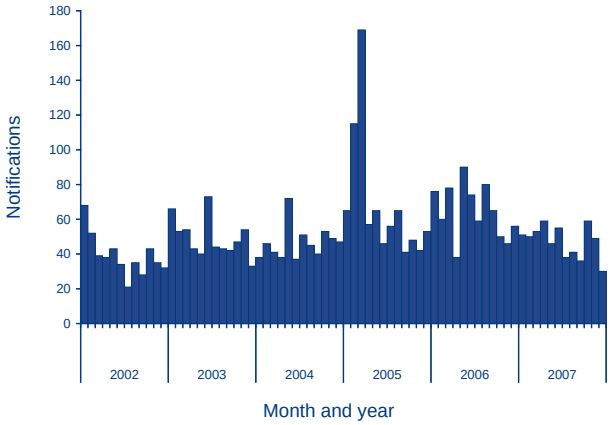


Figure 56: Notifications of malaria, Australia, 2007, by age group and sex

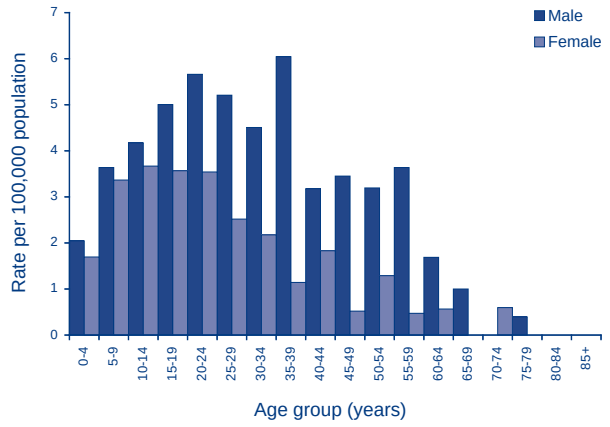


Table 18: Notifications of malaria, Australia, 2007, by parasite type and state or territory

Parasite type	State or territory								Aust	Type (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Plasmodium falciparum	2	32	24	88	17	10	39	51	263	46
Plasmodium malariae	0	2	0	2	0	0	3	3	10	2
Plasmodium ovale	1	2	0	5	0	0	4	6	18	3
Plasmodium vivax	7	58	5	98	7	3	63	12	253	45
Plasmodium species	2	3	0	0	0	0	0	11	16	3
Mixed *P. falciparum* and other species*	0	0	0	0	0	1	4	0	5	1
Mixed other species*	0	0	0	0	0	0	0	2	2	0
Total	12	97	29	193	24	14	113	85	567	

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

Zoonoses

Zoonoses are 'those diseases and infection which are naturally transmitted between vertebrate animals and man'.[58] Approximately 60%–70% of emerging human infectious diseases are zoonoses[59,60] and more than 70% of emerging zoonoses originate from wildlife.[59] 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'.[61]

In 2007, zoonotic diseases notifiable to NNDSS were anthrax, Australian bat lyssaviral or lyssaviral (unspecified) infection, brucellosis, leptospirosis, ornithosis, Q fever, and tularaemia. During 2007, 687 notifications of zoonotic disease (0.5% of total notifications) were made to NNDSS. Queensland accounted for 40% (278 cases) of the zoonotic diseases. Notification numbers were generally higher in males (72%, 497 cases). Notifications of cases aged less than 15 years accounted for 4%, (27 cases) of all notifications. There were no cases of tularaemia reported to NNDSS during 2007.

Anthrax

Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts.[17] Anthrax has a low prevalence in animals, and occurs only sporadically in Australia.[62] It can be an occupational hazard for veterinarians, and agriculture and wildlife workers who handle infected animals.

One case of cutaneous anthrax was reported to NNDSS in 2007. The case was a male knackery worker from northern Victoria, and who had contact with 2 cattle that were subsequently confirmed to have died of anthrax.[63] Over the previous 10 years, only 2 other human cases of anthrax had been reported in Australia, both the cutaneous form, in 1998 and 2006 respectively.[19, 64] Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

In 2007, 13 outbreaks of anthrax were reported in livestock. Twelve outbreaks occurred in New South Wales, where cases have been known to occur in the past, and one in Central Victoria. In all cases, properties were subject to the recommended protocol of quarantine, carcass incineration, site disinfection and vaccination of in-contact animals. All movements from affected properties were traced to ensure that relevant product did not enter the export and domestic food production chains.[62]

Australian bat lyssaviral and lyssaviral (unspecified) infections

No cases of either Australian bat lyssaviral or lyssaviral (unspecified) infections were notified during 2007. Previously, 2 known cases of human infection with Australian bat lyssavirus were fatal and occurred in 1996 and 1998 following close contact with an infected bat.[19]

Surveillance indicates Australian bat lyssavirus infection is and may have been present in Australian bats 15 years prior to its first detection. Sick and injured bats (opportunistic specimens) and change in seasonality and bat ecology pose an increased public health risk.[65]

Brucellosis

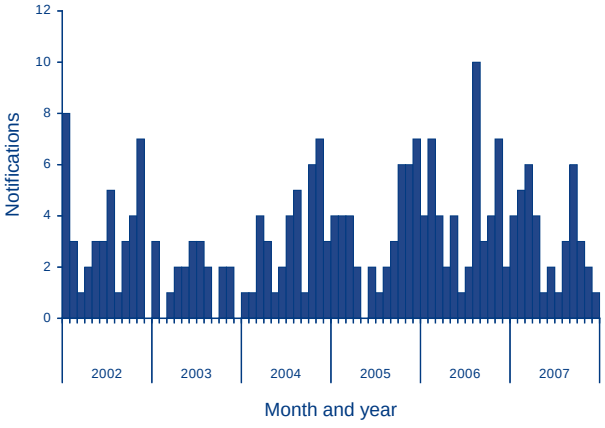
Brucellosis is mainly an occupational disease for farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[17]

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 2007, 38 cases of brucellosis were reported to NNDSS; a national notification rate of 0.2 cases per 100,000 population. Cases were from Queensland (30 cases), New South Wales (4 cases) and 1 case each from South Australia, Victoria, Tasmania and Western Australia. There was little change in the number of notifications of brucellosis over the last 6 years (Figure 57). In Australia, the notification rate for brucellosis in 2007 was lower than in 2006 (0.28 and 0.24, respectively). The highest notification rate of 53 cases per 100,000 population was reported from the Central West Statistical Division of Queensland. The majority of cases were male (n=28) and aged between 20 and 54 years (n=34).

Species data were available for 39% of notifications (n=15). Of these, nine were *B. suis* (all from Queensland), and 4 cases were *B. melitensis* (a single case in Queensland, Tasmania, Victoria, Western Australia). Each of the notified cases of *B. melitensis* were reported to have had recent history of overseas travel. A single case of *B. abortus* (South Australia) was notified to NNDSS. This case had a recent history of overseas travel to Iraq and reported eating numerous unpasteurised milk products.

Figure 57: Notifications of brucellosis, Australia, 2002 to 2007, by month and year of onset



Bovine brucellosis (*B. abortus*) was eradicated from the Australian cattle herd in 1989 and is presently considered an exotic animal disease in Australia.[62] Caprine and ovine brucellosis (caused by *B. melitensis*) has never been reported in Australian sheep or goats.[62] Swine brucellosis (caused by *B. suis*) is confined to small areas of northern Australia, where it occurs in feral pigs, with human cases predominantly seen in recreational feral pig hunters.[62,66]

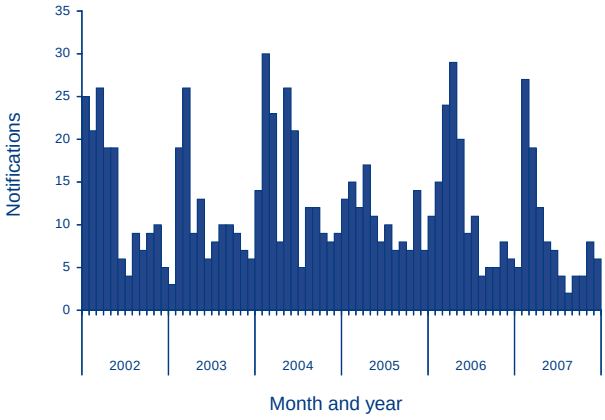
Leptospirosis

Leptospirosis is caused by spirochaetes of the genus, *Leptospira*, which is found in the renal tubules of wild and domestic animals. In affected areas, where there is exposure to infected urine of domestic and wild animals, this disease can be an occupational and recreational hazard.[17]

Between 2002 and 2006 (Figure 58), annual leptospirosis notifications ranged from 126 (2003) to 177 (2004), with 106 notifications in 2007 (0.5 cases per 100,000 population). Cases were reported in all jurisdictions except the Australian Capital Territory.

In 2007, the majority of notifications were from Queensland (75 notifications, 1.8 cases per 100,000 population). Ninety per cent of leptospirosis cases were male (n=95) and the majority of cases were aged between 15 and 54 years (n=84). The highest notification rate of 21 cases per 100,000 population was reported from the Far North Statistical Division of Queensland.

Figure 58: Notifications of leptospirosis, Australia, 2002 to 2007, by month and year of onset



Ornithosis

Ornithosis is caused by *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] Human to human transmission is rare.

In 2007, there were 92 ornithosis infections notified to NNDSS, giving a national rate of 0.4 cases per 100,000 population. In Australia, the notification rate for ornithosis in 2007 was lower than in 2006 (0.4 and 0.8, respectively). Between 2002 and 2006, the annual number of ornithosis notifications ranged from 239 (2004) to 164 (2005) (Figure 59).

Victoria had the highest number of notifications (50 notifications, 1.4 cases per 100,000 population). Notifications were also received from New South Wales (34 cases), Western Australia (3 cases), South Australia (2 cases), Queensland (2 cases), and Tasmania (1 case). The majority of cases were male (n=59, 64%). All cases were aged 15 years or older and 78% of cases were 40 years or over (Figure 60).

At risk groups of people contracting ornithosis include bird owners, pet shop employees, veterinarians, poultry-processing workers, zoo workers and taxidermists. Older adults and pregnant women may have a more severe illness.[67] An outbreak in the Blue Mountains in June 2002 was novel in that infections were predominantly associated with wild birds, rather than with pet birds and aviaries as generally reported in the scientific literature.[68] The risk factors for the cases of ornithosis notified in 2007 is unknown.

Figure 59: Notifications of ornithosis, Australia, 2002 to 2007, by month and year of diagnosis

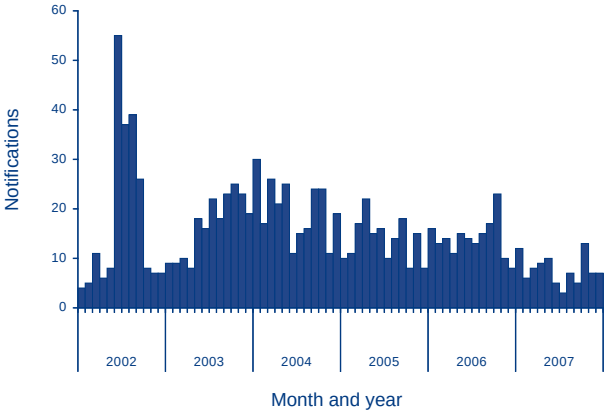
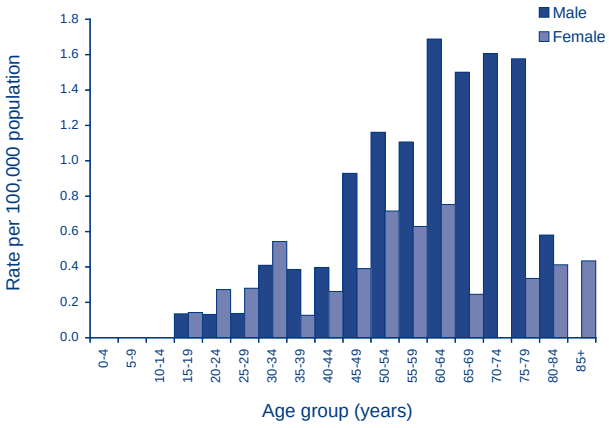


Figure 60: Notification rate for ornithosis, Australia, 2007, by age group and sex

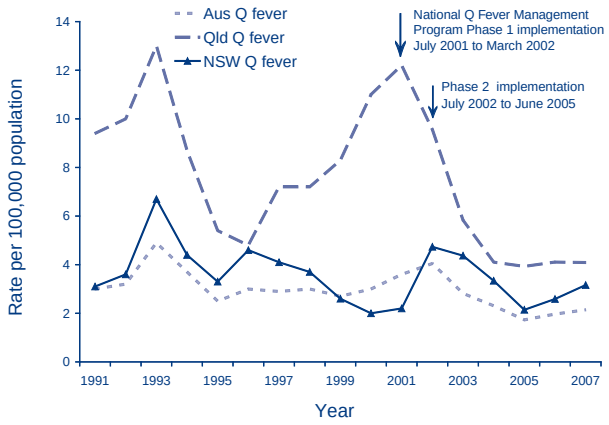


Q fever

Q fever is caused by *Coxiella burnetii*. Primary reservoirs of these bacteria are cattle, sheep and goats. The organisms are resistant to heat, drying and many common disinfectants, which enables the bacteria to survive for long periods in the environment. The mode of transmission to humans is commonly through the airborne route in dust, but it can also occur though direct contact with infected animals and other contaminated material. Humans are often very susceptible to the disease, and very few organisms may be required to cause infection. Person-to-person transmission is rare.[17]

In 2007, 450 cases of Q fever were notified to NNDSS, representing a national rate of 2.1 cases per 100,000 population (Figure 61). Between 1991 and 2001, and prior to the introduction of the National Q Fever Management Program, Q fever notification rates ranged between 2.5 cases per 100,000 population and 4.9 cases per 100,000 population. In Australia, the notification rate for Q fever in 2007 was similar to 2006 (2.1 and 2.10, respectively). Between 2002 and 2006, the annual number of Q fever notifications ranged from 795 (2002) to 353 (2005).

Figure 61: Notification rate for Q fever, Australia, New South Wales and Queensland, 1991 to 2007

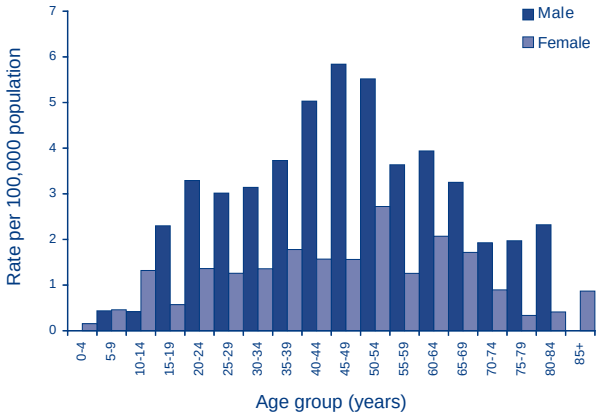


The highest rates of notifications were from Queensland (171 notifications, 4.1 cases per 100,000 population) and New South Wales (215 notifications, 3.2 cases per 100,000 population). The highest notification rate of 88 cases per 100,000 population was reported from the South West Statistical Division of Queensland (Map 11).

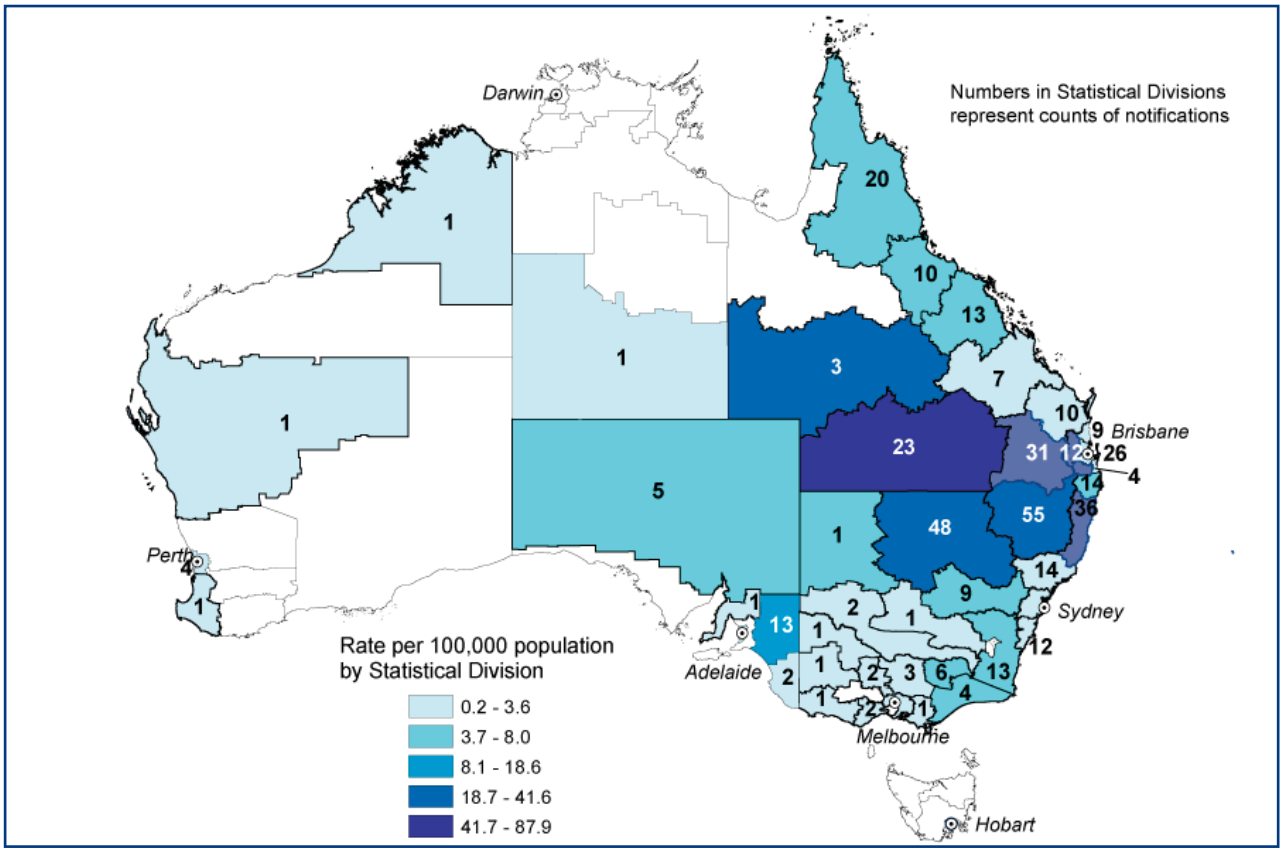
The highest age specific rates (Figure 62) of Q fever were in the 45–49 years age group for males (44 cases, 5.8 cases per 100,000 population), and in the 50–54 years age groups for females (1.8 cases per 100,000 population). There were 19 cases reported in people aged less than 15 years. Seventy per cent of cases were male (314 cases).

The National Q Fever Management Program commenced in July 2001. The program provided funding for free vaccine for people at risk of Q fever from their work environment.[69] Production of the Q fever vaccine in Australia ceased at the end of 2005.[70] At the end of 2006, the Australian Ministers for Health and Agriculture announced funding for CSL Limited to recommence production of the Q fever vaccine.[70] Vaccine from the new facility will commence in 2009. Adults at risk, including abattoir workers, farmers, veterinarians, stockyard workers, shearers, animal transporters and many others exposed to cattle, sheep or goats or their products should be considered for vaccination. The vaccine is not recommended for children under 15 years of age.[13]

Figure 62: Notification rate for Q fever, Australia, 2007, by age group and sex



Map 11: Notification rates for Q fever, Australia, 2007, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2007 and classified as 'other bacterial infections' in the NNDSS. A total of 1,762 notifications were included in this group in 2007, which accounted for 1.2% of all the notifications to NNDSS, a similar total and proportion as in 2006 (1,866 notifications and 1.3% of total).

Legionellosis

Legionellosis includes notifications of infections caused by all *Legionella* species. There were 307 notifications of legionellosis diagnosed in 2007, giving a national rate of 1.5 cases per 100,000 population. This was a decrease from the 350 cases reported in 2006. State and territory notification rates ranged from 0.6 cases per 100,000 population in Tasmania to 3.8 cases per 100,000 population in Western Australia. Compared with 2006, notification rates in 2007 increased in the Australian Capital Territory (1.2 cases per 100,000 population; 293% increase), New South Wales (1.5 cases per 100,000 population; 33% increase) and Queensland (1.2 cases per 100,000 population, 27% increase). A decrease in notification rates for 2007 compared with 2006 was seen in South Australia (1.1 cases per 100,000 population; 74% decrease), Victoria (0.8 cases per 100,000 population; 40% decrease) and Western Australia (3.8 cases per 100,000 population; 13% decrease). There was a negligible change in the notification rates for the Northern Territory and Tasmania from 2006 to 2007 (1.4 and 0.6 cases per 100,000 population, respectively).

In 2007, the highest number of legionellosis cases was diagnosed in June (36 cases, 12%) of all legionellosis notifications received and December (38 cases, 12%). These peaks were slightly later compared with previous years in which the highest numbers of notifications have generally been observed in autumn and spring months (Figure 63). Notifications of legionellosis by month of diagnosis have ranged between 14 and 43 cases between 2002 and 2007.

In 2007, males accounted for 196 (64%) of the 307 notified cases of legionellosis. There was 1 case of legionellosis (identified as *L. pneumophila*) in a child under the age of 5 years. Overall, the highest age specific notification rate was in the 80–84 years age group, with 6.5 cases per 100,000 population. The highest age specific notification rate among males was for the 80–84 years age group (10.4 cases per 100,000 population, 18 cases) and in females for the 65–69 years age group (3.9 cases per 100,000 population, 16 cases) (Figure 64).

Figure 63: Notifications of legionellosis, Australia, 2002 to 2007, by month of diagnosis

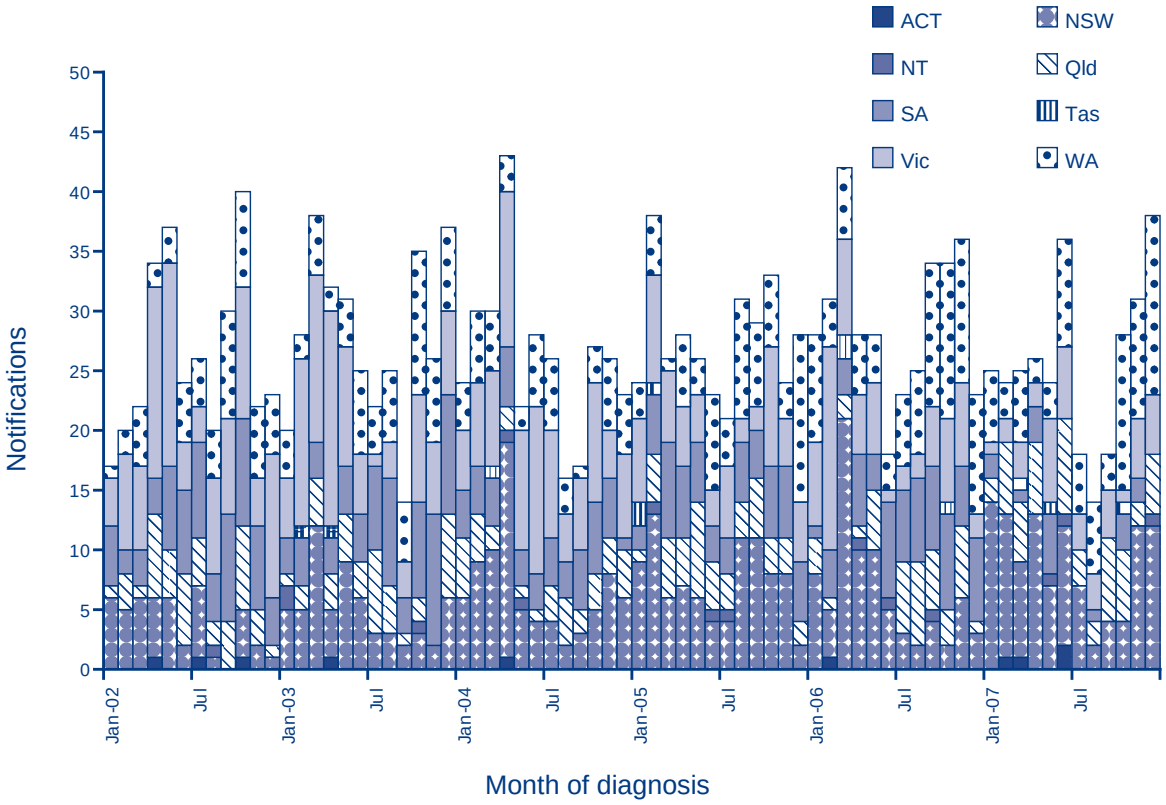
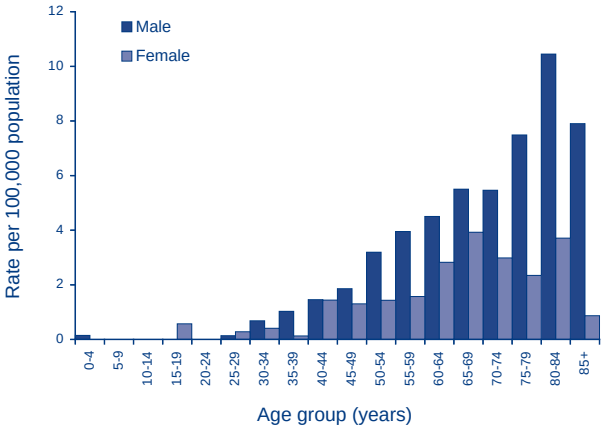


Figure 64: Notification rate for legionellosis, Australia, 2007, by age group and sex



Data on the causative species were available for 277 (90%) of the 307 legionellosis cases. Of these, 141 (51%) cases were identified as *L. pneumophila*, 134 (48%) were *L. longbeachae* and 2 (1%) cases were *L. micdadei* (Table 19).

Of the 141 *L. pneumophila* notifications, serogroup data were available for 75 cases (53%); 73 (97%) of those further typed were *L. pneumophila* serogroup 1.

There were significant differences in the geographic distribution of *L. longbeachae* and *L. pneumophila*, with *L. longbeachae* infections comprising the majority of legionellosis notifications from South Australia and Western Australia, while *L. pneumophila* were the most common infecting species in the eastern States (Queensland, New South Wales and Victoria).

Data on the death of legionellosis cases were available for 144 (47%) notifications. There were 5 reported deaths due to legionellosis in Australia in 2007, giving a case fatality rate of 3.5%. The age range for the deaths was between 58 and 89 years. The break down of deaths by jurisdiction and infecting *Legionella* species is shown in Table 20. There were 2 deaths associated with *L. longbeachae* infection (both in Western Australia), giving a case fatality rate of 1.5%. Three patients with *L. pneumophila* infections died (all from New South Wales), giving a case fatality rate of 2.1%. Case fatality rates may be inaccurate given the large proportion of cases without details of death outcomes.

The number of deaths decreased in 2007, relative to 2006, when there were 9 deaths reported. In 2006, data of death outcomes was reported for 66% of cases, this may in part account for the decrease in reported deaths. The number of deaths associated with legionellosis fell or remained constant in all jurisdictions except New South Wales where there were three more deaths in 2007 than in 2006.

Table 19: Notifications of legionellosis, Australia, 2007, by species and state or territory

Species	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Legionella longbeachae	0	29	2	10	14	2	6	71	134
Legionella micdadei	0	1	0	0	0	0	1	0	2
Legionella pneumophila	0	73	1	28	3	1	34	1	141
Unknown species	4	2	0	14	0	0	1	9	30
Total	4	105	3	52	17	3	42	81	307

Table 20: Deaths due to legionellosis by species, Australia, 2007, by state or territory

Species	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Legionella longbeachae	0	0	0	0	0	0	0	2	2
Legionella micdadei	0	0	0	0	0	0	0	0	0
Legionella pneumophila	0	3	0	0	0	0	0	0	3
Unknown species	0	0	0	0	0	0	0	0	0
Total deaths	0	3	0	0	0	0	0	2	5
Total cases	4	105	3	52	17	3	42	81	307
Number of cases with death status reported	0 (0%)	4 (4%)	3 (100%)	0 (0%)	11 (65%)	3 (100%)	42 (100%)	81 (100%)	144 (47%)

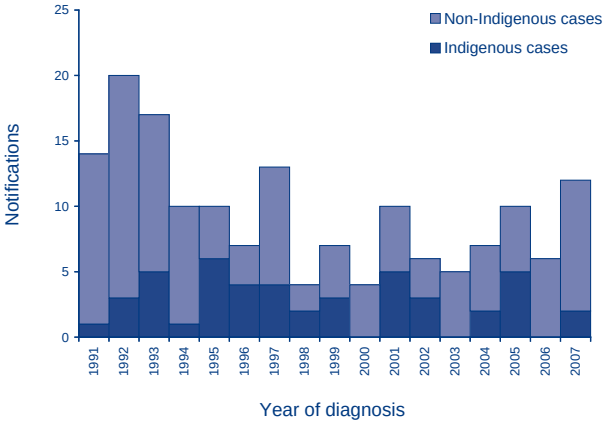
Two large outbreaks of *L. pneumophila* were reported in 2007. One was a cluster of 6 cases related to a contaminated cooling tower near Circular Quay in Sydney.[71] A second was a cluster of 9 cases linked to a cooling tower in the western suburbs in Melbourne.[72]

Leprosy

Leprosy is a chronic infection of the skin and peripheral nerves with the bacterium *Mycobacterium leprae*. Leprosy is a rare disease in Australia, with the majority of cases occurring among migrants to Australia from leprosy endemic countries and occasional cases from Indigenous communities. Trends in the number of leprosy notifications in Indigenous and non-Indigenous Australians and the overall rate are shown in Figure 65.

In 2007, 12 cases of leprosy were notified compared with 6 cases in 2006. There were 4 cases in New South Wales; 2 cases in each of South Australia, Victoria and Western Australia; and a single case in both Queensland and Tasmania. Two of the cases were in Indigenous Australians. The notification from Tasmania was detected in a recent arrival from Africa, who had spent the previous 4 years in Uganda.

Figure 65: Notifications of leprosy, Australia, 1991 to 2007, by indigenous status

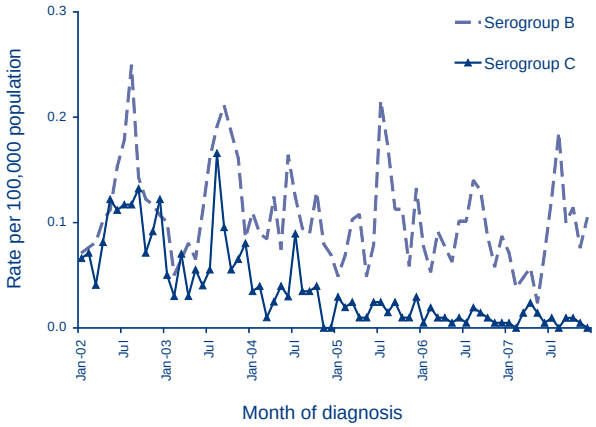


Invasive meningococcal disease

In 2007, there were 304 notifications of invasive meningococcal disease in Australia, a decrease from 317 in 2006. The total number of notifications in 2007 was the lowest since 1996. A decline or stabilisation in notifications was seen in all jurisdictions except New South Wales (112 cases, a 5% increase) and Queensland (75 cases, a 10% increase). The national notification rate in 2007 was 1.4 cases per 100,000 population. The highest rate of notification was reported from the Northern Territory (2.8 cases per 100,000 population; 6 cases).

In 2007, males accounted for 154 of the 304 notified cases of invasive meningococcal disease, giving a male to female ratio of 1:1. As observed in previous years, the largest number of cases, for serogroups B and C, were diagnosed in winter and spring (Figure 66). The majority of cases (285, 94%) were confirmed, through the isolation of *Neisseria meningitidis*, with an additional 19 cases (6%) notified by probable diagnosis, based on clinical symptoms only.

Figure 66: Trends in notification rates (annualised) of invasive meningococcal disease, Australia, 2002 to 2007, by serogroup and month of diagnosis



Of the 285 confirmed invasive meningococcal disease notifications in 2007, 255 (89%) were further typed. Of these, 213 (84%) were serogroup B, 20 (8%) were serogroup C and 22 (9%) were infections with serogroup W (9), serogroup X (1) and serogroup Y (12) (Table 21). In comparison, in 2006, 84% (265/317) of notified cases were serogrouped. Of these serogrouped notified cases 221 (83%) were serogroup B and 46 (15%) were serogroup C. Historically in Australia, serogroups B and C have been the major cause of invasive meningococcal disease.

Serogroup C infections were largely confined to Victoria, New South Wales and Queensland in 2007, similar to recent previous years when it has also been more predominant in the eastern states.

The highest age specific invasive meningococcal disease notification rate in 2007 was in children aged 0–4 years with a rate of 8.1 cases per 100,000 population (108 cases). Of the cases reported in this age group, 74% (80/108) were due to serogroup B infections, which represents the highest age specific

rate for serogroup B infection across all age groups, at a rate of 6.0 cases per 100,000 population. Figure 67 shows the decline in rates of serogroup B infections in most age groups over the period from 2002 to 2007, the greatest of which is in the 0–4 years age group, from 8.9 cases per 100,000 population in 2002.

There has been a marked decrease in notification rates for invasive meningococcal disease caused by serogroup C since 2003, when the National Meningococcal C Vaccination Program was introduced (Figure 68). Under the program, all children turning 12 months of age have been eligible to receive free meningococcal C vaccine since 2003. The program also provided free meningococcal C vaccine for all children and adolescents who were aged 1–19 years in 2003 until 30 June 2006.[13] The greatest decline in the rate of serogroup C disease since the introduction of the program is in the 15–19 years age group, from 4.9 cases per 100,000 population in 2002 (67 cases) to 0.1 cases per 100,000 population in 2007 (2 cases). The rates in the 20–24 years age group fell also from 2.5 cases per 100,000 population (33 cases) to 0.2 cases per 100,000 population (3 cases) over the same period. Rates in the 0–4 years age group fell from 2 cases per 100,000 population in 2002 (26 cases) to 0.3 cases per 100,000 population (4 cases) in 2007.

Death data for meningococcal cases were available for 123 (40%) notifications. There were 9 deaths due to meningococcal disease in 2007, corresponding to a case fatality rate of 30%. The break down of deaths by state or territory and serogroup is shown in Table 22. There were 5 deaths due to serogroup B (case fatality rate of 2.3%) and 3 deaths due to serogroup C disease (case fatality rate of 15.0%). Overall there was a decrease in deaths of meningococcal cases from 12 deaths in 2006 (death data was provided for 45% of cases in 2006).

Laboratory based meningococcal surveillance

The Australian Meningococcal Surveillance Programme (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using agreed standard methodology to determine the phenotype (serogroup,

Figure 67: Notification rate for serogroup B invasive meningococcal disease, Australia, 2002 to 2007, by age group

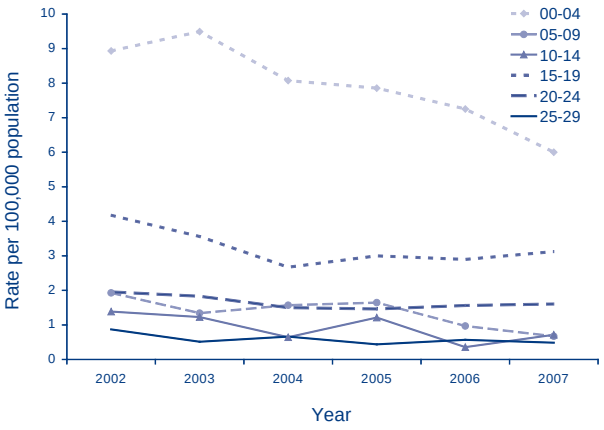


Figure 68: Notification rate for serogroup C invasive meningococcal disease infection, Australia, 2002 to 2007, by age group

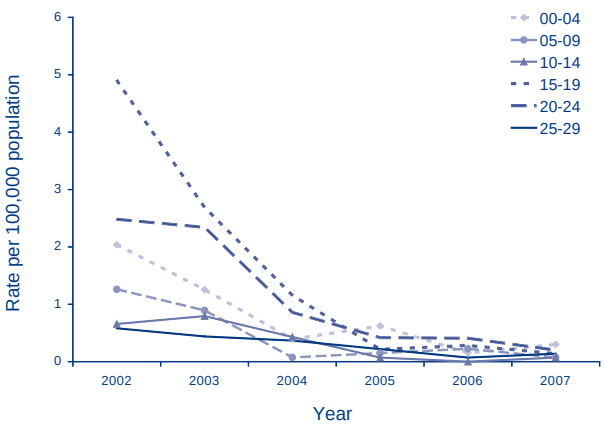


Table 21: Notifications of invasive meningococcal disease, Australia, 2007, by serogroup and state or territory

Species	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Serogroup B	3	77	4	59	0	3	48	19	213
Serogroup C	0	10	2	6	0	0	2	0	20
Other serogroups*	0	7	0	6	0	2	7	0	22
Unknown serogroup	0	18	0	4	15	0	11	1	49
Total	3	112	6	75	15	5	68	20	304

* Serogroup W (9), serogroup X (1) and serogroup Y (12).

Table 22: Deaths due to meningococcal infection, Australia, 2007, by serogroup and state or territory

Species	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Serogroup B	0	0	0	4	0	0	1	0	5
Serogroup C	0	3	0	0	0	0	0	0	3
Serogroup W135	0	0	0	0	0	0	0	0	0
Serogroup unknown	0	1	0	0	0	0	0	0	1
Total deaths	0	4	0	4	0	0	1	0	9
Total cases	3	112	6	75	15	5	68	20	304
Number of cases with death status reported	0 (0%)	4 (4%)	6 (100%)	5 (7%)	15 (100%)	5 (100%)	68 (100%)	20 (100%)	123 (40%)

Laboratory based meningococcal surveillance

serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics. The results of laboratory surveillance in 2007 have recently been published.[73]

In 2007, a total of 242 laboratory confirmed cases of invasive meningococcal disease were reported by the AMSP. Consistent with the NNDSS data, the AMSP reported that 85% (192 cases) were identified as serogroup B and 6.2% (14 cases) were serogroup C. No evidence of meningococcal capsular 'switching' was detected. About two-thirds of all isolates showed decreased susceptibility to penicillin (MIC 0.06–0.5 mg/L). All isolates remained susceptible to rifampicin. One serogroup B isolate had decreased susceptibility to ciprofloxacin.

Tuberculosis

While Australia has one of the lowest rates of tuberculosis (TB) in the world, the disease remains a public health problem in those born overseas and for Indigenous Australians. In 2007, 1,139 TB notifications were received by NNDSS, a rate of 5.4 cases per 100,000 population. In 2006, there were 1,193 cases notified nationally, a rate of 5.8 cases per 100,000 population. The notification rate for TB was higher than the national average in the Northern Territory (24.7 cases per 100,000 population; 53 cases), while the lowest rate occurred in Tasmania (1.2 cases per 100,000 population; 6 cases).

Further details and analysis of TB notifications in 2007 can be found in the TB annual report to be published in the next edition of *CDI*.

Acknowledgements

The authors wish to thank the following people for their contribution to this report.

Alison Milton and Katrina Choi, Office of Health Protection, Department of Health and Ageing, Australian Capital Territory
Mohammad Habib and Kate Ward, NSW Health Department
Rebecca Hundy, ACT Health
Carolien Giele and Gary Dowse, Western Australia Department of Health
Joy Copland, Department of Health, South Australia
David Coleman, Tasmanian Department of Health and Human Services
Russell Stafford, Queensland Health

Appendices

Appendix 1: Mid-year estimate of Australian population, 2007, by state or territory

	State or territory								Aus
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Male	168,286	3,411,349	111,564	2,087,631	782,397	243,329	2,574,901	1,063,849	10,443,306
Female	171,475	3,476,665	103,365	2,093,800	801,800	250,042	2,629,925	1,042,270	10,569,342
Total	339,761	6,888,014	214,929	4,181,431	1,584,197	493,371	5,204,826	2,106,119	21,012,648

Appendix 2: Mid-year estimate of Australian population, 2007, by state or territory and age group

Age group	State or territory								Aus
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
0-4	21,520	439,789	17,796	273,762	91,141	30,826	323,304	135,058	1,333,196
5-9	20,394	439,395	17,294	279,148	95,059	31,554	321,503	136,950	1,341,297
10-14	21,378	453,954	16,675	292,031	101,242	34,139	336,480	144,378	1,400,277
15-19	24,582	464,616	16,109	292,467	106,055	34,020	352,964	149,326	1,440,139
20-24	29,940	475,958	17,577	300,294	110,292	31,330	375,415	153,180	1,493,986
25-29	28,475	476,275	18,205	284,620	99,082	27,850	366,075	143,719	1,444,301
30-34	26,090	488,027	18,043	290,632	100,396	28,907	370,569	145,398	1,468,062
35-39	26,224	505,006	18,020	311,163	112,606	33,811	398,081	159,872	1,564,783
40-44	24,548	493,360	16,356	301,893	113,996	34,164	378,267	157,233	1,519,817
45-49	24,741	497,145	15,504	301,625	116,684	37,270	373,230	155,505	1,521,704
50-54	22,597	451,038	13,775	273,427	108,731	34,981	339,190	143,217	1,386,956
55-59	20,494	411,232	11,292	254,406	101,507	32,915	308,318	128,738	1,268,902
60-64	15,596	350,168	7,746	214,454	86,285	28,585	258,711	102,723	1,064,268
65-69	10,589	268,492	4,790	158,228	65,666	21,673	200,574	77,505	807,517
70-74	7,838	220,260	2,563	120,039	54,739	17,149	164,073	59,602	646,263
75-79	6,218	190,522	1,674	99,364	49,065	14,404	141,800	49,147	552,194
80-84	4,837	143,766	911	72,712	38,991	10,846	107,534	35,264	414,861
85+	3,700	119,011	599	61,166	32,660	8,947	88,738	29,304	344,125
Total	339,761	6,888,014	214,929	4,181,431	1,584,197	493,371	5,204,826	2,106,119	21,012,648

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2007, by notifiable disease

Disease	Aboriginal but not Torres Strait Islander origin	Torres Strait Islander but not Aboriginal origin	Aboriginal and Torres Strait Islander origin	Not indigenous	Not stated (blank/ missing/null)	Total	Complete	Number complete	Number incomplete
Anthrax				1	-	1	100	1	-
Botulism				1	-	1	100	1	-
Donovanosis	2			1	-	3	100	3	-
Kunjin virus infection				1	-	1	100	1	-
Poliomyelitis				1	-	1	100	1	-
Rubella - congenital				2	-	2	100	2	-
Typhoid				87	3	90	97	87	3
Tuberculosis	23	7		1,066	43	1,139	96	1,096	43
Meningococcal infection	26	6		260	12	304	96	292	12
Haemophilus influenzae type b	7			9	1	17	94	16	1
Syphilis < 2 years duration	186	4	2	1,107	82	1,381	94	1,299	82
Leprosy	2			9	1	12	92	11	1
Measles	1			10	1	12	92	11	1
Haemolytic uraemic syndrome				17	2	19	89	17	2
Legionellosis	7			254	46	307	85	261	46
Varicella zoster (chickenpox)	157	12	3	1,224	255	1,651	85	1,396	255
Hepatitis E				15	3	18	83	15	3
Hepatitis C (incident)	27			268	60	355	83	295	60
Hepatitis A				136	29	165	82	136	29
Listeriosis				41	9	50	82	41	9
Varicella zoster (shingles)	53	9	2	1,186	297	1,547	81	1,250	297
Ornithosis				74	18	92	80	74	18
Pneumococcal disease (invasive)	146	8	4	1,004	313	1,475	79	1,162	313
Hepatitis B (incident)	14	2		207	64	287	78	223	64
Shigellosis	266	1	3	190	137	597	77	460	137
Malaria	2	1		430	134	567	76	433	134
Leptospirosis	6	1	1	72	26	106	75	80	26
Hepatitis D	2			23	9	34	74	25	9

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2007, by notifiable disease, *continued*

Disease	Aboriginal but not Torres Strait Islander origin	Torres Strait Islander but not Aboriginal origin	Aboriginal and Torres Strait Islander origin	Not indigenous	Not stated (blank/ missing/null)	Total	Complete	Number complete	Number incomplete
STEC, VTEC	3			75	29	107	73	78	29
Rubella				26	10	36	72	26	10
Gonococcal infection	3,323	156	39	1,810	2,277	7,605	70	5,328	2,277
Brucellosis	1			25	12	38	68	26	12
Mumps	125		1	270	183	579	68	396	183
Dengue virus infection	3			210	101	314	68	213	101
Q fever	13	2		288	147	450	67	303	147
Cholera				2	1	3	67	2	1
Tetanus				2	1	3	67	2	1
Syphilis > 2 years or unspecified duration	319	18	3	712	566	1,618	65	1,052	566
Syphilis - congenital	3			2	3	8	63	5	3
Cryptosporidiosis	186	2	1	1,471	1,150	2,810	59	1,660	1,150
Arbovirus infection (NEC)				11	11	22	50	11	11
Salmonellosis	436	16	12	4,126	4,894	9,484	48	4,590	4,894
Campylobacteriosis	185	5	4	7,727	9,063	16,984	47	7,921	9,063
Hepatitis B (unspecified)	296	39	5	2,705	3,872	6,917	44	3,045	3,872
Chlamydial infection	4,339	562	146	17,720	29,092	51,859	44	22,767	29,092
Influenza (laboratory confirmed)	270	18	18	4,124	5,973	10,403	43	4,430	5,973
Hepatitis C (unspecified)	491	4	13	3,729	7,740	11,977	35	4,237	7,740
Pertussis	35	4	1	1,832	3,452	5,324	35	1,872	3,452
Ross River virus infection	64	6	3	1,252	2,878	4,203	32	1,325	2,878
Varicella zoster (unspecified)	57	6	2	1,099	3,134	4,298	27	1,164	3,134
Barmah Forest virus infection	18	4		413	1,281	1,716	25	435	1,281

Abbreviations

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
APSU	Australian Paediatric Surveillance Unit
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
DENV	dengue virus
DHS	Department of Human Services (Victoria)
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HPV	human papilloma virus
HUS	haemolytic uraemic syndrome
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
PEC	Poliovirus Expert Committee
PCR	polymerase chain reaction
PPNG	penicillinase-producing' *Neisseria gonorrhoeae*
QRNG	quinolone resistant *Neisseria gonorrhoeae*
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
WPV	wild-type polio virus

Author details

Coordinating author: Conan Liu
Data management: Stefan Stirzaker
Mapping: Dougald Knuckey
Bloodborne diseases: Kate Robinson
Gastrointestinal diseases: Katrina Knope
Quarantinable diseases: Gerard Fitzsimmons, Jennifer Wall, Katrina Knope
Sexually transmissible infections: Kate Robinson
Vaccine preventable diseases: Katrina Roper, Nicolee Martin, Anna Reynolds, Rhonda Owen, Aurysia Hii, Christina Barry
Vectorborne diseases: Phil Wright, Gerard Fitzsimmons
Zoonoses: Gerard Fitzsimmons, Lance Sanders
Other bacterial infections: Christina Barry, James Fielding

With contributions from:

National organisations

Communicable Diseases Network Australia and subcommittees
Australian Childhood Immunisation Register
Australian Gonococcal Surveillance Programme
Australian Meningococcal Surveillance Programme
Australian Sentinel Practice Research Network
Australian Quarantine Inspection Service
National Centre in HIV Epidemiology and Clinical Research
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 Unit, ACT Health, Australian Capital Territory
Communicable Diseases Surveillance and Control Unit, NSW Health Department, New South Wales
Centre for Disease Control, Northern Territory Department of Health and Community Services, Northern Territory
Communicable Diseases Unit, Queensland Health, Queensland
Communicable Disease Control Branch, South Australian Department of Health, South Australia
Communicable Diseases Prevention Unit, Department of Health and Human Services, Tasmania
Communicable Diseases Section, Department of Human Services, Victoria
Communicable Diseases Control Directorate, Department of Health, Western Australia

Corresponding author: Mr Conan Liu, Vaccine Preventable Diseases Surveillance Section, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 4898, MDP 14, CANBERRA, ACT 2601. Telephone: +61 2 6289 2712. Facsimile: +61 2 6289 2600. Email: conan.liu@health.gov.au

References

1. National Health Security Act, No 174. 2007. Available from: http://www.comlaw.gov.au/ComLaw/Legislation/Act1.nsf/0/A005BA0145A00248CA25736A00126AA5?OpenDocument Accessed February 2009.
2. National Notifiable Diseases List. 2008. Available from: http://www.comlaw.gov.au/ComLaw/legislation/LegislativeInstrument1.nsf/0/7162D634C6DD1BAACA25740B0079D6B8?OpenDocument Accessed February 2009.
3. National Health Security Agreement. 2008. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-nhs-agreement.htm Accessed February 2009.
4. National Centre in HIV Epidemiology and Clinical Research. *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report, 2008*: National Centre in HIV Epidemiology and Clinical Research, The University of New South Wales, Sydney; 2008.
5. Klug GM, Boyd A, Lewis V, McGlade AR, Roberts H, Douglass SL, *et al*. Surveillance of Cruetzfeldt-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 Accessed February 2009.
7. Australian Bureau of Statistics. Population by age and sex, Australian states and territories, Table 9, Estimated Resident Population By Single Year of Age, Australia. Canberra: Australian Bureau of Statistics; 2008. Report No.: 3201.0
8. Australian Bureau of Statistics. Statistical Geography Volume 1 – Australian Standard Geographical Classification. Canberra: Australian Bureau of Statistics; 2006. Report No.: 1216.0.
9. Australian Bureau of Statistics. Population by age and sex, regions of Australia. Canberra: Australian Bureau of Statistics; 2007. Report No: 3235.0.
10. Australian Bureau of Statistics. Postal Area Concordances, August 2006 Canberra: Australian Bureau of Statistics; 2006. Report No.: 2905.0.55.001.
11. Oxenford C. Current practices surrounding reporting of notifiable diseases by laboratories to State and Territory Health Departments, Bound volume, Masters of Applied Epidemiology Scholar; 2005.
12. Australian Institute of Health and Welfare. National Health Data Dictionary 13.3; 2008.
13. Australian Technical Advisory Group on Immunisation. *The Australian Immunisation Handbook* 9th edn. Canberra, Australia: Department of Health and Ageing; 2008.
14. OzFoodNet Working Group. 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.
15. Stephens N, Stafford R, Fullerton K. Risk factors for sporadic *Campylobacter* infection in children aged 0–4 years in Australia: A multi-centre prospective case-control study. In; Submitted to Foodborne Pathogens and Disease.
16. Australian Bureau of Statistics. Experimental Estimates of Aboriginal and Torres Strait Islander Australians, June 2006, ABS. cat no 3238.0.55.001. Canberra: ABS; 2007.
17. *Control of Communicable Diseases Manual*. 18 edn. Washington: American Public Health Association, USA; 2004.
18. Lewis HC, Kirk M, Ethelberg S, Stafford R, Olsen K, Nielsen EM, *et al*. Outbreaks of shigellosis in Denmark and Australia associated with imported baby corn, August 2007—final summary. *Euro Surveill* 2007;12(10):E071004 071002.
19. 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.

20. Cumpston JHL. *Health and disease in Australia*. Canberra: Australian Government Publishing Service; 1989.
21. 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.
22. World Health Organization. The Global Eradication of Smallpox: Final Report of the Global Commission for the Certification of Smallpox Eradications, Geneva, December 1979. Geneva; 1980.
23. 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.
24. World Health Organization. Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to the World Health Organization. 2009. Available from: http://www.who.int/csr/disease/avian_influenza/country/cases_table_2009_02_05/en/index.html Accessed on 9 February 2009.
25. Australian Government Department of Health and Ageing. Factsheets: Viral haemorrhagic fever. 2009. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-communic-factsheets-vhf.htm Accessed on 9 February 2009.
26. Chen M, Fairley C, 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.
27. 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.
28. Australian Institute of Health and Welfare. Age-standardised rate – Identifying and definitional attributes. 2005. Available from: http://meteor.aihw.gov.au/content/index.phtml/itemId/327276 Accessed on 12 February 2009.
29. 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.
30. Queensland Health. Queensland HIV, Hepatitis C and Sexually Transmissible Infections Strategy: 2005–2011. Queensland Health; 2005.
31. Australian Government Department of Health and Ageing. National Sexually Transmissible Infections Strategy: 2005–2008. Canberra: Commonwealth of Australia; 2005.
32. Chen M, Donovan B. Genital *Chlamydia trachomatis* infection in Australia: epidemiology and clinical implications. *Sex Health* 2004;1(4):189–196.
33. Bowden FJ. Donovanosis in Australia: going, going. *Sex Transm Infect* 2005;81(5):365–366.
34. The Australian Gonococcal Surveillance Programme. Annual report of the Australian Gonococcal Surveillance Programme, 2007. *Commun Dis Intell* 2008;32(2):227–231.
35. 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.
36. Jin F, Prestage G, Zablotska I, Rawstorne P, Kippax S, Donovan T, *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):387–399.
37. Fairley C, Hocking J, Medland N. Syphilis: back on the rise, but not unstoppable. *Med J Aust* 2005;183(4):172–173.
38. Kirkwood C, Cannan D, Boniface K, Bishop R, Barnes G, Australian Rotavirus Surveillance Group. Australian Rotavirus Surveillance Program annual report, 2007/2008. *Commun Dis Intell* 2008;32(4):425–429.
39. Gidding HF, Backhouse JL, Burgess MA, Gilbert GL. Immunity to diphtheria and tetanus in Australia: a national serosurvey. *Med J Aust* 2005;183:301–304.
40. World Health Organization. Immunization surveillance, assessment and monitoring. Diphtheria reported cases. 2009. Available from: http://www.who.int/immunization_monitoring/en/globalsummary/timeseries/tsincidencedip.htm Accessed on February 2009.
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:S1–152.
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. Owen R, Barr IG, Pengilley A, Liu C, Paterson B, Kaczmarek M. Annual report of the National Influenza Surveillance Scheme, 2007. *Commun Dis Intell* 2008;32(2):208–226.
44. Walcott J, Fearnley E, CDC Darwin. Immunisation coverage rates in the 10–49 year age group in a remote Indigenous community in the Northern Territory experiencing a mumps outbreak—a clinical audit. *The Northern Territory Disease Control Bulletin* 2007;14(3):1–4.
45. Australian Government Department of Health and Ageing. Communicable diseases surveillance: Highlights for 4th quarter, 2007. Mumps. *Commun Dis Intell* 2008;32(1):106.
46. 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.
47. World Health Organization. Global Polio Eradication Initiative annual report. Geneva: World Health Organization; 2007.
48. 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;28:69–73.
49. 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;93:1274–1276.
50. 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;45:435–438.
51. Hunt JM, Lumley J. Top end rural and remote Indigenous women: an Australian population group vulnerable to rubella. *Commun Dis Intell* 2004;28:499–503.
52. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.

53. 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.

54. 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.

55. Liu C, Begg K, Johansen C, Whelan P, Kurucz N, Melville L, et al. Communicable Diseases Network Australia National Arbovirus and Malaria Advisory Committee annual report, 2006–07. *Commun Dis Intell* 2008;32(1):31–47.

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

57. Liu C, Broom AK, Kurucz N, Whelan PI. Communicable Diseases Network Australia: National Arbovirus and Malaria Advisory Committee annual report, 2004–05. *Commun Dis Intell* 2005;29(4):341–357.

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

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

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

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

62. Animal Health Australia. Animal Health in Australia 2007. Canberra; 2008.

63. Fielding J. Zoonoses: Anthrax. *Vic Infect Dis Bull* 2007(10):47.

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

65. Field H. The ecology of Hendra virus and Australian bat lyssavirus. 2004. Available from: http://espace.library.uq.edu.au/eserv.php?pid=UQ:13859&dsID=field_thesis_05.pdf Accessed on 01/08/07.

66. Sweeny AL, Beard FH. Queensland Health Notifiable Diseases Report 2002–2006. Brisbane: Communicable Diseases Branch, Brisbane: Queensland Health.; 2009 (In press).

67. Victorian Department of Human Services. Blue Book. Revised Edition 2005. Available from: http://www.health.vic.gov.au/ideas/bluebook Accessed on 15 August 2007.

68. NSW Department of Health. Communicable diseases report. *NSW Public Health Bulletin* 2002;14(3):63–67.

69. Australian Government Department of Health and Ageing. Q Fever Management Program. 2008. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/q-fever-man Accessed on 20 February 2009.

70. Marmion B. Q fever: the long journey to control by vaccination. *Med J Aust* 2007;186(4):164–166.

71. NSW Department of Health. Communicable Diseases Report. *NSW Public Health Bulletin* 2007;18(7–8).

72. Victorian Department of Human Services. Surveillance report. *Victorian Infectious Diseases Bulletin* 2007:10.

73. The Australian Meningococcal Surveillance Programme – Amended. Annual report of the Australian Meningococcal Surveillance Programme, 2007. *Commun Dis Intell* 2009;33(1):9.

Arboviral diseases and malaria in Australia, 2007/08: Annual report of the National Arbovirus and Malaria Advisory Committee

Gerard J Fitzsimmons, Phil Wright, Cheryl A Johansen, Peter I Whelan and the National Arbovirus and Malaria Advisory Committee

Abstract

The National Notifiable Diseases Surveillance System (NNDSS) received 8,671 notifications of diseases transmitted by mosquitoes in Australia for the season 1 July 2007 to 30 June 2008. This represented a 39% increase from the annual average of 6,259 notifications for the previous 5 years. The alphaviruses, Barmah Forest and Ross River, accounted for 7,760 (89%) of these notifications during the 2007/08 season and represents an increase when compared with the mean of the past 5 seasons. Detection of flavivirus seroconversions in sentinel chicken flocks across Australia provides an early warning of increased levels of Murray Valley encephalitis virus (MVEV) and Kunjin virus activity. Unusual MVEV activity in mosquitoes and sentinel chicken flocks was reported in south-east Australia during the 2007/08 season. Two cases of MVEV were reported, one each from New South Wales and Western Australia. There were 365 notifications of dengue virus infection that were acquired overseas compared with an average of 164 overseas-acquired dengue cases per annum reported to NNDSS over the 5 seasons from 2002/03 to 2006/07. There were no reports of locally-acquired malaria notified in Australia and 505 notified cases of overseas-acquired malaria during the season 2007/08. The exotic dengue vector *Aedes aegypti* was first detected on Groote Eylandt, Northern Territory in October 2006 and led to a 2-year *Ae. aegypti* eradication project. The successful eradication of *Ae. aegypti* from Groote Eylandt was officially announced in May 2008. The success of the program was due to the selection of appropriate chemicals that were successful in treating mosquito adults, larvae and egg infested receptacles. This annual report presents information on diseases transmitted by mosquitoes in Australia and notified to NNDSS. *Commun Dis Intell* 2009;33:155–169.

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 nationally notifiable mosquito-borne disease in Australia for the season 1 July 2007 to 30 June 2008. It includes those diseases caused by alphaviruses (Barmah Forest and Ross River), flaviviruses (dengue, Murray Valley encephalitis, Kunjin, Japanese encephalitis and yellow fever) and malaria. Human cases of arbovirus infection and malaria are monitored using the National Notifiable Diseases Surveillance System (NNDSS).

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. The 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. Currently, NAMAC provides expert technical advice on arboviruses and malaria to the Australian Health Protection Committee through the Communicable Diseases Network of Australia. It also assists in the detection, management and control of real or potential outbreaks of arboviral and malarial disease. Members of the committee have expertise in disease surveillance, virology, vector control and quarantine, and represent agencies with a substantial interest in this area.

Methods

All Australian states and territories require doctors and/or pathology laboratories to notify cases of infectious diseases that are important to public health. State and territory health departments transfer these notifications regularly to NNDSS. 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 in November 2008 and analysed by date of diagnosis. The dataset represents a 'snap shot', and numbers in this report may vary slightly from those reported in other

NNDSS sources. Detailed notes on the interpretation of NNDSS and case definitions are available in the 2006 NNDSS annual report.[1] Case definitions are also available from http://www.health.gov.au/casedefinitions. The report includes information on the following diseases transmitted by mosquitoes:

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

To compare notifications in 2007/08 to historical totals, crude numbers and rates of notification were compared either to the mean of the previous 5 years or to data from the previous year. The Australian Bureau of Statistics (ABS) estimated resident populations for Australia and each state or territory at June 2007 was used to calculate rates of notification.

Additional information was available from a survey conducted with some state and territory public health surveillance managers. The survey sought to determine the place of acquisition for overseas-acquired cases of dengue virus infections.

Maps were produced based on residential postcode and notifications were summed for their respective area (Statistical Division or NT Statistical Sub-Division). Rates were calculated using ABS estimated populations for Australia as at June 2007. Total notified cases per area and ranges for the disease rate are represented on each map. Detailed notes on the production of maps in this report are available from the 2007 NNDSS annual report.

Results

During the 2007/08 season, there were 8,671 notifications of diseases transmitted by mosquitoes. This represented a 39% increase from the average of 6,259 notifications for the previous 5 years. A summary of the number and rates of these mosquito-borne diseases is shown in Table 1. There were no reported cases of Japanese encephalitis or yellow fever during the season.

Alphaviruses

Alphaviruses are single-stranded RNA viruses, members of which can cause disease epidemics characterised by fever, rash and polyarthritis. There are a variety of mosquito vectors for Barmah Forest virus (BFV) and Ross River virus (RRV), which breed and transmit viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[2]

During the 2007/08 season, there were 7,760 notifications of alphaviruses (BFV and RRV) of which RRV infections accounted for 74% (n=5,747).

Barmah Forest virus infections

There were 2,013 notifications of BFV infections notified to NNDSS during the 2007/08 season. Fifty-eight per cent of BFV notifications were reported from Queensland (n=1,160) and 27% from New South Wales (n=550). The annual notification rate for the 2007/08 season (Table 1) was 9.6 cases per 100,000 population, which was a 34% increase over the mean rate for the previous 5 years (7.2 per 100,000 population). The highest age specific rate for males was 21 per 100,000 population; reported in the 55–59 year age group and the highest rate for females was 17 per 100,000 population; reported in the 45–49 year age group. A similar number of males and females with BFV were notified to NNDSS (M=1,031:F=981).

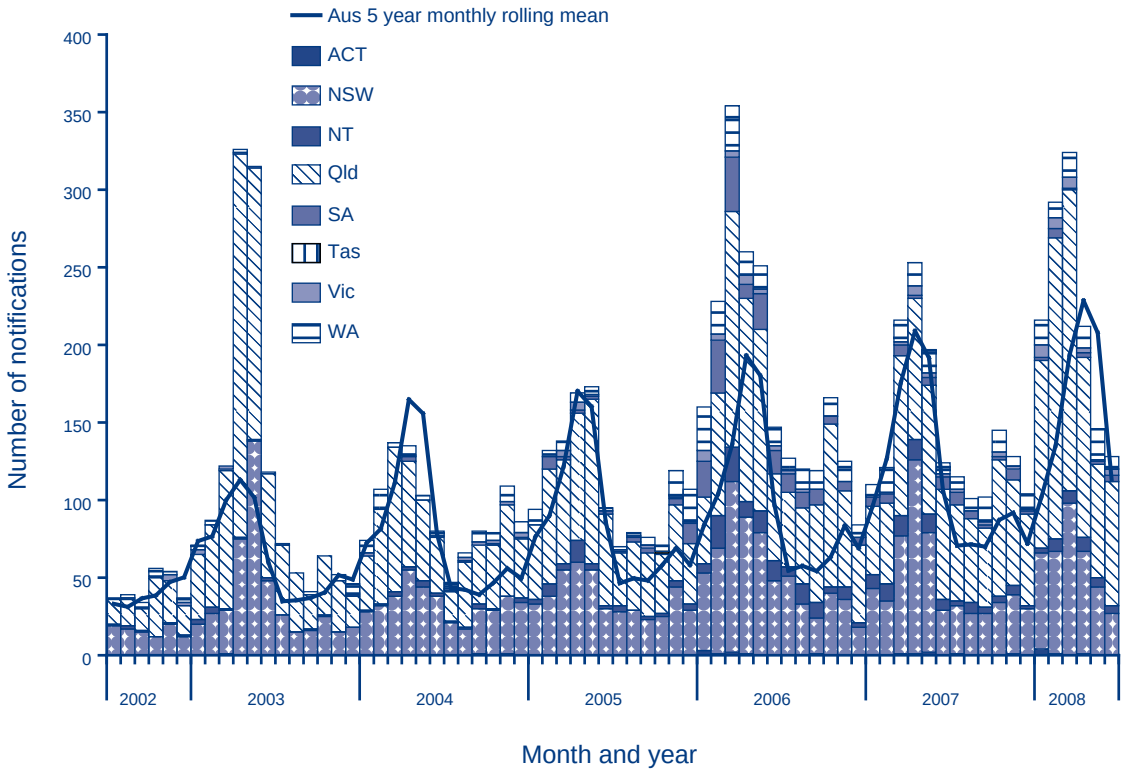
Figure 1 shows that as in previous years, there was a marked seasonal trend with the highest number of notifications being diagnosed in February (292) and March (324). The number of notifications per month exceeded the 5-year rolling mean from July 2007 to March 2008.

The highest rates of BFV notifications were reported by the Northern Territory (30 cases per 100,000 population), and Queensland (18.8 cases per 100,000 population). Cases were reported in all jurisdictions except for Tasmania. All jurisdictions apart from South Australia and Tasmania reported an increase in notifications when compared with the previous 5-year period. Queensland reported 1,160 notifications compared with a 5-year average of 732 cases. The Australian Capital Territory reported 8 notifications compared with a 5-year average of 3 cases. Notification rates for BFV by geographic location are shown in Map 1. These locations represent the place of residence of a notified case and not necessarily the place of acquisition of infection. The highest regional BFV notification rate was reported in the Central West Statistical division of Queensland (70 cases per 100,000 population). Six of the top 10 rates of BFV notification by region in Australia occurred in Queensland in the 2007/08 season.

Ross River virus infections

There were 5,747 cases of RRV infection notified during the season 2007/08 (Table 1). The annual notification rate for the 2007/08 season was 27.3 cases per 100,000 population, which was a 47% increase over the mean rate of the previous 5 years (18.6 per 100,000 population). Fifty-one per cent of RRV notifications were reported from Queensland (n=2,906)

Figure 1: Number of notified cases of Barmah Forest virus infection, Australia, 1 July 2002 to 30 June 2008, by date of diagnosis and state or territory



Map 1: Number of notified cases and rate of Barmah Forest virus infection, Australia, 1 July 2007 to 30 June 2008, by Statistical Division

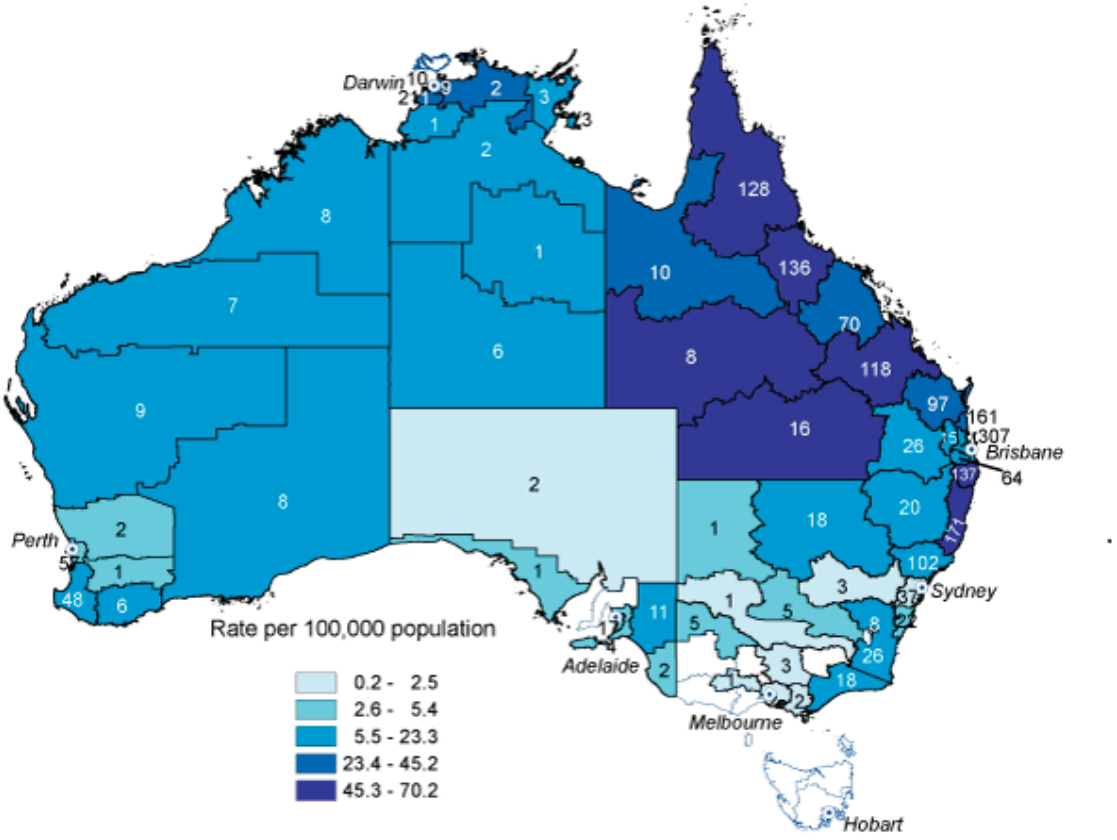


Table 1: Number of notified cases, rate and 5-year mean rate per 100,000 population of mosquito-borne diseases, Australia, 2002/03 to 2007/08, by date of diagnosis, disease and state or territory

Disease		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Alphaviruses										
Barmah Forest virus infection	Notified cases 2007/08	8	550	65	1,160	44	0	40	146	2,013
	Rate, 07/08	2.4	8.0	30.2	27.7	2.8	0.0	0.8	6.9	9.6
	Mean rate, 2002/03–06/07	0.9	7.2	28.3	18.8	3.5	0.0	0.4	4.6	7.2
Ross River virus infection	Notified cases 2007/08	20	1,220	255	2,906	196	77	237	836	5,747
	Rate, 07/08	5.9	17.7	118.6	69.5	12.4	15.6	4.6	39.7	27.3
	Mean rate, 2002/03–06/07	1.7	10.4	103.3	50.0	8.7	2.0	1.9	32.8	18.6
Flaviviruses										
Arbovirus infection (NEC*)	Notified cases 2007/08	0	0	0	13	0	0	3	0	16
	Rate, 07/08	0.0	0.0	0.0	0.3	0.0	0.0	0.1	0.0	0.1
	Mean rate, 2002/03–06/07	0.0	0.1	0.0	0.8	0.0	0.0	0.1	0.0	0.2
Dengue virus infection	Notified cases 2007/08	4	104	26	105	35	4	14	95	387
	Rate, 07/08	1.2	1.5	12.1	2.5	2.2	0.8	0.3	4.5	1.8
	Mean rate, 2002/03–06/07	1.5	0.8	8.6	6.7	0.5	0.1	0.2	0.9	1.8
Japanese encephalitis virus infection	Notified cases 2007/08	0	0	0	0	0	0	0	0	0
	Rate, 07/08	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Mean rate, 2002/03–06/07	0.0	0.0	0.0	0.01	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	Notified cases 2007/08	0	0	0	0	0	0	1	0	1
	Rate, 07/08	0.0	0.0	0.0	0.0	0.0	0.0	0.02	0.0	0.0
	Mean rate, 2002/03–06/07	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Murray Valley encephalitis virus infection	Notified cases 2007/08	0	1	0	0	0	0	0	1	2
	Rate, 07/08	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Mean rate, 2002/03–06/07	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0
Other										
Malaria	Notified cases 2007/08	14	111	23	165	18	9	88	77	505
	Rate, 07/08	4.1	1.6	10.7	3.9	1.1	1.8	1.7	3.7	2.4
	Mean rate, 2002/03–06/07	4.3	1.9	21.6	6.3	1.9	4.5	1.8	3.5	3.2

The Table does not include 2 chikungunya virus infections reported to the National Notifiable Diseases Surveillance System during the 2007/08 season.

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

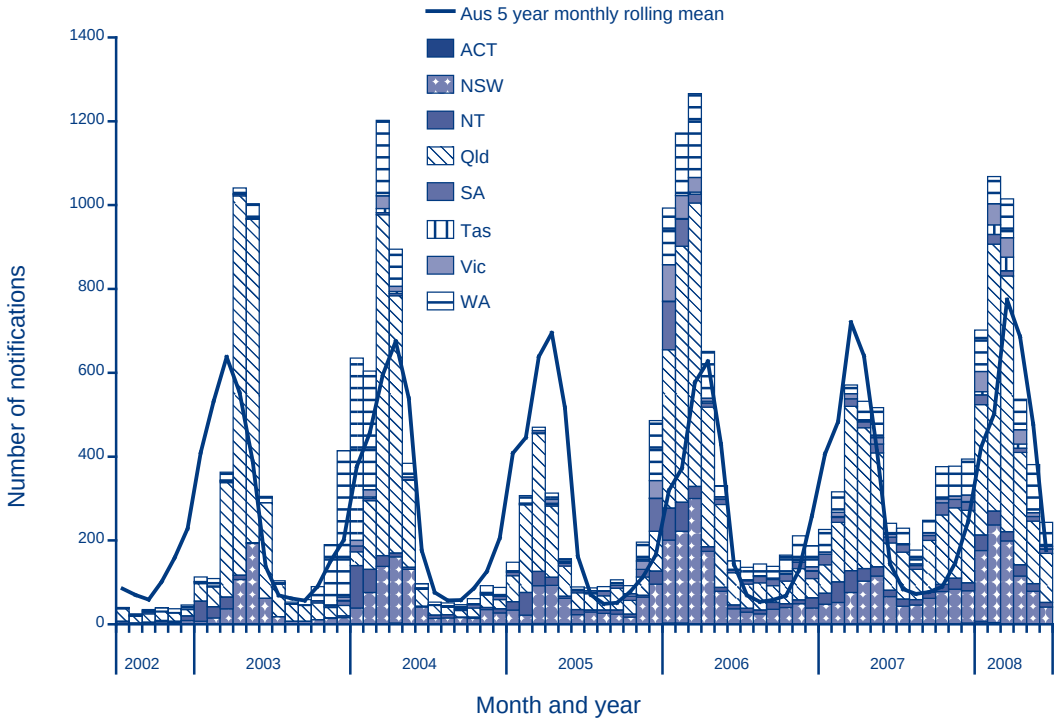
NEC Not elsewhere classified.

and 21% from New South Wales (n=1,220). The highest age and sex specific rate was reported in the 40–44 year age group (males:49 per 100,000 population and females:48 per 100,000 population). A similar number of males and females with RRV were notified to NNDSS (M=2,733:F=3,014). Figure 2 shows that as in previous years, there was a marked seasonal trend with the highest number of notifications being diagnosed in February (n=1,068) and March (n=1,015). The number of notifications per month exceeded the 5-year rolling mean from July 2007 to March 2008.

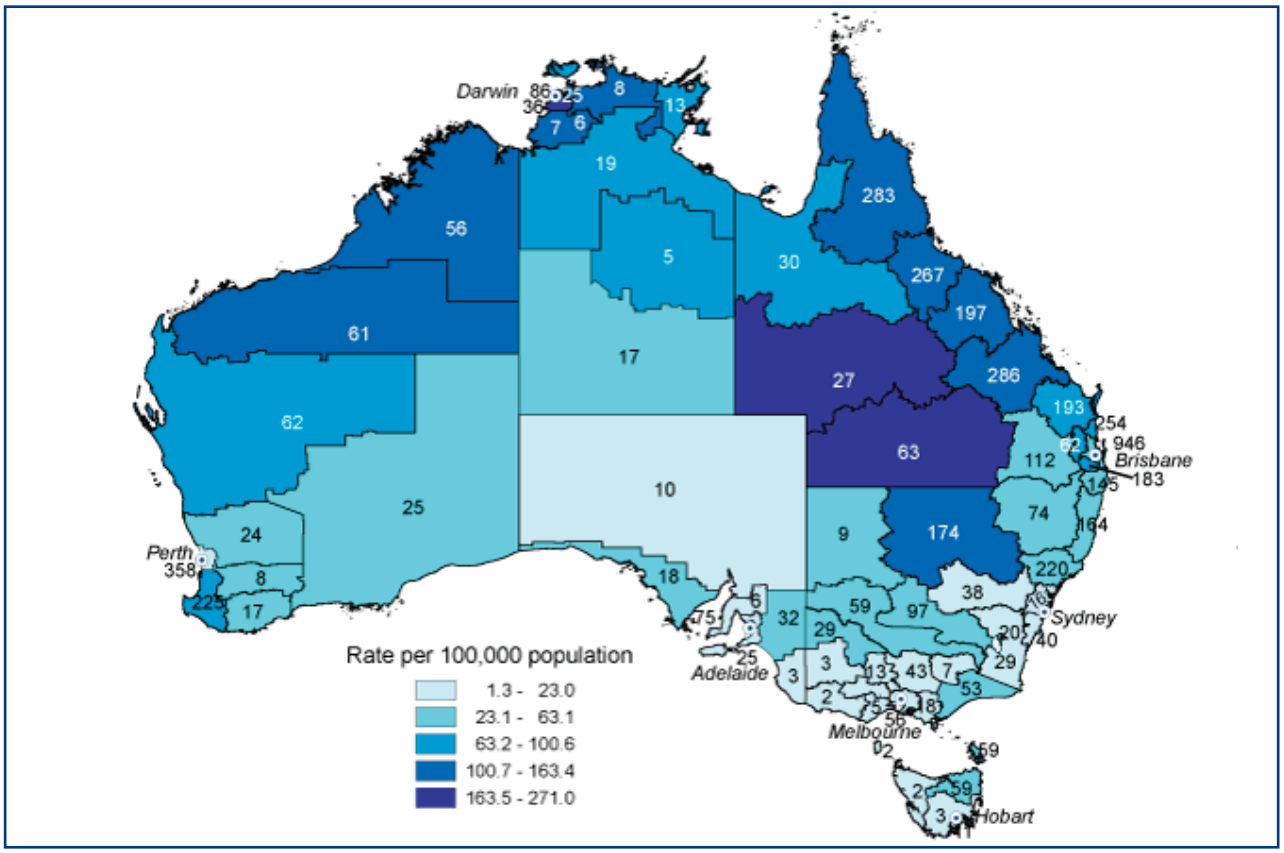
Notification rates ranged from 4.6 per 100,000 population in Victoria to 118 per 100,000 population in the Northern Territory. All jurisdictions reported an increase in notifications when compared with the previous 5-year period. Tasmania reported 77 notifications compared with a 5-year average of 10 cases. The Australian Capital Territory reported 20 notifications compared with a 5-year average of 6 cases.

Notification rates for RRV by the place of residence of a notified case are shown in Map 2. These locations do not represent the place of acquisition of infection. The highest regional RRV notification rates were reported in the Finniss area of the Northern Territory (271 cases per 100,000 population) and the South West region of Queensland (241 per 100,000

Figure 2: Number of notified cases of Ross River virus infection, Australia, 1 July 2002 to 30 June 2008, by date of diagnosis and state or territory



Map 2: Number of notified cases and rate of Ross River virus infection, Australia, 1 July 2007 to 30 June 2008, by Statistical Division



population). Four of the top 10 rates of RRV notification by region in Australia occurred in the Northern Territory during the 2007/08 season.

Chikungunya virus infection

Chikungunya virus is a member of the alphavirus genus in the family *Togaviridae*. It belongs to the Semliki Forest virus complex. It is found epidemically in many parts of South East Asia and in Africa. Chikungunya causes illness characterised by an abrupt onset of fever, rash and severe joint pain (chikungunya is Bantu of the Makonde people of south-east Tanzania for 'that which bends up', reflecting the bent over appearance of those with 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.[3]

In Australia, the known competent vectors for chikungunya virus include *Ae. aegypti*, which occurs in northern Queensland, and *Ae. albopictus,* which is found on Cocos, Christmas and the Torres Strait Islands. Other Australian mosquitoes are possible vectors, but there are no data on the competence of these at present.

There have been confirmed cases of imported chikungunya virus infection into Australia from viraemic travellers during the recent epidemic in the Indian Ocean. Outbreaks in near neighbouring countries such as Indonesia and Papua New Guinea could potentially increase the numbers of viraemic travellers returning to Australia and hence introduce the disease. Northern Australia has a suitable climate and environmental parameters for its introduction. Chikungunya virus infection is a notifiable disease in all jurisdictions other than Queensland and Tasmania. There were 2 cases of overseas-acquired chikungunya infection reported to NNDSS during the 2007/08 season.

Flaviviruses

There were 406 notifications of flavivirus infection during 2007/08 of which dengue virus (DENV) infections accounted for 95% (n=387). Arbovirus infections not elsewhere classified (NEC), accounted for 16 notifications and included 5 cases of Kokobera. The remaining flavivirus notifications included 2 cases of Murray Valley encephalitis (MVEV) and a single case of Kunjin (KUNV) (Table 1).

Sentinel flavivirus surveillance programs

The sentinel chicken program is a program involving Western Australia, New South Wales, Victoria and the Northern Territory that is designed to detect flavivirus activity including the endemic arboviruses MVEV and KUNV, as well as exotic arboviruses such as Japanese encephalitis. Sentinel chicken flocks provide an early warning of increased flavivirus activity in 4 Australian states.[4] The location of sentinel chicken sites during the season is shown in Map 3.

Northern Territory

The current Northern Territory sentinel chicken program commenced in January 1992 and replaced an earlier program run by the Australian Quarantine and Inspection Service (AQIS). 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.

Sentinel chicken flocks are presently located at Leanyer, Howard Springs, Coastal Plains Research Station, Katherine, Nhulunbuy, Tennant Creek, Jabiru, Alice Springs (2), Nathan River, Robinson River and Alyangula (Map 3). DPIFM officers or volunteers usually bleed flocks once a month and the samples are tested for MVEV and KUNV. When chickens from a flock show new antibodies to MVEV during a prime risk period, a media warning is issued for the general area for the risk period. These warnings advise residents of the need to take added precautions to avoid mosquito bites.

Chickens are replaced at least annually and more frequently if birds die or a large proportion seroconvert. They are well positioned to detect flavivirus activity near the principal towns of the Northern Territory and hence provide a timely and accurate indication of risk to people in those towns.

During the 2007/08 season, MVEV activity was detected in the Adelaide River region in February and April, in Katherine in March and April, in Nathan River in February and March and in Robinson River in May (last bleed prior to MVE detection was in December 2007).

KUNV activity occurred in all regions except in East Arnhem and the Barkly region, with chickens seroconverting to KUNV between August 2007 and May 2008. It is notable that probable KUNV activity recorded in the Alice Springs flocks in February and April 2008 were the first since the last seroconversions in 2000/01. However, the titres for both seroconversions were low and could not be confirmed as KUNV with certainty. The lack of MVEV and the low KUNV activity in Alice Springs is thought to be

associated with the draining of the Ilparpa Swamp, as well as the relatively low summer rainfall in Alice Springs.

There were no cases of locally-acquired flavivirus infections notified in the Northern Territory during the 2007/08 season despite the activity reported above in sentinel chicken flocks.

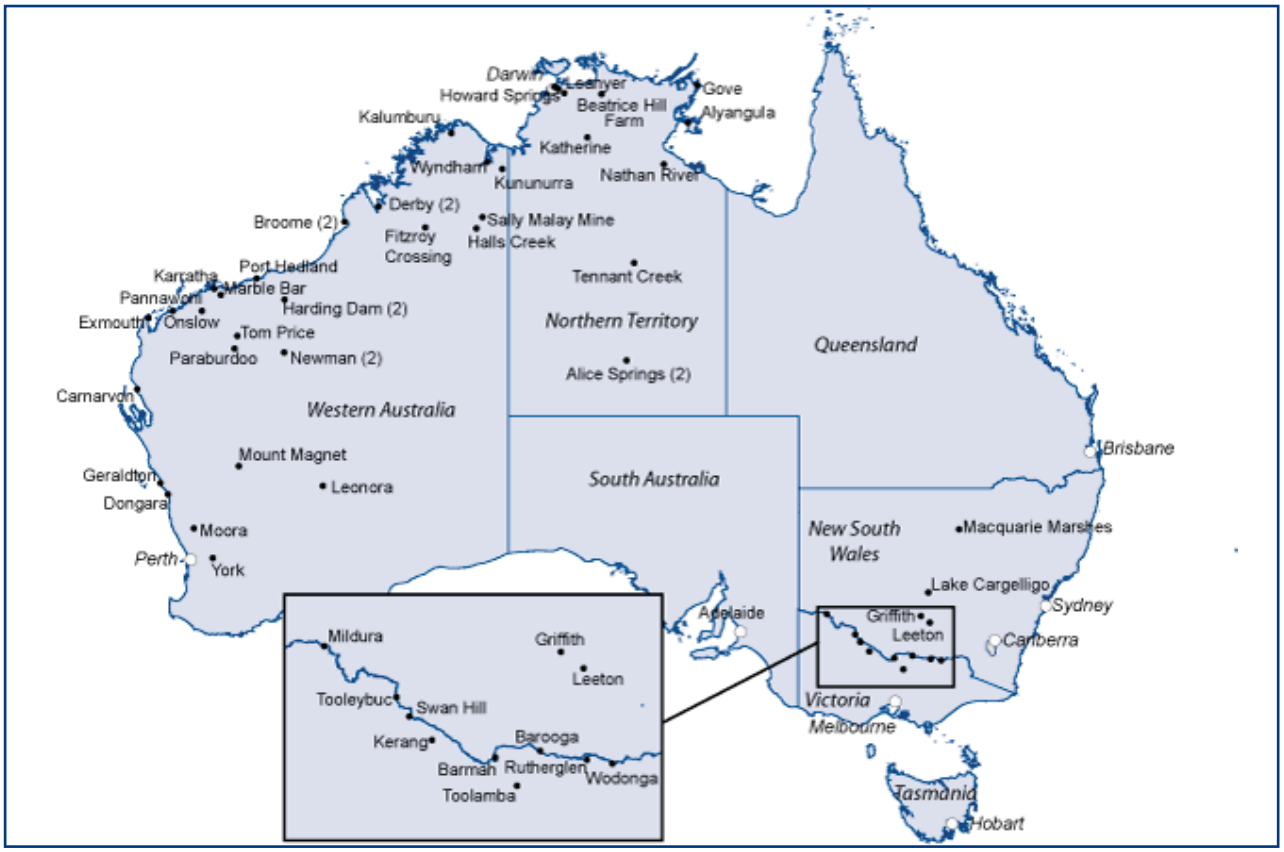
Western Australia

The flavivirus sentinel chicken program in Western Australia is undertaken by the Arbovirus Surveillance and Research Laboratory (ASRL) at The University of Western Australia, on behalf of the Western Australia Department of Health. Many state and local government authorities and community volunteers also take part in the program. Twenty-nine sentinel chicken flocks are located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Central Coastal regions of Western Australia (Map 3). Blood samples from the chickens are collected 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.[5]

In general, rainfall was average to below average between July and November 2007. Seasonal wet season rainfall and the passage of several tropical cyclones through northern Western Australia resulted in above average to well above average rainfall in the Kimberley, Pilbara and Gascoyne regions between December 2007 and March 2008. The western Pilbara and Gascoyne regions also experienced above average rainfall in April. Elsewhere, generally average to below average rainfall was recorded between April and June 2008.

A total of 3,499 serum samples from the 29 Western Australia sentinel chicken flocks were tested for antibodies to flaviviruses during 2007/08.[6] Seroconversions were detected in 48 (1.4%) of the samples. Twenty-three seroconversions detected between July and September 2007 were associated with prolonged flavivirus activity from the previous (2006/07) wet season. KUNV and MVEV were responsible for seven and eight of the seroconversions, respectively. MVEV was more active in the Kimberley region, whereas KUNV was more prevalent in the Pilbara region.

Map 3: Sentinel chicken testing sites, Australia, 2007/08



Overall, the level of MVEV activity during 2007/08 was lower than the previous year, however the level of MVEV activity in the Pilbara region was higher between January and June 2008 than the corresponding period in the previous year. KUNV activity was lower than the previous year in the Kimberley and Pilbara regions. The first activity associated with the 2007/08 wet season occurred in February 2008 when MVEV was detected at Kununurra in the north-east Kimberley region. MVEV activity was subsequently detected at Wyndham, Fitzroy Crossing, Derby and Broome, and the low-level activity continued through to June. KUNV was detected at Kununurra and Broome in May 2008.

In total there were 17 seroconversions to MVEV and 2 seroconversions to KUNV in the Kimberley region between January and June 2008. In the Pilbara region, 3 MVEV seroconversions were detected in the Pannawonica sentinel chicken flock in March 2008, and 2 MVEV infections were detected in the Ophthalmia chickens (near Newman) in April. No KUNV activity was detected in the Pilbara sentinel chickens between January and June 2008, and no flavivirus activity was detected south of Newman during 2007/08. This is the 2nd consecutive season of very low MVEV activity and the 4th year since there was a moderately high number of seroconversions to KUNV in Western Australia, in 2003/04.[7] A number of unidentified flavivirus infections were detected at several locations in the Kimberley and Pilbara regions between July and September 2007. These were possibly due to activity of other flaviviruses that were isolated from mosquitoes collected in northern Western Australia in past seasons.

Media releases were issued by the Western Australia Department of Health on 19 March and 7 April 2008, following the initial detections of MVEV in the Kimberley and Pilbara regions, respectively. A 3rd media release was issued on 29 April 2008 following the death of a resident at Kununurra, in the north-east Kimberley region, after developing MVE. This is the 1st fatal case of MVE in Western Australia since a large outbreak of MVE in 2000.[8]

New South Wales

A total of 1,601 samples were received from 7 sentinel chicken flocks in New South Wales over a 6-month period in 2007/08. There were 4 seroconversions to MVEV and four to KUNV.[9]

There was one human case of MVEV reported from the Macquarie Area Health Service in a 60+ year-old male who developed minor symptoms and made a full recovery. This was the 1st case of MVEV in New South Wales since 1974.[10] The onset date of symptoms was reported as 16 March 2008. The last reported case of KUNV from New South Wales was notified in May 2001.[9]

Victoria

Approximately 3,200 samples were received from 10 sentinel chicken flocks in Victoria over a 4-month period in 2007/08. In March 2008, 2 sentinel chickens in Kerang and 5 chickens in Mildura seroconverted to MVEV. By the end of April another 5 chickens from Kerang, 16 chickens from Mildura and one from Barooga were positive for MVEV.[11] These were the 1st detections of the virus in sentinel chickens since the mid-1970s.

There were no human cases of MVEV reported from Victoria in 2007/08 and none recorded in NNDSS. One human case of KUNV was notified from Victoria in late 2007 in a male tourist who had arrived from Israel with a 5-day history of illness. Further investigations resulted in the reclassification of the diagnosis as West Nile Virus (WNV). This is the first report of a laboratory- confirmed West Nile Virus (New York 99) infection in Australia. Although KUNV is a sub-type of WNV, it does not occur in Israel. The case was almost certainly infected in Israel where WNV is endemic.[12]

Japanese encephalitis virus infections

The AQIS Northern Australia Quarantine Strategy continues to undertake limited surveillance for transmission of Japanese encephalitis virus (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.[13] These animals provide more reliable information than feral or backyard survey samples as they are considered naïve to flaviviruses and can be sampled repeatedly to demonstrate any change in titre.[14] Pigs sampled during a survey of antibodies to JEV in animals in the Torres Strait in 2008 also showed no serological exposure to JEV. AQIS continues to work closely with Queensland Health in relation to the risks of JEV in the region.[13] There were no cases of JEV notified to NNDSS in Australia during 2007/08.

Dengue virus infection

There were 387 cases of dengue virus infection notified during the season of 2007/08. The annual notification rate for the season was 1.8 per 100,000 population, which was similar to the mean rate of the previous 5 years (Table 1). In Australia, imported cases of dengue virus infection are reported each year with occasional local transmission. Local transmission is restricted to areas of northern Queensland

where the key mosquito vector, *Ae. aegypti*, is present. In early 2004, 2 deaths were reported in Australia due to dengue virus infection. These were the 1st deaths attributed to dengue in over 100 years.[15] Figure 3 shows the number of notifications reported by jurisdictions.

Locally-acquired dengue virus infection

Dengue outbreaks in Australia in recent times have been due to importation of the virus by a viraemic tourist or returning resident from a dengue endemic area overseas. Dengue is spread from person to person via the mosquito vector *Aedes aegypti*. Cases of dengue acquired from overseas are of particular importance in north Queensland because of the presence of the *Ae. aegypti* mosquito species that can transmit dengue infection to humans. *Ae. aegypti* is a common mosquito species in north Queensland but dengue is not endemic. A female mosquito can only become infected with dengue after biting an infected human who is viraemic with dengue. It is important to rapidly diagnose the disease in returning residents and tourists to prevent local spread in Queensland.[16] Table 2 shows that 22 cases were locally acquired in north Queensland during the season 2007/08. The Mossman/Port Douglas dengue fever outbreak started in February 2008 and included 22 confirmed cases. The DENV serotype for this outbreak was Type 3 in 19 of the 22 cases.

Overseas-acquired dengue virus infection

During the 2007/08 season, there were 365 notifications of dengue virus infection acquired overseas compared with 203 notifications in the previous season (Table 2). On average, there were 164 overseas-acquired dengue cases per annum reported to NNDSS over the 5 seasons from 2002/03 to 2006/07.

Country of acquisition was available for 250 (68%) cases of overseas-acquired dengue reported to NNDSS (Table 3). Indonesia (including Bali) was reported as the place of acquisition for 104 (28%) cases and involved all 4 dengue serotypes. Cases identified travel to 25 other destinations, which reflect the worldwide distribution of dengue virus infection. Other countries most commonly recorded include Thailand (34), Tonga (18), India (12), Vietnam (11) and Papua New Guinea (9). The infecting DENV serotype was determined for 98 (27%) of the 365 overseas-acquired dengue cases. All 4 serotypes were reported including 35 cases of DENV serotype 1.

The Western Australian Department of Health investigated 70 cases notified between 1 January 2007 and 1 February 2008. Of these, travel to Indonesia was reported by 41 (59%) cases with 31 people confirming travel to Bali. The public health response included alerting the Western Australian public

Figure 3: Number of notified cases of dengue virus infection, local and overseas acquired, Australia, 1 July 2002 to 30 June 2008, by date of diagnosis and state or territory

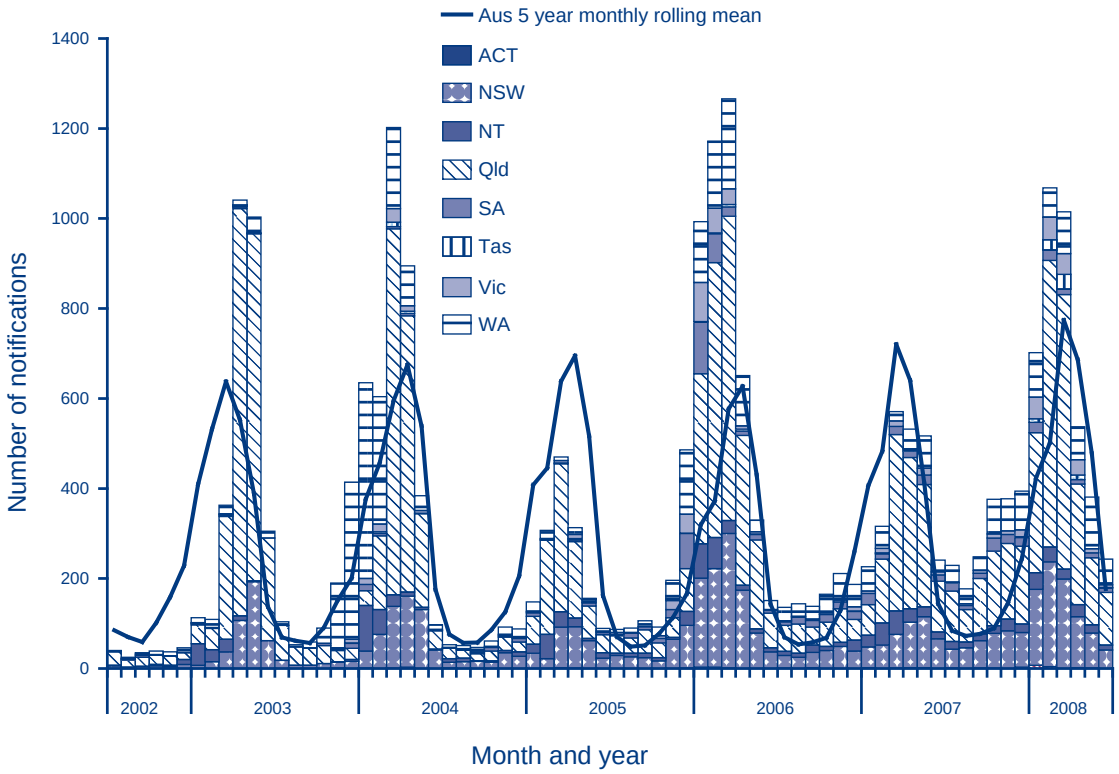


Table 2: Number of notified cases of dengue virus infection, Australia, 1 July 2002 to 30 June 2008, by date of diagnosis, place of acquisition and state or territory

Place of acquisition	Season	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Locally acquired	2002/03	0	0	0	472	0	0	0	0	472
	2003/04	0	0	0	418	0	0	0	0	418
	2004/05	0	0	0	72	0	0	0	2*	74
	2005/06	0	0	0	42	0	0	0	0	42
	2006/07	0	0	0	46	0	0	0	0	46
	2007/08	0	0	0	22	0	0	0	0	22
Total		0	0	0	1,072	0	0	0	2	1,074
Overseas acquired	2002/03	6	82	18	71	8	0	12	21	218
	2003/04	8	33	23	42	7	2	13	6	134
	2004/05	1	33	16	41	3	0	8	11	113
	2005/06	7	54	16	33	10	0	13	20	153
	2006/07	2	72	15	66	12	0	9	27	203
	2007/08	4	104	26	83	35	4	14	95	365
Total		28	378	114	336	75	6	69	180	1,186

* Cases acquired their infection while visiting Queensland.

Table 3: Overseas-acquired dengue notifications, Australia, 1 July 2007 to 30 June 2008, by date of diagnosis, serotype and reported country of acquisition

Country of acquisition	Total	Untyped	Dengue serotype				
			Type 1	Type 2	Type 2 & 3	Type 3	Type 4
Country unknown	115	102	5	4	0	2	2
Indonesia	104	65	6	7	0	8	18
Thailand	34	24	6	2	0	2	0
Tonga	18	16	1	1	0	0	0
India	12	6	1	0	0	5	0
Vietnam	11	7	3	0	0	1	0
Papua New Guinea	9	4	4	0	0	0	1
Malaysia	9	5	3	1	0	0	0
Philippines	8	7	0	0	0	1	0
French Polynesia	5	3	2	0	0	0	0
Singapore	5	2	1	0	1	0	1
Sri Lanka	5	4	0	1	0	0	0
Laos	4	3	1	0	0	0	0
New Caledonia	3	3	0	0	0	0	0
Fiji	3	3	0	0	0	0	0
Cambodia	3	2	1	0	0	0	0
East Timor	3	2	0	0	0	0	1
Bangladesh	3	2	0	1	0	0	0
Solomon Islands	2	2	0	0	0	0	0
Samoa, American	2	2	0	0	0	0	0
North Africa	2	0	1	1	0	0	0
Kiribati	1	0	0	0	0	0	1
Nauru	1	0	0	0	0	0	1
Cook Islands	1	1	0	0	0	0	0
China	1	1	0	0	0	0	0
Brazil	1	1	0	0	0	0	0
Total	365	267	35	18	1	19	25

via print and radio media, of the need for preventive measures in dengue endemic areas. Western Australian doctors (general practitioners, emergency departments, infectious disease physicians, and travel doctors) and laboratories were alerted via a communicable disease bulletin.[17]

Malaria

Malaria is a serious acute febrile illness which can be transmitted from person to person through the bite of an infected mosquito. It is caused by a parasite called *Plasmodium* that includes 4 species – *vivax*, *falciparum*, *malariae* and *ovale*.[18] There were 505 cases of overseas-acquired malaria notified in Australia during the season 2007/08 and no reports of locally-acquired malaria. The annual notification rate for the 2007/08 season was 2.4 per 100,000 population, which was a decrease when compared with the mean rate of the previous 5 years of 3.2 per 100,000 population (Table 1).

Figure 4 shows that as in previous years, there was no seasonal trend. The highest number of notifications occurred in October (58) and February (54). The number of notifications per month exceeded the 5-year rolling mean in October and November 2007.

Notification rates ranged from 1.8 per 100,000 population in Victoria to 10.7 per 100,000 population in the Northern Territory (Figure 5). All jurisdictions reported a decrease in notifications when compared with the previous 5 years other than for Western Australia (3.6 to 3.7 per 100,000 population). Queensland reported 165 notifications compared with a 5 year average of 247 cases. The male to female ratio during 2007/08 was 1:0.5 (68%

Figure 5: Notification rates of malaria infections, 2007/08, compared with the mean of the past 5 financial years, by date of diagnosis and state or territory

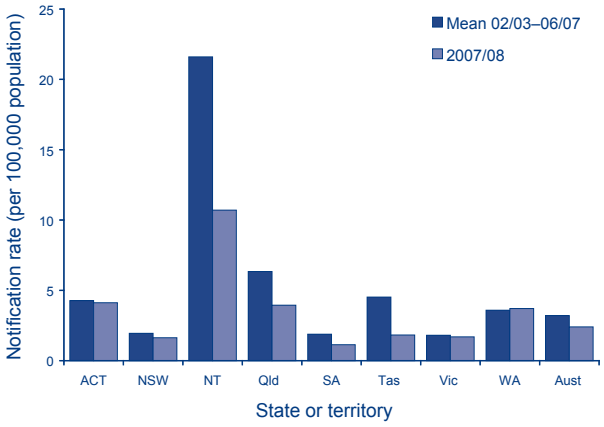
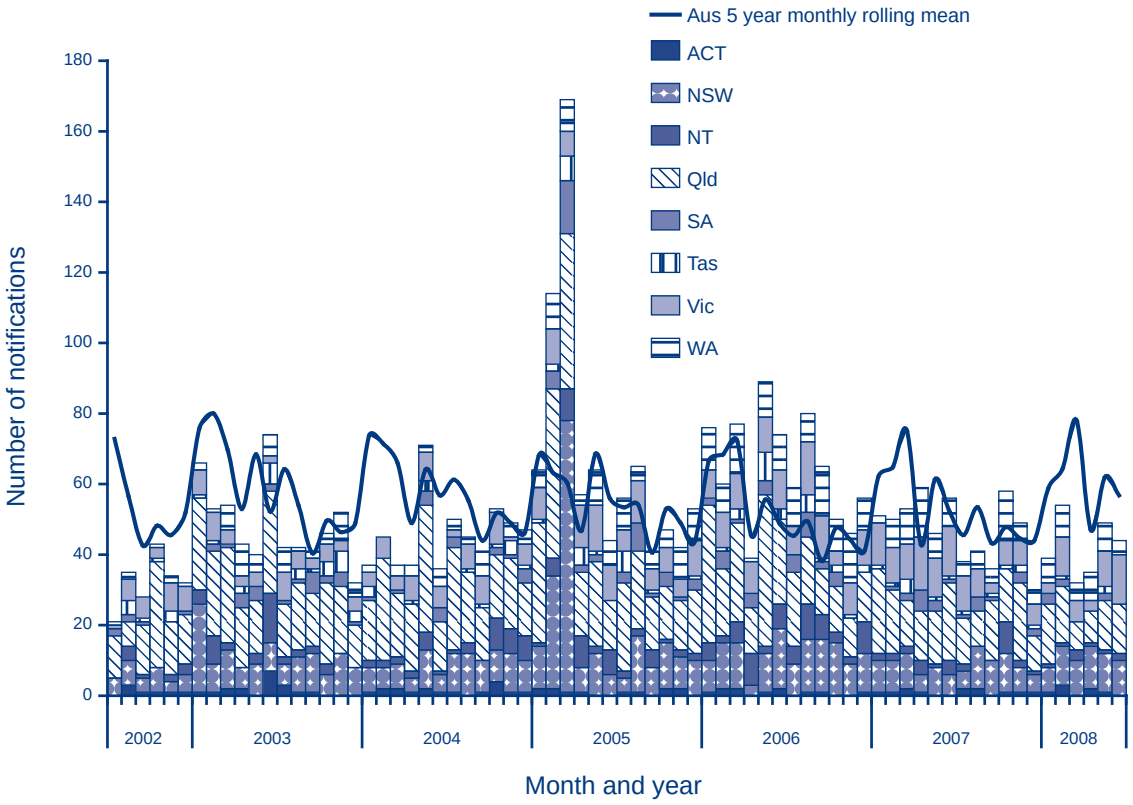


Figure 4: Number of notified cases of malaria infection, Australia, 1 July 2002 to 30 June 2008, by date of diagnosis and state or territory



of notifications were male, which was consistent with the past 5 years). The highest age specific rate for males was 6.4 per 100,000 population; reported in the 20–24 year age group and the highest rate for females was 3.6 per 100,000 population; reported the in 25–29 year age group.

The infecting *Plasmodium* species was reported for 98% of malaria notifications in 2007/08 (Table 4). Of these 505 notifications, *P. falciparum* (46%) and *P. vivax* (48%) were the predominant species.

The country of acquisition was available for 112 (22%) cases of malaria reported to NNDSS (Table 5). Papua New Guinea was reported as the country of acquisition for 80 (28%) cases and included both *falciparum* and *vivax* species. Cases identified travel to 14 other destinations. They included the Solomon Islands (8), Tanzania (5), and Indonesia (4).

Exotic Vector Eradication Program on Groote Eylandt

The exotic dengue vector *Ae. aegypti* was first detected on Groote Eylandt, Northern Territory on 20 October 2006. The Australian Government Department of Health and Ageing (DoHA) agreed in March 2007 to assist with funding for a 2 year *Ae. aegypti* eradication project.

In 2007/08, the *Ae. aegypti* survey and control operations on Groote Eylandt continued to the end of field operations in March 2008. The activities included: adult mosquito control by receptacle and harbourage site spraying, receptacle (breeding site) treatment and larval and adult mosquito surveys. *Ae. aegypti* is a domestic breeder and breeds in water filled receptacles such as tyres, pot plant drip trays, buckets, drums and tins around houses.[16,19] All potential breeding sites were sampled for larvae, which were identified by Northern Territory medical entomology staff.

Increased surveillance and control activities were similar to those used in a previous eradication program at Tennant Creek[20] with the exception that receptacles were primarily treated with alpha cypermethrin instead of bifenthrin. The Groote Eylandt Mining Company (GEMCO) assisted with the survey and control activities.

The last interception of *Ae. aegypti* was in the Alyangula residential area, Northern Territory on 4 June 2007. This was the single property found to have *Ae. aegypti* during the 4th round of survey and treatment. The following 3 rounds of survey and treatment and the Targeted Potential Breeding Site Survey this year, along with broad scale surveys and trapping over a wet season, indicated that the *Ae. aegypti* mosquito population had been eradicated.

Discussion

This report summarises the surveillance of nationally notifiable mosquito-borne disease in Australia for the season 1 July 2007 to 30 June 2008. Of particular concern were overseas-acquired dengue infections and the unusual MVEV activity in surveillance programs in south-east Australia.

Australia experienced an increased number of overseas-acquired dengue virus infections during the season 1 July 2007 to 30 June 2008. Cases of dengue acquired from overseas are of particular importance in north Queensland because of the presence the *Ae. aegypti* mosquito species that can transmit dengue infection to humans.[21] Much of the rise in overseas-acquired dengue virus infections over the past few years can be attributed to disease activity in the Asia Pacific region. Other possible explanations include increased numbers of Australians travelling to dengue-affected areas or changes to diagnostic methods. Regardless, travellers require a doctors' advice prior to travel, outlining the risk of mosquito-borne disease and the required precautions. The World Health Organization has warned of a spreading threat of dengue outbreaks in the Asia Pacific region and urged for a more compre-

Table 4: Overseas-acquired malaria cases, Australia, 1 July 2007 to 30 June 2008, by date of diagnosis, Plasmodium species and state or territory

Plasmodium species	Type (%)	State or territory								Aust
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Plasmodium falciparum	46	2	39	20	70	12	5	30	53	231
Plasmodium vivax	48	10	64	3	88	5	3	54	17	244
Other *Plasmodium* species	3	0	6	0	5	0	0	2	3	16
Mixed *Plasmodium* species	1	0	0	0	0	0	1	2	1	4
Plasmodium species unspecified	2	2	2	0	2	1	0	0	3	10
Total		14	111	23	165	18	9	88	77	505

Table 5: Overseas-acquired malaria cases, Australia, 1 July 2007 to 30 June 2008, by date of diagnosis, country of acquisition and Plasmodium species

Country of acquisition	Total	*Plasmodium* species				
		Not specified	*Falciparum*	*Vivax*	Other *Plasmodium* species	Mixed *Plasmodium* species
Country unknown	393	10	185	180	15	3
Papua New Guinea	80	0	29	50	1	0
Solomon Islands	8	0	0	8	0	0
Tanzania	5	0	5	0	0	0
Indonesia	4	0	2	2	0	0
Kenya	3	0	3	0	0	0
Sudan	2	0	2	0	0	0
Sierra Leone	2	0	1	0	0	1
Vanuatu	1	0	0	1	0	0
East Timor	1	0	0	1	0	0
India	1	0	0	1	0	0
Congo	1	0	1	0	0	0
Nigeria	1	0	1	0	0	0
Burundi	1	0	1	0	0	0
Madagascar	1	0	0	1	0	0
Uganda	1	0	1	0	0	0
Total	505	10	231	244	16	4

hensive approach to mosquito control and disease prevention. The control of dengue and its vectors are important to Australia's health security.[22]

The successful eradication of *Ae. aegypti* from Groote Eylandt was officially announced on 8 May 2008.[23] A number of factors contributed to the successful eradication. A monitoring program enabled early detection of the exotic mosquito incursion and allowed a very quick field response. Assistance from government staff and volunteers aided the initial field response. Rapid access to funding allowed a quick response for program activation, while waiting for approval of funds from DoHA. The success of the program was due to the selection of appropriate chemicals that were successful in treating mosquito adults, larvae and egg infested receptacles. The program incorporated a thorough and repeated larval search of every possible place, treatment of every possible water receptacle and a good evaluation of potential *Ae. aegypti* presence with the aid of ovitraps, carbon dioxide baited encephalitis vector surveillance traps and larval searching of high risk locations. The assistance given by GEMCO and other enterprises on Groote Eylandt and the residents of the various communities also contributed to the successful eradication of *Ae. aegypti*. Increased monitoring for *Ae. aegypti* at Alyangula residential and Alyangula port/industrial areas, and in other areas of the Northern Territory have been implemented and will continue.

Whilst MVEV activity is regularly reported in mosquitoes and sentinel chicken flocks in northern Western Australia and the Northern Territory, it is unusual for activity in mosquitoes and sentinel chicken flocks to occur in south-east Australia. During the season, seroconversions in sentinel chickens in Victoria and New South Wales first indicated the presence of MVEV in February 2008. MVEV was also detected in a chicken in South Australia in May and in horses in Victoria. A human case (fully recovered) of MVEV was also reported from Macquarie Marshes in New South Wales in March 2008. NAMAC considered the level of MVEV activity reported in 2007/08 and in particular its wide geographical distribution to be unusual. The virus detections in mosquito and animals in south-east Australia is also perplexing as the recent drought in south-east Australia caused lower than usual numbers of mosquitoes in the region. NAMAC members note that the reason for last year's MVEV activity is not known, which highlights the gap in knowledge about the epidemiology of MVEV in Australia. Given this recent MVEV activity, NAMAC is reviewing the current guidelines for responding to an outbreak of MVE. Issues in the guidelines currently being reviewed include the nature of MVEV disease, surveillance and detection of an outbreak, investigation of an outbreak source, actions for containment of an outbreak, and actions arising from the initial detection of an outbreak.

The limitations of surveillance data used in this report are referred to in detailed notes on the interpretation of NNDSS, which is available in the 2006 annual report.[1] A 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 assume a clinically compatible arthritic infection. A case may 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.[2,4] Human surveillance of 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.

Arboviral and malaria disease surveillance provides information that assists in the assessment of the effect of mosquito-borne disease in Australia. The monitoring of these diseases has many benefits, including identifying the source of infection and risk factors for illness. Ongoing efforts to strengthen the quality of these data will ensure better use by agencies to prevent and control illness.

Acknowledgements

The National Arbovirus and Malaria Advisory Committee members are (in alphabetical order): Bart Currie, Peter Dagg, Peter Daniels, Jenny Firman, Gerard Fitzsimmons, Julie Hall, Rogan Lee, Mike Lindsay, Conan Liu, John Mackenzie, Rodney Moran, Scott Ritchie, Richard Russell, Christine Selvey, David Smith, James Walker, Peter Whelan, Craig Williams, Jennifer Wall, Susan Barker and Phil Wright (Secretariat).

We would also like to thank:

Alison Milton, Dougald Knuckey, Jennie Hood and Margaret Curran, Office of Health Protection, Australian Government Department of Health and Ageing;

State and territory public health communicable disease surveillance managers.

Sentinel reports were provided by:

Cheryl Johansen and technical staff of the Arbovirus Surveillance and Research Laboratory and the Mosquito-Borne Disease Control Branch, Western Australian Department of Health

The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program, Institute of Clinical Pathology and Medical Research, University of Sydney and Westmead Hospital

Peter Whelan, Nina Kurucz, Northern Territory Department of Health and Families, and Lorna Melville, Northern Territory Department of Primary Industry, Fisheries and Mines

Author details

Gerard Fitzsimmons[1]
Phil Wright[1]
Cheryl Johansen[2]
Peter Whelan[3]

1. Office of Health Protection, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
2. Arbovirus Surveillance and Research Laboratory, Discipline of Microbiology and Immunology, The University of Western Australia, Western Australia
3. Medical Entomology, Communicable Disease Control, Northern Territory Department of Health and Families, Northern Territory

Corresponding author: Mr Gerard Fitzsimmons, Zoonoses, Foodborne and Emerging Infectious Diseases, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 9848, MDP 14, CANBERRA, ACT 2601. Telephone: +61 2 6289 2748. Facsimile: +61 2 6289 2500. Email: Gerard.Fitzsimmons@health.gov.au

References

1. 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, 2006. *Commun Dis Intell* 2008;32(2):139–207.
2. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.
3. 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.
4. 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.
5. 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(2–3):201–210.
6. Johansen C, Avery V, Power S, Zammit C, Masters L, Frestel S, et al. *The University of Western Australia Arbovirus Surveillance and Research Laboratory Annual Report: 2007–2008*. Discipline of Microbiology and Immunology, The University of Western Australia; 2008.

7. Broom A, Johansen C, Power S, Sturrock K, Maley F, Susai V, et al. *Western Australian Arbovirus Surveillance and Research Program Annual Report: 2003–2004*. Discipline of Microbiology and Immunology: The University of Western Australia.; 2004.
8. 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.
9. Doggett S, Clancy J, Haniotis J, Webb C, Russell RC, Hueston L, et al. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program 2007–2008 Annual Report*. Department of Medical Entomology, Institute of Clinical Pathology and Medical Research, Westmead Hospital; 2008.
10. Russell RC, Doggett S. Murray Valley encephalitis virus and Kunjin virus. Institute of Clinical Pathology and Medical Research, [online]. Available from: http://medent.usyd.edu.au/arbovirus/viruses/murrayvalleyencephalitisand-kunjin.htm Accessed on 28 May 2009.
11. Moran R. Flavivirus detection. *Victorian Infectious Diseases Bulletin* 2008;11(2):45.
12. Rogers BA, Hueston L, Ratnam I. Imported West Nile virus encephalitis in an Israeli tourist. *Med J Aust* 2009;191(4):232-234.
13. Animal Health Australia. Animal Health in Australia 2008. Canberra, Australia; 2009. Available from: http://www.animalhealthaustralia.com.au/aahc/index.cfm?F220A69A-C79B-07C7-B0ED-56BE52309051 Accessed May 2009.
14. Animal Health Australia. Animal Health in Australia 2006. Canberra, Australia; 2007.
15. McBride WJ. Deaths associated with dengue haemorrhagic fever: the first in Australia in over a century. *Med J Aust* 2005;183(1):35–37.
16. Queensland Health. *Dengue Fever Management Plan for North Queensland 2005–2010*. Cairns, Queensland: Tropical Public Health Unit Network, Queensland Health; 2005.
17. Western Australian Department of Health. Dengue fever risk in Bali. Disease WAtch 2008;12(2):2.
18. Heymann D, ed. *Control of Communicable Diseases Manual,* 18th edn. Washington: American Public Health Association; 2004.
19. Whelan PI, Kulbac M, Bowbridge D, Krause V. The eradication of *Aedes aegypti* from Groote Eylandt, Australia 2006–2008. *Arbo Res Aust*. In press 2009.
20. Northern Territory Government Department of Health and Community Services. *Aedes Aegypti* eradication Project Tennant Creek Northern Territory NT Department of Health and Community Services, *Aedes Aegypti* Eradication Project Tennant Creek Northern Territory; August 2006.
21. Russell RC, Currie BJ, Lindsay MD, Mackenzie JS, Ritchie SA, Whelan PI. Dengue and climate change in Australia: predictions for the future should incorporate knowledge from the past. *Med J Aust* 2009;190(5):265–268.
22. Sweeny AL, Beard FH. Queensland Health Notifiable Diseases Report, 2002–2006. Brisbane: Communicable Diseases Branch, Brisbane: Queensland Health. In press 2009.
23. Northern Territory Government Department of Health and Community Services. Dengue Mosquito eradicated on Groote Eylandt, Media release 9 May 2008.
24. Public Health Laboratory Network. Alphavirus and flavivirus laboratory case definitions. 2001. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-phlncd-flavivirus.htm Accessed on 11 June 2009.

Immunisation coverage annual report, 2007

Brynley Hull, Shelley Deeks, Rob Menzies, Peter McIntyre

Executive summary

Currently, the routine reporting of immunisation coverage data from the Australian Childhood Immunisation Register is done for 3 key milestone ages, nationally and by jurisdiction, at quarterly intervals. This reporting is limited in a number of ways as timeliness of receipt of vaccines is not captured, not all vaccines currently in the National Immunisation Program (NIP) are included and coverage is reported only at the jurisdictional level, with information about smaller geographic units not provided. The aim of this 1st annual immunisation coverage report is to highlight important trends and significant issues including overall immunisation coverage by Indigenous status and for individual vaccines in the NIP; timeliness of immunisation; and immunisation coverage mapping in smaller geographic areas.

The data in this report reveals that Immunise Australia Program coverage targets have been reached for children at both 12 and 24 months of age and are being approached for children at 6 years of age. With up to 3% of Australian parents not immunising their children for philosophical or religious reasons, it will be difficult for 'fully immunised' coverage estimates to exceed 95%, especially as the reporting of immunisation encounters is still not totally complete.

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. This is likely related to the removal of the 18-month booster dose of DTP, as well as the impact of immunisation incentives. Coverage estimates for the 6-year age group also increased noticeably in June 2006. A possible factor in this increase in coverage is the introduction of the multivalent combination vaccine Infanrix-IPV in November 2005.

A number of vaccines in the NIP are not included when determining 'fully immunised' status or eligibility for incentive payments. Despite this, coverage data for the 7vPCV and meningococcal C vaccines is comparable with currently reported vaccines, while coverage for varicella is lower.

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and pneumococcal polysaccharide vaccine) remains sub-optimal. This has been previously reported for other vaccines for both children and adults.

Although coverage data reveal that most children eventually complete the scheduled vaccination series by the 24-month milestone, many 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 increased slightly.

Although Indigenous children in Australia have coverage levels that are similar to non-Indigenous children at 24 months of age, the disparity in delay in receipt of vaccination between Indigenous and non-Indigenous children, which is up to 17% for the 3rd dose of DTP and 7vPCV, remains a challenge.

Rationale for an annual immunisation coverage report

Currently, the routine reporting of immunisation coverage data from the Australian Childhood Immunisation Register (ACIR) is done for 3 key milestone ages, nationally and by jurisdiction, at quarterly intervals and published in the *Communicable Disease Intelligence* (*CDI*) journal.[1,2] The age milestones are 12 months (for vaccines due at 6 months), 24 months (for vaccines due at 12 months), and 6 years (for vaccines due at 4 years). From the beginning of 2008, immunisation coverage for vaccinations due by 4 years of age has been assessed earlier, at 5 years rather than 6 years of age.[3] This reporting is limited in a number of ways. First, timeliness of receipt of recommended vaccines is not captured; second, these coverage calculations do not include all vaccines currently in the National Immunisation Program (NIP); and third, coverage is reported only at jurisdictional level and information about smaller geographic units is not provided.

Timeliness of immunisation

The most widely accepted indicator of national immunisation coverage internationally is the proportion of children who have received all recommended vaccines by 24 months of age, as prescribed by the World Health Organization,[4] but this does not capture late immunisation, which may be substantial by 24 months of age. Late acquisition of immunity due to delay in immunisation is especially important for a number of severe infections of young infants, such as pertussis and invasive disease due to *Haemophilus influenzae* type b (Hib) or *Streptococcus pneumoniae*. Immunisation at the earliest appropriate age (timeliness) is thus an important public health goal, especially for countries such as Australia where high levels of vaccine

coverage at milestone ages have been achieved. However, published reports on timeliness of vaccine administration are limited primarily to the United States of America[5–12] and 1 report from Sweden,[13] and methods of measuring timeliness have varied.

Reporting on all National Immunisation Program vaccines

Table 1 shows the Australian National Immunisation Program Schedule (NIPS) in 2007. Only those vaccines that were on the schedule prior to 1993 were considered when determining whether a child is 'fully immunised' for the calculation of coverage rates and payment of parental and provider incentives. The Australian Government had not made a decision to include vaccines added after this date in the assessment of vaccination status.

The vaccines included in the assessment of vaccination status were: diphtheria, *Haemophilus influenzae* b (Hib), hepatitis B, measles, mumps, pertussis, polio, rubella and tetanus. Vaccines not included are: meningococcal C vaccine (Men C), 7-valent pneumococcal conjugate vaccine (7vPCV), and rotavirus vaccine. Varicella vaccine was not included for coverage assessment but, in 2007, eligible immunisation providers received an information payment (up to $6) and a Service Incentive Payment (SIP) ($18.50) for reporting completion of the NIP 18-month schedule point, at which varicella vaccine was given.[14,15]

Other vaccines that were not included in the assessment of vaccination status for coverage or payment eligibility purposes were NIP vaccines recommended for specific populations, that is hepatitis A and 23-valent pneumococcal polysaccharide (23vPPV) vaccines, and non-NIP vaccines such as Bacillus Calmette-Guérin (BCG).

Geographic units for reporting of immunisation coverage

Data at the local, jurisdictional, and national level is necessary for comprehensive planning and delivery of immunisation programs. Without this information, public health administrators cannot reliably detect low rates of immunisation in specific geographic areas or populations, especially with respect to prevention of disease outbreaks in communities. While national immunisation coverage may be above the targets for 12 and 24 months of age at the national and jurisdictional level, less is known about coverage in smaller regions within jurisdictions. To date, mapping has revealed pockets of low coverage in inner urban and some rural areas, which are likely to be more a result of reporting problems in the former.[16] Immunisation coverage maps based on ACIR data by region are of interest to a range of national stakeholders but are not routinely published.

Two of the important and unique features of the ACIR are the ability to record a conscientious objection to immunisation and the ability to calculate the percentage of children who have no vaccines recorded. A previous unpublished National Centre for Immunisation Research and Surveillance (NCIRS) study found that having no vaccines recorded on the ACIR was a good proxy for conscientious objection to immunisation, although lack of reporting by providers may sometimes be the cause. Examining trends in both the percentage of children with no vaccines recorded and the percentage

Table 1: Australian National Immunisation Program Schedule for children in 2007

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

* Diphtheria-tetanus-acellular pertussis/Hep B vaccine from May 2000 (Pathway 1).

† Hib PRP-OMP/hep B from May 2000 (Pathway 2).

‡ Hib PRP-OMP (Pathway 1) from May 2000.

§ Aboriginal and Torres Strait Islander children in high risk areas.

‖ 23-valent pneumococcal polysaccharide vaccine for Aboriginal and Torres Strait Islander children in high prevalence jurisdictions only from September 2003.

¶ 3rd dose of vaccine is dependent on vaccine brand used in state or territory.

of registered conscientious objectors to immunisation provides a more complete picture of geographic areas particularly affected by under-immunisation.

In recent years, the NCIRS has published a number of reports[4,17–28] examining aspects of immunisation coverage in Australia, including coverage for different vaccines, at different ages and for Indigenous children, as well as timeliness of immunisation and small area mapping. NCIRS has also assessed coverage rates for NIP vaccines not routinely reported in *CDI*; however, these have not been routinely published. The aim of this 1st annual immunisation coverage report is to combine all these data in one document, highlighting important trends and significant issues over the preceding 12 months. These include overall immunisation coverage by Indigenous status and for individual vaccines included on the NIP; timeliness of immunisation; and immunisation coverage mapping in smaller geographic areas.

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January 1996, and includes all children under the age of 7 years enrolled in Medicare.[19] 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.[19] 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 nightly from the Medicare database 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 is 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.

For an immunisation to be recorded on the Register as a valid dose, it must be given in accordance with current National Health and Medical Research Council guidelines published in *The Australian Immunisation Handbook*.[29] 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)[30] since the ACIR's inception, with each cohort defined by date of birth in 3-month age groups. 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 6 years of age (for vaccines due at 4–5 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 6th birthday are considered.[30] 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.[21,22]

Full year cohorts are predominantly used in the analyses in this report, also with a minimum 3-month lag for late notifications. These cohorts are children born between 1 January and 31 December 2006 for the 12-month milestone age; children born between 1 January and 31 December 2005 for the 24-month milestone age; and children born between 1 January and 31 December 2001 for the 6-year (72-month) milestone age. Three-month cohorts are also used but for time trend analyses only.

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 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of Hib vaccine, and 2 or 3 doses of hepatitis B vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of Hib vaccine, 2 or 3 doses of hepatitis B vaccine, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 6 years of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Immunisation coverage estimates were also calculated for individual NIP vaccines, including the 6 NIP vaccines not routinely reported in *CDI*. They were: 3 doses of 7vPCV due by 12 months of age; 1 dose of varicella vaccine and 1 dose of meningococcal C vaccine due by 24 months of

age; 2 doses of hepatitis A vaccine in Indigenous children due by 30 months of age; and 1 dose of 23-valent pneumococcal polysaccharide vaccine in Indigenous children due by 36 months of age. In addition, 1 dose of rotavirus vaccine due by 4 months of age was assessed. Although this is not the complete series for rotavirus vaccine, 1 dose was assessed as the vaccine was only added to the NIP in July 2007, the year under surveillance for this report. The complete rotavirus vaccine series will be assessed in subsequent annual reports.

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 'no delay' (age-appropriately immunised), vaccines received 'too early' (greater than 30 days prior to when it was due), vaccine dose 'not recorded', vaccine received 'acceptably early' (within 30 days prior to when it was due), 'delay of between 1 to 6 months', or 'delay greater than 6 months'. However, we have only reported on the latter 2 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 then 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.[31] 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', combining children with unknown Indigenous status and those recorded as 'non-Indigenous' for the latter category. Coverage estimate time trends are presented from 2004 only, due to poor rates of reporting Indigenous status prior to then.[32]

Small area coverage

Coverage was calculated for ABS-defined Statistical Subdivisions (SSD).[33] 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. The total number of children included in each cohort was approximately 275,000. Coverage was calculated using the cohort method described in *CDI*, March 1998.[30]

Maps were created using version 7 of the MapInfo mapping software[34] and the ABS Census Boundary Information. As postcode is the only geographical indicator on the ACIR, the ABS Postal Area to SLA 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.[35]

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 2006. At the time of data extraction on 31 March 2008, 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.

Results

Coverage estimates

Overall

The 2007 coverage estimates, calculated for full-year birth cohorts, for the 3 milestone ages of 12 months, 24 months and 6 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

Table 2: Percentage of children in 2007 immunised at 12 months of age, by vaccine and state or territory*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,636	93,305	3,633	57,759	18,511	6,237	68,181	28,612	280,874
Diphtheria, tetanus, pertussis (%)	94.1	92.0	91.0	92.0	91.7	92.6	92.9	89.7	92.0
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	94.9	95.0	93.9	94.4	95.5	94.9	93.4	94.5
Hepatitis B (%)	96.1	94.8	95.5	93.8	94.4	95.4	94.8	93.3	94.4
Fully immunised (%)	93.9	91.6	90.6	91.1	90.9	92.3	91.9	89.1	91.3

* For the birth cohort born in 2006, assessment date 31 March 2008.

Table 3: Percentage of children in 2007 immunised at 24 months of age, by vaccine and state or territory*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,378	90,908	3,602	57,278	17,942	6,029	65,428	27,393	272,958
Diphtheria, tetanus, pertussis (%)	95.4	95.1	96.1	94.6	95.2	96.6	95.8	94.2	95.1
Poliomyelitis (%)	95.4	95.0	96.0	94.6	95.1	96.5	95.8	94.1	95.1
Haemophilus influenzae type b (%)	95.2	94.9	94.5	93.7	93.9	96.4	94.7	93.7	94.4
Hepatitis B (%)	96.0	95.8	97.1	95.5	95.8	96.7	96.4	95.1	95.9
Measles, mumps, rubella (%)	94.4	93.9	95.5	93.7	94.1	95.6	94.9	93.0	94.1
Fully immunised (%)	93.4	92.5	93.6	92.2	92.8	95.0	93.8	91.0	92.7

* For the birth cohort born in 2005, assessment date 31 March 2008.

Table 4: Percentage of children in 2007 immunised at 6 years of age, by vaccine and state or territory*

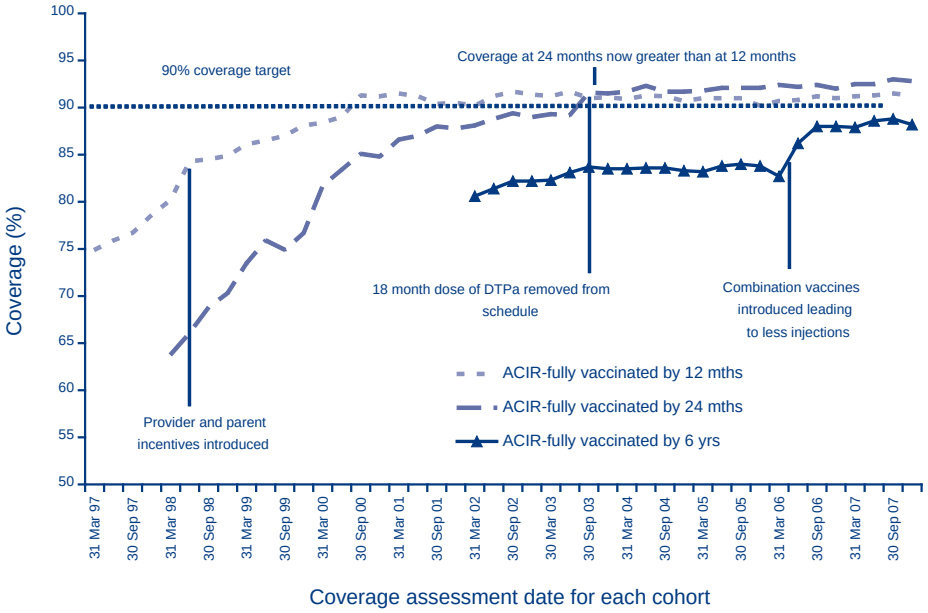
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,153	88,221	3,615	55,185	18,139	6,012	63,806	26,753	265,884
Diphtheria, tetanus, pertussis (%)	90.4	89.1	87.9	88.8	87.3	89.4	91.4	85.4	89.1
Poliomyelitis (%)	90.5	89.0	87.8	88.9	87.3	89.3	91.5	85.5	89.1
Measles, mumps, rubella (%)	90.1	89.1	88.0	88.8	87.2	89.5	91.4	85.5	89.1
Fully immunised (%)	89.5	88.3	87.2	88.1	86.7	88.7	90.9	84.5	88.4

* For the birth cohort born in 2001, assessment date 31 March 2008.

90%. Recorded coverage for the 6-year age group is approaching, but still below, the target. The trends in 'fully immunised' childhood vaccination coverage in Australia at 12 months, 24 months, and at 6 years of age are shown in Figure 1. Coverage was calculated for 42 consecutive 3-month cohorts born from 1 January 1996 to 31 December 2006. For all vaccines due by 1 year of age, coverage estimates increased steadily from 75% for the 1st cohort to 91% by the 42nd cohort, assessed on 31 December 2007. For all vaccines due by 24 months of age, coverage estimates also increased steadily from 64% for the 1st cohort to 92.8% by December 2007. Coverage estimates for all vaccines due by 6 years of age were first reported in *CDI* in 2002, and have also increased steadily from 80.6% in early 2002 to 87.3% in late 2007.

Coverage estimates for the 24-month age group increased substantially and suddenly in

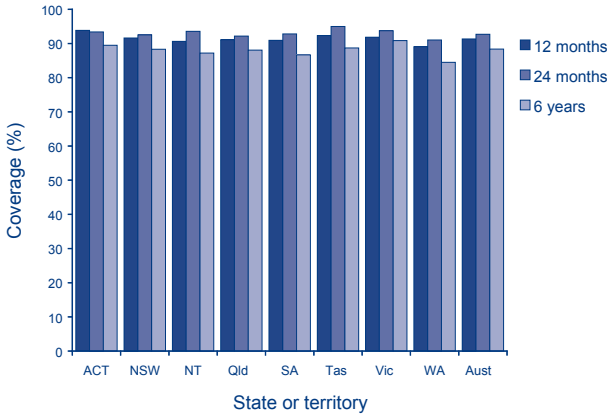
Figure 1: Trends in 'fully immunised' vaccination coverage, Australia, 1997 to 2007, by age cohort



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. Coverage estimates for the 6-year age group also had a noticeable increase in June 2006, corresponding with the introduction of combination vaccines.

Figure 2: 'Fully immunised' vaccination coverage estimates, 2007, by state or territory



Estimates of the proportion of children classified as 'fully immunised' by state and territory for all 3 milestones are shown in Figure 2. There are variations in 'fully immunised' coverage for all 3 milestones. Almost all jurisdictions, except for Western Australia, reached the Immunise Australia Program target of 90% coverage for the 1st and 2nd milestone vaccines. However, only 1 jurisdiction, Victoria, attained the 90% coverage target for the 3rd milestone at 6 years of age.

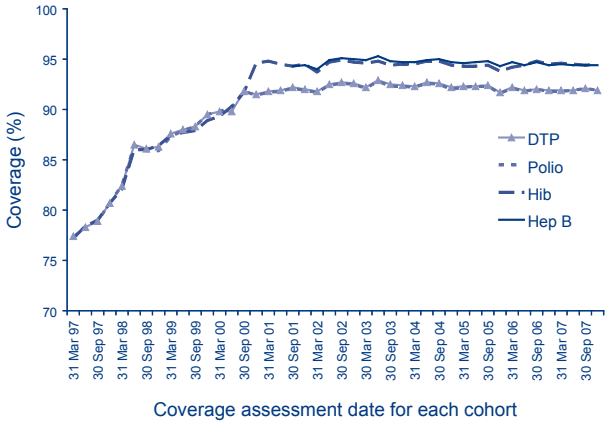
Individual vaccines

The trends in childhood vaccination coverage in Australia for individual vaccines due at 12 months of age (DTP, polio, Hib and hepatitis B) are shown in Figure 3, calculated for consecutive 3-month cohorts born from 1 January 1996 to 31 December 2006. Coverage estimates for all vaccines remained relatively stable throughout the latter part of 2001 to 2007. Coverage for the Hib and hepatitis B vaccines is 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 due at 24 months of age (DTP, polio, Hib, hepatitis B and MMR) are shown in Figure 4, calculated for consecutive 3-month cohorts born from 1 January 1996 to 31 December

2005. 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, while coverage was lowest for the MMR and Hib vaccines.

Figure 3: Trends in vaccination coverage estimates for individual vaccines at 12 months of age (DTP, polio, hepatitis B and Hib)*

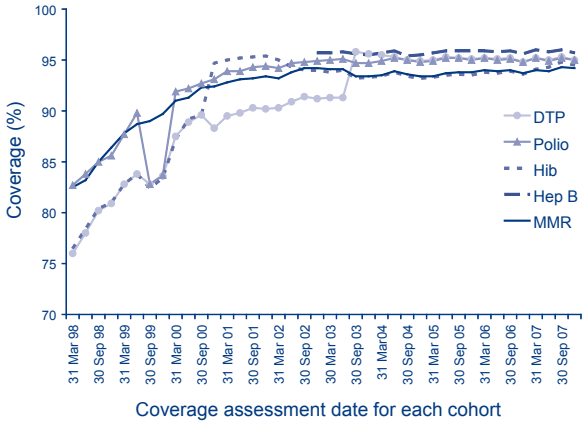


* 3rd dose of DTP and polio, 2nd or 3rd dose of Hib and Hep B

By 3-month birth cohorts born between 1 January 1996 and 31 December 2006. Coverage assessment date was 12 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Figure 4: Trends in vaccination coverage estimates for individual vaccines at 24 months of age (DTP, polio, hepatitis B, Hib and MMR)*



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

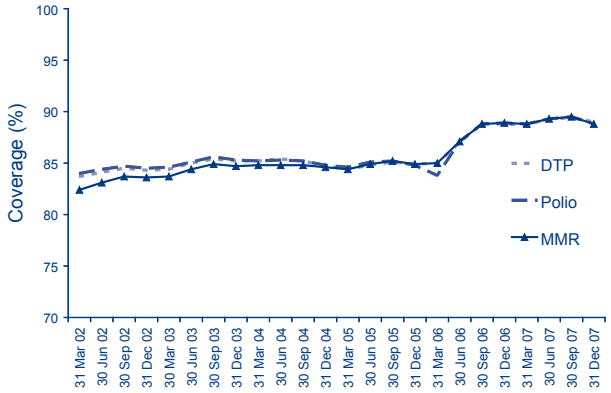
By 3-month birth cohorts born between 1 January 1996 and 31 December 2005. Coverage assessment date was 24 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

However, coverage for all vaccines currently assessed at this age has been consistent for each of them, as evidenced by the very flat curves over this time.

The trends in childhood vaccination coverage in Australia for individual vaccines due by 6 years of age (DTP, polio and MMR) are shown in Figure 5, calculated for consecutive 3-month cohorts born from 1 January 1996 to 31 December 2001. 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 be 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.

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



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

By 3-month birth cohorts born between 1 January 1996 and 31 December 2001. Coverage assessment date was 72 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Coverage estimates for Indigenous children

Vaccination coverage estimates in 2007 for the 3 milestone ages for individual vaccines by Indigenous status are shown in Table 5. These show that coverage is lower for Indigenous children than non-Indigenous at all 3 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

Table 5: Vaccination coverage estimates, 2007, by age, vaccine and Indigenous status

Vaccine	Milestone age	Indigenous	Non-Indigenous
DTP	12 months*	84.8	92.2
	24 months†	94.0	94.8
	6 years‡	87.7	88.9
Polio	12 months*	84.8	92.2
	24 months†	93.9	94.8
	6 years‡	87.9	89.1
Hib	12 months*	92.2	94.5
	24 months†	91.8	93.6
	6 years‡	N/A§	N/A§
Hep B	12 months*	92.6	94.3
	24 months†	97.0	95.5
	6 years‡	N/A§	N/A§
MMR	12 months*	N/A§	N/A§
	24 months†	93.6	93.9
	6 years‡	88.2	89.0

* Birth cohort born 1 January 2006 – 31 December 2006, data as at 31 March 2008.

† Birth cohort born 1 January 2005 – 31 December 2005, data as at 31 March 2008.

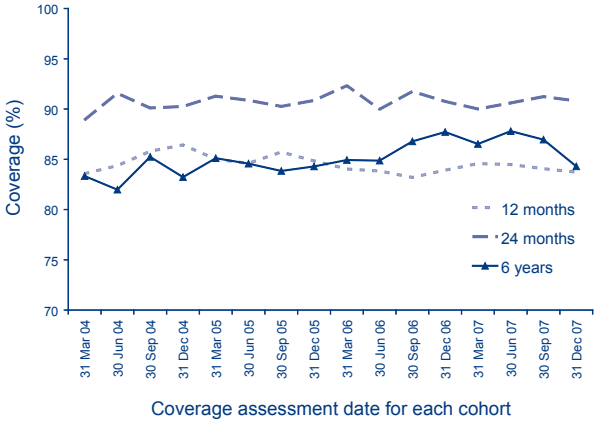
‡ Birth cohort born 1 January 2001 – 31 December 2001, data as at 31 March 2008.

§ Not included in coverage estimates for that group.

24 months of age for most vaccines being very similar 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 for Indigenous children since 2004 are shown in Figure 6. Coverage was calculated for consecutive 3-month cohorts assessed from 1 March 2004 to 31 December 2007. Coverage for all vaccines due by 24 months of age has consistently remained higher than at 12 months

Figure 6: Trends in 'fully immunised' vaccination coverage for Indigenous children in Australia, 2004 to 2007, by age cohorts



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.

Table 6 shows 'fully immunised' vaccination coverage estimates in 2007 for Indigenous children at the 3 milestone ages by state or territory. At age 12 months, the proportion of Indigenous children fully vaccinated was 84.2%, compared with 91.3% for all Australian children (i.e. includes both Indigenous and non-Indigenous children) and was lower among Indigenous children in all jurisdictions. The extent of the difference varied among jurisdictions, reaching more than 10 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 90.7% for Indigenous and 92.7% for all Australian children nationally (Table 6).

At 6 years of age, the proportion recorded as being 'fully vaccinated' was generally lower than that at earlier age milestones. There was little difference between Indigenous and all Australian children at the national level (86.4% and 88.4%, respectively)

Table 6: Percentage of Indigenous children fully immunised at 12 months, 24 months and 6 years of age, 2007, by state or territory *

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
12 months – fully immunised (%)	87.2	84.9	87.6	85.6	78.1	91.5	86.8	76.9	84.2
24 months – fully immunised (%)	91.8	91.4	93.7	91.5	88.6	93.6	91.3	85.1	90.7
6 years— fully immunised (%)	80.2	84.7	92.1	91.1	73.8	85.9	87.4	79.4	86.4

* Assessment date 31 March 2008

while, for individual jurisdictions, coverage in Indigenous children ranged from 13% lower (in South Australia) to 5% higher (in the Northern Territory) than in all Australian children (Table 6).

Timeliness of the 3rd dose of DTP and the 1st dose of MMR vaccine by Indigenous status and remoteness is shown in Table 7. For both vaccines, the proportion with long delays (i.e. greater than 6 months) was 2–4 times higher in Indigenous children than in non-Indigenous children, with greater differentials in accessible than in remote areas. A similar pattern was seen for delays of 1–6 months. When we examined the degree of vaccination delay among Indigenous children only by remoteness, we found that, for the 3rd dose of DTP vaccine, the proportion with short delays was greater among Indigenous children residing in remote areas than in accessible areas (36% versus 31%).

Coverage for National Immunisation Program vaccines not routinely reported

7vPCV

7vPCV was first added to the NIP in January 2005. Figure 7 shows that 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 of 7vPCV is similar in all jurisdictions at greater than or approaching 90% (Table 8).

Rotavirus

Rotavirus vaccine was added to the NIP in July 2007 so there is only a small series of trend data available. Assessment is limited to receipt of the 1st dose by 4 months of age and is currently lower than that for 7vPCV. Reported coverage for 1 dose of rotavirus at 4 months of age does vary by jurisdiction, from 80.3% in Tasmania to 87.2% and 91% in the Northern Territory and Australian Capital Territory, respectively (Table 8).

Meningococcal C

Meningococcal C vaccine was added to the NIP in January 2003. Figure 8 shows that 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 a high of 93%. There is little variation by jurisdiction with all jurisdictions greater than 92% (Table 8).

Figure 7: Trends in coverage for 7vPCV and rotavirus vaccines

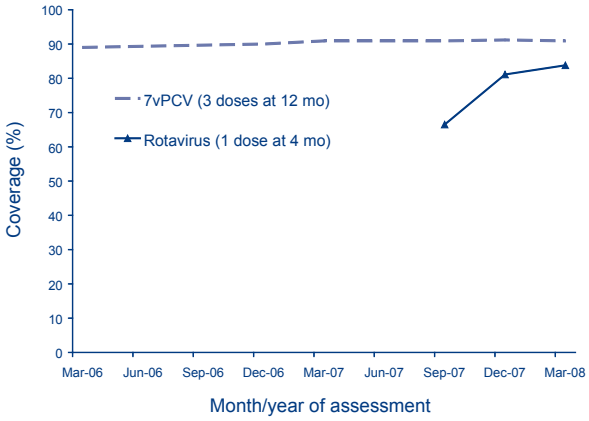


Figure 8: Trends in coverage for meningococcal C (Men C) and varicella vaccines

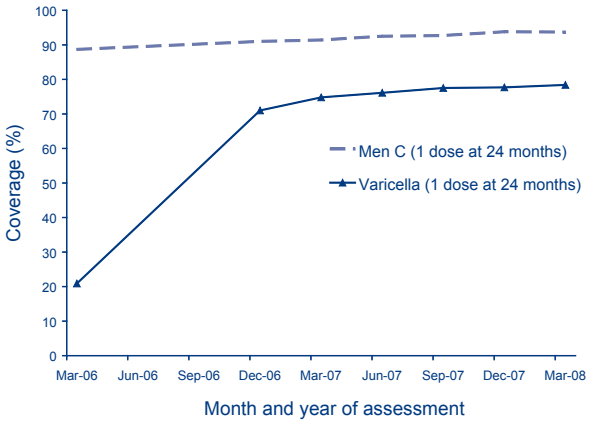


Table 7: Vaccination delay, by Indigenous and remoteness status for the cohort of children born in 2005, Australia

Vaccine dose	Indigenous status	Remoteness	1–6 months delay %	> 6 months delay %
DTP3	Indigenous	Accessible	31	11
		Remote	36	9
	Non-Indigenous	Accessible	19	3
		Remote	20	3
MMR1	Indigenous	Accessible	34	7
		Remote	32	6
	Non-Indigenous	Accessible	27	3
		Remote	27	2

Varicella

Varicella vaccine was added to the NIP in November 2005. Figure 8 shows coverage for this vaccine has consistently been 10–15 percentage points lower than that for meningococcal C vaccine, with coverage just below 80% for the latest assessment. This is probably partly due to the shorter time varicella has been on the NIP, the presence of pre-existing immunity as a reason for non-vaccination and lower acceptance by parents and doctors of the need for varicella vaccination, 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. Varicella vaccine coverage does vary by jurisdiction from 75.8% in Western Australia to greater than 81% in Queensland and Tasmania (Table 8). Data are also available from the ACIR on the numbers 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 slowly increased from 109 in January 2006 to a peak of 2,440 in July 2007, but have decreased since then to around 1,000 reports per quarter (not shown). However, the number of natural immunity reports as a proportion of the ACIR child population is small and would not have an important effect on varicella coverage estimates.

Hepatitis A

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 under 50% (Figure 9). There is a large variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 23.6% in South Australia to a high of 79.9% in the Northern Territory (Table 8).

23vPPV

The 23vPPV has been available in Australia since 1983 and recommended for Indigenous children in those 4 jurisdictions as a booster at 18–24 months of age since 2001; coverage has consistently been

Figure 9: Trends in coverage for hepatitis A and pneumococcal polysaccharide (23vPPV) vaccines for Indigenous children

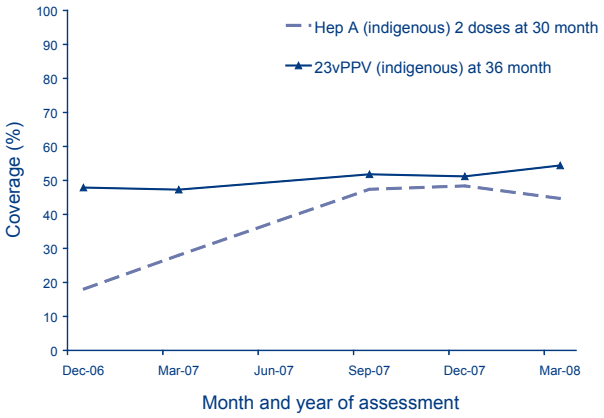


Table 8: Vaccination coverage for 7vPCV, rotavirus, meningococcal C, varicella, hepatitis A (Indigenous only) and 23vPPV (Indigenous only) in 2007, by state or territory, assessment date 31 March 2008

State or territory	Vaccine type					
	7vPCV*	Rotavirus†	Men C‡	Varicella§	Hep A (Indigenous only)‖	23vPPV (Indigenous only)¶
ACT	93.8	91.0	94.8	76.0	na	na
NSW	90.9	83.4	93.4	76.8	na	na
NT	90.5	87.2	94.4	80.5	79.9	74.2
Qld	91.1	84.5	93.5	81.8	32.3	50.3
SA	90.2	85.9	93.6	78.7	23.6	37.0
Tas	91.8	80.3	93.8	81.1	na	na
Vic	91.7	83.7	94.5	78.4	na	na
WA	88.5	81.5	92.7	75.8	50.1	53.5
Aust	90.9	83.8	93.7	78.4	44.7	54.4

* 3 doses at 12 months of age.
† 1 dose at 4 months of age.
‡ 1 dose at 24 months of age.
§ 1 dose at 24 months of age.
‖ 2 doses at 30 months of age.
¶ 1 dose at 36 months of age.

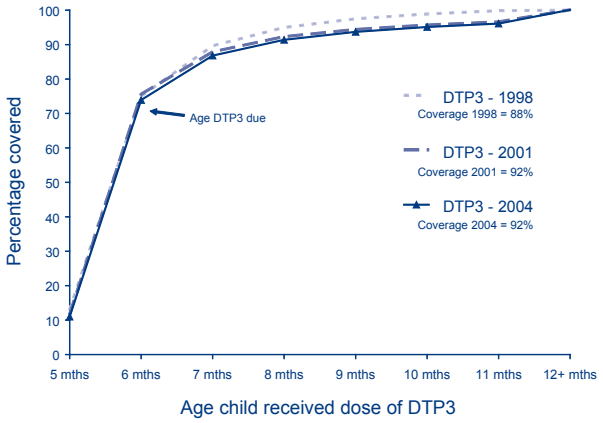
around 50% (Figure 9). There is a large variation in 23vPPV coverage by jurisdiction from a low of 37% in South Australia to a high of 74.2% in the Northern Territory (Table 8).

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.

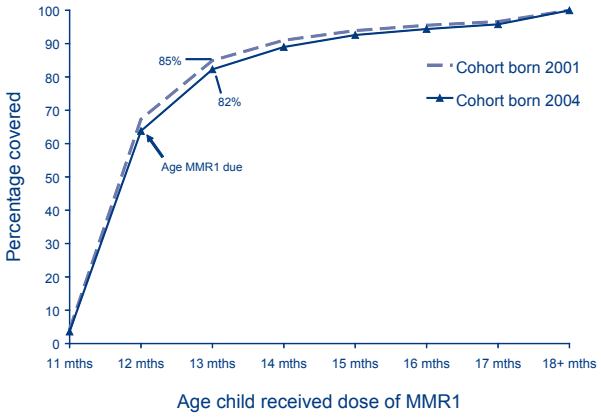
Since 1998, the proportion with timely receipt of DTP vaccine has decreased slightly, although coverage increased over this period from 88% to 92% (Figure 10). Across the 4-year period, 2001–2004, timely receipt of 1 dose of MMR vaccine also decreased by 3 percentage points, although estimated coverage by 24 months of age remained stable at almost 94% (Figure 11).

Figure 10: Trends in timeliness of the 3rd dose of DTP vaccine (DTP3) – cohorts born in 1998, 2001 and 2004*



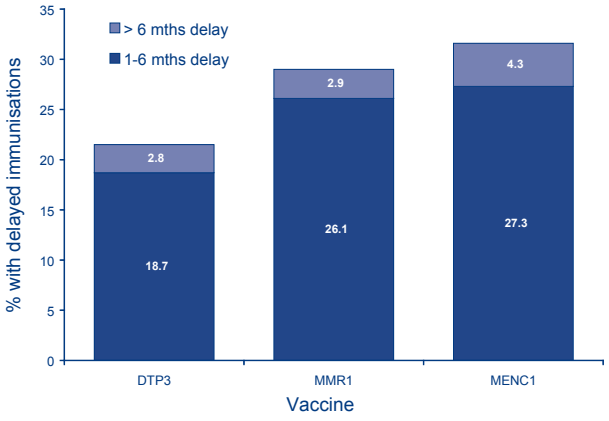
* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.

Figure 11: Trends in timeliness of the 1st dose of MMR vaccine (MMR1) – cohorts born in 2001 and 2004*



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

Figure 12: 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 2004

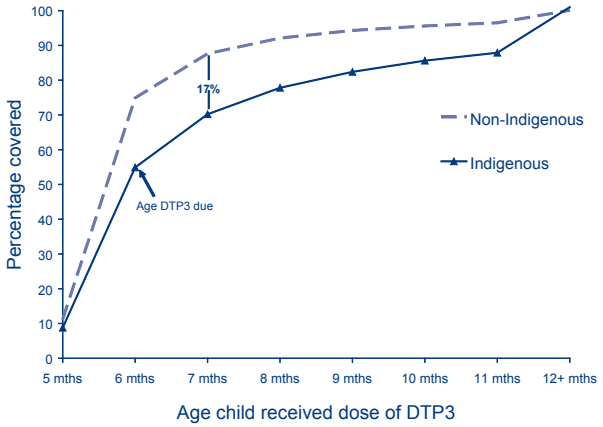


A comparison of vaccination delay for the 3rd dose of DTP, due at 6 months of age, and the 1st doses of MMR and meningococcal C, due at 12 months of age, for the 2004 cohort is shown in Figure 12. As demonstrated in previous studies, the proportion with vaccination delay increased with vaccine doses given at an older age. The greatest proportion with any delay was seen with meningococcal C vaccine with more than 30% of doses given late and over 4% given more than 6 months late.

Figures 13 and 14 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 a 17% differential at 7 months of age. A similar 17% differential was seen for the 3rd dose of 7vPCV (not shown). 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.

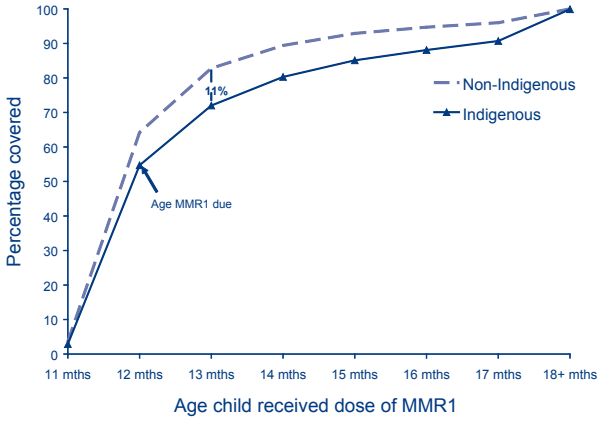
Vaccination delay for Indigenous children for 7vPCV varied by jurisdiction, with greater delays in Western Australia and South Australia (Figure 15). There were no important differences in vaccination delay for non-Indigenous children by jurisdiction (not shown).

Figure 13: Timeliness of the 3rd dose of DTP vaccine (DTP3) by Indigenous status – cohort born in 2004*



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

Figure 14: Timeliness of the 1st dose of MMR vaccine (MMR1) by Indigenous status – cohort born in 2004*



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

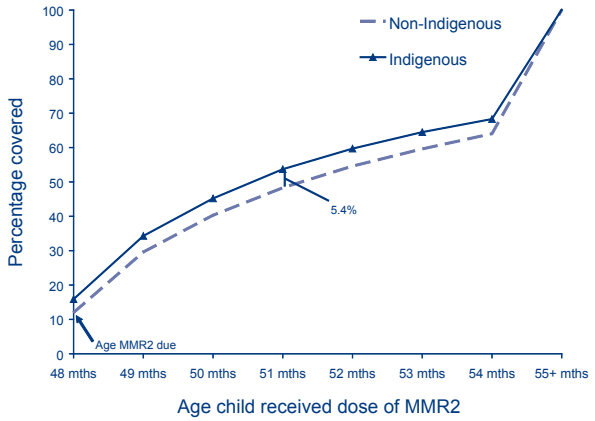
In contrast to earlier ages, analysis of timeliness of immunisation for a vaccine due at 4 years of age, the 2nd dose of MMR, showed more delay in receiving this vaccine for non-Indigenous children than Indigenous children, with a 5.4% differential at 4 years and 3 months of age (Figure 16).

Small area coverage

'Fully immunised' coverage for Australia by SSD for the 12-month, 24-month and 6-year milestone age groups, respectively, is shown in Figures 17–19. All 3 maps demonstrate that immunisation coverage in Australia in 2007 varies substantially within

Figure 15: Vaccination delay for Indigenous children for the 3rd dose of 7vPCV in selected jurisdictions – cohort born in 2005

Figure 16: Timeliness of the 2nd dose of MMR vaccine (MMR2) by Indigenous status – cohort born in 2001*



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

jurisdictions, with some having recorded coverage below the level required to prevent outbreaks of some highly contagious diseases such as measles.

The proportions of children recorded as conscientious objectors and with no vaccines recorded are presented by SSD in Figures 20 and 21, respectively. No vaccines recorded may represent either non-immunisation (parents refusing any vaccines and also not registered as a conscientious objector) or, and probably much less commonly, non-reporting by a provider. The percentage of children with no vaccines recorded nationally (3.4%) is greater than those recorded as conscientious objectors (1.1%).

The map of the proportion of conscientious objectors to immunisation (Figure 20) shows pockets of high levels of objection within jurisdictions in

Figure 17: 'Fully immunised' coverage at 12 months of age, Australia, 2008, by Statistical Sub-Division

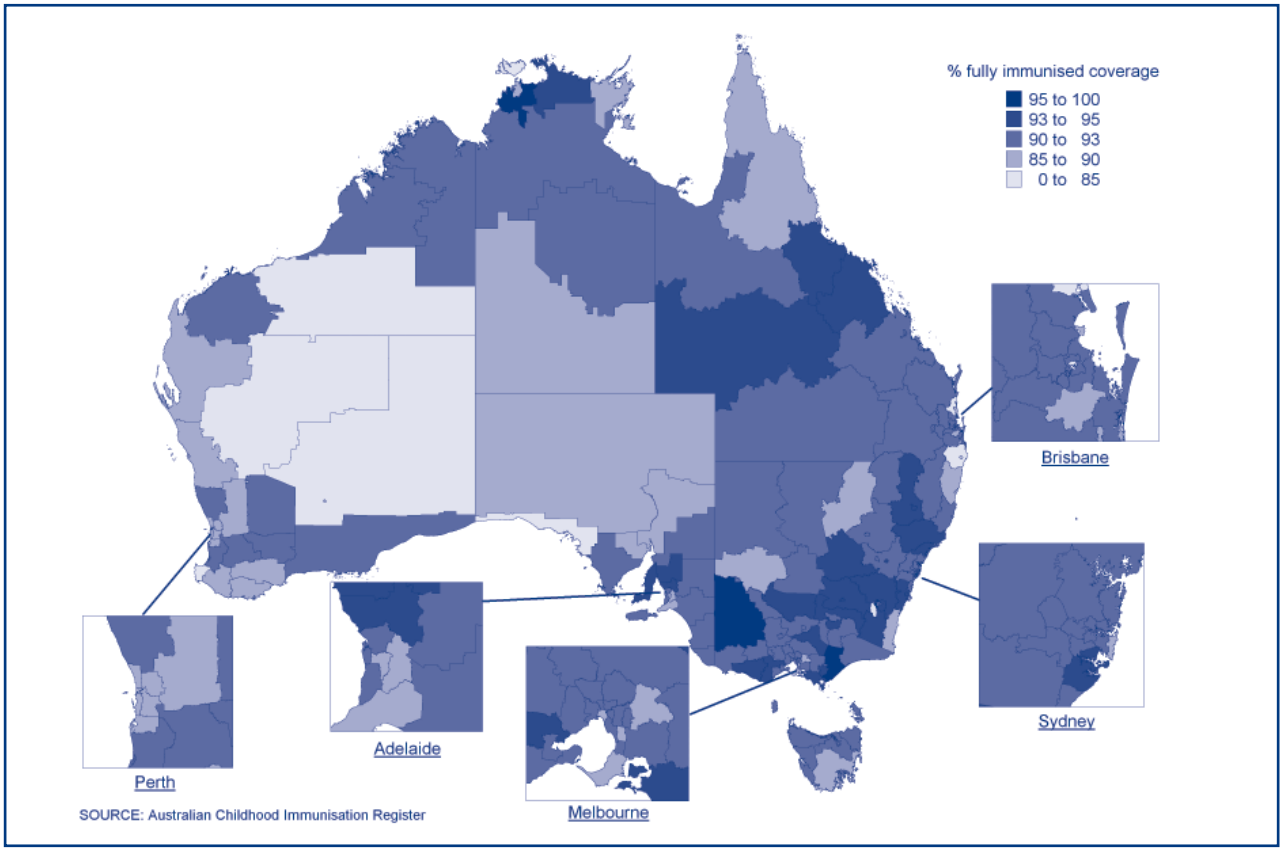


Figure 18: 'Fully immunised' coverage at 24 months of age, Australia, 2008, by Statistical Sub-Division

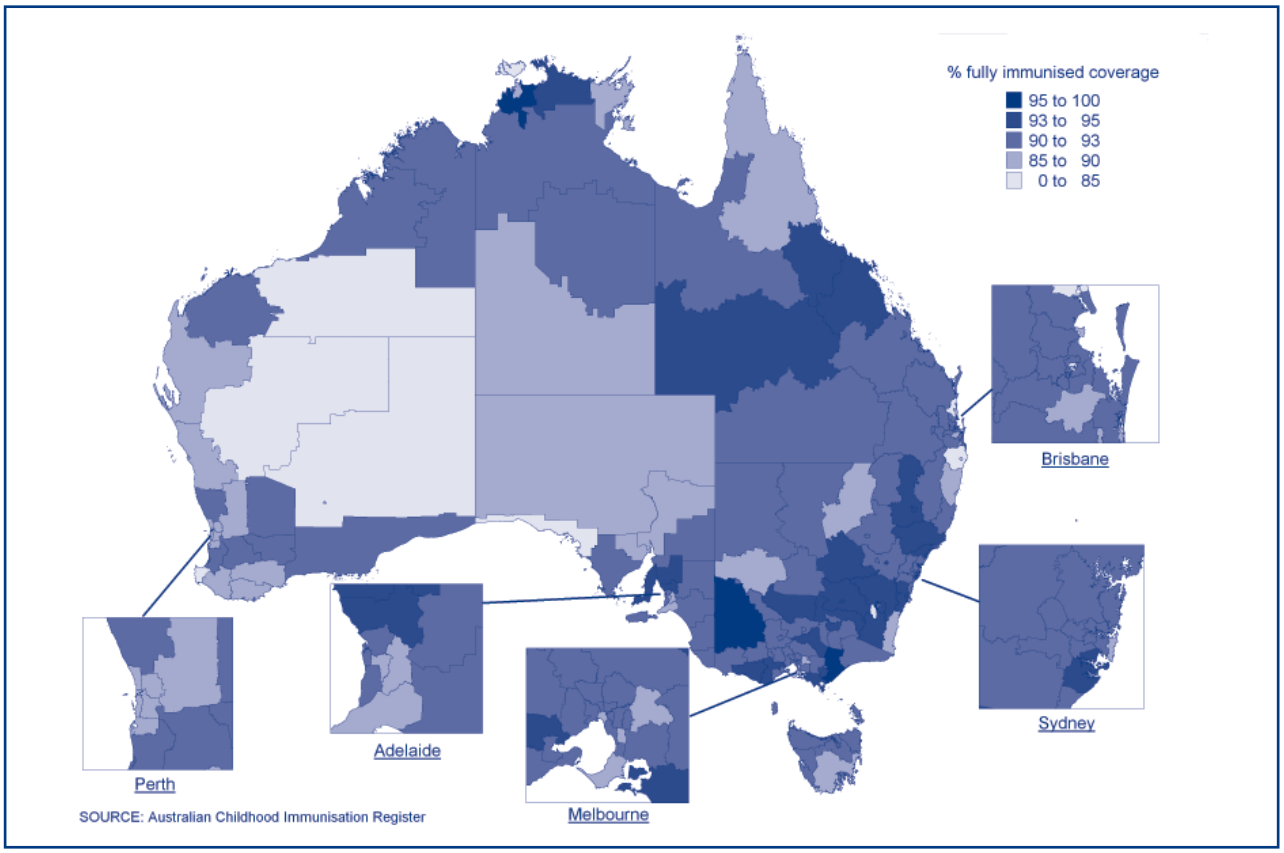


Figure 19: 'Fully immunised' coverage at 6 years of age, Australia, 2008, by Statistical Sub-Division

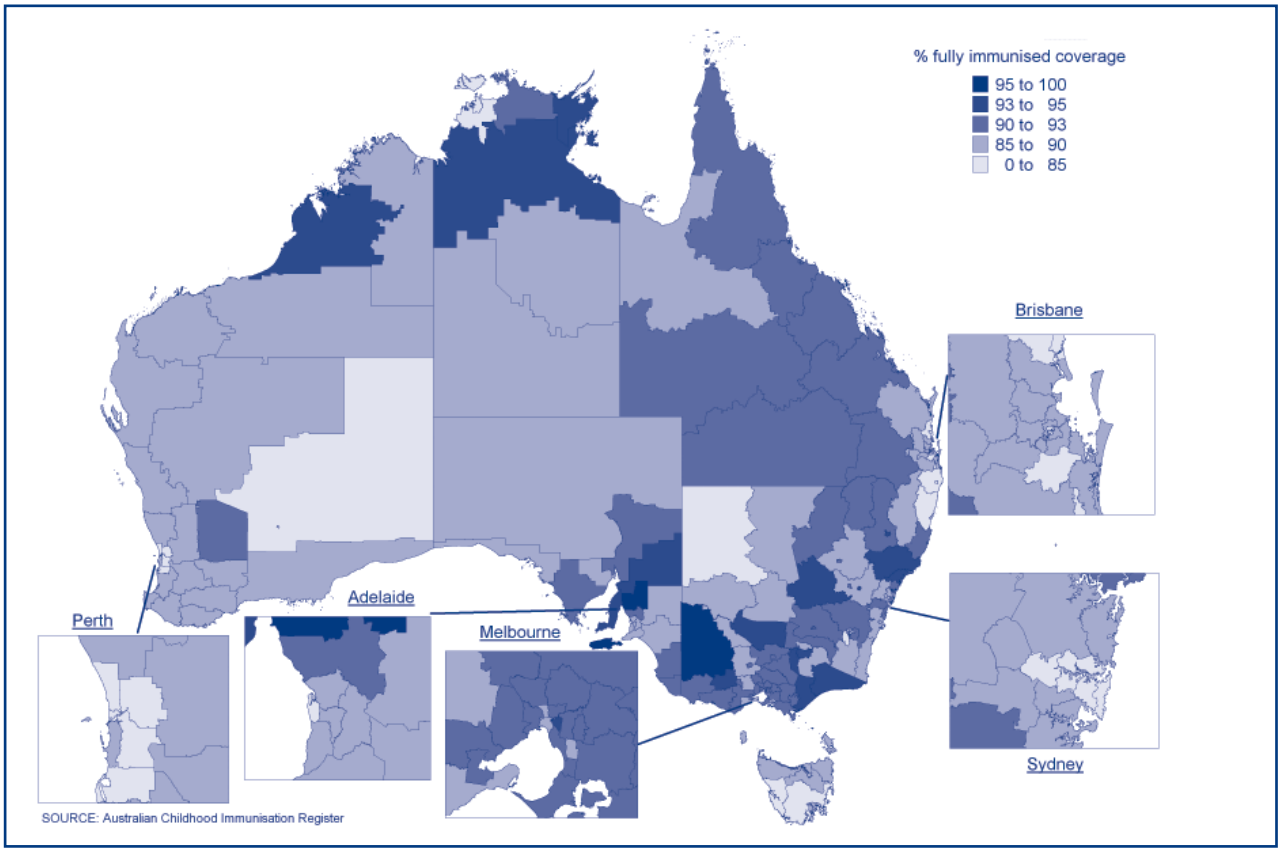
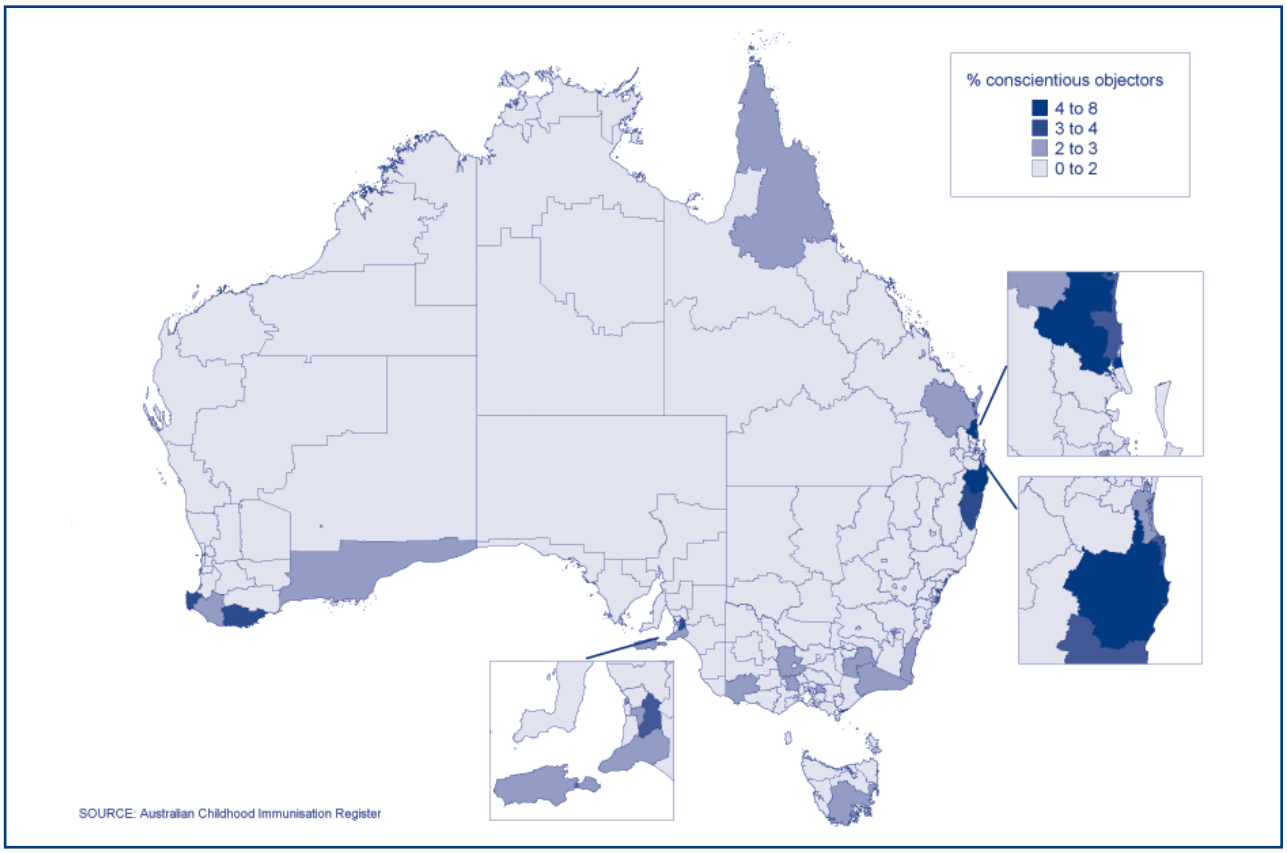


Figure 20: Proportion of official conscientious objectors to immunisation, Australia, 2007 (cohort born 1 January 2001 to 31 December 2006)



2007, particularly in coastal areas of south-east Queensland, northern New South Wales, Adelaide and south-western 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 21) 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 22. 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

Since its inception, the ACIR has grown to hold records for over 5.8 million children and receives reports from over 21,000 providers of immuni-

Figure 22: Proportion of immunisations on the ACIR given by various provider types, by state or territory, 2007

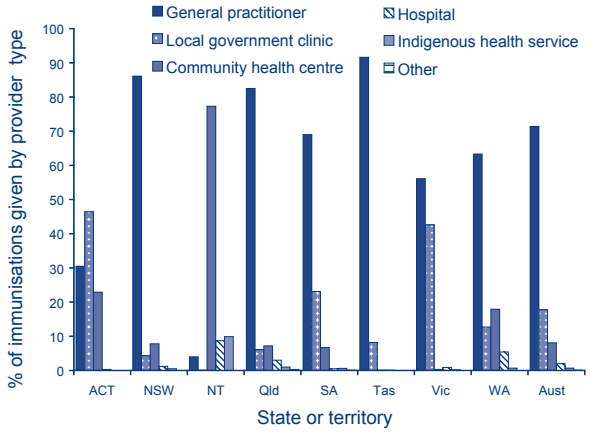
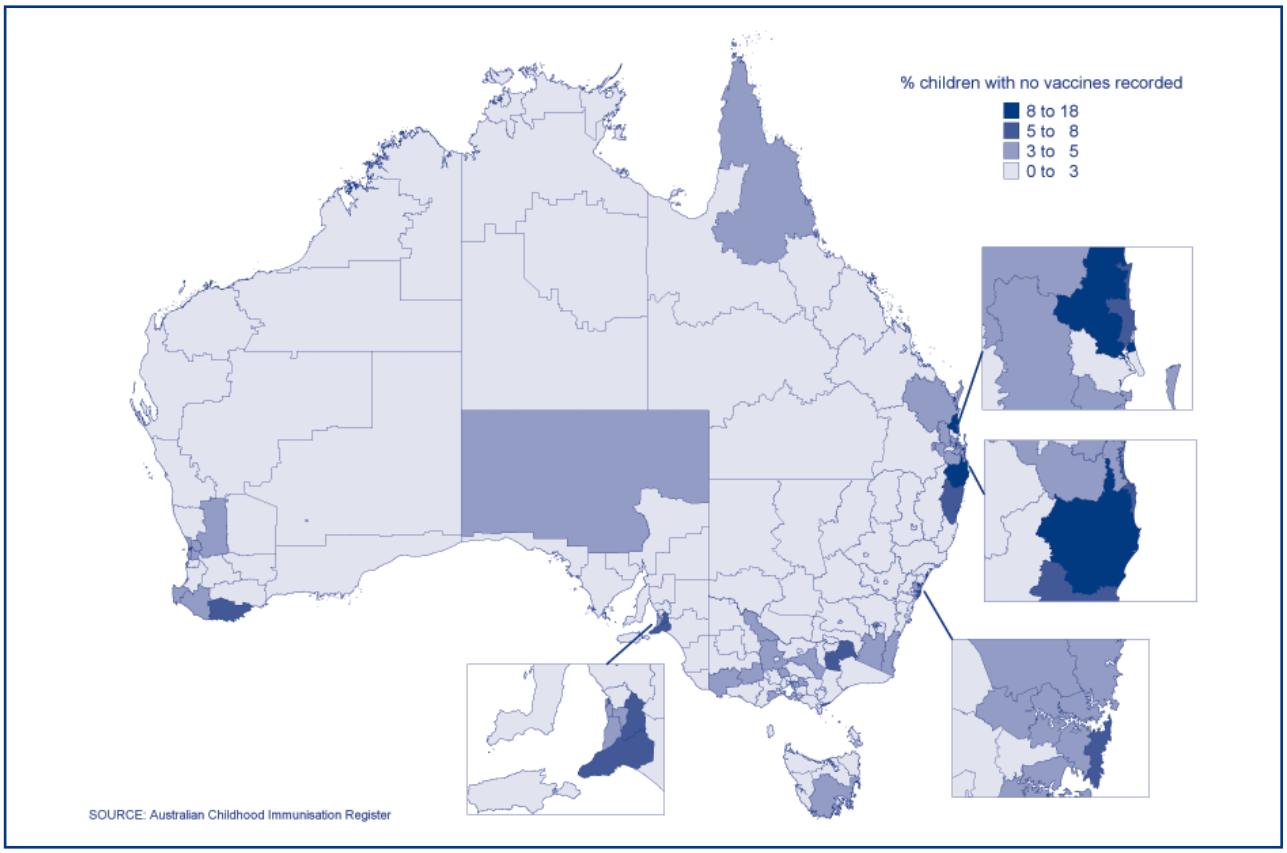


Figure 21: Proportion of children with no vaccines recorded on the ACIR, Australia, 2007 (cohort born 1 January 2001 to 31 December 2006)



sation services. It has become an increasingly valuable resource for administering and facilitating the National Immunisation Program as well as providing immunisation coverage data to monitor program performance. One of the main strengths of the ACIR is that it is a virtual census of children under 7 years of age in Australia, and, as such, has served as a model for other developed countries.[36,37]

However, there are some limitations to ACIR data. The only socio-demographic data collected by the ACIR are the age, postcode, sex and the Indigenous status of the child. This means public health researchers who use data from the ACIR to conduct research into immunisation coverage and other related issues are quite limited in the scope of the research they can undertake. Linkage with other datasets should be pursued in future to enhance its value in assessing control of vaccine preventable diseases. There are also limitations in data quality, which mainly relate to provider reporting. Improvements in coverage estimates can reflect improvements in provider reporting and timeliness as well as in actual coverage.

These data reveal that Immunise Australia Program coverage targets have been reached for children both 12 and 24 months of age and are being approached for children 6 years of age. With up to 3% of Australian parents not immunising their children for philosophical or religious reasons,[28] it will be difficult for 'fully immunised' coverage estimates to exceed 95%, especially as the reporting of immunisation encounters is still not totally complete.

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. This is likely related to the removal of the 18-month booster dose of DTP, as well as the impact of immunisation incentives. Coverage estimates for the 6-year age group also increased noticeably in June 2006. A possible factor in this increase in coverage is the introduction of the multivalent combination vaccine Infanrix-IPV in November 2005, which decreased the number of vaccines needing to be given and recorded on the ACIR. Other factors that may have had an impact at the local level include promotional campaigns targeting childcare or school entry.

Immunisation incentives have also positively impacted coverage estimates over time. 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 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 budget, 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. This policy change was designed to encourage immunisations in the older age groups where coverage estimates are lower compared with the younger age groups. It remains to be seen whether this will impact coverage for vaccines due after 24 months of age. In the same budget it was announced that GPII Service Incentive Payments would be removed from the incentives program in immunisation. It will be important to monitor the impact of these changes in future reports.

A number of vaccines in the NIP are not included when calculating 'fully immunised' status or in determining eligibility for incentive payments. Despite this, coverage data for the 7vPCV and meningococcal C vaccines is comparable with currently reported vaccines, while coverage for varicella is lower. However, there are variations by state and territory. 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' should be considered, although addition of more antigens will inevitably decrease coverage estimates for the various 'fully immunised' age categories.

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and pneumococcal polysaccharide vaccine) remain sub-optimal. This has been previously reported for other vaccines for both children[26] and adults,[38] and could be at least partly due to a lack of provider knowledge about the recommendations, poor identification of Indigenous children, and poor notification to the ACIR of vaccines for which there are no incentive payments. 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. Similarly, differences in the scheduling of pneumococcal polysaccharide vaccine by jurisdiction may partially explain the variation in coverage seen, 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 increased slightly. 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.

Author details

Brynley Hull
Shelley Deeks
Rob Menzies
Peter McIntyre

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, Westmead, New South Wales

Correspondence: Mr Brynley Hull, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead, Locked Bag 4001, WESTMEAD NSW 2145.

References

1. Commonwealth of Australia. Communicable Diseases Surveillance – additional reports: Childhood immunisation coverage. *Commun Dis Intell* 2008;32:122–123.
2. 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.
3. Commonwealth of Australia. Communicable Disease Surveillance – additional reports: Australian childhood immunisation coverage. *Commun Dis Intell* 2008;32:357–359.
4. Hull BP, McIntyre PB. Timeliness of childhood immunisation in Australia. *Vaccine* 2006;24:4403–4408.
5. Dombkowski KJ, Lantz PM, Freed GL. The need for surveillance of delay in age-appropriate immunization. *Am J Prev Med* 2002;23:36–42.
6. Strine TW, Luman ET, Okoro CA, McCauley MM, Barker LE. Predictors of age-appropriate receipt of DTaP dose 4. *Am J Prev Med* 2003;25:45–49.
7. Hanna JN, Wakefield JE, Doolan CJ, Messner JL. Childhood immunisation: factors associated with failure to complete the recommended schedule by two years of age. *Aust J Public Health* 1994;18:15–21.
8. Cotter JJ, Bramble JD, Bovbjerg VE, Pugh CB, McClish DK, Tipton G, et al. Timeliness of immunizations of children in a Medicaid primary care case management managed care program. *J Natl Med Assoc* 2002;94:833–840.
9. Trauth JM, Zimmerman RK, Musa D, Mainzer H, Nutini JF. Do beliefs of inner-city parents about disease and vaccine risks affect immunization? *J Natl Med Assoc* 2002;94:820–832.
10. Ehresmann KR, White KE, Hedberg CW, Anderson E, Korlath JA, Moore KA, et al. A statewide survey of immunization rates in Minnesota school age children: implications for targeted assessment and prevention strategies. *Pediatr Infect Dis J* 1998;17:711–716.
11. Luman ET, McCauley MM, Stokley S, Chu SY, Pickering LK. Timeliness of childhood immunizations. *Pediatrics* 2002;110:935–939.
12. Luman ET, Barker LE, Shaw KM, McCauley MM, Buehler JW, Pickering LK. Timeliness of childhood vaccinations in the United States: days undervaccinated and number of vaccines delayed. *JAMA* 2005;293:1204–1211.
13. Dannetun E, Tegnell A, Hermansson G, Torner A, Giesecke J. Timeliness of MMR vaccination—influence on vaccination coverage. *Vaccine* 2004;22:4228–4232.
14. Australian Government Department of Human Services, Medicare Australia. General Practice Immunisation Incentives (GPII) Scheme. 2007. Available from: http://www.medicareaustralia.gov.au/provider/incentives/gpii/index.shtml Accessed on 18 December 2007.
15. Australian Government Department of Human Services, Medicare Australia. GPII Scheme: Service Incentive Payments (SIP). 2007. Available from: http://www.medicareaustralia.gov.au/provider/incentives/gpii/service-payments.shtml Accessed on 6 December 2007.
16. Hull B, McIntyre P. Mapping immunisation coverage and conscientious objectors to immunisation in NSW. *N S W Public Health Bull* 2003;14:8–12.
17. Hull B, Lawrence G, MacIntyre CR, McIntyre P. Immunisation coverage: Australia 2001. Canberra: Commonwealth Department of Health and Ageing, 2002.
18. Hull B, McIntyre P. Mapping immunisation coverage and conscientious objectors to immunisation in NSW. *N S W Public Health Bull* 2003;14:8–12.
19. 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;28:55–60.
20. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Immunisation coverage in Australia corrected for underreporting to the Australian Childhood Immunisation Register. *Aust N Z J Public Health* 2003;27:533–538.
21. 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;24:17–21.
22. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Estimating immunisation coverage: is the 'third dose assumption' still valid? *Commun Dis Intell* 2003;27:357–361.
23. Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? *Commun Dis Intell* 2004;28:238–243.
24. 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;28:47–52.
25. 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;32:1041–1043.
26. 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;32:257–260.
27. Lawrence GL, MacIntyre CR, Hull BP, McIntyre PB. Effectiveness of the linkage of child care and maternity payments to childhood immunisation. *Vaccine* 2004;22:2345–2350.

28. Lawrence GL, Hull BP, MacIntyre CR, McIntyre PB. Reasons for incomplete immunisation among Australian children. A national survey of parents. *Aust Fam Physician* 2004;33:568–571.

29. National Health and Medical Research Council. The Australian immunisation handbook. 9th ed. Canberra: Australian Government Department of Health and Ageing, 2008.

30. O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. *Commun Dis Intell* 1998;22:36–37.

31. Department of Health and Aged Care. Measuring Remoteness: Accessibility/Remoteness Index of Australia (ARIA). Occasional Papers, New Series No.14. Canberra: Department of Health and Aged Care, 2001.

32. 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;31:283–287.

33. Australian Bureau of Statistics (ABS). Australian Standard Geographical Classification (ASGC), 2001. Cat. no. 1216.0. Canberra: ABS, 2001.

34. MapInfo [computer program]. Version 7. New York: MapInfo Corporation, 2002.

35. Australian Bureau of Statistics (ABS). Statistical Subdivision from Postal Area 2006 Concordance. Canberra: ABS, 2007. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/39433889d406eeb9ca2570610019e9a5/5942283858e38743ca25730c00009f2e!OpenDocument Accessed on 6 December 2008.

36. New Zealand Ministry of Health. Overview of the National Immunisation Register. 2004. Available from: http://www.moh.govt.nz/moh.nsf/0/FA74067C640C0F0FCC256E58000B2089/$File/OverviewoftheNationalImmunisationRegister.pdf Accessed on 6 December 2007.

37. Canavan BC, Kurilo M, Moss T, McLaren R, Berry K, Thomas C, et al. Immunization information systems progress—United States, 2005. *MMWR Morb Mortal Wkly Rep* 2006;55:1327–1329.

38. 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.

Surveillance of Creutzfeldt-Jakob disease in Australia: 2009 update

Genevieve M Klug, Alison Boyd, Victoria Lewis, Amelia McGlade, Christiane Stehmann, Colin L Masters, Steven J Collins

Abstract

In Australia, the occurrence of all human transmissible spongiform encephalopathies (TSEs) is surveyed by the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). While prospective surveillance commenced in October 1993, the ANCJDR also retrospectively ascertained cases that occurred between 1970 and 1993. During the surveillance period of 1 April 2008 to 31 March 2009, the ANCJDR received 90 suspect TSE case notifications, which is slightly increased from previous annual surveillance periods. Based on the total number of probable and definite CJD cases, ascertained between 1993 and 2009, the Australian age-adjusted mortality rate is 1.18 deaths per million per year. In this short report, we provide updated Australian human TSE figures and discuss a recently published investigation of geographical TSE clustering in regional New South Wales. *Commun Dis Intell* 2009;33:188–191.

Keywords: Creutzfeldt-Jakob disease, transmissible spongiform encephalopathies

Introduction

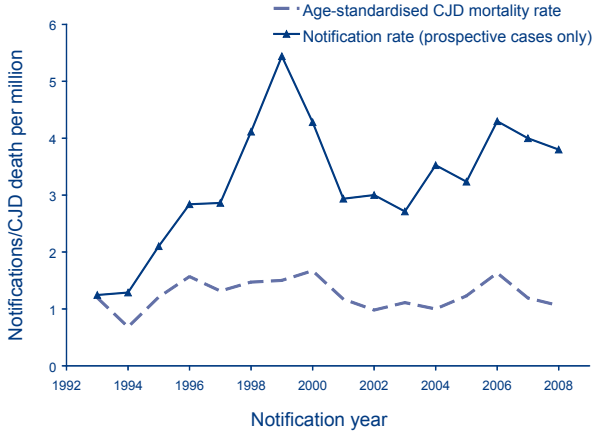
Transmissible spongiform encephalopathies (TSEs) comprise of a group of rare, neurodegenerative diseases with a reported worldwide incidence of approximately 1 case per million per year. TSEs encompass invariably fatal diseases: Creutzfeldt-Jakob disease (CJD), Gerstmann Sträussler-Sheinker syndrome, fatal familial insomnia and variant CJD (vCJD). Although the disease is classified as transmissible, most cases arise sporadically, with no plausible explanation. The remaining cases are related to either a genetic basis or an iatrogenic association through medical intervention. The World Health Organization case definition criteria form the basis for disease classification[1] and include the requirement of neuropathologic assessment of brain tissue for definite cases (either through biopsy or autopsy), while 'probable cases' are classified in accordance with recognised and validated clinical criteria.[2] 'Possible case' classification is based on defined criteria where there is a strong suspicion of CJD, but insufficient investigational evidence to support a probable classification and for this reason these cases are excluded from the following statistical analysis. CJD and vCJD have been notifiable in all Australian states and territories since June 2006 and are two of the 69 communicable diseases under national surveillance as defined by the National Notifiable Diseases List.

The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established in 1993 to provide nation-wide surveillance for all human TSE regardless of aetiology, and to offer specialised diagnostic services, including cerebrospinal fluid 14-3-3 protein analysis.

Australian National Creutzfeldt-Jakob Disease Registry surveillance update

Since 1 October 1993, the ANCJDR has been notified of 1,345 suspected cases of CJD, arising in both the prospective and retrospective ascertainment periods. For the prospective period, the average annual rate of suspect cases notified to the ANCJDR was 3.1 per million per year. Fluctuations in these annual notification rates have been observed (Figure 1) and the reasons for these have been discussed previously.[3] More recently, the rate of notifications from 2006 to 2008 has been sustained at a higher level in comparison to the longer term average observed for 1993–2008. The increased number of notifications is most likely underpinned by increased referrals to the ANCJDR of cerebrospinal fluid for 14-3-3 protein testing, particularly for the 2007–2008 period where CSF referrals have increased by 43% in comparison with the previous 6 year average. This sustained increased level of CSF referrals has given the ANCJDR confidence that the introduction of 'fee-for-service' from 1 January 2007 has not detrimentally affected CSF referrals and consequently rates of suspect case notification, as initially speculated.

Figure 1: Annual age-standardised Creutzfeldt-Jakob disease mortality rates and suspect case* notification rates, 1993 to 2008



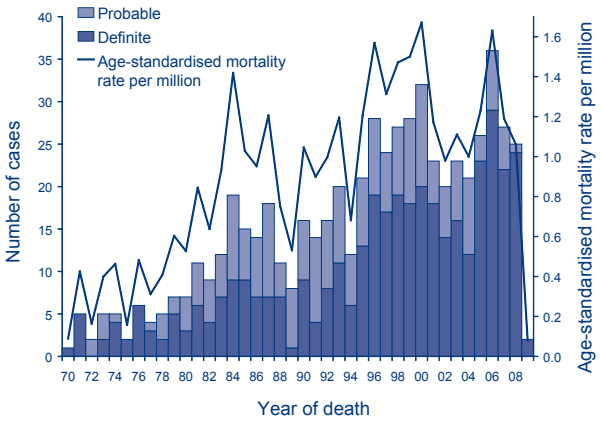
* Prospectively ascertained cases only.

During the 2008–2009 reporting period to 31 March 2009, 90 suspect cases were added to the Register, which is a 17% increase from the previous surveillance year. Of these 90 suspect case notifications, 20 have been reclassified as definite TSE cases. A further 15 cases that were notified prior to 1 April 2008 have also been confirmed as definite (11) or probable (4) TSE since the last update. The Register has a large group of cases still under investigation (184), with the majority of these still alive as at 31 March 2009. Despite active investigation of all suspect cases, the number of incomplete cases continues to expand, although final outcomes for the large majority of all suspect cases have been obtained through detailed investigation. The number of incomplete cases is of concern to the ANCJDR as for many of these cases, a conclusive outcome may not be achievable. Follow-up can be difficult for cases that have been notified several years previously. In 541 cases, CJD has been excluded while 608 cases have been classified as definite (395) or probable (213) CJD and a further 12 cases fulfil the possible case definition (Table 1). A sustained elevation of the annual CJD incidence and the proportions of autopsy-confirmed cases for the surveillance years of 2005–2008 has been observed (Figure 2). As previously discussed, this relates to the growing number of notifications and pro-active approach of the ANCJDR of seeking autopsy in all clinically suspect cases.[3]

The aetiologic proportions of all Australian TSE cases are consistent with previous observations.[3] Cases classified as sporadic CJD comprise 90.5% of all Australian cases, while 8.2% of cases are genetic and the remaining 1.3% are iatrogenic. During the 2008–2009 surveillance period, 4 new cases of familial TSE were classified, while no further iatrogenic CJD and no cases of vCJD were identified in Australia.

Based on the 608 definite and probable cases, TSE incidence peaks at 4.9 cases per million per year in the 65–69 year age group, an incidence almost 5 times the reported global incidence. As sporadic cases comprise the majority of cases, the peak incidence in this group closely aligns with overall TSE rates. Since the last surveillance period, the median age at death for sporadic cases has remained unchanged; 66 years (males, 65 years; females, 67 years), the proportion of female sporadic cases has remained consistent at 53% and their median duration of disease similar at 4 months. A slightly shorter disease duration is observed in males (median, 3 months). Genetically determined TSEs have a younger age at death (medians, overall 59 years; males, 51 years; females, 62 years) and longer illness duration (medians, overall 6 months; males, 4 months; females, 7.5 months) when compared to sporadic cases. The sex ratio for familial cases is slightly biased towards females with 58% of the cases being female.

Figure 2: Number and age-standardised mortality rate for definite and probable cases of Creutzfeldt-Jakob disease, Australian National Creutzfeldt-Jakob Disease Registry, 1970 to 2009*



* To 31 March 2009.

Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australia.

Table 1: Classification of cases by the Australian National Creutzfeldt-Jakob Disease Registry, 1 January 1970 to 31 March 2009

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	350	40	5*	0	0	395
Probable	199	10	4	0	0	213
Possible	11	0	1	0	0	12
Incomplete	0	0	0	0	184†	184
Total	560	50	10	0	184	804

* Includes 1 definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

† Includes 128 living cases.

Based on all definite and probable TSE cases, the average, age-adjusted mortality rate in Australia for the 1970–2009 period is 0.88 deaths per million per year. A restriction of the timeframe to the prospective surveillance period of 1993–2009 provides a more reliable representation of the true national figures with a mortality rate of 1.18 deaths per million per year. By individual state and territory, the TSE mortality rates (Table 2) in some jurisdictions have altered since the previous update.[3] Notably, the average age-standardised mortality rates during 1993–2009 have increased in Western Australia, the Australian Capital Territory and the Northern Territory, while in Tasmania, the rate has continued to decline and is currently around half the rate observed in Victoria and Western Australia (Table 2). This strongly suggests case under-ascertainment in Tasmania for this specific period. In the remaining states and territories, no significant changes in the longer-term mortality rate average (1993–2009) have occurred since last reported.

Analysis of incidence rates by state and territory over the last decade highlights the strengths and weaknesses of surveillance in the various regions (Table 2). In Victoria, case numbers have remained constant over this period and have resulted in the highest mortality rate in Australia. In contrast, a lower than expected mortality rate in Queensland, South Australia and Tasmania was observed. This decline was concerning as the 10-year timeframe provided us with a recent snapshot of confirmed cases, excluding the diluting influence of the earlier prospective surveillance years. Broadened surveillance and diagnostic responsibilities, changes to privacy legislation around 2000 and less accessible autopsy services in various regions may have contributed to the lower mortality in the specific states.

Suspect case notification between this and the previous reporting period have remained stable in the Australian Capital Territory, the Northern Territory, South Australia and Western Australia (Figure 3). In contrast, a 70% increase in notifications has been observed in Victoria. While the total number of CSF referrals arising in Victoria has remained unchanged, there has been an increase in the number of clinically suspect TSE cases added to the Register, derived from CSF referrals. Marked declines in notifications in the large populations of New South Wales and Queensland, and to a lesser degree in Tasmania, were observed in 2008. The impact of these lower notifications may be reflected in a reduced number of confirmed CJD cases for this period. A contractual agreement between the ANCJDR and Queensland Health Department was established in May 2008 to evaluate all cases of suspect TSE. The impact of this agreement on TSE incidence in Queensland will be of particular interest.

Figure 3: Prospective, suspect CJD case notifications to the Australian National Creutzfeldt-Jakob Disease Registry, 1993 to 2008, by state and territory

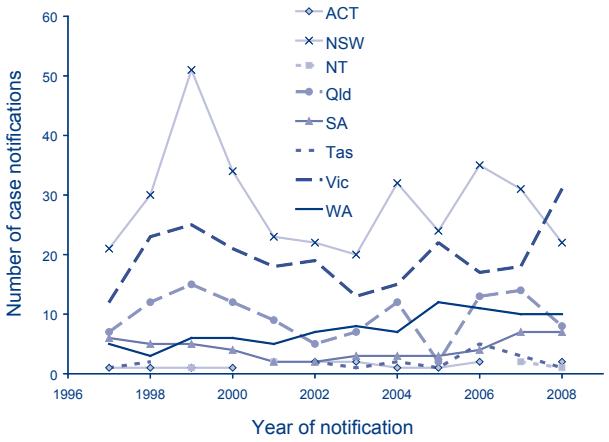


Table 2: Transmissible spongiform encephalopathy deaths and mortality rate, by state and territory

State or territory	TSE cases by year of death										Total TSE deaths	Mean age-adjusted mortality rate (deaths/million/year)	
	00	01	02	03	04	05	06	07	08	09		00–09*	93–09*
ACT			1		1		1		2		5	1.42	1.32
NSW	12	9	7	7	11	10	11	10	5	1	83	1.19	1.18
NT							2	1			3	0.96	0.88
Qld	7	3	3	3			6	4	2		28	0.69	0.97
SA	2			1	2		1	3	3		12	0.73	1.09
Tas			2			1	2				5	0.93	0.67
Vic	9	10	5	9	5	11	9	5	11	1	75	1.45	1.37
WA	2	1	2	3	2	4	4	6			24	1.13	1.35
Australia	32	23	20	23	21	26	36	27	25	2	235	1.11	1.18

* Includes all deaths occurring between 1 January 1993 or 1 January 2000 and 31 March 2009.

Analysis of a potential Creutzfeldt-Jakob disease cluster

During 2008, the ANCJDR published findings from an investigation conducted assessing an increased number of sporadic CJD cases within a coastal region of New South Wales during the period 1993–2006.[4] Statistical analysis identified a spatially significant cluster in 3 contiguous statistical local areas, consisting of 13 definite and 1 probable CJD case. An epidemiological review of ANCJDR case data for the 14 cases did not reveal a plausible cross-over or point source transmission event to explain the cluster of cases.

One potential hypothesis for the significant finding related to the region's clinicians and their management of potential CJD cases. To investigate this theory, further evaluation was undertaken comparing the regional area with the entire state, emphasising rates of referrals for 14-3-3 CSF testing, rates of case notification to the ANCJDR and suspect CJD post-mortem rates. These observations were chosen to objectively quantify an intensity of surveillance and how this relates to incidence rates.

Our analyses demonstrated that the cluster area maintained a higher level of surveillance and clinical awareness compared with the entire State of New South Wales. The population-based rate of notification of all suspect cases to the ANCJDR was 68% greater in the cluster area than for New South Wales (age-adjusted RR_{MH}: 1.68, 95% CI=1.36–2.10) and similarly, the population-based rate of request for CSF testing was 59% greater than the state referral rate (age-adjusted RR_{MH}: 1.59, 95% CI=1.25–2.02). No difference between the likelihood of a suspect case being confirmed as probable or definite CJD (all types or sporadic only) was observed, suggesting that once CJD was questioned as a diagnosis in a clinical setting, the likelihood of a case being assessed for CJD classification was no different in the circumscribed area to the entire state. In contrast, a difference did exist in the proportion of cases that were assessed by neuropathological examination (biopsy or autopsy), with the cluster area having an almost two and half times greater neuropathological examination rate in suspect cases compared with New South Wales (age-adjusted RR_{MH}, 2.34, 95% CI=1.56–3.51). Simply stated, approximately double the intensity of surveillance translated to a doubling of the CJD incidence rate.

One of the distinguishing features of the 14 cluster cases provided another key piece of supporting evidence for enhanced surveillance. The cohort displayed a significantly older age at death when compared with sporadic CJD cases from New South Wales and Australia overall. Analysis of autopsy data in Austria, where autopsy of all suspect CJD cases is mandatory, suggests global under-ascertainment of older age CJD cases.[5] Hence, the ability to detect older and less typical cases in this cluster region suggests clinicians manifested a greater than usual suspicion of CJD and atypical presentations.

These findings have provided us with a hypothesis that intensity of surveillance for rare disorders can be quantified and this can positively correlate with higher incidence. It further suggests that the true incidence of CJD in Australia may be almost twice the currently observed average rate of 1.18 cases per million per year. A further exploration of this hypothesis is needed within and between individual nations and may give us an improved understanding of methodologies for optimal surveillance for rare conditions such as CJD.

Acknowledgements

The ANCJDR wishes to thank families, medical practitioners and associated staff for their generous support of Australian CJD surveillance. The ANCJDR also thanks Dr Handan Wand, Dr Matthew Law and Professor John Kaldor (National Centre in HIV Epidemiology and Clinical Research at the University of New South Wales) for their expert epidemiological and statistical support.

Author details

Miss Genevieve M Klug, Research Assistant
Ms Alison Boyd, Registry Co-ordinator
Miss Victoria Lewis, Research Assistant
Miss Amelia McGlade, Research Assistant
Dr Christiane Stehmann– Administrative Assistant
Prof. Colin L Masters, Director
Assoc. Prof. Steven J Collins, Director

Corresponding author: Miss Genevieve Klug, Australian National Creutzfeldt-Jakob Disease Registry, Department of Pathology, The University of Melbourne, Victoria 3010. Telephone: +61 8344 1949. Facsimile: +61 9349 5105. Email: gmjak@unimelb.edu.au

References

1. World Health Organization. WHO manual for surveillance of human transmissible spongiform encephalopathies including variant Creutzfeldt-Jakob disease, 2003.
2. Will RG. Prion related disorders. *J R Coll Physicians Lond* 1999;33(4):311–315.
3. 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.
4. Klug GM, Wand H, Boyd A, Law M, Whyte S, Kaldor J, et al. Enhanced geographically restricted surveillance simulates sporadic Creutzfeldt-Jakob disease cluster. *Brain* 2009;132:493–501.
5. Gelpi E, Heinzl H, Hoftberger R, Unterberger U, Strobel T, Voigtlander T, et al. Creutzfeldt-Jakob disease in Austria: an autopsy-controlled study. *Neuroepidemiology* 2008;30(4):215–221.

Peer-reviewed articles

Encephalitis in Australia, 1979–2006: trends and aetiologies

Clare Huppatz, Paul M Kelly, Christopher Levi, Craig Dalton, David Williams, David N Durrheim

Abstract

The acute encephalitis syndrome has heralded the emergence of multiple virulent pathogens, including Murray Valley encephalitis, Hendra virus and Australian bat lyssavirus, which may result in severe morbidity and mortality. In Australia, encephalitis is not notifiable and there has been no analysis of trends in encephalitis death rates or causation. Australian Bureau of Statistics mortality and population data for the period 1979–2006 were obtained and cause of death data were extracted using ICD-9 (1979–1998) and ICD-10 (1999–2006) codes that included all relevant encephalitis related diagnoses. Encephalitis-associated deaths were analysed by cause, year, age and gender. Between 1979 and 2006 there were 1,118 encephalitis-associated deaths in Australia. The average annual death rate was 2.3 per 1 million population (range 1.3–3.6). There was a significant decline in encephalitis-associated deaths, particularly due to 'known' pathogens (4.3% decline per year, 95%CI 3.1–5.4%, $P<0.0001$). The aetiology of 576 deaths were unknown and the proportion of deaths due to 'unknown' encephalitis increased from 47.0% between 1979 and 1992, to 57.2% from 1993 to 2006. Downward trends in encephalitis deaths due to 'known' causes can largely be explained by changes in treatment and prevention methods, particularly for herpes encephalitis (use of acyclovir), and measles encephalitis and subacute sclerosing panencephalitis (measles vaccination). The high proportion of encephalitis deaths from 'unknown' pathogens in Australia highlights the importance of monitoring encephalitis morbidity and mortality with a view to improving pathogen diagnosis and identifying emerging infectious diseases. *Commun Dis Intell* 2009;33:192–197.

Keywords: encephalitis; viral encephalitis; infectious encephalitis; Australia; emerging infectious disease

Introduction

The syndrome of encephalitis frequently presents with fever, headache and an altered level of consciousness,[1,2] signifying the underlying pathology of an inflammatory process in the brain's parenchyma.[1,3,4] Treatment in many cases is supportive, and outcomes often include severe morbidity and even death.

A variety of pathogens cause encephalitis, with herpes simplex virus being the most commonly identified pathogen reported in developed countries.[5,6] While many encephalitis causing pathogens do not have an effective treatment, herpes simplex virus is a notable exception. Acyclovir significantly reduces the mortality associated with herpes encephalitis[7,8] from 70% when untreated to approximately 25%–30%.[2,9,10] Other pathogens, such as rabies and rabies-like viruses, carry a mortality approaching 100%.[11]

Often no pathogen is identified in people presenting with the encephalitis syndrome.[12–14] This lack of pathogen diagnosis makes this syndrome a public health challenge, as effective control measures usually depend on an understanding of the underlying epidemiology. In the last 70 years there has been a global increase in emerging infectious diseases (EIDs),[15] including several zoonoses and arboviruses that cause an encephalitis syndrome. Some of these pathogens (such as West Nile virus and Nipah virus) have caused large outbreaks allowing relatively rapid detection and public health action.[16,17] In Australia the emergence of Australian bat lyssavirus[18] and Hendra virus,[19] which presented with very few cases rather than large outbreaks, highlights the need to adequately investigate all cases of encephalitis, so that novel pathogens are not missed.

Several studies from the United States of America (USA) and Europe have considered the burden of disease associated with encephalitis and trends in causation over time. Khetsuriani et al analysed national data for the USA between 1988 and 1997 to estimate the burden of both viral and non-viral encephalitis hospitalisations.[14] They found that the hospitalisation rate for encephalitis in the USA was 7.3 per 100,000 population and 59.5% did not have a specific cause identified.[14] During the study period a case-fatality rate of 7.4% was recorded.[14]

A trend analysis of USA mortality statistics over a longer period showed that the rate of encephalitis related deaths in the USA population between 1979 and 1998 remained stable.[20] This was shown to be

largely due to the impact of HIV infection trends while there was a 27% decline in the rate of encephalitis related deaths for non-HIV infected people, which fell from 4.7 per 1 million between 1979 and 1988 to 3.6 per 1 million between 1989 and 1998.[20] The authors attributed this latter decrease in part to the widespread use of acyclovir, which resulted in a decline in deaths from herpes encephalitis.[20]

A review from the United Kingdom (UK) of viral encephalitis hospitalisations over the period 1989 to 1998, found a hospitalisation rate of 1.5 cases per 100,000 population.[12] During the study period, there were 419 deaths, of which 50% were attributed to an unknown viral aetiology.[12] A Finnish study found that a similar proportion (49%) of patients hospitalised with encephalitis between 1967 and 1991 had no aetiology identified.[21]

A recent investigation into the causes of encephalitis hospitalisation in New South Wales between 1990 and 2006 found an average annual rate of encephalitis hospitalisation of 5.2 per 100,000 (range, 4.2–6.7), with 69.6% of the total admissions due to 'unknown' pathogens (Huppatz et al, unpublished). The case fatality rate for encephalitis during this time period was 4.6% (Huppatz et al, unpublished).

In Australia, public health surveillance of encephalitis is limited to laboratory confirmed encephalitis cases due to a limited number of specific pathogens. There is no active or passive surveillance for encephalitis as a syndrome, and the burden of disease associated with encephalitis is not known. This study aimed to document the burden and trends in encephalitis-associated deaths in Australia between 1979 and 2006.

Methods

Mortality and population data for Australia were obtained for the 28-year period 1979 to 2006 from the Health Outcomes and Information Statistical Toolkit (HOIST), a collection of databases maintained by the Epidemiology and Surveillance Branch of the NSW Department of Health. Datasets used were from the Australian Bureau of Statistics 'population' library and 'death' library, the latter of which allows data extraction using International Classification of Diseases (ICD) codes. Encephalitis-associated deaths were extracted using ICD-9 (1979–1998) and ICD-10 (1999–2006) codes. To maximise comparability, data prior to 1979, which used ICD-8 codes, were not included and data after 2006 were not included, as it was incomplete at the time of analysis.

An encephalitis-associated death was defined as a death for which the primary cause of death was an ICD-9 or ICD-10 code for encephalitis (Table). These codes were further classified by investigators as 'known pathogen' or 'unknown pathogen' codes (Table).

Table: ICD-9 and ICD-10 codes most frequently used for primary cause of death for encephalitis-associated deaths, Australia, 1979 to 2006

Primary cause of death ICD Code (ICD 9; ICD 10)	Number of deaths	% of total
All encephalitis deaths	1,118	100
Known pathogen codes	542	48.5
Herpesviral encephalitis (54.3; B00.4)	300	26.8
Subacute sclerosing panencephalitis (46.2; A81.1)	81	7.2
Measles – postmeasles encephalitis (55.0; B05.0)	44	3.9
Late effects/sequelae of viral encephalitis (139.0; B94.1)	26	2.3
Zoster encephalitis (B02.0 in ICD 10)	17	1.5
Australian encephalitis (0.62.4; A83.4)	13	1.2
Other specified non-arthropod-borne viral diseases of the central nervous system (49.8; A85.8)	13	1.2
Listerial meningitis and meningoencephalitis (A32.1 in ICD 10)	11	1.0
Varicella encephalitis (052.0; B01.1)	8	0.7
Meningococcal infection – meningococcal encephalitis (36.1 in ICD 9)	6	0.5
Other known pathogen codes (= 10 codes)	23	2.1
Unknown pathogen codes	576	51.5
Encephalitis, myelitis, and encephalomyelitis – unspecified cause of encephalitis (323.9; G04.9)	327	29.2
Unspecified non-arthropod-borne viral diseases of the central nervous system or unspecified viral encephalitis (49.9; A86)	238	21.3
Encephalitis, myelitis, and encephalomyelitis – other cause of encephalitis (323.8; G04.8)	11	1.0

Encephalitis-associated deaths were analysed (using SAS version 8) by aetiological category, year, age and gender. Death rates for total, and 'known' and 'unknown' encephalitis deaths were calculated for individual years using mid-year population data from the Australian Bureau of Statistics. Negative binomial regression was used to analyse trends in the crude death rate of all encephalitis deaths and those due to 'known' and 'unknown' aetiology. The regression was repeated with adjustment for population age groups over time.

To compare death rates over time, the average age-specific death rates for the periods 1979–1985, 1986–1992, 1993–1999 and 2000–2006 were calculated using the average number of deaths in each 10-year age group during each time period and the population in the mid-point of each time period. All rates were expressed per 1 million population and the 7 year time periods were chosen to manage small numbers in shorter time periods. Small numbers of deaths in each category precluded determination of rates for individual causes. The annual number of deaths due to the most commonly identified causes were compared over the time periods specified above.

Ethical approval was given by the Hunter New England and the Australian National University Human Research Ethics Committees.

Results

From January 1979 to December 2006, there were 1,118 deaths in Australia with a primary cause of death recorded as encephalitis. The average number of deaths per year was 40.0 (range=26–52). The aetiology of 576 deaths (51.5% of total deaths) were unknown. During the 28 year study period the proportion of deaths due to 'unknown' encephalitis increased from 47.0% between 1979 and 1992 to 57.2% from 1993 to 2006.

The average annual rate of encephalitis deaths was 2.3 per 1 million (range 1.3–3.6). Males accounted for 51.7% of encephalitis deaths.

There was a decline in the crude death rate of all encephalitis deaths and those in the 'known' category (Figure 1) between 1979 and 2006. A negative binomial regression model demonstrated a significant decline of 2.3% per annum in the total encephalitis death rate (95%CI 1.6–3.2%, $P<0.0001$) and a decline of 4.0% (95%CI 3.0–5.0%, $P<0.0001$) in 'known' encephalitis deaths. There was no significant decline in the 'unknown' encephalitis deaths (0.72% decline, $P=0.12$) (Figure 1). When adjusted for age, the decline for total deaths was 3.0% per annum (95%CI 2.1–3.8%, $P<0.0001$) and for 'known' deaths was 4.3% (95%CI 3.1–5.4%, $P<0.0001$). A small decline was found for the 'unknown' death category (1.5% decline, 95%CI 0.4–2.7%, $P<0.01$).

Figure 1: Death rate (per 1 million population) from total encephalitis, 'known' and 'unknown' causes, Australia, 1979 to 2006

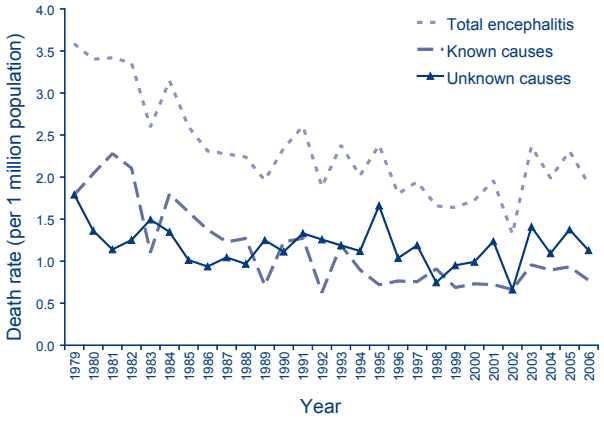
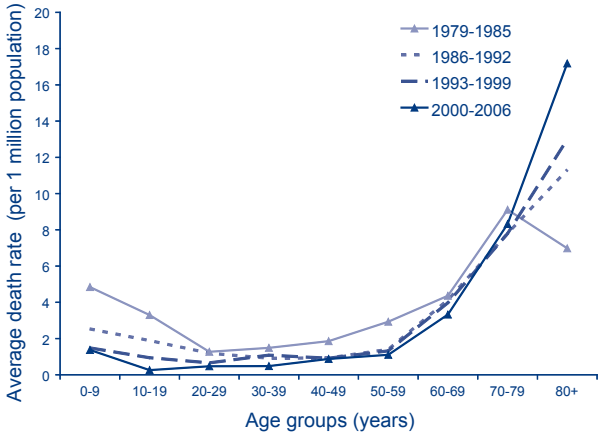


Figure 2 presents the average age-specific death rates for the time periods 1979–1985, 1986–1992, 1993–1999 and 2000–2006. The death rate in all age groups below 60 years decreased, with the most marked reduction in the younger age group (0–9 years). In the oldest age group (80+ years) the death rate increased over time.

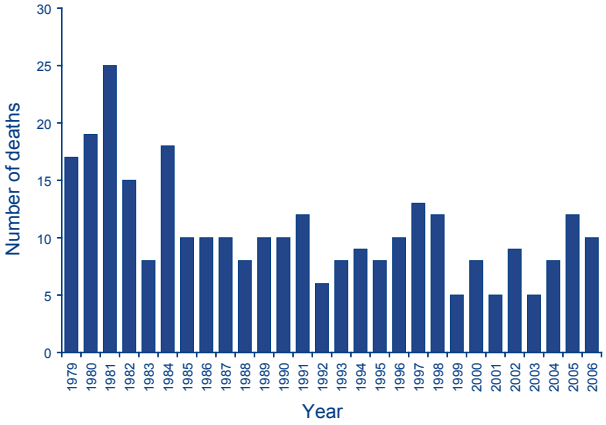
The most frequently identified cause of encephalitis was herpes simplex virus infection, which accounted for 300 deaths (26.8%) (Table). The average number of deaths decreased from 16.0 per year between 1979 and 1985, to 9.4 per year between 1986 and 1992 (Figure 3). Subsequently, the average number of

Figure 2: Trend in average death rates (per 1 million population) from encephalitis, Australia, 1979 to 2006, by 10 year age groups



deaths from herpes encephalitis remained relatively constant (9.3 per year, 1993–1999 and 8.1 per year, 2000–2006).

Figure 3: Number of herpes encephalitis deaths per year, Australia, 1979 to 2006

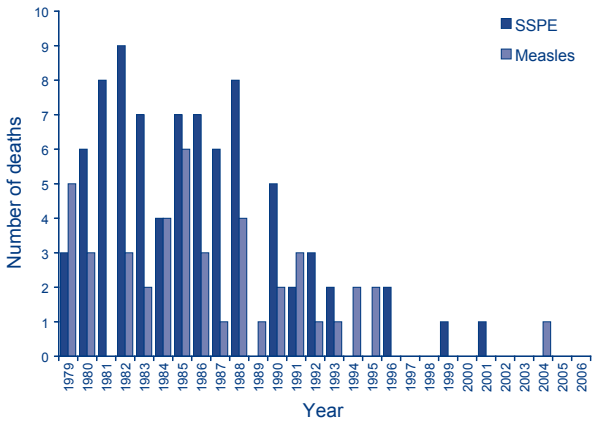


Over the study period, there were 81 deaths (7.2% of total encephalitis deaths) from subacute sclerosing panencephalitis (SSPE) (Table), with the average number of SSPE deaths decreasing from 6.3 per year between 1979 and 1985, to 4.4 per year in 1993 to 1999 (Figure 4). After 1999, the average deaths per year from SSPE was less than one. Measles encephalitis showed a similar decline (Figure 4), decreasing from 3.3 deaths per year (1979–1985) to 2 deaths per year (1986–1992), to fewer than 1 death per year after 1992.

Discussion

There were a total of 1,118 deaths associated with encephalitis in Australia in the 28-year period, 1979 to 2006, with an annual death rate of 2.3 per

Figure 4: Number of subacute sclerosing panencephalitis and measles encephalitis deaths per year, Australia, 1979 to 2006



1 million population (range 1.3–3.6). As 51.5% did not have a pathogen diagnosis, this raises questions about the potential aetiologies of encephalitis deaths in Australia. The increase in the proportion of deaths due to 'unknown' encephalitis from 47.0% to 57.2% over the study period, provides further motivation for improving pathogen identification. The wider availability of polymerase chain reaction confirmation for herpes simplex encephalitis over the study period to explain cases that may have previously been coded as 'unknown' may mean that there has been an even greater relative increase in encephalitis due to unknown causes.

The changes in death rates by age group over time are likely to represent improvements in medical care during the study period. The decrease in death rates observed in children, aged 0–9 years is likely to be in part due to improvements in paediatric intensive care.[22,23] Similarly, the increase in death rates seen in older people (>60 years) may be the result of Australia's ageing population living longer due to improvements in treatment for chronic diseases such as cancer, heart disease and diabetes,[24] with older people being more prone to immunosuppression and hence susceptible to diseases such as herpes encephalitis, *Listeria meningoencephalitis* and zoster encephalitis. Unfortunately, the small number of deaths from individual pathogens precludes further detailed analysis. It is worth noting that an improvement in death rate may not necessarily reflect improvement in morbidity for some causes of encephalitis that have important neurological sequelae.

Australia has seen a small decline in the death rate (3.0% decrease) from encephalitis, largely due to a decline in the 'known' causes (4.3% decrease) explained by changes in medical care and preventative health activities for two of the more common causative organisms. The observed decline in herpes encephalitis can be attributed to the increasing use of acyclovir in the late 1980s.[25] A similar finding was reported from the USA between 1978 and 1998.[20]

The decline in deaths due to measles encephalitis (Figure 4) can be attributed to the increasing uptake of measles vaccination throughout the 1980s, accelerated by Australia's first national measles vaccination campaign in 1987.[26] SSPE is a rare complication of measles and is a neurodegenerative disorder caused by the persistence of a defective measles virus in the central nervous system.[27] SSPE has a reported incubation period of 6–8 years following infection with measles,[28] so it is not surprising that a decline in SSPE was observed in the 1990s. The near disappearance of SSPE and measles encephalitis reflects the success of immunisation with measles vaccine in Australia.

The death rate due to encephalitis in Australia is lower than estimates from the USA (2.3 per 1 million in Australia versus 5.1–5.3 per 1 million), however this may in part be due to a difference in study methodology, as the USA study included deaths for which encephalitis was listed anywhere on the death record, whereas our study included only those deaths for which encephalitis was the primary cause.[20] In addition, the USA had a higher rate of HIV infection, which accounted for many more deaths in that study. The non-HIV related encephalitis death rate in the USA decreased between 1979 and 1988, and 1989 to 1998, from 4.7 to 3.6 per 1 million population.

Interestingly, the USA study found a much higher rate of 'unknown' encephalitis deaths (81.5–86.2%), compared to Australian data. Again, it is difficult to know if this may be a consequence of the data collection, with less likelihood of a pathogen being recorded if encephalitis was not the primary cause of death. Our data showed a proportion of 'unknown' encephalitis deaths more in keeping with the findings from the UK, which found 50.0% of encephalitis deaths were from an unknown aetiology.[12]

There are several limitations associated with using ICD coded death certificate data to estimate disease burden. During the time period of this study, the ICD coding system changed with several new encephalitis-associated codes appearing in ICD-10 (1999–2006) that had not been present in ICD-9 (1979–1998), such as those for zoster encephalitis and *Listeria meningoencephalitis*. Patients with these conditions prior to 1999 must have been coded using different codes, however, it can not be definitively determined which were used. Fortunately, such patients account for a small proportion (<3%) of all encephalitis deaths (Table). In addition to limitations due to the ICD coding changes, there may have been differences in diagnostic criteria for encephalitis used by clinicians during the period 1979–2006. Finally, because we used only primary cause of death data, we may have underestimated encephalitis-associated deaths, particularly in people with co-existing medical conditions, who died from another cause (for example a myocardial infarction) while they were ill with encephalitis.

Globally, there has been a dramatic increase in recognition of EIDs since 1940.[15] Many EIDs have been due to zoonotic or arboviral pathogens[15] and presented with outbreaks of an encephalitis syndrome, including West Nile virus, Hendra virus, Nipah virus, Murray Valley encephalitis and Japanese encephalitis.[29] The high proportion (51.5%) of encephalitis deaths from 'unknown' pathogens in Australia raises questions about our capacity to detect novel pathogens presenting as encephalitis. This highlights the importance of monitoring trends in encephalitis morbidity and mortality in Australia with a view to improving pathogen diagnosis for encephalitis and rapidly identifying novel emerging encephalitis-causing pathogens that demand public health action.

Acknowledgements

The authors wish to gratefully acknowledge the assistance of Mark Clements (Research Fellow, National Centre for Epidemiology and Population Health), Megan Valentine (Statistician, Hunter New England Population Health), Staff at the Epidemiology and Surveillance Branch of the NSW Department of Health, the Australian Government Department of Health and Ageing for funding the Master of Applied Epidemiology Program (CH & PMK) and the National Health and Medical Research Council for partially funding PMK.

Author details

Dr Clare Huppatz, Master of Applied Epidemiology Scholar[1,2]
Assoc Prof Paul M Kelly, Director, Master Applied Epidemiology Program[2]
Assoc Prof Christopher Levi, Senior Staff Specialist Neurologist, Director of Acute Stroke Services[3]
Dr Craig Dalton, Public Health Physician[4]
Assoc Prof David Williams, Director of Neurology[3]
Prof David N Durrheim, Service Director, Health Protection[1,4]

1. Hunter New England Population Health, NSW Department of Health, Wallsend, New South Wales
2. National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital Territory.
3. John Hunter Hospital, Hunter New England Health, Newcastle, New South Wales
4. Hunter Medical Research Institute, Newcastle, New South Wales

Corresponding author: Professor David Durrheim, Service Director, Health Protection, Hunter New England Population Health, Locked Bag 10, WALLSEND NSW 2287. Telephone: +61 2 4924 6395. Facsimile: +61 2 4924 6215. Email: David.Durrheim@hnehealth.nsw.gov.au

References

1. Chaudhuri A, Kennedy PG. Diagnosis and treatment of viral encephalitis. *Postgrad Med J* 2002;78:575–583.
2. Whitley RJ, Gnann JW. Viral encephalitis: familiar infections and emerging pathogens. *Lancet* 2002;359:507–513.
3. Steiner I, Budka H, Chaudhuri A, Koskiniemi M, Sainio K, Salonen O, et al. Viral encephalitis: a review of diagnostic methods and guidelines for management. *Eur J Neurol* 2005;12:331–343.
4. Whitley RJ. Viral encephalitis. *N Engl J Med* 1990;323:242–250.
5. Whitley RJ. Herpes simplex virus infections of the central nervous system. A review. *Am J Med* 1988;85:61–67.
6. Whitley RJ, Lakeman F. Herpes simplex virus infections of the central nervous system: therapeutic and diagnostic considerations. *Clin Infect Dis* 1995;20:414–420.

7. Whitley RJ, Alford CA, Hirsch MS, Schooley RT, Luby JP, Aoki FY, et al. Vidarabine versus acyclovir therapy in herpes simplex encephalitis. *N Engl J Med* 1986;314:144–149.
8. Whitley R, Arvin A, Prober C, Burchett S, Corey L, Powell D, et al. A controlled trial comparing vidarabine with acyclovir in neonatal herpes simplex virus infection. Infectious Diseases Collaborative Antiviral Study Group. *N Engl J Med* 1991;324:444–449.
9. Levitz RE. Herpes simplex encephalitis: a review. *Heart Lung* 1998;27:209–212.
10. Wutzler P. Antiviral therapy of herpes simplex and varicella-zoster virus infections. *Intervirology* 1997;40:343–356.
11. Noah DL, Drenzek CL, Smith JS, Krebs JW, Orciari L, Shaddock J, et al. Epidemiology of human rabies in the United States, 1980 to 1996. *Ann Intern Med* 1998;128:922–930.
12. Davison KL, Crowcroft NS, Ramsay ME, Brown DW, Andrews NJ. Viral encephalitis in England, 1989–1998: what did we miss? *Emerg Infect Dis* 2003;9:234–240.
13. Glaser CA, Honarmand S, Anderson LJ, Schnurr DP, Forghani B, Cossen CK, et al. Beyond viruses: clinical profiles and etiologies associated with encephalitis. *Clin Infect Dis* 2006;43:1565–1577.
14. Khetsuriani N, Holman RC, Anderson LJ. Burden of encephalitis-associated hospitalizations in the United States, 1988–1997. *Clin Infect Dis* 2002;35:175–182.
15. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, et al. Global trends in emerging infectious diseases. *Nature* 2008;451:990–993.
16. Campbell GL, Marfin AA, Lanciotti RS, Gubler DJ. West Nile virus. *Lancet Infect Dis* 2002;2:519–529.
17. Bellini WJ, Harcourt BH, Bowden N, Rota PA. Nipah virus: an emergent paramyxovirus causing severe encephalitis in humans. *J Neurovirol* 2005;11:481–487.
18. Speare R, Skerratt L, Foster R, Berger L, Hooper P, Lunt R, et al. Australian bat lyssavirus infection in three fruit bats from north Queensland. *Commun Dis Intell* 1997;21:117–120.
19. Barclay AJ, Paton DJ. Hendra (equine morbillivirus). *Vet J* 2000;160:169–176.
20. Khetsuriani N, Holman RC, Lamonte-Fowlkes AC, Selik RM, Anderson LJ. Trends in encephalitis-associated deaths in the United States. *Epidemiol Infect* 2007;135:583–591.
21. Rantalaiho T, Farkkila M, Vaheri A, Koskiniemi M. Acute encephalitis from 1967 to 1991. *J Neurol Sci* 2001;184:169–177.
22. Tilford JM, Roberson PK, Lensing S, Fiser DH. Differences in pediatric ICU mortality risk over time. *Crit Care Med* 1998;26:1737–1743.
23. Taylor A, Butt W, Ciardulli M. The functional outcome and quality of life of children after admission to an intensive care unit. *Intensive Care Med* 2003;29:795–800.
24. Taylor R, Lewis M, Powles J. The Australian mortality decline: cause-specific mortality 1907–1990. *Aust N Z J Public Health* 1998;22:37–44.
25. Victorian Drug Usage Advisory Committee. Antibiotic Guidelines. 5th ed. Melbourne, Victoria, Australia: Victorian Medical Postgraduate Foundation Therapeutics Committee; 1987.
26. Turnbull FM, Burgess MA, McIntyre PB, Lambert SB, Gilbert GL, Gidding HF, et al. The Australian Measles Control Campaign, 1998. *Bull World Health Organ* 2001;79:882–888.
27. Garg RK. Subacute sclerosing panencephalitis. *Postgrad Med J* 2002;78:63–70.
28. Wong EH, Hui AC, Mok VC, Leung H, Chan RC, Wong KS, et al. A young man who kept falling over. *Lancet* 2008;372:418.
29. Solomon T. Exotic and emerging viral encephalitides. *Curr Opin Neurol* 2003;16:411–418.

Dengue in north Queensland, 2005–2008

Jeffrey N Hanna, Scott A Ritchie, Ann R Richards, Jan L Humphreys, Brian L Montgomery, Gerhard JM Ehlers, Alyssa T Pyke, Carmel T Taylor

Abstract

The dengue vector, the mosquito *Aedes aegypti*, is present in urban settings in north Queensland, thereby putting the region at risk of outbreaks of dengue. This review describes some features of the 9 outbreaks of dengue that occurred in north Queensland over the 4 years, 2005–2008. *Commun Dis Intell* 2009;33:198–203.

Keywords: *Aedes aegypti*, dengue, north Queensland, mosquitoes

Introduction

Although dengue viruses are not endemic in north Queensland, the vector mosquito, *Aedes aegypti*, is present in urban settings in the region and on some Torres Strait islands. This means that north Queensland is prone to outbreaks of dengue, each one apparently initiated by a viraemic traveller from an endemic country.[1–4] Of concern, these outbreaks have become more frequent over the past 2 decades.[4] This review describes some features of the 9 outbreaks of dengue that occurred in north Queensland between 2005 and 2008.

Methods

The methods have been described elsewhere.[1–4] Briefly, following notification of laboratory-confirmed cases, details are ascertained about each case, including where the infection may have been acquired and where the case may have been infectious to the mosquito vector, *Ae. aegypti*. Mosquito control measures are then prioritised and implemented based upon this information. A dengue serotype-specific IgM enzyme-linked immunosorbent assay[5] has proven to be very useful in confirming cases and identifying the infecting serotype.[4] Phylogenetic analyses of dengue viruses isolated from cases have proven very useful in understanding possible links between outbreaks and the possible initial sources of the outbreak viruses.[2–4]

Results

2005

In late February, a case of locally-acquired dengue in a resident of Thursday Island in the Torres Strait was notified to the Tropical Population Health Services (TPHS). Further cases were identified, and the outbreak soon spread to 2 other islands. The infecting virus was identified as serotype 4; the 3 islands had all been affected by serotype 2 outbreaks in prior recent years.[1,4] This outbreak in the Torres Strait lasted for a total of 7 weeks, and there was a total of 56 confirmed cases (Figure 1). The first known case in the outbreak was, retrospectively, recognised as having an onset 10 days prior to the initial notification.

In early May, TPHS was notified of a case of locally-acquired dengue from the suburb of Currajong in Townsville. Although there was no sudden increase in numbers, 4 more cases occurred in that suburb over the next month (Figure 1). Indeed, this outbreak was characterised by a small number of cases – 18 in total – over a prolonged period, a total of 22 weeks, with considerable intervals between some of the cases. The first known case was retrospectively recognised as having an onset 24 days prior to the initial notification. The infecting virus was also identified as serotype 4.

Phylogenetic analyses indicated that the 2 outbreaks, although both caused by serotype 4 viruses, involved quite different genotypes. The Torres Strait virus was almost identical not only to an isolate from Indonesia, but also to a dengue 4 virus isolated in 2002 from a Cairns resident who had recently been to Indonesia.[3] The Townsville virus was almost identical to dengue serotype 4 viruses recently isolated in the Philippines (Figure 2).

2006

In mid-January, TPHS was notified of a case of locally-acquired dengue from the suburb of

Figure 1: Dengue serotype 4 outbreaks in the Torres Strait and Townsville, 2005

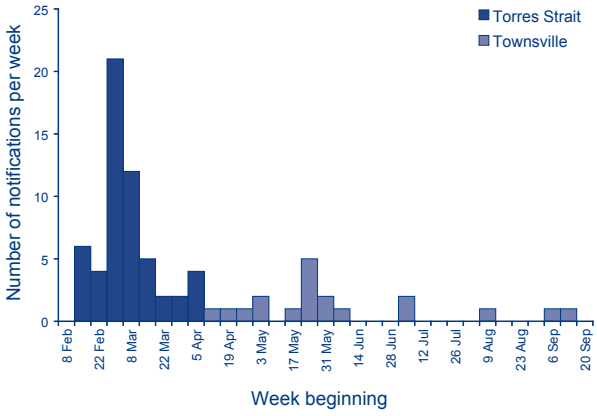
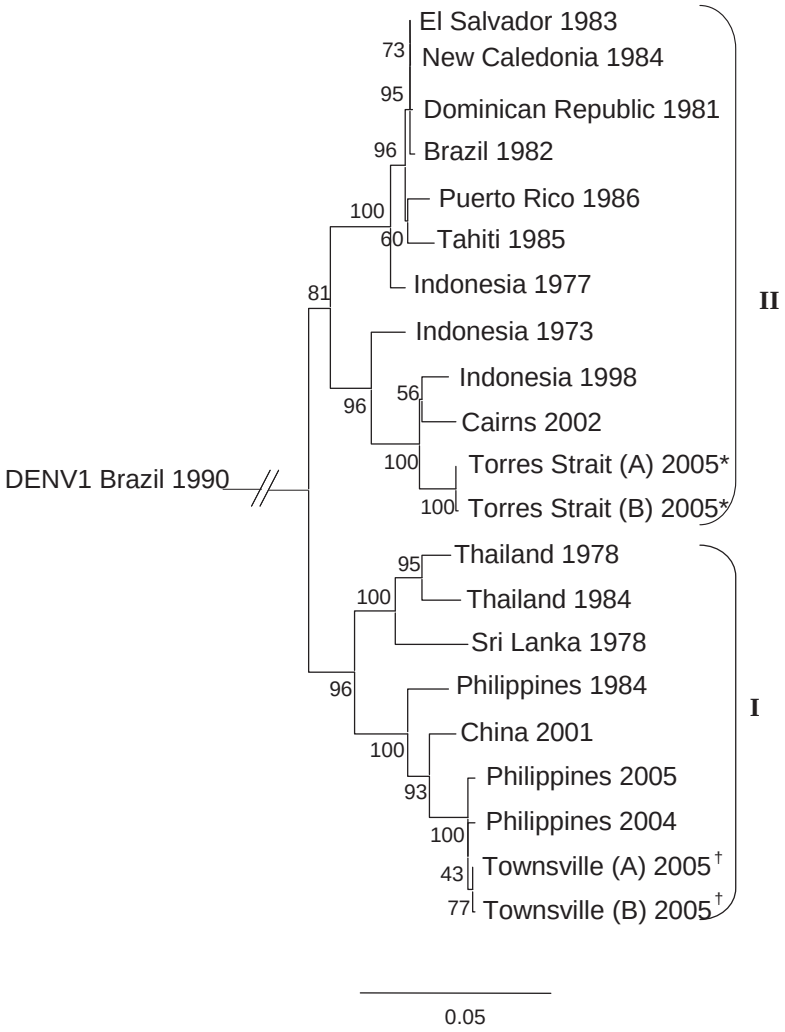


Figure 2: Phylogenetic relationships of the 2005 dengue serotype 4 viruses responsible for the Torres Strait* and Townsville† outbreaks of dengue



Cranbrook in Townsville. Although the outbreak affected residents of the older housing in this suburb, it lasted for only 6 weeks and there were only 8 confirmed cases. The earliest recognised case had an onset 23 days before the initial notification and the infecting virus was identified as serotype 3 (Figure 3).

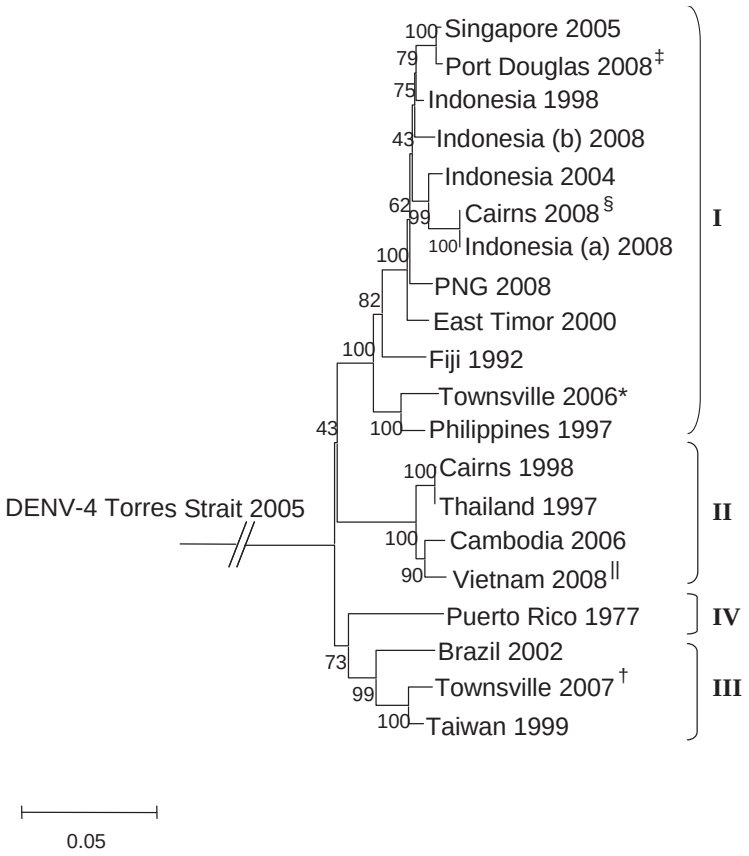
At the beginning of March, TPHS was notified of a case of locally-acquired dengue from Gordonvale, a township just south of Cairns. Two cases were recognised – one retrospectively – as occurring in the latter half of February but no more were recognised in the next 6 weeks. Severe tropical cyclone Larry occurred on 20 March, which not only demolished some general practices in the vicinity, but also resulted in vector control activities being diverted away from Gordonvale to areas more severely affected by the disaster. It was recognised in early April that the outbreak had not ceased, and that it had spread into several Cairns suburbs, Manunda in particular.

The outbreak had a total duration of 18 weeks, and included a total of 29 cases with 20 in Gordonvale residents. The earliest recognised case had an onset 17 days before the initial notification and the infecting virus was identified as serotype 2. A phylogenetic analysis indicated that the virus was virtually identical to isolates from a large outbreak that affected the Torres Strait in 2003 and Cairns in 2004. This prior outbreak was presumed to have been initiated by a traveller from Papua New Guinea.[4]

2007

In late March TPHS was notified of a case of locally-acquired dengue in a resident of the suburb of Cranbrook in Townsville. This location and the identification of the infecting virus as serotype 3 raised some concerns that the virus may have somehow 'overwintered' unrecognised in this suburb. However, the case had visited a residence in South Townsville, and several other people in that residence had recently been ill with an acute febrile

Figure 3: Phylogenetic relationships of the dengue serotype 3 viruses responsible for the outbreaks in Townsville in 2006* and 2007† and in Port Douglas‡ and Cairns§ in 2008

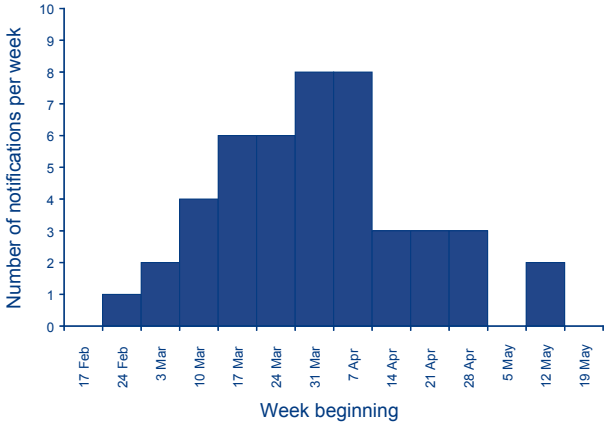


The case imported from Vietnam‖ into Port Douglas in early 2008 is also shown

illness. It was soon recognised that this outbreak was centred on South Townsville with no evidence of transmission in Cranbrook. South Townsville is an older, inner city suburb adjacent to Railway Estate, which was the centre for a large epidemic of serotype 2 dengue in the early 1990s.[6]

The first retrospectively recognised case had an onset 26 days before the initial notification, so the outbreak in South Townsville was well established before it was recognised on 21 March (Figure 4). The outbreak lasted for 12 weeks with a total of 46 confirmed cases, and although it moved into another suburb (West End) there was no evidence that Railway Estate was affected. A phylogenetic analysis demonstrated that the virus was quite different to the dengue serotype 3 virus isolated during the 2006 outbreak in Townsville; indeed the 2 viruses belonged to different genotypes (Figure 3).

Figure 4: Dengue serotype 3 outbreak in Townsville, 2007



2008

In late January TPHS was notified of an imported (from Vietnam) case of dengue in a resident of Port Douglas, a small beach resort situated about 60 km north of Cairns. Port Douglas was severely affected in 1998–1999 during an epidemic of dengue serotype 3.[2] The case was subsequently shown to have been infected by serotype 3 virus. Because the person was acutely ill, and therefore viraemic in Port Douglas, local medical practitioners were alerted to the possibility of subsequent local transmission and mosquito control activities were undertaken around the case's residence and place of work in Mossman, a nearby town.

About a week later TPHS was informed of a locally-acquired case of dengue in a Mossman resident who worked in Port Douglas. This first recognised locally-acquired case worked at a beachfront venue popular with both local residents and visitors. It soon became apparent that there was local transmission occurring in Port Douglas, with an initial focus around the venue. The outbreak eventually included 22 cases, and lasted for 10 weeks. Four of the cases were probably acquired in Mossman, the rest in Port Douglas.

Although the onsets of the illnesses of the imported case and the first recognised locally-acquired case were only 11 days apart, it was not possible to epidemiologically link the imported case to the venue. This apparent paradox was resolved when the phylogenetic analyses revealed that the imported serotype 3 and the outbreak serotype 3 viruses were quite different – indeed belonging to different genotypes – and were therefore not linked (Figure 3). The initial source of the outbreak was never recognised, but it was presumably a viraemic patron of the beachside venue.

In early November TPHS was notified of a locally-acquired case of dengue in a Cairns resident. The onset of his symptoms was 14 days before the notification. Mosquito surveys revealed a very low risk of transmission of dengue at his residence and at several locations around Cairns where he had worked as a tradesman, and no insights could be gained into where he may have acquired the infection. His dengue was caused by a serotype 2 virus and because he had been in Cairns for the duration of his viraemia, local medical practitioners were alerted to the possibility of further cases.

In late November TPHS was notified of a locally-acquired case of dengue in a resident of Cairns North, an older suburb not far from the city centre. Multiple cases were soon recognised in adjacent properties in that suburb, and dengue serotype 3 was identified as the infecting virus. Investigations revealed that the earliest case was an adult male who had become unwell 5 days after returning to Australia from Kalimantan (Indonesian Borneo); although he had been quite unwell for 4–5 days, he did not seek medical attention. He lived in an unscreened highset 'Queenslander' residence that provided ready access for *Ae. aegypti* mosquitoes, which were abundant in the vicinity. Subsequent serological tests indicated that he had recently had a dengue serotype 3 infection.

The hot and sultry weather, with some pre-monsoonal rains at that time, were ideal for *Ae. aegypti* proliferation and would have led to a relatively short extrinsic incubation period of the dengue virus. Indeed, the first recognised locally-acquired case had an onset 10 days after that of the case who apparently had imported the infection from Indonesia, and who was assumed to have initiated the outbreak. Not surprisingly the outbreak expanded rapidly and soon spread to 3 other suburbs: Clifton Beach (25 km north of central Cairns), Whitfield and Parramatta Park. The latter 2 suburbs have been involved in several previous outbreaks of dengue,[2,4] most recently an outbreak of serotype 2 dengue in 2003–04.[4] By the end of 2008, 5 quite separate Cairns suburbs were affected and 98 cases of dengue had been confirmed.

Cases of dengue serotype 3 that were acquired in Cairns were reported from numerous other sites, both within and outside Queensland, reflecting the continuous movement of people, particularly towards the end of the year. In late December, a locally-acquired case of dengue serotype 3 was confirmed from the suburb of Belgian Gardens in Townsville. Molecular analyses indicated that the dengue 3 viruses circulating in Cairns and Townsville were identical, providing strong evidence that an unknown traveller from Cairns had introduced the virus into Belgian Gardens. Both the Cairns and Townsville dengue serotype 3 outbreaks continued into 2009.

Just before the end of the year TPHS was notified of a case of locally-acquired dengue in a resident of the suburb of North Ward in Townsville; there was a 9 day interval between the onset of his symptoms and the notification. Dengue serotype 1 was identified as the infecting virus, and subsequent molecular analyses indicated that the virus was very closely related to recent dengue serotype 1 isolates from Singapore (data not shown). The importation that led to this outbreak was not identified. By the end of the year 4 more cases, all apparently acquired in North Ward, had been notified. This outbreak also continued into 2009.

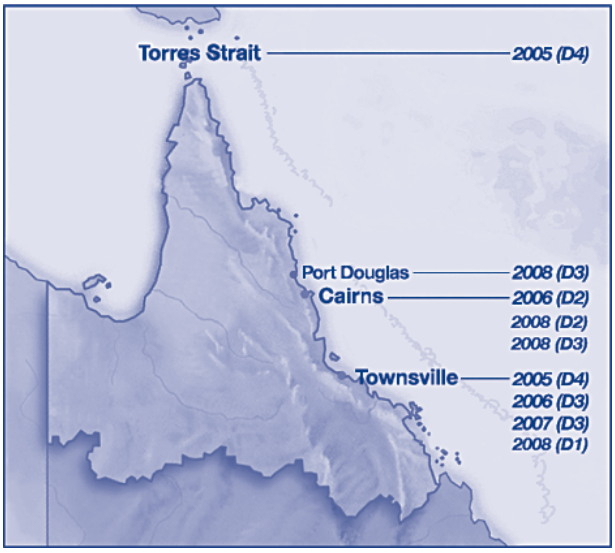
Discussion

The experiences over these 4 years indicate how vulnerable the region is to outbreaks of dengue. There were outbreaks in Townsville every year over the 4 years (Map).

Furthermore, all 4 serotypes of dengue viruses were involved in the outbreaks that occurred over the 4 years (Map). Since many hundreds, probably thousands, of people in the region have had a previous dengue infection, there is the not insignificant likelihood of severe complications such as dengue shock syndrome should secondary infections occur.

A common feature in most (7) of the outbreaks was a considerable delay (mean 18.9 [range 9–26] days) between the onset of the first known cases and the

Map: Outbreaks of dengue, north Queensland, 2005–2008



D1 = dengue serotype 1, D2 = dengue serotype 2, D3 = dengue serotype 3, D4 = dengue serotype 4.

recognition of the outbreaks. This delay, which obviously can delay the mosquito control measures, has understandably been described as the 'Achilles heel' of dengue control in north Queensland.[7] In an effort to reduce the delay, a new rapid diagnostic test was used for the first time during the dengue outbreaks that occurred in late 2008. This enzyme-linked immunosorbent assay can detect a protein (the NS1 protein) produced by replicating dengue viruses early during the course of the dengue illness.[8] The early experiences with this test, being used locally in north Queensland, appear promising with a rapid turnaround of the test results.

The molecular analyses have again proven to be extremely powerful and useful tools allowing clear understanding of the relationships between dengue viruses of the same serotype. They have enabled links between outbreaks to be clarified (Figures 2 and 3), and links between importations of dengue and subsequent outbreaks to be defined (Figure 3). The latter will continue to be important as there were 43 recognised viraemic importations of dengue into north Queensland over the 4 years (data not shown).

Mosquito control strategies have evolved, and will continue to do so, as experience is gained from each outbreak.[7] This evolution has led to a refinement of a comprehensive approach incorporating larval control, targeted interior spraying with synthetic insecticides and the use of lethal ovitraps.[7] A recent innovation has been the rapid deployment of biodegradable lethal ovitraps in and around the residences (and other relevant premises) of dengue cases.[9] The use of lethal ovitraps has improved response times and reduced insecticide use compared to earlier responses that utilised much wider use of interior spraying.[7,9] This has been enhanced by the new (Queensland) *Public Health Act 2005* that allows authorised public health officers to access yards and set lethal ovitraps when the residents are not present.

Following the large outbreak of dengue serotype 3, which led to 275 cases in the Port Douglas/Mossman area in 1998–1999,[2] the local government environmental health officer worked with the local communities on a program to reduce local *Ae. aegypti* populations. Although a considerable proportion of the local population would have been immune, it is encouraging that there were only 22 cases in the 2008 Port Douglas/Mossman outbreak. Similarly, following the large outbreak of dengue serotype 2, which led to 171 cases on Thursday Island in 2003–2004,[4] there was a massive clean-up of large items (that were potential *Ae. aegypti* breeding sites) on the island.[4] Again, it is encouraging that there were only 34 cases acquired in Thursday Island in the 2005 outbreak.

These 2 observations suggest that proactive programs designed to reduce *Ae. aegypti* breeding and therefore *Ae. aegypti* populations can reduce the size of dengue outbreaks in north Queensland, and therefore perhaps even prevent outbreaks from occurring. In 2005 it was stated that 'Clearly, more preventive *Ae. aegypti* control would have to be done in inter-epidemic periods to prevent explosive transmission'.[7] This message still stands; unless local authorities and local communities adopt innovative preventive measures – especially removal of *Ae. aegypti* breeding sites – outbreaks of dengue, some very large with considerable morbidity and occasional mortality, will continue to occur in north Queensland.

Author details

Jeffrey N Hanna, Public Health Physician[1]
Scott A Ritchie, Medical Entomologist[1]
Ann R Richards, Public Health Nursing Officer[1]
Jan L Humphreys, Public Health Nursing Officer[2]
Brian L Montgomery, Medical Entomologist[1]
Gerhard JM Ehlers, Medical Entomologist[2]
Alyssa T Pyke, Scientist[3]
Carmel T Taylor, Scientist[3]

1. Tropical Population Health Service, Queensland Health, Cairns, Queensland
2. Tropical Population Health Service, Queensland Health, Aitkenvale, Queensland
3. Public Health Virology, Queensland Health Forensic and Scientific Services, Coopers Plains, Queensland

Corresponding author: Dr J Hanna, Tropical Population Health Service, PO Box 1103, CAIRNS QLD 4870. Telephone: +61 7 4050 3604. Facsimile:+61 7 4031 1440. Email: Jeffrey_hanna@health.qld.gov.au

References

1. Hanna JN, Ritchie SA, Merritt AD, van den Hurk AF, Phillips DA, Serafin IL, et al. Two contiguous outbreaks of dengue type 2 in north Queensland. *Med J Aust* 1998;168(5):221–225.
2. Hanna JN, Ritchie SA, Phillips DA, Serafin IL, Hills SL, van den Hurk AF, et al. An epidemic of dengue 3 in Far North Queensland, 1997–1999. *Med J Aust* 2001;174(4):178–182.
3. 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.
4. Hanna JN, Ritchie SA, Richards AR, Taylor CT, Pyke AT, Montgomery BL, et al. Multiple outbreaks of dengue serotype 2 in north Queensland, 2003/04. *Aust N Z J Public Health* 2006;30(3):220–225.
5. Taylor C, Simmons R, Smith I. Development of immunoglobulin M capture enzyme-linked immunosorbent assay to differentiate human flavivirus infections occurring in Australia. *Clin Diagn Lab Immunol* 2005;12:371–374.
6. Streatfield R, Sinclair D, Bielby G, Sheridan J, Pearce M, Phillips D. Dengue serotype 2 epidemic, Townsville, 1992–93. *Commun Dis Intell* 1993;17:330–332.
7. Ritchie SA. Evolution of dengue control strategies in north Queensland, Australia. *Arbovirus Res Aust* 2005;9:324–330.
8. Chuansumrit A, Chaiyaratana W, Pongthanapisith V, Tangnararatchakit K, Lertwongrath S, Yoksan S. The use of dengue nonstructural protein 1 antigen for the early diagnosis during the febrile stage in patients with dengue infection. *Pediatr Infect Dis J* 2008;27(1):43–48.
9. Ritchie SA, Long SA, McCaffrey N, Key C, Lonergan G, Williams CR. A biodegradable lethal ovitrap for control of container-breeding *Aedes*. *J Am Mosq Control Assoc* 2008;24(1):47–53.

Rotavirus epidemiology in Queensland during the pre-vaccine era

Suzy J Campbell, Michael D Nissen, Stephen B Lambert

Abstract

Rotavirus, the most common cause of severe gastroenteritis in early childhood, is now a vaccine preventable disease with immunisation added to the Australian publicly funded schedule in July 2007. To better understand rotavirus epidemiology in Queensland prior to vaccine introduction, we used 3 routinely-collected data sources. We analysed hospital records of all children less than 5 years of age admitted to Queensland hospitals between July 2001 and June 2006 with any rotavirus-specific code or with an acute gastroenteritis (AGE) code in the principal field. We linked a sample of public hospital admission records to laboratory test requests to determine the extent of diagnostic testing for causes of AGE. Finally, we analysed rotavirus notifications for the same age group between December 2005 and December 2006. Hospitalisation and notification data both identified Indigenous children as having a higher burden of rotavirus illness than non-Indigenous children. Hospitalisations occurred disproportionately in Indigenous children, at a younger age, and resulted in a longer duration of stay. AGE hospitalisations occurred more commonly than rotavirus admissions, but the seasonal trend mirrored rotavirus data. Linking a sample of hospitalisations with laboratory testing data showed that, for admissions having a rotavirus-specific discharge code, 89% had laboratory-confirmed rotavirus infection. In the pre-vaccine era, rotavirus had the greatest impact in the young and Indigenous. Using routinely collected data, it should be possible to monitor the impact of vaccine introduction in Queensland. *Commun Dis Intell* 2009;33:204–208.

Keywords: rotavirus, epidemiology, Queensland, Indigenous, separation, hospitalisation, diagnostic, laboratory testing, acute gastroenteritis

Introduction

Rotavirus is the most common cause of acute gastroenteritis in children. Prior to vaccine introduction, it is estimated the virus was responsible for up to 50% of diarrhoea hospitalisations in childhood, with approximately 10,000 Australian children hospitalised each year.[1] These figures considerably underestimate overall burden of disease as they miss community-managed illness: only the more serious cases of childhood gastroenteritis are likely to result in hospital admission. Of these, it is likely there is significant under-identification of rotavirus, as laboratory confirmation is not required and this is not undertaken for all acute gastroenteritis (AGE) admissions. This means our understanding of the burden of rotavirus infection in children in Australia is incomplete.

We have now entered a new era of rotavirus epidemiology, with the prospect of disease prevention through vaccination. Two rotavirus vaccines are licensed for use in Australia: Rotarix (GSK Biologicals), a single strain, live attenuated human rotavirus strain, and RotaTeq (Merck, distributed in Australia by CSL Biotherapies), a pentavalent, live human-bovine rotavirus reassortant. Both have excellent efficacy in reducing severe rotavirus gastroenteritis.[2,3] Queensland children born on or after 1 May 2007 were eligible to receive 3 doses of RotaTeq from 1 July 2007.

Since June 1999 the National Rotavirus Reference Centre has been reporting strain surveillance for rotavirus.[4] Rotavirus research within Australia has provided epidemiologic data[1,5,6] including national population based estimates of rotavirus hospitalisation rates,[7,8] and the direct economic cost of these has been calculated.[1,9] Queensland has not consistently contributed rotavirus samples to the National Rotavirus Reference Centre, and little is known about the epidemiology of rotavirus in Queensland. To better understand rotavirus epidemiology in Queensland children during the pre-vaccine era, and provide a baseline for future comparisons, we used 3 sources of routinely collected data — hospitalisations, laboratory testing, and notification data.

Methods

These studies were approved by The University of Queensland Medical Research Ethics Committee, the Royal Children's Hospital and Health Service District Human Research Ethics Committee, and the Queensland Health Corporate Office Human Research Ethics Committee. An application to use data for research purposes under the *Public Health Act 2005* was approved by Queensland's Chief Health Officer.

Hospital discharge coding

We retrieved data for analysis from the Queensland Health Patient Admitted Dataset. All private and public hospital separations in children less than 5 years of age in Queensland, with a rotavirus-specific principal or other diagnosis code (ICD-10-AM code A08.0)[10] from 1 July 2001 to 30 June 2006 were included. We examined age and sex-specific hospitalisation rates, reported Indigenous status, seasonality, and average length of stay (ALOS), as they were recorded in the dataset. The 95% confidence intervals around ALOS and percentage of Indigenous rotavirus notifications were calculated using Stata 9.0 (Stata Corp, College Station, Texas).

We compared rotavirus-coded hospitalisations with the burden due to related and less specific AGE admissions and to allow comparison with other published data.[1,11] We retrieved data on a range of AGE codes where this was the principal diagnosis. The codes used were (ICD-10-AM):[10]

- Bacterial: A00 Cholera, A01 Typhoid and paratyphoid fevers, A02 Other salmonella infections (excluding A02.2), A03 Shigellosis, A04 Other bacterial intestinal infections, excluding A05 Other bacterial foodborne intoxications;
- Protozoal: A06 Amoebiasis (excluding A06.4, A06.5, A06.6, A06.7, A06.8), A07 Other protozoal intestinal disease;
- Viral: A08 Viral and other specified intestinal infections; and
- Non-specific: A09 Diarrhoea and gastroenteritis of presumed infectious origin, and K52 Other non-infective gastroenteritis and colitis.

Data linkage with laboratory testing

A sample of all AGE-related records (including rotavirus) were systematically selected from the merged hospital dataset and linked to the Queensland Health Auslab database to determine the extent of diagnostic testing for causes of AGE, and rotavirus specifically. Records were sorted by separation date, and every 10th separation was chosen for data linkage. Where a record was not from a Queensland Health hospital, meaning any associated stool testing may not have been done in a Queensland Health laboratory, the record was excluded from the analysis without replacement. Testing records for each child were viewed to determine if any faecal testing was done during the admission of interest (including, specifically, whether rotavirus testing had been done), and whether there was a conclusive result. We compared these findings with the hospital discharge code data.

Notifications

Laboratory-confirmed rotavirus became notifiable in Queensland on 1 December 2005, in accordance with the *Public Health Act 2005*.[12] Data on all children aged less than 5 years with a rotavirus notification between December 2005 and December 2006 were collected from the Queensland Health Notifiable Conditions (NOCS) database and analysed.

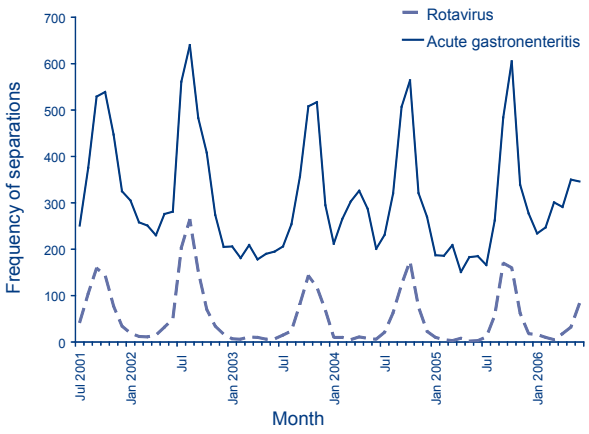
Results

Hospital discharge coding

Between July 2001 and June 2006, there were 3,139 hospital separations in Queensland children aged less than 5 years with the rotavirus-specific discharge code in any field. With a mean 2001–2006 birth cohort of 49,676,[13] this means during the period under review, approximately 1.3% of children were hospitalised for rotavirus. Rotavirus was the principal diagnosis for 2,808 (90%) discharges. Numbers of hospitalisations with a rotavirus code in any position varied year-to-year: the year from 1 July 2002 to 30 June 2003 was a peak year with 787 hospitalisations, and 1 July 2003 to 30 June 2004 a low year with 500 hospitalisations (Figure 1). In each year, the highest number of separations occurred in children between one and two years of age, and overall and in each year of age, there were more separations for males than females (overall male:female ratio 1.27:1).

During the review period there were 18,743 admissions in the same age group with a non-rotavirus AGE code as the principal diagnosis. In the merged dataset, rotavirus made up 14% of combined admissions for AGE or rotavirus in this age group.

Figure 1: All rotavirus-specific separations and discharges with acute gastroenteritis code* as principal diagnosis in children aged less than 5 years, Queensland, 1 July 2001 to 30 June 2006, by month and year

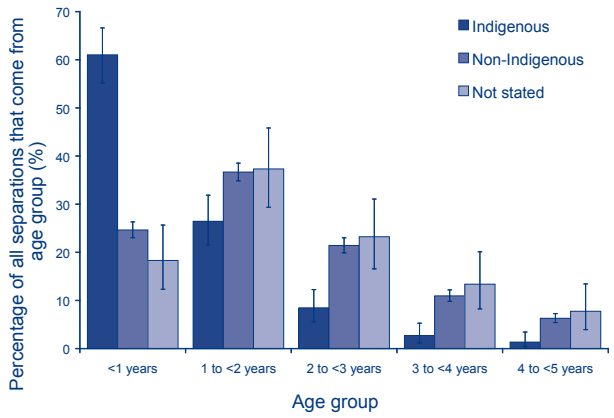


* Excludes rotavirus-specific separations.

Rotavirus separations exhibited a distinct winter seasonal peak between the months of July and November of each year. AGE data had a general pattern that mirrored rotavirus separations, other than for a small autumnal peak in AGE during 2004 (Figure 1).

Rotavirus hospitalisation disproportionately affects Indigenous children. Nearly one tenth (9.4%) of hospitalisations in children less than 5 years of age were identified as occurring in Indigenous children; this compares to the Australian Bureau of Statistics census data, which show Indigenous children make up 6.4% of the general population in Queensland for this age group.[14] This finding varied by age group, with Indigenous children making up 21% of rotavirus admissions in children less than 1 year of age. This resulted in Indigenous children having a younger age at hospitalisation during the review period. Sixty-one per cent of recorded Indigenous hospitalisations for children less than 5 years of age occurred in the 1st year of life, compared with 25% in the non-Indigenous cohort (Figure 2). Indigenous children also had a longer average length of stay when admitted with rotavirus. The ALOS for discharges that had rotavirus as the principal diagnosis for Indigenous children was 2.9 days (95% CI 2.89 to 2.96) compared with 2.1 days (95% CI 2.09 to 2.11) in non-Indigenous children.

Figure 2: Percentage of all rotavirus separations (with 95% confidence interval bars) from each age group, in children aged less than 5 years, Queensland, 1 July 2001 to 30 June 2006, by year of age and Indigenous status



Data linkage with laboratory testing

The systematic sampling method yielded 2,188 patient records for potential analysis. Of these, 578 records were excluded as they were from non-Queensland Health facilities (and therefore had no Auslab record), and 5 records were excluded as the Auslab record did not match the Patient Admitted Data Collection age for the patient. We linked 1,605 hospital discharge records with the laboratory testing history for that child: 222 rotavirus (any field) admissions and 1,383 non-rotavirus AGE (principal field) admissions.

Of the 222 rotavirus admissions: 7 (3%) had no record identified on Auslab; 11 (5%) had no faecal testing performed; and 204 had faecal testing performed (92%). For those that had faecal test results available, 201 (91%) had a rotavirus test result available, and 197 (89%) were positive for rotavirus. Of the 1,383 non-rotavirus AGE admissions: 183 (13%) had no record identified on Auslab; 523 (38%) had no faecal testing performed; and 677 had faecal testing performed (49%). For those that had faecal test results available, 428 (31%) had a rotavirus test result available, and 113 (8%) were positive for rotavirus. Various different ordering patterns were evident, with some children having up to 4 faecal specimens sent for analysis during an AGE admission without rotavirus testing being requested.

Notifications

There were 2,156 rotavirus notifications to NOCS in children aged less than 5 years in Queensland from December 2005 to December 2006. The highest numbers were from children aged 1 to 2 years (40%), followed by notifications in children aged less than 1 year (26%). Males at all ages had more notifications than females with an overall male:female ratio of 1.2:1.

Indigenous status was not reported for 42% of notifications. In the remainder, Indigenous children were over-represented, making up 11% of notifications.

The month with the highest number of rotavirus notifications was September 2006, 566 (26%), and there was a winter/spring seasonal peak between June and December 2006—93% of notifications were made during these months.

Discussion

Rotavirus is a ubiquitous pathogen of early childhood, affecting nearly all children by their 3rd birthday.[15,16] Hygiene improvements have had little to no effect in reducing the incidence of severe gastroenteritis in developed countries, making preventative vaccination the intervention of choice.[17] In large, phase III studies, both Rotarix and RotaTeq provided protection against any rotavirus disease (efficacy: 73%–74%) and severe disease (efficacy: 98%–100%).[17]

On 1 July 2007, Australian children born on or after 1 May 2007 were eligible for rotavirus vaccination on the National Immunisation Program, with either vaccine.[18] Given the impact of recurrent epidemics, and the high burden of disease and hospitalisations, the Northern Territory Government implemented an earlier program, providing Rotarix vaccine from 1 October 2006 for all children born on or after 1 August 2006.[19]

There are few consolidated rotavirus data from the pre-vaccine era available for Queensland. Prior to vaccine use in Australia, there has generally been little in the way of systematic surveillance of rotavirus disease and impact. Most surveillance information has been localised and comes from specific research projects. National hospitalisation data have been used intermittently to estimate the impact of severe rotavirus illness.[1,7,8] There is also little published literature on the potential impact of different diagnostic and laboratory testing patterns for rotavirus infection in hospitalised children with gastroenteritis. Rotavirus only became a notifiable condition in Queensland in December 2005.

We used routinely-collected data in an attempt to fill this gap about rotavirus epidemiology in Queensland. Our findings highlight the disproportionate impact of rotavirus in 2 groups: those under the age of 2 years, and Indigenous children. Overall, rotavirus hospitalisations and notifications peaked between the 1st and 2nd birthday. From hospitalisation and notification data, Indigenous children were at higher risk of being notified with or hospitalised due to rotavirus, and when hospitalisation occurred, it was at an earlier age and for longer than in non-Indigenous cohorts. These findings are similar to trends seen in the Northern Territory, where Indigenous children less than 5 years of age had an increased relative risk of 2.7 for rotavirus notification between 1995 and 2004.[20] Issues with timeliness of vaccine delivery for Indigenous children have been highlighted.[21–23] Due to the theoretical concern regarding intussusception following rotavirus vaccination, the rotavirus vaccines have restricted delivery times with the last (2nd) dose of Rotarix due by 24 weeks of age and the last (3rd) dose of RotaTeq by 32 weeks of age. To maximise any impact vaccines may have, delivering the full course in a timely manner to all children, but particularly to Indigenous children, should be a public health priority.

Based on data-linkage with laboratory testing data, a rotavirus-specific code in any discharge field position was supported by laboratory confirmation 89% of the time. Unless there are systematic changes in general coding procedures, this should mean tracking rotavirus-related hospitalisations is a reasonably specific means of assessing vaccine impact in the future. Internationally, other studies have also validated use of the rotavirus-specific code with between 91%[24] and 100%[25] of rotavirus-coded hospitalisations being supported by laboratory confirmation. Further, based on our findings, we believe hospital discharge codes considerably underestimate the burden of disease. Of non-rotavirus AGE-coded hospitalisations that had a rotavirus test performed, 113 (26.4%) were also found to be positive. We found that half the children with an AGE-related hospitalisation either had no Auslab record or no record of rotavirus testing for the AGE admission performed. It is possible these children had rotavirus testing performed at a privately pathology service; the results of such testing would not be available through the Queensland Health system. The seasonal pattern of AGE hospitalisations follows rotavirus closely, and in the absence of specific rotavirus testing, it is possible a proportion of these admissions are misclassified to less-specific AGE codes. Comparing hospital discharge coding with other sources of data has previously identified issues of sensitivity and specificity.[26] Again, assuming no systematic change in coding methods, it may be possible to observe rotavirus vaccine being effective in reducing not only rotavirus-specific hospitalisations, but admissions identified by less-specific AGE coding. A recent report of a retrospective United States of America cohort study showed that 3 doses of the now withdrawn RotaShield vaccine (Wyeth) had an effectiveness of preventing emergency department presentation and hospitalisation for all-cause AGE of 43% and 83%, respectively.[27]

Routinely collected data, in the absence of systematic changes in reporting or collection, are a simple and efficient method for monitoring the impact of new vaccines. Such data make it possible to fill in gaps in our understanding of disease epidemiology when results from prior targeted research are not available. Hospitalisation discharge data lack timeliness, but historical data allow for the monitoring of trends. The recent addition of rotavirus to the list of notifiable diseases, and the use of laboratory testing data should provide timely data on which to judge the impact of rotavirus vaccine in Australia.

Acknowledgements

Many thanks to Mr Jim Snodgrass, Principal Client Services Officer, Health Information Branch, Queensland Health and Ms Megan Hennessy, Data Manager, Surveillance Team, Communicable Diseases Branch, Queensland Health, for providing data. We would also like to acknowledge the valuable support and assistance provided by Ms Karen Peterson, Manager, Immunisation, Queensland Health and Dr Christine Selvey, Senior Director, Communicable Diseases Branch, Queensland Health, during this research.

Author details

Suzy J Campbell[1,2]
Michael D Nissen[3,4,5]
Stephen B Lambert[3,4]

1 School of Population Health, University of Queensland
2 Communicable Diseases Branch, Queensland Health
3 Queensland Paediatric Infectious Diseases Laboratory, Queensland Children's Medical Research Institute, Royal Children's Hospital
4 Clinical Medical Virology Centre, University of Queensland
5 Clinical and Statewide Services, Queensland Health

Corresponding author: Ms Suzy Campbell, Principal Policy Advisor, Immunisation, Communicable Diseases Branch, Queensland Health, GPO Box 48, BRISBANE QLD 4001. Telephone: +61 7 3328 9701. Email: Suzy_Campbell@health.qld.gov.au

References

1. Carlin JB, Chondros P, Masendycz P, Bugg H, Bishop RF, Barnes GL. Rotavirus infection and rates of hospitalisation for acute gastroenteritis in young children in Australia, 1993–1996. *Med J Aust* 1998;169(5):252–256.
2. Ruiz-Palacios GM, Pérez-Schael I, Velázquez FR, Abate H, Breuer T, Clemens SC, et al. Safety and efficacy of an attenuated vaccine against severe rotavirus gastroenteritis. *N Engl J Med* 2006;354(1):11–22.
3. Vesikari T, Matson DO, Dennehy P, Van Damme P, Santosham M, Rodriguez Z, et al. Safety and efficacy of a pentavalent human-bovine (WC3) reassortant rotavirus vaccine. *N Engl J Med* 2006;354(1):23–33.
4. Kirkwood CD, Bogdanovic-Sakran N, Cannan D, Bishop RF, Barnes GL. National Rotavirus Surveillance Program annual report, 2004–05. *Commun Dis Intell* 2006;30(1):133–136.
5. Ferson MJ. Hospitalisations for rotavirus gastroenteritis among children under five years of age in New South Wales. *Med J Aust* 1996;164(5):273–276.
6. Liddle JL, Burgess MA, Gilbert GL, Hanson RM, McIntyre PB, Bishop RF, et al. Rotavirus gastroenteritis: impact on young children, their families and the health care system. *Med J Aust* 1997;167(6):304–307.
7. 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:S69–S73.
8. Newall AT, Macintyre R, Wang H, Hull B, Macartney K. Burden of severe rotavirus disease in Australia. *J Paed Child Health* 2006;42(9):521–527.
9. Galati JC, Harsley S, Richmond P, Carlin JB. The burden of rotavirus-related illness among young children on the Australian health care system. *Aust N Z J Public Health* 2006;30(5):416–421.
10. Commonwealth of Australia. The International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (ICD-10-AM). Sydney: National Centre for Classification in Health, Faculty of Health Sciences, The University of Sydney. 2006.
11. Charles MD, Holman RC, Curns AT, Parashar UD, Glass RI, Bresee JS. Hospitalizations associated with rotavirus gastroenteritis in the United States, 1993–2002. *Pediatr Infect Dis J* 2006;25(6):489–93.
12. Office of the Queensland Parliamentary Counsel. *Public Health Act 2005*. Available from: http://www.legislation.qld.gov.au/LEGISLTN/CURRENT/P/PubHealA05.pdf. Accessed 14 January 2009.
13. Australian Bureau of Statistics. 3301.0 – Births, Australia, 2007. Canberra: Australian Bureau of Statistics. 2008. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3301.02007?OpenDocument. Accessed 14 May 2009.
14. Australian Bureau of Statistics. Queensland (State) age by indigenous status and sex. In: 2006 Census of Population and Housing. Cat. No. 2068.0 – 2006 Census Tables. Canberra: Australian Bureau of Statistics. 2006.
15. Kirkwood C, Bogdanovic-Sakran N, Barnes G, Bishop R. Rotavirus serotype G9P[8] and acute gastroenteritis outbreak in children, northern Australia. *Emerg Infect Dis* 2004;10(9):1593–1600
16. Bresee JS, Glass RI, Ivanoff B, Gentsch JR. Current status and future priorities for rotavirus vaccine development, evaluation and implementation in developing countries. *Vaccine* 1999;17(18):2207–2222
17. Grimwood K, Lambert SB. Rotavirus vaccines opportunities and challenges. *Hum Vaccin* 2009;5(1):4–16.
18. National Health and Medical Research Council. *The Australian Immunisation Handbook,* 9th edn. National Health and Medical Research Council. Canberra; 2008.
19. Nagy C, Roberts C, Cook H, Krause V. Introducing rotavirus vaccine in the Northern Territory. *The Northern Territory Disease Control Bulletin* 2007;14(1):3–7. Available from: http://www.nt.gov.au/health/docs/cdc_bulletin_mar_2007.pdf. Accessed 09 January 2009.
20. Schultz R. Rotavirus gastroenteritis in the Northern Territory, 1995–2004. *Med J Aust* 2006;185(7):354–356.
21. O'Grady K, Krause V, Andrews R. Immunisation coverage in Australian Indigenous children: time to move the goal posts. *Vaccine* 2009;27(2):307–312.
22. Menzies R, Turnour C, Chui 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:S1-S67.
23. Vlack S, Foster R, Menzies R, Williams G, Shannon C, Riley I. Immunisation coverage of Queensland Indigenous two-year-old children by cluster sampling and by register. *Aust N Z J Public Health* 2007;31(1):67–72.
24. Hsu VP, Staat MA, Roberts N, Thieman C, Bernstein DI, Bresee J, et al. Use of active surveillance to validate international classification of diseases code estimates of rotavirus hospitalizations in children. *Pediatrics* 2005;115(1):78–82.
25. Riordan FA, Quigley T. Estimating hospital admissions due to rotavirus gastroenteritis from hospital episode statistics. *J Infect* 2004;49(1):13–16.
26. Clothier HJ, Vu T, Sundararajan V, Andrews RM, Counahan M, Tallis GF, et al. Invasive pneumococcal disease in Victoria: a better measurement of the true incidence. *Epidemiol Infect* 2007;136:225–231.
27. Tate JE, Curns AT, Cortese MM, Weintraub ES, Hambidge S, Zangwill KM, et al. Burden of acute gastroenteritis hospitalizations and emergency department visits in US children that is potentially preventable by rotavirus vaccination: a probe study using the now-withdrawn RotaShield vaccine. *Pediatrics* 2009;123(3):744–749.

The burden of childhood influenza in a tertiary paediatric setting

David Lester-Smith, Yvonne A Zurynski, Robert Booy, Marino S Festa, Alison M Kesson, Elizabeth J Elliott

Abstract

Influenza is usually considered a mild winter-time illness but can be associated with a range of serious complications. We undertook a retrospective medical record review to study the impact of admissions of children with laboratory-confirmed influenza to The Children's Hospital at Westmead, Sydney, during 2007. One hundred and twenty-two children were identified, representing 530 hospital admission days. There was no clearly documented evidence of influenza vaccination for any patient eligible for vaccination. Fever (97.5%) and cough (69.7%) were the most frequent manifestations. Admissions occurred almost entirely between June and September with a peak in July (n=61, 50%). Two-thirds of the children were aged less than 2 years (median 1.5 years). Most (61.5%) had an underlying chronic medical disorder. Lumbar puncture was performed in 28 (23%) children, mostly infants aged less than 3 months (n=18). Antibiotics were commonly prescribed (67.2%), but use of available influenza-specific antiviral agents was uncommon (13.1%). The nosocomial infection rate was 9.8% and the clinical staff vaccination rate was low (less than 30%). Pneumonia was the most common complication (12.3%). No influenza-related deaths occurred. Influenza in young children poses a significant burden to health care services, tertiary admissions representing the tip-of-the-iceberg. Vaccination rates are inappropriately low in both eligible patients and hospital clinical staff. Early 'point of care' testing, use of influenza-specific antiviral agents, and extension of current vaccination schedules to include all children aged six to 23 months could considerably reduce over-investigation, unnecessary use of antibiotics and the health care impact of influenza. *Commun Dis Intell* 2009;33:209–215.

Keywords: influenza, child, hospitalisation, immunisation

Introduction

Influenza is a common illness of childhood and the burden of disease is highest among pre-school children[1,2] with attack rates up to 20%–30% each year in child care settings.[3] There is a wide range of symptom severity from minor respiratory illness to life-threatening multi-system complications and death.[4–13] Children act as a major viral reservoir during epidemics, transmitting infection to both their families and the community. Increasingly, the health care and wider socioeconomic costs of influenza are being recognised.[14,15] Economic modelling in the United States of America (USA) estimates the annual health care bill at $87.1 billion (CI $47.2–$149.5).[16] Australian data show that 82 cases per 100,000 hospitalisations and 0.2 per 100,000 deaths can be attributed to influenza in children aged less than 5 years.[17]

The current Australian Immunisation Schedule recommends influenza vaccination only for children at high risk for influenza and its complications; not routinely for all children.[18] This is in contrast to the USA where the American Advisory Committee on Immunization Practices (ACIP) recommends that all children aged 6 months to 18 years are immunised annually.[19] A recent systematic review by Matheson et al[20] confirmed the beneficial role of the neuraminidase inhibitors (Zanamivir and Oseltamivir) for treatment and probable prevention of influenza complications in children.

What is already known on this subject?

Influenza is a common infectious disease of childhood, widely regarded as a mild illness.

In Australia influenza vaccination is recommended only for children at high-risk of complications.

Point of care testing and influenza-specific antiviral agents are available and may reduce the annual impact of influenza on health care services and the wider community.

What this study adds

Influenza is a frequent cause of both hospital and intensive care admission: over 100 children admitted to one tertiary hospital in a single season, 10% required intensive care and over 500 hospital bed-days were occupied.

A large proportion of children did not have a risk factor and therefore were not eligible for influenza vaccination, so consideration of universal vaccination is required for more effective prevention.

Point of care testing and influenza-specific antiviral agents are rarely used and thus many children are managed with unnecessary antibiotics and invasive procedures such as lumbar puncture.

The 2007 influenza season was unusually severe with a number of paediatric deaths reported and higher than expected rates of hospitalisation, both in the USA[21] and Australia (www9.health.gov.au/cda/Source/Rpt_4.cfm). The Children's Hospital at Westmead is a large, tertiary paediatric teaching hospital in Sydney, Australia, with a bed capacity of 339 and serving a population of 549,760 children aged less than 16 years.[22] Our objective was to determine the burden of influenza admissions on this hospital during the 2007 influenza season.

Method

A retrospective medical record review was undertaken of all children admitted to the hospital with laboratory confirmed influenza A or B between 1 January and 31 December 2007, inclusive. Patients were identified by reviewing virology records. Data extracted from the records included patient characteristics, clinical presentation, underlying medical conditions, investigations, management and outcome.

A laboratory-confirmed case was defined as any child with influenza virus identified by direct immunofluorescence (DFA) from a nasopharyngeal aspirate (NPA). Samples negative on the initial DFA screen are subsequently cultured but culture-positive patients were not included in our study. NPA specimen testing is available on a daily basis and results available the same day, including weekends, during peak months, June to September.

Hospital infection control policy requires either isolation or co-location of children testing positive for influenza; however, it is possible that some DFA negative cases, which were not isolated, had influenza not yet confirmed by culture, which takes several days.

Nosocomial infection was conservatively defined as the onset of signs and symptoms at greater than 72 hours after admission to hospital. Fever was defined as an auxiliary temperature greater than 37.5°C, dyspnoea as increased respiratory effort or oxygen saturation less than 95%, and encephalopathy as an altered state of consciousness. Pneumonia was recorded if it was confirmed by chest radiograph. Secondary bacterial infections were defined by a positive culture from an appropriate clinical specimen. The remaining clinical manifestations were recorded as they appeared in the medical record.

Daily bed costs were estimated at $420 for a general paediatric bed and $1,250 for a paediatric intensive care unit bed. These figures do not include any additional costs e.g. medications, pathology, imaging and allied health costs.

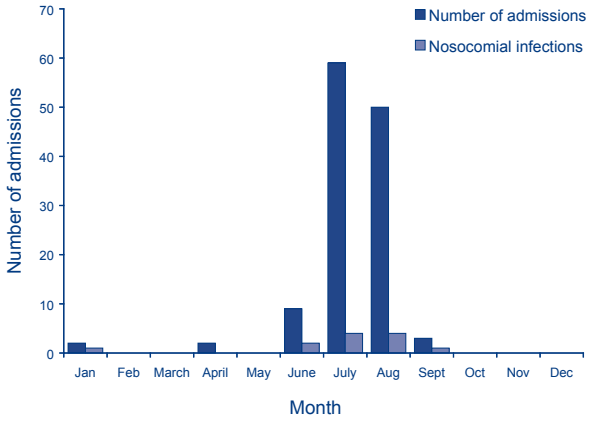
Ethical approval for this study was obtained from the Children's Hospital at Westmead Research Ethics committee (MR 2007-10-08). Data were analysed using descriptive statistics (SPSS v15).

Results

Of 155 influenza cases identified from virology records, 122 were admitted to hospital or were in hospital for another reason when they acquired influenza. Most children (119) had influenza A (H3N2) of the Brisbane/10/2007-like type and only three had influenza B: 1 Florida/4/2006-like type and 2 Malaysia/2506/2004-like types.

The majority (95.6%) of admissions occurred during the winter months, June to September (Figure 1), with a striking peak of admissions in July (n=61, 50%).

Figure 1: Seasonal variation in admissions for influenza and nosocomial infections



Patient characteristics

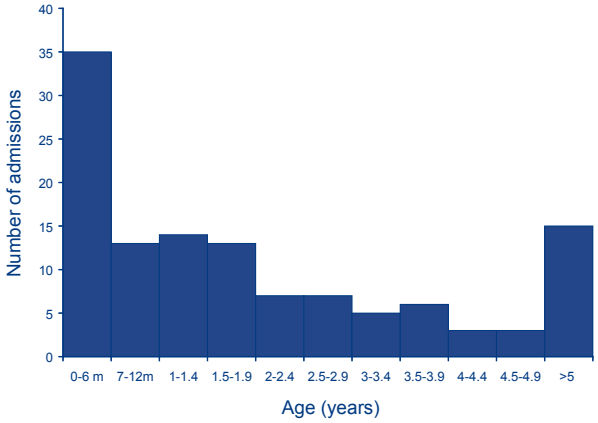
Of the 122 children admitted to hospital, 75 (61.5%) were aged less than 2 years and almost 35 (30%) were aged less than 6 months (Figure 2). The median age was 1.4 years (range: 1 week to 19.4 years) and 69 (56.6%) were male. Two patients identified as Aboriginal or Torres Strait Islander.

Influenza vaccination status was clearly recorded for only 1 unvaccinated child. There were no readily retrievable and clearly documented instances of prior influenza vaccination recorded in the other 121 cases.

Pre-existing chronic medical conditions were common (61.5%). Overall, 17% of children had primary or secondary immunodeficiencies, mostly associated with chemotherapy. Other chronic conditions included respiratory (n=12; 9.8%), neurological (n=8; 6.6%), neuromuscular (n=6;

4.9%), cardiac (n=5; 4.1%), endocrine/metabolic (n=4; 3.3%), gastrointestinal (n=3; 2.5%), renal (n=3; 2.5%); and haematological disorders (n=1; 0.8%). Seven babies were born preterm but none had been diagnosed with chronic lung disease or any other ongoing condition. Six other cases had other chronic complex medical disorders not categorised above. The proportion of children with chronic conditions increased from 20% in babies aged less than 6 months, to 55% in children aged between 6 months and 5 years and to 80% in children aged 5 years or older.

Figure 2: Age distribution of children admitted with influenza, 2007, by age group



Clinical presentation

Three-quarters of children first presented to medical services within 48 hours of the onset of symptoms, to either a primary health care setting or a hospital emergency department. Twelve patients required intensive care and nine of these needed ventilatory support for pneumonia, shock or apnoea. Three patients required emergency retrieval from another New South Wales hospital to our intensive care unit.

The main clinical manifestations at presentation were fever (mean temperature 38.8°C) and evidence of either an upper or lower respiratory tract infection indicated by cough, coryza or dyspnoea (Table 1). Only one half of cases (49.2%) had the triad of fever, cough and coryza and 14 (11.5%) presented with fever alone. One patient had dyspnoea alone whilst two were asymptomatic and diagnosed from a routine NPA following an inter-hospital transfer. Other presenting symptoms included malaise, vomiting, petechial rash and seizures (Table 1). Two of the patients with seizures were subsequently diagnosed with encephalopathy.

Nosocomial infection occurred in 12 (9.8%) patients (Figure 1). Five of these infections occurred

Table 1: Patient characteristics and clinical presentation

Patient characteristics (n=122)	n	%
Male	69	56.6
Median age (range)	1.4 (0.02 to 19.4)	
ATSI*	2	1.6
Influenza A	119	97.5
Immunocompromised	21	17.2
Other chronic disorder	54	44.3
Nosocomial infection	12	9.8
Documented influenza vaccination	1	0.08
Clinical presentation		
Fever	119	97.5
Cough	85	69.7
Coryza	79	64.8
Dyspnoea	29	23.8
Fever, cough and coryza	60	49.2
Fever alone	14	11.5
Other symptoms		
Malaise	31	25.4
Vomiting	16	13.1
Petechial rash	13	10.7
Mottled	8	6.6
Diarrhoea	8	6.6
Seizure	7	5.7
Headache	6	4.9
Sore throat	5	4.1
Apnoea	4	3.3
Myalgia	3	2.5

* Aboriginal or Torres Strait Islander.

in children admitted to 1 ward but there was no temporal association among these cases (Figure 1). The influenza vaccination rate for clinical staff at the Children's Hospital at Westmead for 2007 was estimated at 32%.

Investigations and management

Full blood count was performed at presentation in 113 (92.6%) admissions. The median total white cell count and neutrophil count were within the normal range (8.6 and 4.4 $*10^9$/L respectively). C-reactive protein was measured in 21 (17.2%) cases and the median C-reactive protein was mildly elevated at 21.7 mg/L (normal range 0–10 mg/L). Creatine kinase (measured in 2 patients with myalgia) was 223 in one and over 200,000 u/L (24–215u/L) in the other, and was associated with rhabdomyolysis and acute renal failure in the latter case.

Lumbar puncture was performed as an initial investigation in 28 (23%) children: none had a positive culture from the cerebrospinal fluid. The majority (18 of 28; 64%) of patients undergoing lumbar puncture were aged 3 months or less. Cerebral imaging was uncommon (5 occasions) and in only 1 child did the scan show new changes, consistent with a diagnosis of acute necrotising encephalopathy of childhood.

Antibiotics were the most common class of medication prescribed and two-thirds of patients received either oral or intravenous antibiotics. Third generation cephalosporins were the most commonly prescribed antibiotics (Table 2). Children most likely to receive antibiotics were infants aged under 3 months and presenting with fever (n=16). The documented reasons for commencing antibiotics are outlined in Table 2. In 5 cases there was no clearly documented rationale for starting antibiotics.

Supplemental oxygen was required by 23% of children: all presented with dyspnoea, were desaturated in room air and diagnosed with either pneumonia or bronchiolitis.

Overall, the prescription of specific anti-influenza viral agents was uncommon (n=16; 13.1%), except in the oncology and bone marrow transplant (BMT) setting where Oseltamivir was prescribed in 11 patients. Only 5 non-oncology/BMT patients received antiviral medication.

Complications and outcome

A wide range of complications occurred, most commonly pneumonia (n=15; 12.3%), requiring ventilatory support in 6 patients (Table 3). Other complications included encephalopathy (2) and rhabdomyolysis with associated acute renal failure (1).

Four children had co-infection with pertussis (confirmed by polymerase chain reaction) and two had co-infection with respiratory syncytial virus (RSV) on NPA. One patient had a positive blood culture (enterococcus) from an infected central venous access port, unrelated to the influenza illness.

The overall length of hospital stay ranged from 1 day to 50 days (median stay 2 days). The median length of stay for ICU patients was 2 days (range 1–25 days).

No deaths were attributed to influenza in our hospital during 2007. Only 1 child had residual physical problems following influenza due to the requirement for multiple fasciotomies for compartment syndrome, secondary to severe rhabdomyolysis, but was making good progress at follow-up.

Table 2: Investigations and management

Investigations	n	Median (range)
WCC (*10^9/L)	113	8.6 (0.5–57.9)
Neutrophils (*10^9/L)	113	4.4 (0.2–47.5)
CRP (mg/L)	21	21.7 (0–500)
		%
Lumbar puncture	28	22.9
Positive culture	Nil	N/A
Age <3 months	18	64.0
Age 3–6 months	3	10.7
Age 6–24 months	5	17.9
Age >24 months	2	7.1
CT head	5	4.1
New CT findings	1	0.8
Management		
Oxygen	28	23.0
Antibiotics	82	67.2
Oral	8	6.6
Intravenous	74	60.6
Antibiotic indication		
Fever < 3 months	16	13.1
Immunocompromised	15	12.3
Pneumonia	14	11.5
Petechial rash	10	8.2
Shock (fluid bolus)	8	6.6
General practitioner initiated	5	4.1
No clear indication	5	4.1
Raised WCC	3	2.5
Other indication	14	11.5
Antibiotic usage	82	67.2
Cefotaxime/ceftriaxone	25	20.5
Gentamicin	23	18.9
Ampicillin	20	16.4
Benzylpenicillin	16	13.1
Oncology 1st line*	13	10.7
Other	17	13.9
Antiviral agent (Oseltamivir)	16	13.1
Oncology	11	9.0
Other indications†	5	4.1

WCC=white cell count, CRP=C-reactive protein, CT=computerised tomography, N/A=not applicable

* Timentin, gentamicin, cephalothin.

† Other indications = post liver transplantation (1 case), steroid use for glomerulonephritis (1 case), metabolic disorder (2 cases) and general patient (1 case).

Table 3: Complications, co-infection and outcome

Complications*	n	%
Pneumonia	15	12.3
Shock (requiring fluid bolus)	13	10.7
Ventilated (IPPV+CPAP)	9	7.4
Bronchiolitis	8	6.6
Rapid deterioration	4	3.3
Inotrope use	3	2.5
Seizures	3	2.5
Clotting abnormality	3	2.5
Encephalopathy	2	1.6
Myositis	2	1.6
Co-infection		
Pertussis (PCR)	4	3.3
RSV	2	1.6
Coliform UTI	2	1.6
Strep pyogenes T/S	1	0.8
Pseudomonas sputum	1	0.8
Enterobacter CVL	1	0.8
Outcome		
Discharged alive	122	100
Associated disability	1	0.8
Length of stay	**Median (range)**	
Hospital	2 (1–50)	
PICU (12 patients)	2 (1–25)	

IPPV=intermittent positive pressure ventilation, CPAP=continuous positive airway pressure, RSV=Respiratory syncytial virus, T/S= throat swab, CVL=central venous line, UTI=urinary tract infection, PICU=paediatric intensive care unit, PCR=polymerase chain reaction.

* Some patients had more than 1 complication; complications occurring in only 1 patient included rhabdomyolysis, acute renal failure, compartment syndrome, thrombocytopenia, hypoglycaemia and pansinusitis.

Discussion

We have demonstrated the considerable impact of laboratory-confirmed influenza in children admitted to a tertiary children's hospital in Sydney, Australia. This is the largest reported case series we know of from a single tertiary paediatric centre during 1 influenza season, and equates to a total of 530 hospital admission days (including 52 intensive care admission days). This imposes a significant burden on hospital resources, equivalent to an estimated $264,000 AUD in direct bed costs alone. Our data are likely to underestimate the true burden of hospitalisation as in some children the disease will go unrecognised and we have no estimates of indirect costs such as lost productivity due to parental work absences.

The main burden of influenza in our cohort was among children under 2 years of age. Clinical manifestations were mainly respiratory, but only one half presented with the triad of fever, cough and coryza. A diagnosis of influenza should also be considered in the context of fever alone or fever plus 1 respiratory symptom. Influenza-related complications occurred in almost one-third of patients, with pneumonia being the most common. Influenza-associated encephalopathy was diagnosed in 2 patients and is a rare but well described serious complication.[11,23,24] Severe rhabdomyolysis[4,25] leading to acute renal failure and compartment syndrome (necessitating fasciotomies) is also a rare but well recognised serious complication, and occurred in 1 patient. No deaths were attributed to influenza in 2007, in contrast to 2003 when 3 deaths occurred in our hospital.[12]

Despite evidence that the early use of anti-influenza medication reduces the duration of illness and influenza complications, particularly otitis media,[20] only 13.1% of eligible children in our series received anti-influenza drugs. This suggests we are presently omitting a proven therapy. Children most likely to receive anti-influenza drugs were immunocompromised, mainly in the oncology and BMT clinical setting. Currently in Australia, Oseltamivir (Tamiflu™) is licensed for use in children aged 1 year or over by the Therapeutic Goods Administration of Australia, leaving 48 (39.3%) children in our series ineligible for treatment. There is a need for clinical trials to evaluate the role of anti-influenza medications in infants. No resistance to Oseltamivir of influenza A isolates was reported in Australia during winter 2007. This is in contrast to the growing resistance to Oseltamivir in 2009 isolates in the Northern Hemisphere, further emphasising the importance of vaccination.

'Point of care testing' (POCT) or 'near-patient' testing allows for the rapid diagnosis and treatment of influenza in the primary health care setting or at the inpatient's bedside.[26] Its use remains uncommon: only 1 patient in our series was thus diagnosed. This allowed treatment with an antiviral agent and potentially interrupted transmission of the disease. The majority of children in our series (77%) had first presented to medical services (usually their GP) within 48 hours of onset of symptoms and early diagnosis would have allowed initiation of antiviral treatment and potentially prevented admission in age-eligible patients.

Although influenza is known to predispose to secondary bacterial infection[27–30] there were no documented significant bacterial infections in this series. Despite this, over two-thirds of children received antibiotics, mainly third generation cephalosporins. In the absence of rapid POCT for influenza, there was a clear rationale for the instigation of antibiotics in all but five of the cases.

Free universal influenza vaccination of young children is not included on the National Immunisation Program[18] in Australia, although children with chronic medical problems are eligible. Documentation of the influenza vaccination status of eligible children in our study was poor; it was clearly documented for only 1 child. The Australian Childhood Immunisation Register does not collect information about influenza vaccinations in children because this vaccine is not on the Australian Immunisation Schedule. Nevertheless, it is noteworthy that amongst children requiring intensive care in our series, only three of 12 were eligible for vaccination due to a pre-existing chronic condition. The remainder were either previously healthy or aged less than 6 months and thus ineligible.[18] Overall, 28.7% of children were aged less than 6 months and not eligible for vaccination, due to the limited immunogenicity of the vaccine documented in this age group.[31]

A relatively high nosocomial influenza infection rate (9.8%) was found[32–34] and five of these cases occurred in 1 ward. During 2007 the hospital clinical staff influenza vaccination rate was estimated at only 32%. This is likely to be an overestimate as there were 431 recorded instances of hospital-based influenza vaccination out of a total of 1,346 full-time-equivalent medical, nursing and allied health staff and this does not account for the high proportion of part-time workers. This is despite a high-visibility staff vaccination campaign and a mobile staff vaccination clinic. Increased staff immunisation uptake along with stricter adherence to hand-hygiene may reduce this nosocomial infection rate. The possible impact of co-locating NPA negative, but true influenza, cases with other children on nosocomial infection rates is uncertain but potentially significant.

Current practice in the USA is for all children aged 6 months to 18 years to be offered vaccination annually against influenza.[19] Milne et al[12] and Isaacs[35] called for a similarly inclusive schedule to be adopted in Australia and our study adds further evidence to support this. Extending the immunisation schedule to include all children aged 6–23 months[35] would reduce the burden of influenza in the community and on health care services. Meanwhile, in view of the high proportion of children with medical co-morbidities admitted to hospital due to influenza it is important that health professionals involved in the care of such children ensure that this vulnerable group are immunised annually.

The heavy burden of influenza admissions to a tertiary paediatric centre is well demonstrated by this retrospective study, representing the tip-of-the-iceberg of the total number of community cases. Indeed, the number of admissions is most likely an underestimate as some cases will go unrecognised. A high nosocomial infection rate stresses the importance of annual staff influenza vaccination and hand hygiene. The development of treatment protocols and education of health professionals is needed to optimise the diagnosis and treatment of influenza, to reduce the use of antibiotics, over-investigation and to increase vaccination and the use of influenza-specific antiviral agents in eligible children. Economic evaluation of an extended immunisation schedule, point-of-care testing for influenza and routine use of anti-influenza medication in eligible patients is now necessary to help inform health policy.

Acknowledgements

The activities of the Australian Paediatric Surveillance Unit are supported by the Australian Government Department of Health and Ageing; the National Health and Medical Research Council Enabling Grant No: 402784; the National Health and Medical Research Council Practitioner Fellowship no: 457084 (E Elliott); the Discipline of Paediatrics and Child Health, Faculty of Medicine, University of Sydney; and the Royal Australasian College of Physicians.

The researchers involved in this study are independent from the funders.

Author details

David Lester-Smith[1,2]
Yvonne A Zurynski[1,2]
Robert Booy[2,3]
Marino S Festa[4]
Alison M Kesson[2,5]
Elizabeth J Elliott[1,2]

1. Australian Paediatric Surveillance Unit; The Children's Hospital at Westmead, New South Wales
2. Discipline of Paediatrics and Child Health, The University of Sydney, New South Wales
3. National Centre for Immunisation Research and Surveillance, The Children's Hospital at Westmead, New South Wales
4. Paediatric Intensive Care Unit, The Children's Hospital at Westmead, New South Wales
5. Department of Infectious Diseases and Microbiology, The Children's Hospital at Westmead, New South Wales

Corresponding author: Dr David Lester-Smith, Research Fellow, Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Locked Bag 4001, WESTMEAD NSW 2145. Telephone: +61 2 9845 1202 / 3005. Facsimile: +61 2 9845 3082. Email: davidl7@chw.edu.au

References

1. Ploin D, Gillet Y, Morfin F, Fouilhoux A, Billaud G, Liberas S, et al. Influenza burden in febrile infants and young children in a pediatric emergency department. *Pediatr Infect Dis J* 2007;26(2):142–147.
2. Smitherman HF, Caviness AC, Macias CG. Retrospective review of serious bacterial infections in infants who are 0 to 36 months of age and have influenza A infection. *Pediatrics* 2005;115(3):710–718.

3. Iskander M, Booy R, Lambert S. The burden of influenza in children. *Current Opinion Infect Dis* 2007;20(3):259–263.
4. Agyeman P, Duppenthaler A, Heininger U, Aebi C. Influenza-associated myositis in children. *Infection* 2004;32(4):199–203.
5. Aymard M, Valette M, Luciani J, Sentinel Physicians from the Grippe et Infections Respiratoires Aigües Pédiatriques Network. Burden of influenza in children: preliminary data from a pilot survey network on community diseases. *Pediatr Infect Dis J* 2003;22(10:Suppl):S211–214.
6. Bhat N, Wright JG, Broder KR, Murray EL, Greenberg ME, Glover MJ, et al. Influenza-associated deaths among children in the United States, 2003–2004. *N Engl J Med* 2005;353(24):2559–2567.
7. Centers for Disease Control and Prevention. Update: influenza-associated deaths reported among children aged <18 years--United States, 2003–04 influenza season. *MMWR Morb Mortal Wkly Rep* 2004;52(51–52):1254–1255.
8. Grose C. The puzzling picture of acute necrotizing encephalopathy after influenza A and B virus infection in young children. *Pediatr Infect Dis J* 2004;23(3):253–254.
9. Heikkinen T, Silvennoinen H, Peltola V, Ziegler T, Vainionpaa R, Vuorinen T, et al. Burden of influenza in children in the community. *J Infect Dis* 2004;190(8):1369–1373.
10. Kappagoda C, Isaacs D, Mellis C, Peat J, De Silva L, O'Connell A. Critical influenza virus infection. *J Paediatr Child Health* 2000;36(4):318–321.
11. Maricich SM, Neul JL, Lotze TE, Cazacu AC, Uyeki TM, Demmler GJ, et al. Neurologic complications associated with influenza A in children during the 2003–2004 influenza season in Houston, Texas. *Pediatr* 2004;114(5):e626–e633.
12. Milne BG, Williams S, May ML, Kesson AM, Gillis J, Burgess MA. Influenza A associated morbidity and mortality in a paediatric intensive care unit. *Commun Dis Intell* 2004;28(4):504–509.
13. Poehling KA, Edwards KM, Weinberg GA, Szilagyi P, Staat MA, Iwane MK, et al. The underrecognized burden of influenza in young children. *N Engl J Med* 2006;355(1):31–40.
14. Keren R, Zaoutis TE, Saddlemire S, Luan XQ, Coffin SE. Direct medical cost of influenza-related hospitalizations in children. *Pediatr* 2006;118(5):e1321–e1327.
15. Tsolia MN, Logotheti I. Papadopoulos NG, Mavrikou M, Spyridis NP, Drossatou P, et al. Impact of influenza infection in healthy children examined as outpatients and their families. *Vaccine* 2006;24(33–34):5970–5976.
16. Molinari NA, Ortega-Sanchez IR, Messonnier ML, Thompson WW, Wortley PM, Weintraub E, et al. The annual impact of seasonal influenza in the US: measuring disease burden and costs. *Vaccine* 2007;25(27):5086–5096.
17. 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-152.
18. National Health and Medical Research Council. 3.9 Influenza. *The Australian Immunisation Handbook*. 2007. National Health and Medical Research Council; Canberra: pp160–169.
19. Fiore AE, Shay DK, Haber P, Iskander JK, Uyeki TM, Mootrey G, et al. Prevention and control of influenza. Recommendations of the Advisory Committee on Immunization Practices (ACIP), 2007. *MMWR Morb Mortal Wkly Rep* 2007;56(RR–6):1–54.
20. Matheson NJ, Harnden AR, Perera R, Sheikh A, Symmonds-Abrahams M. Neuraminidase inhibitors for preventing and treating influenza in children. *Cochrane Database Syst Rev* 2007;(1):CD002744.
21. Centers for Disease Control and Prevention. Update: influenza activity--United States and worldwide, May 20–September 15, 2007. *MMWR Morb Mortal Wkly Rep* 2007;56(38):1001–1004.
22. Childrens Hospital at Westmead. Demographic and child health profile. The Children's Hospital at Westmead Healthcare Services Plan 2007–2011. 2008: pp16–18.
23. Mizuguchi M, Yamanouchi H, Ichiyama T, Shiomi M. Acute encephalopathy associated with influenza and other viral infections. *Acta Neurol Scand* 2007;186:45–56.
24. Newland JG, Laurich VM, Rosenquist AW, Heydon K, Licht DJ, Keren R, et al. Neurologic complications in children hospitalized with influenza: characteristics, incidence, and risk factors. *J Pediatr* 2007;150(3):306–310.
25. Augustin SL, Horton S, Thuys C, Bennett M, Claessen C, Brizard C. The use of extracorporeal life support in the treatment of influenza-associated myositis/rhabdomyolysis. *Perfusion* 2006;21(2):121–125.
26. Poehling KA. Zhu Y. Tang YW. Edwards K. Accuracy and impact of a point-of-care rapid influenza test in young children with respiratory illnesses. *Archives Pediatr Adolescent Med* 160(7):713–718, 2006.
27. Hageman JC, Uyeki TM, Francis JS, Jernigan DB, Wheeler JG, Bridges CB, et al. Severe community-acquired pneumonia due to *Staphylococcus aureus,* 2003–04 influenza season. *Emerg Infect Dis* 2006;12(6):894–899.
28. Jensen ES, Lundbye-Christensen S, Samuelsson S, Sorensen HT, Schonheyder HC. A 20-year ecological study of the temporal association between influenza and meningococcal disease. *Eur J Epidemiol* 2004;19(2):181–187.
29. O'Brien KL, Walters MI, Sellman J, Quinlisk P, Regnery H, Schwartz B, et al. Severe pneumococcal pneumonia in previously healthy children: the role of preceding influenza infection. *Clin Infect Dis* 2000;30(5):784–789.
30. Podewils LJ, Liedtke LA, McDonald LC, Hageman JC, Strausbaugh LJ, Fischer TK, et al. A national survey of severe influenza-associated complications among children and adults, 2003–2004. *Clin Infect Dis* 2005;40(11):1693–1696.
31. Neuzil KM, Edwards KM. Influenza vaccines in children. [Review] [52 refs]. *Seminars Pediatr Infect Dis* 2002;13(3):174–181.
32. Maltezou HC, Drancourt M. Nosocomial influenza in children. [Review] [71 refs]. *J Hosp Infect* 2003;55(2):83–91.
33. Slinger R, Dennis P. Nosocomial influenza at a Canadian pediatric hospital from 1995 to 1999: opportunities for prevention. *Infect Control Hosp Epidemiol* 2002;23(10):627–629.
34. Evans ME, Hall KL, Berry SE. Influenza control in acute care hospitals. *Am J Infect Control* 1997;25(4):357–362.
35. Isaacs D. Should all Australian children be vaccinated against influenza? Questions of cost-effectiveness, vaccine efficacy and feasibility are yet to be answered. *Med J Aust* 2005;182(11):553–554.

Prevalence of transmitted HIV drug resistance since the availability of highly active antiretroviral therapy

Jessica S Russell, Doris Chibo, Matthew B Kaye, Megan L Gooey, Louise A Carolan, Anastasia Papadakis, Jodie A Nicholls, Chris J Birch

Abstract

We investigated the prevalence of HIV-1-associated transmitted drug resistance (TDR) in Victoria from the time of first availability of highly active antiretroviral therapy. Drug resistance genotyping was performed on virus present in blood samples collected from individuals with serologically confirmed primary infection, between 1996 and 2007. The significance of any mutations detected was interpreted according to a standardised list of drug resistance mutations. The main outcomes measured were the prevalence by year of TDR to any antiretroviral drug class, the numbers of infected individuals with TDR involving multiple drug classes, and the resistance mutations implicated in all cases. There was an average annual prevalence of TDR of 16%, predominantly associated with nucleoside and non-nucleoside reverse transcriptase (RT) inhibitors and most commonly occurring at codons 41, 103 and 215 in the RT. The prevalence of thymidine-associated mutations remained high throughout the period of study. While mutations known to cause resistance to protease inhibitors were uncommon, they were present in several individuals infected with virus resistant to multiple drug classes. The prevalence of TDR in Victoria is similar to geographical locations outside Australia where HIV-specific drug treatment is widely available. Primary infection with drug resistant HIV is a future treatment issue for the individual patient and for the wider population at risk of infection. At this time TDR shows no sign of waning and our data support recent treatment guidelines recommending baseline testing for TDR before therapy is initiated. *Commun Dis Intell* 2009;33:216–220.

Keywords: HIV, AIDS, drug resistance

Introduction

The availability of highly active antiretroviral therapy (HAART) has produced significant decreases in the morbidity and mortality of patients infected with HIV. However, drug resistance is generated in a proportion of treated patients and may be directly transmitted from them to treatment-naïve individuals at the time of their primary infection. This process and its outcome is referred to as transmitted drug resistance (TDR).

Reports on TDR published during the last 10 years show average prevalences in developed countries ranging from 10 to 20% where use of HAART is widespread, with some variation from year-to-year in certain locations.[1–5] In regions where HAART-usage is less common, for example some areas of Asia and Africa, TDR has also been documented.[6,7] TDR has been regularly demonstrated for 3 antiretroviral drug classes, the nucleoside and non-nucleoside reverse transcriptase inhibitors (NRTIs and NNRTIs, respectively) and protease inhibitors (PIs).[1,8] A single case of transmitted resistance to the fusion inhibitor enfuvirtide has been reported.[9] Several cases of acquired resistance to the first available HIV integrase inhibitor (raltegravir) have been described[10] but there are no reported cases of transmitted resistance to this drug.

The presence of resistance mutations in HIV strains transmitted at the time of infection theoretically diminishes the efficacy of individual drugs to which the mutations apply and may lower the genetic barrier to other drugs in the same class. While a large clinical study has shown the clinical impact of TDR to be subtle during first-line therapy,[11] a number of reports suggest it limits treatment options and clinical response is improved by genotype-directed therapy.[2,12] The impact of TDR on second-line and subsequent treatments is largely unknown. Drug resistance testing prior to commencement of antiretroviral (ARV) drug therapy is now incorporated in the HIV treatment guidelines of many countries including Australia, where it is recommended as a baseline test even if treatment is not being considered immediately.[13]

Evidence for TDR in the above studies has been obtained by genotyping. This is also the recommended method in Australia because of its availability in State HIV reference laboratories. The interpretation applied to specific mutations detected by genotyping may influence the reported prevalence, and a standardised mutation list has been proposed that will enable TDR surveillance programs, which are increasing in number and

location, to produce comparable estimates of TDR rates.[14] It is important, therefore, that laboratory testing reliably distinguishes recently infected cases from newly diagnosed cases.

This report presents the results of 12 years of surveillance of TDR in Victorian patients, commencing in 1996 when HAART became widely used for the first time, and concluding with cases to the end of 2007. The results have important implications for first-line therapy in many patients and emphasise the continuing need for baseline resistance testing as part of HIV clinical practice.

Methods

Genotyping for TDR was performed on available plasma samples from individuals infected with HIV according to one of the following test results: western blot evolving from negative or indeterminate profile to full profile within 12 months, full western blot profile following a negative enzyme immunoassay (EIA) within 12 months, or (only for patients infected in 1999 and 2000) a detuned EIA test result suggesting recent infection.[15] A total of 466 patient samples were tested by the Victorian Infectious Diseases Reference Laboratory (VIDRL), representing approximately 15% of likely new cases of infection over the study period. Because the VIDRL is the reference laboratory for HIV diagnosis in Victoria, this cohort is not thought to be subject to referral bias. The testing period included the years 1996 to 2007 inclusive. Statistical analysis was undertaken using the Pearson chi squared test. Formal ethical approval for the study was not sought. Testing was performed as a result of doctors' requests for genotyping or, prior to the widespread availability of genotyping, as part of the laboratory's surveillance role.

Genotyping of the reverse transcriptase and protease regions was performed on all samples as previously described.[16] Genotyping of the HIV gp41 region for enfuvirtide susceptibility was performed only on samples from the year 2000 onwards as previously described.[17] Testing for evidence of TDR associated with raltegravir therapy was not undertaken. Inclusion of mutations as being associated with TDR was made according to a proposed standardised list of drug-resistance mutations.[14] To enable comparison of the prevalence of transmitted mutations with that of acquired mutations, the results of genotyping performed since 1996 on samples from more than 1,500 patients with acquired drug resistance, were extracted from our electronic laboratory reporting system to generate a database providing the frequency of all recognised resistance mutations. These results were then compared to those obtained on patients with known recent infections according to the above criteria.

Results

Genotypic evidence for TDR was present in 75 of 466 (16%) recently infected individuals tested between 1996 and 2007, inclusive (Table 1). Of the cohort investigated 449 were males and 17 were females. The median age was 35. The majority of individuals were infected via homosexual transmission. Of the 75 patients in whom resistant virus was detected, 72 (96%) were male. All cases involved subtype B HIV strains except for 1 female infected with subtype C virus and a male with a subtype CRF01-AE infection.

Although some fluctuation in the peak annual prevalence of TDR occurred (33% in 1996 versus 9% in 2007, Table 1), in the intervening years the prevalence was broadly stable and overall there was no statistically significant difference from year to year ($P=0.75$). Resistance was mainly associated with NRTIs and involved 50 cases (67%). Resistance to NNRTIs was present in 27 cases (36%), while resistance to PIs was relatively uncommon (10 cases, 13%). Ten patients were infected with HIV strains resistant to more than 1 drug class and two of these were infected with virus resistant to 3 classes. There were no cases of resistance to enfuvirtide.

Codons in RT and protease associated with TDR are shown in Table 2. Mutations associated with resistance to PIs were rare. The 2 most common individual mutations, M41L and K103N, were present in 25% and 28% of TDR cases, respectively, compared with 29% and 19%, respectively, in cases of acquired resistance. Thymidine-associated mutations (TAMS) 41L, 67N, 70R, 210W, 215Y (plus any 215-revertant) and K219Q were also very common, with no obvious evidence for a decline in their prevalence over time (Table 1). Although T215Y mutations were less common in the TDR population than in patients with acquired resistance (4% versus 23%, respectively), the inclusion of 215-revertants raised the incidence of any mutation at codon 215 to 45% in the TDR cohort.

Five of the 75 (7%) cases involved a methionine (M) mutation at codon 184, a prevalence considerably lower than observed in patients in our database with acquired resistance (35%).

Discussion

Over the 12 years of this investigation the overall prevalence of TDR in Victoria was 16% and this was mainly associated with treatment involving NRTIs, zidovudine in particular. More than half the TDR cases we identified were infected with virus containing TAMs, although in 1996 and 2007 none were detected. However, in each subsequent year including 2008 (results not shown), these mutations were once again present in some cases. Therefore it appears that many of the cases in this

Table 1: Number and percentage of cases of transmitted drug resistance, the drug classes implicated according to the mutations detected and the number of cases infected with viruses containing thymidine-associated mutations, 1996 to 2007

Year	Number of tests performed	TDR patients		Drug classes						TAMs	
		n	%	PI	NRTI	NNRTI	PI/NRTI	NRTI/ NNRTI	All classes	n	%
1996	9	3	33		3					3	100
1997	12	1	8	1						0	0
1998	14	3	21		2			1		2	67
1999	27	7	26		6			1		7	100
2000	63	12	17	1	8	1	1		1	10	83
2001	21	3	14		2	1				2	67
2002	48	7	15	2	3	1		1		3	43
2003	38	6	16		1	4	1			2	33
2004	43	7	16		5			2		6	86
2005	55	8	15	1	4	2			1	5	63
2006	72	12	17		6	5		1		5	42
2007	64	6	9	1		5				0	0
Total	466	75	16	6	40	19	2	6	2	45	60

TDR Transmitted drug resistance.
TAMs Thymidine-associated mutations.
PI Protease inhibitor.
NRTI Nucleoside reverse transcriptase inhibitor.
NNRTI Non-nucleoside reverse transcriptase inhibitor.

Table 2: Resistance mutations associated with transmitted drug resistance cases

NRTI		NNRTI		PI	
Mutation	n	Mutation	n	Mutation	n
T215S/D/C/E/I	31	K103N	21	L90M	4
M41L	19	Y181C	4	M46I	3
K219Q	7	L100I	1	I84V	2
M184V	5	V106A	1	L24I	1
L210W	5	Y188H	1	G48V	1
D67N	4	Y188L	1	I54L	1
K70R	4	L190S	1	I54T	1
T215Y	3			I54V	1
M184I	2			V82A	1
K65R	1				
L74V	1				
V75A	1				
V75M	1				

Mutations were included according to a published list.[14] The numbers indicated refer to the total number of individuals with this mutation during the study period. Some individuals had more than one mutation.

PI Protease inhibitor.
NRTI Nucleoside reverse transcriptase inhibitor.
NNRTI Non-nucleoside reverse transcriptase inhibitor.

and other studies occur as a result of the long-term use of zidovudine, including monotherapy and dual therapy prior to the advent of HAART.

As more potent drugs have become available for use in clinical practice and the once-widespread use of zidovudine has declined, a concomitant decrease in TDR prevalence might have been expected. However, despite increasing use of abacavir/lamivudine and tenofovir/emtricitabine in first-line therapy in recent years, we identified only 1 case involving the K65R mutation associated with tenofovir resistance[18] and the M184V resistance mutation remained uncommon. In our acquired resistance database, M184V is the single most common mutation, present in nearly one third of all patients with resistance. However it was only detected in 7% of TDR cases, which is consistent with previous reports. This observation is likely to be related to a combination of reduced transmission efficiency associated with low viral titre and poor replicative fitness of these mutants in the transmitter, as well as impaired fitness in the absence of a selective drug pressure in the TDR cases.[19]

It is unclear why the prevalence of K103N mutations was higher in TDR cases than cases of acquired resistance and it is possible that the apparent dif-

ference between the 2 populations is coincidental. Nevertheless, we have previously reported the common occurrence of both mutations in patients with untreated primary HIV infection in Melbourne,[19] suggesting that viruses with a K103N mutation may be preferentially transmitted.

In contrast to the many reports from Europe and the United States of America, only 1 study on TDR in Australia has been described.[21] It showed a low and stable rate of resistance to PIs (consistent with this and many other studies in a variety of geographical locations) and a decrease in resistance to inhibitors of the HIV RT between 1992 and 2001, a period overlapping introduction of HAART to Australia in 1996. This decline is likely to reflect the addition of NNRTIs and PIs to ARV treatment that until that time comprised a choice of zidovudine, didanosine or zalcitabine. The increased antiviral potency associated with HAART is likely to have reduced resistance rates associated with monotherapy and, as a consequence, the incidence of TDR. From 1997, the rates of TDR in Sydney and Melbourne have been similar.[21] Nevertheless there are differences between the data gathered in these cities when the study times overlapped. In particular, several patients living in Melbourne were infected with virus resistant to multiple drug classes whereas none were seen in Sydney during the study period examined. Whether this difference has been sustained will require further studies on patients with primary infection in Sydney.

There are some drawbacks to investigations of the type we describe. In particular we studied only a proportion of the total number of newly infected patients detected on an annual basis in Victoria. In addition, the time post-infection of blood samples for genotyping varied from patient to patient, possibly biasing the proportions of individual mutations observed. This limits any generalisation that can be made between mutations considered to be transmitted versus those likely to be acquired as a result of failure of drug therapy. Finally the genotyping method employed, while standard in most reference laboratories undertaking such studies, has limited sensitivity. As such the overall TDR prevalence is possibly underestimated.

With the exception of a small number of published studies,[5,21,22] investigations on the prevalence of TDR have involved relatively short time frames post the availability of HAART. This approach highlights the importance of TDR but does not show changes in either its prevalence or the ARV drug classes implicated, both of which would be predicted to evolve as new drugs become available clinically and the use of some drugs declines. Our study is one of the longest reported to date on the prevalence of TDR. As such, it reveals a stable prevalence in Victoria over 12 years on a background of a high incidence of TAMs. Despite the relatively stable TDR rate in this location, the overall prevalence of 16% highlights the need for baseline resistance testing prior to commencing HAART.

Acknowledgements

The investigation of enfuvirtide-associated mutations in TDR cases was supported by a grant from Roche Products Pty Ltd under its Fuzeon Initiatives for National Discoveries Awards (2005). We thank Dr Gillian Hales (Roche Products) for her interest in the study. We also thank Dr Fraser Drummond for supplying the dates of first clinical use of antiretroviral drugs in Australia.

Author details

Jessica S Russell, Medical Scientist
Doris Chibo, Senior Medical Scientist
Matthew B Kaye, Medical Scientist
Megan L Gooey, Medical Scientist
Louise A Carolan, Medical Scientist
Anastasia Papadakis, Medical Scientist
Jodie A Nicholls, Medical Scientist
Chris J Birch, Senior Medical Scientist

HIV Characterisation Laboratory, Victorian Infectious Diseases Reference Laboratory, North Melbourne Victoria

Corresponding author: Ms Jessica Russell, Victorian Infectious Diseases Reference Laboratory, 10 Wreckyn Street, NORTH MELBOURNE VIC 3051. Telephone: +61 3 9342 2628. Facsimile: +61 3 9342 2666. Email: jessica.russell@mh.org.au

References

1. Grant RM, Hecht FM, Warmerdam M, Liu L, Liegler T, Petropoulos CJ, et al. Time trends in primary HIV-1 drug resistance among recently infected persons. *JAMA* 2002;288(2):181–188.
2. Little SJ, Holte S, Routy JP, Daar ES, Markowitz M, Collier AC, et al. Antiviral drug resistance among patients recently infected with HIV. *N Engl J Med* 2002;347(6):385–394.
3. Simon V, Vanderhoeven J, Hurley A, Ramratnam B, Louie M, Dawson K, et al. Evolving patterns of HIV-1 resistance to antiretroviral agents in newly infected individuals. *AIDS* 2002;16(11):1511–1519.
4. Ibe S, Hotta N, Takeo U, Tawada Y, Mamiya N, Yamanaka K, et al. Prevalence of drug-resistant human immunodeficiency virus type 1 in therapy-naïve patients and usefulness of genotype testing. *Microbiol Immunol* 2003;47(7):499–505.
5. Shet A, Berry L, Mohri H, Mehandru S, Chung C, Kim A, et al. Tracking the prevalence of transmitted antiretroviral drug-resistant HIV-1: a decade of experience. *J Acquir Immune Defic Syndr* 2006;41(4):439–446.
6. Choi JY, Kim EJ, Park YK, Lee JS, Kim SS. National survey for drug-resistant variants in newly diagnosed antiretroviral drug-naïve patients with HIV/AIDS in South Korea: 1999–2005. *J Acquir Immune Defic Syndr* 2008;49(3):237–242.

7. Barth RE, Wensing AM, Tempelman HA, Moraba R, Schuurman R, Hoepelman AI. Rapid accumulation of nonnucleoside reverse transcriptase inhibitor-associated resistance: evidence of transmitted resistance in rural South Africa. *AIDS* 2008;22(8):2210–2212.
8. Masquelier B, Bhaskaran K, Pillay D, Gifford R, Balestre E, Jørgensen LB, et al. Prevalence of transmitted HIV-1 drug resistance and the role of resistance algorithms: data from seroconverters in the CASCADE collaboration from 1987 to 2003. *J Acquire Immune Defic Syndr* 2005;40(5):505–511.
9. Peuchant O, Capdepont S, Ragnaud JM, Aurillac-Lavignolle V, Thiébaut R, Fleury H, et al. Primary resistance to enfuvirtide (T20) in recently infected, antiretroviral-naïve patients from the ANRS Aquitane cohort. *Antivir Ther* 2007,12(4):559–562.
10. Malet I, Delelis O, Valantin MA, Montes B, Soulie C, Wirden M, et al. Mutations associated with failure of Raltegravir treatment affect integrase sensitivity to the inhibitor *in vitro*. *Antimicrob Agents Chemother* 2008;52(4):1351–1358.
11. Pillay D, Bhaskaran K, Jurriaans S, Prins M, Masquelier B, Dabis F, et al. The impact of transmitted drug resistance on the natural history of HIV infection and response to first-line therapy. *AIDS* 2006,20(1):21–28.
12. Oette M, Kaiser R, Däumer M, Petch R, Fätkenheuer G, Carls H, et al. Primary HIV drug resistance and efficacy of first-line antiretroviral therapy guided by resistance testing. *J Acquire Immune Defic Syndr* 2006;41(5):573–581.
13. Australasian Society for HIV Medicine. Australian Commentary on Guidelines for the Use of Antiretroviral Agents in HIV-1-infected Adults and Adolescents [Online]. Available from: http://www.ashm.org.au/images/publications/guidelines/dhhsadult_04_03_09%20-%20final.pdf
14. Shafer RW, Rhee SY, Pillay D, Miller V, Sandstrom P, Schapiro JM, et al. HIV-1 protease and reverse transcriptase mutations for drug resistance surveillance. *AIDS* 2007;21(2):215–223.
15. Guy RJ, Breschkin AM, Keenan CM, Catton MG, Enriquez AM, Hellard ME. Improving HIV surveillance in Victoria: the role of the detuned enzyme immunoassay. *J Acquir Immune Defic Syndr* 2005; 38(4):495–499.
16. Middleton T, Smith D, Larder B, Law M, Birch C. Baseline antiretroviral drug susceptibility influences treatment response in patients receiving saquinavir-enhancing therapy. *HIV Clin Trials* 2001;2(6):445–452.
17. Chibo D, Roth N, Roulet V, Skrabal K, Gooey M, Carolan L, et al. Virological fitness of HIV in patients with resistance to enfuvirtide. *AIDS* 2007;21(14):1974–1977.
18. White KL, Margot NA, Wrin T, Petropoulos CJ, Miller MD, Naeger LK. Molecular mechanisms of resistance to human immunodeficiency virus type 1 with reverse transcriptase mutations K65R and K65R+M184V and their effects on enzyme function and viral replication capacity. *Antimicrob Agents Chemother* 2002;46(11):3437–3446.
19. Wainberg MA. The impact of the M184V substitution on drug resistance and viral fitness. *Expert Rev Anti Infect Ther* 2004;2(1):147–151.
20. Kaye M, Chibo D, Birch C. Phylogenetic investigation of transmission pathways of drug-resistant HIV-1 utilising pol sequences derived from resistance testing. *J Acquir Immune Defic Syndr* 2008;49(1):9–16.
21. Ammaranond P, Cunningham P, Oelrichs R, Suzuki K, Harris C, Leas L, et al. No increase in protease resistance and a decrease in reverse transcriptase resistance mutations in primary HIV-1 infection: 1992–2001. *AIDS* 2003;17(2):264–267.
22. Yerly S, von Wyl V, Ledergerber B, Böni J, Schüpbach J, Bürgisser P, et al. Transmission of HIV-1 drug resistance in Switzerland: a 10-year molecular epidemiology survey. *AIDS* 2007;21(16):2223–2229.

Short reports

A community outbreak of meningococcal serogroup B disease in Western Sydney: the challenges of identification and significance

Andrew Jardine, George Truman, Vicky Sheppeard, Denise Gibbons, Jane Thomas, Kathryn Weston

Abstract

The Communicable Diseases Network Australia guidelines provide information for early clinical and public health management of meningococcal disease, including community outbreaks. While community outbreaks of meningococcal serogroup C infections have been reported, community outbreaks of meningococcal serogroup B infections have not been declared in Australia. Three cases of meningococcal serogroup B disease occurred in 2 adjacent suburbs in western Sydney in Spring 2008. Although the temporal and geographic proximity of these cases fulfilled the criteria for a community outbreak, difficulties in establishing an epidemiological or serosubgroup link, and arbitrary definition of the term 'community' provide challenges for identifying such outbreaks. In addition, the declaration of a community outbreak of meningococcal B infection does not provide guidance for the public health response because a vaccine is not available and community-wide prophylaxis is not recommended. *Commun Dis Intell* 2009;33:221–224.

Keywords: meningococcal disease, outbreak, public health management, Neisseria meningitidis, prevention and control

Introduction

The Communicable Diseases Network Australia (CDNA) guidelines[1] define a community outbreak of meningococcal disease as 'three or more confirmed cases with onset in a 3 month interval, where the available microbiological characterisation of the organisms is the same, *and* incidence is at least 10 per 100,000 total community population in a 3 month interval' (p 52). Community outbreaks tend to be more difficult to define and manage than organisation-based outbreaks for several reasons. Firstly, it is difficult to distinguish a community outbreak from normal fluctuations in disease incidence because of the arbitrary nature of choosing geographic boundaries for defining the population at risk in metropolitan areas. Secondly, if there is no obvious epidemiological link between cases, the public health response may include conducting expensive and logistically challenging mass vaccination clinics. The CDNA guidelines explicitly state that 'Community-wide clearance antibiotics should not be used' (p 57). Finally, other consequent public health activities such as active surveillance and mass media alerts can result in stigmatisation of the defined community.

The average annual notification rate of meningococcal infections (all serogroups) has decreased similarly in both New South Wales and Australia from approximately 3.5 to 1.5 per 100,000 population since the meningococcal serogroup C (MSC) vaccine was added to the National Immunisation Program in 2003. A number of organisation-based outbreaks of MSC were reported in Australia prior to or just after the vaccine was introduced. These were in secondary schools,[2,3,4] a university college,[5] and clustered within a family.[6] Two MSC community clusters have been described previously in western Sydney, the first a community outbreak in Campbelltown[7] and the second associated with a nightclub in Penrith.[8]

Since the introduction of the MSC vaccine, meningococcal serogroup B (MSB) has been most commonly identified in laboratory confirmed cases both in New South Wales and nationally, accounting for 68% and 80% of notifications respectively in 2006.[9] However, MSB only causes sporadic cases in Australia.[10] Isaac-Toua and colleagues reported a number of community-based cases of MSB within a 3 month period in the Australian Capital Territory in early 2004.[11] However, they did not have sufficient numbers to reach the criterion of 10 cases per 100,000 population and therefore did not declare a community outbreak of MSB.

Methods and results

Description of the outbreak and public health investigation

Three cases of MSB disease occurred in 2 adjacent suburbs in the Penrith Local Government Area (LGA) in western Sydney with a combined population of approximately 28,000.[12] This equated to

10.7 cases per 100,000 population across the 2 suburbs within 1 month, which met the CDNA definition of a community outbreak.[1] A summary of critical events in the outbreak is shown in the Figure.

Case 1 (female, aged 9) had complained of a sore stomach and headache during the days prior to presenting to a general practitioner on 26 September 2008. By this time further symptoms had developed (rash, numb legs) which suggested meningococcal disease. The patient was immediately treated with intramuscular benzyl penicillin and taken to hospital. Although a blood culture was attempted, there was no bacterial growth.

Case 2 (female, aged 2), from the same suburb as case 1, became ill on 28 September 2008 and progressively worsened over the next few days until she was admitted to hospital with suspected gastroenteritis on 29 September 2008. The diagnosis was changed to meningococcal infection the following day as classic symptoms (stiff neck and photophobia) became apparent. A blood culture was requested and gram negative diplococci were identified.

Case 3 (female, aged 13), from a suburb adjacent to cases 1 and 2, saw a general practitioner on 18 October 2008 with fever and vomiting and was diagnosed with viral gastroenteritis. However the patient deteriorated rapidly and was admitted to the Intensive Care Unit of a local hospital a few hours later. A lumbar puncture was carried out and gram negative diplococci were identified in the cerebrospinal fluid.

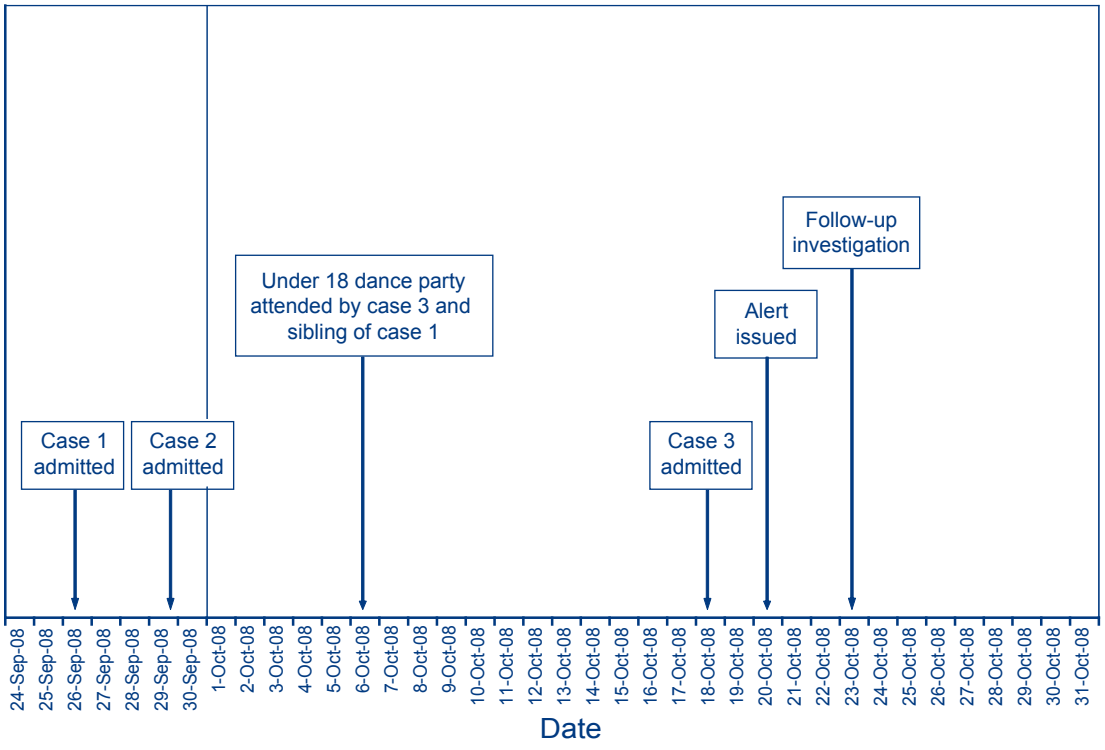
All cases fully recovered after a period of seven to 10 days in hospital with treatment in accordance with the CDNA guidelines.

Routine case histories were taken at the time of notification to the public health unit using the NSW Health Department Meningococcal Disease questionnaire,[13] noting in particular any special functions or extra-curricular school activities. Contacts of cases were identified and managed in accordance with Australian guidelines, including the provision of information and clearance antibiotics.

All cases were fully vaccinated for age against MSC, confirmed by the Australian Childhood Immunisation Register records for cases 1 and 2, and school program vaccination records for case 3. Case 1 had only attended school on 1 day in the week before symptom onset, due to absence with earlier symptoms of stomach ache and headache. Case 2 was too young to attend school and was not in child care. Case 3 attended school in the week prior to symptom onset, but not the same school as case 1.

Polymerase chain reaction testing of blood (n=1) or cerebrospinal fluid (n=2) samples from each case confirmed *Neisseria meningitis* serogroup B in all

Figure: Time line of investigation into meningococcal serogroup B cluster in western Sydney



3 cases. The isolate on case 2 was serosubtyped as B:4:P1.4. Further typing on cases 1 and 3 was not possible as isolates were not available.

The community outbreak was declared following the 3rd case and on 20 October 2008 a public health alert was issued to all general practitioners, emergency departments, paediatricians and infectious diseases specialists in the Sydney West Area Health Service. The alert outlined the unusual increase in cases of invasive meningococcal disease and requested that all suspected cases be notified to the public health unit as soon as possible following the implementation of antibiotics and initial investigations.

A follow-up epidemiological investigation was instigated to identify common links or exposures between the cases. The primary carer of each case was re-interviewed on 23 October 2008 to seek additional information to that collected at the time the case history was taken. No direct connections were apparent and only 2 tenuous associations could be identified. Cases 2 and 3 had close contacts in the building and construction industry, but no associations between these contacts could be identified. A sibling of case 1 also attended the same under-18 dance party on 6 October 2008 as case 3, however the sibling had already completed a course of clearance antibiotics at the time of the party.

Discussion

A community outbreak of MSB occurred in the Penrith LGA of western Sydney with 10.7 cases per 100,000 population within a month. However, we were unable to identify any common exposures or direct links between the cases. The finding that two of the 3 cases had close contacts in the construction industry is not surprising as this is a common occupation in the region.[12] The dance party which occurred on 6 October 2008, after the sibling of case 1 had received clearance antibiotics and over a week before case 3 developed symptoms, excludes the possibility of the sibling transmitting MSB to case 3. However, the event provides a possible opportunity for transmission of the meningococcus from a contact of the sibling to case 3 and earlier to case 1. Further exploration of this scenario was not possible due to the unavailability of data.

In addition to failing to establish an epidemiological link between the cases, a major limitation of this investigation was that serosubtyping of the MSB isolates for two of the cases could not be carried out as one could not be cultured and the other isolate was not available for processing in the typing laboratory. In the absence of an epidemiological link, the establishment of a biological link would assist interpretation. If the cases had different serosubtypes, then they would be sporadic cases whereas cases with the same serosubtype could provide evidence for a cluster. Whether the cases were sporadic or part of a cluster remains undetermined.

The serosubtype identified from case 2 (B:4:P1.4) is commonly identified in sporadic cases in the eastern states of Australia with 14 cases in 2006 and 12 in 2007.[9] The same serosubtype has also been implicated in MSB outbreaks in New Zealand and elsewhere for many years.[14] It will be important to obtain isolates for serosubtyping in all local cases of MSB to monitor the occurrence of this strain in New South Wales.

Although the 3 cases described here were linked in time and place, the public health investigations highlighted the difficulty in defining a geographic boundary for a community outbreak in a suburban area. The 2 suburbs in question are contiguous with other suburbs without clear natural or man-made boundaries. We chose to calculate incidence rates based on the population of the 2 suburbs of residence, however a lower incidence rate would result if other geographical boundaries (such as local government area or all adjacent suburbs) were selected. Nevertheless, the choice of the 2 suburbs of residence as the population denominator makes epidemiological sense in that the 2 adjacent suburbs had a common public bus service, and one of the suburbs acted as a hub with a suburban shopping centre, local library and other services, and large sporting fields.

In addition to the challenges in identifying the presence of a community outbreak, it is difficult to determine the significance of the outbreak when MSB is involved. Mass or risk-group-specific vaccination programs are not possible because of a lack of a suitable vaccine in Australia. In addition, clearance antibiotics are not recommended for community-wide application,[1] and therefore the possible public health response is limited.

Despite the success of the MSC vaccine and subsequent reduction in numbers of cases caused by that serotype, MSB rates have remained relatively constant in the community over the last 10 years. This study demonstrates that community outbreaks of MSB as well as sporadic cases can occur, but identification of the source or location of the source of a community outbreak of MSB can be difficult, as can the planning and implementation of an adequate public health response.

Acknowledgements

The Master of Applied Epidemiology Program is funded by the Australian Government Department of Health and Ageing.

Author details

Andrew Jardine, Master of Applied Epidemiology Scholar[1]
George Truman, Public Health Epidemiologist[2]
Vicky Sheppeard, Manager[2]
Denise Gibbons, Immunisation and Surveillance Officer[2]
Jane Thomas, Immunisation Co-ordinator[2]
Kathryn Weston, Senior Surveillance Officer[2]

1. National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital
2. Communicable Diseases and Immunisation Team, Sydney West Area Health Service, Penrith, New South Wales

Corresponding author: Mr George Truman, Public Health Epidemiologist, Communicable Diseases and Immunisation Team, Sydney West Area Health Service, PO Box 63, PENRITH NSW 2751. Telephone +61 2 4734 2022. Facsimile: +61 2 4734 3300. Email: trumang@wahs.nsw.gov.au

References

1. Communicable Diseases Network Australia. Guidelines for the early clinical and public health management of meningococcal disease in Australia. Canberra: Commonwealth of Australia; 2007.
2. Davison RP, Lovegrove DR, Selvey LA, Smith HV. Using the national guidelines to manage a meningococcal group C outbreak in a Brisbane boarding school—some discretionary judgements are needed. *Commun Dis Intell* 2003;27(4):520–523.
3. Miles TA, Lewis PR, Cook L, Bruderlin KI. An outbreak of meningococcal disease in a secondary school—implications for public health practice. *Commun Dis Intell* 2004;28(3):345–347.
4. Robinson P, Taylor K, Tallis G, Carnie J, Rouch G, Griffith J, et al. An outbreak of serogroup C meningococcal disease associated with a secondary school. *Commun Dis Intell* 2001;25(3):121–125.
5. Ferson M, Young M, Hansen G, Post J, Tapsall J, Shultz T, et al. Unusual cluster of mild invasive serogroup C meningococcal infection in a university college. *Commun Dis Intell* 1999;23(10):261–264.
6. Young MK, McCall BJ, Smith HV, Looke D. A family cluster of serogroup C meningococcal disease. *Commun Dis Intell* 2004;28(4):496–498.
7. Chant K, Stewart G, Brown J, Munro R, Toouli G, Kociuba K. A cluster of meningococcal cases in Campbelltown. *NSW Public Health Bulletin* 1992;3:93–94.
8. Jelfs J, Jalaludin B, Munro R, Patel M, Kerr M, Daley D, et al. A cluster of meningococcal disease in western Sydney, Australia initially associated with a nightclub. *Epidemiol Infect* 1998;120(3):263–270.
9. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2007. *Commun Dis Intell* 2008;32(3):299–307.
10. Jelfs J, Munro R. Epidemiology of meningococcal disease in Australia. *J Paediatr Child Health* 2001;37(5):S3–S6.
11. Isaac-Toua G, Guest C, Hiam R, Passaris I. Public health management of increased incidence of meningococcal disease in the Australian Capital Territory: 2003 to 2004. *Commun Dis Intell* 2007;31(1):112–118.
12. Australian Bureau of Statistics. 2006 census of population and housing: census data by location. Available from: http://www.censusdata.abs.gov.au/ABSNavigation/prenav/LocationSearch Accessed on 19 November 2008.
13. NSW Department of Health. Meningococcal disease questionnaire form. Available from: http://www.health.nsw.gov.au/resources/publichealth/infectious/diseases/Menigococcal/imd_core_data_form.pdf Accessed September 2008.
14. Baker MG, Martin DR, Kieft CEM, Lennon D. A 10-year serogroup B meningococcal disease epidemic in New Zealand: Descriptive epidemiology, 1991–2000. *J Paediatr Child Health* 2001;37(5):S13–S19.

Measles status in Australia, and outbreaks in the first quarter of 2009

Nicolee Martin, A Ruth Foxwell

Introduction

Measles is an acute, highly communicable viral disease spread by respiratory secretions that may lead to serious complications such as diarrhoea, otitis media, pneumonia or encephalitis.[1] In the past, measles infection was a common childhood illness but as a result of national immunisation campaigns is now rare in Australia.[2] Measles remains endemic wherever vaccination coverage is low and is one of the leading causes of vaccine preventable death in children worldwide.[3]

Historically, measles epidemics occur worldwide every 1 to 5 years with vaccination decreasing the number of cases in an outbreak. However, new birth cohorts or immigration can result in subsequent new epidemics.[4] The critical requirements for epidemics include a community size of approximately 250,000 to 500,000 and approximately 15 to 20 secondary cases arising from every index case.[5] While the measles virus can be divided into a number of genetically different types, and there is molecular evolutionary change, so far this has not resulted in high levels of genetic variation[4] and therefore, all genotypes can be neutralised by antibodies produced from the one strain.[6]

Genotypes are divided by the genetic differences in one small part of the carboxy-terminus of the nucleocapsid (N) gene and the whole length of the haemagglutinin (H) gene (one of the proteins responsible for binding the virus to the host cell).[7] Differences in nucleotide sequence by 2.5% in the N or 2.0% in the H gene will result in the classification of a new genotype.[7] The World Health Organization (WHO) classifies the measles viruses on the basis of clades (letters) and subtypes (numbers) with approximately 8 clades and 23 sub-types. While some of the genotypes are widespread, there are enough that are geographically distinct to make them a useful epidemiologic tool. Communities with lower levels of vaccination coverage and frequent outbreaks have been found to have fewer circulating strains at any one time. However, where endemicity is constant, single genotypes with several lineages tend to co-exist.[6] In Australia, since the early to mid 1990s no one genotype has appeared repeatedly, indicating the absence of an endemic circulating strain.[8]

This report reflects on the current status of measles elimination in Australia and examines a number of measles outbreaks in the 1st quarter of 2009.

Measles elimination

In 2003, the WHO Regional Office for the Western Pacific (WPRO) nominated 2012 as the target date for measles elimination, defined as the absence of transmission of endemic measles virus.[9] It should be noted that even if measles elimination is achieved in a sizeable geographic area such as the WPRO Region, high levels of vaccination would continue to be needed in order to prevent re-introduction of the virus from other areas. Measles cases will continue to occur until measles is eradicated, defined as interruption of measles transmission globally, after which vaccination could be ceased.[10] While global measles eradication is potentially possible, it is difficult to achieve because of the highly infectious nature of the virus and the susceptibility of infants during the period of time between waning maternal antibody resistance and their 1st routine dose of a measles containing vaccine at 12 months of age.[11]

WPRO indicators to track progress towards elimination include: a very low incidence of measles of less than 1 case per million population, not including imported cases; high quality case-based measles surveillance, which includes national reporting of non-measles suspected cases; high population immunity demonstrated by a very high vaccination coverage, defined as greater than or equal to 95% of the population receiving 2 doses of the measles-mumps-rubella (MMR) vaccine; greater than or equal to 80% of outbreaks having transmission of less than 10 cases; and the absence of an endemic measles virus genotype.[12,13]

In 1998, the National Measles Surveillance Strategy (NMSS) was developed in order to prepare for measles elimination in Australia. The main objectives of the measles elimination initiative were to cease measles related morbidity and mortality by interrupting indigenous transmission of measles and to prevent the re-introduction of measles by maintaining uniformly low levels of population susceptibility. High vaccination coverage of greater than 95% for each new birth cohort and low susceptibility levels, particularly in closed environments such as schools,

where rates of contact are high, were needed to achieve these objectives. In particular, the NMSS highlighted the need for coverage to be consistent across the population in order to prevent 'pockets of susceptible persons' from sustaining endemic measles transmission.[14] A widespread school based measles vaccination campaign was started in 1998 and was followed by a change in the routine vaccination schedule which moved the 2nd dose of MMR from between 10 and 16 years to 4 years of age. A second catch up dose for primary school aged children was provided for those between 5 and 12 years of age and born between 1986 and 2003.[14] This strategy significantly reduced the incidence of measles in Australia. The continued high MMR 2-dose coverage under the National Immunisation Program has ensured a high level of population immunity and enabled Australia to move into a measles elimination phase.[15]

Population immunity

Vaccination coverage targets for Australia's measles elimination strategy identified in the NMSS, were 95% for children with at least 1 dose, and 90% with 2 doses of measles containing vaccine (MCV) at school entry by 2001.[14] The latest figures from the 2007 annual immunisation coverage report reveal that 94.1% of 2 year olds (born in 2005) were fully immunised (i.e. received 1 dose of a MCV) and 89.1% of 5 year olds (born in 2001) were fully immunised (i.e. received 2 doses of a MCV).[16] These figures may underestimate the actual coverage of 1 dose of a MCV by 3%–5% and 2 doses by 5%–10% indicating that the NMSS targets may have been met.[8] In addition, incomplete reporting of immunisation encounters and the small proportion of people for which vaccination is medically contraindicated makes it difficult for the coverage estimates to exceed 95%.[15]

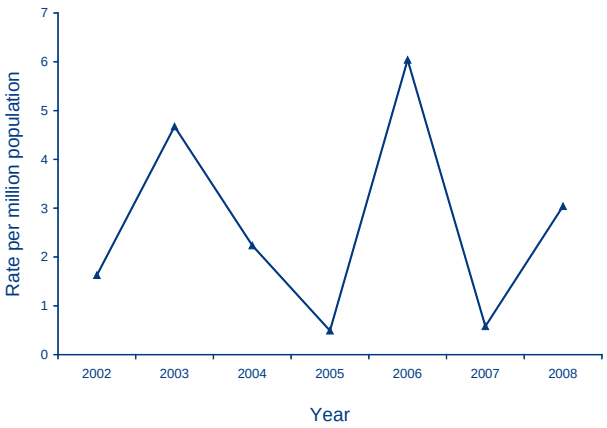
The 2007 annual immunisation coverage report states that up to 3% of Australian parents are not immunising their children for religious or philosophical reasons. However, these levels can be much higher in local areas of some jurisdictions, particularly coastal areas of south-east Queensland, northern New South Wales, the Adelaide Hills and the south-west of Western Australia.[16]

The 2002 measles serosurvey conducted by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) estimated that 94% of the Australian population was immune to measles. However, it also identified susceptible age groups with only 65% of 1 year olds, 88.5% of 2 to 4 year olds, and 87% of 20–24 year olds demonstrating measles immunity at this time.[8] Infants are at high risk until they have had their 1st vaccination at 12 months of age. Young adults born between 1968 and 1982 are a particularly susceptible cohort as many missed being vaccinated as infants when coverage was still low and the risk of exposure and subsequent development of natural immunity was declining. Of this group, those born between 1978 and 1982 (or those who were 20–24 years of age at the time of the 2002 serosurvey) have been identified as at increased risk because during their childhood a 2nd dose was not yet recommended and they were not targeted as part of the catch-up campaign in 1998.[2,9,15] An evaluation of age-specific measles susceptibility in Australia, undertaken as part of a wider evaluation, which included 17 European countries, also identified adolescents and young adults between 10 and 39 years of age as being susceptible to measles outbreaks.[17] This is not the case for those born before 1968, 97% of whom demonstrated measles immunity in the 2002 serosurvey.[8]

Incidence

Measles notification rates in Australia have been progressively decreasing since 1994. Since 2002, measles incidence in Australia has ranged between 0.5 and 6 cases per million population[18] with the WHO target for measles elimination of less than 1 case per million population reached in both 2005 and 2007. For those years in which rates exceeded the WHO target, case-based investigation, including analysis of genotypes, indicate that most were either imported or linked to an imported case.[8] For example, in 2006 there was a large multi-state outbreak that was associated with a travelling spiritual group from a country where measles is endemic.[19] A high proportion of those who attended tour meetings were opposed to vaccination.[8] In 2008, there was another import associated outbreak in New South Wales.[20] Both of these outbreaks led to rates above the WHO target (Figure 1).

Figure 1: Measles notification rates per 1 million population, Australia, 2002 to 2008



High quality surveillance

In Australia, a measles case definition was formally adopted by all states and territories in 2004 as part of the revision and development of standard surveillance case definitions for all nationally notifiable diseases.[21] Public health agencies in each jurisdiction are responsible for the follow-up of all measles and suspected measles cases. While information on measles suspected cases are not collected at the national level, and therefore do not meet this WHO elimination indicator,[9] it is collected and notified on suspicion at the jurisdictional level. National measles guidelines for public health units outline requirements that include the recording of all suspected, probable and confirmed measles cases on the notifiable diseases data base in each jurisdiction.[22] Confirmed measles cases are all notified to the National Notifiable Diseases Surveillance System (NNDSS). Researchers at NCIRS concluded in a paper published in November 2008 that despite not reporting non-measles suspected cases at the national level, Australia has satisfied multiple criteria that justify the formal declaration of measles elimination.[8]

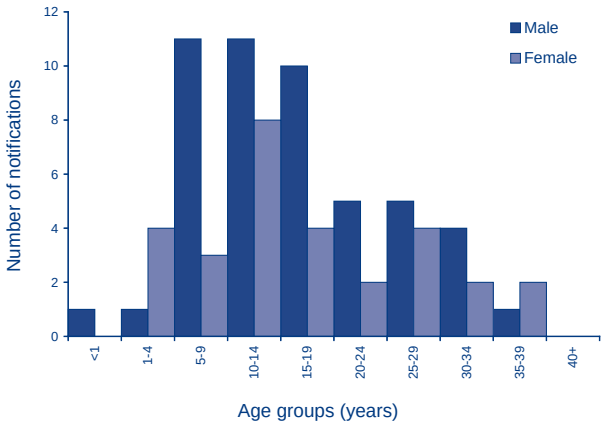
Measles cases in Australia, 1st quarter 2009

Measles outbreaks still occur in Australia when susceptible populations are exposed to the measles virus. These outbreaks are usually associated with a case imported from overseas, as has occurred in the 1st quarter of 2009 when separate importations of measles led to large outbreaks in Queensland and Victoria. Based on information provided by the Victorian Department of Human Services and Queensland Health and to the NNDSS as at 31 March 2009, both Queensland and Victoria had one and 2 outbreaks respectively in which local transmission exceeded 10 cases. However, approximately 84% (16/19) of outbreaks in this quarter had less than 10 cases, which meets the WHO elimination criteria of greater than or equal to 80% of outbreaks having transmission of less than 10 cases.

Between 1 January and 31 March 2009, 78 cases of measles were reported in Australia, 5.5 times the quarterly 5-year rolling mean (n=14.2). This compared with 65 cases notified for all of 2008. The majority of cases were from Victoria (n=33) and Queensland (n=31), with lower numbers reported from New South Wales (n=7), Tasmania (n=2), Western Australia (n=2), South Australia (n=1), the Australian Capital Territory (n=1) and the Northern Territory (n=1). Of the 78 cases, 49 (63%) were male and 29 (37%) were female with ages ranging from less than 1 year to 38 years (Figure 2).

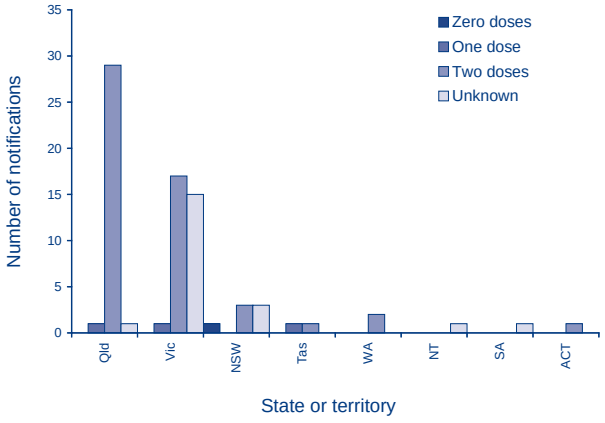
The number of vaccine doses was known for 57 of the 78 cases, of which none had received 2 doses of

Figure 2: Notifications of measles, Australia, 1 January to 31 March 2009, by age and sex



a MCV, four (7%) had received 1 dose and 53 (93%) had received no doses: the remaining 21 cases were of unknown vaccination status (Figure 3). Of the 4 cases who had received 1 dose of a MCV, two were adults born in 1971 and 1983 respectively and as such part of the susceptible cohort mentioned above; and one was a 3-year-old child, not yet due their second dose.

Figure 3: Notifications of measles, Australia, 1 January to 31 March 2009, by state or territory and number of vaccine doses



Recent travel, resulting in the acquisition of measles overseas, accounted for 22% (17/78) of the cases. Importation of cases occurred from India (n=6), Vietnam (n=4), Thailand (n=2) and one each from the Philippines, France, the United States of America, New Zealand (related to an outbreak cluster where the index case was imported from Vietnam) and Iran. Of the 17 imported cases, 13 (76%) were young adults between 17 and 34 years of age. Sixty-one of the cases were locally acquired resulting in an annualised rate of 1.14 cases per 100,000 popula-

tion (or 11.4 cases per million population) in 2009 compared with 0.23 cases per 100,000 population (or 2.3 cases per million) for all of 2008.

While measles cases were reported from all states and territories during the 1st quarter of 2009, imported cases only led to locally acquired cases in Queensland, Victoria and New South Wales with only Queensland and Victoria reporting outbreaks of greater than 10 cases. Importantly, isolated imported cases into Western Australia, South Australia, the Australian Capital Territory and the Northern Territory did not result in any ongoing transmission in those states and territories.

Outbreaks of less than 10 cases during the 1st quarter of 2009

A Tasmanian case imported from India and identified as genotype D4 was epidemiologically linked to 2 locally acquired cases, both 31-year-old males, one each from Victoria and Queensland. The measles virus in this cluster of three was 6 base pairs different from the D4 identified in the larger Queensland outbreak described below. Of 4 imported cases in New South Wales, three resulted in local secondary transmission to 1 case as at 31 March 2009.

Outbreaks of more than 10 cases during the first quarter of 2009

Queensland had a total of 31 cases of measles notified in the 1st quarter of 2009. Of these, 25 cases in a Sunshine Coast high school were linked to an imported case from India diagnosed on 12 January 2009, and were of genotype D4. None of the 25 cases were vaccinated at the time of exposure. In this case an outbreak occurred amongst a cohort of unvaccinated children despite the vaccination coverage in the overall geographical area being estimated at greater than 90% (assessed at 24 months of age for the birth cohort 1/10/05 to 30/9/06)(Map). This highlights the risk of imported disease resulting in a localised outbreak with the potential for sustained transmission when uniform MMR coverage is not achieved. An additional 6 cases were notified from Queensland during this period, of which three were imported and the remaining three were locally acquired, including the case linked to the imported Tasmanian case described above.

In Victoria, 4 outbreaks of measles were identified and were linked to 4 separate imported cases. The 1st Victorian outbreak involved 11 cases and began with an imported case from Iran diagnosed on 2 January 2009 and resulted in 3 generations of locally acquired transmission. The measles virus identified was genotype H1. Five cases were epidemiologically linked to the index case from Iran however, an additional two and 3 separately linked cases, all H1, could not be definitively linked to each other or the imported case. Of the H1 cases, six were unvaccinated, four were of unknown vaccination status and 1 case was partially vaccinated with 1 dose of a MCV.

A 2nd cluster of 20 cases, identified as genotype D8, began in Victoria with an imported case from India diagnosed on 5 February 2009. This outbreak resulted in 5 generations of local transmission by the end of March. In this cluster, 9 cases were unvaccinated and 11 were of unknown vaccination status.

The additional 2 clusters included 1 imported case in an 11-month-old baby (too young for vaccination) from Thailand, identified as genotype D9, which had no ongoing local transmission, and a 31-year-old man epidemiologically linked to the imported case in Tasmania described above.

A timeline of measles notifications by state and outbreak is presented in Figure 4 for the main Queensland and Victorian outbreaks. A clear chain of transmission in the 3 main clusters (the D4 school based outbreak in Queensland, H1 and D8 clusters in Victoria) can be seen with exposure periods, onset date and infectious periods outlined for each case based on information provided to NNDSS.

Public health measures by the relevant jurisdictions, including media releases, contact tracing, case isolation and offering prophylactic vaccination and normal human immunoglobulin for contacts with confirmed or suspected measles where indicated, minimised the spread of local transmission in the affected areas.

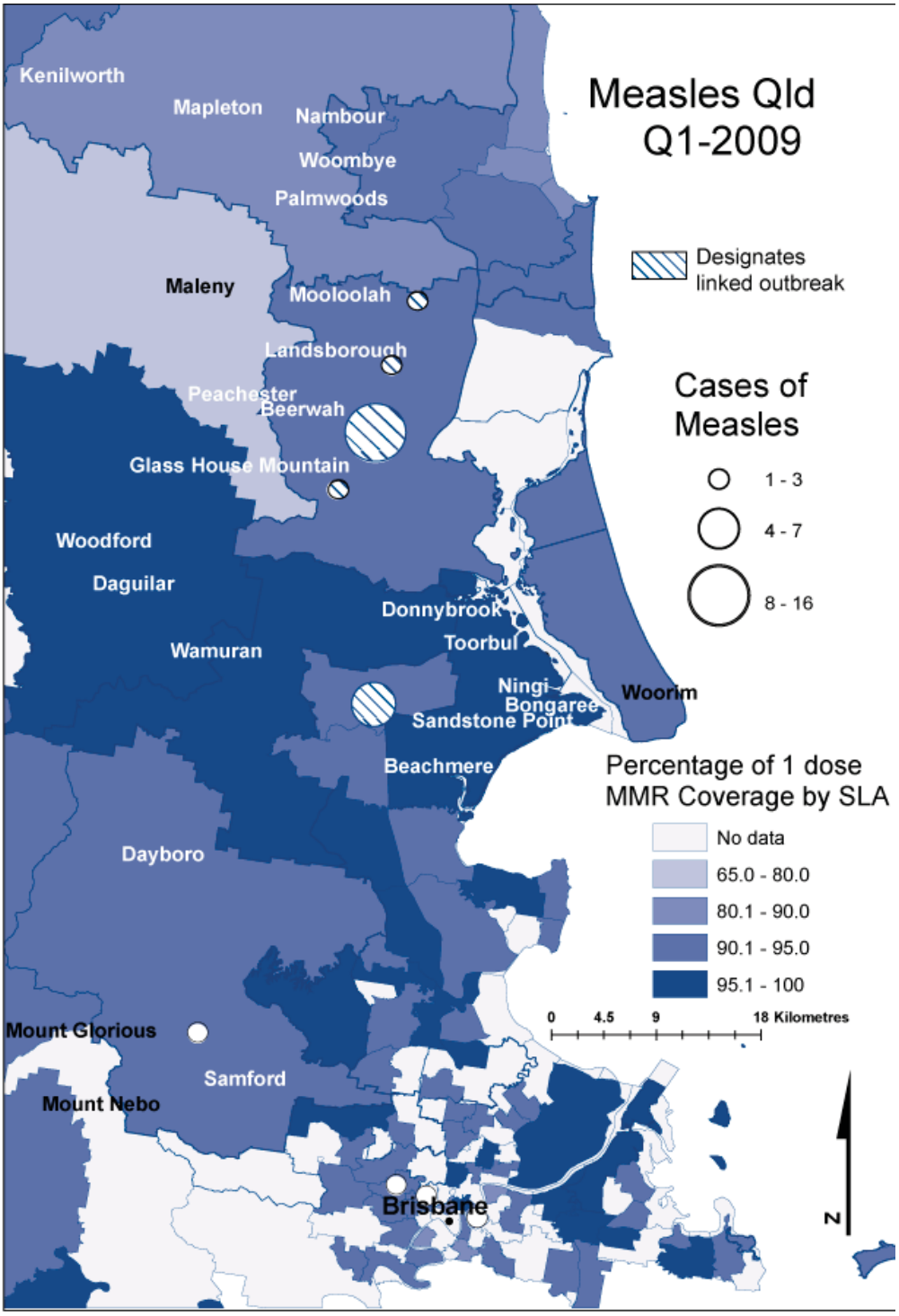
Conclusion

The rapid public health response in each jurisdiction to 17 separate measles importations during the 1st quarter of 2009 has prevented sustained measles virus transmission and highlights the capability of Australia's disease surveillance systems.

Adolescents and young adults have been identified as a susceptible cohort with young adult travellers a major source of imported infection during the 1st quarter of 2009. It has been suggested that prevention of outbreaks in this susceptible age group may require strategies such as one-off targeted mass vaccination campaigns, requirements for up-to-date vaccination records for entry to further education or overseas travel.[9,15,17]

It will also continue to be critical during this elimination phase of measles control that molecular analysis occurs in routine and outbreak investigations to identify the genotype of each new cluster and the

Map: Percentage of 1 dose measles-mumps-rubella coverage* assessed at 24 months of age (birth cohort 1/10/05 to 30/9/06), by Queensland Statistical Local Area and postcode of residence



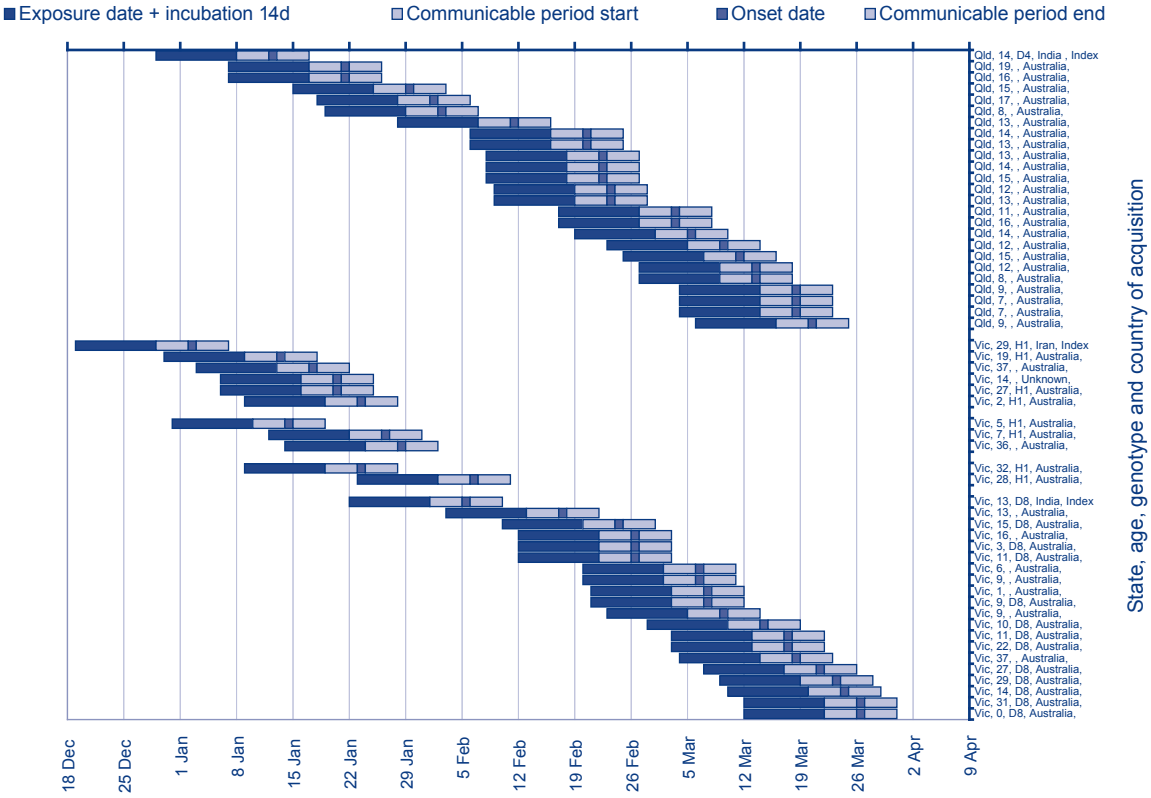
* Some Statistical Local Area 100% coverage figures are imprecise estimates as they are based on small numbers of children.

Source: B. Hull, Australian Centre for Immunisation Research unpublished data, July 2009.

origin of the measles virus in order to demonstrate the absence of sustained transmission of 1 genotype in Australia.[8]

In summary, although evidence suggests that endemic measles has been eliminated from Australia with the absence of an endemic circulating genotype, we will continue to be at risk of outbreaks among susceptible populations associated with imported cases from time to time. Ongoing efforts to maintain uniformly high levels of immunisation coverage across all regions of Australia are therefore

Figure 4: Timeline of measles outbreaks greater than 10 cases, National Notifiable Diseases Surveillance System, Victoria and Queensland (n=56), 1 January to 31 March 2009, by infectious period



required. Enhanced surveillance to identify every new case of measles and track genotypes continues to be essential in order to move Australia from the current phase of elimination to measles eradication by 2012.

Acknowledgements

The authors would like to thank the following people from the Department of Health and Ageing: Dougald Knuckey for his time and skill in producing the Queensland MMR coverage map; Kate Robinson for her comments and editorial input; and Conan Liu for comments and demonstrating the use of the modified Gantt chart. In addition we would like to thank Brynley Hull from NCIRS, Lisa Hall from Queensland Health and the following staff from the Victorian Department of Human Services: James Fielding, Margaret Hill, Pauline Lynch, Marion Moloney, Anne Murphy and Kleete Simpson and from the Victorian Infectious Diseases Reference Laboratory: Jennie Leydon, Thomas Tran and staff from the Serology and Viral Identification laboratories for their invaluable input.

The Master of Applied Epidemiology Program is funded by the Australian Government Department of Health and Ageing.

Author details

Nicolee Martin[1]

A Ruth Foxwell[1,2]

1. Vaccine Preventable Disease Surveillance, Department of Health and Ageing, Canberra, Australian Capital Territory
2. Master of Applied Epidemiology scholar, National Centre for Epidemiology and Public Health, Australian National University, Canberra, Australian Capital Territory

References

1. Heymann DL. Ed. *Control of Communicable Diseases Manual* 19th Edn. Washington DC: American Public Health Association; 2008.
2. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 9th Edn. Canberra: Commonwealth of Australia; 2008. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/5335A7AB925D3E39CA25742100194409/$File/handbook-9.pdf
3. The Measles Initiative [online]. Available from: http://www.measlesinitiative.org/index3.asp Accessed 18 May 2009.
4. Pomeroy LW, Bjornstad ON, Holmes EC. The evolutionary and epidemiological dynamics of the paramyxoviridae. *J Mol Evol* 2008;66:98–106.
5. Griffin DE. Measles virus. In Fields BN, Knipe DM, Howley PM, Eds. *Virology*. Philidelphia: Lippincott Williams and Wilkons; 2001. p.1401–1441.

6. Riddell MA, Rota JS, Rota PA. Review of the temporal and geographical distribution of measles virus genotypes in the prevaccine and postvaccine eras. *J Virology.*2005;2:87–95.
7. World Health Organization. Nomenclature for describing the genetic characteristics of wild-type measles virus (update, Part 1). *Wkly Epidemiol Rec* 2001;76:242–247.
8. Heywood AE, Gidding HF, Riddell MA, McIntyre PB, MacIntyre CR, Kelly HA. Elimination of endemic measles transmission in Australia. *Bull World Health Organ* 2009;87:64–71.
9. World Health Organization. Progress towards the 2012 measles elimination goal in WHO's Western Pacific Region, 1990–2008. *Wkly Epidemiol Rec* 2009;27(84):269–280.
10. Measles eradication: Recommendations from a meeting cosponsored by the World Health Organization, the Pan American Health Organization, and CDC. *MMWR Morbid Mortal Wkly Rep* 1997;46(RR11):1-20.
11. Fenner, F. Candidate viral diseases for elimination or eradication. *MMWR Morbid Mortal Wkly Rep* 1999;48(SU01):86-90.
12. World Health Organization Regional Office for the Western Pacific. Monitoring measles surveillance and progress towards measles elimination. *Measles Bulletin*. 2007;13:1–6. Available from: http://www.wpro.who.int/NR/rdonlyres/7BE6353C-7D82-4368-A300-57-DB3F38148D/0/MeasBulletinIssue13.pdf
13. World Health Organization Regional Office for the Western Pacific. Field guidelines for measles elimination. Geneva: 2004. Available from: http://www.wpro.who.int/publications/pub_929061126x.htm
14. Heath T, Burgess M, McIntyre P, Catton M. The national measles surveillance strategy. *Commun Dis Intell*1999;23:41–50.
15. Gidding H, Wood J, MacIntyre CR, Kelly H, Lambert SB, Gilbert GL, et al. Sustained measles elimination in Australia and priorities for long-term maintenance. *Vaccine* 2007;25:3574–3580.
16. Hull B, Deeks SL, Menzies R, McIntyre P. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Annual Immunisation Coverage Report, 2007. *Commun Dis Intell* 2009;32 (2): 169–186.
17. Andrews N, Tischer A, Siedler A, Pebody RG, Barbara C, Cotter S, et al. Towards elimination: measles susceptibility in Australia and 17 European countries. *Bull World Health Organ* 2008;86(3):197–204.
18. Department of Health and Ageing. National Notifiable Disease Surveillance System. Available from www.health.gov.au/nndss Accessed 14 April 2009.
19. Sheppeard V, Forssman B, Ferson MJ, Moreira C, Campbell-Lloyd S, Dwyer DE, McAnulty J. Vaccine failures and vaccine effectiveness in children during measles outbreaks in New South Wales, March-May 2006. *Commun Dis Intell*. 2009;33(1):21-26.
20. Australian Government Department of Health and Ageing. Communicable Diseases Surveillance. Highlights for 1st quarter, 2008. *Commun Dis Intell* 2008;32(2):274–276.
21. National Surveillance Case Definitions for the Australian National Notifiable Diseases Surveillance System Canberra: Department of Health and Ageing. Available from: http://www.health.gov.au/casedefinitions Accessed on 27 July 2009.
22. Australian Government Department of Health and Ageing. Series of National Guidelines. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cdnasongs.htm Accessed on 18 August 2009.

Quarterly reports

OzFoodNet quarterly report, 1 January to 31 March 2009

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food occurring in Australia from 1 January to 31 March 2009.

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 first quarter of 2009, OzFoodNet sites reported 322 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 4,520 people, of whom 158 were hospitalised. There were 10 deaths reported during these outbreaks. The majority (65%, n=209) of outbreaks were due to person-to-person transmission (Table 1).

Table 1: Mode of transmission for outbreaks of gastrointestinal illness, OzFoodNet sites, 1 January to 31 March 2009

Transmission mode	Number of outbreaks	Percentage of total
Foodborne	43	14
Person-to-person	209	65
Recreational water	9	3
Unknown – *Salmonella* cluster	4	1
Unknown – other pathogen cluster	10	3
Unknown	47	15
Total	322	100

Foodborne disease outbreaks

There were 43 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 495 people and resulted in 70 hospitalisations. There were no reported deaths during these outbreaks. This compares with 31 foodborne outbreaks for the 1st quarter of 2008 and 29 outbreaks for the 4th quarter of 2008.

Salmonella was responsible for 27 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype (79%, n=22). There were 15 outbreaks due to *S.* Typhimurium phage type 170, two due to *S.* Typhimurium 44 and one each due to *S.* Typhimurium 135a, U302 and 197. There were 2 outbreaks of *S.* Typhimurium where phage typing was not reported. There was 1 outbreak each due to *S.* Montevideo, *S.* Chester, *S.* Saintpaul, *S.* Singapore and *S.* Virchow.

Of the remaining 16 outbreaks, five were due to foodborne toxins, including 2 *Clostridium perfringens* outbreaks, 2 outbreaks of fish-associated histamine poisoning and 1 ciguatera fish poisoning outbreak. There were 2 outbreaks due to norovirus and 1 outbreak due to *Campylobacter* infection. The remaining 8 outbreaks were of unknown aetiology.

Fifteen outbreaks (35%) reported in this quarter were associated with food prepared in restaurants, 8 (19%) associated with aged care facilities, 6 (14%) private residences, 4 (9%) commercial caterers, and 3 (7%) takeaway premises. Individual outbreaks were associated with food prepared at a bakery, camp, from primary produce, childcare centre, other institution, nationally franchised fast food restaurant, and a school.

To investigate these outbreaks, sites conducted 5 cohort studies, 2 case control studies, and collected descriptive case series data for 36 investigations. As evidence for the implicated vehicle, investigators collected microbiological evidence in 1 outbreak, analytical epidemiological evidence in 3 outbreaks,

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2009 (n=43)

State or territory	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicle
ACT	February	Private residence	Histamine poisoning	2	1	D	Tuna steak
	February	Restaurant	*S.* Typhimurium 170	20	0	A	Tiramisu dessert
	March	Private residence	*S.* Typhimurium 170	5	0	D	Zucchini bake
NSW	January	Aged care facility	*S.* Typhimurium 170	4	0	D	Unknown
	January	Bakery	*S.* Typhimurium 170	9	1	D	Chocolate, custard and cream cakes
	January	Institution – other	*S.* Typhimurium 170	40	5	A	Hollandaise sauce
	January	National franchised fast food	*S.* Typhimurium 170	3	1	D	Unknown
	January	Private residence	*S.* Typhimurium 170	68	14	AM	Home made raw egg mayonnaise
	January	Private residence	*S.* Typhimurium 170	4	1	D	Unknown
	January	Restaurant	Unknown	2	0	D	Unknown
	January	Restaurant	*S.* Chester	13	2	AM	Chilli sauce
	January	Restaurant	Histamine poisoning	2	1	D	Tinned anchovies imported from Morocco
	January	Takeaway	*S.* Typhimurium 170	2	1	D	Suspected chicken salad roll with homemade mayonnaise
	February	Aged care facility	*C. perfringens*	25	0	D	Suspected vegetable gravy
	February	Takeaway	Unknown	6	6	D	Unknown
	February	Restaurant	Unknown	5	0	D	Unknown
	February	Takeaway	*S.* Typhimurium	3	1	D	Unknown
	February	School	*S.* Typhimurium 170	37	0	D	Unknown
	March	Aged care facility	*S.* Typhimurium 170	7	2	D	Unknown
	March	Commercial caterer	*S.* Montevideo	10	2	D	Catered Indonesian foods
	March	Restaurant	Unknown	10	0	D	Unknown
	March	Restaurant	*S.* Typhimurium 170	2	1	D	Fijian chicken
	March	Restaurant	*S.* Typhimurium 170	33	13	AM	Fried icecream
	March	Restaurant	*S.* Virchow	3	1	D	Unknown
	March	Restaurant	*Campylobacter*	4	0	D	Suspected hickory steak with chips and salad
NT	March	Private residence	*S.* Typhimurium U302	2	0	D	Suspected tiramisu
Qld	February	Aged care facility	*S.* Typhimurium	3	Unknown	D	Unknown
	February	Restaurant	Unknown	6	0	D	Unknown
	February	Commercial caterer	Norovirus	20	1	A	Unknown
	February	Primary produce	Ciguatera fish poisoning	3	2	D	Spanish mackerel

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicle
Qld, *cont'd*	January	Aged care facility	*S.* Typhimurium 44	20	4	AM	Suspected vitamised foods and scrambled eggs
	January	Aged care facility	*S.* Typhimurium 135a	3	0	D	Unknown
	January	Private residence	Norovirus	10	1	D	Unknown
Vic	February	Commercial caterer	*S.* Typhimurium 170	4	1	D	Unknown
	February	Restaurant	Unknown	10	0	D	Suspected stews and casseroles
	February	Restaurant	*S.* Typhimurium 197	2	2	D	Unknown
	March	Aged care facility	*C. perfringens*	22	0	D	Suspected vitamised meals
	March	Camp	Unknown	13	0	D	Unknown
	March	Child care centre	*S.* Typhimurium 170	18	1	D	Unknown
	March	Private residence	*S.* Typhimurium 44	7	1	D	Unknown
WA	February	Restaurant	*S.* Saintpaul	7	1	M	Fried icecream
	February	Aged care facility	Unknown	16	0	D	Unknown
	March	Restaurant	*S.* Singapore	10	3	D	Unknown

* No foodborne outbreaks were reported by Tasmania or South Australia during the quarter.

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.

and both analytical epidemiological and microbiological evidence in 4 outbreaks. Descriptive evidence only was obtained in 36 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred in this quarter. Tasmania and South Australia did not report any foodborne outbreaks during this quarter.

Australian Capital Territory

The Australian Capital Territory reported 4 outbreaks of foodborne illness during the quarter, three of which were due to *Salmonella* and one due to histamine poisoning.

Twenty people became ill following meals at a restaurant and *S.* Typhimurium 170 was isolated from stool specimens of 8 cases. A case control study showed that illness was associated with eating dessert containing raw eggs. Environmental investigation did not identify any positive foods or environmental samples. Traceback identified an egg producer/supplier in the Australian Capital Territory, although the same serotype and phage type of *Salmonella* was not isolated. In the 2nd outbreak, 6 people in a single family became ill, with 75% (3/4) of children confirmed with *S.* Typhimurium 170 infection. No source was identified for the cluster. The 3rd outbreak affected a family of five infected with *S.* Montevideo attending a birthday party in New South Wales (reported in New South Wales report).

An outbreak of histamine poisoning affected 2 people after eating tuna steaks purchased from an Australian Capital Territory fishmonger. Traceback identified a Queensland supplier, which was referred to Queensland Health.

New South Wales

New South Wales reported 22 foodborne or suspected foodborne disease outbreaks in the 1st quarter of 2009, with 13 of these being due to *Salmonella*. Ten

of these outbreaks were due to *S.* Typhimurium 170 with closely related multi-locus variable tandem repeat analysis (MLVA) profiles, including:

- 11% (33/308) of customers eating at a Japanese teppanyaki restaurant where people became ill from eating fried icecream (RR: 257.88, 95% CI: 36.39–1827.54). *S.* Typhimurium 170 MLVA type 3-9-7-12-523 was cultured from 12 cases with a matching strain cultured from samples of fried icecream, raw beef and a dish cloth from the restaurant.
- 50% (2/4) of Spanish restaurant customers where people were infected with *S.* Typhimurium 170 (MLVA 3-10-7-9-523). No food samples were tested and the source of the *Salmonella* was never identified.
- 27% (4/15) of aged care facility residents infected with *S.* Typhimurium 170 (MLVA 3-9-7-12-523); the source was never identified.
- 60% (3/5) of children in a single household infected with *S.* Typhimurium 170 (MLVA 3-9-7-13-523), which was suspected to be due to bacon and beef burger meals at a franchise. No source was identified for this outbreak.
- 57% (34/59) of female boarders and 38% (3/8) of staff of a school infected with *S.* Typhimurium 170 (MLVA type 3-9-8-12-523). No source was identified, despite intensive epidemiological and environmental investigations.
- 40% (40/100) of people living in a retirement village infected with *S.* Typhimurium 170 (MLVA type 3-9-8-12-523), which was associated with consumption of Hollandaise sauce prepared with raw eggs (RR: 2.0, 95% CI: 0.6-7.4).
- illness amongst a group of approximately 120 people attending a barbecue at a bowling club who were infected with *S.* Typhimurium 170 (MLVA type 3-9-8-12-523). In a cohort study, 82% (68/83) of people were ill with gastroenteritis, which was associated with consumption of lettuce (RR=1.4 95%CI 1.0-2.0) and Russian salad (RR=1.8 95%CI 1.2-2.9). The Russian salad was prepared with homemade raw egg mayonnaise, which was positive for the same strain of *S.* Typhimurium 170 as that infecting patients.
- 2 people infected with *S.* Typhimurium 170 (MLVA type 3-9-8-12-523) after eating chicken salad with mayonnaise from a café; a specific source wasn't identified.
- 4% (7/162) of residents at an aged care facility infected with *S.* Typhimurium 170 (MLVA type 3-9-8-12-523), with 2 residents hospitalised. All food samples and environmental swabs were negative for *Salmonella* and no source was identified.
- 5 people from 2 families sharing a barbecue were infected with *S.* Typhimurium 170 (MLVA type 3-15-16-14-523). No source of the outbreak was identified.

New South Wales also reported 3 outbreaks due to other serotypes of *Salmonella*. Fifty per cent (10/20) of people attending a birthday party developed illness due to *S.* Montevideo. A family from the Australian Capital Territory was also affected in this outbreak. Indonesian food, including chicken skewers, was served by a caterer as well as a home-made birthday cake and no source was identified. In another outbreak, 14 people from 6 groups were infected with *S.* Chester on 3 consecutive days after eating chilli sauce prepared at a restaurant. Two food handlers who prepared the chilli sauce were asymptomatically infected with *S.* Chester and were excluded from work. Chilli sauce and raw chillies were positive for *S.* Chester, although it was not possible to trace the source of chillies. In the 3rd outbreak, three of 4 people from different households developed gastroenteritis after eating chilli crab in a Chinese restaurant; one had *S.* Virchow isolated from their stool. Foods were negative for *Salmonella* and no source was identified.

An outbreak of histamine poisoning affected two out of 8 people after eating Nicoise salad with tinned anchovies and tuna. Elevated histamine (360 mg/kg) was detected in cans of anchovies (imported from Morocco) at the restaurant.

Seventeen per cent (25/146) of residents of an aged care facility were ill with *Clostridium perfringens* intoxication, with enterotoxin A detected in 5 stool samples. The New South Wales Food Authority sampled vegetable gravy that had been inadequately stored, which contained moderate levels of both *C. perfringens* and *Bacillus cereus.*

An outbreak of *Campylobacter* affected 33% (4/12) of people following a meal of steak, chips and salad at a franchise, although no source of the outbreak was identified.

New South Wales health reported a further 4 outbreaks of gastroenteritis of unknown aetiology.

Northern Territory

The Northern Territory reported 1 outbreak of foodborne or suspected foodborne illness during the quarter.

Two people experienced gastroenteritis following a dinner party of 6 people at a private residence. One case tested positive for *S.* Typhimurium U302. The food vehicle was suspected to be tiramisu made with raw eggs.

Queensland

Queensland reported 7 outbreaks of foodborne or suspected foodborne illness during this quarter: *S.* Typhimurium caused 3 outbreaks, norovirus caused 2 outbreaks, ciguatera fish poisoning caused 1 outbreak and there was 1 outbreak where no aetiological agent was identified.

All 3 *Salmonella* outbreaks during the 1st quarter occurred in aged care facilities. In January 2009, 3 residents of a Fraser Coast nursing home were infected with *S.* Typhimurium 135a (MLVA profile 1-3-4-21-3), although no source of infection was identified. Also in January, 20 residents of a Gold Coast nursing home were infected with *S.* Typhimurium 44 (MLVA profile 1-1-19-14-3). The same strain of *S.* Typhimurium was isolated from a swab of the kitchen blender and from several food samples, including vitamised meals and scrambled eggs, although the source of contamination wasn't definitively identified. In February 2009, 2 female residents of a Brisbane aged care facility and a male resident of an adjacent facility were notified with *S.* Typhimurium. All cases consumed egg meals prior to illness; however, no vehicle or source of infection was identified.

Queensland reported 2 foodborne outbreaks due to norovirus during the quarter. The 1st occurred in January and affected 83% (10/12) of people from 3 families who shared a common meal. Onset dates of illness suggested likely foodborne transmission, although no source of infection was identified. In the 2nd norovirus outbreak, 68% (20/29) of people attending a work conference became ill after consuming sandwiches and bakery items. Hot cross buns (RR 2.2, 95% CI 1.0–4.8) were associated with illness and a mixture of person-to-person and foodborne transmission was suspected.

In February 2009, 3 people were affected by ciguatera fish poisoning after eating Spanish mackerel steaks. The median incubation period was 6 hours (range 4–9 hours) and 2 case patients were hospitalised. The fish (>20 kg) was purchased from a Brisbane market and had been caught off Mooloolaba by a private fisherman.

Six people became ill with gastroenteritis after eating at a Gold Coast restaurant in February 2009. No faecal specimens were collected, although *B. cereus* was detected in 83% (5/6) swabs from the kitchen. No vehicle or source of infection was identified.

Victoria

Victoria reported 7 outbreaks of foodborne or suspected foodborne illness this quarter.

There were 3 foodborne outbreaks in February 2009. The 1st affected a group of people staying at a motel where 24% (10/42) of people eating a buffet meal became ill with diarrhoea. Clinical illness was consistent with *C. perfringens* intoxication and 1 faecal specimen was positive for *C. perfringens* enterotoxin. In the 2nd outbreak, 2 people who regularly ate food at the same restaurant were infected with *S.* Typhimurium 197, although no source was identified. In the 3rd outbreak, 4% (3/80) of people attending a 40th wedding anniversary were infected with *S.* Typhimurium 170, along with an employee. No source was identified for the outbreak.

In March, 22 residents of an aged care home experienced diarrhoea, with most people becoming ill on a single day. Ten of the cases had a vitamised diet representing 33% of all of the people in the facility on this diet, although the association was not significant (RR 1.6, 95%CI 0.79–3.4). Ten faecal specimens were positive for *C. perfringens* enterotoxin.

Routine surveillance identified an outbreak of *S.* Typhimurium 170 amongst 17 children attending the same child care centre. One staff member was also affected. No source was identified for the outbreak. In March, gastrointestinal illness affected a group of 93 people attending a bushwalking club weekend. The attack rate was 43% amongst 30 people responding to a questionnaire and no source for the outbreak was identified. The illness was consistent with a bacterial intoxication. Routine surveillance identified an outbreak of *S.* Typhimurium 44 affecting 5 people attending a birthday party at a private home, although no source was identified.

Western Australia

Western Australia reported 3 outbreaks during the 1st quarter of 2009.

Seven cases of *S.* Saintpaul were associated with eating at an Asian restaurant in January 2009, with cases reporting incubation periods ranging from 3.5 hours to 10 days. Eighty-six per cent (6/7) of cases had eaten fried icecream, and one case had eaten a red bean dessert with no icecream. People attending the restaurant during this period were contacted but no further cases were identified. Fried icecream was positive for *S.* Saintpaul, which was indistinguishable from human strains by pulsed-field gel electrophoresis (PFGE). The source of *S.* Saintpaul contamination of the fried icecream was not identified.

In February 2009, 33% (16/48) of residents of a high care unit in an aged care facility experienced diarrhoea with onset of illness over a 2 day period and a median duration of 1.5 days. Two staff members were ill with diarrhoea and vomiting. Consuming

vitamised food was strongly associated with illness (OR 11.5, CI 1.9-116.6). Thirteen faecal samples were negative for common bacterial and viral pathogens, as well as bacterial toxins. Two stools were positive for *C. perfringens* but had different PFGE profiles. There were no remaining food samples from the period prior to onset of illness, and more recent food samples that had been vitamised were negative for common bacterial pathogens and toxin. The aetiological agent and source of infection were not identified.

Ten cases of *S.* Singapore were notified in February and March 2009. Five cases had eaten at the same outlet of an Asian franchise restaurant and another case ate at a different outlet of the same franchise. There were no reports of staff illness and chicken meat samples were negative for *Salmonella*. The source of *S.* Singapore from the restaurant franchise was not identified.

Cluster investigations

During the 1st quarter of 2009, OzFoodNet investigated a multi-jurisdictional outbreak in Shiga toxin-producing *Escherichia coli* (STEC) O157 infection. The Microbiological Diagnostic Unit Public Health Laboratory typed isolates using PFGE and phage typing and identified that amongst STEC O157 cases, there was a distinct cluster of 14 cases in Queensland (3 cases), New South Wales (3 cases), Victoria (1 case), and South Australia (7 cases). OzFoodNet epidemiologists identified several foods of interest through hypothesis generating interviews, but there were no common brands and the number of cases declined before an analytical study could be performed.

During the 1st quarter of 2009, jurisdictions reported increases in cryptosporidiosis, including the Australian Capital Territory with 68 cases in the 1st quarter of 2009 compared with seven for same time period in 2008, and Queensland with 1,036 cases in the 1st quarter of 2009 compared with 241 in 2008. Queensland reported that a cluster of 12 cases of cryptosporidiosis in the Sunshine Coast in March had swum at the same aquatic centre, where 2 water samples were positive for *Cryptosporidium*. The Northern Territory reported clusters of cases of cryptosporidiosis in remote communities.

Several clusters of *Salmonella* were investigated during the quarter, including serotypes Montevideo, Singapore, Virchow, subspecies 1, Waycross, and Typhimurium phage types 197, 9, 44 and 170/108. Victoria investigated a cluster of 4 cases of locally acquired typhoid infection where case patients shopped at the same food store. There were no secondary cases among families of the 4 cases. Despite intensive investigations, no source was identified.

In addition, jurisdictions investigated clusters of a range of other enteric infections during the quarter, including: yersiniosis and listeriosis in Queensland, multi-drug resistant *Shigella sonnei* biotype G in Victoria, *Shigella sonnei* biotype A, and *Shigella flexneri* 3b and STEC O157 (with a different PFGE to the multi-jurisdictional outbreak) in Western Australia.

Comments

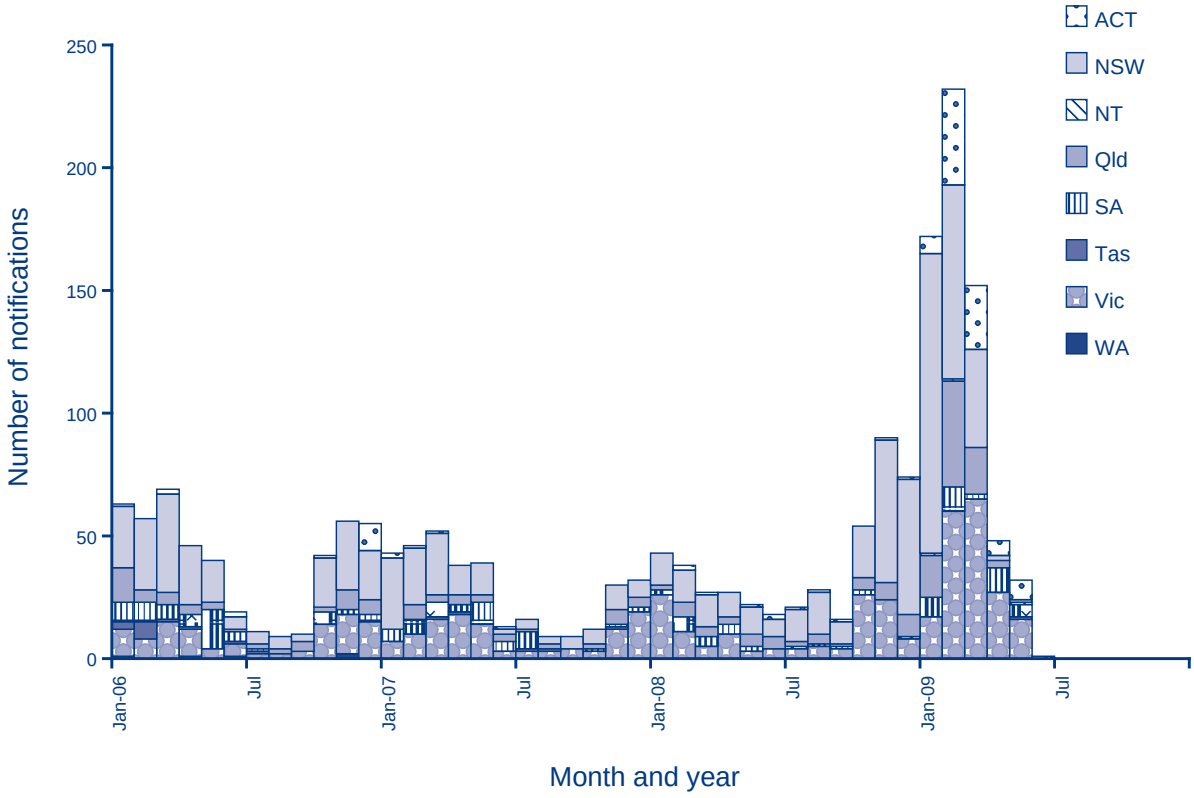
There were a large number of foodborne outbreaks (n=43) during the quarter when compared with the previous quarter and the same quarter in 2008. The main cause of the increase was a large increase in both sporadic cases and point source outbreaks due to *S.* Typhimurium 170/108 (Figure). In total, there were 15 outbreaks of *S.* Typhimurium 170/108 this quarter, with 11 occurring in New South Wales. As a result of the large number of outbreaks and increase in sporadic notifications of this phage type, OzFoodNet initiated a multi-jurisdictional outbreak investigation in April 2009.

The majority of these *S.* Typhimurium 170/108 outbreaks were suspected to be caused by contaminated eggs, which are a consistent cause of *Salmonella* outbreaks in Australia.[1,2] This is partly because eggs are a commonly consumed food, but also due to the endemic nature of *Salmonella* in egg-laying flocks. It is important that consumers and the food service industry recognise the risks associated with raw or partially cooked eggs. Food Standards Australia New Zealand is currently preparing a primary production and processing standard for the egg production sector to improve food safety relating to eggs in Australia.[1]

During the quarter there were 8 outbreaks of foodborne disease in aged care facilities, which is higher than previous years. Five of these outbreaks were due to *Salmonella,* which reflects the general upsurge in salmonellosis outbreaks during summer and autumn. Outbreaks of gastroenteritis are common amongst aged care facility residents, but most of these are non-foodborne.[3] In Australia, a new food standard was introduced that was designed to protect populations that may be more vulnerable to foodborne infections, such as those living in aged care facilities.[4]

During the quarter, jurisdictions used a variety of typing schema and nomenclature for *Salmonella,* which made it difficult to determine whether increases in infections in 1 jurisdiction related to that occurring in other jurisdictions. For the investigation into the multi-jurisdictional outbreak of STEC, it was necessary to send isolates or specimens from several jurisdictions to a single laboratory due to the complicated nature of testing. Laboratories

Figure: Notifications of Salmonella Typhimurium 170/108 to state and territory health departments, National Notifiable Diseases Surveillance System, Australia, 2006 to June 2009



Data were extracted on 25 June 2009.

are fundamental to foodborne disease investigation and it is important to understand the impact of changes in laboratory testing on outbreak detection and investigation.[5]

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* for their continuing work during this quarter.

The OzFoodNet Working Group is (in alphabetical order): Robert Bell (Qld), Barry Combs (WA), Craig Dalton (Hunter New England), Lindy Fritsche (NSW), Kathleen Fullerton (DoHA), Robyn Gibbs (WA), Joy Gregory (Vic), Gillian Hall (NCEPH), Michelle Harlock (NT), Geoff Hogg (MDU), Libby Gruszynski (HNE), Katina Kardamanidis (NSW), Martyn Kirk (DoHA), Katrina Knope (DoHA), Karin Lalor (Vic), Narelle Marro (DAFF), Tony Merritt (Hunter New England), Cameron Moffatt (ACT), Jennie Musto (NSW), Lillian Mwanri (SA), Bev Paterson (Hunter New England), Nevada Pingault (WA), Jane Raupach (SA), Mark Salter (FSANZ), Craig Shadbolt (NSWFA), Frances Sheehan (Qld), Russell Stafford (Qld), Nicola Stephens (Tas), Hassan Vally (NCEPH), Mark Veitch (MDU).

Author details

Correspondence: Mr Martyn Kirk, Senior Epidemiologist, OzFoodNet, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 9848, MDP 14, CANBERRA ACT 2601. Telephone: +61 2 6289 2746. Facsimile: +61 2 6289 2500. Email: martyn.kirk@health.gov.au

References

1. 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;32:400–424.
2. Stephens N, Coleman D, Shaw K. Recurring outbreaks of *Salmonella* Typhimurium phage type 135 associated with the consumption of products containing raw egg in Tasmania. *Commun Dis Intell* 2008;32:466–468.
3. Ryan MJ, Wall PG, Adak GK, Evans HS, Cowden JM. Outbreaks of infectious intestinal disease in residential institutions in England and Wales 1992–1994. *J Infect* 1997;34:49–54.
4. Kirk MD, McKay I, Hall GV, Dalton CB, Stafford R, Unicomb L, et al. Food safety: foodborne disease in Australia: the OzFoodNet experience. *Clin Infect Dis* 2008;47:392–400.
5. Kafatos G, Andrews N, Gillespie IA, Charlett A, Adak GK, De Pinna E, et al. Impact of reduced numbers of isolates phage-typed on the detection of *Salmonella* outbreaks. *Epidemiol Infect* 2009;137:821–827.

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 50,978 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January and 31 March 2009 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia where data is not collected.
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 January to 31 March 2009, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2009†	Total 4th quarter 2008	Total 1st quarter 2008	Last 5 years mean 1st quarter	Year to date 2009	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	1	0	0.4	0	0.4	0.0
Hepatitis B (incident)	0	8	2	10	2	2	16	0	40	50	56	70.2	40	70.2	0.6
Hepatitis B (unspecified)	37	848	44	263	94	16	515	192	2,009	1,742	1,550	1,576.6	2,009	1,576.6	1.3
Hepatitis C (incident)	2	8	1	NN	4	10	40	2	67	111	84	94.0	67	94.0	0.7
Hepatitis C (unspecified)	46	1,273	46	760	130	68	608	294	3,225	2,846	2,727	3,084.0	3,225	3,084.0	1.0
Hepatitis D	0	3	0	6	0	0	1	0	10	6	12	8.6	10	8.6	1.2
Gastrointestinal diseases															
Botulism	0	0	0	1	0	0	0	0	1	0	0	0.6	1	0.6	1.7
Campylobacteriosis§	96	NN	69	1,276	411	136	1,453	586	4,027	4,081	4,647	4,336.0	4,027	4,336.0	0.9
Cryptosporidiosis	71	937	69	1,048	39	8	567	121	2,860	452	756	1,036.0	2,860	1,036.0	2.8
Haemolytic uraemic syndrome	0	1	0	0	2	0	0	0	3	11	8	5.4	3	5.4	0.6
Hepatitis A	1	22	0	9	4	0	26	7	69	48	85	85.2	69	85.2	0.8
Hepatitis E	0	8	0	3	0	0	4	1	16	9	15	11.4	16	11.4	1.4
Listeriosis	1	7	0	5	4	2	8	2	29	9	28	19.6	29	19.6	1.5
Salmonellosis	0	9	0	12	24	0	5	3	53	40	31	22.8	53	22.8	2.3
Shigellosis	105	1,124	125	1,040	168	56	491	282	3,391	2,132	2,889	2,975.6	3,391	2,975.6	1.1
STEC, VTEC‖	3	56	30	27	18	1	35	48	218	206	240	190.8	218	190.8	1.1
Typhoid	1	14	0	5	0	1	14	2	37	24	35	29.2	37	29.2	1.3
Quarantinable diseases															
Cholera	0	2	0	0	0	0	0	0	2	4	0	0.8	2	0.8	2.5
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 January to 31 March 2009, by date of diagnosis,* continued

Disease	State or territory								Total 1st quarter 2009†	Total 4th quarter 2008	Total 1st quarter 2008	Last 5 years mean 1st quarter	Year to date 2009	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	231	3,737	567	4,196	894	337	3,437	2,200	15,599	14,256	14,474	11,947.2	15,599	11,947.2	1.3
Donovanosis	0	0	0	0	0	0	0	0	0	0	1	2.0	0	2.0	0.0
Gonococcal infection	14	432	377	448	128	4	429	385	2,217	1,834	1,951	2,045.0	2,217	2,045.0	1.1
Syphilis (all)	13	379	39	84	6	4	218	50	793	780	810	671.8	793	671.8	1.2
Syphilis < two years duration	2	136	10	37	6	0	101	25	317	291	347	231.4	317	231.4	1.4
Syphilis >two years or unspecified duration	11	243	29	47	NDP	4	117	25	476	489	463	440.4	476	440.4	1.1
Syphilis - congenital	0	0	2	0	0	0	0	0	2	3	0	2.6	2	2.6	0.8
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	4	0	2	0	0	1	1	8	5	5	4.0	8	4.0	2.0
Influenza (laboratory confirmed)	12	239	16	97	15	2	33	57	471	1,562	433	290.0	471	290.0	1.6
Measles	1	7	1	31	1	2	33	2	78	1	33	14.2	78	14.2	5.5
Mumps	0	8	2	7	5	1	27	6	56	36	141	61.0	56	61.0	0.9
Pertussis	118	5,376	79	1,231	681	136	688	184	8,493	7,425	1,542	1,675.0	8,493	1,675.0	5.1
Pneumococcal disease (invasive)	7	60	12	31	18	2	54	21	205	353	206	243.2	205	243.2	0.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	1	0	2	1	0	3	1	8	10	5	6.4	8	6.4	1.3
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0.0
Tetanus	0	2	0	0	0	0	1	0	3	0	3	1.2	3	1.2	2.5
Varicella zoster (chickenpox)	0	NN	20	34	85	8	79	84	310	767	271	269.0	310	269.0	1.5
Varicella zoster (shingles)	3	NN	21	68	246	29	148	135	650	754	552	401.0	650	401.0	2.7
Varicella zoster (unspecified)	16	NN	0	990	76	24	409	244	1,759	1,383	957	970.0	1,759	970.0	3.0
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	13	0	0	1	0	14	12	7	16.2	14	16.2	0.9
Barmah Forest virus infection	0	117	51	304	12	1	8	75	568	424	823	538.8	568	538.8	1.1
Dengue virus infection	7	58	12	881	8	1	10	36	1,013	211	159	127.8	1,013	127.8	7.9
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0.0
Kunjin virus infection	0	0	1	1	0	0	0	0	2	0	0	1.2	2	1.2	1.7
Malaria	1	26	2	57	3	3	22	19	133	129	124	191.4	133	191.4	0.7
Murray Valley encephalitis virus infection	0	0	1	0	0	0	0	1	2	0	1	0.8	2	0.8	2.5
Ross River virus infection	0	168	197	720	68	10	31	445	1,639	937	2,780	2,138.0	1,639	2,138.0	0.8

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2009, by date of diagnosis,* continued

Disease	State or territory								Total 1st quarter 2009†	Total 4th quarter 2008	Total 1st quarter 2008	Last 5 years mean 1st quarter	Year to date 2009	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0.4	0.0
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	1	0	6	1	0	0	1	9	12	8	11.0	9	11.0	0.8
Leptospirosis	1	4	3	57	0	0	0	1	66	23	43	50.0	66	50.0	1.3
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	5	0	0	1	0	10	0	16	19	23	40.6	16	40.6	0.4
Q fever	0	48	1	46	2	0	1	0	98	99	111	103.6	98	103.6	0.9
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Other bacterial infections															
Legionellosis	1	19	2	14	7	0	9	12	64	76	62	82.0	64	82.0	0.8
Leprosy	0	0	0	0	0	0	1	0	1	3	3	3.2	1	3.2	0.3
Meningococcal infection**	0	16	2	11	4	1	9	4	47	64	42	65.8	47	65.8	0.7
Tuberculosis	3	94	5	49	18	2	104	33	308	362	289	266.4	308	266.4	1.2
Total	791	15,124	1,799	14,028	3,180	876	9,549	5,631	50,978	43,792	39,255	35,279.0	50,978	35,279.0	1.4

* 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 syphilis <2 years; syphilis >2 years or unspecified duration based on 5 years data.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

|| Infections 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 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 January to 31 March 2009, 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 (incident)	0.0	0.5	3.6	0.9	0.5	1.6	1.2	0.0	0.7
Hepatitis B (unspecified)	43.0	48.7	80.0	24.6	23.5	12.8	38.9	35.5	37.6
Hepatitis C (incident)	2.3	0.5	1.8	0.0	1.0	8.0	3.0	0.4	1.6
Hepatitis C (unspecified)	53.5	73.1	83.7	71.0	32.5	54.6	45.9	54.4	60.4
Hepatitis D	0.0	0.2	0.0	0.6	0.0	0.0	0.1	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	111.6	0.0	125.5	119.3	102.6	109.2	109.7	108.4	111.8
Cryptosporidiosis	82.5	53.8	125.5	98.0	9.7	6.4	42.8	22.4	53.5
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.5	0.0	0.0	0.0	0.1
Hepatitis A	1.2	1.3	0.0	0.8	1.0	0.0	2.0	1.3	1.3
Hepatitis E	0.0	0.5	0.0	0.3	0.0	0.0	0.3	0.2	0.3
Listeriosis	1.2	0.4	0.0	0.5	1.0	1.6	0.6	0.4	0.5
Salmonellosis	0.0	0.5	0.0	1.1	6.0	0.0	0.4	0.6	1.0
Shigellosis	122.0	64.5	227.3	97.2	42.0	45.0	37.1	52.1	63.5
STEC, VTEC‡	3.5	3.2	54.6	2.5	4.5	0.8	2.6	8.9	4.1
Typhoid	1.2	0.8	0.0	0.5	0.0	0.8	1.1	0.4	0.7
Quarantinable diseases									
Cholera	0.0	0.1	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§	268.4	214.5	1,031.2	392.2	223.2	270.6	259.5	406.8	292.0
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	16.3	24.8	685.6	41.9	32.0	3.2	32.4	71.2	41.5
Syphilis (all)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis <2 years duration	0.0	0.0	3.6	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis >2 years or unspecified duration	2.3	7.8	18.2	3.5	1.5	0.0	7.6	4.6	5.9
Syphilis - congenital	12.8	14.0	52.7	4.4	0.0	3.2	8.8	4.6	9.6
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.2	0.0	0.2	0.0	0.0	0.1	0.2	0.1
Influenza (laboratory confirmed)	13.9	13.7	29.1	9.1	3.7	1.6	2.5	10.5	8.8
Measles	1.2	0.4	1.8	2.9	0.2	1.6	2.5	0.4	1.5
Mumps	0.0	0.5	3.6	0.7	1.2	0.8	2.0	1.1	1.0
Pertussis	137.1	308.6	143.7	115.1	170.1	109.2	51.9	34.0	159.0
Pneumococcal disease (invasive)	8.1	3.4	21.8	2.9	4.5	1.6	4.1	3.9	3.8

Table 3: Notification rates of diseases, 1 January to 31 March 2009, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.2	0.2	0.0	0.2	0.2	0.1
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Varicella zoster (chickenpox)	0.0	0.0	36.4	3.2	21.2	6.4	6.0	15.5	8.6
Varicella zoster (shingles)	3.5	0.0	38.2	6.4	61.4	23.3	11.2	25.0	18.1
Varicella zoster (unspecified)	18.6	0.0	0.0	92.5	19.0	19.3	30.9	45.1	48.8
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	1.2	0.0	0.0	0.1	0.0	0.3
Barmah Forest virus infection	0.0	6.7	92.7	28.4	3.0	0.8	0.6	13.9	10.6
Dengue virus infection	8.1	3.3	21.8	82.3	2.0	0.8	0.8	6.7	19.0
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	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	1.2	1.5	3.6	5.3	0.7	2.4	1.7	3.5	2.5
Murray Valley encephalitis virus infection	0.0	0.0	1.8	0.0	0.0	0.0	0.0	0.2	0.0
Ross River virus infection	0.0	9.6	358.3	67.3	17.0	8.0	2.3	82.3	30.7
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.6	0.2	0.0	0.0	0.2	0.2
Leptospirosis	1.2	0.2	5.5	5.3	0.0	0.0	0.0	0.2	1.2
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.3	0.0	0.0	0.2	0.0	0.8	0.0	0.3
Q fever	0.0	2.8	1.8	4.3	0.5	0.0	0.1	0.0	1.8
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	1.2	1.1	3.6	1.3	1.7	0.0	0.7	2.2	1.2
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Meningococcal infection‖	0.0	0.9	3.6	1.0	1.0	0.8	0.7	0.7	0.9
Tuberculosis	3.5	5.4	9.1	4.6	4.5	1.6	7.9	6.1	5.8

* 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 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 8,456 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2009 (Tables 4 and 5).

Table 4: Virology and serology laboratory reports, 1 January to 31 March 2009, by state or territory,* and total reports for the year†

	State or territory								This period 2009	This period 2008	Year to date 2009	Year to date 2008
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	4	–	10	2	–	23	–	39	13	39	13
Mumps virus	–	–	1	1	3	–	6	1	12	19	12	19
Rubella virus	–	–	–	2	2	–	–	–	4	6	4	6
Hepatitis viruses												
Hepatitis A virus	–	1	–	5	2	–	–	–	8	23	8	23
Hepatitis D virus	–	–	–	–	6	–	1	–	7	9	7	9
Hepatitis E virus	–	–	–	2	–	–	1	–	3	2	3	2
Arboviruses												
Ross River virus	–	5	1	280	109	2	1	2	400	796	400	796
Barmah Forest virus	–	7	–	79	19	–	1	–	106	250	106	250
Flavivirus (unspecified)	–	9	–	140	–	–	–	–	149	30	149	30
Adenoviruses												
Adenovirus not typed/ pending	1	54	–	163	182	–	5	–	405	349	405	349
Herpesviruses												
Cytomegalovirus	5	58	–	161	116	6	13	–	359	366	359	366
Varicella-zoster virus	1	63	2	468	185	3	10	–	752	760	752	760
Epstein-Barr virus	–	13	–	303	287	5	4	–	612	655	612	655
Other DNA viruses												
Parvovirus	–	4	–	39	9	–	14	–	66	80	66	80
Picornavirus family												
Coxsackievirus A9	–	5	–	–	–	–	–	–	5	1	5	1
Coxsackievirus A16	–	3	–	–	–	–	–	–	3		3	
Echovirus type 4	–	1	–	–	–	–	–	–	1	1	1	1
Echovirus type 9	–	1	–	–	–	–	–	–	1	4	1	4
Echovirus type 30	–	7	–	–	–	–	–	–	7	–	7	–
Rhinovirus (all types)	–	24	–	–	–	–	–	–	24	38	24	38
Enterovirus not typed/ pending	–	27	–	5	3	2	1	–	38	111	38	111
Picornavirus not typed	–	–	–	–	–	3	1	–	4	1	4	1
Ortho/paramyxoviruses												
Influenza A virus	–	25	–	43	16	–	2	–	86	49	86	49
Influenza B virus	1	17	–	10	7	–	–	–	35	22	35	22
Parainfluenza virus type 1	–	2	–	–	1	–	–	–	3	63	3	63
Parainfluenza virus type 2	–	4	–	8	2	–	–	–	14	10	14	10
Parainfluenza virus type 3	–	19	–	2	20	–	3	–	44	7	44	7
Respiratory syncytial virus	–	108	–	82	54	–	6	1	251	205	251	205

Table 4: Virology and serology laboratory reports, 1 January to 31 March 2009, by state or territory,* and total reports for the year,† *continued*

	State or territory								This period 2009	This period 2008	Year to date 2009	Year to date 2008
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	–	–	–	–	83	–	–	–	83	4	83	4
Rotavirus	–	7	–	–	53	3	–	–	63	109	63	109
Norwalk agent	–	16	–	–	–	–	–	–	16	17	16	17
Other												
Chlamydia trachomatis not typed	3	338	1	1,448	555	11	22	1	2,379	2,357	2,379	2,357
Chlamydia pneumoniae	–	–	–	1	–	–	1	–	2	–	2	–
Chlamydia psittaci	–	1	–	2	–	–	20	–	23	22	23	22
Chlamydia spp typing pending	–	2	–	–	–	–	–	–	2	–	2	–
Chlamydia species	–	2	–	–	–	–	3	–	5	2	5	2
Mycoplasma pneumoniae	–	6	–	128	50	3	66	–	253	207	253	207
Mycoplasma hominis	–	2	–	–	–	–	–	–	2	2	2	2
Coxiella burnetii (Q fever)	–	2	–	12	17	–	–	–	61	80	61	80
Rickettsia prowazeki	–	–	–	–	1	–	–	–	1	–	1	–
Rickettsia - spotted fever group	–	1	–	20	1	–	2	–	38	28	38	28
Streptococcus group A	–	4	–	172	–	–	1	1	178	272	178	272
Brucella abortus	–	–	–	–	1	–	–	–	1	–	1	–
Brucella species	–	–	–	4	–	–	–	–	4	8	4	8
Bordetella pertussis	1	532	–	338	488	2	1	–	1,362	198	1,362	198
Legionella pneumophila	–	2	–	3	–	–	1	–	6	7	6	7
Legionella longbeachae	–	–	–	–	1	–	2	–	3	4	3	4
Legionella species	–	–	–	3	–	–	2	–	5	1	5	1
Cryptococcus species	–	4	–	2	4	–	–	–	10	7	10	7
Leptospira species	–	–	–	16	2	–	–	–	18	30	18	30
Treponema pallidum	1	66	1	227	172	–	21	–	488	565	488	565
Toxoplasma gondii	–	1	–	4	1	1	–	–	7	1	7	1
Echinococcus granulosus	–	–	–	–	8	–	–	–	8	7	8	7
Total	13	1,447	6	4,183	2,462	41	234	6	8,456	7,798	8,456	7,798

* State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

† Data presented are for reports with reports dates in the current period.

– No data received this period.

Table 5: Virology and serology reports, 1 January to 31 March 2009,* by laboratory

State or territory	Laboratory	January 2009	February 2009	March 2009	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	263	292	357	912
	New Children's Hospital, Westmead	46	48	68	162
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	8	28	20	56
	South West Area Pathology Service, Liverpool	32	42	55	129
Queensland	Queensland Medical Laboratory, West End	1,631	1,253	1,522	4,406
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	724	830	907	2,461
Tasmania	Northern Tasmanian Pathology Service, Launceston	12	16	6	34
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	21	19	24	64
	Monash Medical Centre, Melbourne	3	5	12	20
	Royal Children's Hospital, Melbourne	–	–	–	–
	Victorian Infectious Diseases Reference Laboratory, Fairfield	83	44	85	212
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	–	–	–	–
Total		2,823	2,577	3,056	8,456

* The complete list of laboratories reporting for the 12 months, January to December 2009, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic data collection was established in 2006 and currently, further development of ASPREN is in progress to create an automatic reporting system.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2009, four conditions are being monitored. They include influenza like illness, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2008;32:135.*

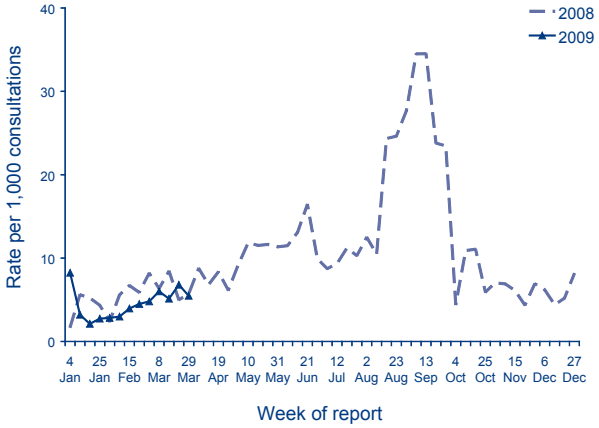
Data on influenza-like illness, gastroenteritis, chickenpox and shingles from 1 January to 31 March 2009 compared with 2007, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 and 4, respectively.

Reporting period 1 January to 31 March 2009

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 100 general practitioners contributed data to ASPREN in the 1st quarter of 2009. Each week an average of 73 general practitioners provided information to ASPREN at an average of 6,735 (range 3,764 to 7,400) consultations per week.

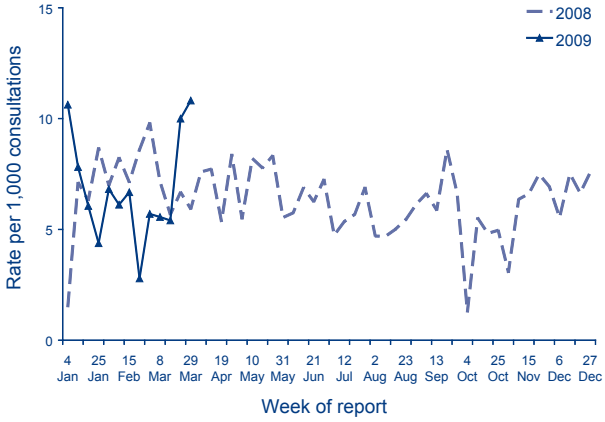
ILI rates reported from 1 January to 31 March 2009 were lower (2–8 cases per 1,000) compared with the same reporting period in 2007 (4–11 cases per 1,000 consultations (Figure 1).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2008 to 31 March 2009, by week of report



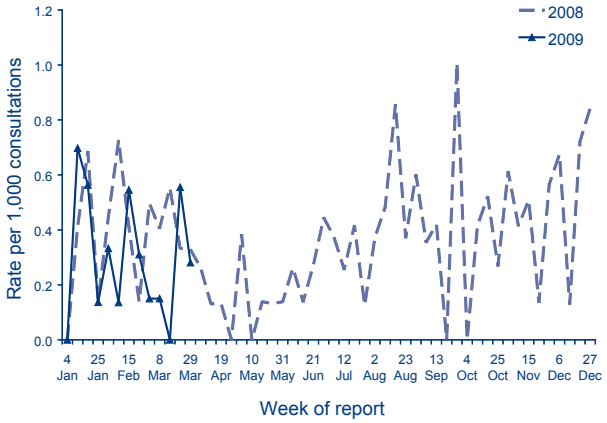
During this reporting period, consultation rates for gastroenteritis ranged from 3 to 11 cases per 1,000 consultations. Rates of gastroenteritis at the end of the 1st quarter of 2009 were approximately double compared with the same period in 2007 (Figure 2).

Figure 2: Consultation rates for gastroenteritis, ASPREN, 1 January 2008 to 31 March 2009, by week of report



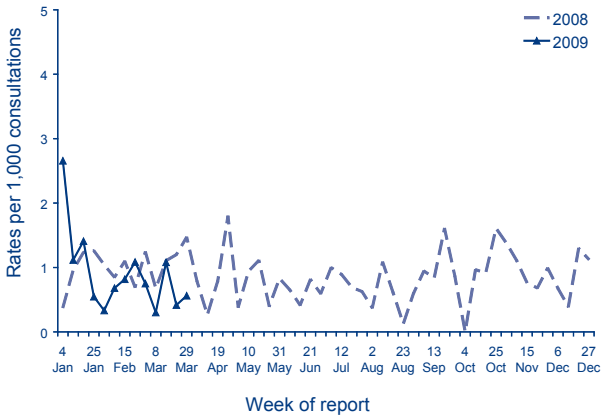
Varicella infections were reported at a similar rate for the 1st quarter of 2009 compared with the same period in 2008. From 1 January to 31 March 2009, recorded rates for chickenpox were between 0 and 1 case per 1,000 consultations (Figure 3).

Figure 3: Consultation rates for chickenpox, ASPREN, 1 January 2008 to 31 March 2009, by week of report



In the first quarter of 2009, reported rates for shingles were between less than 1 to 2.7 cases per 1,000 consultations (Figure 4).

Figure 4: Consultation rates for shingles, ASPREN, 1 January 2008 to 31 March 2009, by week of report



Australian childhood immunisation coverage

Tables 1, 2 and 3 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 October and 31 December 2007, at 24 months of age for the cohort born between 1 October and 31 December 2006, and at 5 years of age for the cohort born between 1 October and 31 December 2002 according to the National Immunisation Program Schedule. However from March 2002 to December 2007, coverage for vaccines due at 4 years of age was assessed at the 6-year milestone age.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2008;32:134–135 and for a full description of the methodology used by the Register see* Commun Dis Intell *1998;22:36-37.*

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1435, Email: brynleyh@chw.edu.au

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of *Haemophilus influenzae* type b (Hib) vaccine, and 2 or 3 doses of hepatitis B vaccine. '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 Hib vaccine, 2 or 3 doses of hepatitis B vaccine 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 four or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased slightly by 0.4 of a percentage point to 91.7% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia decreased slightly by 0.2 of a percentage point to 92.5 (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Immunisation coverage for children 'fully immunised' at 5 years of age for Australia increased for the 2nd consecutive quarter, by 1.3 percentage points, to 80.7% (Table 3). This increase nationally appears to be driven by important increases in coverage for all individual vaccines due at 4 years of age in the 2 largest populated jurisdictions, New South Wales (3.4 percentage points), and Victoria (1.8 percentage point). Various jurisdictional-specific strategies and local efforts including data quality improvements through data cleaning may have had an effect.

Table 1: Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2007; assessment date 31 March 2009

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,194	23,966	922	14,795	4,830	1,621	17,779	7,471	72,578
Diphtheria, tetanus, pertussis (%)	95.0	92.5	90.4	91.6	91.9	92.2	92.9	89.6	92.1
Poliomyelitis (%)	95.0	92.5	90.4	91.5	91.8	92.2	92.9	89.6	92.1
Haemophilus influenzae type b (%)	96.6	95.2	93.8	94.1	94.3	94.6	95.4	93.2	94.7
Hepatitis B (%)	96.6	95.1	94.3	93.9	94.1	94.6	95.2	93.3	94.6
Fully immunised (%)	94.8	92.2	90.0	91.2	91.5	92.2	92.4	89.1	91.7
Change in fully immunised since last quarter (%)	+1.1	+0.8	-0.2	+0.4	-0.3	+0.2	+0.6	-0.9	+0.4

Table 2: Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2006; assessment date 31 March 2009*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,111	23,522	896	14,375	4,663	1,613	17,539	7,266	70,985
Diphtheria, tetanus, pertussis (%)	96.0	94.7	96.1	94.5	94.0	95.0	95.7	93.8	94.8
Poliomyelitis (%)	96.1	94.6	96.1	94.5	94.0	94.9	95.7	93.7	94.8
Haemophilus influenzae type b (%)	95.9	94.8	94.5	93.8	92.8	95.0	94.6	93.6	94.3
Measles, mumps, rubella (%)	95.1	93.4	95.0	93.5	93.4	94.5	94.7	92.8	93.8
Hepatitis B (%)	96.3	95.5	97.2	95.4	94.9	96.0	96.3	94.4	95.5
Fully immunised (%)	93.9	92.3	93.8	92.2	91.9	93.4	93.6	90.9	92.5
Change in fully immunised since last quarter (%)	-0.6	-0.4	+0.8	0.0	-0.8	-1.3	-0.2	+1.0	-0.2

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2008;32:289.

Table 3: Percentage of children immunised at 5 years of age,* preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2003; assessment date 31 March 2009

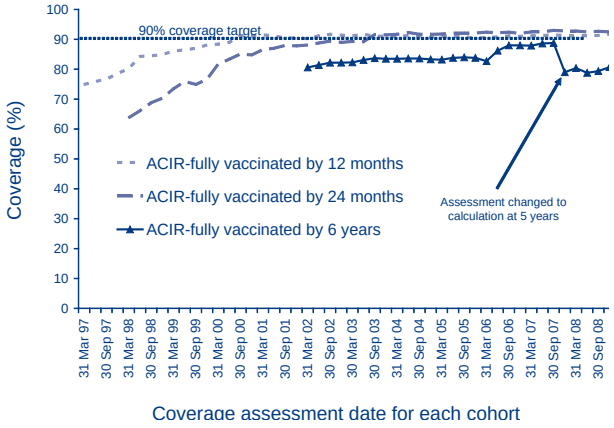
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,144	22,083	817	13,781	4,526	1,539	16,487	6,759	67,136
Diphtheria, tetanus, pertussis (%)	85.7	79.2	82.6	81.8	76.2	83.2	86.3	79.6	81.6
Poliomyelitis (%)	85.2	79.1	82.5	81.6	76.0	83.2	86.2	79.6	81.4
Measles, mumps, rubella (%)	85.1	78.8	82.6	81.4	75.9	82.6	86.0	79.4	81.2
Fully immunised (%)	84.4	78.4	81.9	80.9	75.4	82.2	85.6	78.6	80.7
Change in fully immunised since last quarter (%)	-0.8	+3.4	-2.9	-0.6	-0.0	+1.4	+1.8	-1.6	+1.3

Due to a calculation error by ACIR, the coverage estimates for the past 5 quarters for the 5-year age group have been incorrect. Assessment was made at 66 months rather than 60 months, which inflated the estimates. The age of assessment for vaccines due at 4 years of age makes a critical difference to coverage estimates for these vaccines. Corrected tables are published in the erratum at the end of the section.

Figure 5 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 6 years (5 years from March 2008), although cover-

age for vaccines due at 4 years decreases significantly due to the above-mentioned change in assessment age. It should also be noted that, currently, coverage for the vaccines added to the NIP since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months) are not included in the 12 or 24 months coverage data respectively.

Figure 5: Trends in vaccination coverage, Australia, 1997 to 31 December 2008, by age cohorts



Meningococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Meningococcal Surveillance Programme.

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence. For more information see Commun Dis Intell *2008;32:135.*

Laboratory confirmed cases of invasive meningococcal disease for the period I January to 31 March 2009, are included in this issue of Communicable Diseases Intelligence *(Table 4).*

Table 4: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2009, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	09													0	
	08													0	
New South Wales	09			12		3				1				16	
	08			4		1		1						6	
Northern Territory	09			2		1								3	
	08					1								1	
Queensland	09			11										11	
	08			16		2								18	
South Australia	09			4										4	
	08			2										2	
Tasmania	09													0	
	08													0	
Victoria	09			5		1						2		8	
	08			4										4	
Western Australia	09			2		2								4	
	08			3								1		4	
Total	09	0		36		7		0		1		2		46	
	08			30		4		1				1		36	

Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick NSW 2031 for the Australian Gonococcal Surveillance Programme.

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] *Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see* Commun Dis Intell *2008;32:134.*

Reporting period 1 January to 31 March 2009

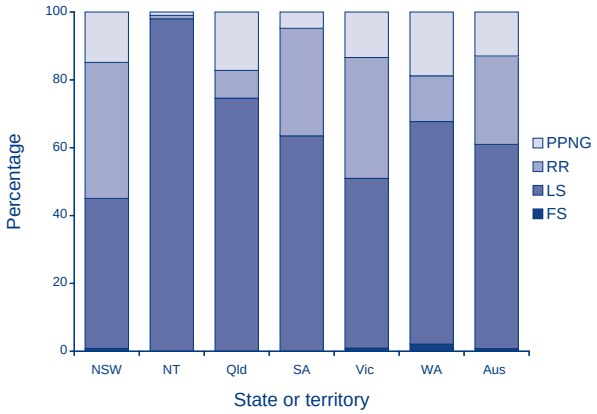
The AGSP laboratories received a total of 875 isolates in this quarter, of which 856 underwent susceptibility testing. This number was 76 more than the 799 isolates reported in this period in 2008. About 27% of this total was from New South Wales, 25% from Victoria, 16% from Queensland, 12% each from Western Australia and the Northern Territory and 7% from South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter, 336 (39%) of all isolates examined were penicillin resistant by one or more mechanisms. One hundred and eleven (13%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 223 (26%) penicillin resistant by chromosomal mechanisms, (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from 2% in the Northern Territory to 56% in New South Wales. In this quarter in 2008, 45% of isolates were penicillin resistant by any mechanism and in 2007, 39%. The decrease in penicillin resistant strains to 2007 proportions was the result of decreased numbers of gonococci with chromosomally mediated resistance.

Figure 6 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≤ 1 mg/L) or else PPNG, aggregated for Australia and by state 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 6: Categorisation of gonococci isolated in Australia, 1 January to 31 March 2009, 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 97 CMRP (41%) and 36 PPNG (15%). Victoria had 77 (36%) CMRP and 29 (13%) PPNG. Queensland had higher numbers of PPNG, 23 (17%), but fewer CMRP, 11 (8%). Western Australia also had higher numbers of PPNG 18 (19%) than CMRP, 13 (14%). One CMRP and 1 PPNG strain were found in the Northern Territory. Two CMRP and 1 PPNG were found in the Australian Capital Territory and 2 CMRP and no PPNG were reported from Tasmania. Of note was the decrease in penicillin resistant strains in South Australia in this quarter. This was a decrease to 36.5%, comprising 20 CMRP (31.75%) and 3 PPNG (4.75%). Corresponding proportions in 2008 were 5% PPNG and 70.7% CMRP.

Ceftriaxone

Ten isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, five in New South Wales, three in Queensland and two in South Australia. This is compared with eight nationally in the first quarter of 2008.

Spectinomycin

All isolates were susceptible to this injectable agent. This antibiotic is no longer available in Australia.

Quinolone antibiotics

The total number (397) and proportion (46%) of quinolone resistant *N. gonorrhoeae* (QRNG) was lower than data reported in recent quarters that reported high levels of resistance to this group of antibiotics (Figure 7). In the equivalent period in 2008, there were 415 (53%) QRNG. All but seven of the 397 QRNG detected in this quarter had ciprofloxacin MICs of 1 mg/L or more and 340 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. The highest number of QRNG was found in New South Wales (152) and this represented 64% of all isolates. One hundred and thirty-eight QRNG in Victoria also represented a high (64%) proportion of all isolates in that state. In Queensland, there were 41 (31%) QRNG, and 33 (34%) in Western Australia. The 23 (37%) QRNG in South Australia was a marked decrease in number compared with the 83 (84%) QRNG in the same quarter in 2008, and parallels the decrease in penicillin resistance also noted in that jurisdiction in this quarter. Six QRNG were detected in the Australian Capital Territory and two in Tasmania. A single QRNG was detected in the Northern Territory.

Figure 7: The distribution of quinolone resistant isolates of Neisseria gonorrhoeae in Australia, 1 January to 31 March 2009, 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 (157) and proportion (18%) of high level tetracycline resistance (TRNG) detected increased when compared with the 2008 data (135 TRNG, 17%). TRNG were found in all states and territories except Tasmania, and elsewhere represented between 2% (South Australia) and 33% 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 Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 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, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2009;33:83.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2008, as reported to 30 September 2008, and for 1 July to 30 September 2008, as reported to 30 December 2008, are included in this issue of Communicable Diseases Intelligence *(Tables 5, 6, 7 and 8).*

Table 5: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2008, 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 2008	This period 2007	YTD 2008	YTD 2007
HIV diagnoses	Female	1	11	1	13	3	0	14	8	51	32	78	64
	Male	0	83	3	49	7	1	65	16	224	231	464	483
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	94	4	62	10	1	79	24	275	263	542	547
AIDS diagnoses	Female	0	0	0	0	0	0	1	0	1	4	3	7
	Male	0	2	0	3	0	0	10	2	17	40	53	75
	Total*	0	2	0	3	0	0	11	2	18	45	56	83
AIDS deaths	Female	0	2	0	0	0	0	0	0	2	4	11	8
	Male	0	0	1	0	0	0	3	0	4	8	42	81
	Total*	0	2	1	0	0	0	3	0	6	12	53	90

* Totals include people whose sex was reported as transgender.

Table 6: Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2008, and reported by 30 September 2008, by sex and state or territory

	Sex	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	33	948	24	317	116	13	420	232	2,103
	Male	273	14,001	142	2,990	996	115	5,634	1,304	25,455
	Not reported	0	230	0	0	0	0	22	0	252
	Total*	306	15,207	166	3,316	1,113	128	6,098	1,543	27,867
AIDS diagnoses	Female	10	264	4	73	32	4	118	42	547
	Male	94	5,522	46	1,065	413	55	2,078	438	9,711
	Total*	104	5,804	50	1,140	446	59	2,209	482	10,294
AIDS deaths	Female	7	141	1	43	20	2	64	29	307
	Male	73	3,601	31	677	280	33	1,432	299	6,426
	Total*	80	3,753	32	722	300	35	1,505	329	6,756

* Totals include people whose sex was reported as transgender.

Table 7: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 July to 30 September 2008, 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 2008	This period 2007	YTD 2008	YTD 2007
HIV diagnoses	Female	0	10	1	6	0	0	8	5	30	36	107	101
	Male	1	71	1	43	12	0	61	12	201	210	667	696
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	81	2	49	12	0	69	17	231	247	774	798
AIDS diagnoses	Female	0	0	0	0	0	0	1	0	1	3	4	10
	Male	0	0	0	5	1	0	10	2	18	33	73	108
	Total*	0	0	0	5	1	0	11	2	19	36	77	119
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	3	3	7
	Male	0	1	0	0	0	1	4	0	6	10	18	33
	Total*	0	1	0	0	0	1	4	0	6	13	21	40

* Totals include people whose sex was reported as transgender.

Table 8: Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 September 2008, and reported by 31 December 2008, by sex and state or territory

	Sex	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	34	958	25	323	116	13	426	237	2,132
	Male	274	14,078	143	3,033	1,008	115	5,694	1,316	25,661
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	308	15,294	168	3,365	1,125	128	6,164	1,560	28,112
AIDS diagnoses	Female	10	265	4	74	32	4	119	42	550
	Male	94	5,524	46	1,076	414	55	2,093	439	9,741
	Total*	104	5,807	50	1,152	447	59	2,225	483	10,327
AIDS deaths	Female	7	141	1	43	20	2	64	29	307
	Male	73	3,601	31	677	280	33	1,432	299	6,426
	Total*	80	3,753	32	722	300	35	1,505	329	6,756

* Totals include people whose sex was reported as transgender.

ERRATUM: CHILDHOOD IMMUNISATION COVERAGE

Due to a calculation error by ACIR, the coverage estimates for the past 5 quarters for the 5-year age group have been incorrect. Assessment was made at 66 months rather than 60 months, which inflated the estimates. The age of assessment for vaccines due at 4 years of age makes a critical difference to coverage estimates for these vaccines. Corrected tables are reproduced below. The change in fully immunised status has not been calculated for Table 1.

Table 1: Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2002; assessment date 31 March 2008

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,062	22,069	876	13,108	4,356	1,488	16,088	6,570	65,617
Diphtheria, tetanus, pertussis (%)	86.7	77.2	80.8	81.5	75.2	80.9	83.8	78.3	79.9
Poliomyelitis (%)	86.5	76.9	80.8	81.3	74.8	80.6	83.7	78.0	79.7
Measles, mumps, rubella (%)	86.2	76.7	81.5	81.2	74.6	80.5	83.5	77.9	79.5
Fully immunised (%)	85.8	76.2	80.4	80.6	74.1	79.8	83.1	77.2	79.0

Table 2: 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 2003; assessment date 30 June 2008

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,028	21,494	942	13,525	4,293	1,420	15,502	6,615	64,819
Diphtheria, tetanus, pertussis (%)	87.6	80.2	81.6	82.5	73.5	81.1	85.1	77.9	81.3
Poliomyelitis (%)	87.2	79.9	81.6	82.3	73.5	80.9	85.0	77.6	81.1
Measles, mumps, rubella (%)	86.7	79.6	81.2	82.2	73.5	80.6	84.6	77.7	80.9
Fully immunised (%)	86.4	79.1	80.7	81.7	73.0	79.9	84.3	76.8	80.4
Change in fully immunised since last quarter (%)	+0.6	+2.9	+0.3	+1.1	-1.1	+0.2	+1.2	-0.3	+1.4

Table 3: Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2003; assessment date 30 September 2008

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children									
Diphtheria, tetanus, pertussis (%)	88.3	77.1	80.8	82.0	75.6	82.6	82.1	78.0	79.6
Poliomyelitis (%)	88.1	76.9	80.7	81.8	75.2	82.7	82.0	77.9	79.4
Measles, mumps, rubella (%)	87.6	76.6	80.8	81.6	75.2	82.3	81.7	77.7	79.2
Fully immunised (%)	87.4	76.2	80.2	81.2	74.8	82.0	81.3	77.0	78.8
Change in fully immunised since last quarter (%)	+1.0	-2.9	-0.5	-0.6	+1.8	+2.1	-3.1	+0.1	-1.7

Table 4: Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2003; assessment date 31 December 2008

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,106	23,056	815	14,277	4,807	1,494	16,903	6,706	69,164
Diphtheria, tetanus, pertussis (%)	85.8	75.7	85.5	82.3	76.0	81.6	84.6	81.2	80.2
Poliomyelitis (%)	86.0	75.7	85.4	82.2	75.9	81.5	84.5	81.0	80.1
Measles, mumps, rubella (%)	85.7	75.5	84.9	81.8	75.8	81.1	84.1	80.7	79.8
Fully immunised (%)	85.3	75.0	84.8	81.4	75.5	80.8	83.8	80.1	79.4
Change in fully immunised since last quarter (%)	-2.1	-1.2	+4.6	+0.3	+0.6	-1.2	+2.5	+3.2	+0.6