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Communicable Diseases Intelligence

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Annual reports

Australia's notifiable disease status, 2009: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

In 2009, 65 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 236,291 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, an increase of 48% on the number of notifications in 2008. This increase was largely due to cases of influenza A(H1N1) pandemic 2009. In 2009, the most frequently notified diseases were vaccine preventable diseases (101,627 notifications, 43% of total notifications), sexually transmissible infections (73,399 notifications, 31% of total notifications), and gastrointestinal diseases (31,697 notifications, 13% of total notifications). There were 18,861 notifications of bloodborne diseases; 8,232 notifications of vectorborne diseases; 1,919 notifications of other bacterial infections; 552 notifications of zoonoses and 4 notifications of quarantinable diseases. *Commun Dis Intell* 2011;35(2):61–131.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2009, 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] 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. Under the Agreement, in 2009 states and territories forwarded de-identified data on the nationally agreed set of 65 communicable diseases to the Department of Health and Ageing for the purposes of national communicable disease surveillance, although not all 65 diseases were notifiable in each jurisdiction. Data were renewed electronically from states and territories, daily or several times a week. The system was complemented by other surveillance systems, which provided enhanced information on various diseases, including four that are not reported to the National Notifiable Diseases Surveillance System (NNDSS).

In 2009, the NNDSS core dataset included the following 5 mandatory data fields: unique record reference number; notifying state or territory; disease code; confirmation status and the date when the public health unit was notified (notification receive date).

In addition, the following core but non-mandatory data fields were supplied where possible: date of birth; age at onset; sex; Indigenous status; postcode of residence; disease onset date; date when the medical practitioner signed the notification form (notification date), death status, date of specimen collection and outbreak reference number (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case were collected and reported to NNDSS. Data quality was monitored by the Office of Health Protection and the National Surveillance Committee (NSC) and there was a continual process of improving the national consistency of communicable disease surveillance through the daily, fortnightly and quarterly review of these data.

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

Newly diagnosed HIV infection and AIDS were notifiable conditions in each state or territory health jurisdiction in 2009 and were forwarded to the National HIV Registry and National AIDS Registry at the Kirby Institute (formerly known as the National Centre in HIV Epidemiology and Clinical Research). Further information can be found in the Kirby Institute'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.

Notification rates for each notifiable disease were calculated using the estimated 2009 mid-year resident population supplied by the Australian Bureau of Statistics[7] (ABS) (Appendix 1 and Appendix 2). Where diseases were not notifiable in a state or territory, national rates were adjusted by excluding the population of that jurisdiction from the denominator. For some diseases, age adjusted rates were calculated using either the direct method of standardisation for gastrointestinal diseases, or indirect method for sexually transmissible infections, with 2006 Census data as the standard population, (Map 1, Table 1).[8] The Northern Territory was represented by Statistical Subdivisions (SSD) and in the case of Greater Darwin, by the combination of the Tiwi Islands, Darwin, Palmerston and Litchfield SSD. This combination helped preserve confidentiality while improving legibility at the printed map scale. The geocode 77777 for Greater Darwin is nominal.

Notifications were summed by the postcode weighting calculated by the ABS Postcode Concordance.[9] These ABS concordance data were used to proportionally allocate notifications into SDs/SSDs according to the percentage of the population of the postcode living in the region. The total notifications per region are displayed in the relevant area.

Disease rates were calculated per 100,000 population for the relevant areas using ABS population data.[7] Rates were mapped for different SDs and ordered into five groups using the Jenks Natural Breaks method whereby the largest breaks between natural clusters of ordered data were identified and used as class boundaries. A class '0' was added to account for areas with no notifications, for a total of six rate classes per map. Note that the classification is data dependent and changes from map to map.

Notes on interpretation

The present report is based on 2009 'finalised' data from each state or territory agreed upon in July 2010 and represents a snap shot of the year after duplicate records and incorrect or incomplete data were removed. Therefore, numbers in this report may vary slightly from the numbers 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 often represent a proportion (the 'notified fraction') of the total incidence (Figure 1) and this has to be taken into account when interpreting NNDSS data. Moreover, the notified fraction varies by disease, by jurisdiction and by time.

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

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 2009 data.

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

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 of data completeness was defined as:

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

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

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

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

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

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

9=Not stated

Figure 1: Communicable diseases notifiable fraction

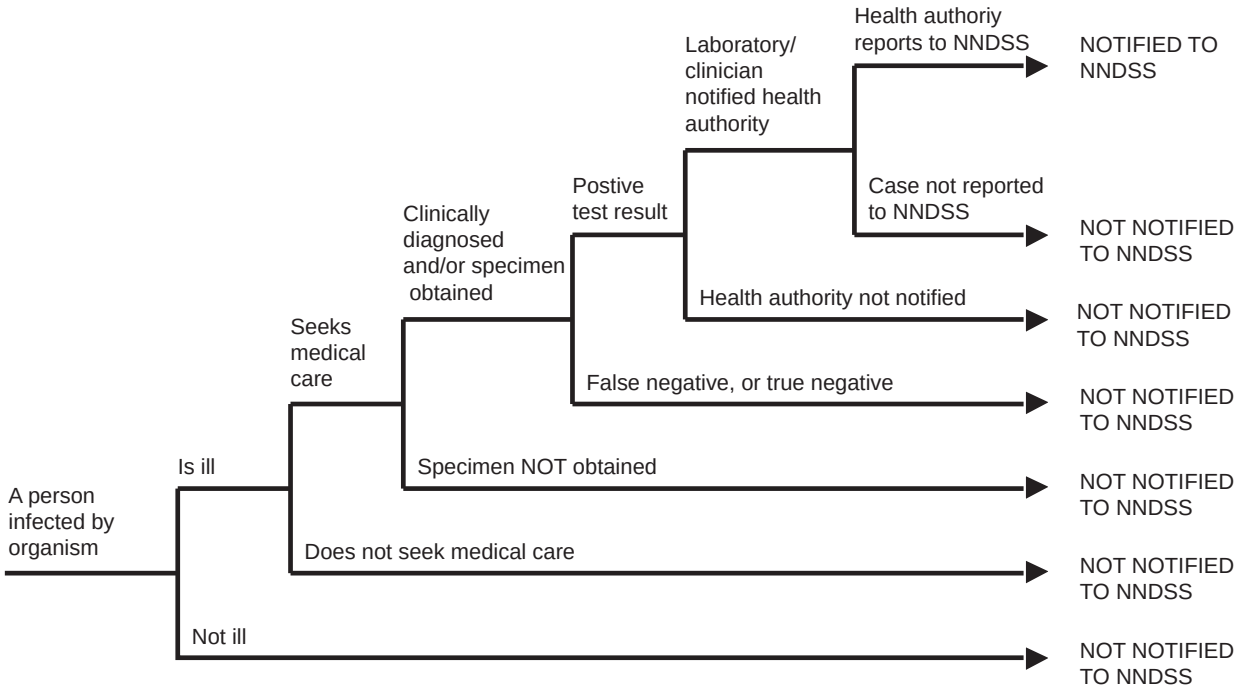
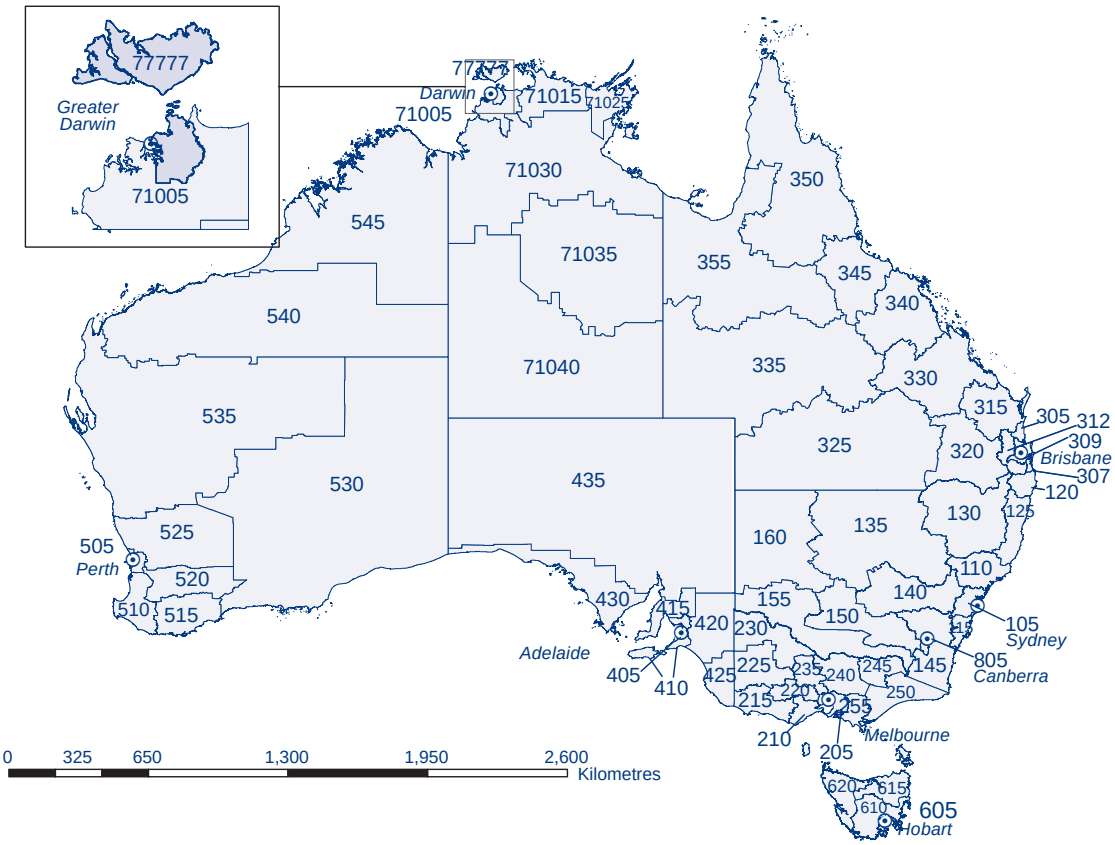


Table 1: Australian population by Statistical Division and Statistical Subdivision for the Northern Territory, 2009

SD code	Statistical Division	Population
Australian Capital Territory		
805	Canberra	351,868
New South Wales		
105	Sydney	4,504,469
110	Hunter	644,279
115	Illawarra	431,160
120	Richmond–Tweed	241,954
125	Mid-North Coast	309,588
130	Northern	184,822
135	North Western	118,535
140	Central West	183,157
145	South Eastern	216,593
150	Murrumbidgee	158,593
155	Murray	118,540
160	Far West	22,731
Northern Territory (Subdivisions)		
71005	Finniss	2,865
71015	Alligator	6,806
71025	East Arnhem	16,070
71030	Lower Top End NT	23,868
71040	Central NT	40,967
77777	Greater Darwin	127,285
Queensland		
305	Brisbane	2,004,262
307	Gold Coast	515,157
309	Sunshine Coast	323,423
312	West Moreton	94,660
315	Wide Bay–Burnett	287,425
320	Darling Downs	237,211
325	South West	26,277
330	Fitzroy	220,714
335	Central West	12,270
340	Mackay	172,735
345	Northern	227,340
350	Far North	269,650
355	North West	33,979
South Australia		
405	Adelaide	1,187,466
410	Outer Adelaide	136,623
415	Yorke and Lower North	47,052
420	Murray Lands	70,426
425	South East	65,978
430	Eyre	35,556
435	Northern	80,489
Tasmania		
605	Greater Hobart	212,019
610	Southern	37,456
615	Northern	141,434
620	Mersey–Lyell	112,383
Victoria		
205	Melbourne	3,995,537
210	Barwon	285,096
215	Western District	106,268
220	Central Highlands	155,585
225	Wimmera	50,878
230	Mallee	94,736
235	Loddon	183,659
240	Goulburn	210,114
245	Ovens–Murray	99,872
250	East Gippsland	86,812
255	Gippsland	174,671
Western Australia		
505	Perth	1,658,992
510	South West	246,202
515	Lower Great Southern	58,851
520	Upper Great Southern	19,169
525	Midlands	55,730
530	South Eastern	58,727
535	Central	64,849
540	Pilbara	47,528
545	Kimberley	35,009
Other territories		–
Australia	**Total**	**21,944,741**

Source: Australian Bureau of Statistics. Population by Age and Sex, Regions of Australia, 2009; 2010. ABS Catalogue: 3235.0.[8]

Map 1: Australian Bureau of Statistics Statistical Division codes, Australia, and Statistical Subdivision codes, Northern Territory, 2009



Notes on cases definitions

Each notifiable disease is governed by a national surveillance case definition for reporting to the 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. These case definitions are reviewed by the Case Definitions Working Group* (CDWG) and seeks to be consistent with the Public Health Laboratory Network laboratory case definitions.

The national surveillance case definitions and their review status are available from http://www.health.gov.au/casedefinitions

Results

There were 236,291 communicable disease notifications received by NNDSS in 2009 (Table 3).

In 2009, the most frequently notified diseases were vaccine preventable diseases (101,627 notifications, 43.0% of total notifications), sexually transmissible infections (73,399 notifications, 31.1% of total notifications), and gastrointestinal diseases (31,697 notifications, 13.4% of total notifications).

There were 18,861 notifications of bloodborne diseases; 8,232 notifications of vectorborne diseases; 1,919 notifications of other bacterial infections; 552 notifications of zoonoses and 4 notifications of quarantinable diseases. In 2009, the total number of notifications was the highest recorded in NNDSS

Table 3: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2009, by disease category rank order

Disease category	Number	%
Vaccine preventable diseases	101,627	43.0
Sexually transmitted infections	73,399	31.1
Gastrointestinal diseases	31,697	13.4
Bloodborne diseases	18,861	8.0
Vectorborne diseases	8,232	3.5
Other bacterial diseases	1,919	0.8
Zoonoses	552	0.2
Quarantinable diseases	4	0.0
Total	236,291	100.0

* The Case Definitions Working Group is a working group of the Communicable Diseases Network Australia.

Table 2: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2009

Disease	Data received from
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions, except Western Australia
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions, except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions, except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infections	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions, except South Australia
Syphilis – congenital	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella – congenital	All jurisdictions
Tetanus	All jurisdictions

Table 2 *continued*: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2009

Disease	Data received from
Vaccine preventable diseases, *continued*	
Varicella zoster (chickenpox)	All jurisdictions, except NSW
Varicella zoster (shingles)	All jurisdictions, except NSW
Varicella zoster (unspecified)	All jurisdictions, except NSW
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 disease (invasive)	All jurisdictions
Tuberculosis	All jurisdictions

No new diseases were added to the disease list in 2009.

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

NEC Not elsewhere classified.

since the surveillance system commenced data collection in 1991. There was an increase of 48% compared with notifications in 2008 (Figure 2). This increase was largely due to cases of influenza A(H1N1) pandemic 2009.

Notifications and notification rates per 100,000 population for each disease by state or territory, in 2009, are shown in Table 4 and Table 5 respectively. Trends in notifications and rates per 100,000 population for the period 2004 to 2009 are shown in Table 6.

The year in which diseases became notifiable to NNDSS in each jurisdiction is shown in Table 7.

Figure 2: Notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2009, by year of diagnosis

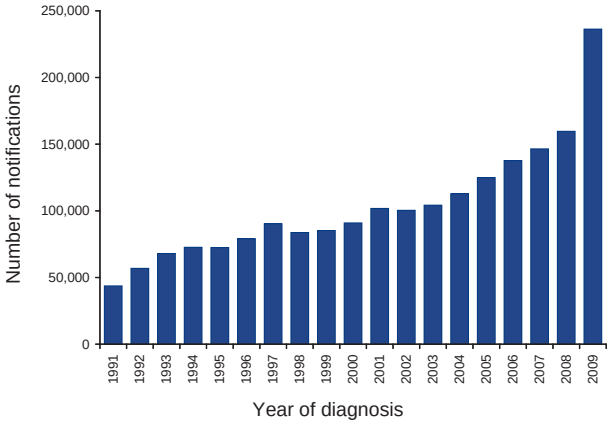


Table 4: Notifications of communicable diseases, Australia, 2009, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0
Hepatitis B (newly acquired)*	5	36	4	49	9	8	88	39	238
Hepatitis B (unspecified)†	101	2,651	152	1,022	447	77	1,948	709	7,107
Hepatitis C (newly acquired)*	7	41	5	NN	45	21	188	94	401
Hepatitis C (unspecified)†,‡	158	3,913	160	2,709	503	262	2,322	1,054	11,081
Hepatitis D	0	9	0	13	0	0	12	0	34
Gastrointestinal diseases									
Botulism	0	0	0	1	0	0	0	0	1
Campylobacteriosis§	357	NN	205	4,610	1,755	626	5,838	2,582	15,973
Cryptosporidiosis	106	1,463	150	1,460	106	66	1,039	235	4,625
Haemolytic uraemic syndrome	0	4	0	2	4	0	2	0	12
Hepatitis A	6	98	1	56	59	5	303	35	563
Hepatitis E	0	17	0	3	0	0	8	5	33
Listeriosis	2	26	0	14	4	3	27	15	91
Salmonellosis	225	2,736	487	2,471	681	166	1,647	1,120	9,533
Shigellosis	8	156	85	115	51	2	85	120	622
STEC,VTEC‖	0	21	1	23	62	0	16	6	130
Typhoid fever	2	47	0	13	2	1	42	8	115
Quarantinable diseases									
Cholera	0	3	0	0	0	0	1	0	4
Human 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¶,**	941	14,948	2,115	16,721	3,757	1,453	13,889	8,836	62,660
Donovanosis	0	0	0	1	0	0	0	0	1
Gonococcal infection**	55	1,655	1,504	1,570	400	21	1,515	1,339	8,059
Syphilis – all**,††	33	910	137	475	53	28	858	182	2,676
Syphilis <2 years duration**	11	522	38	179	53	10	390	88	1,291
Syphilis >2 years or unspecified duration†,**	22	388	99	296	NDP	18	468	94	1,385
Syphilis – congenital**	0	0	3	0	0	0	0	0	3
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	6	0	6	1	0	2	4	19
Influenza (laboratory confirmed)	1,259	12,393	1,967	18,363	10,752	1,305	6,990	5,533	58,562
Measles	1	19	1	32	3	2	36	10	104
Mumps	0	40	13	34	12	1	45	20	165
Pertussis	351	12,436	215	6,216	5,346	616	3,778	778	29,736
Pneumococcal disease (invasive)	29	477	86	270	145	35	368	149	1,559
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	0	7	0	6	3	0	6	5	27
Rubella – congenital	0	0	0	0	0	0	0	0	0
Tetanus	0	2	0	0	0	0	1	0	3

Table 4, *continued*: Notifications of communicable diseases, Australia, 2009, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *continued*									
Varicella zoster (chickenpox)	2	NN	87	153	475	34	530	318	1,599
Varicella zoster (shingles)	12	NN	112	259	1,045	117	575	539	2,659
Varicella zoster (unspecified)	66	NN	3	3,835	280	80	1,847	866	6,977
Vectorborne diseases									
Arbovirus infection (NEC)	0	0	0	23	0	0	3	0	26
Barmah Forest virus infection	3	359	117	799	36	3	15	154	1,486
Dengue virus infection	17	132	27	1,036	17	2	38	133	1,402
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0
Kunjin virus infection‡‡	0	0	1	1	0	0	0	0	2
Malaria	3	92	14	185	32	5	113	82	526
Murray Valley encephalitis virus infection‡‡	0	0	1	1	0	0	0	2	4
Ross River virus infection	2	912	427	2,154	326	29	85	851	4,786
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australia bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	4	0	22	2	0	3	1	32
Leptospirosis	2	18	4	110	0	0	11	1	146
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	22	0	0	3	0	38	2	65
Q fever	0	139	3	131	9	0	25	2	309
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial diseases									
Legionellosis	4	94	3	56	44	0	50	51	302
Leprosy	0	0	0	2	0	0	1	0	3
Meningococcal infection§§	2	96	6	60	22	3	42	28	259
Tuberculosis	23	488	28	218	58	9	419	112	1,355
Total	**3,782**	**56,470**	**8,124**	**65,300**	**26,550**	**4,980**	**44,849**	**26,020**	**236,075**

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

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

‡ In Queensland, includes incident hepatitis C cases.

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

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

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

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Does not include congenital syphilis.

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

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

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Table 5: Notification rates for nationally notifiable communicable diseases, Australia, 2009, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	1.4	0.5	1.8	1.1	0.6	1.6	1.6	1.7	1.1
Hepatitis B (unspecified)†	28.8	37.3	67.6	23.2	27.5	15.3	35.9	31.7	32.5
Hepatitis C (newly acquired)*	2.0	0.6	2.2	NN	2.8	4.2	3.5	4.2	2.3
Hepatitis C (unspecified)†,‡	45.0	55.1	71.2	61.5	31.0	52.1	42.8	47.1	50.7
Hepatitis D	0.0	0.1	0.0	0.3	0.0	0.0	0.2	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	101.7	NN	91.2	104.6	108.2	124.5	107.6	115.4	108.1
Cryptosporidiosis	30.2	20.6	66.7	33.1	6.5	13.1	19.1	10.5	21.1
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.2	0.0	0.0	0.0	0.1
Hepatitis A	1.7	1.4	0.4	1.3	3.6	1.0	5.6	1.6	2.6
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.1	0.2	0.2
Listeriosis	0.6	0.4	0.0	0.3	0.2	0.6	0.5	0.7	0.4
Salmonellosis	64.1	38.5	216.6	56.1	42.0	33.0	30.3	50.1	43.6
Shigellosis	2.3	2.2	37.8	2.6	3.1	0.4	1.6	5.4	2.8
STEC,VTEC‖	0.0	0.3	0.4	0.5	3.9	0.0	0.3	0.3	0.6
Typhoid fever	0.6	0.7	0.0	0.3	0.1	0.2	0.8	0.4	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Human 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¶,**	268.0	210.5	940.6	379.4	231.5	289.1	255.9	395.0	286.4
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	15.7	23.3	668.9	35.6	24.7	4.2	27.9	59.9	36.8
Syphilis – all**,††	9.4	12.8	60.9	10.8	3.3	5.6	15.8	8.1	12.2
Syphilis <2 years duration**	3.1	7.4	16.9	4.1	3.3	2.0	7.2	3.9	5.9
Syphilis >2 years or unspecified duration†,**	6.3	5.5	44.0	6.7	NDP	3.6	8.6	4.2	6.8
Syphilis – congenital**	0.0	0.0	1.3	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.1	0.0	0.0	0.2	0.1
Influenza (laboratory confirmed)	358.5	174.6	874.8	416.7	662.6	259.6	128.8	247.4	267.7
Measles	0.3	0.3	0.4	0.7	0.2	0.4	0.7	0.4	0.5
Mumps	0.0	0.6	5.8	0.8	0.7	0.2	0.8	0.9	0.8
Pertussis	99.9	175.2	95.6	141.1	329.4	122.6	69.6	34.8	135.9
Pneumococcal disease (invasive)	8.3	6.7	38.2	6.1	8.9	7.0	6.8	6.7	7.1
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	0.1	0.2	0.0	0.1	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	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 5 *continued*: Notification rates for nationally notifiable communicable diseases, Australia, 2009, by state or territory

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Varicella zoster (chickenpox)	0.6	NN	38.7	3.5	29.3	6.8	9.8	14.2	10.8
Varicella zoster (shingles)	3.4	NN	49.8	5.9	64.4	23.3	10.6	24.1	18.0
Varicella zoster (unspecified)	18.8	NN	1.3	87.0	17.3	15.9	34.0	38.7	47.2
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.5	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	0.9	5.1	52.0	18.1	2.2	0.6	0.3	6.9	6.8
Dengue virus infection	4.8	1.9	12.0	23.5	1.0	0.4	0.7	5.9	6.4
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	0.4	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	0.9	1.3	6.2	4.2	2.0	1.0	2.1	3.7	2.4
Murray Valley encephalitis virus infection‡‡	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1	0.0
Ross River virus infection	0.6	12.8	189.9	48.9	20.1	5.8	1.6	38.0	21.9
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.5	0.1	0.0	0.1	0.0	0.1
Leptospirosis	0.6	0.3	1.8	2.5	0.0	0.0	0.2	0.0	0.7
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.3	0.0	0.0	0.2	0.0	0.7	0.1	0.3
Q fever	0.0	2.0	1.3	3.0	0.6	0.0	0.5	0.1	1.4
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	1.1	1.3	1.3	1.3	2.7	0.0	0.9	2.3	1.4
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection§§	0.6	1.4	2.7	1.4	1.4	0.6	0.8	1.3	1.2
Tuberculosis	6.5	6.9	12.5	4.9	3.6	1.8	7.7	5.0	6.2

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

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

‡ In Queensland, includes incident hepatitis C cases.

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

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

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

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Does not include congenital syphilis.

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

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

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Table 6: Notifications and notification rate for communicable diseases, Australia, 2004 to 2009, (per 100,000 population)

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2004	2005	2006	2007	2008	2009			2004	2005	2006	2007	2008	2009
Bloodborne diseases														
Hepatitis (NEC)	0	1	1	0	1	0	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	283	253	292	294	258	238	276.0	0.9	1.4	1.2	1.4	1.4	1.2	1.1
Hepatitis B (unspecified)†	5,641	6,264	6,224	6,847	6,518	7,107	6,298.8	1.1	28.0	30.7	30.1	32.5	30.4	32.5
Hepatitis C (newly acquired)*	457	379	442	384	362	401	404.8	1.0	2.8	2.3	2.7	2.3	2.1	2.3
Hepatitis C (unspecified)†‡	12,348	11,901	11,863	11,868	11,098	11,081	11,815.6	0.9	61.3	58.4	57.3	56.3	51.8	50.7
Hepatitis D	29	32	30	34	42	34	33.4	1.0	0.1	0.2	0.1	0.2	0.2	0.2
Gastrointestinal diseases														
Botulism	1	3	1	1	0	1	1.2	0.8	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	15,586	16,498	15,420	16,995	15,524	15,973	16,004.6	1.0	116.1	121.0	111.1	120.0	107.5	108.1
Cryptosporidiosis	1,676	3,213	3,200	2,810	2,003	4,625	2,580.4	1.8	8.3	15.8	15.5	13.3	9.3	21.1
Haemolytic uraemic syndrome	16	20	14	19	31	12	20.0	0.6	0.1	0.1	0.1	0.1	0.1	0.1
Hepatitis A	319	327	281	165	277	563	273.8	2.1	1.6	1.6	1.4	0.8	1.3	2.6
Hepatitis E	28	30	24	18	44	33	28.8	1.1	0.1	0.1	0.1	0.1	0.2	0.2
Listeriosis	67	54	61	50	68	91	60.0	1.5	0.3	0.3	0.3	0.2	0.3	0.4
Salmonellosis	7,841	8,422	8,252	9,529	8,303	9,533	8,469.4	1.1	39.0	41.3	39.9	45.2	38.7	43.6
Shigellosis	520	729	546	600	829	622	644.8	1.0	2.6	3.6	2.6	2.8	3.9	2.8
STEC, VTEC‖	49	86	70	106	106	130	83.4	1.6	0.2	0.4	0.3	0.5	0.5	0.6
Typhoid fever	73	52	77	90	105	115	79.4	1.5	0.4	0.3	0.4	0.4	0.5	0.5
Quarantinable diseases														
Cholera	5	3	3	4	4	4	3.8	1.1	0.0	0.0	0.0	0.0	0.0	0.0
Human pathogenic avian influenza in humans	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 6 *continued*: Notifications and notification rate for communicable diseases, Australia, 2004 to 2009, (per 100,000 population)

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2004	2005	2006	2007	2008	2009			2004	2005	2006	2007	2008	2009
Sexually transmissible infections														
Chlamydial infection[¶, **]	36,169	41,293	47,425	52,009	58,449	62,660	47,069.0	1.3	179.7	202.5	229.1	246.8	272.7	286.4
Donovanosis	10	13	6	3	2	1	6.8	0.1	0.0	0.1	0.0	0.0	0.0	0.0
Gonococcal infection**	7,170	8,070	8,565	7,685	7,655	8,059	7,829.0	1.0	35.6	39.6	41.4	36.5	35.7	36.8
Syphilis – all**,††	2,065	1,934	2,197	2,758	2,674	2,676	2,325.6	1.2	10.3	9.5	10.6	13.1	12.5	12.2
Syphilis <2 years duration**	628	653	883	1,412	1,310	1,291	977.2	1.3	3.1	3.2	4.3	6.7	6.1	5.9
Syphilis >2 years or unspecified duration**	1,437	1,281	1,314	1,346	1,364	1,385	1,348.4	1.0	7.1	6.8	6.9	6.9	6.9	6.8
Syphilis – congenital**	13	17	13	7	6	3	11.2	0.3	0.1	0.1	0.1	0.0	0.0	0.0
Vaccine preventable diseases														
Diphtheria	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	15	17	22	17	25	19	19.2	1.0	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)[‡‡]	2,135	4,557	3,254	10,445	9,130	58,562	5,904.2	10.0	10.6	22.3	15.7	49.6	42.6	267.7
Measles	44	10	125	12	65	104	51.2	2.1	0.2	0.0	0.6	0.1	0.3	0.5
Mumps	102	240	275	586	285	165	297.6	0.6	0.5	1.2	1.3	2.8	1.3	0.8
Pertussis	8,748	11,164	9,764	4,862	14,285	29,736	9,764.6	3.0	43.5	54.7	47.2	23.1	66.7	135.9
Pneumococcal disease (invasive)	2,372	1,692	1,453	1,479	1,634	1,559	1,726.0	0.9	11.8	8.3	7.0	7.0	7.6	7.1
Poliomyelitis	0	0	0	1	0	0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	31	29	59	34	36	27	37.8	0.7	0.2	0.1	0.3	0.2	0.2	0.1
Rubella – congenital	1	1	0	2	0	0	0.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	6	2	3	3	4	3	3.6	0.8	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)[§§]	NN	16	1,558	1,668	1,795	1,599	1,259.3	1.3	NN	0.2	17.8	18.6	19.7	10.8
Varicella zoster (shingles)[§§]	NN	7	1,092	1,561	2,309	2,659	1,242.3	2.1	NN	0.1	12.5	17.4	25.3	18.0
Varicella zoster (unspecified)[§§]	NN	141	3,678	4,286	4,415	6,977	3,130.0	2.2	NN	1.6	42.0	47.9	48.3	47.2
Vectorborne diseases														
Arbovirus infection (NEC)[‖‖]	66	28	32	24	26	26	35.2	0.7	0.3	0.1	0.2	0.1	0.1	0.1
Barmah Forest virus infection	1,100	1,322	2,133	1,715	2,097	1,486	1,673.4	0.9	5.5	6.5	10.3	8.1	9.8	6.8
Dengue virus infection	351	220	189	316	562	1,402	327.6	4.3	1.7	1.1	0.9	1.5	2.6	6.4
Japanese encephalitis virus infection	1	0	0	0	1	0	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection[¶¶]	6	1	3	1	1	2	2.4	0.8	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	545	817	770	568	529	526	645.8	0.8	2.7	4.0	3.7	2.7	2.5	2.4
Murray Valley encephalitis virus infection[¶¶]	1	2	1	0	2	4	1.2	3.3	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	4,205	2,538	5,544	4,202	5,652	4,786	4,428.2	1.1	20.9	12.4	26.8	19.9	26.4	21.9

Table 6 *continued*: Notifications and notification rate for communicable diseases, Australia, 2004 to 2009, (per 100,000 population)

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2004	2005	2006	2007	2008	2009			2004	2005	2006	2007	2008	2009
Zoonoses														
Anthrax	0	0	1	1	0	0	0.4	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	0.0	0.0
Brucellosis	38	41	51	38	47	32	43.0	0.7	0.2	0.2	0.2	0.2	0.2	0.1
Leptospirosis	177	129	145	108	112	146	134.2	1.1	0.9	0.6	0.7	0.5	0.5	0.7
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	239	164	165	93	102	65	152.6	0.4	1.2	0.8	0.8	0.4	0.5	0.3
Q fever	460	352	410	448	376	309	409.2	0.8	2.3	1.7	2.0	2.1	1.8	1.4
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	312	331	349	306	272	302	314.0	1.0	1.6	1.6	1.7	1.5	1.3	1.4
Leprosy	6	10	7	13	11	3	9.4	0.3	0.0	0.0	0.0	0.1	0.1	0.0
Meningococcal infection***	406	392	318	305	285	259	341.2	0.8	2.0	1.9	1.5	1.4	1.3	1.2
Tuberculosis	1,056	1,078	1,205	1,133	1,203	1,355	1,135.0	1.2	5.2	5.3	5.8	5.4	5.6	6.2
Total	**112,791**	**124,895**	**137,613**	**146,503**	**159,620**	**236,075**								

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

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

‡ In Queensland, includes incident hepatitis C cases.

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

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

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

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Does not include congenital syphilis.

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

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

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

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

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

NEC Not elsewhere classified.

NN Not notifiable.

Table 7: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Bloodborne diseases										
Hepatitis (NEC)	1991	1991	1991	1991	1991	1991	1991	NN	1991 to present	WA do not report
Hepatitis B (newly acquired)	1995	1993	1993	1994	1993	1993	1993	1994	1995 to present	ACT did not report 1994
Hepatitis B (unspecified)	1991	1991	2004	1994	1991	1991	1991	1991	1991 to present	
Hepatitis C (newly acquired)	1995	1993	2005	NN	1993	1995	1997	1995	1993 to present	All jurisdictions except Qld
Hepatitis C (unspecified)	1991	1991	1991	1991	1994	1991	1991	1993	1995 to present	Includes reports of incident hepatitis C, 1991 to 1994
Hepatitis D	1999	1999	1999	1997	1999	1999	1999	2001	1999 to present	WA did not report 1999–2000
Gastrointestinal diseases										
Botulism	1992	1998	1998	1997	1993	1992	1992	2001	1992 to present	State reporting started as shown
Campylobacteriosis	1991	NN	1991	1991	1991	1991	1991	1991	1991 to present	NSW do not report
Cryptosporidiosis	2001	2001	2001	1996	2001	2001	2001	2001	2001 to present	
Haemolytic uraemic syndrome	1999	1999	1999	1997	1999	1999	1999	1999	1999 to present	
Hepatitis A	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Hepatitis E	1999	1999	1999	1999	1999	1999	1999	2001	1999 to present	WA did not report 1999–2000
Listeriosis	1991	1991	1994	1991	1992	1991	1991	1991	1991 to present	SA did not report 1991 NT did not report 1991–1993
Salmonellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Shigellosis	1991	2001	1991	1997	1991	1991	1991	1991	1991 to present	NSW did not report 1991–2000 Qld did not report 1991–2006
STEC, VTEC	1999	1999	1999	2002	1999	1999	1999	2001	1999 to present	Qld did not report 1991–2002 WA did not report 1999–2001
Typhoid†	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Quarantinable diseases										
Cholera	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Highly pathogenic avian influenza in humans	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Plague	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rabies	1993	1997	1991	1991	1991	1991	1991	1991	1991 to present	
Severe acute respiratory syndrome	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Smallpox	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Viral haemorrhagic fever	1993	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Yellow fever	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Sexually transmissible infections										
Chlamydial infection (NEC)	1993	1991	1991	1991	1993	1991	1991	1993	1994 to present	NSW did not report 1994–1998
Donovanosis	1991	2002	1991	1991	2002	1993	1991	1991	1991 to present	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Gonococcal infection‡	1991	1993	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis – all§	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

Table 7 *continued*: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Sexually transmissible infections, *continued*										
Syphilis <2 years	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis >2 years or unspecified duration	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis – congenital	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Vaccine preventable diseases										
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Haemophilus influenzae type b	1991	1991	1991	1991	1991	1991	1991	1994	1991 to present	WA did not report 1991–1993
Influenza (laboratory confirmed)	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Measles	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Mumps	1992	1992	1995	1997–1998; 2002	1994	1995	1992	1994	1995 to present	Qld did not report (1995–1996 & 1999–2000)
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Pneumococcal disease (invasive)	2001	2001	2001	1997	2001	2001	2001	2001	2001 to present	
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rubella‖	1991	1991	1993	1991	1993	1995	1992	1994	1993 to present	Tasmania did not report 1993–1994
Rubella – congenital	2003	2003	2003	1997	2003	2003	2003	2003	2003 to present	
Tetanus	1991	1991	1991	1985	1991	1991	1991	1991	1991 to present	Qld did not report 1991–1993
Varicella zoster (chickenpox)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Varicella zoster (shingles)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Varicella zoster (unspecified)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Vectorborne diseases										
Barmah Forest virus infection	1995	1995	1997	1995	1995	1995	1995	1995	1995 to present	
Dengue virus infection	1993	1991	1991	1991	1991	1991	1991	1995	1991 to present	ACT did not report 1991–1992
Arbovirus infection (NEC)¶,**	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	Includes JEV, MVEV and Kunjin 1991–2000
Japanese encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Kunjin virus	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Reported under MVEV in ACT
Malaria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Murray Valley encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Combined with Kunjin in ACT
Ross River virus infection	1993	1993	1991	1991	1993	1993	1991	1991	1993 to present	
Zoonoses										
Anthrax	2001	2001	2001	1991	2002	2001	2001	2001	2001 to present	
Australian bat lyssavirus	2001	2001	2001	1998	2001	2001	2001	2001	2001 to present	
Brucellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

Table 7 *continued*: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

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

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

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

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

§ Includes syphilis – congenital from 1991 to 2002.

|| Includes rubella – congenital from 1991 to 2002.

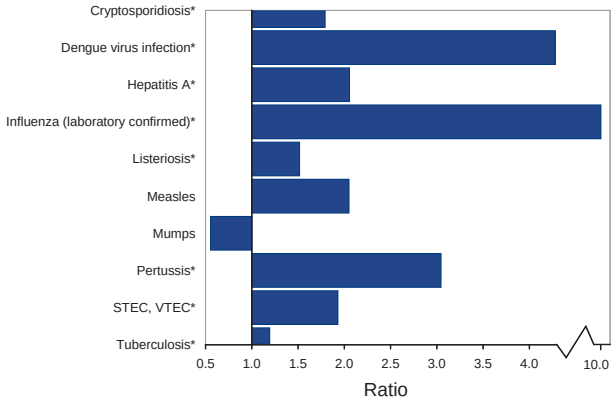
¶ Before 1997, includes Ross River virus infection, dengue virus infection and Barmah Forest virus infection.

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

NN Not notifiable

The major changes in communicable disease notifications in 2009 are shown in Figure 3 as the ratio of notifications in 2009 to the mean number of notifications for the previous 5 years. Notifications of dengue virus infection, influenza (laboratory confirmed), pertussis and hepatitis A were highest since 2004 and exceeded the expected range (5-year mean plus 2 standard deviations). Notifications of mumps, measles, tuberculosis and Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC) were within the historical range.

Figure 3: Comparison of total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2009, with the previous 5-year mean



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

Data completeness

The case's sex was complete in 99.7% of notifications and age at onset in close to 100% of notifications (Table 8). In 2009, Indigenous status was complete in 49.6% of notifications, and this varied by jurisdiction. Indigenous status was complete for 93.1% of data reported in the Northern Territory, 86.2% in Western Australia and 79.5% South Australia. Indigenous status was complete for less than 50% in the remaining jurisdictions.

Data completeness on Indigenous status also varied by disease (Appendix 3). There were 5 diseases for which notifications were 100% complete for Indigenous status.[10] A further 7 diseases equalled or exceeded 90% completeness for Indigenous status. Of the 18 priority diseases agreed to by CDNA and the NSC in 2009 for improving Indigenous identification, seven had an Indigenous completeness that

Table 8: Completeness of National Notifiable Diseases Surveillance System data received, Australia, 2009, by state or territory*

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total notifications	3,782	56,686	8,124	65,300	26,549	4,980	44,850	26,020	236,291
Sex									
Unknown/missing	1	395	12	27	3	8	318	2	766
Per cent complete	100.0	99.3	99.9	100.0	100.0	99.8	99.3	100.0	99.7
Age at onset									
Unknown/missing	0	6	8	0	1	2	74	1	88
Per cent complete	100.0	100.0	99.9	100.0	100.0	100.0	99.8	100.0	100.0
Indigenous status									
Unknown/missing	3,105	43,347	564	36,648	5,433	2,595	23,720	3,596	119,008
Per cent complete	17.9	23.5	93.1	43.9	79.5	47.9	47.1	86.2	49.6

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

exceeded 90% (donovanosis, *Haemophilus influenzae* type b, congenital syphilis, meningococcal infection, syphilis less than 2 years duration, tuberculosis and hepatitis A). The diseases for which there was less than 90% Indigenous completeness included dengue virus infection, gonococcal infection, leprosy, measles, pneumococcal disease (invasive), and shigellosis. In 2009, CDNA set target thresholds of 95% completeness for key diseases and 80% completeness for the remainder of the notifiable diseases.

Bloodborne diseases

In 2009, the bloodborne viruses reported to the NNDSS were hepatitis B, C, and D. Both hepatitis B and C cases are notified to the NNDSS as either 'newly acquired', where evidence was available that the infection was acquired within 24 months prior to diagnosis; or 'greater than 2 years or unspecified' period of infection. These categories were reported from all states and territories except Queensland where all cases of hepatitis C, including newly acquired, were reported as 'greater than 2 years or unspecified'. The determination of a case as 'newly acquired' is heavily reliant on public health follow-up, with the method and intensity of follow-up varying by jurisdiction and over time.

In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence or incidence. Testing policies[11] and screening programs, including the preferential testing of high risk populations such as persons in prison, injecting drug users and persons from countries with a high prevalence of hepatitis B or C, may contribute to these changes.

Information on exposure factors relating to the most likely source(s) or risk factors of infection for hepatitis B and C was reported in a subset of diagnoses of newly acquired infections. The collection of these enhanced data are also dependant on the level of public health follow-up, which is variable by jurisdiction and over time.

Further information regarding the surveillance of these infections are described within the hepatitis B and hepatitis C sections.

Notifications of HIV and AIDS diagnoses are reported directly to the Kirby Institute, which maintains the National HIV Registry and the National AIDS Registry. Information on national HIV/AIDS surveillance can be obtained from the Kirby Institute website at www.nchecr.unsw.edu.au

Hepatitis B

Hepatitis B notifications are classified as either 'newly acquired' (infection acquired within 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified). The classification of hepatitis B cases is primarily based on serological evidence or evidence of a previously negative test within the 24 months prior to diagnosis. In 2009, there were 7,345 notifications of hepatitis B (both newly acquired and unspecified), equating to a rate of 33.6 notifications per 100,000 population. Following a peak in hepatitis B notifications between 2000 and 2001 (41.3 and 39.9 per 100,000 population, respectively), the overall hepatitis B notification rate declined and remained relatively stable at around 32 notifications per 100,000 population between 2003 and 2009 (Figure 4). Of the jurisdictions, the

Northern Territory recorded the highest rate of hepatitis B notifications in 2009 (69.4 per 100,000 population), followed by New South Wales (37.8 per 100,000 population) and Victoria (37.5 per 100,000 population).

Since the introduction of the adolescent hepatitis B vaccination program for children aged 10–13 years in 1997 and the universal infant program in 2000,[12] there has been a general decline in overall hepatitis B notification rates, especially amongst the 15–19 and 20–29 years age groups. In 2009, one notification of newly acquired hepatitis B and 5 notifications of hepatitis B (unspecified) were reported in children in the 0–4 years age group, representing 0.4% and 0.1% of hepatitis notifications in these categories, respectively. Approximately 93% of the 2008 Australian birth cohort received the full course of the hepatitis B vaccine.[13–17]

Newly acquired hepatitis B notifications

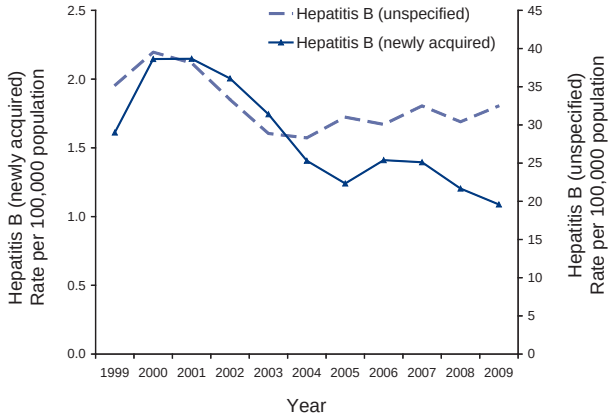
In 2009, 238 newly acquired hepatitis B notifications (1.1 per 100,000 population) were reported to the NNDSS, which was fewer than the number reported in 2008 (258 notifications; 1.2 per 100,000 population). The 2009 notification rate was the lowest since 1999, following a peak of 2.1 notifications per 100,000 population between 2000 and 2001 (Figure 4).

Nationally, the proportion of all hepatitis B notifications in 2009 that were documented as newly acquired cases was 3.2%, compared with 3.8% in 2008. The proportion of newly acquired infections compared with total hepatitis B infections varied substantially: Tasmania (9%); the Australian Capital Territory, Queensland and Western Australia (5%); Victoria (4%); the Northern Territory (3%); South Australia (2%); and New South Wales (1%). The highest notification rates of newly acquired hepatitis B infection were reported from the Northern Territory (1.8 per 100,000 population), closely followed by Western Australia (1.7 per 100,000 population) and Victoria and Tasmania (1.6 per 100,000 population). The identification and classification of newly acquired hepatitis B is reliant upon public health follow-up, the extent of which varies between jurisdictions and over time.

In 2009, the highest notification rate of newly acquired hepatitis B infection was observed in the 25–29 years age group amongst males (3.9 per 100,000 population) and in the 35–39 years age group amongst females (2.5 per 100,000 population) (Figure 5). Overall, notifications of newly acquired hepatitis B infection were higher amongst males, with a male to female ratio of 1.9:1.

Trends in newly acquired hepatitis B infection by year and age group are shown in Figure 6. Between 2000 and 2009, the notification rate of newly acquired hepatitis B fell substantially amongst persons aged 15–19 years (87%) and 20–29 years (71%). These trends occurred in both sexes.

Figure 4: Notification rate for newly acquired hepatitis B* and unspecified hepatitis B,† Australia, 1999 to 2009, by year‡

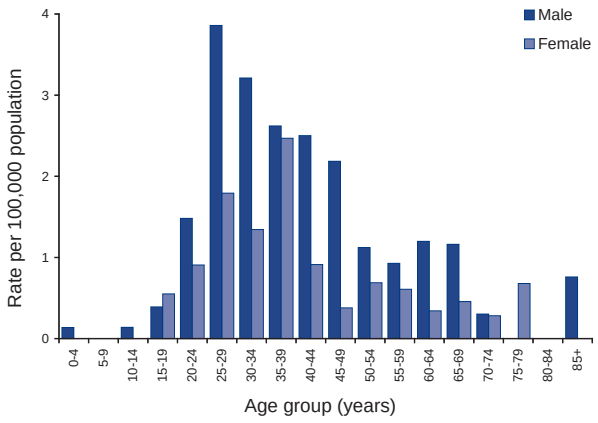


* Data for newly acquired hepatitis B for the Northern Territory (1999–2004) includes some unspecified hepatitis B cases.

† Data for unspecified hepatitis B for all jurisdictions except the Northern Territory between 1999 and 2004.

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

Figure 5: Notification rate for newly acquired hepatitis B, Australia, 2009, by age group and sex

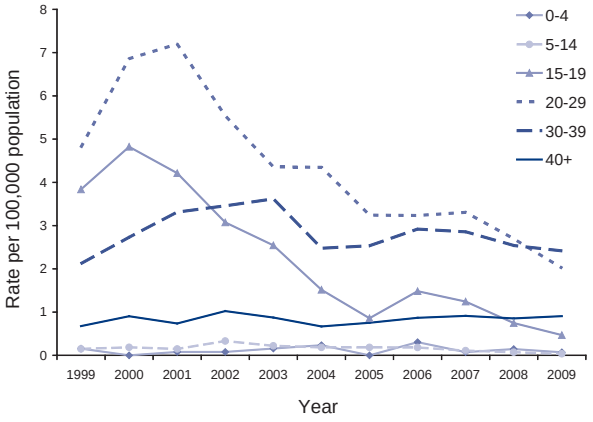


Of the 238 newly acquired hepatitis B notifications reported in 2009, the exposure history of 150 notifications from all jurisdictions except Queensland and Western Australia were assessed† (Table 9). In

† Prior to 2009 enhanced hepatitis B surveillance data were reported to the National Centre in HIV Epidemiology and Clinical Research from health authorities in jurisdictions.

2009, 59% of these notifications had at least one risk factor recorded, with the source of exposure not recorded or unable to be determined for the remainder of these cases. Between 2006 and 2009, the proportion of notifications associated with injecting drug use declined from 43%18 to 33%. The proportion of diagnoses reporting a history of heterosexual contact with a hepatitis B positive partner also decreased from 21% in 2005 to 12% in 2009. Additional information was also collected on the country of birth of newly acquired cases from all jurisdictions except Queensland. The majority of these newly acquired diagnoses occurred amongst Australian-born persons, and the proportion of overseas-born people with hepatitis B was similar to the proportion of overseas-born people in the Australian population.[18]

Figure 6: Notification rate for newly acquired hepatitis B,* Australia, 1999 to 2009, by age group and year



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

Table 9: Newly acquired hepatitis B notifications, selected jurisdictions,* 2009, by sex and exposure category†

Exposure category	Number of exposure factors reported†			Percentage‡ of notifications* (n = 150)
	Male	Female	Total	
Injecting drug use	32	18	50	33.3
Imprisonment	7	1	8	5.3
Skin penetration procedure	10	6	16	10.7
Tattoos	9	4	13	8.7
Ear or body piercing	–	2	2	1.3
Acupuncture	1	–	1	0.7
Healthcare exposure	7	5	12	8.0
Surgical Work	4	2	6	4.0
Major Dental Surgery	–	2	2	1.3
Blood/tissue recipient	2	1	3	2.0
Haemodialysis	1	–	1	0.7
Sexual contact – hepatitis B positive partner	14	9	23	15.3
Opposite sex	11	9	20	13.3
Same sex	3	–	3	2.0
Household contact	5	6	11	7.3
Needlestick or bio-hazardous injury§	1	–	1	0.7
Other	22	8	30	20.0
Sexual contact – unknown hepatitis B status partner‖	16	4	20	13.3
Notifications with at least one risk factor recorded	60	28	88	58.7
Risk factor unable to be determined	3	2	5	3.3
Unknown (not recorded)	36	21	57	38.0
Total number of exposure factors reported†	137	76	213	–
Total number of notifications*	99	51	150	–

* Notifications from the Australian Capital Territory, New South Wales, the Northern Territory, South Australia, Tasmania and Victoria.

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

‡ The denominator used to calculate the percentage is based on the total number of notifications from all jurisdictions, except Queensland and Western Australia. As more than one exposure category for each notification could be recorded, the total percentage does equate to 100%.

§ Includes both occupational and non-occupational exposures.

‖ Established through analysis of free text field.

Unspecified hepatitis B notifications

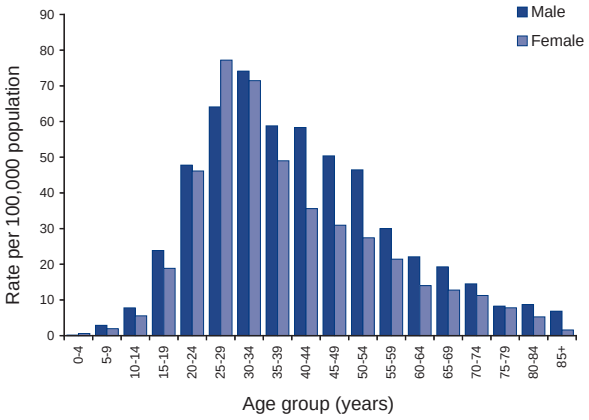
In 2009, a total of 7,107 notifications of unspecified hepatitis B infection were reported to the NNDSS, compared with 6,518 notifications in 2008. The Northern Territory recorded the highest notification rate (67.6 per 100,000 population), followed by New South Wales (37.3 per 100,000 population) and Victoria (35.9 per 100,000 population).

The notification rate of hepatitis B (unspecified) has declined by 23% since 2001 (39.5 per 100,000 population) with the lowest annual rate observed in 2004 (28.5 per 100,000 population) (Figure 4). Since 2001, there has been a slight upward trend in the notification rate for hepatitis B (unspecified), with a rate of 32.5 per 100,000 population observed in 2009.

In 2009, sex was recorded in 7,019 of the 7,107 notifications (99%). The male to female ratio of notifications was 1.2:1. Notification rates were highest in males aged 25 to 44 years, peaking in the 30–34 years age group (74.1 notifications per 100,000 population). Among females, notification rates were highest in the 25–29 years age group (77.2 per 100,000 population), followed by the 30–34 years age group (71.5 per 100,000 population) (Figure 7).

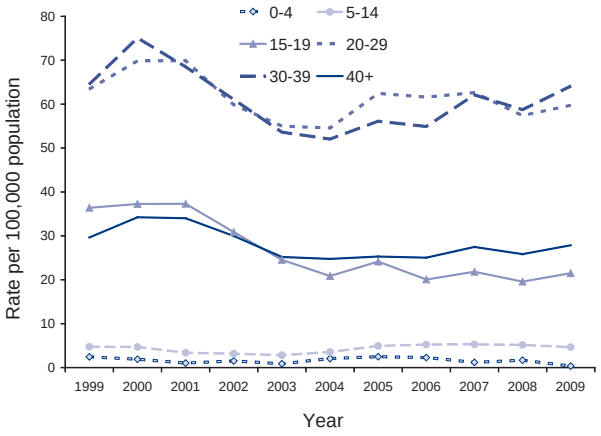
Trends in hepatitis B (unspecified) infection by year and age group are shown in Figure 8. Rates across most age groups were slightly higher in 2009 compared with 2008, with the 30–39 years age group increasing by 9.2% (58.7 to 64.1 notifications per 100,000 population). The highest notification rates continued to be observed amongst the 20–29 and 30–39 years age groups (59.7 and 64.1 per 100,000 population, respectively).

Figure 7: Notification rate for unspecified hepatitis B, Australia, 2009, by age group and sex*



* Excluding 94 cases whose sex or age were not reported.

Figure 8: Notification rate for unspecified hepatitis B,* Australia, 1999 to 2009, by age group and year



* Data for hepatitis B (unspecified) from all states except the Northern Territory between 1999 and 2004.

Hepatitis C

Hepatitis C notifications are classified as either 'newly acquired' (infection acquired within 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified). Current testing methods cannot distinguish between newly acquired (incident) and chronic infections (greater than 2 years or unspecified). The identification of newly acquired cases is therefore dependent on evidence of a negative test result within 24 months prior to laboratory diagnosis or clinical hepatitis within the 24 months prior to a positive diagnostic test where other causes of acute hepatitis have been excluded. Ascertainment of a person's hepatitis C testing and clinical history usually requires active follow-up by public health units.

Between 1999 and 2009, total hepatitis C notification rates declined by 49.5% (104 to 52.5 notifications per 100,000 population), with the greatest reductions observed between 1999 and 2002 (24% decline) (Figure 9). These reductions followed a peak in case notifications associated with the detection and accounting of prevalent cases that occurred in the late 1990s through the expansion of testing in high risk groups.[19] The continuing decline in the notification rate may be attributable to reductions in risk behaviours related to injecting drug use, especially amongst young people, and the implementation of needle exchange programs.[19,20]

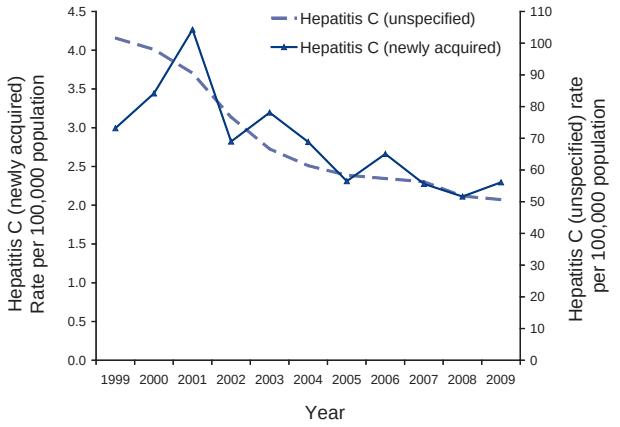
Although initial infection with the hepatitis C virus is asymptomatic or mildly symptomatic in more than 90% of cases, approximately 50%–80% of cases will go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.[21] In 2009, it

was estimated that 291,000 people living in Australia had been exposed to the hepatitis C virus. Of these, approximately 165,000 had chronic hepatitis C infection and early liver disease, 46,000 had chronic hepatitis C infection with moderate liver disease, 5,900 were living with hepatitis C related cirrhosis and 74,000 had cleared their infection.[18]

Newly acquired hepatitis C notifications

Notifications of newly acquired hepatitis C were reported from all jurisdictions except Queensland, where all cases of hepatitis C, regardless of whether they are newly acquired, are reported as unspecified. There were 401 newly acquired hepatitis C notifications reported in 2009 (362 notifications in 2008), giving a notification rate of 2.3 per 100,000 population (Figure 9).

Figure 9: Notification rate for newly acquired hepatitis C* and unspecified hepatitis C,† Australia, 1999 to 2009, by year



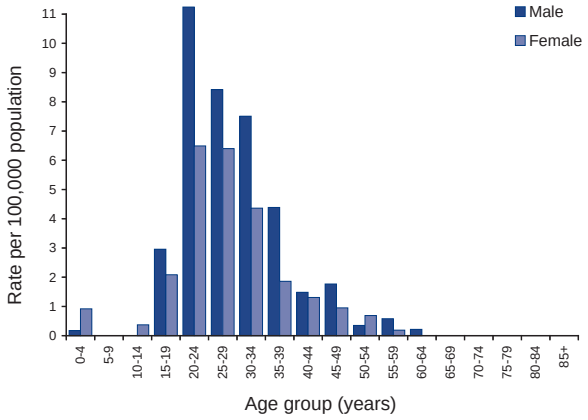
* Data for newly acquired hepatitis C from all states and territories except Queensland 1999 to 2009 and the Northern Territory 1999 to 2002.

† Data for unspecified hepatitis C provided from Queensland (1999–2009) and the Northern Territory (1999–2002) includes both newly acquired and unspecified hepatitis C cases.

Of all hepatitis C notifications in 2009, 3.5% were identified as newly acquired infections, which is comparable to previous years. Amongst jurisdictions, the proportion of newly acquired infections compared with total hepatitis C diagnoses varied substantially, with 8% in South Australia, Western Australia and Victoria; 7% in Tasmania; 4% in the Australian Capital Territory; 3% in the Northern Territory, and 1% in New South Wales. The highest rates of newly acquired hepatitis C infection were reported in Tasmania and Western Australia (4.2 per 100,000 population). The identification and classification of newly acquired hepatitis C is reliant upon public health follow-up to identify testing and clinical histories. The method and extent of case follow-up, and the population groups targeted, vary among jurisdictions, with newly acquired infection more likely to be detected in population groups that are tested frequently, such as those in prison settings.

Notification rates of newly acquired hepatitis C were highest in males in the 20–24 years age group followed by the 25–29 and 30–34 years age groups (11.2, 8.4 and 7.5 per 100,000 population, respectively). Peaks in the female population occurred in the 20–24 and 25–29 years age groups at around 6.5 notifications per 100,000 population (Figure 10).

Figure 10: Notification rate for newly acquired hepatitis C, Australia,* 2009, by age group and sex



* Data from all states and territories except Queensland.

Trends in the age distribution of newly acquired hepatitis C infection are shown in Figure 11. While rates for individual age groups vary from year to year, declines continue to be observed in the 15–19 and 20–29 years age groups. Annual rates in the other age groups continued to be relatively stable over the 1999 to 2009 period.

Exposure history surveillance data for all newly acquired hepatitis C notifications reported in 2009 were assessed from all jurisdictions except Queensland (Table 10). In 2009, 80% of these notifications had at least one risk factor recorded, with the source of exposure not recorded or unable to be determined for the remainder of these cases. Approximately 67% of notifications had a history of injecting drug use (42% of which with injecting drug use in the 24 months prior to diagnosis), and 19% had been detained in a correctional facility within the 24 months prior to diagnosis. Screening rates are generally higher in the prison entry population than the general population. A screening survey of prison entrants conducted over a two-week period in 2007 found that the prevalence of hepatitis C, based on hepatitis C antibody detection, was 35%.[13]

Table 10: Newly acquired hepatitis C notifications, selected jurisdictions,* 2009, by sex and exposure category†

Exposure category	Number of exposure factors reported†			Percentage‡ of notifications* (n=401)
	Male	Female	Total	
Injecting drug use	166	101	267	66.6
Imprisonment	68	8	76	19.0
Skin penetration procedure	48	24	72	18.0
Tattoos	34	13	47	11.7
Ear or body piercing	13	10	23	5.7
Acupuncture	1	1	2	0.5
Healthcare exposure	4	12	16	4.0
Surgical Work	3	11	14	3.5
Major Dental Surgery	1	1	2	0.5
Blood/tissue recipient	–	–	–	0.0
Sexual contact – hepatitis C positive partner	14	25	39	9.7
Opposite sex	12	24	36	9.0
Same sex	2	1	3	0.7
Household contact	6	12	18	4.5
Perinatal transmission	11	12	23	5.7
Needlestick or bio-hazardous injury§	3	2	5	1.2
Other	6	9	15	3.7
Notifications with at least one risk factor	193	127	320	79.8
Risk factor unable to be determined	11	6	17	4.2
Unknown (not recorded)	41	23	64	16.0
Total number of exposure factors reported†	378	234	612	–
Total number of notifications*	245	156	401	–

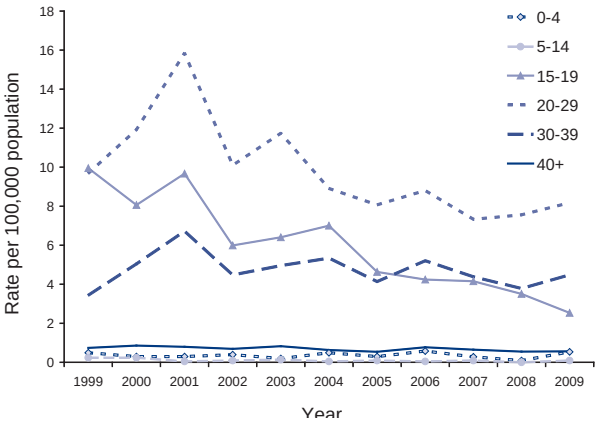
* Includes diagnoses in the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria, Western Australia and the Northern Territory.

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

‡ The denominator used to calculate the percentage is based on the total number of notifications from all jurisdictions, except Queensland. As more than one exposure category for each case could be recorded, the total percentage does not equate to 100%.

§ Includes both occupational and non-occupational exposures.

Figure 11: Notification rate for newly acquired hepatitis C, Australia,* 1999 to 2009, by age group and year



* Data from all states and territories except Queensland (1999–2009) and the Northern Territory (1999–2002).

Unspecified hepatitis C notifications

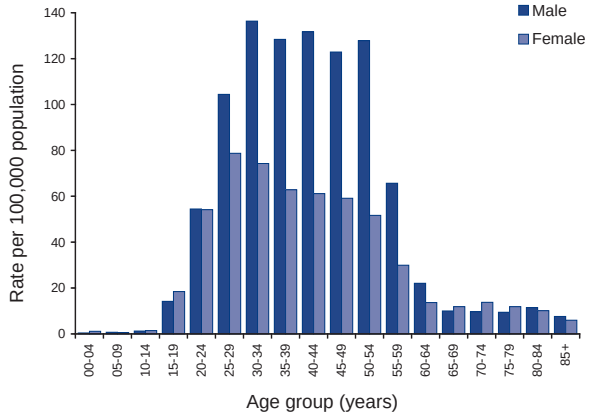
In 2009, 11,081 unspecified hepatitis C infections were notified to the NNDSS (50.7 per 100,000 population) compared with 11,098 notifications in 2008 (51.8 per 100,000 population).

The national notification rate for unspecified hepatitis C infection declined from 101.6 per 100,000 population in 1999 to 50.7 per 100,000 population in 2009 (Figure 9). Several factors may account for the decrease: changes in surveillance practices, including duplicate notification checking; a gradual decline in the prevalent group of hepatitis C cases accumulated prior to the introduction of hepatitis C testing in the early 1990s; and general reductions in risk behaviours related to injecting drug use, including the implementation of needle exchange programs.[18–20]

In 2009, the Northern Territory continued to have the highest notification rate (71.2 per 100,000 population) followed by New South Wales (55.1 per 100,000 population) and Tasmania (52.1 per 100,000 population). Queensland's rate was also high, at 61.5 per 100,000 population, however this included both newly acquired and unspecified cases.

The male to female ratio remained consistent with historical trends at 1.7:1. Amongst males, notification rates were highest across the age group range 30–34 to 50–54 years at around 129 per 100,000 population (range: 127.8 to 136.3). In the female population, notification rates were highest in the 25–29 and 30–34 years age groups, at 78.7 and 74.3 per 100,000 population respectively (Figure 12).

Figure 12: Notification rate for unspecified hepatitis C,* Australia, 2009, by age group and sex†



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

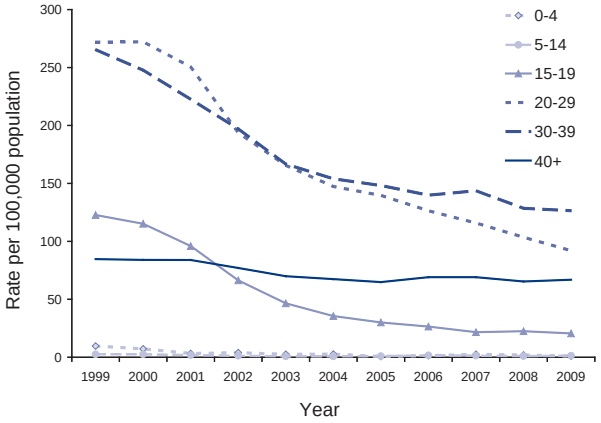
† Excludes 60 cases whose age or sex was not reported.

Trends in the age distribution of unspecified hepatitis C infection are shown in Figure 13. Between 2000 and 2009, the notification rate of unspecified hepatitis C declined by 82% amongst the 15–19 years age group, by 66% amongst the 20–29 years age group and by 49% in the 30–39 years age group. Trends in the 0–4 and the 40 years and over age groups have remained relatively stable over the past 10 years.

Hepatitis D

Hepatitis D is a defective single-stranded ribonucleic acid virus (RNA) that replicates in the presence of the hepatitis B virus. Hepatitis D infection can occur either as a co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection.[21] 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.

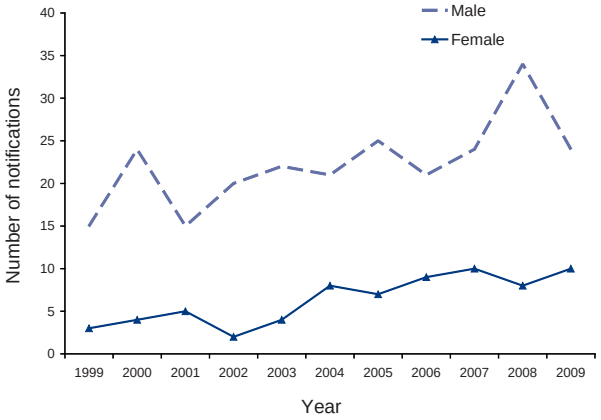
Figure 13: Notification rate for unspecified hepatitis C,* Australia, 1999 to 2009, by age group and year



* Data provided from Queensland (1999–2009) and the Northern Territory (1999–2002) includes both newly acquired and unspecified hepatitis C cases.

In Australia, the rate of hepatitis D remains low. In 2009, there were 34 notifications of hepatitis D (0.2 per 100,000 population) reported from Queensland (13), Victoria (12) and New South Wales (9). Over the past 5 years, notifications of hepatitis D have continued to remain relatively stable at around 34 notifications per year (range: 29 to 42), and over this time the male to female ratio was around 3:1 (Figure 14).

Figure 14: Notifications of hepatitis D, Australia, 1999 to 2009, by sex



Gastrointestinal diseases

In 2009, gastrointestinal diseases notified to NNDSS were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga toxin-producing *Escherichia coli* (STEC) infections and typhoid.

Overall notifications of gastrointestinal diseases increased 16% from 27,308 in 2008 to 31,697 in 2009. Notifications of cryptosporidiosis, hepatitis A, listeriosis and STEC were notably increased compared with the 5-year mean (exceeded the mean by more than 2 standard deviations).

Australia's enhanced foodborne disease surveillance network, OzFoodNet, monitors the incidence of diseases caused by pathogens commonly transmitted by food, using population-based passive and enhanced surveillance for notifiable gastrointestinal diseases and for outbreaks of gastroenteritis and enteric disease. In 2009, OzFoodNet aggregated and analysed data from NNDSS, supplemented by enhanced surveillance data, on the following 9 diseases or conditions, a proportion of which may be transmitted by food: non-typhoidal salmonellosis, campylobacteriosis, listeriosis, shigellosis, typhoid, STEC infections, botulism, HUS and hepatitis A. The data and results from these analyses are summarised in the following sections but are reported in more detail elsewhere.[22]

Botulism

Botulism is a rare but extremely serious intoxication resulting from toxins produced by *Clostridium botulinum* (commonly toxin types A, B and E). Three forms of botulism are recognised; infant, foodborne and wound.[21] Infant botulism occurs when *C. botulinum* spores are ingested, germinate in the infant's intestine and the organism produces botulinum toxin. It does not include cases where the preformed toxin is ingested, these are considered foodborne.

One case of botulism was reported to NNDSS in 2009; an infant botulism case reported from Queensland.[22] The case was hospitalised in intensive care with onset of symptoms (acute flaccid paralysis) in March 2009. *C. botulinum* toxin was detected in a stool sample and culture by mouse bioassay, and identified as toxin type B. The infant was entirely breast-fed and had not had a bowel motion for approximately 2 weeks prior to admission. It was speculated that the slow transit time within the bowel provided time for the toxin to develop. Treatment included human immunoglobulin for infant botulism obtained from the United States of America.

There were no notifications of botulism reported in 2008 and one in 2007.

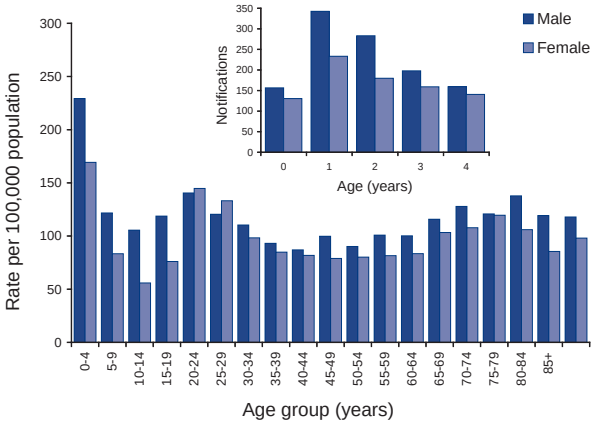
Campylobacteriosis

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

In 2009, there were 15,973 notifications of campylobacteriosis, similar to the 15,535 notifications reported in 2008. The national rate of campylobacteriosis notifications was also similar to the previous year, with 108.1 notifications per 100,000 population in 2009 compared with 107.5 per 100,000 in 2008.

Notification rates were highest amongst males in nearly all age groups. The highest age specific rate for both males and females was in infants aged 1 year (343 and 233 notifications per 100,000 population, respectively) with additional peaks in the 20–29 and 70–84 year age-groups (Figure 15).

Figure 15: Notification rate for campylobacteriosis, Australia, 2009, by age group and sex. Inset: age and sex in children aged under 5 years



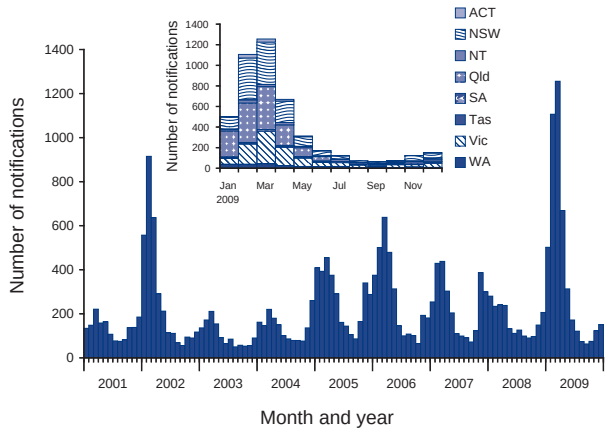
Cryptosporidiosis

In 2009, 4,625 notifications of cryptosporidiosis were reported to NNDSS, a national rate of 21.1 notifications per 100,000 population. This represents a 130% increase over the 2,003 notifications reported in 2008 and is the largest number reported since the disease became nationally notifiable in 2001 (Figure 16). Cryptosporidiosis notifications fluctuate from year to year, and notifications are most numerous in autumn and summer, with some regional variation.

The highest notification rate was in the Northern Territory, with a rate of 66.7 per 100,000 population (150 notifications). There were 4 recognised outbreaks of cryptosporidiosis in the Northern Territory in 2009, all of them occurring in remote Indigenous communities.

The largest number of notifications was reported from New South Wales (1,463), where notifications were increased by 143% compared with the 5-year mean of 861 notifications for the state. This increase was due to a large outbreak of cryptosporidiosis asso-

Figure 16: Notifications of cryptosporidiosis, Australia, by month and year, 2001 to 2009. Inset: by month and state or territory

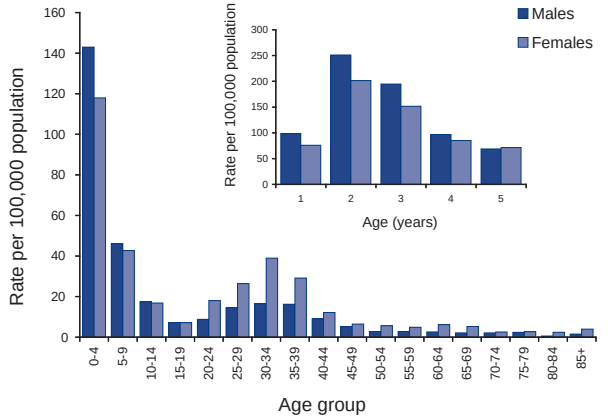


ciated with public swimming pools in early 2009. The NSW Department of Health (NSW Health) issued several public health alerts through the media, and NSW Health's Environmental Health Branch worked with the owners of the affected swimming pools to reduce the risk of further transmission.

The completeness of the Indigenous status field nationally (47.9%) was too low for meaningful analysis, but in the Northern Territory, cases amongst Indigenous people accounted for 64.7% of notifications in 2009, with 94.7% data completeness.

In 2009, 52.9% of nationally notified cases were female. Forty per cent of all notified cases were in children aged under 5 years with notification rates higher amongst males in this age group (Figure 17).

Figure 17: Notification rate for cryptosporidiosis, Australia, 2009, by age group and sex. Inset: age and sex in children aged under 5 years



Haemolytic uraemic syndrome

Haemolytic uraemic syndrome is a rare but serious disease, related to some gastrointestinal infections, and results in chronic complications in 40% of cases.[23] In 2009, there were 12 notifications of HUS (rate 0.05 per 100,000 population) (Table 3), compared with 31 in 2008 and a mean of 20 notifications per year (0.1 per 100,000 population) between 2004 and 2008.

The median age of HUS notifications was 10 years (range 1 to 89 years) and were most frequently reported amongst children aged 0–4 years (Table 11).

HUS can result from an antecedent STEC infection, but may be due to non-enteric infections, or non-infectious causes. An antecedent STEC infection was reported in 41.7% (5) of notified cases. One was associated with a non-Shiga toxin-producing *E. coli* infection, 1 case was associated with *Streptococcus pneumoniae* infection, and no aetiology was reported for the remaining 5 notifications.[22]

Table 11: Notifications of haemolytic uraemic syndrome, Australia, 2009, by age group

Age group	Number of notifications
0–4	4
5–9	2
10–14	2
15–19	0
20–24	0
25–29	1
30–34	0
35–39	0
40–44	0
45–49	0
50–54	0
55–59	0
60–64	1
65–69	0
70–74	0
75–79	1
80–84	0
85+	1

Hepatitis A

In 2009, there were 563 notifications of hepatitis A in Australia, a 104% increase compared with the 277 notifications in 2008 (Table 3). The rate of 2.6 notifications per 100,000 population compared with the 5-year mean of 0.3 per 100,000.[22] This

increase (Figure 18) was largely attributable to an outbreak of locally-acquired infections between 1 March 2009 and 18 March 2010, associated with the consumption of semi-dried tomatoes.[22,24]

Hepatitis A was most frequently notified amongst young adults (Figure 19). The median age of notified cases was 32 years (range 1–88 years). Half (50.4%) of all notified cases were female.

Figure 18: Notifications of hepatitis A, Australia, 1991 to 2008, by year of diagnosis

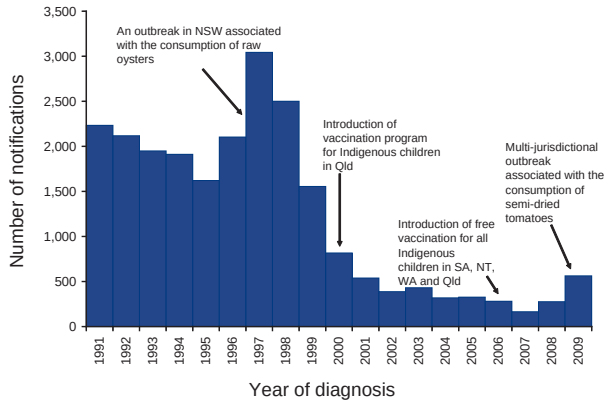
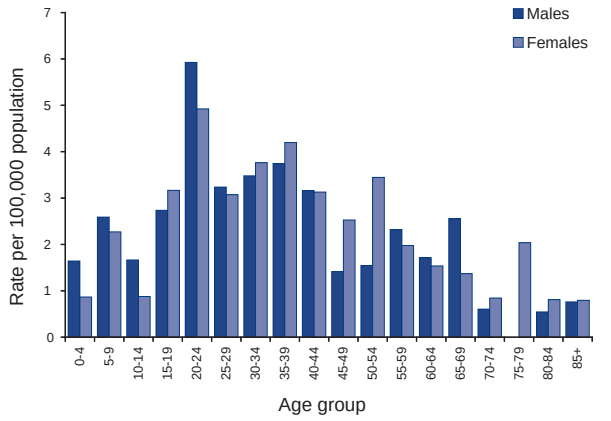


Figure 19: Notification rate for hepatitis A, Australia, 2009, by age group and sex



While overseas travel has been the most frequently reported risk factor for notified cases in recent years,[25] in 2009 a higher than usual proportion of notified cases were locally-acquired (67% in 2009 compared with less than 45% between 2004 and 2008). This increase was due to the semi-dried tomato outbreak (Table 12).

The proportion of notifications of hepatitis A in Australia in Indigenous persons remains low, with only 1% of notifications in 2009 reported as Indigenous, compared with 10%–12% (37–53 notifications) per year between 2003 and 2006, and less than 2% in 2007 and 2008 (0 and 3 notifications respectively). Indigenous status was known for 92% of notifications in 2009. This marked decrease in recent years in the number and proportion of notifications who were Indigenous is likely to be due in part to targeted vaccination programs for Indigenous children commencing in north Queensland in 1999, and the provision of free hepatitis A vaccine for all Indigenous children in the whole of Queensland, South Australia, Western Australia and the Northern Territory from 2006 (Figure 18).[26]

Hepatitis E

In 2009, there were 33 notifications of hepatitis E, compared with 44 notifications in 2008. Hepatitis E in Australia is associated strongly with overseas travel, with 68% (30)[27] and 89% (16/18)[28] of notified cases in 2008 and 2007 respectively known to have been acquired overseas. Data on travel status were not collated nationally in 2009.

Listeriosis

Invasive listeriosis commonly affects the elderly or immunocompromised, and is most common amongst people with serious or terminal underlying illnesses, but also amongst pregnant women and their newborn babies. Foetuses may become infected *in utero*. Laboratory-confirmed infections in a mother and unborn child or a neonate are notified separately in the NNDSS. However,

Table 12: Notifications of hepatitis A, Australia, 2004 to 2009, by place of acquisition

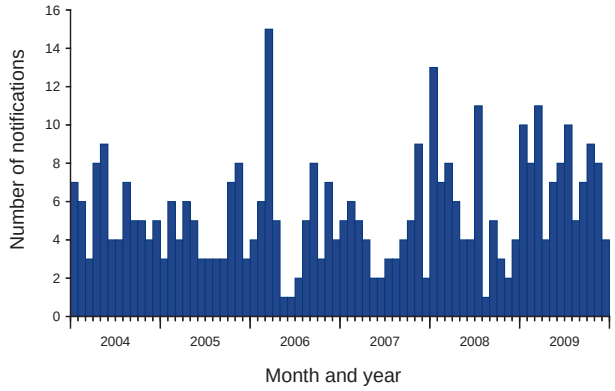
	Locally acquired		Acquired overseas		Unknown	
Year	%	n	%	n	%	n
2004	44.7	143	30.6	98	24.7	79
2005	36.7	121	31.8	105	31.5	104
2006	42.1	120	37.9	108	20.0	57
2007	30.5	50	57.9	95	11.6	19
2008	37.0	102	55.8	154	7.2	20
2009	67.0	377	30.4	171	2.7	15

OzFoodNet counts such pairs as 1 case', with the mother reported as the primary case, leading to differences in numbers from those reported here.

In 2009, 91 notifications of invasive *Listeria monocytogenes* infection were reported to NNDSS (0.4 per 100,000 population) compared with a 5-year historical mean of 60 notifications (0.3 per 100,000) (Figure 20). This increase was in part due to a multi-jurisdictional outbreak of listeriosis that was associated with the consumption of contaminated chicken wraps.[22]

Seventeen of these 91 notified cases (19%) were pregnancy related, occurring in pregnant women and/or their newborn babies. In 2009, 55% (41/74) of the non-pregnancy related cases were female. Fifty-six per cent (51/91) of notifications were in people aged 60 years or more (this group forms 19% of the Australian population) and the highest age-specific notification rate was in people aged 85 years or more (12 notifications, 3.1 per 100,000 population).

Figure 20: Notifications of invasive listeriosis, Australia, 2004 to 2009, by month and year



Salmonellosis (non-typhoidal)

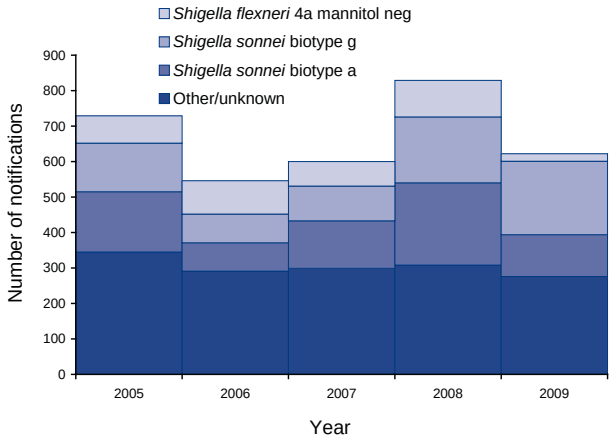
There were 9,533 notifications of salmonellosis in Australia in 2009 representing a rate of 43.6 notifications per 100,000 population, compared with a 5-year mean of 40.8 per 100,000.[22] Notification rates ranged from 30 per 100,000 in Victoria to 217 per 100,000 in the Northern Territory. In 2009, 51% of notifications were in females. Children aged 0–1 year had the highest age specific notification rate (300 per 100,000).

Individual notifications are rarely attributed to a particular source. In Australia, *Salmonella* infections, and in particular serotype Typhimurium, frequently manifest as outbreaks transmitted via contaminated food, and investigation of these outbreaks provides information about high-risk foods to inform policy and regulation. In 2009, OzFoodNet epidemiologists investigated 60 foodborne or suspected foodborne outbreaks of salmonellosis, affecting 771 people, although not all of these were laboratory-confirmed cases.[22] The most frequently reported *Salmonella* serotypes nationally were *S.* Typhimurium (32% of notified cases) and *S.* Enteritidis (18% of notified cases).

Shigellosis

In 2009, 622 notifications of shigellosis were reported, a rate of 2.8 per 100,000 population, similar to the 5-year mean of 3.1 per 100,000. As in previous years, the highest notification rate was in the Northern Territory (37.8 per 100,000), although this was lower than its 5-year mean (74.7 per 100,000).[22] *Shigella sonnei* biotype g (33%; 207) was the most commonly reported biotype in 2009, followed by *S. sonnei* biotype a (19%; 118) (Figure 21).

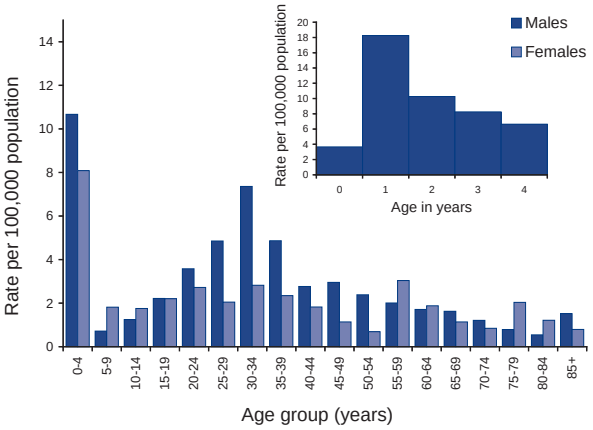
Figure 21: Notifications of shigellosis, Australia, 2005 to 2009, by biotype



Notification rates for shigellosis were highest in males and females aged 0–4 years (10.7 and 8.1 per 100,000, respectively). Secondary peaks were observed in males aged 30–44 years, and in females aged 55–59 years. Amongst children under 5 years of age, the highest notification rates were in children aged 1 year (Figure 22).

Information on Indigenous status was available for 66.6% (414) of notifications. Data completeness on Indigenous status varied by state or territory, with the Australian Capital Territory, New South Wales, Queensland, and South Australia being less than 85% complete. Amongst jurisdictions with greater than 85% completeness, the proportion of notified cases who identified as being of Aboriginal or Torres Strait Island origin was 48.6% (142/292).

Figure 22: Notification rate for shigellosis, Australia, 2009, by age group and sex. Inset: notifications in children aged under 5 years, Australia, 2009



Information on overseas travel status as a risk factor was available for 47.4% (295/622) of notified cases, with 45.8% (135/295) of these reporting overseas travel during the time when they were likely to have been exposed to the infection (Table 13). The most frequently reported countries of acquisition for imported cases were Indonesia (29.6%, 40/135) and India (11.9%, 16/135).[22]

Shiga toxin-producing *Escherichia coli* infections

There were 130 notifications of STEC in Australia in 2009,, a rate of 0.6 notifications per 100,000 population (Table 3) compared with the 5-year mean of 0.4 per 100,000.[22] Thirty-one per cent (40) of notifications in 2009 were known to have been associated with 3 jurisdictional outbreaks and a multi-jurisdictional cluster, which may in part explain the higher overall rate.

Rates of STEC infection are strongly influenced by jurisdictional practices regarding the screening of stool specimens.[29] In particular, South Australia routinely tests all bloody stools by polymerase chain reaction (PCR) for genes coding for Shiga toxins and other virulence factors, making rates for this state the highest in the country at 3.9 per 100,000 population.

In 2009, 56.9% of notified cases were female. The median age of notified cases was 44 years (range 0–91 years). Age specific notification rates were highest in younger (0–19 years) and older (55 years or older) age groups, with 35.4% (46/130) and 41.5% (54/130) respectively falling into these age groups (Figure 23).

Typhoid

There were 115 notifications of *S.* Typhi infection (typhoid) during 2009 (0.5 per 100,000 population), which was slightly higher than the 5-year mean of 0.4 per 100,000.[22]

Figure 23: Notification rate for Shiga toxin-producing *Escherichia coli*, Australia, 2009, by age group

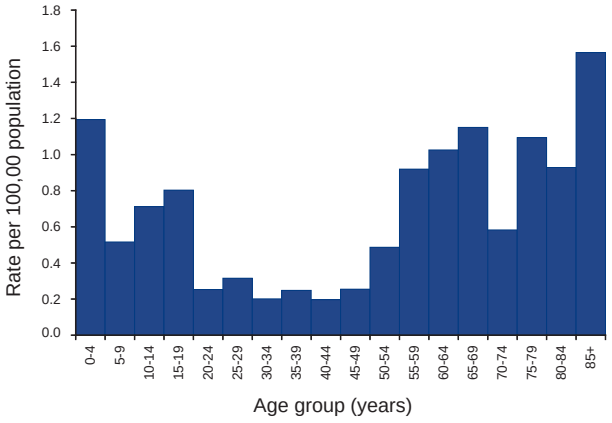


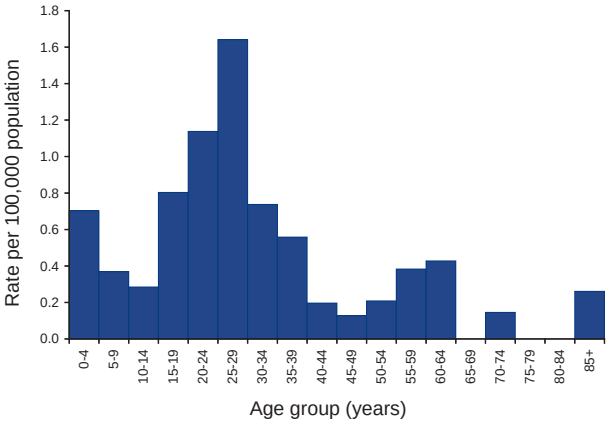
Table 13: Notifications of shigellosis, Australia, 2009, by overseas travel status

State or territory	Overseas travel			Overseas acquired (%)	Total
	Yes	No	Not stated/ unknown		
ACT	6	2	0	75.0	8
NSW	10	1	145	6.4	156
NT	2	4	79	2.4	85
Qld	20	48	47	17.4	115
SA	28	11	12	54.9	51
Tas	2	0	0	100.0	2
Vic	39	40	6	45.9	85
WA	28	54	38	23.3	120
Total	135	160	327	21.7	622

Similar to previous years, overseas travel was the primary risk factor for notified cases in 2009, with 88.7% (102/115) of notified cases known to have been acquired overseas, compared with 92.3% (97/105) in 2008.[27] India continues to be the most frequently reported country of acquisition, accounting for 61.8% (63/102) of overseas acquired cases in 2009, with a range of other countries in South and South East Asia reported as the place of acquisition, each by less than 1% of cases.

Age specific notification rates were highest in the 25–29 years age group (1.6 per 100,000 population) and the 20–24 years (1.1 per 100,000) age group (Figure 24), reflecting higher rates of overseas travel in young adults.

Figure 24: Notification rate for typhoid, Australia, 2009, by age group



Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and notifiable in Australia and to the WHO in 2009 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS), human swine influenza (H1N1) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean–Congo).

Cholera, plague, rabies, smallpox, yellow fever, SARS, HPAIH, H1N1 and viral haemorrhagic fevers are of international public health importance. Travellers are advised to seek information on the risk of contracting these diseases at their destinations and to take appropriate measures. More information on quarantinable diseases and travel health can be found on the following web sites:

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

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

There were no cases of plague, rabies, smallpox, yellow fever, SARS, HPAIH or viral haemorrhagic fevers reported in Australia in 2009. Table 14 provides information on the occurrence of quarantinable diseases in Australia.

Table 14: Australia's status for human quarantinable diseases, 2009

Disease	Status	Date of last record and notes
Cholera	Free	A small number of cases are reported annually and related to overseas travel or imported food products.[27]
Plague	Free	Last case recorded in Australia in 1923.[30]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990.[31]
Smallpox	Free	Last case recorded in Australia in 1938[32]
Yellow fever	Free	No cases recorded on shore in Australia – 5 occasions on which vessels arrived in Australian ports 1892–1915.[30]
SARS	Free	Last case recorded in Australia in 2003.[33]
HPAIH	Free	No cases recorded.[34]
H1N1	Currently circulating as seasonal virus	See vaccine preventable diseases section.
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded.[35]
Marburg	Free	No cases recorded.[35]
Lassa	Free	No cases recorded.[35]
Crimean–Congo	Free	No cases recorded.[35]

Cholera

In 2009, there were 4 notifications of cholera reported to the NNDSS in Australia, three from New South Wales and one from Victoria. All were acquired overseas.

All cases of cholera reported since the commencement of the NNDSS in 1991 have been acquired outside Australia except for 1 case of laboratory-acquired cholera in 1996[36] and 3 cases in 2006.[37] There have been 19 cases of cholera notified between 2004 and 2008 (Table 6).

Sexually transmissible infections

In 2009, the sexually transmissible infections (STIs) reported to the NNDSS were chlamydial infection, donovanosis, gonococcal infection and syphilis. Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance Programme (AGSP), which is a network of specialist laboratories monitoring antimicrobial susceptibility patterns of gonococcal infection, and the Kirby Institute, which maintains the National HIV Registry and the National AIDS Registry.

The national trends in the number and rates of STI notifications reported to the NNDSS between 2004 and 2009 are shown in Table 6. In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence: changes in screening programs,[38,39] the use of less invasive and more sensitive diagnostic tests and periodic public awareness campaigns may influence the number of notifications that occur over time. For some diseases, changes in surveillance practices may also need to be taken into account when interpreting national trends.

Direct age standardised notification rates, using the method described by the Australian Institute of Health and Welfare,[40] were calculated for Indigenous and non-Indigenous notifications for jurisdictions that had Indigenous status data completed for more than 50% of notifications over the period 2004 to 2009. Where the Indigenous status of a notification was not completed, these notifications were counted as non-Indigenous in the analysis. These data however, should be interpreted with caution as STI screening occurs predominately in specific high risk groups, including in Indigenous populations. Previous research into high rates of STIs amongst the Indigenous population in the Northern Territory suggested that the disparity in notification rates could be attributed to more targeted screening programs and poorer access to primary health care services, rather than to increased levels of transmission amongst Indigenous people.[41,42] Similarly, the differences in rates between females and males should be interpreted with caution, as rates of testing for STIs, symptom status, health care-seeking behaviours, and partner notification differ between the sexes.[43]

In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Infections in children may be acquired perinatally (e.g. gonococcal conjunctivitis).[44] Notifications of chlamydial, gonococcal and non-congenital syphilis infections were excluded from analysis where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

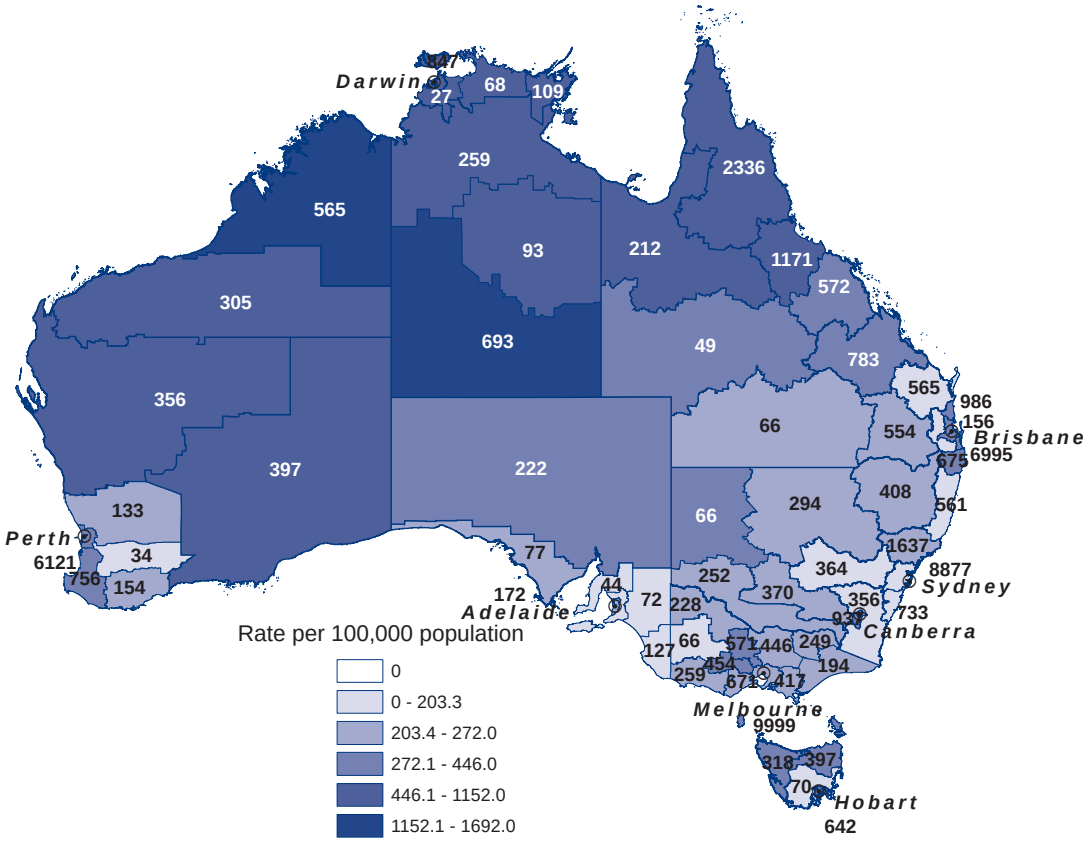
Chlamydial infection

Chlamydial infection continued to be the most commonly notified disease in 2009. Since chlamydial infection became a nationally notifiable disease in 1991 (1997 in New South Wales), the rate has increased in each consecutive year. In 2009, there were a total of 62,660 notifications of chlamydial infection, equating to a rate of 286 per 100,000 population. This represents an increase of 5% compared with the rate reported in 2008 (273 per 100,000 population). Between 2004 and 2009, chlamydial infection notification rates increased by 61%, from 180 to 286 per 100,000 population (Table 6).

Chlamydial infection notification rates were substantially higher than the national rate in the Northern Territory (941 per 100,000 population), Western Australia (395 per 100,000 population) and Queensland (379 per 100,000 population) (Table 5). At a regional level, chlamydial infection notification rates were highest in the Central NT Statistical Subdivision of the Northern Territory and the Kimberley Statistical Division of Western Australia (range: 1,152–1,692 notifications per 100,000 population), noting that notification rates in geographic areas where the estimated residential population and case numbers are small, should be interpreted with caution. Notifications rates were also substantially higher than the national rate (range: 446–1,152 notifications per 100,000 population) in the Statistical Divisions of the Far North West and Northern Queensland, the Pilbara, Central and South Eastern Western Australia, and the remaining Northern Territory Statistical Subdivisions, (Map 2).

In 2009, notification rates of chlamydial infection in males and females were 236 and 336 per 100,000 population respectively. When compared with 2008, notification rates increased by 7% in males and 4% in females. The male to female ratio in 2009 was 0.7:1, which was similar to previous years. Rates in

Map 2: Notification rates and counts* for chlamydial infection, Australia, 2009, by Statistical Division and Statistical Subdivision of residence in the Northern Territory



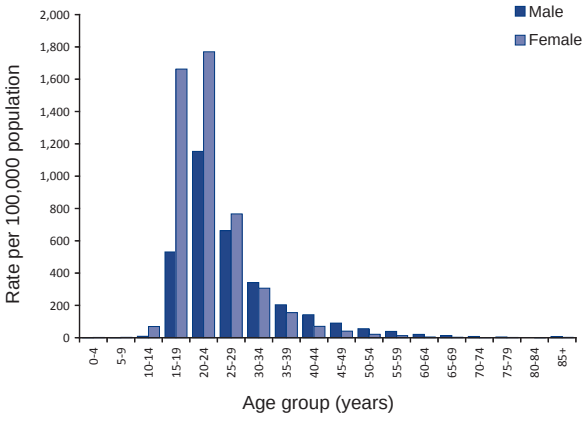
* Numbers in the shaded Statistical Divisions and Statistical Subdivisions represent the count of notifications.

females markedly exceeded those in males, especially in the 10–14 and 15–19 years age groups with ratios of 0.1:1 and 0.3:1, respectively (Figure 25).

Between 2004 and 2009, there was an increasing trend in chalmydia notification rates across all age groups, except the 10–14 years age group, and in both males and females (Figure 26). The greatest increase in notifications rates occurred in both males and females in the 15–19 (10% and 68% respectively) and the 20–29 (55% and 51% respectively) years age groups. These age groups accounted for around 80% of the annual number of notifications over the period 2004 to 2009.

From 2004 to 2009, the rates of chlamydial infection diagnoses increased in both Indigenous and non-Indigenous populations. Nationally in 2009, data on Indigenous status were complete in 49% of notifications; higher than the preceding 5-year average of 44% (range: 40%–48%). It should be noted that the completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Four jurisdictions had greater than 50% completeness of the Indigenous status field across the 2004 to 2009 period: the Northern Territory, South Australia, Tasmania and Western Australia. Among these jurisdictions, the combined age standardised notification rate ratio between Indigenous and non-Indigenous populations in 2009 was 3.6:1, with the disparity in notification rates improving substantially since 2000.

Figure 25: Notification rate for chlamydial infection, Australia, 2009, by age group and sex*



* Excludes 115 notifications for whom age or sex were not reported.

Between 2006 and 2008, rates of chlamydial infection notifications amongst these jurisdictions remained relatively stable at around 1,226 per 100,000 in the Indigenous population, but increased in the non-Indigenous population by 32%. In 2009, the rate of notifications in the Indigenous population declined by 10% compared with 2008, with relatively no change observed in the non-Indigenous population. At the jurisdictional level, between 2008 and 2009, chlamydia notification rates in the Indigenous population decreased in the Northern Territory, South Australia and Western Australia, while rates in their non-Indigenous counterparts remained relatively stable (Figure 27). The overall high Indigenous rates observed in the Northern Territory may be partly explained by high levels of screening, which take place in remote Indigenous communities.

Figure 26: Notification rate for chlamydial infection in persons aged 10–39 years, Australia, 2004 to 2009, by age group and sex

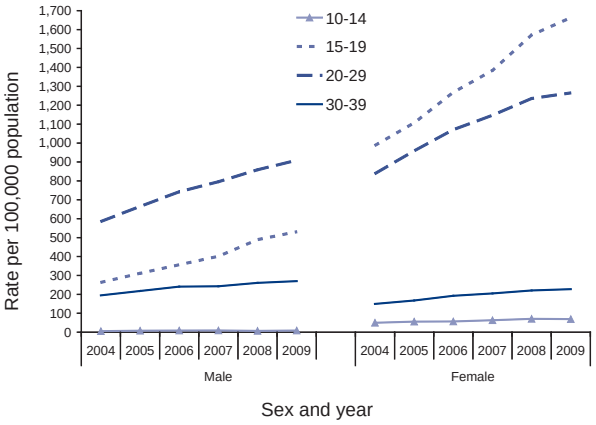
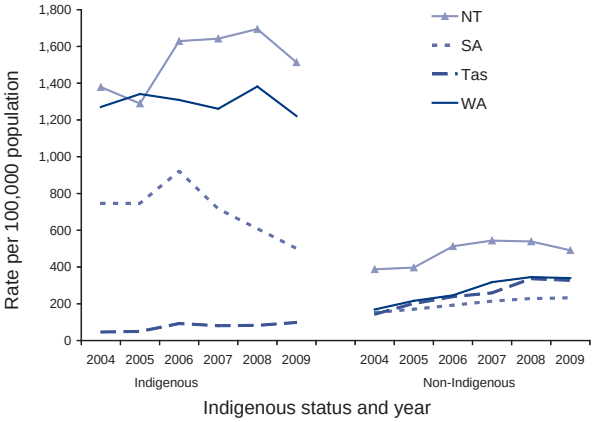


Figure 27: Notification rate for chlamydial infection, selected states and territories,* 2004 to 2009, by Indigenous status



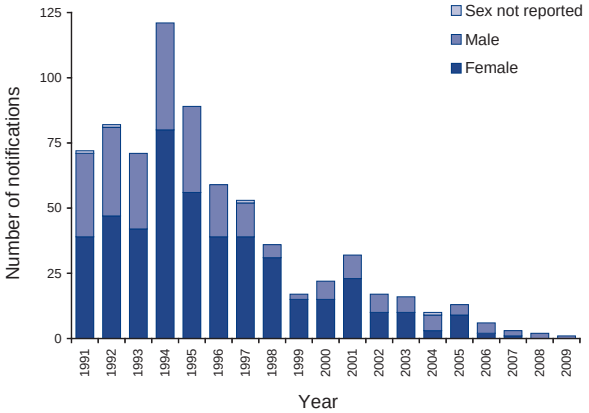
* Includes notifications in the Northern Territory, South Australia, Tasmania and Western Australia where Indigenous status completeness was reported for more than 50% of cases between 2004 and 2009.

Between May 2007 and June 2010, the Australian Department of Health and Ageing funded a pilot program called the Australian Collaboration for Chlamydia Enhanced Surveillance (ACCESS). The aim of the program was to monitor the uptake and outcome of chlamydia testing in Australia through a range of sentinel sites including sexual health services, general practices and laboratories. In 2009, ACCESS identified that chlamydia positivity, amongst people who accessed the sentinel sites, was 10.6% amongst males and 9.3% amongst females, with positivity highest in the 16–19 years age group across most of the sentinel sites.[18, 45] Enhanced surveillance of chlamydial notifications undertaken in Tasmania during 2008 identified that 57% of males presented as asymptomatic compared with 70% of females (personal communication, David Coleman, Tasmanian Department of Health and Human Services, 2 July 2010). Enhanced chlamydial surveillance data in Tasmania for the period 2001 to 2007 noted that females were more likely to have been tested for chlamydial infection as a result of screening, and males were more likely to have been tested when presenting with symptoms or as a result of contact tracing.[43] Therefore, notification rates for chlamydia, and other STIs, are particularly susceptible to overall rates of testing as well as targeted testing in certain high risk population sub-groups.

Donovanosis

Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project.[46] It predominantly occurred in rural and remote Indigenous communities in central and northern Australia and is now relatively uncommon. In 2009, one notification was reported to the NNDSS in an Indigenous male from Queensland (Figure 28).

Figure 28: Notifications of donovanosis, Australia, 1991 to 2009, by sex and year



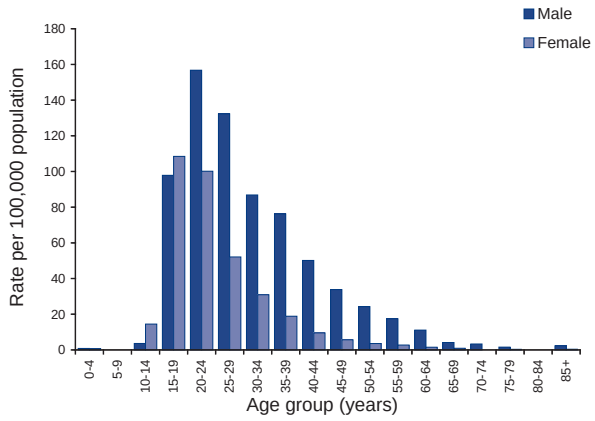
Gonococcal infections

In 2009, 8,059 notifications of gonococcal infection were reported to the NNDSS, equating to a notification rate of 36.8 per 100,000 population. This was a slight increase compared with 2008 (35.7 per 100,000 population). Due to a reporting issue, gonococcal notification data for Queensland is under-reported in 2009 and therefore should be interpreted with caution.

The highest notification rate in 2009 was in the Northern Territory (669 per 100,000 population), which was almost 18 times higher than the national rate (Table 5). Considerable declines in notification rates between 2008 and 2009 were observed in Western Australia (23%), South Australia (20%) and Tasmania (17%). Increases in notification rates for the same period were observed in Victoria (64%) and New South Wales (22%), with the Australian Capital Territory reporting an increase from 6.1 to 15.7 per 100,000 population.

Nationally, there was an increase in the gonococcal infection notification rates in males (6%) and a decrease in females (3%). Gonococcal infection notification rates were over two times higher amongst males compared with females (49.7 and 24.0 per 100,000 population respectively). The male to female rate ratio in 2009 was 2:1, which is similar to the previous 5 years. As in previous years, the exception to this pattern was the Northern Territory, where females had an overall higher notification rate than males (677 compared with 242 per 100,000 population). Nationally, notification rates of gonococcal infection in males exceeded those in females in all age groups except in the 10–14 and 15–19 years age groups (Figure 29).

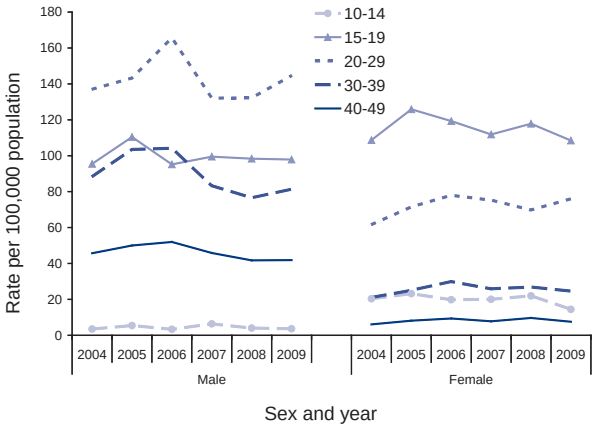
Figure 29: Notification rate for gonococcal infections, Australia, 2009, by age group and sex*



* Excludes 20 notifications for whom age or sex were not reported.

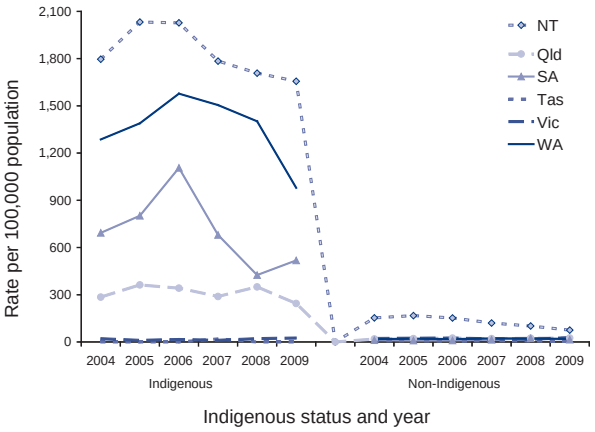
Trends in age specific notification rates show that there was an increase in gonococcal notifications amongst males in the 20–29 years age range in 2009 compared with 2007 and 2008. The notification rate of gonococcal infection in females across most age groups continued to have slight declines, with a small increase observed in the 20–29 years age group between 2008 and 2009 (Figure 30).

Figure 30: Notification rate for gonococcal infection in persons aged 10–49 years, Australia, 2004 to 2009, by age group and sex



In 2009, the data completeness of the Indigenous status field for gonococcal infection notifications was 65%, which was a decrease compared with previous years (around 70%). Six jurisdictions had greater than 50% completeness of the Indigenous status field: the Northern Territory, Queensland, South Australia, Tasmania, Victoria and Western Australia. Amongst these jurisdictions the combined age standardised notification rate for gonococcal infection was 634 per 100,000 in the Indigenous population and 24 per 100,000 in the non-Indigenous population resulting in an Indigenous to non-Indigenous rate ratio of 27:1. Between 2008 and 2009, rates of gonococcal infection notifications in the Indigenous population declined by 40% in Western Australia and 30% in Queensland, with a small decline also observed in the Northern Territory (3%). For the same period, an increase in the Indigenous notification rate of gonococcal infections was observed in South Australia (20%). Rates in the non-Indigenous population remained relatively stable over the 2004 to 2009 period, with declines observed in the Northern Territory between 2006 and 2009 (Figure 31). The overall high Indigenous rates observed in the Northern Territory may be partly explained by high levels of screening which take place in remote Indigenous communities.

Figure 31: Notification rate for gonococcal infection, selected states and territories,* 2004 to 2009, by Indigenous status and year



* Includes notifications in the Northern Territory, Queensland, South Australia, Tasmania, Victoria and Western Australia where Indigenous status completeness was reported for more than 50% of cases over a 5-year period.

Other surveillance of gonococcal infections

The AGSP is the national surveillance system for monitoring the antimicrobial resistance of *Neisseria gonorrhoeae* isolates, via a network of public and private reference laboratories located in each jurisdiction. Susceptibility testing to a core group of antibiotics: penicillin, ceftriaxone, spectinomycin, quinolone and tetracycline is performed on gonococcal isolates using a standardised methodology.

In 2009, the AGSP reported[47] a total of 3,220 gonococcal isolates that were tested for antibiotic susceptibility, representing approximately 40% of gonococcal infection notifications. The decreasing number of gonococcal isolates available for susceptibility testing is affected by the increasing use of non-culture based diagnosis methods.

Of the total number of isolates collected through the AGSP in 2009, there were 2,622 isolates from males, 596 isolates from females (male to female ratio 4.4:1) and there were 2 isolates for which the sex was not reported. In males, 71% of isolates were obtained from the urethra, 17% from the rectum and 10% from the pharynx. In females, the majority of isolates (89%) were obtained from the cervix.

In 2009, approximately 36% of gonococcal isolates had some level of resistance to the penicillins and 43% had some level of resistance to the quinolone antibiotic group. Since 2001, low numbers of isolates with decreased susceptibility to ceftriaxone have been identified in Australia, with 2% of isolates being 'non-susceptible' in 2009. As in previous years, the pattern of gonococcal antibiotic susceptibility differed between states and territories, and rural and urban areas within each jurisdiction,[48] where for example, in remote areas of some jurisdictions with high disease rates, penicillin-based treatments continue to be effective.

Syphilis (non-congenital)

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

In 2009, a total of 2,676 notifications of syphilis infection of all non-congenital categories were reported, representing a notification rate of 12.2 per 100,000 population; a slight decrease compared with 2008 (12.5 per 100,000 population) (Table 6, Figure 32). The Northern Territory continued to have the highest notification rate of syphilis (61 per 100,000 population), although the rate was 47% lower than in 2008. In 2009, there were increases in notification rates in Tasmania (26%), Queensland (15%), Victoria (7%), New South Wales (7%) and South Australia (5%). As in other developed countries, syphilis infection rates have continued to rise in Australia, predominantly affecting men who have sex with men.[49,50]

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

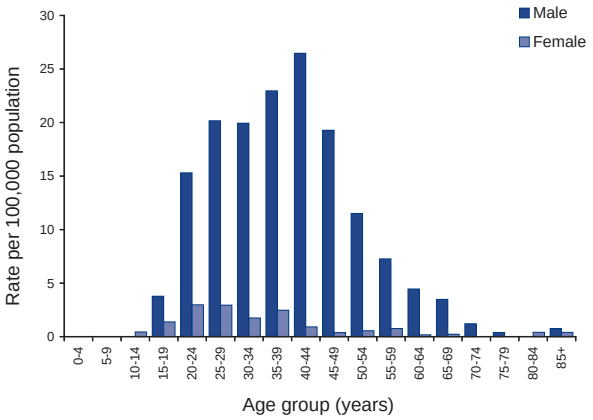
In 2009, 1,291 cases of infectious syphilis (primary, secondary and early latent), less than 2 years duration, were reported to the NNDSS. This represents a notification rate of 5.9 per 100,000 population, a decrease of 4% compared with 2008 (6.1 per 100,000 population) (Table 5). The rate of infectious syphilis notifications increased from 3.1 per 100,000 population in 2004 to 6.7 in 2007 and then declined to 5.9 in 2009 (Figure 32). Although the Northern Territory had the highest notification rate at 17 per 100,000 population in 2009, this was a substantial decrease compared with 2008 (38 per 100,000 population). The decrease was approximately the same in both sexes, even though there continued to be more cases in males than in females.[51]

Nationally, the notification rates of infectious syphilis for males and females were 10.8 and 1.4 per 100,000 population respectively, and represented a male to female ratio of 8:1 (Table 15). Notification

rates in males were highest in the 40–44 years age group (27 per 100,000 population), closely followed by the 35–39 years age group (23 per 100,000), whereas in females the highest notification rates were observed in the 20–24 and 25–29 years age groups (3.0 and 2.9 per 100,000 population respectively) (Figure 33).

Over the period 2005 to 2007, notification rates amongst males increased substantially, in the 20–29, 30–34 and 40–49 years age groups but have remained relatively stable since. The overall increases observed during this period were mainly attributed to men who have sex with men.[18] In females, for the 2004 to 2009 period, rates remained relatively steady, except in the 15–19 years age group where they decreased from a peak of 7.8 per 100,000 population in 2006 to 1.4 per 100,000 population in 2009 (Figure 34).

Figure 33: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2009, by age group and sex



* Excludes 1 notification for whom sex was not reported.

Figure 32: Notification rate for non-congenital syphilis infection (all categories), Australia, 2004 to 2009, by year

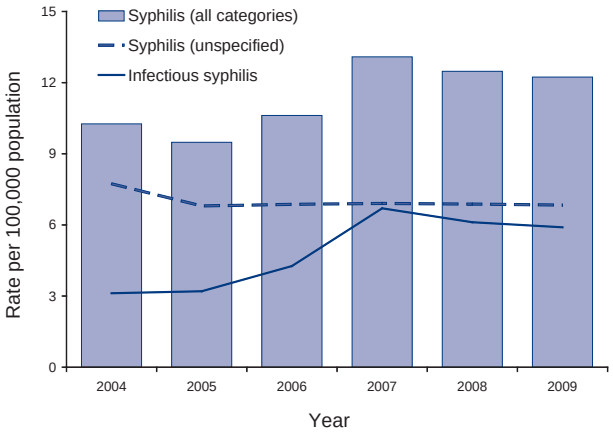


Figure 34: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, in persons aged 10 years or over, Australia, 2004 to 2009, by age group and sex

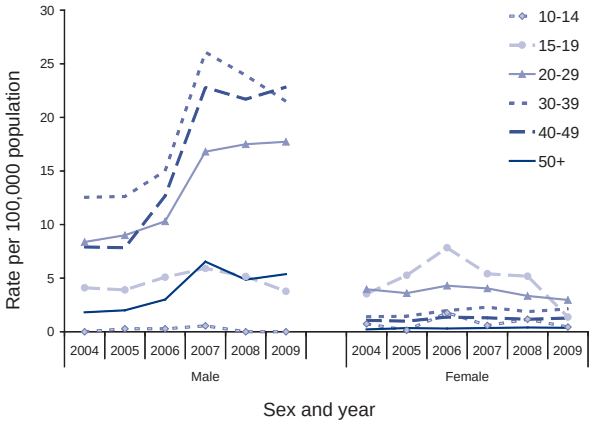


Table 15: Notifications and rates* for infectious syphilis (less than 2 years duration), Australia, 2009, by state or territory and sex†

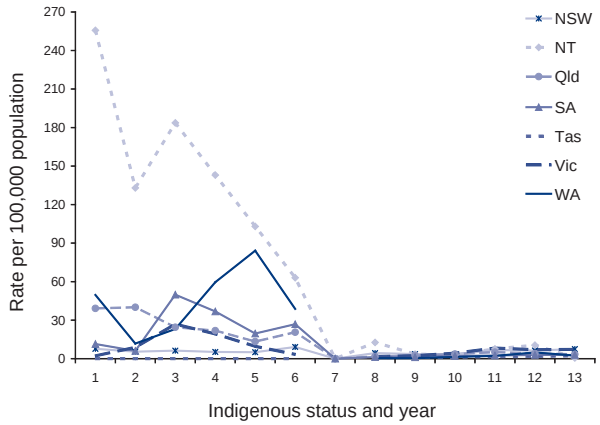
State or territory	Male		Female		Total†	
	Count	Rate*	Count	Rate*	Count	Rate*
ACT/NSW	504	13.7	22	0.6	533	7.2
NT	18	15.4	34	31.4	38	16.9
Qld	168	7.6	20	0.9	179	4.1
SA	44	5.5	7	0.9	53	3.3
Tas	10	4.0	2	0.8	10	2.0
Vic	369	13.7	17	0.6	390	7.2
WA	62	5.5	47	4.3	88	3.9
Total	1,175	10.8	149	1.4	1,291	5.9

* Notification rate per 100,000 population.

† Total includes 1 notification for whom sex was not reported.

In 2009, data on Indigenous status were complete for 96% of infectious syphilis notifications. All jurisdictions except the Australian Capital Territory had greater than 50% completeness of the Indigenous status field between 2004 and 2009. The age standardised notification rate was 23.7 per 100,000 in the Indigenous population and 5.6 per 100,000 in the non-Indigenous population, representing a ratio of 4:1. Age standardised notification rates varied widely across jurisdictions. Since 2006, Indigenous notification rates decreased across all of these jurisdictions except Western Australia, where the notification rate increased between 2005 and 2008 from 11–84 notifications per 100,000 population and declined to 39 per 100,000 population in 2009. This increase in Indigenous rates was largely attributable to an outbreak that occurred in 2008 in the Pilbara region amongst Aboriginal people (Figure 35).[52] Rates of infectious syphilis in the Indigenous population are highest in the 25–29 and 30–34 years age groups, compared with the non-Indigenous population where notification rates are highest in the 40–44 years age group.

Figure 35: Notification rate for infectious syphilis, selected states and territories,* 2004 to 2009, by Indigenous status and year



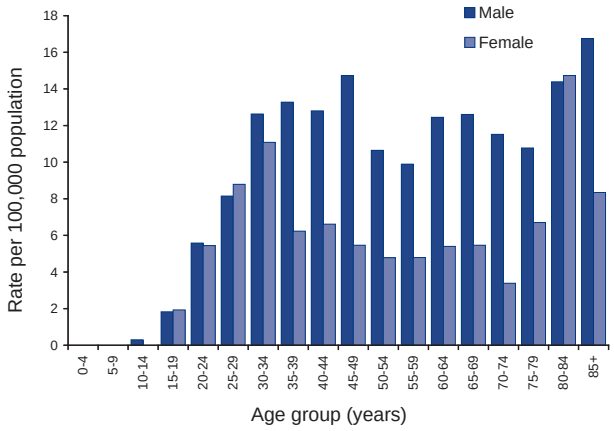
* Includes notifications in the Northern Territory, Queensland, South Australia, Tasmania, Victoria, Western Australia and New South Wales where Indigenous status completeness was reported for more than 50% of cases over a 5-year period.

Syphilis of more than 2 years or unknown duration

In 2009, a total of 1,385 notifications of syphilis of more than 2 years or unknown duration were reported to the NNDSS, giving a notification rate of 6.8 per 100,000 population, which was similar to the rate in 2008 (6.9 per 100,000 population). The Northern Territory continued to have the highest notification rate at 44 per 100,000 population, however, this was a decrease of 43% compared with 2008 (78 per 100,000 population).

In 2009, notification rates of syphilis of more than 2 years or unknown duration in males and females were 8.4 and 5.1 per 100,000 population, respectively (Table 16). Nationally, the male to female ratio was 1.7:1 (Figure 36). The distribution of notification rates across age groups in females was bimodal, with peaks in the 30–34 and 80–84 years age groups. In males, rates remained high from 30 years and over and peaks occurred in the 45–49 and 85 or over age groups. Rates in males were substantially higher than in females, especially in the 35–79 years age groups.

Figure 36: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2009, by age group and sex†



* Data from all states and territories except South Australia.

† Excludes 14 notifications for whom age or sex was not reported.

Over the period 2004 to 2009, notification rates increased amongst males in the 30–39 and 40–49 years age groups, with a substantial decrease observed in the 15–19 years age group. In females for the same period, increases were observed in the 40 years or over age groups and substantial decreases were observed in the 15–19 and 20–29 years age groups (72% and 40% respectively) (Figure 37).

Congenital syphilis

Following a peak of 19 notifications in 2001, notifications of congenital syphilis have continued to decline in 2009 (Figure 38). There were 3 notifications of congenital syphilis reported in 2009, 1 male and 2 females. All 3 notifications were from the Northern Territory. Two of the notifications were Indigenous and one was non-Indigenous.

Table 16: Notifications and rates* for syphilis of more than 2 years or unknown duration, Australia,† 2009, by state or territory and sex

State or territory	Male		Female		Total‡	
	Count	Rate*	Count	Rate*	Count	Rate*
ACT/NSW	248	6.7	159	4.2	410	5.5
NT	52	44.6	47	43.5	99	44.0
Qld	166	7.5	130	5.9	296	6.7
SA	NDP	–	NDP	–	NDP	–
Tas	14	5.6	4	1.6	18	3.6
Vic	314	11.7	145	5.3	468	8.6
WA	57	5.0	37	3.4	94	4.2
Total	851	8.4	522	5.1	1,385	6.8

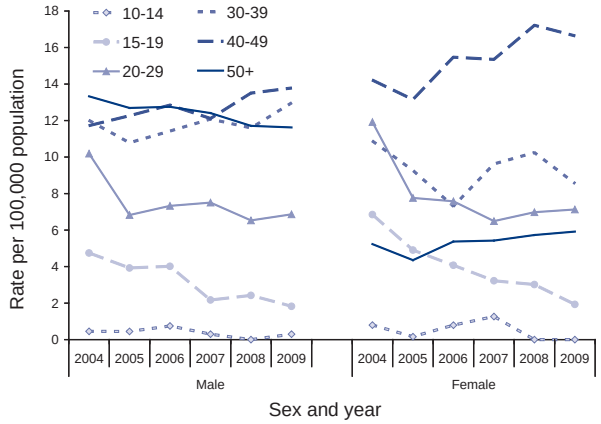
* Notification rate per 100,000 population.

† Data from all states and territories except South Australia.

‡ Total includes 12 notifications for whom sex was not reported.

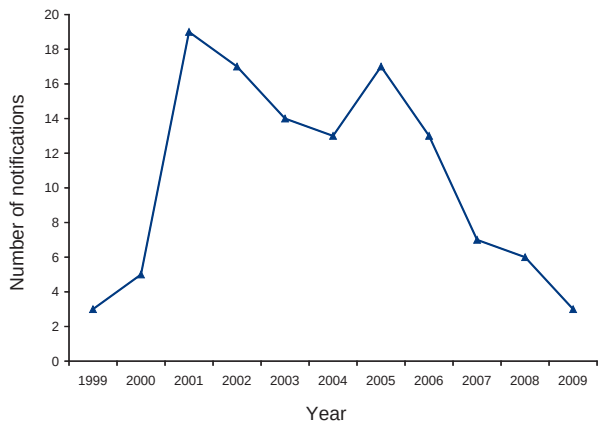
NDP No data provided.

Figure 37: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2004 to 2009, by age group and sex



* Data from all states and territories except South Australia.

Figure 38: Notifications of congenital syphilis, Australia, 1999 to 2009



Vaccine preventable diseases

Introduction

This section summarises the national notification surveillance data for notifiable diseases targeted by the National Immunisation Program (NIP) in 2009. These include diphtheria, invasive *Haemophilus influenzae* type b infection, laboratory-confirmed influenza, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella, tetanus and varicella zoster infections (chickenpox, shingles and unspecified). Data on hepatitis B and invasive meningococcal disease, which are also targeted by the NIP, can be found in this report under 'Bloodborne diseases' and 'Other bacterial infections' respectively. Other vaccine preventable diseases (VPDs) presented in this report include hepatitis A and Q fever under the 'Gastrointestinal' and 'Zoonoses' sections respectively. Rotavirus is not included as it is not a nationally notifiable condition. For more comprehensive reports on historical data, including notifications, hospitalisations and deaths, readers are referred to the regular CDI supplements *Vaccine Preventable Diseases in Australia,* the latest of which has recently been published.[53]

In 2009, there were 101,627 notifications of VPDs (43% of total) reported to the NNDSS. This was 2.9 times more notifications than in 2008 (33,983). Influenza was the most commonly notified VPD (58,778, 58% of total) followed by pertussis (29,736, 29% of total) reflecting the epidemics occurring as a result of these 2 diseases in 2009. The number of notifications and notification rates for VPDs in Australia are shown in Table 3 and Table 4, respectively.

There were no new vaccines added to the NIP in 2009. However, in response to the influenza pandemic experienced during 2009, a monovalent vaccine was developed and distributed through the national Pandemic (H1N1) 2009 Vaccination Program from the end of September 2009 to reduce transmission of the pandemic (H1N1) 2009 influenza virus and protect vulnerable individuals. Whilst the Program initially focused on particular priority groups, including health care workers and those vulnerable to severe health outcomes associated with influenza infection, the vaccine was also made available for free to everyone in Australia who wished to be vaccinated.

Vaccination coverage is an important factor influencing the incidence of VPDs. Since the commencement of the Australian Childhood Immunisation Register in 1996, immunisation coverage in children has been high by international standards, although areas of lower coverage remain, in which there is a potential for VPDs to occur and circulate. These mainly coincide with high levels of conscientious objectors to immunisation including coastal areas of South East Queensland, northern New South Wales, Adelaide and south-western Western Australia. On average, just 3% of children in Australia are not fully vaccinated for age, but in the above areas this proportion is much higher.[54]

Information on receipt of vaccines has historically been recorded on NNDSS using the 'vaccination status' field (full, partial or unvaccinated), plus a field capturing the number of doses. In January 2008 new, more detailed fields were added to record 'vaccine type' and 'vaccination date' for each dose. The new fields were intended to replace the old fields, with a transition period allowing either form of vaccination details. In 2009, four jurisdictions were using the new fields (Northern Territory, Queensland, Tasmania and New South Wales for selected diseases), while the remaining jurisdictions continued to use the old fields. In this report data on receipt of vaccines is presented for each disease combining data from the two different formats. No vaccine is 100% effective, and therefore infections sometimes do occur in fully vaccinated people, and some are reported later in this section. However, effective vaccines do provide a substantially lower chance of becoming infected, and/or reduced severity of disease. Monitoring vaccine failure rates is an important part of evaluating the NIP.

Diphtheria

Diphtheria is an acute illness caused by toxin-producing strains of the bacterium *Corynebacterium diphtheriae*. It normally involves the mucous membranes of the upper respiratory tract producing a membrane that can obstruct the airway. On rare occasions other mucous membranes or the skin can be affected. Diphtheria is spread by respiratory droplets or by direct contact with skin lesions or articles soiled by infected individuals.[21]

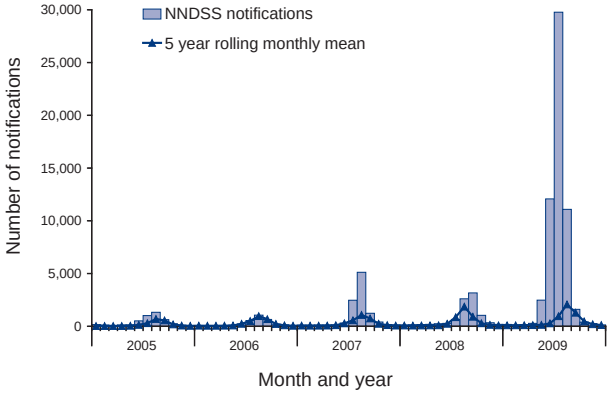
There were no notifications of diphtheria reported to NNDSS in 2009. The last notification of diphtheria reported in Australia was a case of cutaneous diphtheria (which affects the skin) in 2001, the only notification reported since 1992.

Influenza

In April 2009, the WHO announced the emergence of a novel influenza A virus, prompting the declaration of the first public health emergency of international concern since the *International Health Regulations (2005)* came into effect in 2007. The WHO subsequently raised the pandemic influenza alert in June 2009 to phase 6, the pandemic phase. The first notification of the pandemic (H1N1) 2009 influenza virus in Australia occurred in May 2009.

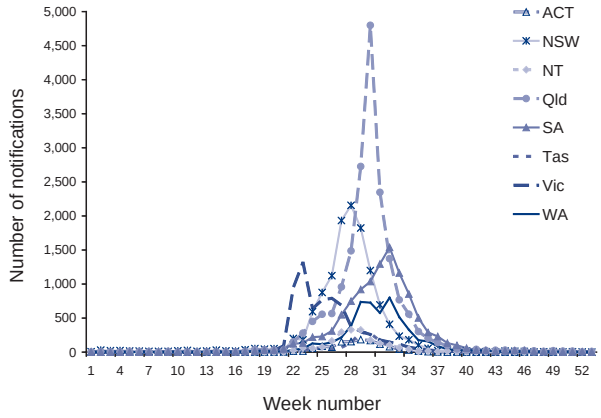
During the pandemic response, influenza notifications were reported by each jurisdiction using NetEpi, a web-based outbreak case reporting system, in addition to NNDSS. A more detailed analysis of enhanced data collected through NetEpi and additional sentinel surveillance systems will be reported in the 2010 National Influenza Surveillance Scheme annual report. The number of notifications in the Australian 2009 influenza season was the highest since national reporting to the NNDSS began in 2001, and substantially higher than in recent years (Figure 39). In 2009, there were 58,562 notifications of laboratory-confirmed influenza, a rate of 268 cases per 100,000 population. The number of notifications was 8.6 times greater than the 5-year mean and peaked in July with 29,770 notifications, but this over-representation is likely, at least in part, to reflect testing and laboratory practices in addition to real differences in the incidence of infection.[55] Notifications

Figure 39: Notifications of laboratory-confirmed influenza, Australia, 2009, by month and year of diagnosis



in the non-seasonal period were also higher than in previous years. Although Queensland continued to account for the highest proportion of all confirmed influenza cases notified (31%) (Figure 40), this figure was lower than previous years (the average for 2005–2008 was 44%, range 38%–54%). Throughout 2009, national testing protocols for each phase of the pandemic response were informed by the influenza SoNG.‡ For example during the 'Protect' phase the influenza SoNG focused on the testing of persons most at risk for severe disease outcomes, including people belonging to identified vulnerable groups and those presenting with severe disease presentation. However, due to local influenza activity and resource availability, testing rates in jurisdictions were variable.

Figure 40: Notifications of laboratory-confirmed influenza, Australia, 2009, by state or territory and week of diagnosis



In 2009, the highest notification rates occurred in the Northern Territory (875 per 100,000 population), followed by South Australia (663 per 100,000 population), Queensland (417 per 100,000 population) and the Australian Capital Territory (359 cases per 100,000 population) (Table 3).

In 2009, distribution of influenza notifications tended to occur in persons aged less than 55 years, with substantially higher rates observed in persons aged less than 30 years, compared with older age groups. In previous years, notifications of laboratory-confirmed influenza were highest in children aged 0–4 years (Figure 41), which represented, on average, 18% of notifications, whereas in 2009 they represented only 12% of notifications. In contrast, notification rates in 2009 were highest in the 5–9, 10–14 and 15–19 years age groups (Figure 42).

Figure 41: Notification rate for laboratory-confirmed influenza, Australia, 2006 to 2009, by age group

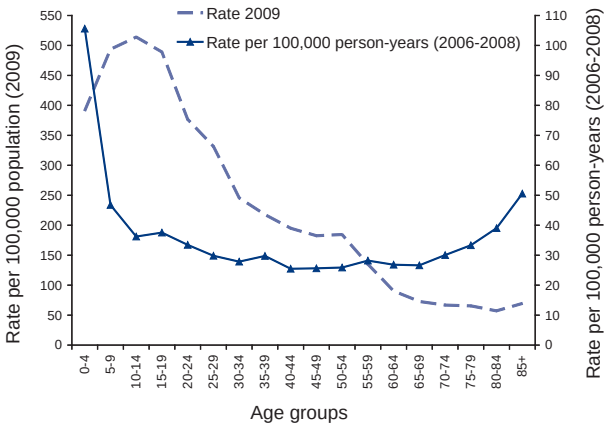
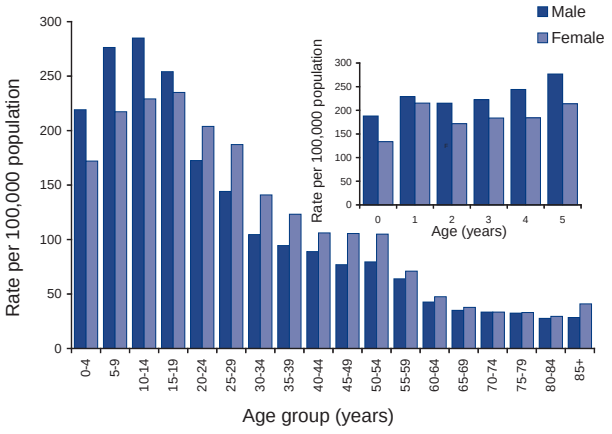


Figure 42: Notification rate for laboratory-confirmed influenza, Australia, 2009, by age group and sex*



* Excludes 128 notifications for whom age or sex were not reported.

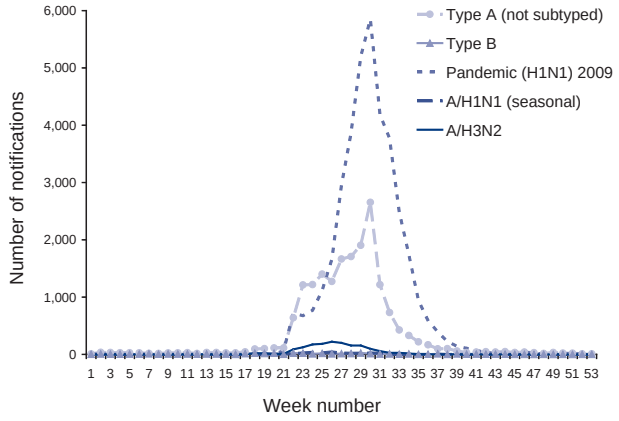
In 2009, 58,411 (99.8%) influenza notifications in the NNDSS and NetEpi had typing data. Of the typed notifications, 64.6% (37,750) were pandemic (H1N1) 2009, 31.4% (18,345) were notified as influenza A not subtyped, 2.8% (1,612) were influenza A/H3N2, 0.7% (410) were influenza B and 0.5% (294) were influenza A/H1N1 (seasonal) (Figure 43).

In 2009, 1,586 influenza virus isolates were subtyped by the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC), representing almost 3% of laboratory-confirmed cases reported to

‡ Series of National Guidelines (SoNG) – developed in consultation with the Communicable Diseases Network Australia and endorsed by the Australian Health Protection Committee. Their purpose is to provide nationally consistent advice and guidance to public health units in responding to a notifiable disease event.

the NNDSS. Pandemic (H1N1) 2009 represented the majority (74%) of isolates subtyped, followed by influenza A(H3N2) (18%), seasonal A(H1N1) (7%) and influenza B (1%).

Figure 43: Notifications of laboratory-confirmed influenza, Australia, 2009, by type and week of diagnosis*



* Notifications of influenza 'untyped' (n = 150) excluded from analysis.

The WHOCC also conducted antigenic characterisation on 884 of the influenza virus isolates, in similar proportions to those subtyped. The majority of pandemic (H1N1) 2009 isolates were characterised as A/California/7/2009-like. Seasonal influenza A(H1N1) viruses of the 2009 vaccine, A/Brisbane/59/2007, circulated sporadically throughout the year in very low numbers, being displaced by the pandemic (H1N1) 2009 strain.[56] Of the circulating influenza A(H3N2) viruses, most were antigenically similar to the 2009 A/Brisbane/10/2007 vaccine component, however the majority of these were low reactor versions indicating some drift in the strain. Although there were only a small number of influenza B viruses detected, antigenic characterisation showed a drift throughout the season in the 2009 vaccine strain, B/Florida/4/2006 (B/Yamagata lineage), to the B/Brisbane/60/2008 (B/Victoria lineage) strain.

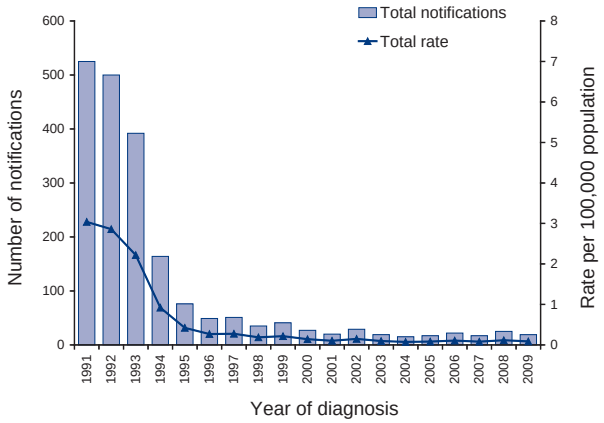
All 3 strains in the 2010 Southern Hemisphere influenza vaccine were different to those previously recommended in the 2009 Southern Hemisphere vaccine. The 2010 vaccine contained A/California/7/2009 (H1N1)-like, A/Perth/16/2009 (H3N2)-like and B/Brisbane/60/2008-like viruses.

Antiviral susceptibility testing for resistance to oseltamivir or zanamivir by enzyme inhibition assay (EIA) was conducted on 587 isolates of the pandemic (H1N1) 2009 strain by the WHOCC during 2009. Of these isolates, four showed resistance to oseltamivir. Molecular analysis of 276 isolates found 9 isolates (including the 4 oseltamivir resistant isolates identified through EIA) with the H275Y mutation, which is known to confer resistance to oseltamivir. Oseltamivir resistance was also found in the majority (36 of 37) seasonal A/H1N1 isolates tested, which is consistent with historical trends. In 2009 there were no reports of antiviral resistance in any of the A(H3N2) or influenza B isolates tested.

Invasive *Haemophilus influenzae* type b disease

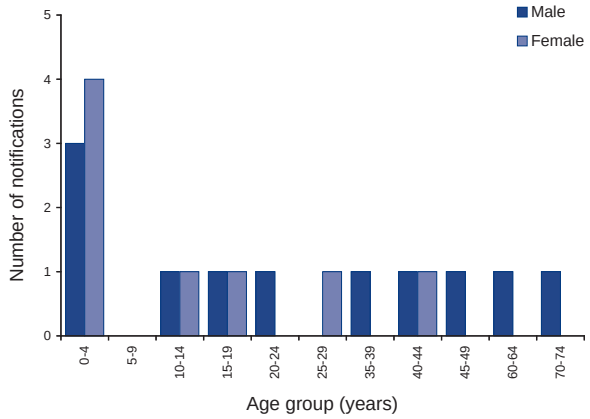
Invasive *Haemophilus influenzae* type b bacteria causes disease with symptoms dependant on which part of the body is infected. These include: septicaemia (infection of the blood stream); meningitis (infection of the membranes around the brain and spinal cord); epiglottitis (severe swelling of the epiglottis at the back of the throat); pneumonia (infection of the lungs); osteomyelitis (infection of the bones and joints) and cellulitis (infection of the tissue under the skin, usually on the face). Since the introduction of the Hib vaccine in 1993, there has been a marked reduction in total Hib notifications in Australia (Figure 44), which now has one of the lowest rates of Hib notifications in the world.[57]

Figure 44: Notifications and rates for invasive *Haemophilus influenzae* type b infection, Australia, 1991 to 2009, by year of diagnosis



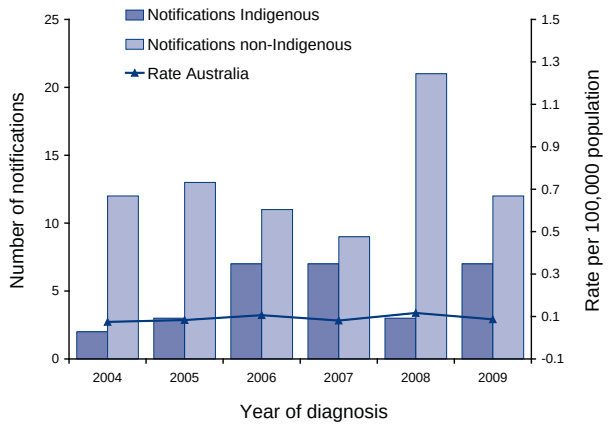
There were 19 notifications of Hib disease in 2009, a rate of 0.1 per 100,000 population, and six fewer than reported in 2008. Thirty-seven per cent (7) of notifications were amongst children less than 5 years of age with the majority of these (6/7) infants aged less than 1 year. The remaining 12 notifications ranged between 13 and 74 years. Fifty-eight per cent (11) were males resulting in a male to female ratio of 1.4:1. (Figure 45).

Figure 45: Notifications of invasive *Haemophilus influenzae* type b infection, Australia, 2009, by age group and sex



Indigenous status was 100% complete for notifications in 2009. Thirty-seven per cent (7/19) were reported as Indigenous and 63% (12/19) were non-Indigenous. The Hib notification rate in 2009 was 1.3 per 100,000 in Indigenous people and 0.1 per 100,000 in non-Indigenous people, a ratio of 13:1. Between 2004 and 2009, Hib rates for Indigenous people ranged between 5.5 and 30.3 times higher than for non-Indigenous people (Figure 46). However, these figures vary widely because of the low number of notifications. This analysis excludes those notifications with an unreported or unknown Indigenous status between 2004 and 2009 (4 for 2006 and 1 for each remaining year).

Figure 46: Notifications and rates for invasive *Haemophilus influenzae* type b infection, Australia, 2004 to 2009, by Indigenous status



All children under the age of 17 years in 2009 were eligible for Hib vaccination in infancy, as Hib vaccines were introduced to the NIP in April 1993 for all children born after February 1993. There were 9 notifications for children less than 17 years of age in 2009. The majority (7/9) of these were one year of age or less of which five were vaccinated and two were not vaccinated. Of the five who were vaccinated four had received 1 dose of a Hib containing vaccine and one had received 2 doses. Although four of these 5 vaccinated cases had received their age appropriate dose of vaccine none of the five had received the full course of recommended vaccine, which includes 3 or 4 doses depending on Indigenous status. The remaining two were a 13-year-old who had received 3 doses and a 14-year-old with unknown vaccination status.

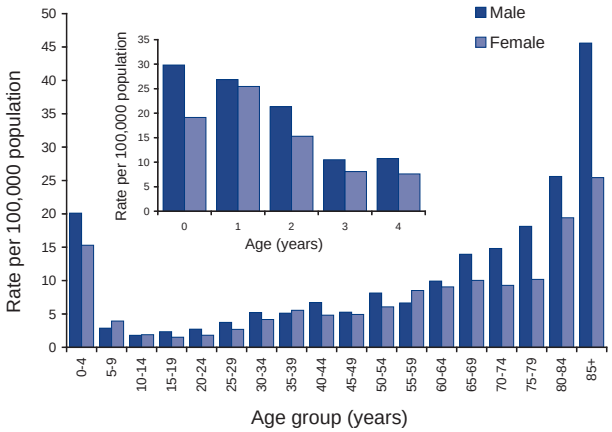
Invasive pneumococcal disease

There were 1,559 notifications of invasive pneumococcal disease (IPD) in Australia in 2009, a rate of 7.1 notifications per 100,000 population. This was a small decrease of 4% from the 1,634 reported in 2008 (7.6 per 100,000). An increase in rates in 2009, compared with 2008, was seen in the Australian Capital Territory (29, 8.3 per 100,000), the Northern Territory (86, 38.2 per 100,000), South Australia (145, 8.9 per 100,000) and Victoria (368, 6.8 per 100,000). A decrease in notifications was noted in Queensland (270, 6.1 per 100,000), New South Wales (477, 6.7 per 100,000), Tasmania (35, 7.0 per 100,000), and Western Australia (149, 6.7 per 100,000).

In 2009, males accounted for 54% (843) of the 1,559 notifications of IPD. In most age groups there were more male than female notifications, resulting in a male to female ratio of 1.2:1. Figure 47 shows that the highest rates of IPD in 2009 were notified in persons aged 85 years or over (32.3 per 100,000) and in children aged 1 year (26.2 per 100,000).

In 2001, the 7vPCV became available for infants and children at high risk of IPD, including Indigenous infants. In 2005 it was added to the NIP for all children up to 2 years of age.[12] Rates of IPD disease

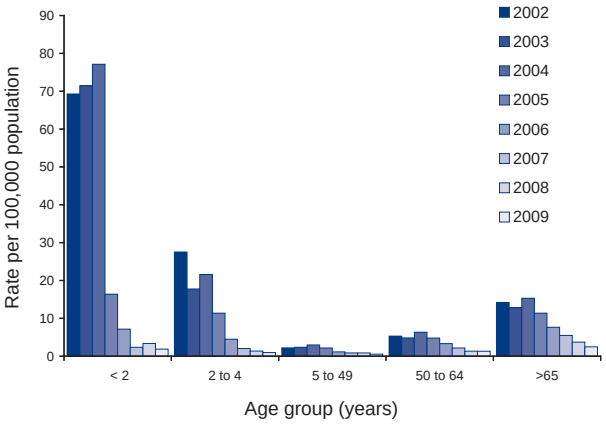
Figure 47: Notification rate for invasive pneumococcal disease, Australia, 2009, by age group and sex



caused by 7vPCV serotypes have declined between 2004 and 2009 from 7.7 to 1.0 per 100,000 (1,548 to 216 notifications). The decline was seen across all age groups (Figure 48). Those aged 65 years or more had the greatest rate of IPDs caused by 7vPCV serotypes in 2009 (72, 2.5 per 100,000) with those aged less than 2 years having a rate of 1.9 per 100,000 (11 notifications).

Additional data were collected on notifications of IPD in all Australian jurisdictions during 2009. More detailed analyses can be found in the IPD annual report series published in CDI.

Figure 48: Notification rate for invasive pneumococcal disease caused by 7vPCV serotypes, Australia, 2002 to 2009, by age group

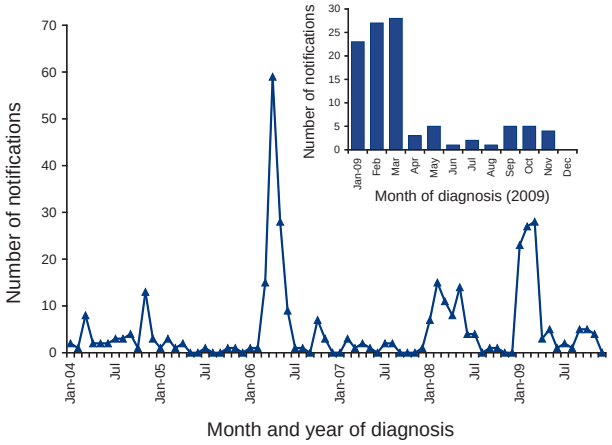


Measles

Measles is a highly infectious, acute viral illness spread by respiratory secretions, including airborne transmission via aerosolised droplets. The prodrome, lasting 2 to 4 days, is characterised by fever and malaise followed by a cough, coryza and conjunctivitis. It is usually followed by a maculopapular rash, which typically begins on the face, and then becomes generalised. Measles can be a severe disease, with complications such as otitis media, pneumonia, and acute encephalitis. Subacute sclerosing panencephalitis (SSPE) is a late, rare (approximately 1 in 100,000 cases) complication of measles,[21] which is always fatal.[12] Evidence suggests that endemic measles has been eliminated from Australia, since at least 2005.[58]

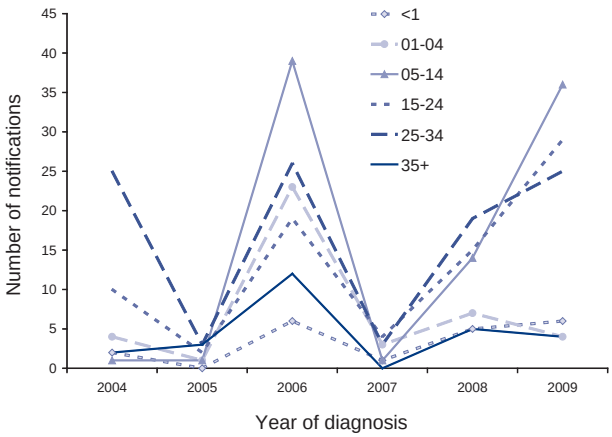
There were 104 notifications of measles reported to NNDSS in 2009, representing a rate of 0.5 notifications per 100,000 population. This represents an increase from the 65 notifications reported in 2008 (0.3 notifications per 100,000 population) and the 12 reported in 2007 (0.1 per 100,000) (Figure 49). In 2009, notifications were reported from all states and territories: Victoria (36), Queensland (32), New South Wales (19), Western Australia (10), South Australia (3), Tasmania (2), the Australian Capital Territory (1) and the Northern Territory (1).

Figure 49: Notifications of measles, Australia, 2004 to 2009, by month and year of diagnosis



In 2009, 63% (66/104) of measles notifications were male. The age at diagnosis ranged from 6 months to 56 years with the median age being 16 years. There was an increase in notifications in three age groups (5–14 years, 15–24 years and 25–34 years) compared with 2008, while the remaining three age groups (< 1 year, 1–4 years and 35+) remained relatively constant compared with 2008 (Figure 50). This increase was highest in the 5–14 years age group with 36 notifications in 2009 compared with 14 in 2008; in part influenced by an outbreak in a Sunshine Coast High School in Queensland, in which 18 cases were in this age group.

Figure 50: Notifications of measles, Australia, 2004 to 2009, by age group and year of diagnosis

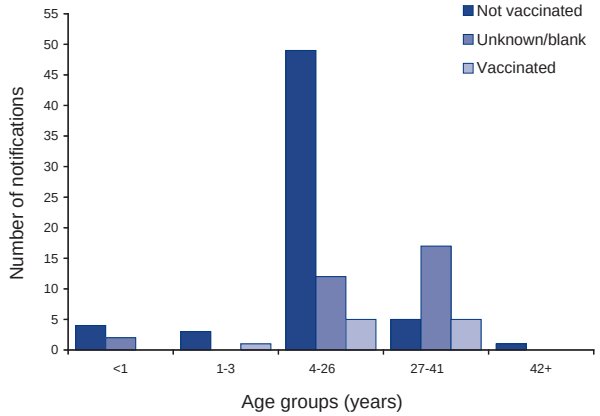


In 2009, 34% (35/104) of notifications were reported as being acquired from overseas including: Vietnam (11), India (6), Thailand (4), the Philippines (2), the United Kingdom (2), the United States of America (2), New Zealand (2) and 1 importation each from Iran, France, South Africa, China, Indonesia and Korea. Of the remaining 69 locally-acquired cases, 62 were epidemiologically linked to the imported cases. There were 3 outbreaks with more than 5 cases during 2009: one with 25 cases in a Sunshine Coast high school in Queensland that was linked to an imported case from India, one with 20 cases in Victoria that was also linked to an imported case from India, and one with 11 cases in Victoria that was linked to an imported case from Iran. Seventy-six of the 104 cases were linked to specific genotypes of which 29 were identified as D4, 26 as D8, 19 as H1 and 2 as D9.

Two doses of the MMR vaccine are funded under the NIP for children and provided at 12 months and 4 years of age. The MMR induces long-term measles immunity in 95% of recipients after a single dose and 99% of recipients after the 2nd dose.[12]

Nationally, there was information on vaccination for 70% (73/104) of notifications in 2009 of which 85% (62/73) were not vaccinated and 15% (11/73) had been vaccinated (1 with 2 doses and 9 with 1 dose and the remaining 1 case with no dose number stated; Figure 51). There were 6 notifications in infants less than 1 year of age at diagnosis who were ineligible for routine vaccination. Only one of the 4 cases in children between one and 3 years of age, who were eligible for 1 dose of the MMR, were vaccinated. Fifty-four notifications with vaccine information available were between four and 26 years of age and eligible for 2 doses of MMR. Ninety-one per cent (49/54) of those were not vaccinated and 9% (5/54) had been vaccinated. Two of those vaccinated in this age group had 2 doses and 2 cases had 1 dose of a measles-containing vaccine

Figure 51: Notifications of measles, Australia, 2009, by age group and vaccination status



and the remaining case had no dose number stated. There were 10 notifications with information on vaccination in the 27–41 years age group. This age group is considered to be a susceptible age cohort because many may have missed being vaccinated as infants when coverage was still low and the risk of natural immunity through exposure was declining. Of these, 50% were not vaccinated. Of the 5 vaccinated cases in this age group, four had 1 dose and the additional case had no dose number stated. The remaining 1 notification was in the 42 years or over age group and not vaccinated.

Mumps

Mumps is an acute viral illness transmitted by the respiratory route in the form of air-borne droplets or by direct contact with saliva of an infected person. A high proportion of mumps infections involve non-specific symptoms including fever, headache, malaise, myalgia and anorexia with approximately one-third of infections being asymptomatic. The characteristic bilateral, or occasionally unilateral, parotid swelling occurs in 60% to 70% of clinical cases.[21]

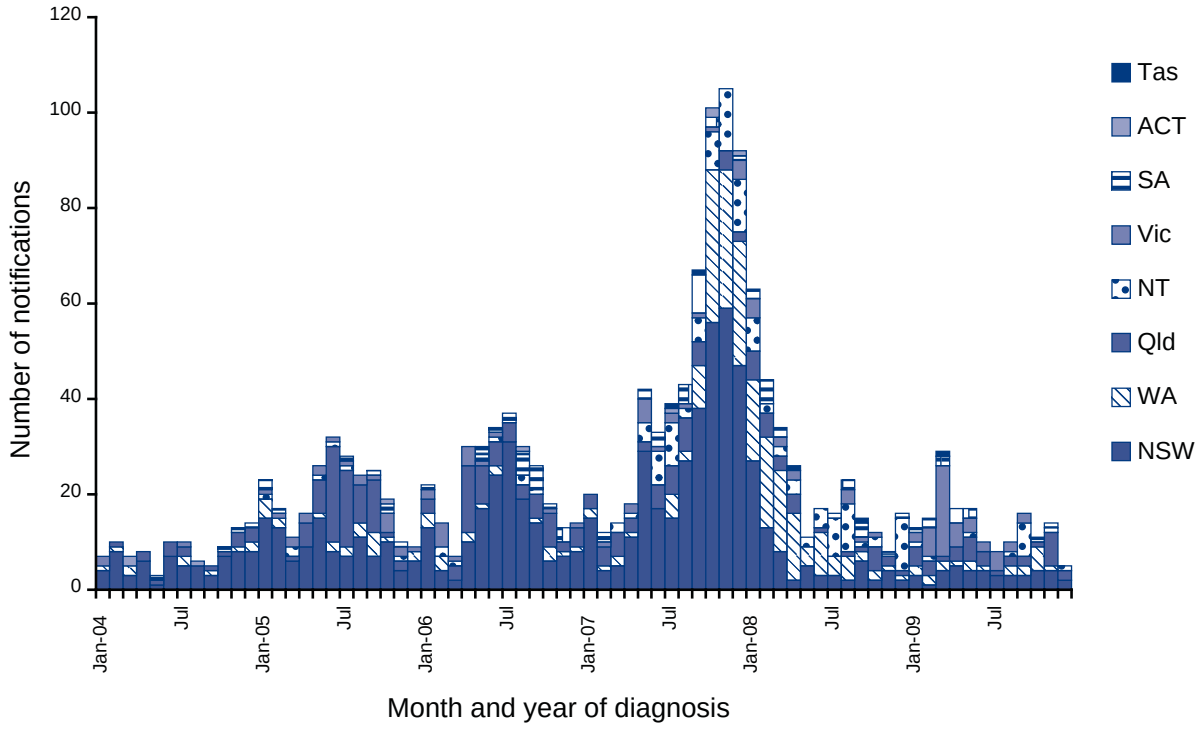
In 2009, there were 165 notifications of mumps (0.8 notifications per 100,000 population), compared with the 285 notifications (1.3 per 100,000) reported in 2008 (Figure 52) and a ratio of 0.6 compared with the 5-year mean. The crude national rate has continued to decrease in 2009 after increasing from 2004 and peaking in 2007 at 2.8 per 100,000 (Figure 53).

Notifications in 2009 were reported from all jurisdictions except the Australian Capital Territory, with 27% (45/165) from Victoria, 24% (40/165) from New South Wales and 21% (34/165) from Queensland. The highest rate was in the Northern Territory with 5.8 notifications per 100,000 population (13 notifications) followed by Western Australia with 0.9 per 100,000 (20 notifications).

There were no large mumps outbreaks in 2009. There were 2 small clusters notified to the NNDSS, the 1st involving 6 locally-acquired cases in Victoria and the 2nd was a localised cluster of 8 cases in a small country town in the Northern Territory.

In 2009, there were notifications of mumps in all age groups with the highest rates amongst adolescents (15–24 years) and young adults (25–34 years; Figure 53) reflecting historical vaccination schedules. Adolescents aged 15–24 years were eligible for 2 doses of a mumps-containing vaccine; however, coverage with the 2nd dose may have been suboptimal for some members of this cohort as they would have no longer been in primary school during the 1998 Measles Control Campaign (MCC). Only a small proportion of the young adults aged 25–34 years would have been eligible for 2 doses of

Figure 52: Notifications of mumps, Australia, 2004 to 2009, by state or territory and month of diagnosis



a mumps-containing vaccine, and many would not have been eligible for 1 dose. However, some of this cohort would have developed natural immunity as exposure to wild virus was still likely when they were young children.[59]

In 2009, the highest rates were for males in the 20–24 and 25–29 years age groups (Figure 54), which is similar to 2008. Sixty per cent of notifications (99/165) were male, which is a similar proportion to the 5-year mean.

Indigenous status was reported for 65% (107/165) of mumps notifications, of which 90% (96/107) were reported as non-Indigenous and 10% (11/107) as Indigenous. This represents a 40% decrease in the proportion of Indigenous vs non-Indigenous notifications in 2009 compared with 2008 in which 50% were reported as Indigenous. The higher rate of Indigenous notifications in 2008 was influenced by an outbreak amongst Indigenous communities in the Kimberley region of Western Australia and the Northern Territory.[60]

The mumps component of the MMR vaccine has been estimated to be the least effective of the 3 components ranging from providing 62%–88% and 85%–95% protection for the 1st and 2nd dose

Figure 53: Notification rate for mumps, Australia, 2004 to 2009, by age group

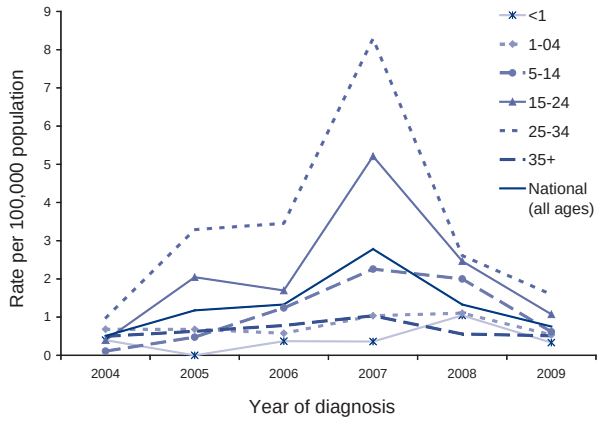
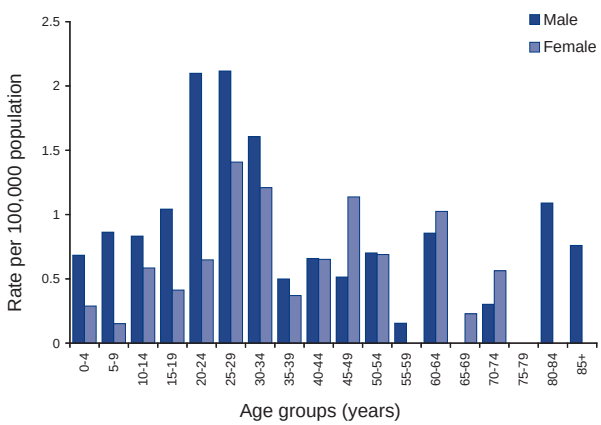


Figure 54: Notification rate for mumps, Australia, 2009, by age group



respectively.[61, 62] Reduced effectiveness of the mumps vaccine has been demonstrated over time such that waning immunity may at least partially account for the proportion of vaccinated mumps cases, and contribute to mumps outbreaks in older vaccinated populations.[62]

Nationally, information on vaccination was available for 34% (56/165) of the notifications, of which 59% (33/56) were not vaccinated and 41% (23/56) were vaccinated. The remaining 66% (109/165) were reported as not applicable or unknown. Of the vaccinated notifications 26% (6/23) had 2 doses and 43% (10/23) reported one dose of a mumps-containing vaccine, with the remaining seven having dosage information missing or unknown. Nine of the 11 Indigenous notifications had a reported vaccination status of which 89% (8/9) were vaccinated, one with 2 doses and seven with 1 dose of a mumps-containing vaccine, and one was not vaccinated.

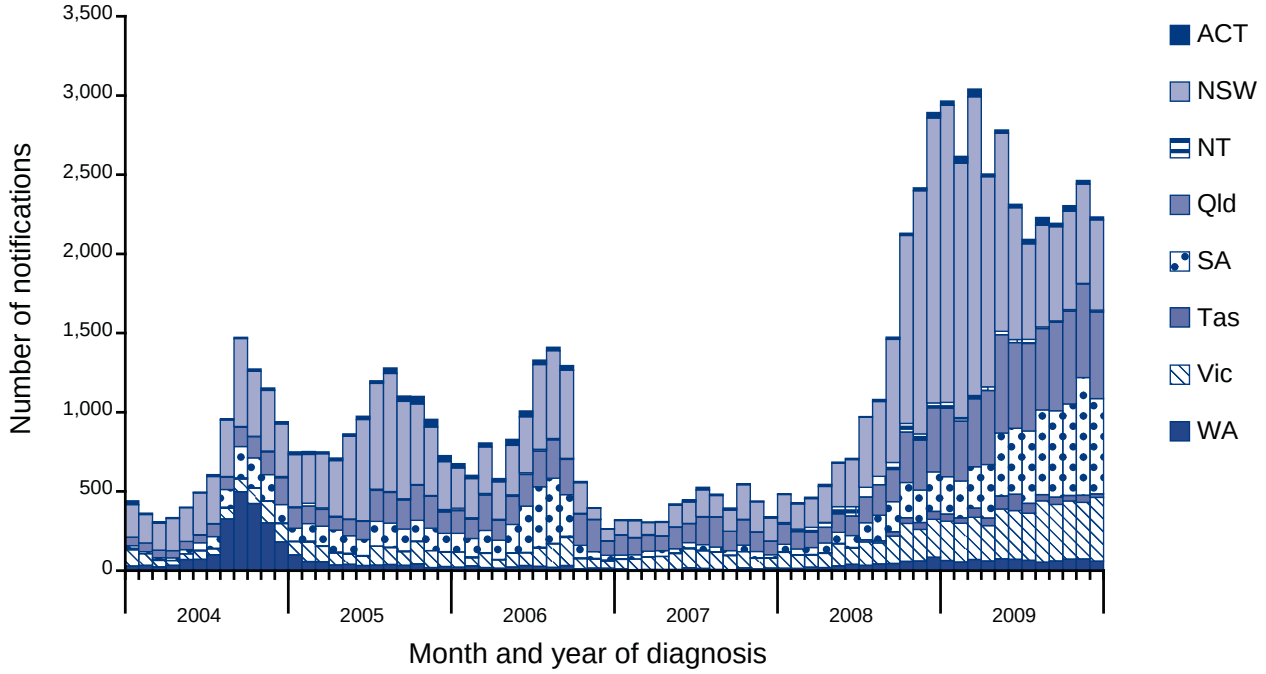
Pertussis

Pertussis is the most common vaccine preventable illness in Australia after influenza. It is a highly infectious disease caused by *Bordetella pertussis* and spread by respiratory droplets. Epidemics occur at regular intervals of approximately 3 to 4 years, which can vary from region to region, on a background of endemic circulation. In vaccinated populations these outbreaks tend to be smaller with less mortality and morbidity than in unvaccinated populations.[21] While pertussis can affect people of any age, infants are at highest risk of more severe disease as maternal antibody does not provide reliable protection and adequate immunity is not achieved through vaccination until receiving a 2nd dose at 4 months of age.[63] The majority of notifications usually occur in the spring and summer months.

In 2009, 29,736 notifications of pertussis including 2 deaths were reported to NNDSS. This represents a notification rate of 135.9 notifications per 100,000 population, a 2-fold increase in notifications compared with 2008 (14,285; 66.7 per 100,000) and 3 times the 5-year mean (9,764). Both deaths were in infants less than 2 months of age and too young to be protected by vaccination. The increase in notifications reflects the Australia-wide epidemic that began in mid-2008. (Figure 55). The causes of this epidemic are unclear but contributing factors may include suboptimal vaccine coverage, improved testing methods and case finding, and waning immunity levels in the vaccinated population.

In response to the nation-wide outbreak, many states and territories implemented public awareness campaigns and funded a booster vaccination program for parents of infants as part of a cocooning strategy to protect vulnerable infants from infection. These jurisdictions included Victoria and Queensland (for parents), the Northern Territory (for parents, carers and siblings of babies under 7 months of age), New South Wales (for parents, grandparents and other carers of infants) and the Australian Capital Territory (for parents and grandparents).

Figure 55: Notifications of pertussis, Australia, 2004 to 2009, by state or territory and month of diagnosis



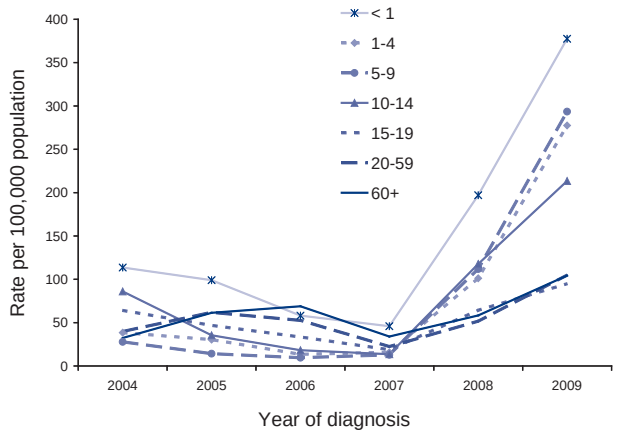
The government convened a Pertussis Working Party of the Australian Technical Advisory Group on Immunisation (ATAGI) in February 2009 to review the NIP schedule in light of the epidemic. ATAGI is continuing to consider all the available scientific evidence including options to optimise protection for babies in particular.

Notification rates in 2009 varied widely with age. Children aged less than 15 years had a higher rate of infection (271.8 notifications per 100,000 population) than those adolescents and adults 15 years of age or over (103.8 per 100,000) giving a rate ratio of 2.6. While this was similar to the 2008 rate ratio of 2.2, it contrasts markedly with the 2007, 2006, and 2005 rate ratios of 0.7, 0.3 and 0.5 respectively, reflecting the higher rate in adults relative to children during those years.

The highest rates amongst children were in infants less than 1 year of age (377.5 notifications per 100,000 population) followed by those aged 5–9 years (293.5 per 100,000) and those aged 1–4 years (277.5 per 100,000). This trend reflects that of 2008 but contrasts with 2007 when age group rates were more closely clustered.

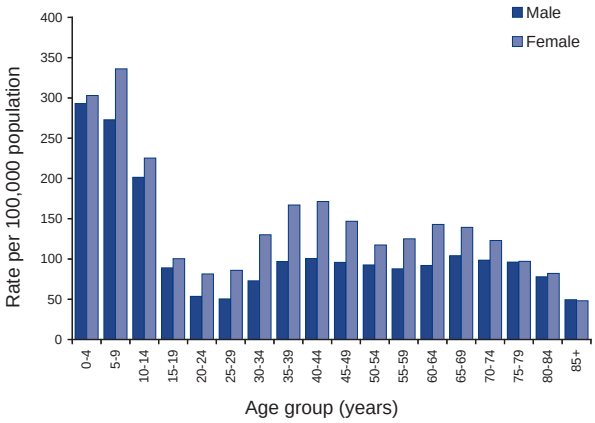
Between 2004 and 2007, a period inclusive of the last national epidemic in 2005/2006, age group notification rates were either trending down or remaining relatively constant but since 2007 rates have been increasing. This increase is most marked amongst those less than 15 years of age (Figure 56).

Figure 56: Notification rate for pertussis, Australia, 2004 to 2009, by age group



Fifty-seven per cent (16,858/29,736) of notifications in 2009 were female and 43% (12,837/29,736) were male, 41 had no sex specified (Figure 57). The highest rate amongst females was in the 5–9 years age group (336.1 per 100,000 population) with the highest rate in males being in the 0–4 years age group (293.0 per 100,000). While rates for both sexes were highest in those aged less than 15 years, the pattern of predominance of female notification rates compared with males occurred in all age groups except for those aged 75 years or over.

Figure 57: Notification rate for pertussis, Australia, 2009, by age group and sex



Follow-up is required in order to determine the vaccination status of individual cases. In a large outbreak, follow-up of all cases is not possible and as per national guidelines, jurisdictions prioritised follow-up to those less than 5 years of age. This age group made up 14% (4,266/29,736) of all notifications in 2009.

While the pertussis vaccine is not 100% effective and does not confer life-long immunity, vaccine effectiveness is estimated to be 68% after receiving 1 dose of vaccine, increasing to 92% after the 2nd dose,[64] and greater than 99% following subsequent doses.[65] Immunity to disease decreases over time post vaccination with estimates of protection remaining for 4–12 years.[64] The current vaccine schedule for pertussis under the NIP includes a dose provided at 2, 4 and 6 months of age followed by a booster at 4 years of age and again at 12–17 years of age (the timing of this last booster dose varies by jurisdiction).

Nationally, information on vaccination was available for 86% (3,676/4,266) of all notifications in children less than 5 years of age of which 80% (2,937/3,676) had received at least 1 pertussis containing vaccine and 18% (658/3,676) were not vaccinated. No data were entered or vaccination status was unknown for 14% (590/4,266) of the total notifications in this age group.

Of those 1,126 notifications less than one year of age, 54% (604/1,126) were vaccinated of which 41% (245/604) had received two or more doses of a per-

tussis vaccine. Twenty-one per cent (236/1,126) were less than 6 weeks of age and too young for their first scheduled dose of vaccine at 2 months.

Of 1,270 notifications for children aged between 3.5 and less than 5 years of age eligible for the first booster dose, only 15% (194/1,270) were reported as having had this 4th dose.

Pertussis notification rates varied considerably by state or territory and residential location. By jurisdiction, rates were highest in South Australia (5,346, 329.4 notifications per 100,000 population) followed by New South Wales (12,436, 175.2 per 100,000), Queensland (6,216, 141.1 per 100,000), Tasmania (616,122.6 per 100,000), the Australian Capital Territory (351, 99.9 per 100,000), the Northern Territory (215, 95.6 per 100,000), Victoria (3,778, 69.6 per 100,000) and Western Australia (778, 34.8 per 100,000).

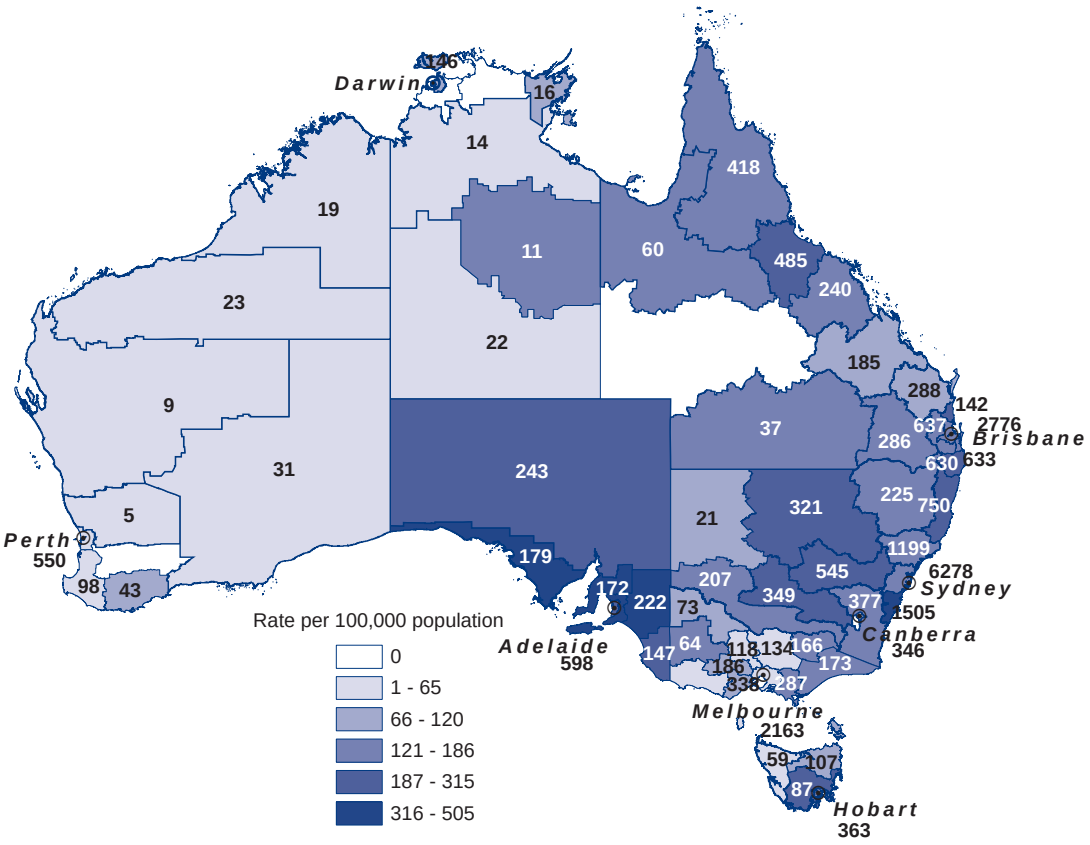
Rates by SD also varied widely across most jurisdictions except for South Australia where they were uniformly high (Map 3). In New South Wales rates were highest in the Illawarra SD (1,505 notifications, 349.1 notifications per 100,000 population), with high rates also reported from coastal, north and central western areas. In Queensland rates were highest in the Northern SD (485, 213.3 per 100,000) followed by the Sunshine Coast (637, 197.0 per 100,000). The East Gippsland and Ovens-Murray SDs had the highest rates in Victoria (173 and 166 respectively, 199.3 and 166.2 per 100,000), while in Tasmania, the Southern and Greater Hobart SDs had the highest rates (87 and 363 respectively, 232.3 and 171.2 per 100,000). Western Australia's rates remained low compared with the rest of Australia in 2009. Rates by SD should be interpreted with caution, as they can be high or low depending on the size of the population.

Poliomyelitis

Poliomyelitis is a highly infectious disease caused by gastrointestinal infection by a poliovirus. Transmission occurs primarily from person to person via the faecal-oral route. In most cases poliovirus infection is not symptomatic however in less than 1% of cases the virus may invade the nervous system and cause acute flaccid paralysis (AFP).[21]

In 2009, there were no notifications of poliomyelitis in Australia, which along with the Western Pacific Region remained poliomyelitis free. Poliomyelitis

Map 3: Notification rates and counts* for pertussis, Australia, 2009, by Statistical Division of residence and Statistical Subdivision for the Northern Territory



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

is a notifiable disease in Australia with clinical and laboratory investigation conducted for cases involving patients of any age with a clinical suspicion of poliomyelitis. Australia follows the WHO protocol for poliomyelitis surveillance and focuses on investigating cases of AFP in children under 15 years of age. The WHO target for AFP surveillance in a polio non-endemic country is 1 case of AFP per 100,000 children aged less than 15 years. Between 1 January and 31 December 2009 there were 54 eligible AFP cases notified to the National Polio Reference Laboratory (NPRL) of which all were classified as non-poliomyelitis. The 2009 non-poliomyelitis AFP rate was 1.17 hence meeting the WHO AFP surveillance indicator for the 6th time since 1995. Details of the 2009 notifications are provided in the 2009 annual report of the Australian NPRL.[66]

Rubella

Rubella is generally a mild and self-limiting viral infectious disease. It is spread from person to person through direct contact with respiratory secretions or via air-borne droplets. Clinically, rubella can be difficult to distinguish from other diseases that cause a febrile rash, such as measles, and is asymptomatic in up to 50% of cases. Rubella infection in pregnancy can cause foetal infection resulting in congenital rubella syndrome (CRS). CRS occurs in up to 90% of infants born to women who are infected during the first 10 weeks of pregnancy and may result in foetal malformations and death.[21]

In 2009, there were 27 notifications of rubella (0.1 notifications per 100,000 population), a decrease compared with the 36 notifications in 2008. Notifications were reported from New South Wales (7), Victoria (6), Queensland (6), Western Australia (5) and South Australia (3). The age profile of rubella notifications in 2009 was similar to 2008. There were small numbers of notifications reported across the age groups with none in infants less than 1 year of age or for adults over 60 years of age (Figure 58). The median age was 29 years and 74% (20/27) of notifications were adults between 20 and 49 years of age. The overall male to female ratio of notified cases in 2009 was 1.7:1, (17 males and 10 females). Of the 10 females, 80% were notified in women of child-bearing age (17–47 years). There were no notifications of CRS reported in 2009.

Figure 59 shows that rubella notifications in different age groups have continued to trend at low levels since 2004, except for a spike amongst the 25–34 years age range in 2006. This spike was primarily due to an increase of notifications from the South Eastern and Central Sydney SDs in New South Wales. It was concentrated in those aged 15–44 years, however there was no single identifiable source for the increase in notifications.[67]

A single dose of rubella vaccine produces an antibody response in more than 95% of recipients and while antibody levels are lower than after natural infection, they are shown to persist for at least 16 years in the absence of endemic disease.[12] Rubella vaccine is included in the combined MMR vaccine and provided under the NIP at 12 months and 4 years of age.

Figure 58: Notifications of rubella, Australia, 2009, by age group and sex

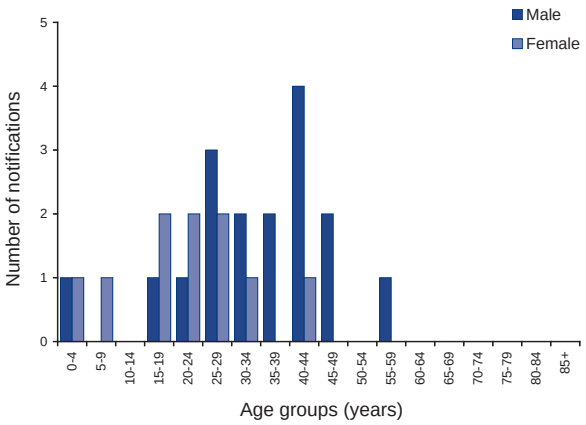
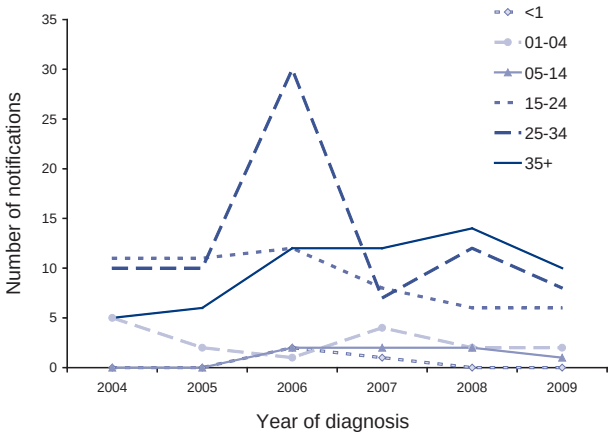


Figure 59: Notifications of rubella, Australia, 2004 to 2009, by age group



Nationally, information on vaccination was available for 48% (13/27) of rubella notifications of which the majority, 62% (8/13), was not vaccinated and 38% (5/13) were vaccinated. The remaining 52% (14/27) were stated as either unknown or blank. Of the 8 male notifications with information on vaccination reported, 71% (5/8) were not vaccinated, all of whom were adults in the 18–44 years age range, and three had received 1 dose of a rubella-containing vaccine. Of the 5 female notifications in 2009 with vaccination information reported, 60% (3/5) were not vaccinated (two were women of child-bearing

age: 17-year-old and 24-year-old) and two had received at least 1 dose of a rubella-containing vaccine (4-year-old and 40-year-old).

None of the rubella notifications in 2009 was identified as Indigenous, although seven of the 27 were of unknown status.

Tetanus

Tetanus is an acute, often fatal, disease caused by the toxin produced by the bacterium *Clostridium tetani*. Tetanus spores usually enter the body through contamination of a wound with soil, street dust or animal or human faeces.[21] The neurotoxin acts on the central nervous system to cause muscle rigidity with painful spasms. Generalised tetanus, the most common form of the disease, is characterised by increased muscle tone and generalised spasms. Early symptoms and signs include increased tone in the jaw muscles, difficulty in swallowing, stiffness or pain in the neck, shoulder and back muscles. In Australia, tetanus is rare, occurring primarily in older adults who have never been vaccinated or were vaccinated in the remote past.[12]

Tetanus vaccination stimulates the production of antitoxin, which protects against the toxin produced by the organism. Complete immunisation (3 primary doses and 2 boosters included for children on the NIP) induces protective levels of antitoxin lasting throughout childhood, but by middle age about 50% of vaccines have low or undetectable levels. It is recommended, though not funded under the NIP, that all adults who reach the age of 50 years and have not received a booster of a tetanus-containing vaccine in the previous 10 years, should have one.[12]

In 2009, there were 3 notifications of tetanus, two from New South Wales and one from Victoria, all over 34 years of age. Of the 3 notifications, 2 male and 1 female, one had been partially vaccinated with 1 dose and the remaining two had no vaccination status recorded.

Varicella zoster virus infections

Chickenpox (also known as varicella) and shingles (also known as herpes zoster) are both caused by the varicella-zoster virus (VZV). VZV is a member of the herpesvirus family and is highly contagious. Chickenpox occurs on initial infection with the virus. The virus then stays dormant in the body's nerve cells and has a 20%–30% chance of reactivating as shingles later in life.[21]

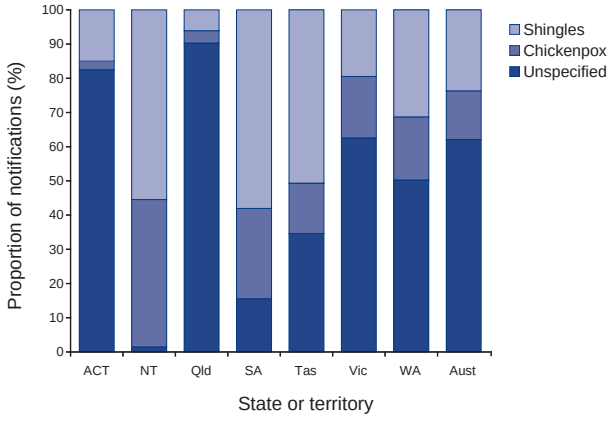
In November 2005, the varicella-zoster vaccine was added to the NIP schedule as a single dose due at 18 months (for children born on or after 1 May 2004), or as a catch-up dose at 10–13 years of age. In 2006, the Communicable Diseases Surveillance Network Australia (CDNA) agreed to make 3 categories of VZV infection notifiable: chickenpox, shingles and varicella infection (unspecified). The year 2009 was the first complete year in which all jurisdictions, except New South Wales, sent VZV data to NNDSS.

In 2009, there were 11,235 VZV notifications from the 7 reporting jurisdictions. This was 10% more than in 2008. Sixty-two per cent (6,977) were unspecified varicella infection, 14% (1,599) were chickenpox and 24% (2,659) were shingles.

Varicella-zoster virus infection (unspecified)

Notifications of unspecified VZV infections are laboratory specimens that are positive for VZV but have not been followed up by the local health authority and distinguished clinically as either chickenpox or shingles. Although varying by jurisdiction (Figure 60), VZV (unspecified) accounted for 62% of all VZV notifications in 2009, an increase compared with 52% of the total in 2008.

Figure 60: Proportion of total notifications for varicella-zoster virus unspecified, chickenpox and shingles, 2009, by state or territory*



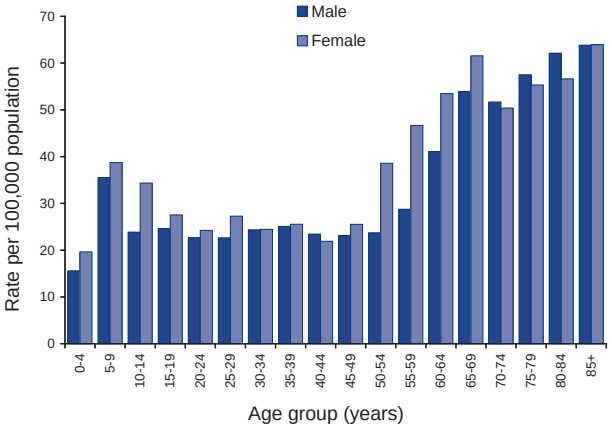
* Excluding New South Wales.

There were 6,977 notifications of VZV infections (unspecified) based on laboratory diagnoses compared with 4,415 in 2008, with a rate of 47.2 notifications per 100,000 population. The high proportion of unspecified VZV infection compared with chickenpox or shingles is attributable to the varying capacity of jurisdictions to follow-up on laboratory notifications to determine the clinical presentation of each case. The highest rates were reported from

Queensland (3,835 notifications, 87.0 per 100,000 population), Western Australia (866, 38.7 per 100,000) and Victoria (1,847, 34.0 per 100,000).

The age and sex distribution of unspecified VZV are shown in Figure 61.

Figure 61: Notification rate for varicella-zoster virus unspecified, Australia,* 2009, by age group and sex



* Excluding New South Wales.

Chickenpox

Chickenpox is a highly contagious infection spread by air-borne transmission of droplets from the upper respiratory tract or from the vesicle fluid of the skin lesions of chickenpox or shingles infections. Chickenpox is usually a mild disease of childhood, however complications occur in approximately 1% of cases. It is more severe in adults and in individuals of any age with impaired immunity, in whom complications, disseminated disease, and fatal illness can occur.[12]

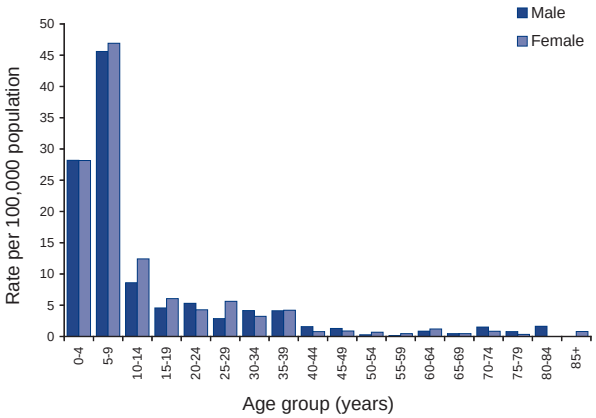
In 2009, there were a total of 1,599 notifications of chickenpox reported compared with 1,795 in 2008 and, a rate of 10.8 notifications per 100,000 population. The highest rates were reported from the Northern Territory (87 notifications, 38.7 per 100,000 population) and South Australia (475, 29.3 per 100,000) reflecting the increased case ascertainment in these jurisdictions due to their practice of following up VZV notifications.

Sixty-four per cent of notifications (1,028) occurred in children aged less than 10 years. The highest rates were in the 5–9 years age group (627 notifications, 46.2 per 100,000 population; Figure 62). In 2009, the rate for children aged less than 4 years (28.2 per 100,000) was approximately half of the 2008 rate (59.0 per 100,000).

Indigenous status was recorded for 98% (1,569) of notifications, the majority of which 82% (1,305) were non-Indigenous.

Of the 1,599 notifications for chickenpox, information on vaccination was available for 30% (543/1,790) and 80% (432/543) of these were unvaccinated.

Figure 62: Notification rate for chickenpox, Australia,* 2009, by age group and sex



* Excluding New South Wales.

Shingles

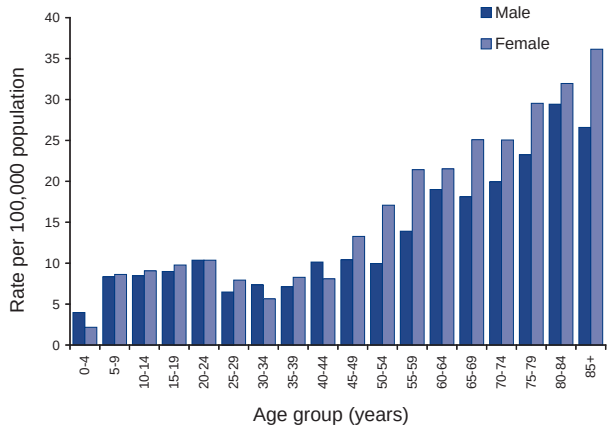
Shingles occurs most commonly with increasing age (> 50 years), impaired immunity, and a history of chickenpox in the first year of life. Reactivation of VZV causing shingles is thought to be due to a decline in cellular immunity to the virus, and in the majority of cases presents clinically as a unilateral vesicular rash in a dermatomal distribution. Associated symptoms may include headache, photophobia, malaise, and an itching, tingling, or severe pain in the affected dermatome. In the majority of patients shingles is an acute and self-limiting disease however, complications develop in approximately 30% of cases, the most common of which is chronic severe pain or post-herpetic neuralgia.[21]

There were 2,659 notifications of shingles reported to NNDSS in 2009, an increase when compared with 2,309 in 2008, and a rate of 18.0 notifications per 100,000 population. The highest rates were in South Australia (1,045, 64.4 per 100,000) and the Northern Territory (112, 49.8 per 100,000).

There were more female notifications (1,470; 55.3%) than males (1,187; 44.7%), which was similar to 2008. The highest rates were in the 85 years or over age group (126, 32.9 per 100,000; Figure 63).

Indigenous status was recorded for 81% (2,166) of notifications, the majority of which 97%, (2,102/2,166) were non-Indigenous.

Figure 63: Notification rate for shingles, Australia,* 2009, by age group and sex



* Excluding New South Wales.

Vectorborne diseases

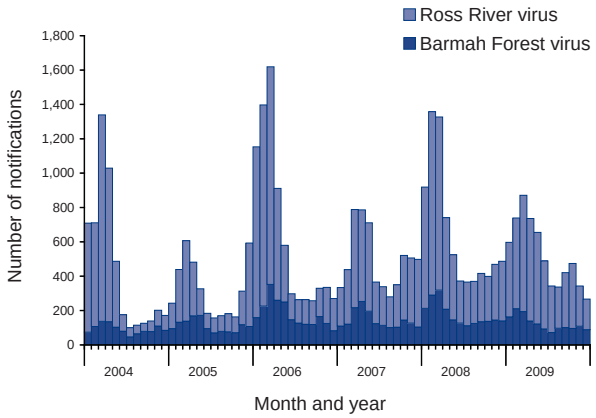
A disease that is transmitted to humans or other animals by an insect or other arthropod is known as a vectorborne disease. Vectors of human disease of most concern in Australia are typically mosquitoes that are able to transmit viruses or parasites to humans.

During 2009, there were 8,232 notifications of mosquito-borne diseases reported to NNDSS (3.5% of total notifications). This was a 7% decrease in the number of notifications compared with 2008 (8,876). The notifiable mosquito-borne diseases include those caused by the alphaviruses (Barmah Forest virus and Ross River virus), flaviviruses (dengue, Murray Valley encephalitis, Kunjin, Japanese encephalitis and yellow fever) and malaria. Yellow fever is reported under quarantinable diseases. Geographical location rates for vectorborne disease notifications represent the place of residence rather than the place of acquisition of infection, although in many instances this may be the same. Further information about these vectorborne diseases can be found in the National Arbovirus and Malaria Advisory Committee Annual (NAMAC) annual report 2008–09.[68]

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 (BFV) and Ross River virus (RRV), which facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[69] In Australia, BFV and RRV are the alphaviruses of major public health significance, accounting for 76% (6,272 notifications) of the total mosquito-borne disease notifications for 2009. Between 2004 and 2009, notifications ranged annually for BFV from 1,100 (2004) to 2,133 (2006), and for RRV from 2,538 (2005) to 5,652 (2008) (Figure 64).

Figure 64: Notifications of Barmah Forest and Ross River virus infections, Australia, 2004 to 2009, by month and year of diagnosis



Barmah Forest virus infection

There were 1,486 notifications of BFV infections notified to NNDSS in 2009, which accounted for 18% of total mosquito-borne disease notifications for the reporting period. Fifty-four per cent of BFV notifications were reported from Queensland (799 notifications) and 24% from New South Wales (359 notifications). BFV notifications during 2009 were 0.9 times the mean for the previous 5 years.

Cases were reported in all jurisdictions. The highest rates of BFV notifications were reported by the Northern Territory (52.0 notifications per 100,000 population) and Queensland (18.1 notifications per 100,000 population). Cases were reported in all jurisdictions. The national BFV notification rate in 2009 was 6.8 notifications per 100,000 population, compared with 9.8 notifications per 100,000 population in 2008. Overall, 55% of BFV notifications reported to NNDSS were males.

Ross River virus infection

There were 4,786 notifications of RRV infections reported to NNDSS in 2009, which accounted for 58% of the total mosquito-borne disease notifications received during this period. The majority of notifications in 2009 were from Queensland (45%,

2,154 notifications) and New South Wales (19%, 912 notifications).

The highest rates of RRV notifications were reported by the Northern Territory (189.9 notifications per 100,000 population) and Queensland (48.9 notifications per 100,000 population). Cases were reported in all jurisdictions. The national RRV notification rate for 2009 was 21.9 notifications per 100,000 population compared with 26.4 notifications per 100,000 population in 2008. Overall, 47% of RRV notifications reported to NNDSS were males.

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 Programme is a surveillance scheme involving New South Wales, the Northern Territory, Victoria and Western Australia. Chicken flocks are located in strategic locations and are regularly tested for antibodies to MVEV and KUNV. This program is designed to provide early warning of flavivirus activity (excluding DENV and JEV).[70] A sentinel chicken surveillance report was published as part of the NAMAC annual report 2008–09.[68]

Murray Valley encephalitis virus infection

During 2009, 4 notifications of MVEV were reported to NNDSS. The 2 MVEV notifications from Western Australia involved a resident of Broome (March 2009) and a resident of Port Hedland (May 2009). Both these cases survived their illness but have long term neurological deficits.[71] The 2 MVEV notifications from the Northern Territory both died as a result of their illness. The 1st case was a long term resident from the Batchelor area (March 2009) and the other was a Queensland resident holidaying at Channel Point (May 2009). Health warnings were given both before and after the cases, with warnings based on vector numbers, rainfall, historical risk periods and/or detections of seroconversions in sentinel chicken flocks. During 2008, 4 notifications of MVEV were reported to NNDSS.

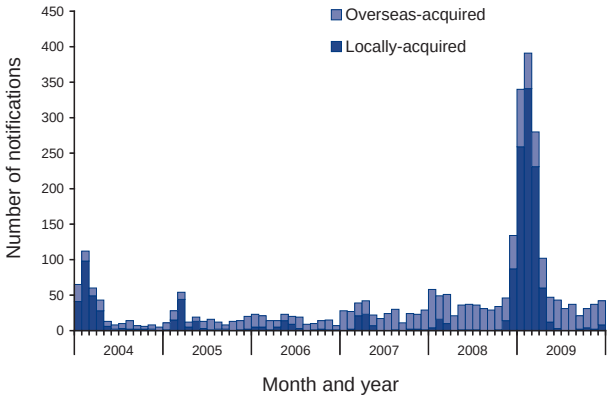
Kunjin virus infection

There were 2 notifications of KUNV reported to NNDSS in 2009, one from Queensland and one from the Northern Territory compared with 1 notification in 2008 from Queensland.

Dengue virus infection

There were 1,402 notifications of DENV infection reported to NNDSS in 2009 (Figure 65), of which 66% were locally acquired (922 notifications) and 34% (480 notifications) were acquired overseas. The number of cases reported in 2009 was a 150% increase in the number of cases reported in 2008 (562).

Figure 65: Notifications of dengue virus infection, Australia, 2004 to 2009, by month and year of diagnosis



Local transmission in Australia is restricted to areas of northern Queensland where the key mosquito vector, *Aedes aegypti*, is present. Dengue is not endemic to Queensland, but outbreaks occur when the virus is imported via international travellers or residents returning home from overseas. Queensland reported 1,036 notifications of DENV in 2009 (74% of all DENV notifications). Locally-acquired cases represented 66% (922) of the total number of dengue notifications in 2009. These were attributed to outbreaks of locally-acquired dengue, involving all 4 serotypes, in north Queensland.

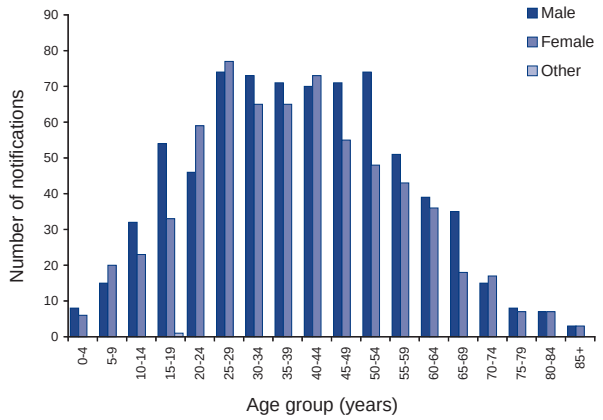
One dengue associated death was reported in March 2009. The last time death due to dengue fever was reported was in early 2004, when 2 deaths were reported in Australia. The latter were the first deaths attributed to dengue in over 100 years.[72]

In 2009, the largest number (99) of dengue notifications was in the 40–44 years age group and 53% were males (Figure 66).

Japanese encephalitis virus infection

There were no notifications of JEV reported to the NNDSS in 2009 compared with 1 notification of JEV in New South Wales in 2008. This case was in a man who had recently travelled to Japan and was the first JEV notification in Australia since 2004.

Figure 66: Notifications of dengue, Australia, 2009, by age group and sex



Arbovirus infections (NEC)

In 2009, there were 26 notifications of arbovirus infection (not elsewhere classified or NEC). There were 23 notifications in Queensland and three in Victoria. Overall, 58% of NEC notifications reported to NNDSS were males.

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 5 species – *vivax, falciparum, malariae*, *knowlesi* and *ovale*.[21] There were 526 notifications of malaria in Australia in 2009, compared with 529 in 2008 (Figure 67). There were no locally-acquired infections in 2009. Since Australia was declared malaria free in 1981 there have been 2 reported locally-transmitted outbreaks in 1986 and 2002 with a total of 15 cases. The majority of notifications in 2009 were reported by Queensland (35%, 185 notifications), Victoria (21%, 113 notifications), New South Wales (18%, 92 notifications), and Western Australia (16%, 82 notifications). Queensland reported that 51 of 185 notifications (28%) were acquired in Papua New Guinea.

The largest number (59) of malaria notifications was in the 20–24 years age group and 71% of malaria notifications were for males (Figure 68).

The infecting *Plasmodium* species was reported for 96% of malaria notifications in 2009 (Table 17). Of these 526 notifications, *P. falciparum* (42%) and *P. vivax* (48%) were the predominant species. New South Wales notified the first case of a fifth species, *Plasmodium knowlesi*, acquired in Indonesian Borneo.[73]

Zoonoses

Zoonoses are 'those diseases and infections which are naturally transmitted between vertebrate animals and man'.[74] Approximately 60%–70% of emerging human infectious diseases are zoonoses[75,76] and

Figure 67: Notifications of malaria (imported cases), Australia, 2004 to 2009, by state or territory and month and year of diagnosis

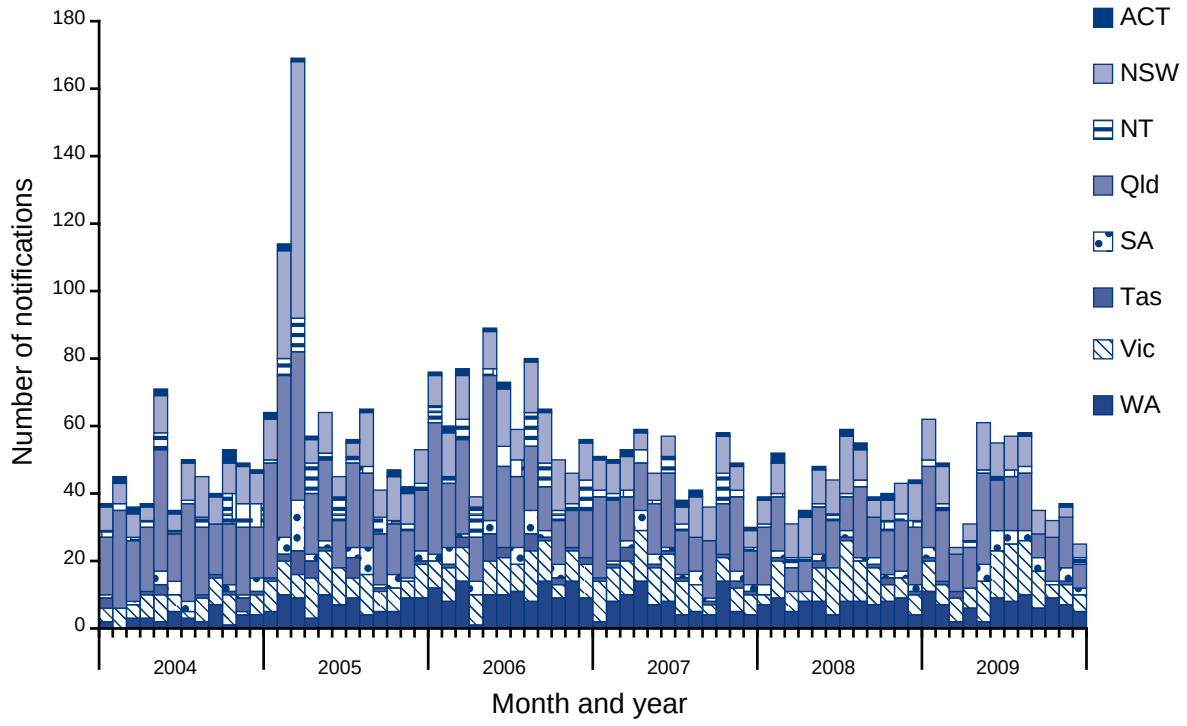
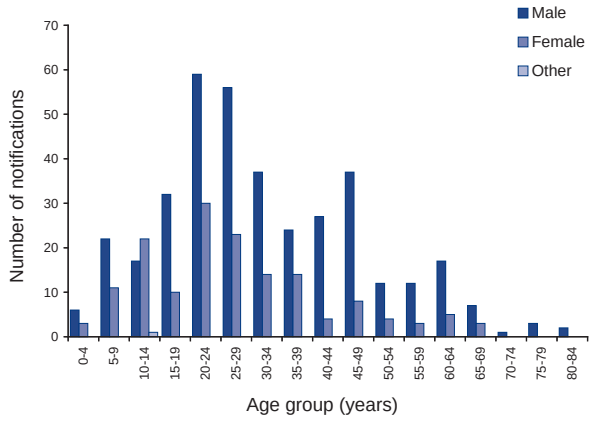


Table 17: Notifications of malaria, Australia, 2009, by parasite type and state or territory

Parasite type	State or territory									Type (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
Plasmodium falciparum	2	43	2	74	13	4	33	47	219	42
Plasmodium knowlesi	0	1	0	0	0	0	0	0	1	0
Plasmodium malariae	0	1	1	2	1	0	3	1	9	2
Plasmodium ovale	0	4	0	3	0	0	2	3	12	2
Plasmodium vivax	1	41	9	91	15	1	71	24	253	48
Plasmodium species	0	0	0	15	1	0	2	2	20	4
Mixed *P. falciparum* and other species*	0	1	2	0	2	0	2	5	11	2
Mixed other species*	0	1	0	0	0	0	0	0	1	0
Total	3	92	14	185	32	5	113	82	526	

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

Figure 68: Notifications of malaria, Australia, 2009, by age group and sex



more than 70% of emerging zoonoses originate from wildlife.[75] 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'.[77]

The zoonoses notifiable to the NNDSS included in this chapter are anthrax, Australian bat lyssavirus or lyssavirus (unspecified) infection, brucellosis, leptospirosis, ornithosis, Q fever, and tularaemia. During 2009, 552 notifications of these zoonotic diseases were made to the NNDSS. Of these, Queensland accounted for 48% (263 notifications) and New South Wales 33% (183 notifications) of the zoonotic diseases. Notification numbers were generally higher in males (78%, 552 notifications). There were only 12 notifications (2%) of zoonotic disease cases aged less than 15 years and 19 notifications (3%) in cases over the age of 70 years.

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

Anthrax

Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts.[21] Anthrax has a low incidence in animals, and occurs only sporadically in Australia.[78] It can be an occupational hazard for veterinarians, and agriculture, wildlife and industry livestock workers who handle infected animals or by-products.

No cases of anthrax were reported to NNDSS in 2009. Over the previous 10 years, only 2 human cases of anthrax were reported in Australia in 2006 and 2007,[79,80] both of which were cutaneous anthrax. Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

In 2009, 5 anthrax incidents were reported in livestock. Three occurred in New South Wales, where cases have been known to occur in the past, and two in north-eastern Victoria. In all instances, properties were subject to the recommended protocol of quarantine, disposal of carcasses, and vaccination and tracing of at-risk animals and their products. During 2009, an 'animal side' immunochromatographic test was used as a rapid anthrax screening test in Victoria to investigate sudden ruminant deaths. The results of this testing were consistent with confirmatory laboratory-based testing.[78]

Australian bat lyssavirus, rabies and lyssavirus (unspecified) infections

Classical rabies virus does not occur in Australia, although a related virus called Australian bat lyssavirus was identified in 1996 and is present in some Australian bats and flying foxes.[81] No notifications of either Australian bat lyssavirus infection (ABL), rabies or lyssavirus (unspecified) infections were reported to the NNDSS during 2009.

Only 2 known cases of ABL infection in humans have been reported in Australia, in 1996 and 1998. Both cases occurred after close contact with an infected bat and both were fatal.[82,83] Surveillance indicates that ABL infection may have been present in Australian bats for at least 15 years prior to its first detection. Sick and injured bats and changes in bat ecology pose an increased public health risk.[84] Bat testing conducted by the Australian Wildlife Health Network between January and December 2009 yielded 12 ABL detections compared with no detections in bats during 2008.[85]

Brucellosis

Brucellosis is mainly an occupational disease for farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[21] However, the most common source of human infection in Queensland, which reported 69% of cases, is infected feral pigs and inadequate measures by feral pig hunters to prevent brucellosis infection.[86]

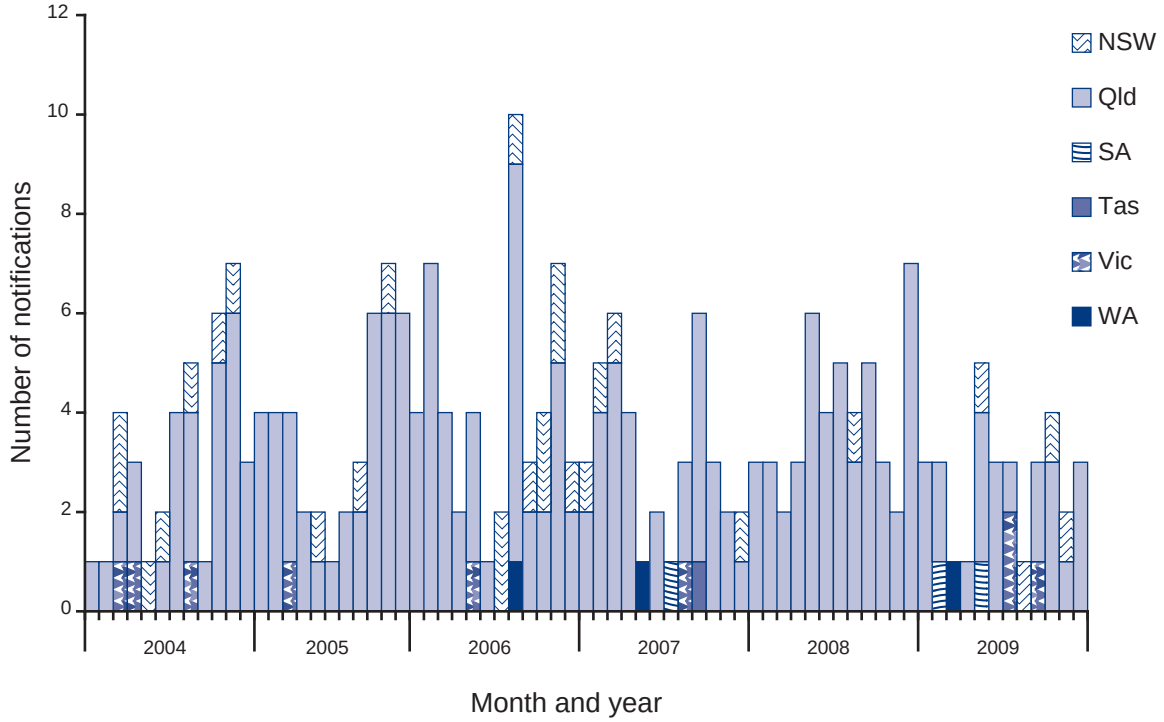
Several *Brucella* species can infect both animals and humans. Infections that can cause illness in humans include *Brucella melitensis* from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle.

In 2009, 32 notifications of brucellosis were reported to the NNDSS; a national notification rate of 0.1 notifications per 100,000 population, compared with 0.2 notifications per 100,000 population in 2008. Queensland reported 22 notifications, with New South Wales reporting four, Victoria three, South Australia two, and Western Australia one. There has been little change in the number of notifications of brucellosis over the last 6 years (Figure 69). In 2009, the majority of notifications were male (27) and aged between 15 and 49 years (25) in 2009.

Species data were available for 38% of notifications (12) of which eight were *B. suis* (all from Queensland). There were 4 imported cases of *B. melitensis* (Egypt, Saudi Arabia, Turkey and Kenya).

Bovine brucellosis (*B. abortus*) was eradicated from the Australian cattle herd in 1989 and is considered to be an exotic animal disease in Australia.[78] Caprine and ovine brucellosis (caused by *B. melitensis*) has never been reported in Australian sheep or goats.[78] Swine brucellosis (caused by *B. suis*) is confined to some areas of Queensland, where it occurs in feral pigs, with human cases predominantly seen in recreational feral pig hunters.[86] Swine brucellosis was not detected in any of Queensland's domestic piggeries during 2009.[78]

Figure 69: Notifications of brucellosis, selected jurisdictions, 2004 to 2009, by state or territory and month and year of diagnosis



Leptospirosis

Leptospirosis is caused by spirochaetes of the genus, *Leptospira,* which is found in the genital tract and renal tubules of domestic and wild animals. In affected areas, where there is exposure to infected urine of domestic and wild animals, this disease can be an occupational and recreational hazard (such as swimming or wading in contaminated water).[21]

Between 2004 and 2009 leptospirosis notifications ranged from 108 (2007) to 177 (2004) annually, with 146 notifications in 2009 (0.7 notifications per 100,000 population). Cases were reported in all jurisdictions except for South Australia and Tasmania (Figure 70). In 2009, the majority of notifications were from Queensland (110 notifications, 2.5 notifications per 100,000 population). Eighty-seven per cent of leptospirosis cases were male (127 notifications) and 82% of all cases were aged between 15 and 54 years (120 notifications).

The World Health Organization/Food and Agriculture Organization/World Organization of Animal Health Collaborating centre for reference and research on leptospirosis provided an annual surveillance report of leptospirosis cases in 2009. The most frequently identified leptospirosis serovars in 2009 were Arborea, Zanoni and Australis. Serovar Arborea was the most frequently reported during 2009, accounting for 29% (43) of all notifications and was a 79% increase on Arborea notifications reported in 2008 (24).[87] The last reported death in Australia attributed to leptospirosis was reported in 2002.[88]

Ornithosis

Ornithosis is caused by infection with the bacterium *Chlamydophila psittaci* and is transmitted to humans by exposure to waterfowl, seabirds, shore birds, pigeons and doves and many psittacine birds. Birds can become carriers of the disease without becoming symptomatic. The mode of transmission to humans is by inhaling bacteria, usually from contaminated dried faeces, nasal or eye secretions and dust from infected birds.[21] Person-to-person transmission is rare.

In 2009, 65 ornithosis infections were notified to NNDSS, giving a national rate of 0.3 notifications per 100,000 population. This was lower than the 2008 rate of 0.5 notifications per 100.000 population. Between 2004 and 2009, the annual number of ornithosis notifications has decreased from 239 to 65 respectively (Figure 71). The annual number of notifications in 2009 represents the lowest total number of ornithosis notifications since 2001.

Victoria had the highest number of notifications (38 notifications, 0.7 per 100,000 population). Notifications were also received from New South Wales (22), South Australia (3) and Western Australia (2). Sixty-five per cent of the notifications in 2009 were male (42 notifications) compared with 2008, where the minority of cases were male

Figure 70: Notifications of leptospirosis, Australia, 2004 to 2009 by state or territory and month and year of onset

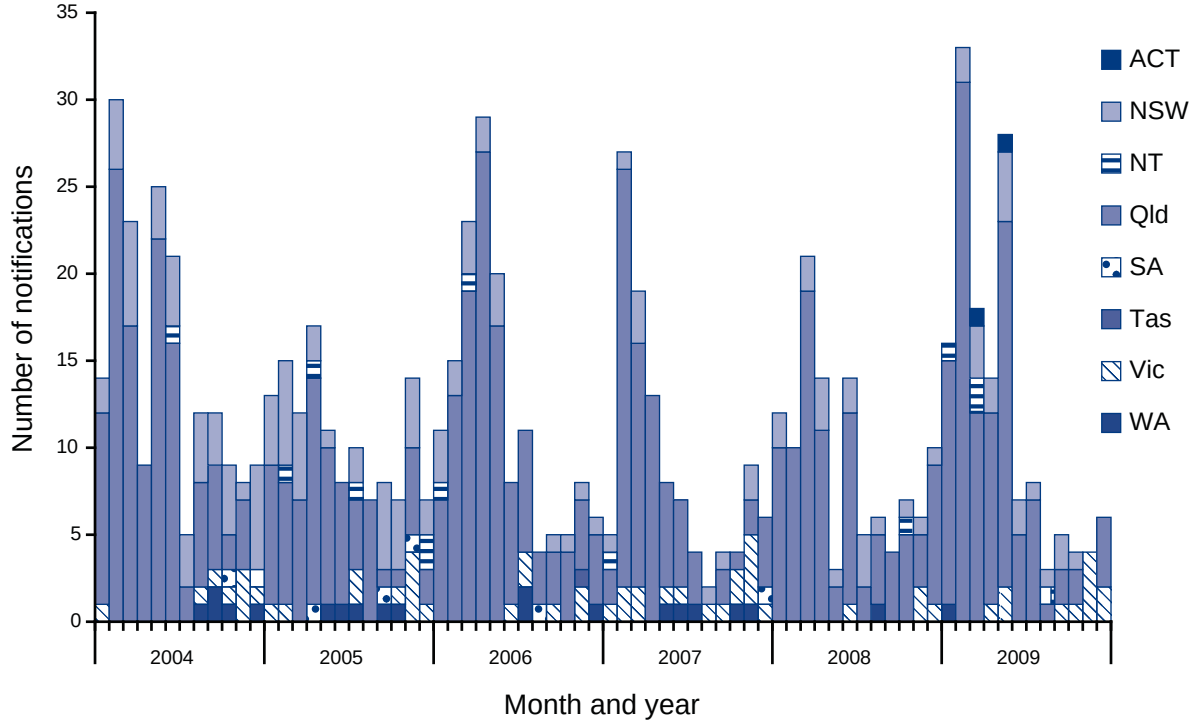
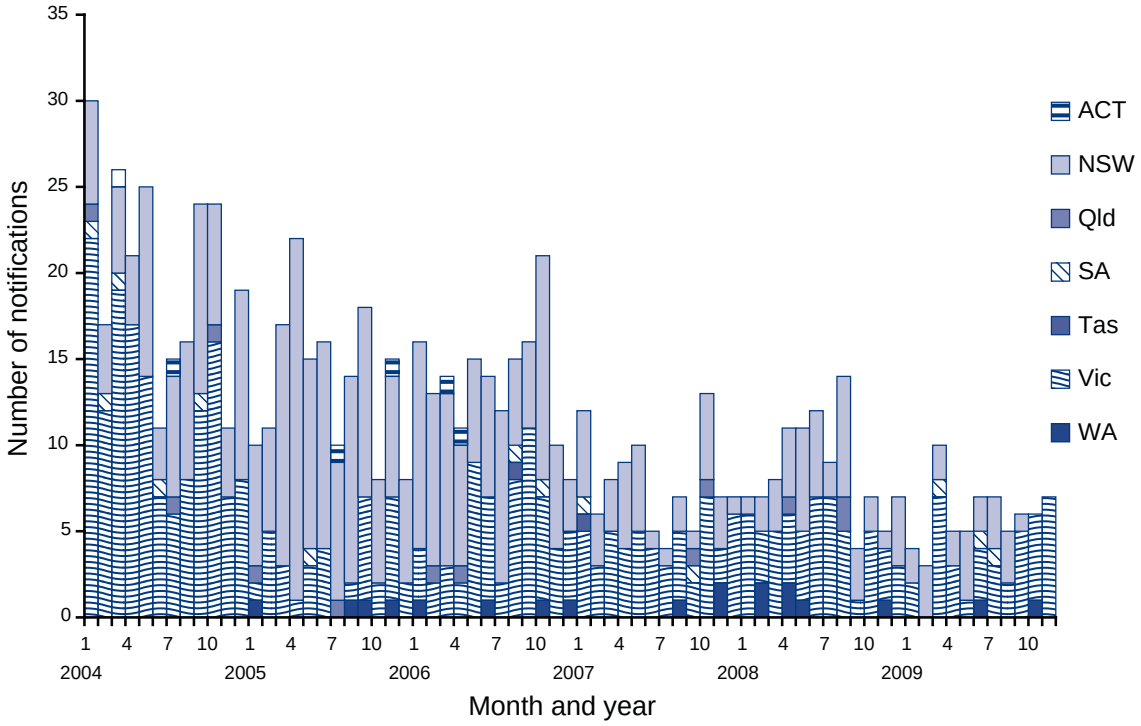


Figure 71: Notifications of ornithosis, Australia (except Northern Territory), 2004 to 2009, by state or territory and month and year of diagnosis



(47%). All notifications were aged 10 years or older and 75% of notifications were aged 40 years or over (Figure 72).

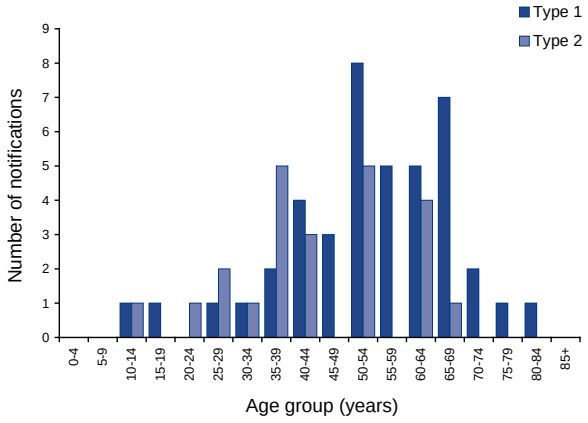
People at risk of contracting ornithosis include bird owners, pet shop employees, veterinarians, poultry-processing workers, zoo workers and taxidermists. Older adults and pregnant women may experience a more severe illness.[89]

Q fever

Q fever is caused by infection with the bacterium, *Coxiella burnetii*. Primary reservoirs of these bacteria are cattle, sheep and goats. These organisms are resistant to heat, drying and many common disinfectants, which enable the bacteria to survive for long periods in the environment. The mode of transmission to humans is most commonly by the airborne route through inhalation of contaminated dust. 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.[21]

In 2009, 309 notifications of Q fever were reported to the NNDSS, representing a national rate of 1.4 notifications per 100,000 population (Figure 73). Between 1991 and 2001, and prior to the introduction of the National Q Fever Management Program, Q fever notification rates ranged between 2.5–4.9 notifications per 100,000 population. The

Figure 72: Notifications of ornithosis, Australia 2009, by age group and sex

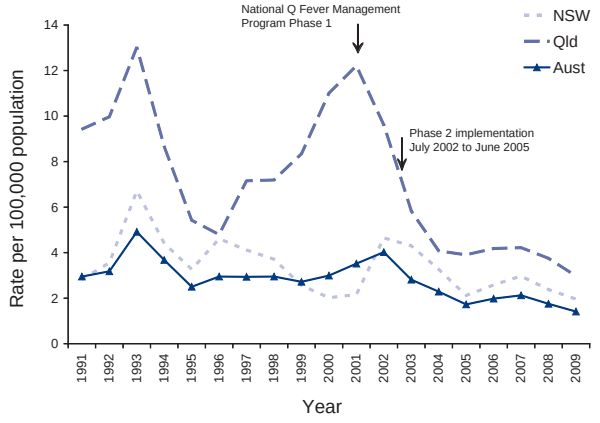


national notification rate for Q fever was lower in 2009 than in 2008 (1.4 and 1.8 notifications per 100,000 population, respectively). Between 2004 and 2009, the annual number of Q fever notifications ranged from 460 to 309 respectively.

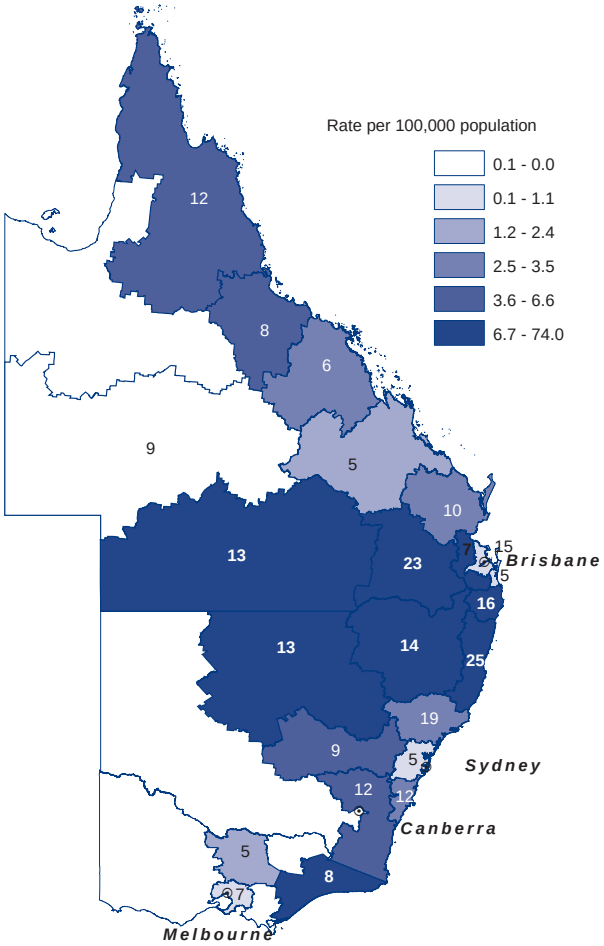
In 2009, the highest notification rates were from Queensland (131 notifications, 3.0 notifications per 100,000 population) and New South Wales (139 notifications, 2.0 notifications per 100,000 population). On a regional basis, the Central West Statistical Division of Queensland had the highest notification rate of 73 notifications per 100,000 population (Map 4). (Note: a small number of cases also occurred in South Australia, the Northern Territory, Victoria and Western Australia).

Seventy-five per cent of notifications reported to the NNDSS were male (232). As in 2008, the highest age specific rates of Q fever for males was in the 55–59 years age group (32 notifications, 5.0 notifications per 100,000 population), and for females was in the 60–64 years age groups (2.1 notifications per 100,000 population). There were 4 notifications reported in people aged less than 15 years.

Figure 73: Notification rate for Q fever, Australia, New South Wales and Queensland, 1991 to 2009



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



The Australian Government has facilitated the availability of the Q fever vaccine. Adults at risk of Q fever infection, including abattoir workers, farmers, veterinarians, stockyard workers, shearers and animal transporters should be considered for vaccination. The administration of the Q fever vaccine requires pre-vaccination screening test to exclude those recipients with a previous (unrecognised) exposure to the organism. A Q fever vaccine may cause an adverse reaction in a person who has already been exposed to the bacterium. Vaccine is not recommended for children under 15 years of age.[12]

Tularaemia

Tularaemia is caused by infection with the bacterium *Francisella tularensis*. The most common modes of transmission are through arthropod bites, handling infected animals, inhalation of infectious aerosols or exposure to contaminated food or water. Small mammals such as rodents, rabbits and hares are often the reservoir host.[26]

There were no notifications of tularaemia in 2009, and there has never been a case notified in Australia.

Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2009 and classified as 'other bacterial infections' in the NNDSS. A total of 1,919 notifications were included in this group in 2009, which accounted for less than 1% of all the notifications to NNDSS, an increase in cases and a similar proportion as in 2008 (1,771 notifications and 1% of total).

Legionellosis

Legionellosis, caused by the bacterium *Legionella,* can take the form of either Legionnaires' disease, a severe form of infection of the lungs or Pontiac fever, a milder influenza-like illness. The species that are most commonly associated with human disease in Australia are *L. pneumophila* and *L. longbeachae*. *Legionella* bacteria are found naturally in low levels in the environment. In the absence of effective environmental treatment *Legionella* organisms can breed to high numbers in air conditioning cooling towers, hot water systems, showerheads, spa pools, fountains or potting mix.

Infections caused by any *Legionella* species are notifiable, provided they meet the national surveillance case definition.[90] There were 302 notifications of

legionellosis reported in 2009, giving a national rate of 1.4 notifications per 100,000 population. This was an 11% increase from the 272 notifications reported in 2008 (1.3 notifications per 100,000 population). State and territory notification rates ranged from 0.9 notifications per 100,000 population in Victoria to 2.7 notifications per 100,000 population in South Australia, with no cases reported in Tasmania in 2009.

Data on the causative species were available for 94% (285) of cases: 57% (171) were *L. longbeachae,* 37% (112) were identified as *L. pneumophila* and 1 (1%) case each of *L. micdad*ei and *L. bozemanii* were reported (Table 18).

Historically, there have been differences in the geographic distribution of *L. longbeachae* and *L. pneumophila*, with *L. longbeachae* making up the majority of notifications from South Australia and Western Australia, while *L. pneumophila* has been the most common infecting species in the eastern States (Queensland, New South Wales and Victoria). However, in 2009 *L. longbeachae* was also notified more frequently than *L. pneumophila* in the eastern States of Queensland and New South Wales.

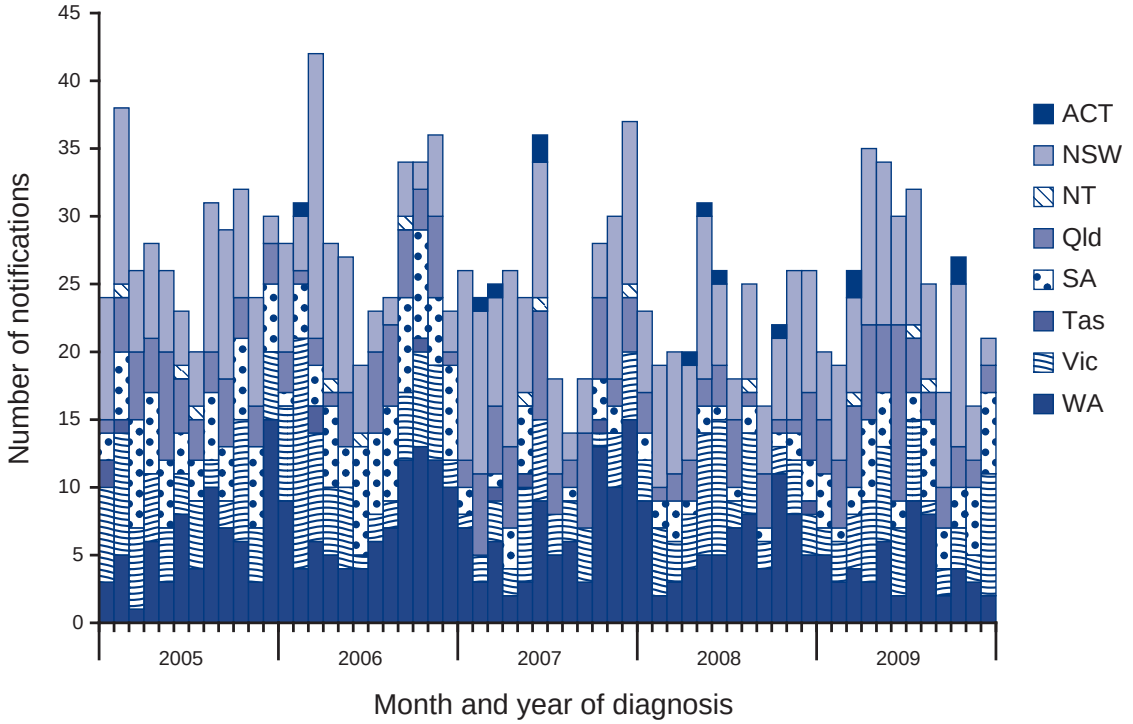
In 2009, diagnoses of legionellosis were highest in April (35 notifications, 12%) and May (34 notifications, 11%) (Figure 74). *L. pneumophila* occurred most frequently in the autumn months, with 45 cases reported over the period March to May 2009 (Figure 75). Twenty cases of *L. pneumophila* were reported in April 2009, the largest number of cases diagnosed in a month since 23 cases were reported in March 2006. *L. longbeachae* cases peaked in winter 2009, with 55 cases reported over the period June to August 2009, including 22 cases in July.

Males accounted for 61% (184) of legionellosis notifications in 2009, with a male to female ratio of 1.6:1. There were no notifications in people under the age of 16 years. The notification rate was highest in the 75–79 years age group (6 per 100,000 population, 33 notifications). The highest age and sex-specific rates were observed in men aged 75–79 years (9.5 per 100,000, 24 notifications) and women aged 70–74 years (4.5 per 100,000 population, 16 cases, Figure 76).

An infecting species analysis by age group showed that 84% (144/171) of *L. longbeachae* notifications were in persons aged 45 years or older, with the highest rate in the 65–69 years age group (3.2 per 100,000 population, 28 notifications). The proportion of *L. pneumophila* infections in persons 45 years or older was also 84% (94/112), with the highest rate in the 70–74 years age group (2.6 per 100,000 population, 18 notifications).

Mortality data were available for 44% (133/302) of notifications. There were 10 reported deaths due to legionellosis in Australia in 2009, which was an increase from 5 reported deaths in 2008. Those who died ranged in age between 62 and 82 years (median

Figure 74: Notifications of legionellosis, Australia, 2005 to 2009, by state or territory and month and year of diagnosis



72 years); 7 deaths were in males and 3 deaths were in females. There were 6 deaths associated with *L. pneumophila* infection and 4 deaths associated with *L. longbeachae* (Table 18). Mortality data should be interpreted with caution given the large proportion of cases without outcome details and the variability across jurisdictions in reporting death to the NNDSS.

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 amongst migrants from leprosy-endemic countries and occasional locally-acquired cases in Indigenous communities. Trends in leprosy notifications in Indigenous and non-Indigenous Australians are shown in Figure 77.

In 2009, 3 leprosy notifications (1 male and 2 females) were received, compared with 11 in 2008. There were 2 notifications in Queensland and one in Victoria. One notification was identified as an Indigenous Australian. The cases were aged 13, 21 and 28 years.

Figure 75: Notifications of legionellosis, Australia, 2005 to 2009, by infecting species, and month and year of diagnosis

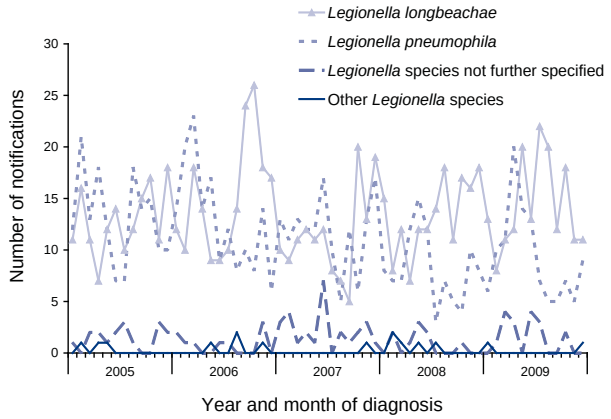


Figure 76: Notification rates for legionellosis, Australia, 2009, by sex and age group

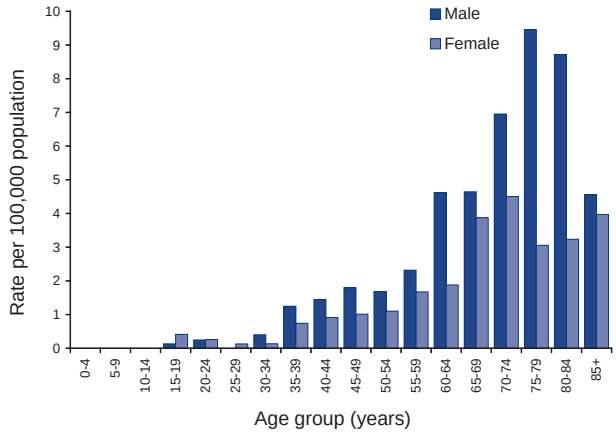


Figure 77: Notifications of leprosy, Australia, 1992 to 2009, by Indigenous status and year of diagnosis

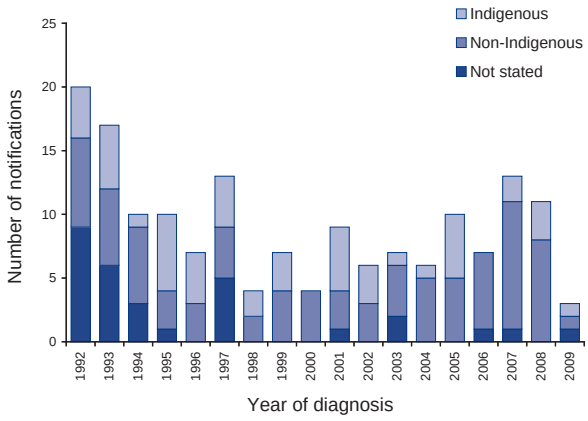


Table 18: Notifications of legionellosis, Australia, 2009, by species and state or territory

Species	State or territory								Aust	Total %
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
*Legionella pneumophila**	0	28	0	24	21	0	32	7	112	37.1
Legionella longbeachae†	0	64	3	28	23	0	10	43	171	56.6
Legionella micdadei	0	0	0	0	0	0	1	0	1	0.3
Legionella bozemanii	0	0	0	0	0	0	1	0	1	0.3
Unknown species	4	2	0	4	0	0	6	1	17	5.6
Total	4	94	3	56	44	0	50	51	302	100.0

* Four deaths.

† Six deaths.

Invasive meningococcal disease

Meningococcal disease is caused by the bacterium *Neisseria meningiditis* and becomes invasive when bacteria enter a normally sterile site, usually the blood (septicaemia), cerebrospinal fluid (meningitis) or both. The bacterium is carried by about 10% of the population without causing disease, and is transmitted via respiratory droplets. It occasionally causes a rapidly progressive serious illness, most commonly in previously healthy children and young adults. There are 13 known serogroups of the meningococcus. Globally, serogroups A, B, C, W135 and Y most commonly cause disease.[21] Historically, *N. meningitidis* serogroups B and C have been the major cause of invasive meningococcal disease (IMD) in Australia. There has been a marked decrease in rates of IMD due to *N. meningitidis* serogroup C infections following the introduction of the National Meningococcal C Vaccination Program in 2003.

In 2009, there were 259 notifications of IMD, a 9% decrease from 285 cases in 2008, and the lowest number of notifications since 1996. Since 2004, notification rates have decreased from 2.0 cases per 100,000 population to 1.2 per 100,000 in 2009.

Males accounted for 54% (139) of IMD notifications in 2009, with a male to female ratio of 1.2:1. Notifications peaked in July. Ninety-six per cent of notified cases (248) met the national case definition as 'confirmed' and the remaining 4% (11) were classified as 'probable', based on clinical symptoms alone.

Eighty-six per cent of IMD notifications (224) in 2009 had serogroup data available of which 88% (1977) were caused by serogroup B organisms, 6% (14) serogroup C (Figure 78), 2% (5) serogroup W135, 4% (8) serogroup Y, and the remaining 16% were either unknown or untypeable (Table 19). In comparison, in 2008 of 285 notifications, 77% (220) were caused by serogroup B organisms, 7% (21) were serogroup C, 3% (8) serogroup W135, 2% (8) were serogroup Y, and 10% (28) were either unknown or untypeable.

The highest age-specific IMD notification rate in 2009 was in children aged 0–4 years (6.4 per 100,000 population). Of the notifications reported in this age group, 81% were serogroup B. Although there is no vaccine available to protect against serogroup B disease, the rate for IMD due to serogroup B organisms has also declined in most age groups over the period 2004 to 2009 (Figure 79). The highest rate for sero-

Figure 78: Notifications of invasive meningococcal disease, Australia, 2004 to 2009, by serogroup and month and year of diagnosis

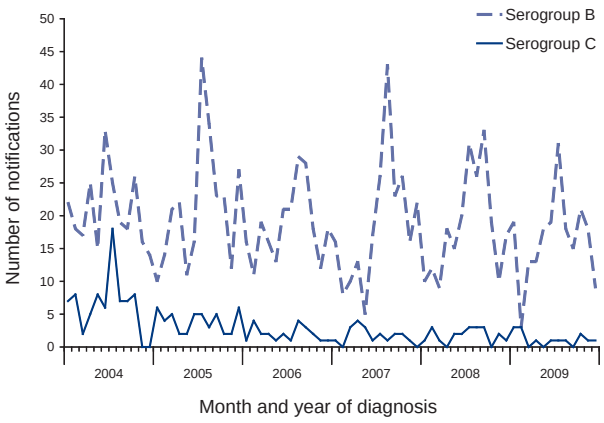


Figure 79: Notification rate for serogroup B invasive meningococcal disease, Australia, 2004 to 2009, by select age group and year of diagnosis

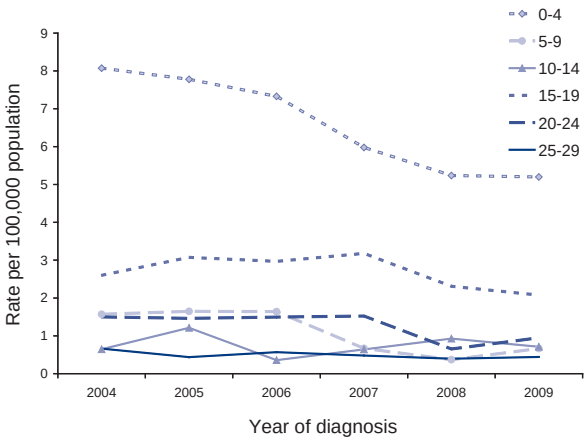


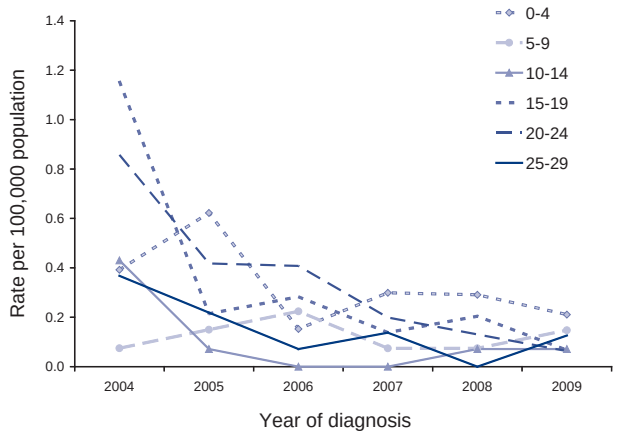
Table 19: Notifications of invasive meningococcal disease, Australia, 2009 by serogroup and state or territory

Serogroup	State or territory									Total (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
Serogroup B	2	58	5	50	0	3	35	24	197	76.1
Serogroup C	0	8	1	2	0	0	1	2	14	5.4
W135	0	5	0	0	0	0	0	0	5	1.9
Y	0	3	0	1	0	0	1	1	8	3.1
Unknown or untyped serogroup	0	22	0	7	22	0	5	1	35	13.5
Total	2	96	6	60	22	3	42	28	259	100.0

group B infection in 2009 was 5.2 per 100,000 population in the 0–4 years age group (74 notifications), representing a 36% decline from 2004 (103 notifications, 8.1 per 100,000). There was a corresponding 56% decline in the 5–9 years age group from 1.6 per 100,000 (21 notifications) in 2004 to 0.7 per 100,000 (9 notifications) in 2009.

Notification rates for IMD due to serogroup C infections remained low in most age groups in 2009 (Figure 80). Since 2004, the largest decline has been in the 15–19 years age group, with 0.1 notifications per 100,000 population (1 notification) in 2009 compared with 1.2 per 100,000 (16 notifications) in 2004; a decline of 92%. Similarly, the rate in the 20–24 years age group fell from 0.9 per 100,000 (12 notifications) to 0.1 per 100,000 (1 notification) over the same period; a 89% decline.

Figure 80: Notification rate for serogroup C invasive meningococcal disease, Australia, 2004 to 2009, by select age group



Mortality data for IMD were available for 98 of the 259 (38%) notifications reported to the NNDSS in 2009. Of these, there were 10 deaths due to IMD (6 serogroup B, 1 serogroup C and 1 serogroup W135). This was an increase from 8 deaths in 2008 (mortality data were provided to the NNDSS for 51% of notifications in 2008). Mortality data should be interpreted with caution given the low level of completeness and the variability across jurisdictions in reporting death as an outcome in NNDSS.

Laboratory based meningococcal disease surveillance

The Australian Meningococcal Surveillance Program (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *N. meningitidis* from cases of IMD in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using standardised methodology to determine the phenotype (serogroup, serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics. The results of laboratory surveillance in 2009 have yet to be published.

Tuberculosis

Tuberculosis (TB) is an infection caused by the bacterium *Mycobacterium tuberculosis*. TB is transmitted by airborne droplets produced by people with pulmonary or respiratory tract TB during coughing or sneezing. While Australia has one of the lowest rates of tuberculosis in the world, the disease remains a public health problem in the overseas-born and Indigenous communities. In 2009, 1,335 TB notifications were received by NNDSS; a rate of 6.2 cases per 100,000 population. In 2008, there were 1,203 notifications (5.6 per 100,000). TB notification rates were higher than the national average in the Australian Capital Territory (6.5 per 100,000), New South Wales (6.9 per 100,000), the Northern Territory (12.5 per 100, 000) and Victoria (7.7 per 100,000. The lowest rate occurred in Tasmania (1.8 per 100,000).

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

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Abbreviations

7vPCV	7 valent pneumococcal conjugate vaccine
23vPPV	23 valent pneumococcal polysaccharide vaccine
ABL	Australian bat lyssavirus
ABS	Australian Bureau of Statistics
ACCESS	Australian Collaboration for Chlamydia Enhanced Surveillance
AFP	acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AIDS	acquired immune deficiency syndrome
AMSP	Australian Meningococcal Surveillance Programme
ANCJDR	Australian National Creutzfeldt-Jakob Disease Registry
ATAGI	Australian Technical Advisory Group on Immunisation
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
DENV	dengue virus
EIA	enzyme inhibition assay
H1N1	influenza A(H1N1) pandemic 2009
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HUS	haemolytic uraemic syndrome
IMD	invasive meningococcal disease
IPD	invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
MMR	measles-mumps-rubella vaccine
MVEV	Murray Valley encephalitis virus
NAMAC	National Arbovirus and Malaria Advisory Committee
NEC	not elsewhere classified
NIP	National Immunisation Program
NN	not notifiable
NNDSS	National Notifiable Diseases System
NPRL	National Polio Reference Laboratory
NSC	National Surveillance Committee
PCR	polymerase chain reaction
RNA	ribonucleic acid virus
RRV	Ross River virus
SARS	severe acute respiratory syndrome
SD	Statistical Division
SoNG	Series of National Guidelines
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*
VZV	Varicella-zoster virus
WHO	World Health Organization
WHOCC	World Health Organization Collaborating Centre for Reference and Research on Influenza
WPR	Western Pacific Region
WPV	wild-type polio virus

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Appendices

Appendix 1: Mid-year estimate of Australian population, 2009, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aus
Males	174,487	3,517,707	116,684	2,203,712	801,487	247,942	2,689,782	1,135,244	10,888,385
Females	176,695	3,582,007	108,164	2,203,111	821,225	254,685	2,737,899	1,101,657	10,986,535
Total	351,182	7,099,714	224,848	4,406,823	1,622,712	502,627	5,427,681	2,236,901	21,874,920

Source: Australian Bureau of Statistics. Population by Age and Sex, Australian States and Territories, Estimated Resident Population By Single Year of Age, Australia. Canberra: Australian Bureau of Statistics; 2009. Report No.: 3201.0.[7]

Appendix 2: Mid-year estimate of Australian population, 2009, by state or territory and age

Age group	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aus*
0–4	23,058	451,548	18,472	304,237	96,417	33,205	346,739	149,790	1,423,608
5–9	20,629	440,537	17,625	285,751	94,254	30,812	325,640	140,621	1,356,035
10–14	21,294	451,099	16,760	296,596	100,898	33,562	336,462	148,121	1,404,996
15–19	24,155	480,422	16,730	309,425	107,799	34,706	364,910	155,481	1,493,798
20–24	30,306	502,865	18,187	316,099	112,913	31,145	405,673	164,471	1,581,787
25–29	30,751	513,319	20,190	317,629	107,629	28,727	401,364	164,619	1,584,403
30–34	26,782	489,605	18,542	296,220	100,201	28,327	377,842	154,174	1,491,830
35–39	27,281	519,020	18,626	327,459	111,616	33,250	406,483	167,562	1,611,487
40–44	24,659	483,559	16,457	307,840	112,989	33,500	384,684	162,456	1,526,360
45–49	25,069	508,821	16,105	315,670	118,111	37,606	384,775	163,245	1,569,605
50–54	22,999	465,704	14,277	285,765	111,319	35,875	352,953	149,350	1,438,428
55–59	20,863	421,682	12,209	261,574	103,406	33,885	317,282	133,967	1,305,071
60–64	17,485	381,364	8,716	236,730	94,230	31,126	284,833	115,151	1,169,759
65–69	11,654	288,240	5,484	172,901	69,792	23,491	213,912	83,461	869,039
70–74	8,502	231,684	2,998	129,878	57,169	18,189	172,827	64,847	686,136
75–79	6,347	187,971	1,710	99,257	47,705	14,490	141,217	49,721	548,443
80–84	4,984	148,998	1,060	76,174	39,969	10,997	111,342	37,213	430,743
85+	4,364	133,276	700	67,618	36,295	9,734	98,743	32,651	383,392
Total	351,182	7,099,714	224,848	4,406,823	1,622,712	502,627	5,427,681	2,236,901	21,874,920

Source: Australian Bureau of Statistics. Population by Age and Sex, Australian States and Territories, Estimated Resident Population By Single Year of Age, Australia. Canberra: Australian Bureau of Statistics; 2009. Report No.: 3201.0.[7]

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2009, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Cholera	0	0	0	4	0	0	4	100.0	4	0
Donovanosis	0	1	0	0	0	0	1	100.0	1	0
Haemophilus influenzae type b	7	0	0	12	0	0	19	100.0	19	0
Tetanus	0	0	0	3	0	0	3	100.0	3	0
Syphilis – congenital	2	0	0	1	0	0	3	100.0	3	0
Murray Valley encephalitis virus infection	1	0	0	3	0	0	4	100.0	4	0
Meningococcal infection	25	1	3	223	7	0	259	97.3	252	7
Syphilis <2 year duration	108	12	5	1,112	53	1	1,291	95.8	1,237	54
Typhoid	1	0	0	110	3	2	116	95.7	111	5
Tuberculosis	23	3	0	1,247	76	6	1,355	93.9	1,273	82
Hepatitis E	0	0	0	31	2	0	33	93.9	31	2
Hepatitis A	8	0	0	513	40	2	563	92.5	521	42
Haemolytic uraemic syndrome	0	0	0	10	1	0	11	90.9	10	1
Hepatitis C (newly acquired)	47	1	1	310	41	1	401	89.5	359	42
Listeriosis	2	0	0	79	8	2	91	89.0	81	10
Hepatitis B (newly acquired)	13	1	0	197	26	1	238	88.7	211	27
Varicella zoster (chickenpox)	104	3	5	1,305	152	30	1,599	88.6	1,417	182
Pneumococcal disease (invasive)	174	2	4	1,154	191	34	1,559	85.6	1,334	225
Legionellosis	3	0	0	247	41	11	302	82.8	250	52
Measles	1	0	0	85	19	0	105	81.9	86	19
Varicella zoster (shingles)	59	3	2	2,102	411	82	2,659	81.5	2,166	493
Ornithosis	1	0	0	51	12	1	65	80.0	52	13
Malaria	1	7	0	404	111	3	526	78.3	412	114
Rubella	0	0	0	20	4	3	27	74.1	20	7
Syphilis >2 years or unspecified duration	226	31	6	757	359	6	1,385	73.6	1,020	365
Leptospirosis	7	1	1	98	39	0	146	73.3	107	39
Shigellosis	193	2	0	226	113	88	622	67.7	421	201
Leprosy	0	1	0	1	1	0	3	66.7	2	1

Appendix 3, *continued*: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2009, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
STEC, VTEC	2	0	0	105	54	0	161	66.5	107	54
Brucellosis	1	0	0	20	11	0	32	65.6	21	11
Q fever	14	0	0	187	104	4	309	65.0	201	108
Gonococcal infection	2,798	165	36	2,236	2,097	727	8,059	65.0	5,235	2,824
Mumps	11	0	0	96	49	9	165	64.8	107	58
Hepatitis D	0	0	0	21	11	2	34	61.8	21	13
Dengue virus infection	39	45	6	721	564	27	1,402	57.8	811	591
Influenza (laboratory confirmed)	3,945	458	173	21,790	19,502	301	46,169	57.1	26,366	19,803
Kunjin virus infection	0	0	0	1	1	0	2	50.0	1	1
Salmonellosis	408	12	10	4,242	3,883	978	9,533	49.0	4,672	4,861
Chlamydial infection	4,376	678	226	25,407	25,532	6,441	62,660	49.0	30,687	31,973
Campylobacteriosis	222	9	10	7,567	7,636	529	15,973	48.9	7,808	8,165
Cryptosporidiosis	252	1	3	1,959	2,175	235	4,625	47.9	2,215	2,410
Pertussis	635	28	25	13,503	13,395	2,150	29,736	47.7	14,191	15,545
Arbovirus infection (NEC)	0	0	0	11	15	0	26	42.3	11	15
Hepatitis B (unspecified)	204	38	4	2,501	3,744	616	7,107	38.7	2,747	4,360
Ross River virus infection	134	12	3	1,644	2,722	271	4,786	37.5	1,793	2,993
Hepatitis C (unspecified)	465	5	14	3,344	6,283	970	11,081	34.5	3,828	7,253
Barmah Forest virus infection	39	4	2	388	995	58	1,486	29.1	433	1,053
Varicella zoster (unspecified)	129	27	7	1,553	5,059	202	6,977	24.6	1,716	5,261

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

TSI Torres Strait Islander

Immunisation coverage annual report, 2009

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Abstract

This, the third annual immunisation coverage report, documents trends during 2009 for a range of standard measures derived from Australian Childhood Immunisation Register data, including overall coverage at standard age milestones and for individual vaccines included on the National Immunisation Program (NIP). Coverage by Indigenous status and mapping by smaller geographic areas as well as trends in timeliness is also summarised according to standard templates. With respect to overall coverage, the Immunise Australia Program targets have been reached for children at 12 and 24 months of age but not for children at 5 years of age. Coverage at 24 months of age exceeds that at 12 months of age, but as receipt of varicella vaccine at 18 months is excluded from calculations of 'fully immunised' this probably represents delayed immunisation, with some contribution from immunisation incentives. Similarly, the decrease in coverage estimates for immunisations due at 4 years of age from March 2008 is primarily due to changing the assessment age from 6 years to 5 years of age from December 2007. With respect to individual vaccines, a number of those available on the NIP are not currently assessed for 'fully immunised' status or for eligibility for incentive payments. These include pneumococcal conjugate and meningococcal C conjugate vaccines, for which coverage is comparable with vaccines that are assessed for 'fully immunised' status, and rotavirus and varicella vaccines for which coverage is lower. Coverage is also suboptimal for vaccines recommended for Indigenous children only (i.e. hepatitis A and pneumococcal polysaccharide vaccine) as previously reported for other vaccines for both children and adults. Delayed receipt of vaccines is an important issue for vaccines recommended for Indigenous children and has not improved among non-Indigenous children despite improvements in coverage at the 24-month milestone. Although Indigenous children in Australia have coverage levels that are similar to non-Indigenous children at 24 months of age, the disparity in delayed vaccination between Indigenous and non-Indigenous children remains a challenge. *Commun Dis Intell* 2011;35(2):132–148.

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

Introduction

This is the third annual Australian Childhood Immunisation Register (ACIR) coverage report, following the first on 2007 data[1] and the second on 2008 data.[2] This series of annual reports was established to consolidate the various forms of regular coverage reports and ad hoc publications produced by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases using ACIR data, highlighting important trends and significant issues over the preceding 12 months.[3–15] It follows the format of the first and second reports, providing a detailed summary for 2009 that includes vaccination coverage at standard milestone ages, coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children, and data for small geographic areas on vaccination coverage and prevalence of conscientious objectors. Readers are referred to the first report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.[1]

This report uses the long-standing international practice of reporting coverage at key milestone ages, to measure coverage against national targets and to track trends over time. No new vaccines were introduced to the NIP during 2009, with the exception of the Northern Territory, where the 10-valent pneumococcal conjugate vaccine at 2, 4, 6 and 12 months of age replaced the 7-valent conjugate and 23-valent polysaccharide vaccines. However, this report does include the 2nd full year of coverage data for rotavirus vaccine, having been introduced in 2007.

Incentives for vaccination and reporting to the Australian Childhood Immunisation Register

The Australian Government, through the Department of Health and Ageing, advises the ACIR whether calculation of coverage of the new vaccines and antigens should be included in the completed schedule assessment for eligibility for payments to parents or immunisation providers. Up to 2008, the ACIR made information payments (up to $6) to all immunisation providers and for general practitioners (GPs), under the General Practice Immunisation Incentive (GPII) scheme. In the 2008–09 Budget, the Australian Government announced that one of the components of the GPII Scheme, the GPII Service Incentive Payment (SIP), would stop from 1 October 2008. Service Incentive Payments ($18.50) were made for reporting a vaccination that completed a schedule point on the NIP,

at 6, 12, 18 months and 5 years.[16] However, the GPII Outcomes Payments, which paid practices that achieve 90 per cent or greater levels for full immunisation, was maintained. The vaccines and antigens required for full immunisation in assessment for the Outcomes Payment in 2009 were the same as in recent years, i.e. diphtheria, *Haemophilus influenzae* type b (Hib), hepatitis B, measles, mumps, pertussis, polio, rubella and tetanus. Vaccines included in the NIP in 2009 but not part of the completed schedule assessment for provider payments were: meningococcal C vaccine (Men C); 7-valent pneumococcal conjugate vaccine (7vPCV); and rotavirus vaccine. Varicella vaccine was also not included for coverage assessment but eligible providers received an information and SIP payment (up to October 2008) for reporting, as varicella vaccine is currently the only vaccine required for completion of the 18-month schedule point. While the ACIR records vaccines given only to Indigenous children in Queensland, Northern Territory, Western Australia and South Australia (hepatitis A and pneumococcal polysaccharide vaccines (23vPPV)) and vaccines not included in the National Immunisation Program , such as bacille Calmette-Guérin, reporting of these vaccines does not attract a GPII payment.

Table 1 shows the Australian National Immunisation Program Schedule in 2009.

In 2004–05, the means test to qualify for the Maternity Immunisation Allowance (MIA) was removed. This payment of $233 per child in 2008, is likely to be substantial enough to provide motivation both to complete immunisation and for parents to prompt their provider to notify any outstanding reports to the ACIR before the child reaches 24 months of age. In the 2008–09 Budget, in addition to the changes mentioned above, it was announced that the MIA payment would be paid in 2 equal amounts of $167, with eligibility for the 2nd payment assessed at 4 and 3 months to 5 years of age. This came into effect during the period of this report in January 2009. To meet the requirements of these changes the ACIR changed the National Due and Overdue Rules for Childhood Immunisation. From 1 January 2009 the overdue rule changed for all children born from 1 January 2005 onwards. The ACIR National Due and Overdue Rules now state that a child is due for their 4-year-old vaccinations at 4 years and overdue at 4 years 1 month of age, instead of overdue at 5 years of age.

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January 1996, by incorporating demographic data from Medicare on all enrolled children under the age of 7 years.[4] 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.[4] Children not enrolled in Medicare can also be added to the ACIR via a supplementary number. Since 2001, immunisations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible immunisation either through the Internet using the Medicare Australia web site or by submitting paper encounter forms, which are scanned at a central location. The existence of medical contraindications and conscientious objection to immunisation 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.

Immunisations recorded on the Register must be rendered in accordance with the guidelines issued by

Table 1: Australian National Immunisation Program Schedule for children in 2009

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

* 3rd dose of *Haemophilus influenzae* type b (Hib) vaccine at 6 months is dependent on vaccine brand used in state or territory.

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

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

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

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

Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state or territory)[18] since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 months), and 5 years of age (for vaccines due at 4 years). A minimum 3-month lag period is allowed for late notification of immunisations to the Register, but only immunisations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.[18] If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.[6,7]

Three-month birth cohorts are used for time trend analyses, while 12-month cohorts are used for other analyses in this report such as for small area coverage analysis and mapping of coverage estimates. A minimum 3-month lag is allowed for late notifications. These cohorts are children born between 1 January and 31 December 2008 for the 12-month milestone age; children born between 1 January and 31 December 2007 for the 24-month milestone age; and children born between 1 January and 31 December 2004 for the 5-year (60-month) milestone age.

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

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

Timeliness

Age-appropriate immunisation was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTP (due at 60 days of age) when he or she was more than 90 days of age was classified as not age-appropriately immunised (i.e. late for the dose). For descriptive purposes, the outcome measure for each dose is categorised as either vaccine dose 'no delay' (age-appropriately immunised), 'delay of between 1 to 6 months', or 'delay greater than 6 months'. Doses received 'too early' (greater than 30 days prior to when it was due), and doses never administered or recorded, were excluded. Timeliness is measured in 12 month cohorts. Children included in the timeliness analysis were assessed at 1–2 years after doses were due to allow time for late vaccinations to be recorded. Therefore, cohorts assessed for timeliness are not the same as those assessed for coverage milestones. 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 and Remoteness Index of Australia (ARIA), which was developed by the Department of Health and Ageing, and proposed as the national standard measure of remoteness for inclusion in the Australian Bureau of Statistics (ABS) 2001 census.[19] The two ARIA categories with most restricted access to services were defined as 'remote' (approximately 2.6% of the Australian population) and all other areas defined 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 two categories of children were considered: 'Indigenous' and 'non-Indigenous', children with unknown Indigenous status were presumed to be 'non-Indigenous'. Coverage estimate time trends are presented from 2004 only, due to poor rates of reporting Indigenous status prior to then.[20]

Small area coverage

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

Conscientious objection and 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. The percentage of children with no vaccines recorded on the ACIR was used as a proxy measure of the number of these children.[15] 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 2003 and 31 December 2008. At the time of data extraction on 31 March 2010, they were between 12 and 72 months of age. This cohort was chosen when calculating proportions so that children under the age of 12 months were not included, to allow sufficient time for registration of objection and to exclude infants late for vaccination.

Results

Coverage estimates

Overall

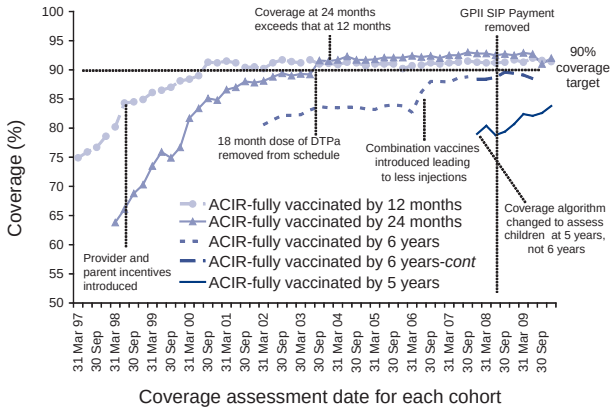
In 2009, coverage estimates for full-year birth cohorts at the 3 milestone ages of 12 months, 24 months and 5 years are provided in Tables 2, 3 and 4. Nationally, 'fully immunised' coverage and coverage for all individual vaccines for the 12-month and 24-month age groups exceeded the Immunise Australia Program's target of 90%. Recorded national coverage for the 5-year age group was well below the target, at 83% for all vaccines and lower in particular jurisdictions.

Figure 1 shows time trends in 'fully immunised' childhood vaccination coverage in Australia, assessed at 12 months, 24 months, and at 60 months of age, for 3-month cohorts born from 1 January 1996 to 31 December 2008. The proportion 'fully immunised' at 1 year of age increased steadily from 75% for the 1st cohort in 1997 to 91.4% by 31 December 2009. At the 24 month milestone, 'fully immunised' coverage estimates also increased steadily from 64% for the 1st cohort to 92% by December 2009. 'Fully immunised' coverage estimates at 6 years of age, for vaccines due at 4 years, were first reported in *CDI* in 2002, and increased steadily from 80.6% in early 2002 to 87.3% in late 2007, including a noticeable increase in June 2006, corresponding with the introduction of combination vaccines. However, from the beginning of 2008, when the assessment age was changed from 6 years to 5 years, 'fully immunised' coverage was substantially lower at 80.7% in December 2008, related to delayed immunisation. However, during 2009, coverage for this age group gradually rose to 83.8%. Figure 1 shows that coverage calculated at 6 years was unchanged at 89% during 2009.

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

There is a clear trend of increasing vaccination coverage over time for all age groups assessed, with the 2 youngest age cohorts having the highest cover-

Figure 1: Trends in 'fully immunised' vaccination coverage, Australia, 1997 to 2009, by age cohort



age. 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 until 2009 where they are now both similar.

Individual vaccines

The trends in childhood vaccination coverage in Australia for individual vaccines at 12 months of age (DTPa, polio, Hib, hepatitis B, rotavirus and

Table 2: Percentage of children immunised at 12 months of age in 2009, by vaccine and state or territory*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,820	97,550	3,807	62,150	19,601	6,533	70,800	30,714	295,975
Diphtheria, tetanus, pertussis (%)	94.4	92.7	90.1	92.2	91.8	92.6	92.9	90.2	92.3
Poliomyelitis (%)	94.3	92.6	90.0	92.2	91.8	92.6	92.9	90.2	92.3
Haemophilus influenzae type b (%)	95.7	95.1	94.6	94.6	94.6	95.0	95.2	93.6	94.8
Hepatitis B (%)	95.6	95.0	95.3	94.4	94.4	94.9	95.0	93.4	94.7
Rotavirus (%)	89.2	86.5	82.4	81.7	83.4	85.4	82.4	82.2	83.9
7vPCV (%)	93.3	91.9	89.7	91.4	91.1	92.2	91.9	89.2	91.5
Fully immunised (%)	93.8	92.2	89.4	91.8	91.4	92.4	92.3	89.5	91.8
Fully immunised (incl rotavirus and 7vPCV) (%)	88.1	84.7	79.9	85.9	86.9	83.9	85.6	80.2	84.8

* For the birth cohort born in 2008.

Table 3: Percentage of children immunised at 24 months of age in 2009, by vaccine and state or territory*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,720	98,175	3,783	62,169	19,704	6,465	71,498	30,641	297,155
Diphtheria, tetanus, pertussis (%)	95.6	94.6	94.8	94.1	94.7	95.0	95.4	93.6	94.6
Poliomyelitis (%)	95.5	94.6	94.8	94.0	94.7	95.0	95.4	93.6	94.6
Haemophilus influenzae type b (%)	95.6	94.9	92.9	93.4	93.9	95.1	94.6	93.0	94.3
Hepatitis B (%)	96.1	95.5	96.1	94.9	95.3	95.9	96.1	94.6	95.4
Measles, mumps, rubella (%)	94.6	93.5	94.9	93.3	94.0	94.4	94.6	92.9	93.8
Varicella (%)	86.6	80.5	82.7	84.5	80.5	82.6	82.4	79.0	81.8
MenC (%)	94.3	93.2	94.1	92.8	93.9	94.3	94.3	92.2	93.4
Fully immunised (%)	93.3	92.1	92.0	91.6	92.3	93.2	93.0	90.3	92.2
Fully immunised (incl varicella and MenC) (%)	85.0	78.5	80.2	82.3	79.0	81.2	80.6	76.6	79.8

* For the birth cohort born in 2007.

Table 4: Percentage of children immunised at 5 years of age in 2009, by vaccine and state or territory*

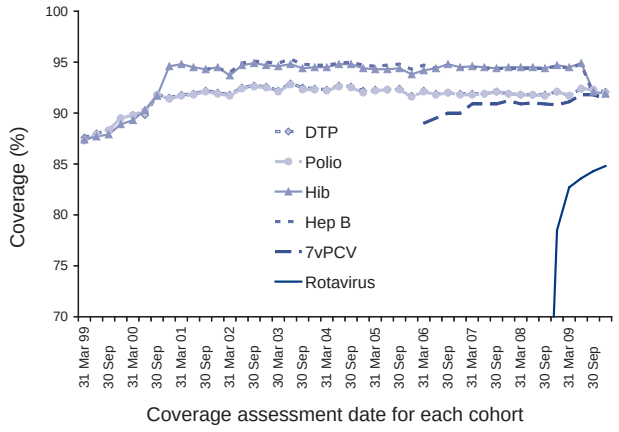
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,379	88,407	3,350	56,160	17,993	5,821	65,164	27,860	269,134
Diphtheria, tetanus, pertussis (%)	86.5	82.6	82.3	83.3	79.4	84.4	86.4	81.5	83.4
Poliomyelitis (%)	86.5	82.6	82.6	83.2	79.5	84.6	86.4	81.5	83.4
Measles, mumps, rubella (%)	86.2	82.4	82.4	83.2	79.3	84.4	86.1	81.2	83.2
Fully immunised (%)	85.8	81.9	81.4	82.5	78.8	83.7	85.7	80.5	82.7

* For the birth cohort born in 2004.

7vPCV) are shown in Figure 2, for 3-month cohorts born from 1 January 1998 to 31 December 2008.

Coverage estimates for all vaccines except Hib and hepatitis B remained relatively stable throughout the latter part of 2001 to 2009. Prior to the change in algorithm to measure coverage that occurred in the latter half of 2009, coverage for the Hib and hepatitis B vaccines at 12 months of age was greater than DTPa and polio. The change led to measures of Hib and hepatitis B vaccine coverage becoming similar to those for DTPa and polio in the last two cohorts of 2009 (Figure 2). Coverage for 7vPCV rose steadily from below 90% in early 2006 to be just below that for all other vaccines due at this age at around 92%, except for rotavirus vaccine. Rotavirus vaccine coverage rose steeply from late 2008 from below 70% to almost 85% in late 2009.

Figure 2: Trends in vaccination coverage estimates for individual vaccines at 12 months of age (DTP, polio, hepatitis B, Hib, 7vPCV and rotavirus)*



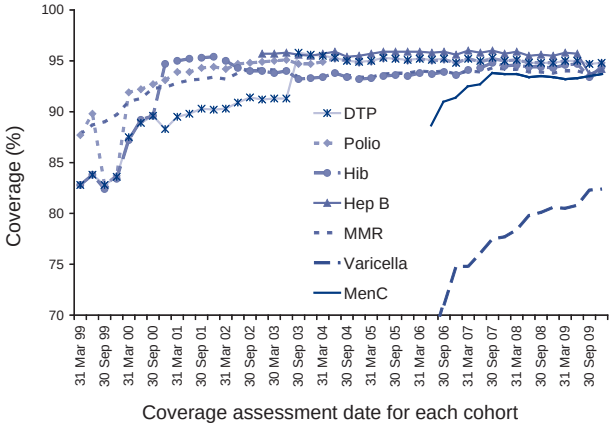
Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2008. Coverage assessment date was 12 months after the last birth date of each cohort.

* 3rd dose of diphtheria-tetanus-pertussis (DTP) and polio, 2nd or 3rd dose of *Haemophilus influenzae* type b (Hib) and hepatitis B.

The trends in childhood vaccination coverage in Australia for individual vaccines at 24 months of age (DTPa, polio, Hib, hepatitis B, MMR, Men C and varicella) are shown in Figure 3, for the 3-month cohorts born from 1 January 1997 to 31 December 2007. For most of the study period, hepatitis B coverage was higher than for all other vaccines, at just under 95%, due to the different coverage algorithm described above. Coverage was lowest for MMR and Hib, the only vaccines that have a 12-month dose used in calculations. The overall coverage estimates for 24-month-olds are more than 90% for all vaccines except varicella.

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



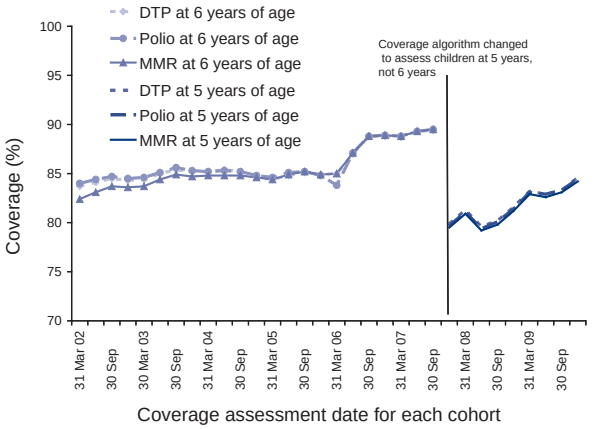
Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2007. Coverage assessment date was 24 months after the last birth date of each cohort.

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

The trends in childhood vaccination coverage in Australia for individual vaccines (DTPa, polio and MMR) at 6 years of age (5 years of age from December 2007) are shown in Figure 4 for the 3-month cohorts born from 1 January 1998 to 31 December 2004. There has been little difference for the different vaccines in recent years.

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



Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2004. Coverage assessment date was 72 months after the last birth date of each cohort up to December 2007 and then 60 months after the last birth date of each cohort.

* 4th dose of diphtheria-tetanus-pertussis (DTP) and polio, 2nd dose of measles-mumps-rubella (MMR).

Coverage estimates and vaccination timeliness for Indigenous children

Vaccination coverage estimates in 2009 for the three 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 the 12-month, 24-month and 5-year age milestones for most vaccines, 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. The coverage differential between Indigenous and non-Indigenous children for individual vaccines varies, with coverage at 24 months of age for most vaccines being almost identical for both groups and greater among Indigenous children for hepatitis B, MMR and Men C vaccines.

The trends in 'fully immunised' childhood vaccination coverage in Australia at 12 months, 24 months, and 6 years of age (5 years of age from December 2007) for Indigenous children since 2004 are shown in Figure 5, for the 3-month cohorts assessed from 1 March 2004 to 31 December 2009. Coverage for all vaccines due by 24 months of age has consistently remained higher than at 12 months and 6 years of age. Since the beginning of 2006,

Figure 5: Trends in 'fully immunised' vaccination coverage for Indigenous children in Australia, 2004 to 2009, by age cohorts

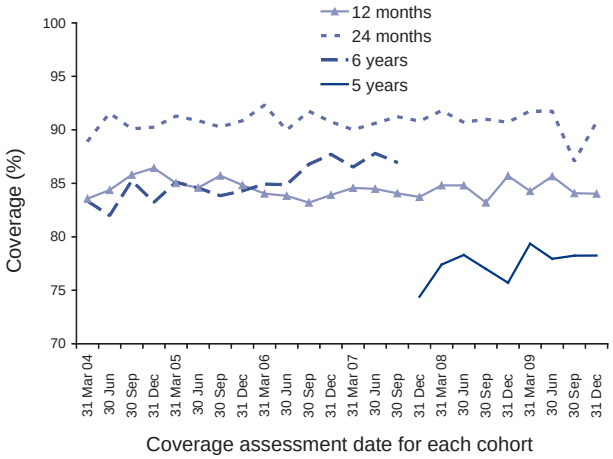


Table 5: Vaccination coverage estimates by age, vaccine and Indigenous status, 2009

Vaccine	Milestone age	Indigenous	Non-Indigenous
DTP	12 months*	85.3	92.7
	24 months†	94.2	94.7
	5 years‡	79.2	83.6
Polio	12 months*	85.2	92.6
	24 months†	94.2	94.6
	5 years‡	79.4	83.6
Hib	12 months*	93.2	94.9
	24 months†	93.1	94.3
	5 years‡	N/A§	N/A§
Hep B	12 months*	93.5	94.7
	24 months†	97.0	95.4
	5 years‡	N/A§	N/A§
MMR	12 months*	N/A§	N/A§
	24 months†	94.1	93.7
	5 years‡	79.6	83.4
Varicella	12 months*	N/A§	N/A§
	24 months†	80.7	81.9
	5 years‡	N/A§	N/A§
Meningococcal C	12 months*	N/A§	N/A§
	24 months†	93.7	93.4
	5 years‡	N/A§	N/A§
7vPCV	12 months*	85.0	91.8
	24 months†	N/A§	N/A§
	5 years‡	N/A§	N/A§
Rotavirus	12 months*	71.5	84.5
	24 months†	N/A§	N/A§
	5 years‡	N/A§	N/A§

* Birth cohort born 1 January to 31 December 2008.
† Birth cohort born 1 January to 31 December 2007.
‡ Birth cohort born 1 January to 31 December 2004.
§ Not included in coverage estimates for that group.

coverage for Indigenous children at 6 years of age was greater than coverage at 12 months of age, until falling below 80% in December 2007 when assessed at 5 years, due to delayed immunisation.

Table 6 shows 'fully immunised' vaccination coverage estimates in 2009 for Indigenous children at the three milestone ages, by state or territory. At age 12 months, the overall proportion of Indigenous children fully vaccinated was 85%, compared with 92.2% for non-Indigenous children. Although coverage was lower among Indigenous children in all jurisdictions, the extent of the difference varied, reaching more than 13 percentage points in Western Australia. However, by age 24 months, coverage disparities between Indigenous and non-Indigenous children had greatly reduced nationally, at 90.6% fully vaccinated for Indigenous and 92.2% for non-Indigenous children.

At 5 years of age, the proportion recorded as being 'fully vaccinated' was lower than that at earlier age milestones. At the national level, the coverage for Indigenous and non-Indigenous children was 78.6% and 82.9%, respectively, a greater disparity than in 2008 (2.7%). There was dramatic variation between individual jurisdictions, ranging from 11.9% lower in Indigenous children in South Australia to 8.2% higher in the Northern Territory, compared with non-Indigenous 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. Vaccination was delayed by more than one month for 30%–34% of Indigenous children and 18%–26% of non-Indigenous children. 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 no great differences between accessible and remote areas or vaccines. Delays of 1–6 months were also more frequent for Indigenous children, although less marked, especially for the 1st dose of MMR. The proportion with short delays was greater among Indigenous children residing in remote areas than in accessible areas for the 3rd dose of DTP vaccine (35% versus 31%), but not for the 1st dose of MMR.

Coverage for National Immunisation Program vaccines not routinely reported elsewhere

7vPCV and rotavirus

7vPCV was first added to the NIP in January 2005. Since coverage was first calculated for this vaccine in early 2006, it has remained high, with a slight increase from 89% to 91.5% (Figure 2). Coverage is similar in all jurisdictions at greater than or approaching 90% (Table 2).

Table 6: Percentage of children fully immunised at 12 months, 24 months and 5 years of age, 2009, by Indigenous status and state or territory

	State or territory								
	ACT	NSW	Vic	Qld	SA	WA	Tas	NT	Australia
12 months – fully immunised (%)*									
Indigenous	92.1	87.4	86.9	85.7	80.2	77.2	89.6	85.8	85.0
Non-Indigenous	93.8	92.4	92.4	92.3	91.8	90.3	92.7	91.9	92.2
12 months – fully immunised (including rotavirus and 7vPCV) (%)									
Indigenous	85.2	76.4	78.0	75.5	74.3	63.2	78.5	69.7	73.7
Non-Indigenous	88.2	85.0	85.7	86.7	87.4	81.3	84.4	86.9	85.4
24 months – fully immunised (%)†									
Indigenous	90.6	91.4	92.7	91.4	90.3	84.4	94.2	92.4	90.6
Non-Indigenous	93.3	92.2	93.0	91.6	92.4	90.7	93.1	91.7	92.2
24 months – fully immunised (including varicella and MenC) (%)									
Indigenous	77.7	76.5	76.4	79.4	74.8	70.3	81.0	82.8	77.3
Non-Indigenous	85.1	78.6	80.7	82.5	79.1	77.0	81.2	78.3	79.3
5 years – fully immunised (%)‡									
Indigenous	85.2	78.0	80.5	79.6	67.3	74.1	80.2	86.1	78.6
Non-Indigenous	85.8	82.0	85.8	82.7	79.2	80.9	83.9	77.9	82.9

* 'Fully immunised' – 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines.

† 'Fully immunised' – 3 or 4 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 3 or four doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine.

‡ 'Fully immunised' – 4 or 5 doses of a DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Table 7: Vaccination delay for the cohort of children born in 2007, Australia, by Indigenous and remoteness status

Vaccine dose	Indigenous status	Remoteness	1–6 months delay %	>6 months delay %
DTP3	Indigenous	Accessible	30	9
		Remote	33	7
	Non-Indigenous	Accessible	18	2
		Remote	18	2
MMR1	Indigenous	Accessible	33	6
		Remote	34	4
	Non-Indigenous	Accessible	26	2
		Remote	26	2

Rotavirus vaccine was added to the NIP in July 2007, so coverage for 2 or 3 doses (depending on vaccine) at 12 months of age could be calculated only from the December 2008 quarter. Rotavirus coverage was lower nationally, and had greater variation between jurisdictions compared to other vaccines given at 2, 4 and 6 months, which may be due to the strict upper age limits for this vaccine. Reported coverage for 2 or 3 doses (Rotarix® versus Rotateq®) of rotavirus vaccine at 12 months of age varied from 81.7% in Queensland (Rotateq®) to 86.5% and 89.2% in New South Wales and the Australian Capital Territory (both Rotarix®) respectively (Table 2).

Meningococcal C and varicella

Meningococcal C vaccine was added to the NIP in January 2003. Since coverage was first calculated for this vaccine in early 2006, it has remained at high levels, with an increase over 2 years from 88% to around 93% (Figure 3). There was little variation by jurisdiction with all jurisdictions experiencing coverage levels greater than 92% (Table 3).

Varicella vaccine was added to the NIP in November 2005. Figure 3 shows reported coverage for this vaccine has consistently been 10–15 percentage points lower than that for meningococcal C vaccine, being just above 80% for the latest assessment. This is probably partly due to the shorter time varicella has been on the NIP and the recommendation to give the vaccine at 18 months of age, which was historically associated with lower coverage when there was a 18-month pertussis booster prior to 2003, as well as a gap when no vaccine was administered at 18 months over 2 years. Reported varicella vaccine coverage also shows considerable variation by jurisdiction from 79% in Western Australia to 86.6% in the Australian Capital Territory (Table 3). Data are also available from the ACIR on the number of reports from GPs stating that children, born since May 2004, have natural immunity to varicella and do not require varicella vaccination. Reports of natural immunity to varicella total greater than 15,000 since May 2004 (not shown), corresponding to approximately 1.1% of the cohort. It is likely that there is under-reporting of presumed natural immunity by GPs but this is unlikely to fully account for lower varicella coverage.

Hepatitis A and 23vPPV

Hepatitis A vaccine was available in Australia prior to the development of the ACIR in 1996 and has been included on the NIP for Indigenous children in the Northern Territory, South Australia, Western Australia, and in Queensland since November 2005, but was used earlier than this in North Queensland. 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 59% in December 2009 (Figure 6). However, the figure for December 2009 is calculated differently from all previous figures. The calculation now gives an equal amount of assessment time for all four relevant jurisdictions, so 2 doses are assessed at 30 months for Western Australia and the Northern Territory and 2 doses are assessed at 36 months for Queensland and South Australia. An additional 9% of children had received 1 dose of hepatitis A vaccine by 18 or 24 months of age, increasing national coverage for at least 1 dose of hepatitis A vaccine to 68% in Indigenous children (Table 8). The 23vPPV has been recommended for Indigenous children in the same 4 jurisdictions as a booster at 18–24 months of age since 2001; coverage has gradually increased from 47% in December 2006 to 55% in December 2009 (Figure 6). There is a large variation in 23vPPV coverage by jurisdiction from a low of 38% in South Australia to a high of 78.9% in the Northern Territory (Table 8). Similarly, there is a variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 36% in South Australia to a high of 86.2% in the Northern Territory (Table 8).

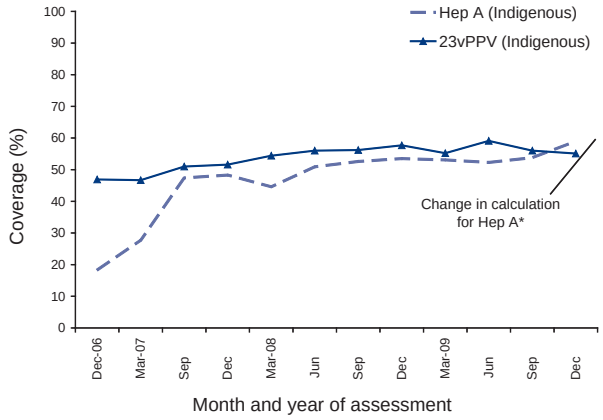
Timeliness of immunisation

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

Since 2001, the proportion with timely receipt of the 3rd dose of DTP vaccine has remained at 88% (Figure 7). Across the 7-year period, 2001–2007, timely receipt of 1 dose of MMR vaccine initially decreased by 3 percentage points but then rose 2 percentage points, although estimated coverage by 24 months of age remained stable at almost 94% (Figure 8).

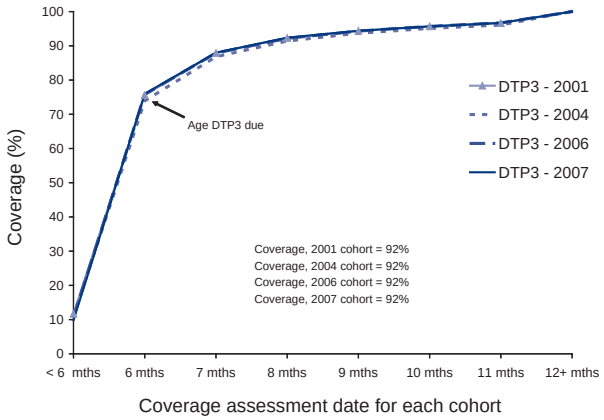
A comparison of vaccination delay for the 3rd dose of DTPa, due at 6 months of age; the 1st doses of MMR and meningococcal C, due at 12 months of age; and the 2nd dose of MMR, due at 4 years of age, is shown in Figure 9. As demonstrated in previous studies, the proportion with vaccination delay increased with vaccine doses given at an older age. The greatest proportion with any delay was seen with the 2nd dose of MMR vaccine with 80% of doses given late and almost 35% given more than 6 months late.

Figure 6: Trends in coverage for hepatitis A* and pneumococcal polysaccharide (23vPPV) vaccines for Indigenous children



* Two doses assessed at 30 months for Western Australia and the Northern Territory. Two doses assessed at 36 months for Queensland and South Australia. (Prior to December 2009, 2 doses assessed at 30 months for Western Australia, the Northern Territory, Queensland and South Australia).

Figure 7: Trends in timeliness of the 3rd dose of DTP vaccine (DTP3) for cohorts born in 2001, 2004, 2006 and 2007*



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

Table 8: Vaccination coverage for 7vPCV, rotavirus, meningococcal C, varicella, hepatitis A (Indigenous only) and 23vPPV (Indigenous only) for the last 3-month cohort assessable in 2009, by state or territory

State or territory	Vaccine type					
	7vPCV*	Rotavirus†	Men C‡	Varicella§	Hep A‖	23vPPV¶
ACT	93.2	88.8	95.0	87.2	Na	Na
NSW	91.5	87.2	93.5	81.1	Na	Na
NT	89.8	82.6	92.1	82.4	86.2 (82.8)	78.9
Qld	91.5	82.6	93.3	85.2	49.3 (60.8)	47.5
SA	90.9	84.1	93.6	81.5	36.0 (44.0)	38.1
Tas	92.4	87.2	94.6	83.3	Na	Na
Vic	92.1	83.8	93.3	82.1	Na	Na
WA	89.2	83.2	94.3	80.6	60.3 (65.9)	57.0
Aust	91.4	84.8	93.7	82.4	59.0 (68.1)**	55.1**

Na Not applicable.

* 3 doses at 12 months of age.

† 2 or 3 doses at 12 months of age.

‡ 1 dose at 24 months of age.

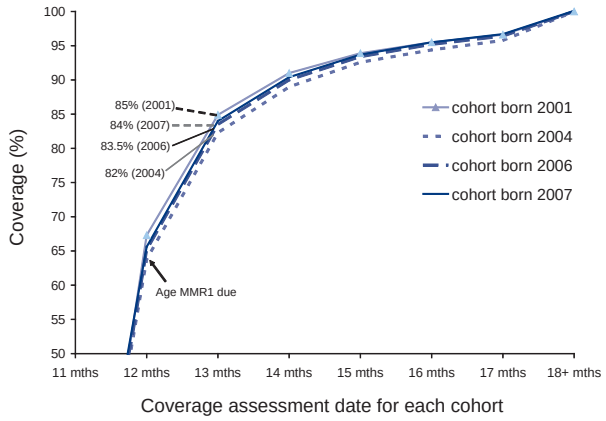
§ 1 dose at 24 months of age.

‖ Indigenous only: 2 doses by 30 months of age for Western Australia and the Northern Territory (1 dose by 18 months of age), 2 doses by 36 months of age for Queensland and South Australia (1 dose by 24 months of age).

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

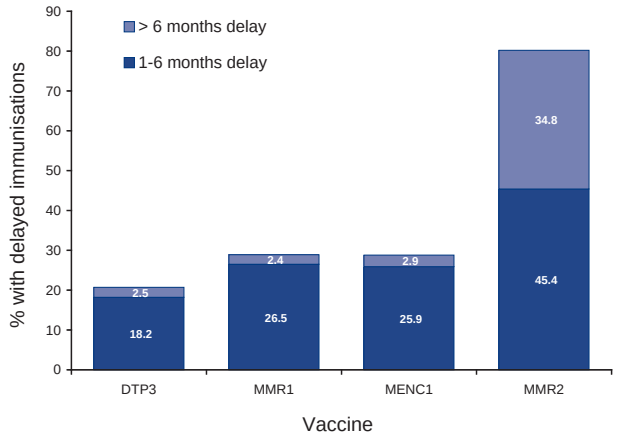
** Northern Territory, Queensland, South Australia and Western Australia only.

Figure 8: Trends in timeliness of the 1st dose of MMR vaccine (MMR1) – cohorts born in 2001, 2004, 2006 and 2007*



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

Figure 9: Vaccination delay for cohorts born in 2007 (DTP3, MMR1, MENC1) and 2003 (MMR2)



DTP3 = 3rd dose of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine.

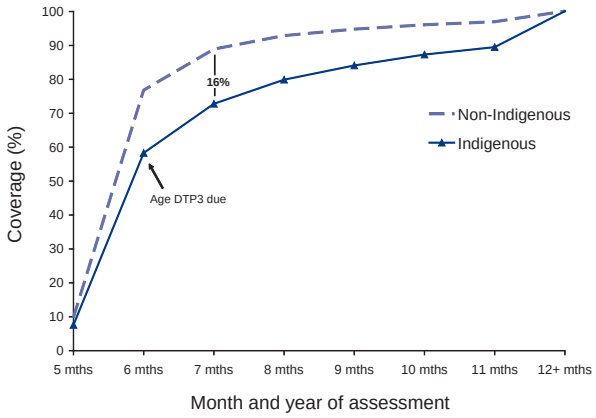
MMR1 = 1st dose of a measles, mumps and rubella vaccine.

MENC1 = 1st dose of a meningococcal C vaccine.

MMR2 = 2nd dose of a measles, mumps and rubella vaccine.

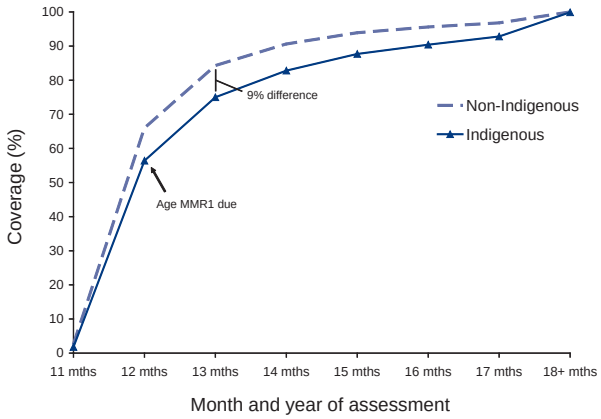
Figures 10 and 11 provide a comparison of timeliness of immunisation between Indigenous and non-Indigenous children in Australia for the 3rd dose of DTPa vaccine, and the 1st dose of MMR vaccine, respectively. For the 3rd dose of DTPa, there was a significantly greater delay for Indigenous children than non-Indigenous children, with a 16% differential at 7 months of age. This is a slight improvement from the previous annual report which found an 18% differential. The same pattern was found for timeliness of the 1st dose of MMR, but with a smaller differential of 9%, again a 2 percentage point improvement from

Figure 10: Timeliness of the 3rd dose of DTP vaccine (DTP3) for the cohort born in 2007,* by Indigenous status



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

Figure 11: Timeliness of the 1st dose of MMR vaccine (MMR1) for the cohort born in 2007,* by Indigenous status



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

the previous report. Although Indigenous children had only slightly lower coverage than non-Indigenous children by 24 months of age, they were more likely to have delayed vaccination.

Vaccination delay for Indigenous children for selected jurisdictions was measured for 7vPCV, with greater delays in Western Australia and South Australia (Figure 12). The proportion of children with long delays in receipt of the 3rd dose of 7vPCV vaccine in Western Australian and South Australian Indigenous children was about twice that in Queensland Indigenous children. There were no important differences in vaccination delay for non-Indigenous children by jurisdiction (not shown).

In contrast to earlier ages, analysis of timeliness of immunisation for a vaccine due at 4 years of age, the 2nd dose of MMR, showed similar delay in receiving this vaccine for non-Indigenous children and Indigenous children, with only a 0.4% differential at 4 years and 3 months of age (Figure 13).

Figure 12: Vaccination delay for Indigenous children for the 3rd dose of 7vPCV in selected jurisdictions, for the cohort born in 2007

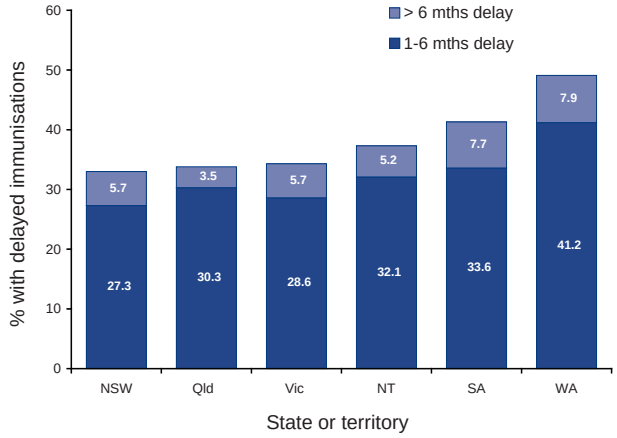
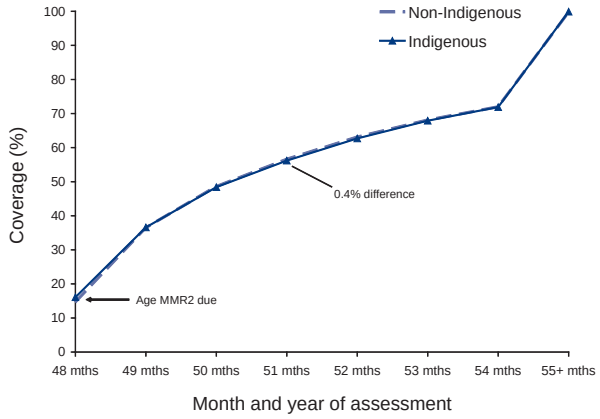


Figure 13: Timeliness of the 2nd dose of MMR vaccine (MMR2) for the cohort born in 2003,* by Indigenous status



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

Small area coverage

'Fully immunised' coverage for Australia by SSD for the 12-month, 24-month and 5-year milestone age groups, respectively, is shown in Figures 14–16. All three maps demonstrate that immunisation coverage in Australia in 2009 varies substantially within jurisdictions, with some areas having recorded coverage below the level required to prevent outbreaks of some highly contagious diseases such as measles. In particular, there are very few small areas in Australia where recorded 'fully immunised' coverage for vaccines due at 4 years of age is above the 90% level required to prevent disease.

The proportions of children recorded as conscientious objectors and with no vaccines recorded are presented by SSD in Figures 17 and 18, respectively. No vaccines recorded may represent either non-immunisation (parents refusing any vaccines) or, and probably much less commonly, non-reporting by a provider. The percentage of children with no vaccines recorded nationally (3.1%) is greater than those recorded as conscientious objectors (1.5%).

The map of the proportion of conscientious objectors to immunisation (Figure 17) shows pockets of high levels of objection within jurisdictions in 2009, particularly in coastal areas of south-east Queensland, northern New South Wales, Adelaide and south-west Western Australia, which would be hidden if these data were reported at broader geographical levels.

The map of the proportion of children with no vaccines recorded (Figure 18) shows some additional areas not evident from maps of official conscientious objection.

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 19. 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 a major number of immunisations nationally is community health centres. Regional differences are marked, with immunisations almost entirely administered by GPs in some jurisdictions, while in others a majority are given by local government and community health clinics.

Discussion

These data reveal that Immunise Australia Program coverage targets have been reached for children both 12 and 24 months of age. However, this is not the case for children 5 years of age where coverage is below the target in all jurisdictions.

Coverage at 24 months of age exceeds that at 12 months of age, and this is likely related to the exclusion of the varicella vaccine at 18 months from

Figure 14: 'Fully immunised' coverage at 12 months of age, by Statistical Subdivision, Australia, 2009

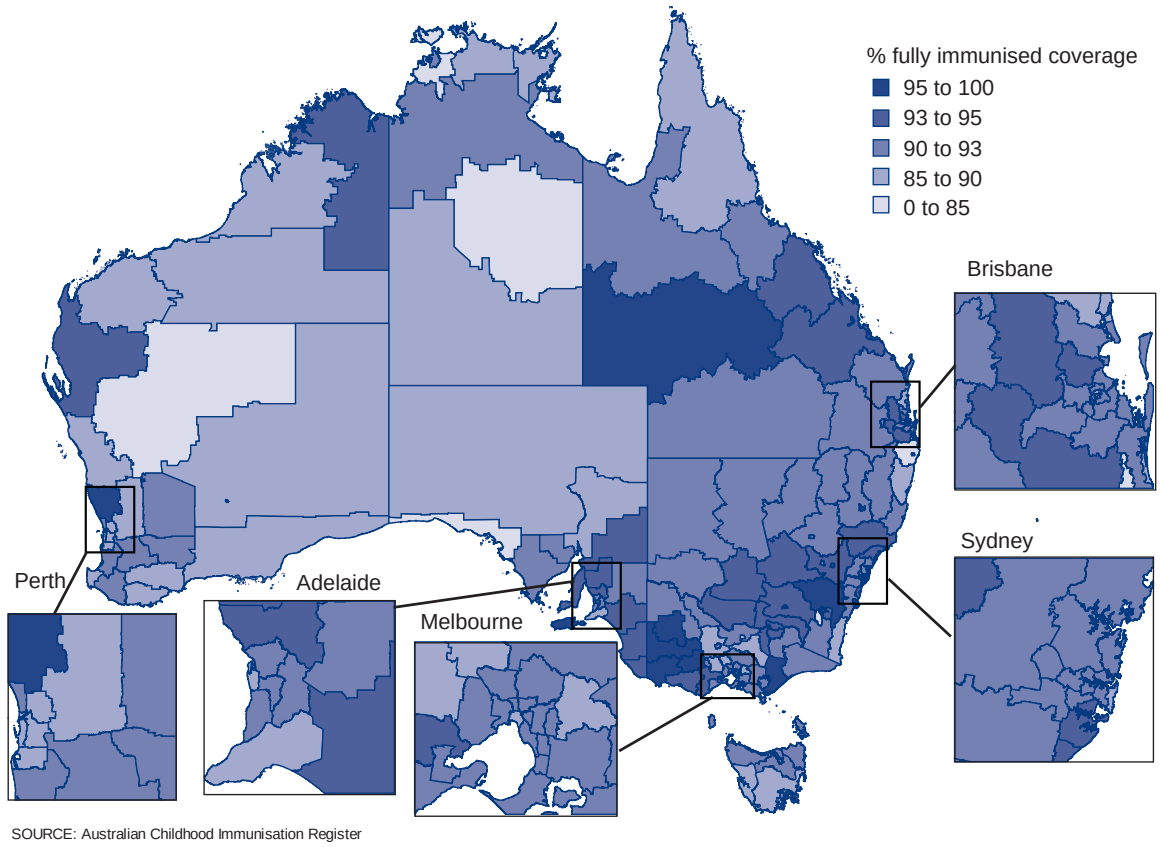


Figure 15: 'Fully immunised' coverage at 24 months of age, by Statistical Subdivision, Australia, 2009

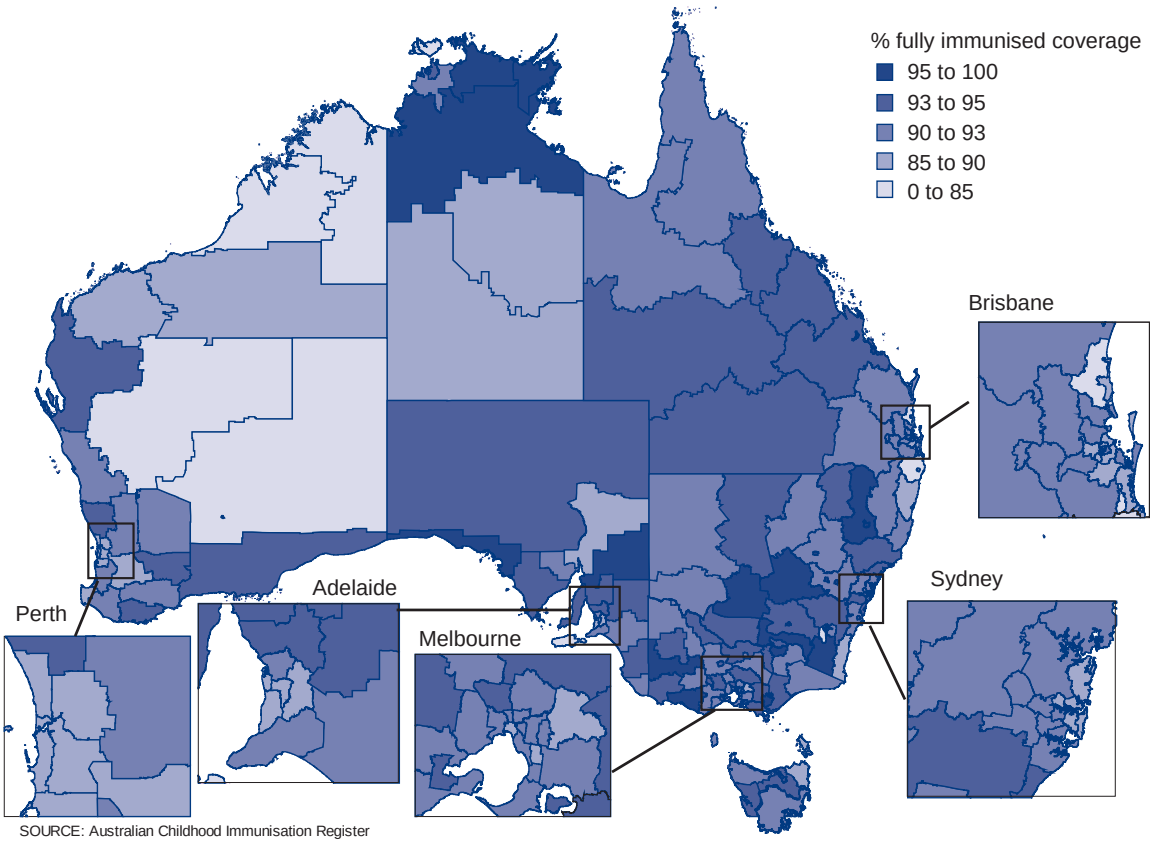


Figure 16: 'Fully immunised' coverage at 5 years of age, by Statistical Subdivision, Australia, 2009

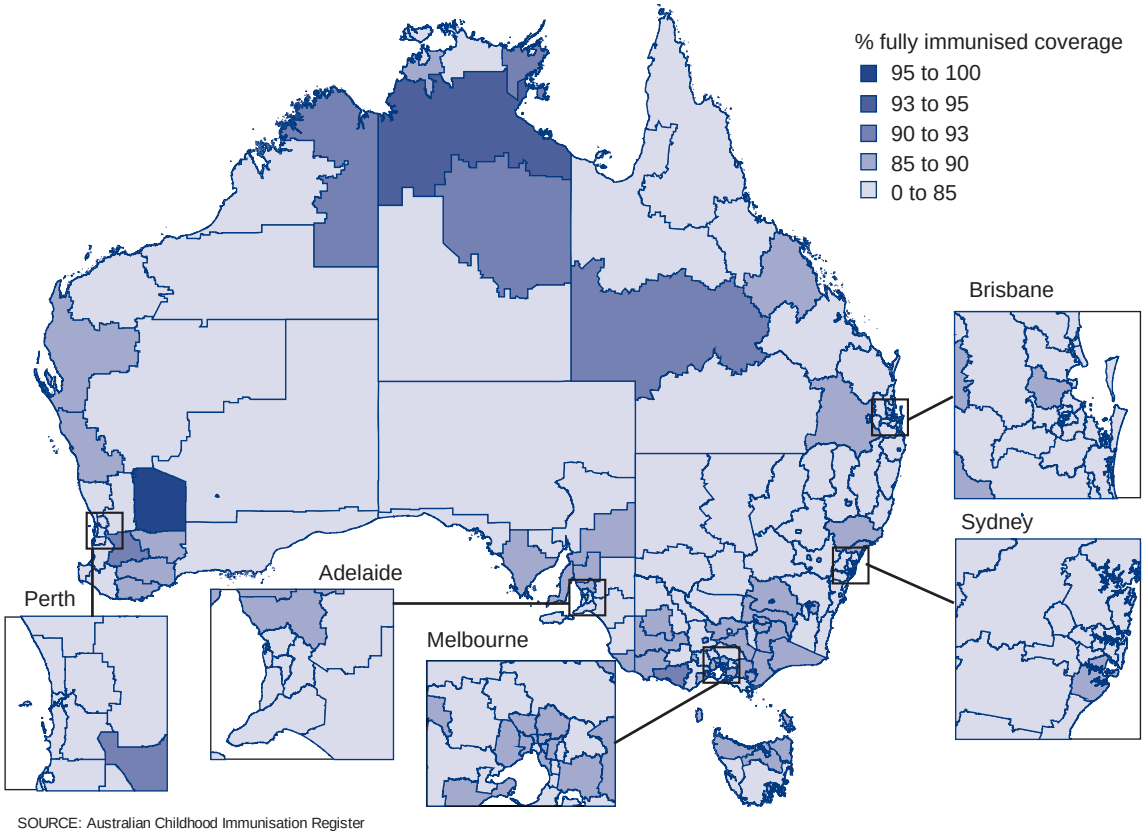


Figure 17: Proportion of official conscientious objectors to immunisation, Australia, 2009 for the cohort born January 2003 to December 2008

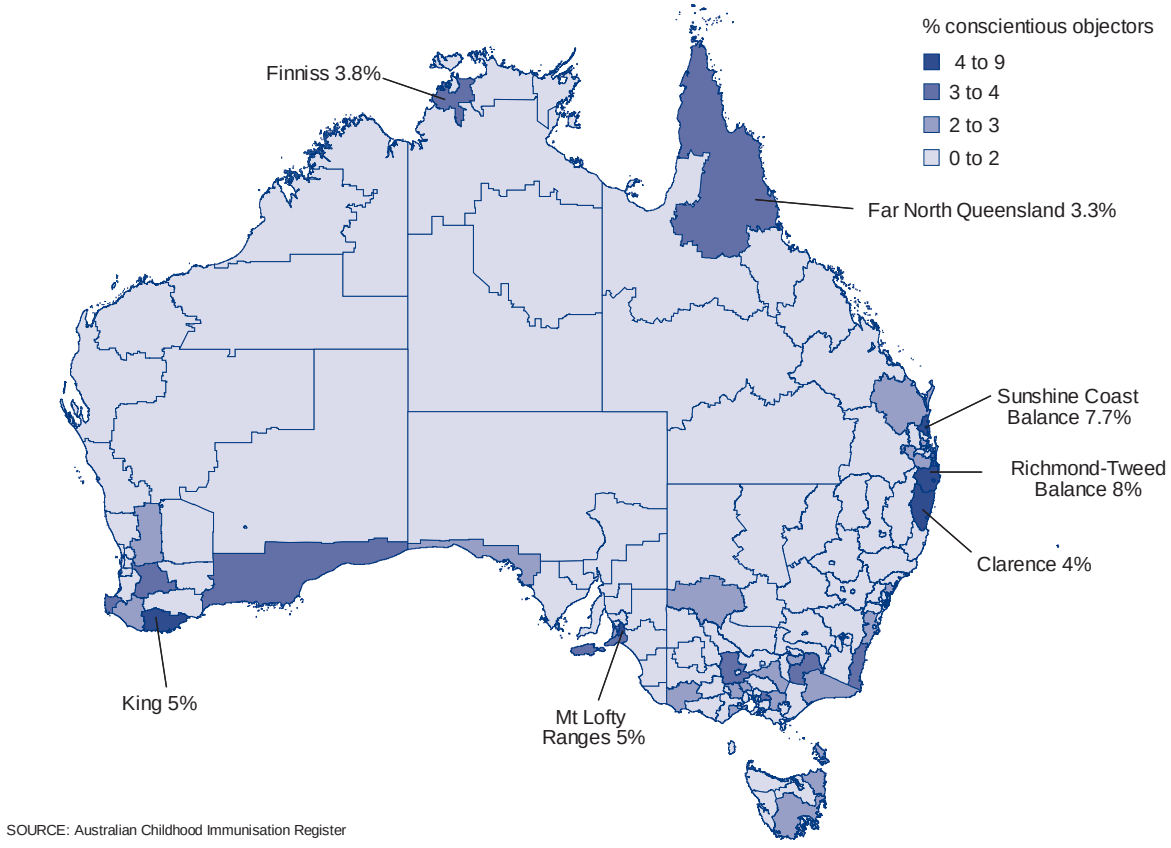
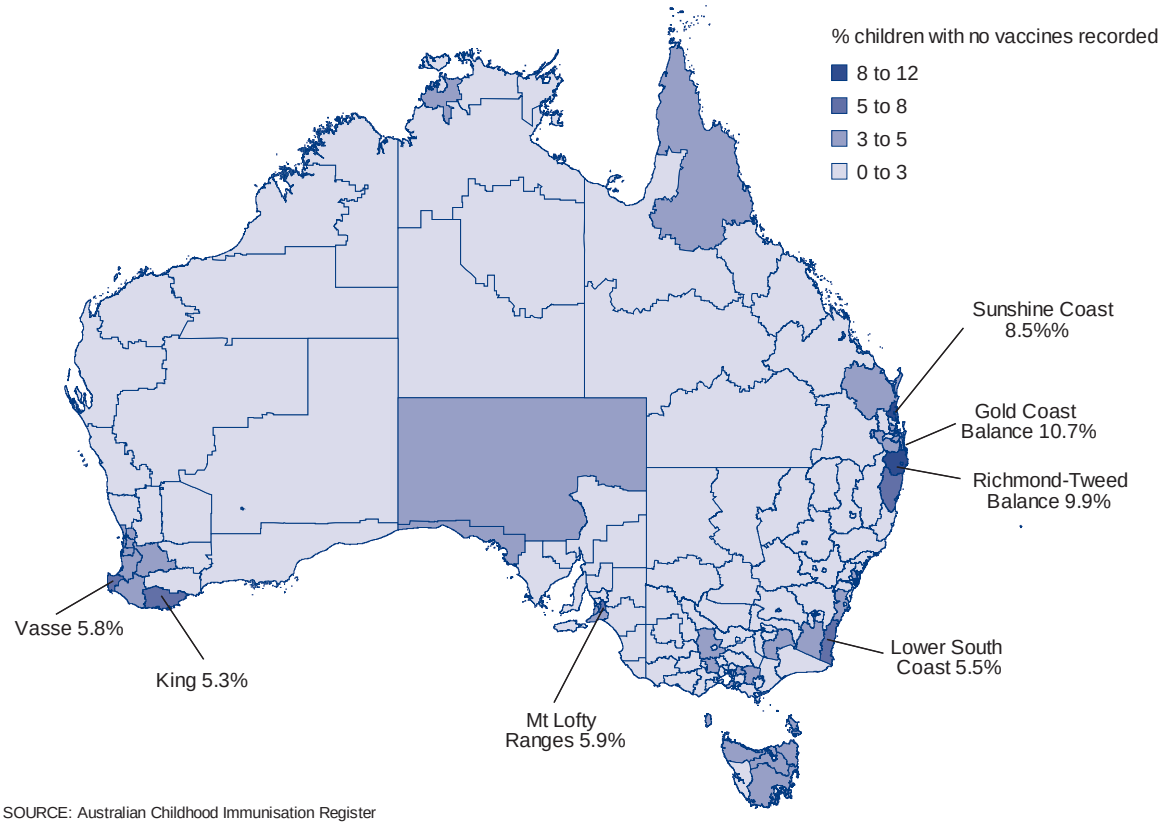
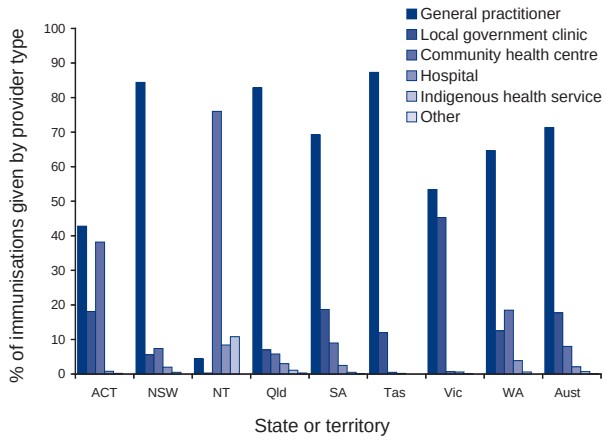


Figure 18: Proportion of children with no vaccines recorded on the Australian Childhood Immunisation Register, Australia, 2009 for the cohort born January 2003 to December 2008



SOURCE: Australian Childhood Immunisation Register

Figure 19: Proportion of immunisations on the Australian Childhood Immunisation Register given by various provider types, by state or territory, 2009



the calculation of 'fully vaccinated', the absence of any other vaccines administered between those ages, and the impact of immunisation incentives. The change in December 2007 in assessment age from 6 to 5 years for vaccines due at 4 years, resulted in lower coverage estimates for vaccines due at this age and has revealed that many children are not fully protected in a timely way for the diseases these vaccines guard against. This has been of particular concern during the pertussis epidemic of 2008 and 2009, when children aged 5 to 9 years were seriously affected.[24]

There is earlier evidence that immunisation incentives positively impacted coverage estimates.[14] However, there have been significant changes to incentives during 2008 for both providers and parents, in particular the removal of SIP payments and the reduction in the age cut-off for eligibility for payments for the 4 year vaccination. The initial analyses in this report provide no evidence of a reduction in coverage associated with the removal of SIP payments, while coverage at 5 years has increased following the age cut-off changes. However, more analysis is required to examine the impact of these changes in more detail.

A number of vaccines that are included in the NIP are not included when calculating 'fully immunised' status or in eligibility for incentive payments. Coverage estimates for 7vPCV and meningococcal C vaccines are comparable with estimates for vaccines that are included in 'fully vaccinated' calculations, but estimates for varicella and rotavirus are substantially lower. For rotavirus vaccines, strict upper age limits for administration may explain lower coverage, whilst varicella is the only vaccine

due at 18-months, and this milestone was historically problematic and lapsed for a 2-year period (2003–2005). The implications also vary. In the case of rotavirus vaccine, coverage of 80% or greater has been associated with substantial herd immunity and decreases in rotavirus hospitalisations in Australia and elsewhere.[25] In contrast, modelling studies suggest that low coverage with varicella vaccine will result in a shift of disease to older age groups with higher disease severity.[26] Inclusion of these and other vaccines included in the NIP in coverage assessments for 'fully immunised', and thereby in eligibility for provider and parent incentives, should be considered to drive higher coverage, especially at 18 months of age.

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and pneumococcal polysaccharide vaccine) remain sub-optimal. The extent of under-reporting to the ACIR for these vaccines is unknown but likely to be more than for 'universal' vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children[13] and adults.[27] Both a lack of provider knowledge about the recommendations for high risk groups, and poor identification of Indigenous children by immunisation providers, are also likely to be important contributing factors. Differences in schedules between jurisdictions may also contribute. Coverage for both vaccines is higher in the Northern Territory and Western Australia, which give the vaccines at 6 months younger (hepatitis A, 12 and 18 months, pneumococcal 18 months), than in Queensland and South Australia (18 and 24, and 24 months). The presence of other vaccines on the schedule at the same age may assist achieving higher coverage, particularly at 12 months and less so at 18 months of age. Failure to receive a 2nd dose by 9% of children also contributed to the low coverage for hepatitis A vaccine. However, protective antibody responses after 1 dose is expected from a majority of children.[28]

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. Vaccination delay as measured in this report has improved only marginally. However, timeliness cannot be measured in the most recent cohort, as time must be allowed for late vaccination to be received. It is expected that improvements in coverage recently seen at 5 years of age will be reflected in later timeliness calculations. However, coverage 12 months after the due date of this vaccine is still less than 85%. Poorer timeliness in Indigenous children has been noted previously in infants. In this report the disparity in coverage between Indigenous and non-Indigenous at children 5 years of age has widened from 2.7% in 2008 to 4.3% in 2009, as improvements in non-Indigenous children were not reproduced in Indigenous children. Delayed vaccination 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.

The ACIR has shown the rapid uptake of new vaccines and consistently high coverage for all vaccines, unlike some other developed countries.[29,30] In comparison with similar countries, reported coverage at 12 months of age is higher,[31] and, with more than 3% of children not vaccinated due to parental objection, targeting of on time vaccination is required to significantly improve the current levels of more than 91% fully immunised at 12 months of age. The reporting of national small area coverage data has not been noted elsewhere. Areas of low coverage have been identified in many remote areas and areas containing higher proportions of conscientious objectors. Vaccination timeliness has been reported elsewhere but not routinely.[8]

In conclusion, data provided by the ACIR in this report reflect continuing successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for varicella and rotavirus vaccines are below that for other vaccines; coverage is low in some small geographic areas; timeliness of vaccination could be improved, particularly for Indigenous infants; and coverage for vaccines recommended only for Indigenous infants is lower than for other vaccines. The ACIR continues to be a very useful tool for administering the NIP and monitoring its implementation.

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Surveillance of Creutzfeldt-Jakob disease in Australia: update to December 2010

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

Abstract

Since the establishment of the Australian National Creutzfeldt-Jakob disease Registry (ANCJDR) its activities have expanded from prospectively investigating additional iatrogenic Creutzfeldt-Jakob disease cases to include: retrospective ascertainment to 1970; provision of expert opinions in the area of infection control management; provide diagnostic testing services for all suspect cases; and maintenance of national and international collaborations in conjunction with routine surveillance responsibilities. An update of the ANCJDR's surveillance activities and outcomes between 1 April and 31 December 2010 is herein presented, including a summation of a recent publication by the ANCJDR. The shorter reporting period is due to a contractual change with the Department of Health and Ageing in 2010, resulting in the reporting timeframe shifting to align with full calendar years. *Commun Dis Intell* 2011;35(2):149–153.

Introduction

In October 1993, the Australian Government Department of Health and Ageing established the Australian National Creutzfeldt-Jakob disease Registry (ANCJDR) and have since charged this unit with the task of the surveillance of human prion diseases in Australia. The formation of this unit was underscored by the recommendations of the Allars Report,[1] which investigated the identification of 4 women who were recipients of human-derived pituitary hormones and who died of Creutzfeldt-Jakob disease (CJD) between 1988 and 1991. CJD is one form of the prion group of neurological disorders, which in humans, includes Gerstmann Sträussler-Sheinker syndrome, fatal familial insomnia, Kuru and variant CJD (vCJD) while bovine spongiform encephalopathy (BSE) in cattle, scrapie in sheep and chronic wasting disease in deer and elk represent principal animal forms of disease. This family of disorders, also known as transmissible spongiform encephalopathies (TSEs), causes a rapidly progressive neurological illness, ultimately leading to death. Globally, the annual incidence of CJD is around 1 case per million, although it is speculated that this may well be higher as has been indicated from several international surveillance centres,[2] where the annual incidence of 2 cases per million per year has been observed. The large majority of CJD cases have no known underlying cause and are thus classified as sporadic. The remaining cases are attributed to iatrogenic transmission or genetic predisposition. All suspect TSE cases referred to the ANCJDR are actively investigated and where possible, classified as definite, probable or possible according to the internationally recognised and validated clinical and neuropathological criteria.[3,4]

ANCJDR surveillance update to 31 December 2010

Notifications

Between 1 April and 31 December 2010 46 new suspect cases of CJD were notified to the ANCJDR. While this figure is reduced from previous reports based on 12 month periods, it is a reflection of the shorter reporting period of 9 months due to a change in contractual timeframes. Of these new suspect cases, nine have been confirmed as definite or probable cases, one has been classified as a possible case and three have been removed from the register. The remaining 33 are still under investigation with 16 of these cases still alive and 17 deceased. Neuropathological examination for nine of the deceased cases is pending.

Since establishment, a total of 1,468 cases of suspect CJD have been notified to the ANCJDR, comprising 308 notifications of case deaths prior to 1993 (retrospective cases) and 1,160 suspects notified prospectively. While this equates to around 80 notifications annually, the notification of prospective cases provides a more accurate estimate of annual national notifications. The average number of suspect case notifications for the period 1993 to 2010 is 64 cases per year or 3.2 cases per million population per year. This is almost 3 times greater than the rate of Australian confirmed cases for the same period (1.2 cases per million per year). Almost half of the prospective notifications stem from referrals for cerebrospinal fluid (CSF) 14-3-3 protein analysis (one of the diagnostic tests offered by the ANCJDR since 1997), while around a third are derived from personal communication from clinicians, family or hospitals. The remaining cases are ascertained through death certificate searches, hospital and health department searches and requests for other diagnostic services such as genetic testing.

By state and territory, analysis of the prospective suspect case notifications shows relatively stable levels since 2006 compared with previous years (Figure 1).

Prior to this report however, Tasmania was an exception to stable notification levels due to declining notifications since 2006. In 2010, 3 cases have been notified to the ANCJDR, returning the number of notifications to expected levels. A reduced number of notifications in New South Wales for 2008–2009 has been sustained in 2010, with around 10 fewer cases being notified for these 3 years compared with the 2006–2007 period.

Case outcomes

Of the 1,468 cases notified to the ANCJDR, 653 of these have been classified as probable or definite CJD cases (Table 1). An additional case of definite iatrogenic CJD is included in Table 1, due to pituitary hormone treatment occurring within Australia. However, due to the non-domestic location of onset and death, this case is not included in the Australian statistical analyses. The remaining, notified cases have been excluded after detailed follow-up investigation (573); are currently under evaluation (229); or have been classified as possible cases (13). Possible cases, classified as clinically likely but unable to meet diagnostic criteria, are excluded from all statistical analyses in this report.

Since the last reporting period, 14 suspect cases have been removed from the register, with 11 of these after neuropathological confirmation. A further definite CJD case, who was initially referred to the register during treatment in Australia, died overseas and was therefore removed from the register and not included as an Australian case due to the non-domestic location at death. For the 9-month reporting period, 20 cases were confirmed as definite cases and four

Figure 1: Prospective, suspect Creutzfeldt-Jakob disease case notifications to the Australian National Creutzfeldt-Jakob Disease Registry, 1997 to 2010, by state or territory

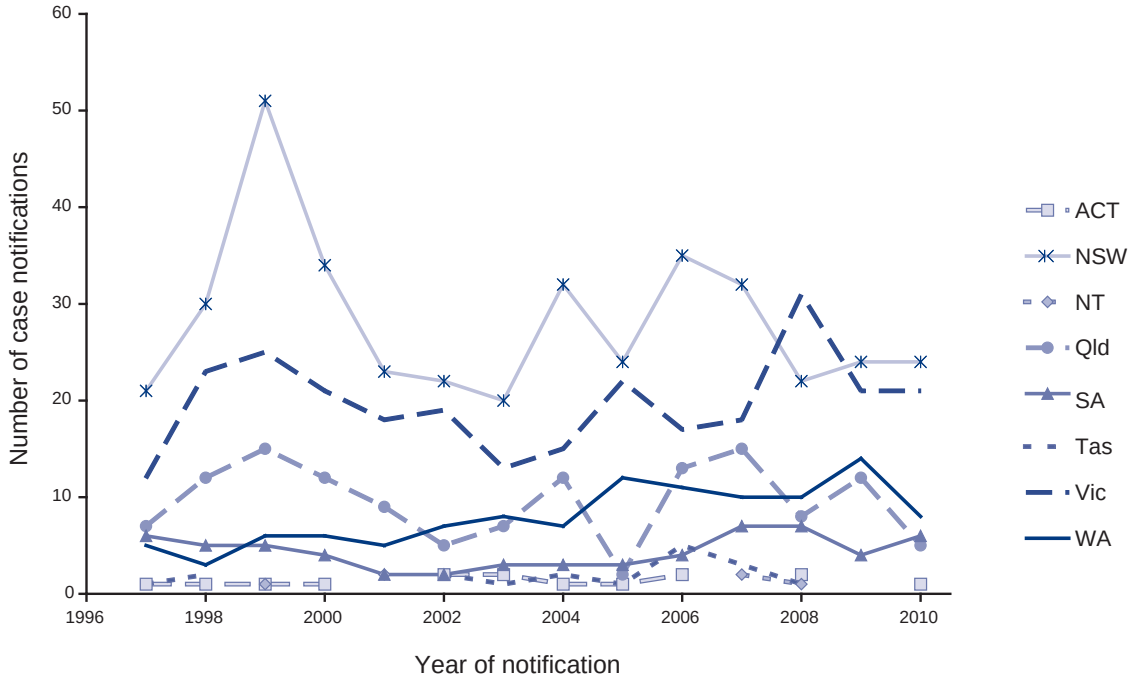


Table 1: Classification of cases by the ANCJDR, 1 January 1970 to 31 December 2010

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	385	43	5*	0	0	433
Probable	207	10	4	0	0	221
Possible	12	0	1	0	0	13
Incomplete	0	0	0	0	229†	229
Total	604	53	10	0	229	896

* 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 159 living cases.

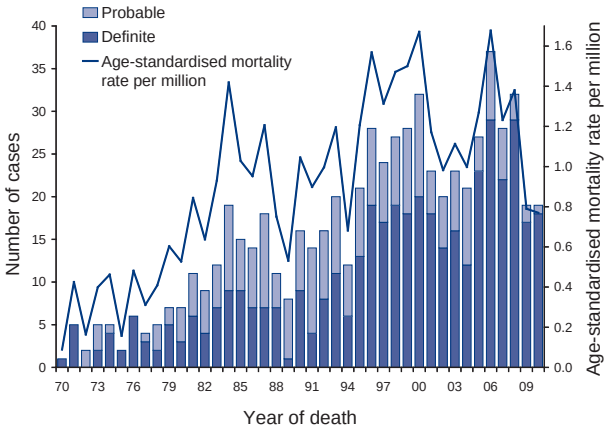
confirmed as probable cases, which is consistent with the number of cases classified in previous full 12 month reporting periods. This finding stems from the greater number of definite cases confirmed after post-mortem examinations performed in 2010 and relates to the reduced turnaround time for post-mortem examinations across some states and territories within this 9-month period, translating into more cases being confirmed in a shorter period of time. Of the new cases, 23 were classified as sporadic cases and one as a familial case.

The annual proportion of suspect cases notified to the ANCJDR where death is known to have occurred and have undergone post-mortem examination, has increased over time. This is to be expected given the active approach that the ANCJDR has undertaken to seek and facilitate post-mortem examinations in recent years. For the 1993 to 2010 period, 60% of all suspect case deaths have undergone post-mortem. It should be noted that this proportion is related only to cases where death is known to have occurred and the ANCJDR is aware that not all deaths are notified. In Victoria, further assistance with post-mortems has been provided through the formalisation of a contractual agreement with the Victorian Department of Health and the ANCJDR. The agreement has led to more expeditious and higher rates of autopsy in this state.

Based on the Australian population, the average crude rate of CJD-related post-mortems between 1993 and 2010 is 1.4 post-mortems per million per year, which is considerable given CJD is a particularly rare condition. By state and territory, the rate ranges from 0.8 CJD post-mortems performed annually per million in Tasmania to 1.5 in both Victoria and New South Wales. Despite the smaller populations in the Tasmania, the Northern Territory and the Australian Capital Territory, the post-mortem rates are all relatively consistent with more populous states and provide a level of confidence that suspect case deaths in these states and territories have a similar likelihood of undergoing post-mortem examination.

As reported previously, the annual incidence of CJD has steadily increased from 1970 to peak in 2000, 2006 and 2008 (Figure 2) with 1.4–1.6 cases per million per year being recorded, equating to 32 to 37 cases per year. For the overall period of 1993 to 2010, an average of 24.5 CJD cases per year are confirmed in Australia and the average age-standardised mortality rate is 1.2 cases per million per year. Although these long term averages align closely with rates observed in other countries with similar surveillance mechanisms in place,[2] it is believed that the incidence in the peak years, more closely reflects the true incidence of CJD in Australia. The ANCJDR therefore aims to achieve this level of case ascertainment.

Figure 2: ANCJDR definite and probable cases 1970 to 2010,* number and age-standardised mortality rate



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

* To 31 December 2010.

Delineation of the total case deaths by state and territory shows absolute numbers reflecting regional population distributions. The annual number of deaths from definite and probable TSE according to state and territory during 2000–2010 is shown (Table 2). The mean age-standardised rates (1993–2010) indicates that there is little variability between the larger regions of Australia with between 1.0–1.5 deaths per million occurring annually. These rates are in alignment with reported figures from other countries with similar surveillance mechanisms as those in Australia.[5] Furthermore, analysis of sporadic CJD standardised mortality ratios indicate that the rate of death was not found to be significantly different in any state or territory compared with the rate in the Australian general population, indicating that no state or territory had a greater or lower risk of CJD.

The highest TSE (all forms) mortality rates (1993–2010) were observed in Victoria and Western Australia (1.4 and 1.5 deaths per million per year, respectively). Previously, the lowest rates of mortality were observed in the Northern Territory and Tasmania and it was postulated that cases were being under-ascertained in these regions. More recently, an increase in confirmed cases in these less populated states and territories has contributed to the re-alignment of mortality rates to that of the larger states and territories. Tasmania continues to have the lowest TSE mortality in Australia; however, as previously discussed[5] an under-ascertainment of cases prior to 2000 may be responsible for skewing the overall incidence. Furthermore, a confirmed CJD case who was a permanent Tasmanian resident, but died interstate was not attributed to Tasmania due to the non-Tasmanian location at death. It should be noted

Table 2: Transmissible spongiform encephalopathy deaths and mortality rate, by state or 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	10		00–10*	93–10*
ACT			1		1		1		2		1	6	1.5	1.4
NSW	12	9	7	7	11	10	11	10	5	8	4	94	1.2	1.2
NT							2	1				3	0.9	0.8
Qld	7	3	3	3			7	2	4	3	1	33	0.7	1.0
SA	2			1	2	1	1	3	5	2	2	19	1.0	1.2
Tas			2			1	2					5	0.9	0.6
Vic	9	10	5	9	5	11	9	6	12	4	9	89	1.5	1.4
WA	2	1	2	3	2	4	4	6	4	2	2	32	1.3	1.5
Australia	32	23	20	23	21	27	37	28	32	19	19	281	1.2	1.2

* Includes all deaths occurring between the complete years 1 January 1993 or 1 January 2000 and 31 December 2010.

that the effect of 2 additional confirmed CJD cases in Tasmania would result in the mortality increasing to 0.9 cases per million per year, re-aligning mortality rates more closely to expected levels.

The age group with the highest mortality from all forms of CJD is amongst those aged 65–69 years where 8.3 deaths per million persons occur annually. From the age of 50, incidence increases to peak at 9.0 deaths per million per year in females in the 65–69 year age group and at 7.6 deaths per million per year in males in the 70–74 year age group (Figure 3). After these gender-specific peak age groups, mortality rates decline for both genders, although it should be noted that an increase in the detection of older age cases in recent years has led to a more rounded decline in the age-specific trend in the older age groups. Females are in slight excess (53%) for all forms of CJD, and this is true for familial (55%) and sporadic (53%) groups. In the small number of Australian iatrogenic cases, an equal number of males and females have been affected overall; 3 female pituitary hormone-related cases, 4 male and 1 female dura mater related cases.

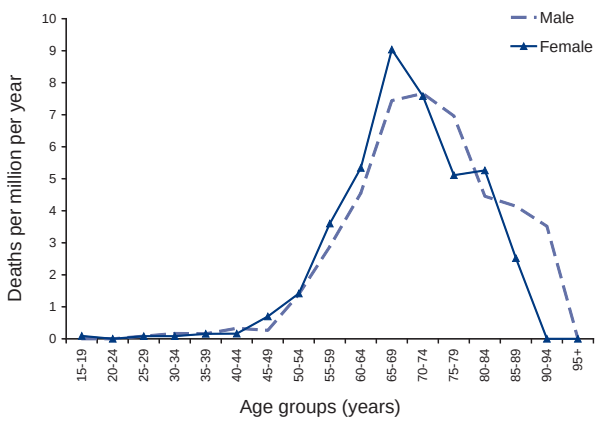
Since the last reporting period, there has been little change in the aetiological proportions of Australian TSE cases with the large majority occurring sporadically (90.7%) and the remainder classified as familial (8.1%) and iatrogenic (1.2%). A slight reduction in the genetic CJD forms has been observed in recent years and while the explanation for this is unclear at present, the incidence of genetic CJD will be closely monitored in future years. There have been no confirmed cases of vCJD, Kuru or further cases of iatrogenic CJD relating to recipients of dura mater grafts or pituitary hormone. The last deaths from iatrogenic CJD occurred in 1991 (pituitary hormone-related CJD) and 2000 (dura mater-related CJD).

As shown in Figure 3, the majority of Australian TSE cases occur after the age of 50 and this is true for all TSE aetiologies. The median age at death in the 653 confirmed cases is 66 years (range, 18–90 years), with the median age younger in familial cases (59 years, range 18–82 years) and iatrogenic cases (39 years, range 26–62 years), but overall closely aligns with the median age death of sporadic cases, given this CJD form represents 90.7% of all cases. Similarly, the median duration of disease from onset to death is 4 months for all cases (range 0.9–192 months) and the sporadic only case group (range 0.9–60 months), yet longer for both the familial case group (6 months, range 1.5–192 months) and the iatrogenic case group (6.5 months, range 2–25 months).

Recent publication

During 2010, the ANCJDR published several articles including an update on pituitary hormone cases in Australia, drawing on the experience in other countries for comparative analysis.[6] In brief,

Figure 3: Age- and sex-specific mortality rates in all Creutzfeldt-Jakob disease cases, 1993 to 2010



this review examined the ongoing risk for individuals who received pituitary hormone extracted from cadavers between 1967 to mid-1985 for the treatment of infertility and short stature under the Australian Human Pituitary Hormone Program. This program ceased in mid-1985 after the recognition of a linkage between treatment and CJD in a recipient in the United States of America. Australia had the lowest rate of pituitary hormone-related CJD cases across the countries compared, with the reasons for this not entirely clear. In addition, given 20 years has passed since the last case of pituitary hormone-related CJD was identified in Australia, the review discusses the current risk for the recipient community and raises the potential for changes to the infection control measures for this recipient cohort in the future.

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Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2008 and 2009

A report of the Australian Mycobacterium Reference Laboratory Network

Richard Lumb, Ivan Bastian, Robyn Carter, Peter Jelfs, Terillee Keehner, Aina Sievers

Abstract

There were 886 and 1,062 bacteriologically-confirmed cases of tuberculosis (TB) in 2008 and 2009, representing an annual rate of 4.1 and 4.9 cases per 100,000 population respectively. Over the 2 years, a total of 23 children aged under 10 years (male n = 13, female n = 10) had bacteriologically confirmed tuberculosis, including 3 children with TB meningitis. Results of *in vitro* drug susceptibility testing were available for 885 of 886 and 1,060 of 1,062 isolates for isoniazid (INH), rifampicin (RIF), ethambutol (EMB), and pyrazinamide (PYZ) in 2008 and 2009 respectively. In 2008, a total of 94 (10.7%) isolates of *Mycobacterium tuberculosis* complex were resistant to at least one of the anti-tuberculosis agents. Any resistance to INH was noted for 76 (8.7%), 23 (2.6%) for RIF, 10 (1.1%) for EMB and 9 (1.0%) for PYZ. Resistance to at least INH and RIF (defined as multidrug-resistant TB (MDR-TB) was detected in 21 (2.4%) isolates. None of the 21 MDR-TB isolates had resistance to either ofloxacin or the injectable agents. In 2009, a total of 168 (15.9%) were resistant to at least one of the anti-TB agents. Any resistance to INH was noted for 150 (14.2%) isolates, 37 (3.5%) for RIF, 5 (0.5%) for EMB and 13 (1.2%) for PYZ. A total of 31 (2.9%) isolates were MDR-TB. In 2009, there were 2 cases of quinolone resistance in MDR-TB from persons born overseas. Mono-resistance to INH was the most commonly detected resistance with 33 and 80 isolates in 2008 and 2009, respectively. Mono-resistance to RIF was infrequently encountered with 2 and 5 isolates in 2008 and 2009 respectively. There were six and 11 MDR-TB patients from the Papua New Guinea (PNG) – Torres Strait Islands (TSI) cross-border region in 2008 and 2009 respectively. The PNG-TSI zone now contributes a substantial proportion of MDR-TB cases to the database. In addition, there were 24 isolates of *Mycobacterium bovis* bacille Calmette Guérin (BCG), 15 were cultured from males (4 aged ≤ 5 years) and from 9 females (5 aged ≤ 5 years). The predominant site of isolation was from vaccination abscess. Eight males (range: 57–87 years) had *M. bovis* BCG isolated from urine or blood culture. *Commun Dis Intell* 2011;35(2):154–161.

Keywords: *Mycobacterium tuberculosis*, *Mycobacterium bovis*, laboratory diagnosis, drug resistance

Introduction

Australia continues to benefit from an effective national tuberculosis (TB) control program delivered through state- and territory-based TB services. The incidence of TB cases is low at between 5–6 cases per 100,000 population.[1] In contrast, the Western Pacific and South East Asia regions of the World Health Organization report far more incident cases (estimated) at 108 (2007) and 181 (2007) per 100,000 population, respectively.[2,3] These two regions account for almost 60% of the global burden of TB.

In Australia, drug resistance is mainly associated with people born in high-burden TB countries within the Western Pacific and South East Asian regions and reflects the performance of national TB programs in these regions.[2,3] Multidrug-resistant TB (MDR-TB) has remained within a low range of 0.5%–2.0%, although recent rises above 2.0% in 2006 (2.4%) and 2007 (2.8%) demand vigilance.[4,5] For the Western Pacific Region, the proportion of new cases with MDR-TB was estimated to be 4% and rising to 24% in re-treatment cases. Cases from China, the Philippines and Vietnam accounted for 97% of the total estimated MDR-TB (new and re-treatment).[2]

There are two sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on TB notifications reported to public health authorities in Australia's states and territories. The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986. Statistics compiled by the AMRLN relate to cases of bacteriologically-confirmed tuberculosis whereas NNDSS data also include cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations. This report describes the bacteriologically-confirmed TB diagnoses for the years 2008 and 2009.

Methods

The data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). Almost all isolates of MTBC were referred to

one of the five laboratories comprising the AMRLN for species identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia Beyond 2000* prepared by the National TB Advisory Committee,[6] were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases. Data include temporary visitors to Australia, immigrants or persons detained in Australia in correctional services facilities, and asylum seekers. For each new bacteriologically-confirmed case, the following information was collected where available: demography: patient identifier, age, sex, HIV status and state of residence; specimen: type, site of collection, date of collection and microscopy result; isolate: *Mycobacterium* species and results of drug susceptibility testing; nucleic acid amplification testing results; and for drug resistant isolates: patient country of origin, and history of previous TB treatment to determine whether resistance was initial or acquired. Data from contributing laboratories were submitted in standard format to the AMRLN coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for 2008 and 2009 supplied by the Australian Bureau of Statistics.[7,8] For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were counted as pulmonary disease. Patients with isolates recovered from multiple sites were counted as pulmonary disease (the most important category for public health purposes) if a sputum, bronchoscopy, or lung biopsy specimen was culture positive. Drug resistance among new cases (proxy for primary resistance) was defined as the presence of resistant isolates of *M. tuberculosis* in patients who, in response to direct questioning, denied having received any prior anti-TB treatment (for more than 1 month) and, in countries where adequate documentation is available, for whom there is no evidence of such a history.[9] Drug resistance among previously treated cases (proxy for acquired resistance) is defined as the presence of resistant isolates of *M. tuberculosis* in cases who, in response to direct questioning, admit having been treated for 1 month or more or, in countries where adequate documentation is available, for whom there is evidence of such a history.[9]

For 2009 onwards, the AMRLN has been requested by the National Tuberculosis Advisory Committee to provide laboratory data on bacteriologically confirmed isolation of *Mycobacterium bovis* (bacille Calmette Guérin) (BCG).

Results

There were 886 and 1,062 bacteriologically-confirmed cases of tuberculosis in 2008 and 2009, representing an annual rate of 4.1 and 4.9 cases per 100,000 population respectively. State-specific reporting rates varied from 0.6 (2008: Tasmania) to 11.4 (2008: Northern Territory) cases per 100,000 population. In 2009, all jurisdictions except South Australia and the Northern Territory recorded increased notification rates over 2008 levels (Table 1).

Causative organism

The overwhelming majority of the MTBC isolated were *M. tuberculosis* with a small number of *Mycobacterium africanum*, *M. bovis* and 'oryx' bacillus identified (Table 2).

Distribution by gender, age, and site of disease

Complete information for gender and age was available for 884 (99.8%) patients and 1,052 (99.1%) in 2008 and 2009 respectively. The distribution of bacteriologically confirmed cases by site is presented in Table 3.

Table 1: Bacteriologically confirmed cases of tuberculosis in Australia, 1998 and 1999, and 2007–2009, cases and rate per 100,000 population, by state or territory

State or territory	2009		2008		2007*		1999*		1998*	
	n	Rate	n	Rate	n	Rate	n	Rate	n	Rate
New South Wales†	409	5.5	327	4.5	343	5.0	291	4.3	289	4.4
Victoria	331	6.1	299	5.6	279	5.4	261	5.5	192	4.1
Queensland	153	3.4	111	2.6	118	2.8	75	2.1	85	2.5
Western Australia	87	3.9	72	3.3	45	2.1	64	3.4	66	3.7
South Australia	51	3.1	49	3.1	46	2.9	46	3.1	40	2.7
Tasmania	7	1.4	3	0.6	8	1.6	2	0.4	6	1.3
Northern Territory	24	10.9	25	11.4	33	15.4	21	10.9	22	11.6
Total	1,062	4.9	886	4.1	872	4.1	760	4.0	700	3.7

* Data from previous reports of the Australian Mycobacterium Reference Laboratory Network.

† Data from the Australian Capital Territory are included with those from New South Wales.

The site of disease was dependent upon age and gender. The overall male:female ratio was 1:0.7 and 1:0.9 for 2008 and 2009 respectively for respiratory isolates. Males were predominant but females accounted for the greatest number of lymphadenitis (Table 4). In 2009, for males, there were two distinct peak age groups in bacteriologically-confirmed rates: a rise to 11.6 cases of TB per 100,000 population at 25–29 years of age and a second peak in the elderly (males aged more than 84 years (up to 20.5 cases of TB per 100,000 population). A similar age distribution in female cases occurred with 12.9 and 3.6 bacteriologically-confirmed TB cases per 100,000 population at the 25–29 and greater than 84 years age groups, respectively. The median age group for patients with bacteriologically-confirmed disease was 30–34 years for both males and females.

Respiratory samples were the predominant culture-positive specimen type with sputum and bronchoscopy specimens being the 2 most common specimen types (Table 3). The most commonly encountered

Table 2: Members of the *Mycobacterium tuberculosis* complex isolated in the years 2008 and 2009

Organism	2009	2008
M. tuberculosis	1,052	881
M. bovis	3	4
M. africanum	3	0
'Oryx' bacillus	4	1
Total	1,062	886
M. bovis (BCG)	24	N/A

N/A Not available

Table 3: Site of specimens smear- and culture-positive for *Mycobacterium tuberculosis* complex, 2008 and 2009

	2009			2008		
		Smear positive			Smear positive	
	N*	n*	%	N*	n*	%
Sputum	472	240	50.8	407	203	49.9
Bronchoscopy	137	39	28.5	119	36	30.3
Lymph node	250	44	17.6	156	31	19.9
Pleural	48	2	4.2†	41	2	4.9‡
Genito-urinary	28		§	27		§
Bone/joint	36		§	21		§
Peritoneal	25		§	29		§
Skin	9		§	8		§
Cerebrospinal fluid	10		§	7		§

* Based on specimens that reported a microscopy result and excludes (i) microscopy not performed or (ii) result unknown.

† One pleural biopsy and 1 fluid were smear positive.

‡ Two pleural biopsies only were smear positive.

§ Percentage of specimens smear positive not calculated due to the small number of cases.

Table 4: Distribution and site of disease, 2008 and 2009, by age and sex

	2009		2008	
Male/female	n	ratio	n	ratio
All	568/493	1:0.9	525/359	1:0.7
Respiratory	356/272	1:0.8	354/189	1:0.5
Lymph node	108/142	1:1.3	62/94	1:1.5
Median age – male				
All	30–34		30–34	
Respiratory	35–39		35–39	
Lymph node	25–29		30–34	
Median age – female				
All	30–34		30–34	
Respiratory	30–34		30–34	
Lymph node	30–34		30–34	

extrapulmonary culture-positive specimen was lymph tissue followed by pleural, peritoneal, bone/joint, and genitourinary tract (Table 3).

In 2008 and 2009, a total of 23 children aged under 10 years (male n = 13, female n = 10) had bacteriologically confirmed tuberculosis (sputum n = 4, gastric aspirate n = 4, lymph node n = 5, bone/joint n = 3, cerebrospinal fluid (CSF) n = 3, and one each from oropharyngeal aspirate, pleura, pus and bronchoscopy.

Association with HIV

For 2008 and 2009, the AMRLN database recorded the HIV status of only 77 (8.7%) and 96 (9.0%) patients. One bacteriologically-confirmed patient was HIV positive in 2009.

Microscopy

Results of acid-fast microscopy were available for 866 of 886 (97.7%) and 1,054 of 1,062 (99.2%) specimens in 2008 and 2009 respectively.

For 2008, smears were positive in 203 of 407 (49.9%) sputum and 36 of 119 (30.3%) bronchoscopy specimens respectively (Table 3). Of 41 pleural specimens (17 biopsy and 24 fluids) that were culture-positive for *M. tuberculosis*, 2 biopsies only were smear-positive. Lymph node specimens were smear-positive in only 31 of 156 (19.9%) cases. The corresponding figures for 2009 were 240 of 472 (50.8%) sputum and 39 of 137 (28.5%) bronchoscopy specimens. Of 48 pleural specimens (17 biopsies and 31 fluids), two (4.2%) were smear positive. Only 44 of 250 (17.6%) lymph node specimens were smear positive (Table 3).

Drug susceptibility testing

Results of *in vitro* drug susceptibility testing (DST) were available for 885 of 886 and 1,060 of 1,062 isolates for isoniazid (INH), rifampicin (RIF), ethambutol (EMB), and pyrazinamide (PYZ) in 2008 and 2009 respectively. A single strain failed to grow on subculture in both years. In 2009, a single strain was mixed with *Mycobacterium fortuitum* and could not be isolated as a pure growth. None of the *M. bovis* isolates (which are inherently PYZ-resistant) were included in the following results. Therefore, a total of 881 and 1,057 isolates were available for DST in 2008 and 2009 respectively. For the first time, streptomycin (STR), when available, was included in the drug resistance profiles (Table 5).

In 2008, a total of 94 (10.7%) isolates of *M. tuberculosis* complex were resistant to at least one of the above anti-tuberculosis agents. Any resistance to INH was noted for 76 (8.7%), 23 (2.6%) for RIF 10 (1.1%) for EMB and 9 (1.0%) for PYZ. Resistance to at least INH and RIF (defined as MDR) was detected in 21 (2.4%) isolates (Table 5). Of the 21 MDR-TB isolates, 16 were from the respiratory tract (sputum n = 13, bronchoscopy n = 3), lymph node (n = 4) and a single isolate from fluid (site not stated). Eight of the MDR-TB-positive sputum specimens were smear-positive as were 2 bronchoscopy samples. The most common drug resistance profile among MDR-TB strains was resistance to INH/RIF only, a trend continuing from previous years (Table 6). Please note that Table 5 has STR-resistance included but that Table 6 does not include STR-resistance as historical data cannot be accessed. Therefore, data distortions

Table 5: Drug resistance profiles, 2008 and 2009

	2009	2008
Total isolates	1,062	886
Total isolates and DST	1,057*	881*
Fully susceptible	889†	787†
Any resistance		
S	61	53
H	150	76
R	37	23
E	5	10
Z	13	9
Mono-resistance		
S	7	16
H	80	33
R	5	2
E	1	0
Z	3	0
Multidrug-resistant tuberculosis		
HR	12	3
HRE	1	0
HRZ	3	1
HREZ	1	1
SHR	9	7
SHRE	0	3
SHRZ	4	2
SHREZ	1	4
Poly-resistant		
SR	1	0
SH	38	19
SE	1	0
HE	0	1
HZ	1	0
SHE	0	1
SHZ	0	1

* Excludes no drug susceptibility testing (DST) available and no *Mycobacterium bovis.*

† Includes streptomycin resistant strains.

Streptomycin (S), isoniazid (H), rifampicin (R), ethambutol (E), pyrazinamide (Z)

occur inevitably and MDR-TB data for 2008 is an excellent example. In Table 5, there were 3 INH/RIF-resistant strains and 7 STR/INH/RIF-resistant strains but 10 INH/RIF-resistant strains recorded in Table 6 where STR-resistance was not considered. None of the 21 MDR-TB isolates in 2008 had concomitant resistance to ofloxacin or a second-line injectable agent (kanamycin, amikacin, capreomycin).

In 2009, a total of 168 (15.9%) isolates were resistant to at least one of the anti-tuberculosis agents. Any resistance to INH was noted for 150 (14.2%), 37 (3.5%) for RIF, 5 (0.5%) for EMB and 13 (1.2%) for PYZ. A total of 31 (2.9%) isolates were MDR-TB. Twenty-three isolates were from the respiratory tract (sputum n = 21, bronchoscopy n = 2), lymph node n = 3, peritoneal n = 2, and one each from pleural biopsy, bone, and CSF. Ten of the MDR-TB-positive sputum specimens were smear-positive and all of the specimens from extrapulmonary sites were smear negative. In 2009, there were 2 cases of quinolone resistance in MDR-TB from persons born overseas (Indonesia, China). In 2008 and 2009, no cases were detected of extensively drug resistant tuberculosis (XDR-TB), defined as MDR-TB strains with additional resistance to a quinolone and a second-line injectable agent.

Overall, mono-resistance to INH was the most-commonly-detected resistance profile with 33 and 80 isolates in 2008 and 2009 respectively (Table 5). Mono-resistance to RIF was infrequently encountered with 2 and 5 isolates in 2008 and 2009 respectively. For 2009, three of the 5 mono-RIF resistant strains were found in patients from the Papua New Guinea–Torres Strait Islands (PNG–TSI) zone (Table 7). Resistance to STR/INH was the most frequent form of poly-resistance with 19 and 38 isolates in 2008 and 2009 respectively (Table 5).

In 2008, 6 MDR-TB patients were PNG nationals from the Western Province and who are able to access the PNG–TSI cross-border region. These patients access health services in outer TSI and receive treatment in Australia. In 2009, another 11 DR-TB patients were from the PNG–TSI zone. The impact of MDR-TB arising from the PNG–TSI zone is demonstrated in the Figure. In 2009, only 10 of 30 PNG–TSI patients had a fully susceptible strain; mono-resistance, including three with RIF-mono-resistance was found in 5 patients, STR/INH-resistance in 4 patients, and 11 had MDR-TB (Table 7).

New or previously treated cases, and country of birth

The majority of drug resistance was considered to be primary acquisition of a drug resistant strain; 47/78 (60.3%) in 2008 and 124/168 (73.8%) in 2009.

Table 6: Drug resistance patterns in multidrug-resistant strains, Australia, 1995 to 2009

Resistance pattern (standard drugs)*	2009	2008	2007	2006	2005	2004	2003	2002	2001	2000	1999	1998	1997	1996	1995
H+R only	21	10	16	16	5	7	4	8	8	3	2	2	6	10	3
H+R+E	1	3	2	1	3	2	2	1	1	1	1	1	1	1	1
H+R+Z	7	3	5	0	1	1	1	1	3	3	1	2	5	4	1
H+R+E+Z	2	5	1	5	3	2	0	2	0	1	0	1	2	0	0
XDR-TB	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
Total (%)	31 (2.9)	21 (2.4)	24 (2.8)	22 (2.4)	12 (1.5)	12 (1.5)	7 (0.9)	12 (1.7)	12 (1.6)	8 (1.0)	4 (0.5)	6 (0.9)	14 (1.9)	15 (2.0)	5 (0.7)

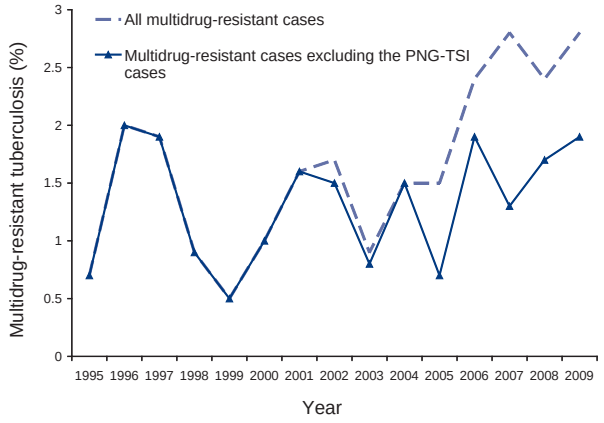
* The streptomycin result was not considered for this table.
H = isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide
XDR-TB Extensively drug resistant tuberculosis

Table 7: Drug resistance profile of *Mycobacterium tuberculosis* isolates from the Papua New Guinea–Torres Strait Islands cross-border region, 2009

Drug resistance	N
Fully susceptible	10
Mono-resistance	
S	1
H	1
R	3
Poly-resistance (not MDR-TB)	
SH	4
Multidrug-resistant tuberculosis	
HR	3
HRZ	1
SHR	6
SHRZ	1
Total	30

Streptomycin (S), isoniazid (H), rifampicin (R), ethambutol (E), pyrazinamide (Z)

Figure: Percentage of multi-drug resistant tuberculosis in Australia: the impact of cases from the Papua New Guinea–Torres Strait Island zone



There were 6 and 12 Australian-born cases of drug-resistant tuberculosis in 2008 and 2009 respectively. The overseas-born persons with drug-resistant disease were from 20 and 27 countries respectively for 2008 and 2009. The countries of birth were predominantly from 5 countries; India, Papua New Guinea, Vietnam, China, The Philippines (data not shown).

Isolation of *Mycobacterium bovis* BCG

There were 24 isolates of *M. bovis* BCG in 2009. Fifteen were cultured from males (4 aged ≤ 5 years) and from 9 females (5 aged ≤ 5 years). The predominant site of isolation was from the vaccination site or axilla (n = 13). Nine of these 13 patients were less than 5 years of age. Eight males (age range: 57–87 years) had *M. bovis* BCG isolated from urine or blood culture (n = 2). The isolation of *M. bovis* BCG from the sputum of a 23-year-old patient was associated with a neck node and draining sinus.

Discussion

The detection of 886 laboratory-confirmed cases of TB in 2008 (i.e. 4.1 cases per 100,000 population) is consistent with previous AMRLN reports with the incidence of bacteriologically confirmed TB generally between 3.5–4.4 cases per 100,000 population (see previous AMRLN reports). In contrast, in 2009, the AMRLN recorded 1,062 cases of bacteriologically confirmed tuberculosis with an incidence of 4.9 cases per 100,000 population, the highest figure recorded since laboratory data were first collected nationally in 1985. All jurisdictions, except the Northern Territory and South Australia had increases in the incidence rate of bacteriologically confirmed disease. Increases in the laboratory diagnosis of respiratory- and lymph node- disease accounted for the majority of additional cases in 2009.

As expected, the number of cases notified to NNDSS was higher than for bacteriologically confirmed TB. There were 1,212 and 1,334 notifications of tuberculosis in 2008 and 2009 respectively compared with 886 (73%) and 1,062 (80%) of cases confirmed bacteriologically.[10] The most frequent reasons postulated for the extra cases reported in the NNDSS database include: diagnosis of childhood and extrapulmonary TB based on clinical, radiological and epidemiological information; and submission of extrapulmonary samples in formalin precluding bacteriological investigations.

The format for documenting drug resistance has changed from previous reports and is now more consistent with requirements of the World Health Organization. For the first time, streptomycin has been included more formally in the drug resistance data and has resulted in some changes to the proportion of strains with drug resistance. In 2008 and 2009, there were an additional 7 and 16 isolates respectively with mono-resistance to streptomycin. This change in analytic methodology results in modest increases in total drug resistance reported (e.g. for 2009, overall drug resistance increased from 15.3% to 15.9% when mono-streptomycin resistance was included).

The rise in the isolation of drug resistance to 15.9% for 2009 was the highest percentage since the MRLN began data collection in 1985. The previous highest had been in the years 1989–1992 where 14.4% of isolates were resistant to at least one of the four anti-

tuberculosis drugs. That report did not consider streptomycin resistance indicating that the overall resistance would have been slightly higher.[11]

MDR-TB remains at a low level but there are reasons for concern. Since 2006, the proportion of MDR-TB isolates has risen above the long-term range of 0.5%–2.0%, due to patients in the PNG–TSI zone presenting for treatment. In 2009, there were 31 (2.9%) cases of MDR-TB but when the 11 PNG–TSI patients were excluded, the proportion decreased to 1.9%. However, the level of drug resistance in the PNG–TSI zone patient group is most disturbing. Of a total of 30 patients identified as being from PNG–TSI, only 10 patients had a fully susceptible strain. Importantly, 3 patients had mono-resistance to rifampicin meaning that 14 of 30 PNG–TSI patients had *in vitro* resistance to at least rifampicin. A further 4 patients had resistance to streptomycin and isoniazid; considered to be a precursor to MDR-TB.[12] Although there is highly likely to be patient bias in the limited data, the figures are of great concern for PNG TB control and for Australia.

The recognition of *M. tuberculosis* isolates with low level rifampicin is another matter of concern. All Australian laboratories are now using the Becton Dickinson MGIT 960 automated liquid culture (MGIT) system for primary isolation and for first- and second-line drug susceptibility testing. A recent paper by Van Deun and colleagues highlighted that isolates with low-level resistance to rifampicin may not be detected by the MGIT system.[13] The *rpoB* mutations associated with low level resistance included Leu511Pro, Asp516Tyr ,His526Leu, His526Ser, Ile572Phe, and 533Pro.[10] Met515Ile has also been associated with low level rifampicin resistance.[14] The authors concluded that low-level but probably clinically relevant rifampicin resistance linked to specific *rpoB* mutations, is easily missed by standard growth-based methods, particularly the automated broth-based systems. The critical rifampicin concentration for the MGIT system is 1.0 mg/L but the problematic strains had minimal inhibitory concentrations 0.13–0.38 mg/L. The frequency of these low level rifampicin resistant isolates is presently unknown. The bacteriologically unfavourable treatment outcomes for most of the borderline rifampicin resistant strains suggest that these specific mutations may have clinical significance. Laboratories undertaking DST using the MGIT system should consider performing *rpoB* sequencing in the following circumstances: (i) all rifampicin resistant strains and (ii) for isolates where isoniazid resistance is reported.

An alternative strategy to sequencing would be wider use of one of the commercially-available molecular tests, Hain Genotype MTBDR*plus*[15,16] or the GeneXpert MTB/RIF.[17,18] Both are able to detect MTBC nucleic acid and to detect gene mutations associated with rifampicin resistance, including low level resistance. The Hain Genotype MTBDR*plus* assay is able to detect mutations associated with resistance to rifampicin (*rpoB*) and isoniazid (*katG*, *inhA*) either directly from processed sputum specimens or from culture.[15,16] The laboratory testing protocol is based on a conventional multiplex nucleic acid amplification followed by reverse hybridisation on a solid phase. For smear positive respiratory specimens, the sensitivity and specificity for rifampicin approaches 100% for both assays. For the Genotype MTBDR*plus* assay, the sensitivity and specificity for isoniazid resistance may also be high but appears to vary between geographic regions depending on the proportion of isolates that are phenotypically isoniazid resistant, but for which no resistance mutation is found in *katG* or *inhA*.[16] The recently released Genotype MTBDR*sl* assay uses the same technology to detect mutations associated with injectable agents (kanamycin, capreomycin, amikacin), quinolones, and ethambutol.[19,20] Using isolates of *M. tuberculosis*, the sensitivity and specificity respectively for quinolones, kanamycin, amikacin, capreomycin, was 87%–90.2% and 90.2%–100%, 77% and 100%, 83.3%–100% and 100%, and 80%–86.8% and 98%–99.1%. The results for ethambutol were disappointing with both studies reporting sensitivities less than 60%.[19,20] For both Genotype assays, the time to obtain results is less than 2 days.

In contrast to the conventional molecular protocols of the Genotype assay, the GeneXpert MTB/RIF is a radical departure with all reactions occurring within a single-use disposable cartridge loaded into a module and controlled via a computer. A technical knowledge of molecular protocols is not required nor are specialised molecular laboratory facilities. Test samples may be direct or processed sputum, or positive cultures. The lysis buffer used early in the testing protocol will inactivate more than 95% of viable tubercle bacilli meaning that the platform may be used outside of the TB laboratory. The assay is stand-alone once the cartridge has been placed into the machine and testing is completed in less than two hours. In a multi-country evaluation, the sensitivity for detecting MTBC nucleic acid in smear positive and smear negative specimens was 98.2% and 72.5% respectively and with 100% specificity. For rifampicin resistance, the assay achieved a sensitivity of 97.6% and specificity of 98.1%.[19] Most of the AMRLN laboratories now have a GeneXpert platform in their laboratory.

The much anticipated merging of the AMRLN and NNDSS databases has experienced further delays due to information technology limitations and transitions in various states. A combined database will not be available before the 2012 dataset at the

earliest. In the interim, Australia must continue to provide a combined prevalence of drug resistance and remains unable to provide comprehensive data to the World Health Organization global reports sub-classifying drug-resistance between new cases and previously-treated patients.

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The Australian Mycobacterium Reference Laboratory Network comprises the Mycobacterium Reference Laboratories at the following facilities:

SA Pathology, Adelaide, South Australia

Queensland Health Pathology Services, Herston Hospitals Complex, Herston, Queensland

Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria

PathWest Laboratory Medicine WA – QEIIMC, Hospital Avenue, Nedlands, Western Australia

Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales.

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Peer-reviewed articles

Norovirus in residential care facilities: Does prompt notification of outbreaks help?

Craig A Davis, Hassan Vally, Frank H Beard

Abstract

Outbreaks of viral gastroenteritis occur regularly in residential care facilities (RCFs), with norovirus being the most common agent. Notification of outbreaks to public health authorities is encouraged in Australia, although there is limited evidence that this results in public health benefit. The aim of this study was to investigate if prompt notification of suspected norovirus outbreaks to public health authorities is associated with a reduction in either the duration or attack rate of outbreaks. Viral gastroenteritis outbreaks notified from Queensland RCFs between 2004 and 2007 were analysed. Foodborne outbreaks were excluded, along with 6 outbreaks where viruses other than norovirus were identified as the causative agent. Of the 264 remaining outbreaks, 70.8% were laboratory-confirmed as being due to norovirus. The average time to notification was 4 days and the average duration of outbreaks was 12 days. Outbreaks notified promptly (within 1 day) were of significantly shorter duration compared with outbreaks notified within 2–3 days ($P < 0.02$) or 4 or more days ($P < 0.001$). Early notification of outbreaks was not significantly associated with a reduced attack rate, however there was a significantly higher attack rate in facilities with less than 150 individuals at risk compared with facilities with 150 or more individuals at risk (30% versus 18%, respectively; $P < 0.001$). The shorter duration of promptly notified outbreaks provides some evidence to support recommendations from best practice guidelines for prompt notification of outbreaks by RCFs. However, further research is needed to unravel the interplay of factors that may influence the severity of viral gastroenteritis outbreaks in RCFs. *Commun Dis Intell* 2011;35(2):162–167.

Keywords: norovirus, viral gastroenteritis, residential care facilities, outbreak management, and notification of outbreaks

Introduction

Viral gastroenteritis affects all age groups, with particularly severe disease occurring in the elderly and people with chronic diseases.[1] Outbreaks of viral gastroenteritis are commonly reported from residential care facilities (RCFs)[2–4] where they may be long-lasting and may result in deaths.[1,5] A number of viruses may cause these outbreaks, including norovirus, rotaviruses, and adenoviruses,[6] although norovirus is by far the most common.[1,4,5]

Norovirus is most commonly associated with outbreaks because it requires only a small infective dose,[5,7–9] may be transmitted via multiple routes (i.e. person-to-person, food, water and environmental sources),[1,9,10] may become established well before the outbreak is identified,[8] immunity is usually short-lived,[9,11,12] is highly stable in the environment,[13,14] and is resistant to disinfectants.[9] Outbreaks in RCFs are also facilitated by factors such as decreased personal hygiene related to immobility, incontinence and dementia.[2,15] Host susceptibility is considered general although variable,[9] and transmission, particularly within outbreak settings, may be enhanced by extended periods of symptoms and viral shedding[1,9,14,16] as well as asymptomatic infection.[14,17] Aerosolisation of viral particles may occur during vomiting[18] and environmental surface contamination is a significant factor contributing to transmission in the enclosed living conditions of institutional facilities.[9,15,19]

Outbreaks cause considerable additional workload and logistical and economic burden for institutional facilities and public health authorities.[20–24] In residential care facilities ill residents require isolation and additional care, and in more serious cases may require hospitalisation. Common areas may be closed to residents and sometimes entire wings or facilities may be closed to visitors. Additional cleaning and infection control measures are required and there are staff productivity costs. For public health authorities, outbreaks trigger a range of investigations (laboratory, environmental and epidemiological), as well as additional surveillance and reporting requirements.[5,25]

A range of public health guidelines in Australia[26–28] advise on how to manage gastroenteritis outbreaks in RCFs, including the management of ill patients, staff and visitors, cleaning and disinfection, and monitoring and investigation. These guidelines encourage notification of outbreaks to public health, although there is little evidence that early identification and intervention are an effective use of public health resources.

There were two aims to this study. Firstly, to describe the epidemiology of notified viral gastroenteritis outbreaks in Queensland from 2004 to 2007 thought to be due to person-to-person transmission of norovirus; and secondly, to determine if prompt notification of outbreaks to public health authorities was associated with reduced severity of outbreaks.

Methods

Data source

The data used in this analysis were an extract of records from the Queensland OzFoodNet outbreak register, a state-wide register of reported foodborne and non-foodborne outbreaks. The data in the register were collected by public health units (PHUs) as part of routine surveillance of outbreaks notified by residential care facilities. Outbreak details were recorded by PHUs using a standard report template and forwarded to OzFoodNet Queensland for inclusion in their outbreak register, which forms part of the national OzFoodNet outbreak register. In this register, an outbreak includes 'two or more people with sudden onset of vomiting or diarrhoea (two or more episodes than is considered normal for the specific individual) within 24 hours'. Records were extracted from the register where transmission was recorded as person-to-person and where the onset date of the first case in the outbreak occurred between 1 January 2004 and 31 December 2007. Foodborne outbreaks were excluded, along with 6 outbreaks where viruses other than norovirus were identified as the causative agent (5 due to rotavirus and 1 due to adenovirus).

Data description

Fields extracted from the OzFoodNet outbreak register included the following: notification (report) date, date of onset of first case, date of onset of last case, total number ill (residents and staff combined), total number at risk of illness (residents and staff combined), number with faecal specimen collected, number laboratory confirmed, number hospitalised, number who died, type of epidemiological investigation, causative organism, and means of transmission. The identification of those who were ill, as well as those at risk of infection, was made by residential care facilities. The numbers reporting symptoms including diarrhoea, bloody diarrhoea, vomiting, nausea, abdominal pain, and fever was also collected. All specimens were tested for norovirus at the Public Health Virology Laboratory, Queensland Health Forensic and Scientific Services, Brisbane using reverse transcriptase polymerase chain reaction.[10]

Time to notification was calculated as the difference in the number of days between the notification date (report date) and the date of onset of symptoms of the first case and categorised as within 1 day, 2–3 days, and 4 or more days. These categories were chosen on the basis of clinical judgment and recommendations in guidelines for prompt notification within 24 hours.[26]

Facility size was calculated as the number of people (residents and staff combined) at risk of infection and was grouped as either less than 150 people or 150 people or more, given that median facility size was 153 people.

Outcome measures included attack rate and duration of outbreak. Both are continuous variables that were further categorised for analysis on the basis of clinical judgment and distribution of data. Attack rate was calculated as a percentage of the number of people who were ill, divided by those at risk of being ill, including both staff and residents of facilities. Attack rate was grouped for analysis as less than 15%, 15%–29% and 30% or more. Duration of outbreak was calculated as the number of days between the onset of the first case and the onset of the last case and categories used in the analysis were less than 9 days, 9–17 days, and 18 days or more.

Data analysis

The effect of the time taken to notify the outbreak and the size of the facility, on attack rates and duration of outbreaks were explored. As these variables were not normally distributed the Kruskal-Wallis test was used to test for statistical significance, with a two-sided *P*-value of less than 0.05 considered statistically significant. Median and inter-quartile ranges (IQR) are also presented. All analyses were carried out using Stata version 9.1.

Ethical approval

Ethical approval for this study was obtained from the Australian National University.

Results

Characteristics of notified outbreaks

A total of 264 outbreaks were notified between 2004 and 2007 (Table). The number of outbreaks in each calendar year ranged from 9 (in 2005) to 144 (in 2007). Peak months for notified outbreaks were June and July with the 6 months from April to September accounting for 74% (197) of all notified outbreaks (Figure).

Of 45,025 people at risk of infection, 9,020 (20%) ill cases were recorded. The median number of cases per outbreak was 28 (range 3–119) and the median number at risk of infection was 151 (range 17–1,026). Overall, there were 154 hospitalisations across

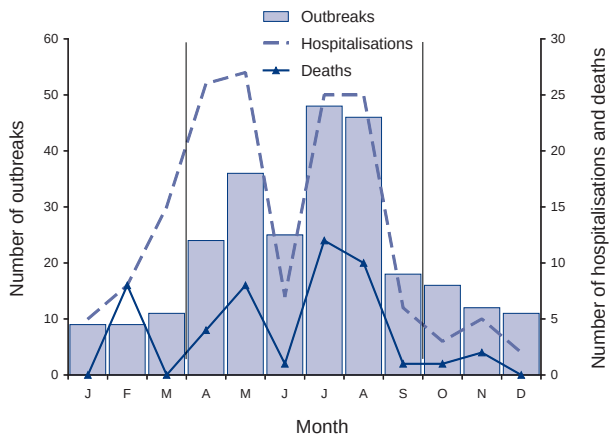
Table: Characteristics of notified viral gastroenteritis outbreaks in residential care facilities, Queensland, 2004 to 2007

Characteristics of notified outbreaks	2004	2005	2006	2007	2004–2007
Number of notified outbreaks	68	9	43	144	264
Total number who were ill	2,715	228	1,486	4,591	9,020
(range)	(3–104)	(7–78)	(5–102)	(3–119)	(3–119)
mean number per outbreak	40	25	35	32	34
median number per outbreak	35	16	29	25	28
Total number who were at-risk	11,528	1,517	6,615	24,680	44,340
(range)	(50–800)	(80–407)	(40–528)	(17–1,026)	(17–1,026)
mean number per outbreak	170	190	158	174	173
median number per outbreak	123	162	154	166	151
Attack rate	23.6%	15.0%	22.5%	18.6%	20.3%
Number of ill who were hospitalised	53	3	21	77	154
mean number per outbreak	0.9	0.4	0.6	0.6	0.6
% of ill who were hospitalised	2.0%	1.3%	1.4%	1.7%	1.7%
Number of cases who died	11	2	3	31	47
mean number per outbreak	0.2	0.3	0.1	0.2	0.2
% of ill who died	0.4%	0.9%	0.2%	0.7%	0.5%

85 (32%) outbreaks and 47 deaths across 33 (12%) outbreaks. The number of hospitalised cases and deaths peaked in June and July with approximately 75% of both occurring between April and September (Figure). Hospitalisation and death rates per 10,000 population were 34.4 and 10.2, respectively.

There were 1,335 faecal specimens collected for testing from 249 (94.3%) outbreaks. Half (50.5%, 674) of all faecal specimens collected tested positive to norovirus. Norovirus was confirmed (one or more stools testing positive) as the cause for 70.8% (187) of outbreaks.

Figure: Seasonality of notified viral gastroenteritis outbreaks in residential care facilities, by month of onset of first case, and associated hospitalisations and deaths, Queensland, 2004 to 2007



Outbreak duration and attack rate

The median number of days to notification of all outbreaks was 3.0 days (range 0–27 days). The median duration of all outbreaks was 11.0 days (range 0–36 days). The median duration of outbreaks notified within 1 day (7.5 days, IQR = 5–13 days) was significantly less ($P = 0.04$) than the median duration of outbreaks notified within 2–3 days (10 days, IQR = 6–14.5 days), and highly significantly less ($P < 0.001$) than the median duration of outbreaks notified after 4 or more days (14 days, IQR = 10–19 days). The median duration of outbreaks for facilities with less than 150 people at risk of infection was 11 days (IQR = 6–15 days) which was similar to that for facilities with 150 or more people at risk (11 days, IQR = 7–17.8 days).

The overall attack rate for all outbreaks included in this study was 20.8%. The median attack rate for outbreaks notified within 1 day (17.9%, IQR = 8.6%–26.5%) was not significantly different to that of outbreaks notified within 2–3 days (22.4%, IQR = 11.8%–36.8%) or notified after 4 or more days (20.7%, IQR = 10.2%–35.3%). The median attack rate for facilities with less than 150 people at risk of infection (24.9%, IQR = 16.8%–41.8%) was significantly higher ($P < 0.001$) than the attack rate for facilities with 150 or more people at risk (15.1%, IQR = 8.5%–26.1%).

Discussion

Our study shows that outbreaks of viral gastroenteritis, either due to or presumed due to norovirus, are

common in RCFs in Queensland and cause considerable burden to RCFs, PHUs and the community. From 2004 to 2007, there were 264 outbreaks notified involving more than 9,000 cases. One in 3 outbreaks involved at least 1 hospitalisation and one in 8 outbreaks involved at least 1 death. Such serious outcomes ensure that these outbreaks continue to attract media attention.

There is little published evidence that early identification of gastroenteritis outbreaks in RCFs and intervention are an effective use of public health resources. One recent study reported a significant reduction in attack rates in staff (but not residents) where the time to implementation of control measures was within 3 days of the onset of symptoms in the first case.[29] Infection control protocols were provided to facilities prior to study commencement, and there was no subsequent public health advice or support in managing the outbreaks. The study reported variable compliance with implementation of recommended control measures.

Our study found that prompt notification of outbreaks (within 1 day) was associated with significantly shorter outbreak duration (7.5 days) compared with outbreaks notified within 2–3 days and 4 or more days (10.0 days ($P < 0.02$) and 14.0 days ($P < 0.001$), respectively), suggesting that the advice provided by PHUs at notification of outbreaks may help to reduce the severity of outbreaks.

While there was also a lower attack rate in outbreaks notified promptly, this finding was not statistically significant. Our inability to demonstrate a significant association between these two parameters may have been due to a lack of study power due to the relatively small number of notified outbreaks over the study period, or confounding. Time to notification of outbreaks was assumed to be a proxy for time to implementation of control measures. However, control measures such as cohorting of ill residents, allocation of dedicated nursing staff, restricted access to common areas and infection control may be implemented prior to the notification of outbreaks.

While time to notification was not associated with attack rate, facility size was found to be inversely related to attack rate. There was a significantly higher attack rate in smaller facilities with less than 150 people at risk than in larger facilities. Smaller facilities may have fewer resources to identify and manage outbreaks, or reduced capacity to isolate the ill. However, facility size (the population at risk of infection) may be subject to measurement bias, particularly in large facilities and particularly in relation to staff numbers.

Two previous studies have reported an increased risk of disease outbreaks (number of outbreaks not severity) with increasing size of facility.[29,30] One of these studies included gastroenteritis outbreaks (mostly due to norovirus)[29] while the other included both respiratory and gastroenteritis outbreaks.[30] Neither of these studies examined the association between size of facility and severity of outbreak (attack rate and duration). Twenty-nine per cent of the outbreaks included in the study were presumed to be due to norovirus, in the absence of laboratory confirmation. As norovirus is by far the most common cause of viral gastroenteritis in RCFs,[1,4,5] it is likely that most outbreaks lacking laboratory confirmation were indeed due to norovirus. This assumption is supported by the finding that only 6 outbreaks required exclusion from the study due to laboratory confirmation of another viral pathogen, while 187 outbreaks were laboratory confirmed as norovirus.

Other factors that should be considered in interpreting our study findings include the potential for variation in PHU advice to RCFs and RCF skills at implementing control measures, and that some of the data reported by RCFs and PHUs may be subject to recall or measurement bias.

Despite these limitations, this study highlights that outbreaks of viral gastroenteritis are common in RCFs and place a considerable burden on residents, the facilities and on public health resources. The study was able to demonstrate an association between prompt notification to public health authorities and a shorter duration of outbreaks. This provides some evidence to support existing guidelines for management of outbreaks of viral gastroenteritis in RCFs which recommend prompt notification to public health authorities and early implementation of control measures.[4,27,28] With an ageing population in Australia and other western countries, it is increasingly important that we gain a better understanding of the risk of norovirus infection in RCF settings.[31] This study however only represents a preliminary exploration of what is a very complex issue. Further research is needed to unravel the interplay of factors including time to notification, timing and effectiveness of control measures, facility attributes including size, design, ease of movement within the facility, and staffing patterns, which may influence the severity of viral gastroenteritis outbreaks in RCFs.

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Feasibility of latent tuberculosis infection diagnosis by interferon-gamma release assay remote from testing facilities

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Abstract

Although the tuberculin skin test (TST) has been the mainstay of the diagnosis of latent tuberculosis infection (LTBI) for many decades, interferon-gamma release assays (IGRAs) are gaining acceptance and are more specific for this diagnosis. The characteristics of one such IGRA, the QuantiFERON®-TB Gold Whole Blood In-Tube, make it feasible for use in a remote setting. This study performed 62 IGRAs with this test on individuals testing positive by TST, in a clinical setting over 3,000 km from the testing laboratory. Of these, 42 patients (68%) recorded negative results, 19 (31%) were positive, with only 1 result (2%) indeterminate. Negative, and therefore discordant in this study, test results were more common in those known to have been previously vaccinated with bacille Calmette-Guérin. These results are consistent with other reports, indicating that this approach to testing is logistically feasible, and has the potential to complement LTBI screening to assist tuberculosis control programs in settings remote from the testing laboratory. *Commun Dis Intell* 2011;35(2):168–171.

Keywords: tuberculin test, tuberculosis, interferon-gamma release assay, rural health services

Introduction

Globally, around one third of the world's population, approximately 2 billion people, is thought to be infected with tuberculosis (TB).[1] Although tuberculosis rates are comparatively low in Australia, the Northern Territory has the highest notification rate of any jurisdiction at around 25 new cases per 100,000 population per year.[2] Tuberculin skin tests (TSTs) have been used for over a century in the diagnosis of latent tuberculosis infection (LTBI), as positive results are associated with increased risk of current or future active disease. However, there are several limitations of TSTs, including the inconvenience of returning to a health-care provider after 48–72 hours for the reading of the result and the subjectivity of this reading. Also, as TSTs use antigens also found in bacille Calmette-Guérin (BCG) and non-tuberculous mycobacteria, false positive results can occur.[3]

New interferon-gamma release assays (IGRAs) measure interferon-γ release from previously sensitised memory T-cells in response to antigens specific to *Mycobacterium tuberculosis,* but found in few non-tuberculous mycobacteria. Such tests, including QuantiFERON®-TB Gold Whole Blood In-Tube, have been found to have improved specificity for *M. tuberculosis* infection, with similar sensitivity to TSTs.[4] Despite this, availability of the test and adequate transfer of specimens must be considered before the test is adopted by a tuberculosis control program servicing a remote area. In particular, the need for incubation to be commenced within 16 hours of specimen collection and continued at 37ºC for a further 16 to 24 hours can be limiting in this setting. For reasons including these, the Northern Territory Centre for Disease Control Tuberculosis Unit continues to use TST as the mainstay of diagnosis of LTBI. This study aimed to determine whether this form of IGRA could be used to produce interpretable results in a location remote from the testing laboratory.

Methods

Subjects were prospectively enrolled from the Chest Clinic at the Centre for Disease Control (CDC), Darwin, with all adults eligible for inclusion if assessed for LTBI from August 2008 to December 2009. Patients were recruited when collection and transport could be co-ordinated with the local laboratory, located on the same campus as the CDC Chest Clinic. Study methods were approved by the Menzies School of Health Research Human Research Ethics Committee and subjects gave written, informed consent.

At the initial consultation, baseline demographic characteristics, indication for testing, results of chest x-ray, Indigenous status and BCG status were recorded. TST was then performed, followed by blood sampling for IGRA. TST was performed with intradermal injection of Tubersol® 0.1 mL (Sanofi Pasteur) and read after 48–72 hours, with results recorded as the transverse diameter of skin induration in millimetres. For the purpose of this study into immunocompetent adults, a positive result was defined as 10 mm or greater of induration regardless of BCG vaccination status.

Subjects recording a positive TST result proceeded to IGRA with the commercial kit QuantiFERON®-TB Gold Whole Blood In-Tube assay (Cellestis, Carnegie, Victoria). Approximately 5 mL of venous whole blood was obtained for testing, from which 1 mL of blood was instilled into each of three tubes (TB antigen, mitogen and nil control) and shaken for 5 seconds. On the same day (< 16 hours), the tubes were placed into incubation at 37°C for 16–24 hours at the Pathology Department of the Royal Darwin Hospital. The next day they were centrifuged at 3,000 revolutions per minute for 15 minutes. The specimens were then stored at 2°C to 8°C and sent refrigerated by air-freight to the Victorian Infectious Diseases Reference Laboratory, (North Melbourne Victoria, Australia) for enzyme-linked immunosorbent assay (ELISA) interferon-γ quantification according to the manufacturer's instructions. All individuals tested were seen again in the CDC Chest Clinic to receive counselling on the implications of their test result and options for treatment.

Multivariate logistic regression was performed for the binary outcome of a positive IGRA result, using the exposure variables of age, gender, BCG status, indication for testing, diameter of TST induration, presence of chest x-ray abnormalities and country of birth risk level for tuberculosis.

Results

A total of 62 subjects with positive TST results were tested with IGRA, with baseline characteristics displayed in Table 1. Patients were predominantly non-Indigenous, BCG vaccinated, young adults with an even gender distribution. Most were assessed in relation to employment, of which health care workers (22) and defence force personnel (16) were the largest groups. Other indications for assessment included external referral for assessment of tuberculosis risk (10) and follow up of a tuberculosis undertaking (5).[5] Chest x-rays were abnormal in 9 (15%) patients, with most changes being focal areas of minor scarring or granulomata. No patients were diagnosed with active tuberculosis. Of the 62 IGRAs obtained, 42 (68%) were negative, 19 (31%) positive and 1 (2%) indeterminate.

No exposure variables were independently associated with the outcome of a positive IGRA result on multivariate analysis ($P > 0.05$). Although not significant on multivariate analysis, a significantly lower proportion of patients with an established history of BCG vaccination was found to be IGRA positive than those without such a history ($P = 0.023$, $\chi 2$, Table 2). Although our guidelines state that 15 mm induration or greater is suggestive of true exposure, rather than BCG effect, this difference was not limited to those with less strongly positive TST results (10–14 mm induration), but was also seen in those with ≥ 15 mm induration.[7]

Table 1: Patient characteristics and interferon-gamma release assays results (n = 62)

Age, median (IQR)	36 (27–41)	
	n	%
Female gender	30	48
Indication for testing		
Health care worker	22	35
Other employment	20	32
Other indication	20	32
Country of birth*		
Australia	34	55
Other lower risk level country	6	10
Medium risk level country	0	
Higher risk level country	22	35
Indigenous status		
Indigenous	2	3
Non-Indigenous	60	97
Bacille Calmette-Guérin status		
Vaccinated	50	81
Unvaccinated	6	10
Uncertain	6	10
Diameter of tuberculin skin tests induration		
10 to 14 mm	23	37
15 to 19 mm	24	39
20 mm and greater	15	24
Chest x-ray abnormality present	9	15
Interferon-gamma release assays result		
Positive	19	31
Negative	42	68
Indeterminate	1	2

* Risk levels as defined by the Australian Government Department of Immigration and Citizenship.[6]

Discussion

Our results were broadly comparable to previous findings in use of IGRAs in less remote settings. In particular, this study reports a low proportion of indeterminate results suggesting that performance of the IFN-γ ELISA at a site remote from the testing laboratory is feasible. This finding is likely to relate to the short duration from venesection to incubation of generally less than 2 hours, as the reduction of this delay is associated with fewer indeterminate results.[8]

The lower proportion of positive IGRAs among BCG vaccinated individuals was also consistent with previous research.[9] However, the fact that patients with a history of BCG and negative IGRA were not primarily those with less strongly positive TSTs (10–14 mm) was unexpected. This finding may not be borne out with larger numbers, and as a reliable date

Table 2: Interferon-gamma release assays results by bacille Calmette-Guérin vaccination status and tuberculin skin tests size

Group	Interferon-gamma release assays result		
	n	Total	%
All tuberculin skin tests results			
BCG vaccinated	12	49	24
BCG unvaccinated*	7	12	58
$\chi^2 = 5.1$, $P = 0.023$			
Tuberculin skin tests 10–14 mm			
BCG vaccinated	3	16	19
BCG unvaccinated*	3	7	43
$\chi^2 = 1.5$, $P = 0.226$			
Tuberculin skin tests ≥15 mm			
BCG vaccinated	9	33	27
BCG unvaccinated*	4	5	80
$\chi^2 = 5.4$, $P = 0.021$			

Indeterminate result excluded from analysis.

* Includes bacille Calmette-Guérin (BCG) status uncertain.

for BCG vaccination could not be established for all subjects, the effect of timing of vaccination could not be assessed. This would be important given the high proportion of health care workers, particularly those on short-term placements, who may have received recent vaccination. However, the number of subjects enrolled limited our ability to study factors associated with increased rates of IGRA positivity.

IGRA tests, in particular QuantiFERON, have been shown to be feasible when used in hospital settings, and in clinics located in close proximity to laboratory testing facilities where processing is performed on the day of phlebotomy.[10] This observation has been expanded to groups including homeless, immigrant, refugee and intravenous drug-users and has been extended to the newer QuantiFERON®-TB Gold test.[11,12]

In the United States of America, a country where BCG has not been used as a recommended tuberculosis control strategy, the Centers for Disease Control and Prevention recommends use of IGRA in preference to TST in all BCG vaccinated individuals. These recommendations include programmatic considerations, in preferring IGRAs to TSTs when targeting groups considered less likely to return after the appropriate interval for TST reading. When delivering a tuberculosis control program over a wide geographical region, remote from testing facilities, guidelines also recommend consideration of transportation of specimens.[13] British guidelines recommend TST as the initial investigation for the diagnosis of LTBI, but confirmation of all positive results with an IGRA is recommended where available.[14] In Australia, the recommend initial investigation for LTBI remains the TST, but jurisdictions are encouraged to undertake further research to better define the role of IGRAs.[15]

The processing of samples for this study was consistent with the manufacturer's recommendation that tubes be incubated within 16 hours of collection and will then be stable for transport for up to 3 days.[16] Reports exist of effective IGRA testing in remote areas, although testing is most commonly performed locally in clinical research.[17] Ravn et al report satisfactory results in a remote Ethiopian setting in which specimens were transported by road for 6–8 hours then resting overnight before separation and testing for response to ESAT-6.[18] A report of a multicentre school-based screening program in Norway using QuantiFERON®-TB Gold In-Tube included rural areas; processing and storing specimens locally, before transport to a central testing facility.[19]

This study documents effective IGRA performance at a considerable distance from final testing, with results available within a reasonable time-frame to allow for effective decision-making. The finding of satisfactory results following local centrifugation and incubation, suggests IGRAs may be a feasible test to complement the Northern Territory tuberculosis control program. The use of IGRA in a low risk group with high rates of BCG vaccination would lead to fewer diagnoses of LTBI and a consequent reduction in the number of patients treated for this condition. However, for the test to be used successfully in remote communities, specimens would still require transportation to Darwin for centrifugation and incubation as early as possible within 16 hours of collection. This would present further logistical issues, the feasibility of which has not been tested. Using IGRAs in urban Darwin, but the TST in remote communities could also be considered but may present programmatic difficulties in training and use of two different diagnostic approaches.

The low rate of indeterminate results is not a consistent finding across all studies,[20] and may not be reliably replicated outside the research setting if familiarity with local processing is not maintained. As this study did not limit the interval between TST and IGRAs, some IGRA results may have been affected by a boosting effect.[21]

In conclusion, IGRA performance in urban Darwin is feasible and provides interpretable results with a low frequency of indeterminate results obtained. Further study will be required before IGRAs can be used in remote communities, away from basic laboratory facilities. However, use of these tests in urban areas, to complement LTBI screening in the Northern Territory tuberculosis control program, may be considered in future guidelines.

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Chronic disease and hospitalisation for pandemic (H1N1) 2009 influenza in Indigenous and non-Indigenous Western Australians

Leigh S Goggin, Dale Carcione, Donna B Mak, Gary K Dowse, Carolien M Giele, David W Smith, Paul V Effler

Abstract

Indigenous and non-Indigenous Western Australians with pandemic (H1N1) 2009 influenza (pH1N1) infection were compared for risk factors, influenza vaccination history, symptoms, use of antiviral medications, and hospitalisation. Data were collected systematically on 856 notified cases with laboratory confirmed pH1N1 infection during the first 10 weeks of pH1N1 virus transmission in Western Australia in 2009. Indigenous people with pH1N1 were approximately 3 times more likely to be hospitalised and were more likely to have a range of underlying medical conditions and be smokers, compared with non-Indigenous cases. Age ($P < 0.001$) and the presence of two or more co-morbidities ($P < 0.001$) were independent predictors of hospitalisation, while Indigenous status was not, indicating that higher pH1N1 hospitalisation rates in Indigenous Australians during the 2009 winter season were attributable to the higher prevalence of underlying chronic disease. These results underscore the need to ensure that influenza vaccination is delivered as widely as possible among those with chronic health conditions. *Commun Dis Intell* 2011;35(2):172–176.

Keywords: pandemic (H1N1) 2009 influenza, chronic disease, hospitalisation, Indigenous, Western Australia

Introduction

It has long been recognised that Indigenous populations are at an increased risk of suffering adverse consequences and complications associated with influenza infection.[1,2] Indigenous populations of Australia, New Zealand, Canada, Brazil, and the Pacific Islands were shown to be particularly vulnerable to pandemic influenza A (H1N1) 2009 (pH1N1) infection, with disproportionately higher rates of hospitalisation and mortality.[3] Although Indigenous populations comprise less than 5% of the total population in Canada and the United States of America, they represented almost 18% of all hospitalisations due to pH1N1 infection.[3] Similarly, in comparison to non-Indigenous populations, the rate of severe acute respiratory illness in Brazilian Amerindians infected with pH1N1 was 4.5 times higher, the hospitalisation rate in New Zealand Maoris was 3 times higher, and the hospitalisation and mortality rate among Australian Aborigines were 7.7 and 5.1 times higher respectively.[3] The Indigenous population in the top end of Australia's Northern Territory were 12 times more likely to be hospitalised and 5 times more likely to be admitted to intensive care.[4] Twenty per cent of 4,808 hospitalisations attributed to pH1N1 during the 2009 winter in Australia were in Indigenous people, equating to an 8-fold higher hospitalisation rate compared with the non-Indigenous population.[5]

From May to September 2009, the notification rate of pH1N1 to the Communicable Disease Control Directorate (CDCD) in the Department of Health in Western Australia was 4 times higher among Indigenous people compared with non-Indigenous people.[6] After notification of the 1st case of pH1N1 in Western Australia in May 2009, the CDCD commenced an investigation of the impact of pH1N1 compared with co-circulating seasonal influenza. This provided an opportunity to compare Indigenous and non-Indigenous cases in terms of demographics, co-morbidities, influenza vaccination history, symptoms, use of antiviral medications and outcomes, and to determine if Indigenous status alone conferred an increased risk of hospitalisation for pH1N1, once other factors had been considered.

Methods

Influenza is a notifiable disease in Western Australia and all cases detected by pathology laboratories are reported to the CDCD. Pandemic H1N1 cases were recruited during the 10 week period from 29 May 2009 (4 days after notification of the 1st confirmed pH1N1 infection in Western Australia) to 7 August 2009, either as consecutive notifications or randomly, as described previously.[7] A case was defined as anyone notified to CDCD with laboratory confirmed pH1N1 diagnosed by nucleic acid testing during the study period. Cases were excluded if they had a co-infection with another influenza virus.

Demographic information for notified influenza cases was obtained from the Western Australian Notifiable Infectious Diseases Database. A ques-

tionnaire was administered via telephone by trained public health nurses to each selected case within 48 hours of receipt of the notification at CDCD. The case was briefed about the investigation and verbal consent was obtained to participate. A total of 6 attempts were made to contact the case after which they were deemed not contactable. The questionnaire gathered information on signs and symptoms of illness, hospitalisation, use of antiviral medications and underlying medical conditions. If a case was unable to answer the questionnaire, an adult household member most familiar with the case was interviewed as a proxy. Hospitalisation status was ascertained at the time of interview and by retrospectively checking all notified cases against a hospital discharge database encompassing all public hospitals and one large private hospital in Western Australia.[7] A case was defined as being hospitalised if they were admitted for one or more nights for illness caused or exacerbated by pH1N1 infection. Indigenous cases were those who identified themselves at time of interview to be of Aboriginal and/or Torres Strait Islander descent. Cases were excluded if Indigenous status was unknown or missing.

Human research ethics committee approval was not required as information was collected as part of the public health response to pH1N1. Statistical analyses were performed using PASW Version 17.0.2 (SPSS Inc., Chicago, IL). Differences in univariate data were assessed by χ^2 tests for proportions and t-tests for continuous variables. Univariate odds ratios (OR) and 95% confidence intervals (95% CI) for factors associated with hospitalisation were calculated using logistic regression. P values < 0.05 were considered statistically significant. Factors significantly associated with hospitalisation in univariate analyses were included in a multivariate logistic regression model with backwards stepwise elimination of variables.

Results

Over the 10-week study period, 984 pH1N1 cases that fulfilled the case definition were selected for inclusion. Of these, 871 cases (88.5%) were interviewed, 107 (10.9%) were not contactable, and 6 (0.6%) refused to participate. Hence, 871 pH1N1 cases were interviewed, but Indigenous status was not determined for 15 (1.7%), leaving 856 pH1N1 cases, of whom 63 (7.4%) were Indigenous. The proportion of Indigenous cases was more than twice as high as in the general Western Australia population (3.3%) (personal communication, Epidemiology Branch, Department of Health, Western Australia; 2009).

Characteristics of Indigenous and non-Indigenous cases are compared in Table 1. There was no difference in age or gender distribution, but Indigenous cases were more likely to be treated with antiviral medications (73% vs 41%, $P < 0.01$) and to be hospitalised (27% vs 10%, $P < 0.01$). There was no difference in the overall frequency of having 'any underlying medical condition or risk factor', but Indigenous cases were significantly more likely to be smokers or to have diabetes, heart disease, or renal disease, compared with non-Indigenous cases. Symptomatology was broadly similar between the two groups, although Indigenous cases were more inclined to report respiratory symptoms, while non-Indigenous cases reported significantly higher frequencies of non-respiratory symptoms including myalgia, headache, diarrhoea and vomiting.

As shown in Table 2, the age difference between hospitalised and non-hospitalised Indigenous cases was nearly 18 years. Among Indigenous cases, those hospitalised had higher frequencies of each individual underlying medical condition, smoking and pregnancy, compared with those not hospitalised. Sixty-three per cent of hospitalised cases reported two or more medical conditions or risk factors, compared with only 7% of those not hospitalised ($P < 0.01$) (Table 2).

Univariate logistic regression analysis for the total study population showed that being Indigenous conferred a significantly higher risk of hospitalisation resulting from pH1N1 infection (OR = 3.2, 95% CI 1.7–5.8), as did age (OR = 1.03, 1.02–1.04) (where 1.03 is the increase in odds per year of age); having any pre-existing medical condition or risk factor (OR = 5.9, 3.5–10.1); diabetes (OR = 4.1, 2.1–7.9); heart disease (OR = 6.4, 3.0–13.7); respiratory disease (OR = 2.3, 1.4–3.5); obesity (OR = 3.2, 1.8–5.8); and two or more medical conditions or risk factors (OR = 7.6, 4.7–12.1).

When the above factors were entered into a stepwise multivariate logistic regression model for all cases, only 2 factors were independent predictors of hospitalisation: having two or more medical conditions or risk factors (OR = 4.9, 95% CI = 2.9–8.2, $P < 0.001$) and age (OR = 1.02, 1.01–1.03, $P = 0.006$).

Discussion

This study has found that Indigenous West Australians with pH1N1 were 3.2 times more likely to be hospitalised than their non-Indigenous peers, which is similar to the rate ratio of hospitalisation reported by Flint et al[4] for the Northern Territory (3.4), and to that reported for the Maori population in New Zealand (3.0).[3] Not surprisingly, compared with non-Indigenous pH1N1 cases, Indigenous people with pH1N1 infection reported a higher prevalence of several underlying medical conditions known to be a risk for worse outcomes from influenza, including diabetes, heart disease,

Table 1: Characteristics of Indigenous and non-Indigenous pandemic (H1N1) 2009 influenza cases, Western Australia, 2009

	Indigenous	Non-Indigenous	*P* value*
Number	**63**	**793**	
Male	35/63 (56%)	397/793 (50%)	NS
Mean age, years (range)	26.0 (1–78)	26.0 (0–85)	NS
Antivirals administered for treatment	46/63 (73%)	325/792 (41%)	<0.01
Vaccinated in 2009 for seasonal influenza	16/50 (32%)	162/673 (24%)	NS
Underlying medical conditions and risk factors			
Any existing medical condition (includes pregnancy and smoking)	32/58 (55%)	323/680 (48%)	NS
Diabetes	10/58 (17%)	35/670 (5%)	<0.01
Heart disease	6/57 (11%)	24/667 (4%)	0.01
Respiratory condition	13/57 (23%)	158/671 (24%)	NS
Renal disease	3/57 (5%)	8/667 (1%)	0.02
Obesity	6/57 (11%)	54/671 (8%)	NS
Pregnancy	2/60 (3%)	33/758 (4%)	NS
Smoker	13/58 (22%)	80/670 (12%)	0.02
Other medical conditions†	7/57 (12%)	22/668 (3%)	<0.01
Multiple medical conditions or risk factors‡	13/58 (22%)	95/680 (14%)	NS
Symptoms			
Influenza-like illness	55/63 (87%)	640/793 (81%)	NS
Cough	61/63 (97%)	674/793 (85%)	<0.01
Pyrexia	42/63 (67%)	440/793 (56%)	NS
Sore throat	39/63 (62%)	442/793 (56%)	NS
Dyspnoea	21/63 (33%)	260/793 (33%)	NS
Coryza	31/63 (49%)	461/793 (58%)	NS
Fatigue	41/63 (65%)	588/793 (74%)	NS
Myalgia	31/63 (49%)	523/793 (66%)	<0.01
Rigors	30/63 (48%)	433/793 (55%)	NS
Headache	31/63 (49%)	503/793 (63%)	0.02
Diarrhoea	5/63 (8%)	155/793 (20%)	0.02
Vomiting	12/63 (19%)	268/793 (34%)	0.02
Outcomes			
Hospitalisation	17/63 (27%)	83/793 (10%)	<0.01
Median number of days hospitalised (range)	4 (2–7)	4 (2–69)	NS

* Pearson χ^2 test; t-test for age and number of days hospitalised.

† Other conditions include neurological diseases, blood disorders, metabolic disorders, and immune system disorders.

‡ Multiple conditions includes two or more of the above listed conditions (includes smoking).

NS Not significant.

Numbers and percentages shown, unless otherwise stated.

and renal disease, and also had a higher prevalence of cigarette smoking. These findings reflect a large body of evidence documenting a greater chronic disease burden in the Indigenous population of Australia.[8,9]

Indigenous persons requiring hospitalisation for pH1N1 infection were on average 18 years older than non-hospitalised cases, and had higher frequencies of most individual chronic medical conditions and risk factors that were examined, including obesity, pregnancy and smoking. Notably, 63% of Indigenous hospitalised cases reported more than one underlying medical condition or risk factor for adverse influenza outcome, as opposed to only 7% of non-hospitalised cases.

Significantly, only age and the presence of two or more medical conditions or risk factors were shown to be independent predictors of hospitalisation

Table 2: Characteristics of Indigenous cases, by hospitalisation status

	Non-hospitalised	Hospitalised	*P* value*
Number	46	17	
Male	27/46 (59%)	8/17 (47%)	NS
Mean age, years (range)	21.2 (1–73)	39.0 (2–78)	0.01
Antivirals administered for treatment	31/46 (67%)	15/17 (88%)	NS
Vaccinated in 2009 for seasonal influenza	13/38 (34%)	3/12 (25%)	NS
Any existing medical condition (includes pregnancy and smoking)	19/42 (45%)	13/16 (81%)	0.01
Pregnancy	1/44 (2%)	1/16 (6%)	NS
Diabetes	3/42 (7%)	7/16 (44%)	<0.01
Heart disease	2/41 (5%)	4/16 (25%)	0.03
Respiratory condition	6/41 (15%)	7/16 (44%)	0.02
Renal disease	1/41 (2%)	2/16 (12%)	NS
Obesity	2/41 (5%)	4/16 (25%)	0.03
Smoker	7/42 (17%)	6/16 (37%)	NS
Other medical conditions†	5/41 (12%)	2/16 (12%)	NS
Multiple medical conditions or risk factors‡	3/42 (7%)	10/16 (63%)	<0.01

* Pearson χ^2 test; t-test for age.

† Other conditions include neurological diseases, blood disorders, metabolic disorders, and immune system disorders.

‡ Multiple conditions includes two or more of the above listed conditions (includes smoking).

NS Not significant.

Numbers and percentages shown, unless otherwise stated

for pH1N1 infection, with Indigenous status not contributing further. This suggests that Indigenous Australians suffered higher rates of pH1N1 hospitalisation only by virtue of their higher prevalence of risk factors for severe disease, rather than due to any innate genetic predisposition or vulnerability.[10]

The finding that Indigenous pH1N1 cases were more likely to have received treatment with antiviral medication is almost certainly due to the prioritisation of antiviral medication to those in the community with influenza-like illness or confirmed pH1N1 infection who were recognised to be at increased risk of severe illness, which included people with underlying medical conditions and Indigenous Australians, and those requiring hospitalisation.[11,12] Reflecting the latter recommendation, 88% of hospitalised Indigenous cases received antiviral treatment.

The differences in the reported symptomatology of pH1N1 infection are interesting, with Indigenous cases more likely to report respiratory symptoms and non-Indigenous cases reporting systemic symptoms more frequently. This may be due to a higher rate of respiratory complications in Indigenous cases, contributing to the higher rate of hospitalisation observed. However, the possibility that the difference reflects cultural differences in reporting cannot be discounted.

There are several potential limitations to this investigation. Previous analyses have demonstrated that the interviewed and non-interviewed pH1N1 cases were very similar, excepting that a higher proportion of hospitalised cases were not interviewed.[7] It is possible that non-interviewed hospitalised cases may have been more unwell, and have had higher prevalence of risk factors for severe disease, but were this so, it is likely that inclusion of such cases would only strengthen the reported findings. Indigenous pH1N1 cases were also relatively under-represented in the study group compared with all notified cases but this is unlikely to have introduced significant bias to these comparisons.

In addition, as this was an investigation of pH1N1 cases detected as a result of healthcare attendance in the community, there was no control over which cases presented and were tested for pH1N1. It is recognised that a significant proportion of pH1N1 cases had asymptomatic or mild infection,[13] so cases identified by health-care attendance and testing are likely to have a higher prevalence of underlying medical conditions and adverse outcomes than the true population infected with the virus. The underlying medical conditions and clinical manifestation data analysed in this investigation were also self-reported, and as such, may be subject to some inaccuracy.

In summary, Indigenous Western Australians with pH1N1 infection were more likely to be hospitalised and had a significantly higher prevalence of underlying medical conditions and risk factors for adverse outcomes of influenza infection, compared with non-Indigenous cases. However, after accounting for age and the presence of two or more co-morbidities and risk factors, Indigenous status did not confer additional risk for hospitalisation for pH1N1 infection. This study highlights the need to ensure that preventative measures, especially influenza vaccination, are delivered as widely as possible among those with chronic medical conditions and risk factors for adverse outcomes of influenza infection.[14]

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Respiratory syncytial virus – the unrecognised cause of health and economic burden among young children in Australia

Geetha Ranmuthugala, Laurie Brown, Brett A Lidbury

Abstract

Respiratory syncytial virus (RSV) presents very similar to influenza and is the principle cause of bronchiolitis in infants and young children worldwide. Yet, there is no systematic monitoring of RSV activity in Australia. This study uses existing published data sources to estimate incidence, hospitalisation rates, and associated costs of RSV among young children in Australia. Published reports from the Laboratory Virology and Serology Reporting Scheme, a passive voluntary surveillance system, and the National Hospital Morbidity Dataset were used to estimate RSV-related age-specific hospitalisation rates in New South Wales and Australia. These estimates and national USA estimates of RSV-related hospitalisation rates were applied to Australian population data to estimate RSV incidence in Australia. Direct economic burden was estimated by applying cost estimates used to derive economic cost associated with the influenza virus. The estimated RSV-related hospitalisation rates ranged from 2.2–4.5 per 1,000 among children less than 5 years of age to 8.7–17.4 per 1,000 among infants. Incidence ranged from 110.0–226.5 per 1,000 among the under five age group to 435.0–869.0 per 1,000 among infants. The total annual direct healthcare cost was estimated to be between $24 million and $50 million. Comparison with the health burdens attributed to the influenza virus and rotavirus suggests that the disease burden caused by RSV is potentially much higher. The limitations associated with using a passive surveillance system to estimate disease burden, and the need to explore further assessments and to monitor RSV activity are discussed. *Commun Dis Intell* 2011;35(2):177–184.

Keywords: respiratory syncytial virus, respiratory tract infections, burden of disease, Australia

Introduction

The Influenza A H1N1 pandemic of 2009 served as a reminder of the importance of monitoring in the detection and management of infectious disease outbreaks. In Australia, laboratory confirmed influenza is by law, a notifiable disease that needs to be reported to the National Notifiable Diseases Surveillance System and is thereby reported routinely. There is however little recognition of the role that the respiratory syncytial virus (RSV) plays in the burden of disease that is attributed to the influenza virus the world over.

With a disease spectrum ranging from rhinitis and otitis media to bronchiolitis and pneumonia, RSV infection presents very similar to influenza and contributes to the influenza-like illness that occurs in the community.[1–2] It is the principal cause of bronchiolitis in infants and young children worldwide, with incidence peaking between 2 and 5 months of age and almost all children being exposed to RSV by the time they reach 3 years of age.[3]

Overseas studies suggest a high disease burden in industrial nations. In the United Kingdom, it is estimated that among the under 5 years age group, general practitioner consultation rates due to RSV are similar to that of influenza, while RSV accounts for more hospital admissions than does influenza (35,540 and 9,967 respectively), particularly among pre-school aged children.[4] In the year 2000 in the United States of America (USA), RSV-related hospital admissions accounted for 17% of all hospitalisation of infants aged 3–12 months.[5] In Japan, it is estimated that a quarter (26.2%) of children aged less than 3 years of age are treated annually for RSV and 13.6% are hospitalised.[6] The medical cost associated with RSV in children less than 5 years of age in the USA in 2000 was estimated to be $US652 million; 60% of this cost attributed to hospitalisations.[5]

In Australia, while a small number of studies have examined viral cultures taken in hospital to help create a picture of RSV burden, there is no systematic collection of data on RSV to estimate the health and economic burden specific to Australia. This study draws on published data to derive an estimate of RSV occurrence in Australia and compares the disease burden with that caused by other highly prevalent viruses.

Methods

RSV is not a notifiable disease in Australia and there is no single data source that accurately measures RSV activity across Australia. The primary data source for this study is the Laboratory Virology and Serology Reporting Scheme (LabVISE), a passive surveillance system involving a network of 17–20 laboratories across Australia voluntarily sub-

mitting monthly reports on the laboratory identification of viruses and other organisms.[7] Specifically, two studies that have examined RSV reporting in LabVISE have been used to estimate the number of cases of RSV-related hospitalisations and hospitalisation rates that occur in Australia.[8–9]

The first of the two LabVISE based studies was published in 2000 and presents the number of RSV cases in New South Wales reported to LabVISE during the period January 1993 and December 1997.[8] The authors, in presenting the number of RSV cases reported to LabVISE, identify that these numbers are 'likely to represent hospitalised cases (and that the data) reflect a pattern of hospitalisation rather than RSV infection in the community.'[8] Given the absence of more comprehensive data sources, and recognising the limitations of using a passive surveillance system to estimate the occurrence of disease, the number reported to LabVISE is used in this study as a surrogate indicator of the number of RSV hospitalisations in New South Wales.

The second study published in 2002 reviewed Australia-wide reporting of RSV as part of viral and non-viral pathogen identifications reported to LabVISE during the 10 year period 1991–2000.[9] As with the RSV reporting for New South Wales, the number of RSV cases reported across Australia is used in this study as a surrogate indicator for RSV hospitalisations in Australia. This study does not provide an age breakdown of RSV occurrence in Australia. Being uncertain about how the New South Wales age distribution of RSV compares with that of Australia, and in view of the fact that the age distribution of the Australian and the USA populations and the New South Wales and the Australian populations are similar,[10] the age distribution of RSV from a national estimate for the USA[5] was applied to the number of RSV cases reported in Australia to estimate RSV hospitalisation rates in Australia (Table 1).

The number of RSV hospitalised cases obtained from the two studies (Table 1) were then applied to the New South Wales and Australian population distributions of 1996 and 2000 to derive RSV hospitalisation rates for New South Wales and Australia (Table 1).

A second data source used in this study to inform the number of RSV-related hospitalisations in Australia is the National Hospital Morbidity Database (NHMD) as reported by the Australian Institute of Health and Welfare (AIHW).[11] The number of hospital separations registered from all states and territories in Australia in the NHMD with a discharge diagnosis of ICD-10-AM codes B97.4 (RSV), J12.1 (RSV pneumonia), J20.5 (acute RSV bronchitis), or J21.0 (acute RSV bronchiolitis) for the year 1999–2000 was obtained via the data cubes published on the AIHW website.[12] Table 2 presents and compares the NHMD derived estimates of hospitalisation rates with those presented in Table 1 and with national USA estimates.

To estimate the incidence of RSV in Australia, the proportion of RSV cases expected to be hospitalised was applied to the hospitalisation rates presented in Table 2. Given the absence of a definite estimate of the proportion of RSV cases that require hospitalisation, the upper end of the published statistics that 0.5%– 2% of all cases with RSV in the infant age group require hospitalisation[3] was used as the proportion. The upper end was used as this would provide a lower estimate of incidence, thereby reducing the likelihood of over-estimating RSV occurrence. Since RSV hospitalisation rates derived from NHMD were higher than the LabVISE estimates (Table 2) and given the fact that NHMD is an actual count of the number of RSV-related hospital separations as opposed to an estimation based on a passive surveillance system, the NHMD derived hospitalisation rates have been used separately to estimate RSV incidence in Australia. The LabVISE estimated hospitalisation rates and the USA national estimate have been combined as a range.

The estimated incidence and hospitalisation rates are used to calculate an economic burden of RSV in Australia. In the absence of data to inform the economic costing, the cost estimation is limited to the direct health care costs associated with hospitalisation. The average cost of a RSV-related hospital admission was taken to be $5,245, which is the Australian Refined Diagnosis Related Groups based average cost of a hospital admission for influenza or pneumonia over the period 1998–2005 (in 2005 dollar value) used to estimate the economic cost of influenza to the Australian health system.[13] Applying the value used to estimate the cost of influenza is justifiable on the basis that influenza and RSV are very similar in terms of clinical presentation and have comparable lengths of stay in hospital (median of 2–4 days for RSV[5,14] and 2 days for influenza[15]). In estimating the total direct healthcare costs associated with RSV infections, it is assumed, based on existing literature,[5] that 60% of direct costs are incurred in hospital.

To place these estimates in perspective, the RSV-related burden of disease estimated in this study was compared against published hospitalisation rates for influenza in Sydney, New South Wales, for the period 1994–2001.[16] Given that the influenza vaccine was introduced in Australia as part of the mass immunisation program in 1999, the period 1994–2001 mostly represents pre-vaccination period for influenza virus and is therefore comparable to the current RSV situation of no vaccination. Also used as a comparator was the number of reports of influenza to LabVISE from 1991–2000.[9] Once again,

Table 1: Estimated respiratory syncytial virus-related hospitalisation rates for New South Wales and Australia, based on reporting to LabVISE

Study	Study years	Study population	Age group (per 1,000 population)	Cases per year	Population		Hospitalisation rate*
Lister[8]	1993–1997	NSW	All ages	770–1,131	6,038,696	(1996 Census)	0.1–0.2 (all ages)
			<3 months	276† (29% of all cases)‡	85,302	(live births, 1996)‖	3.2 per 1,000 live births
			<6 months	504† (53% of all cases)‡	85,302	(live births, 1996)‖	5.9 per 1,000 live births
			<1 year	742† (78% of all cases)‡	85,302	(live births, 1996)‖	8.7 per 1,000 live births
			<5 years	937† (98.5% of all cases)‡	427,696	(1996 Census)	2.2 per 1,000 pop <5 years
Roche[9]	1991–2000	Australia	All ages	2,555 to 4,641	17,892,423	(1996 Census)	0.1–0.3 (all ages)
			<3 months	1,259† (35% of all cases)§	255,445	(live births, 2000)¶	4.9 per 1,000 live births
			<6 months	1,979† (55% of all cases)§	255,445	(live births, 2000)¶	7.7 per 1,000 live births
			<1 year	2,699† (75% of all cases)§	255,445	(live births, 2000)¶	10.6 per 1,000 live births
			<5 years	3,598† (98% of all cases)§	1,273,589	(2000 projections)	2.8 per 1,000 pop <5 years

* Estimating hospitalisation rate is based on using the number of cases reported to the Laboratory Virology and Serology Reporting Scheme (LabVISE) as a surrogate indicator of the number of respiratory syncytial virus hospitalisations.

† Based on an average of the annual number of cases reported in the paper, assuming a normal distribution in the number of cases reported every year. The average for the Lister study was 951 cases per year (for New South Wales)[8] and the Roche study was 3,598 cases per year (for Australia).[9]

‡ Proportion of cases by age group as reported in Lister et al, 2000.[8]

§ Proportions of cases reported in Paramore et al. 2004[5]

‖ NSW Department of Health. *NSW Mothers and Babies 1996. N S W Public Health Bull* 1998;Suppl. (State Health Publication No (ESB) 970146).

¶ Australian Institute of Health and Welfare. Australia's mothers and babies. Canberra: Australian Institute of Health and Welfare; 2000.

this largely constitutes the pre-immunisation period for influenza and is therefore a suitable comparison.

Just as RSV is now recognised to be a major contributor to lower respiratory tract infections in infants and young children, rotavirus is the major cause of gastroenteritis among infants and young children in Australia. In common with RSV, rotavirus transmission in temperate climates is seasonal, occurring predominantly during the winter season. Estimated RSV-related hospitalisation rates are compared with published reports on rotavirus-related hospitalisation rates and disease burden for the period 1991–2002.[11,17] Rotavirus immunisation was not added to the Australian National Immunisation Program until 2007.

Results

In New South Wales a total of 4,665 cases of RSV was reported to LabVISE between January 1993 and December 1997, with the number of cases per year ranging from 770–1,131.[8] Across Australia, RSV was the single most common virus reported to LabVISE during the 10 year period 1991–2000 with a total of 36,346 cases reported, and the annual number of cases ranging from 2,555 to 4,641.[9] Consistent with published literature and similar to influenza,[18–19] annual epidemics occurred in winter and the majority of cases of RSV reporting was for children less than 5 years of age.[9]

The hospitalisation rates estimated based on the number of cases reported to LabVISE, suggest that while the rates for New South Wales were marginally lower than rates based on the 10-year review of Australia-wide reporting of RSV, the estimates for New South Wales and Australia were not dissimilar (Table 1). The difference, if at all, was more among the young infant age group.

Table 2 compares the estimates from the two LabVISE studies with hospitalisation rates derived from the NHMD and the national USA estimates. The NHMD rates are higher than rates estimated from the LabVISE-based studies and are more in keeping with the USA national estimate reported by Paramore et al[5] (Table 2). As a result, the NHMD derived hospitalisation rates were used separately for estimating the RSV incidence for Australia.

Applying the hospitalisation rates derived in Table 2 to the 2006 Australian Census population estimates that up to 5,710 RSV-related hospital episodes may occur every year among children less than 5 years of age in Australia (Table 3). Assuming the upper limit from the published literature that up to 2% of RSV cases are hospitalised,[3] this converts to up to 285,481 cases of RSV occurring in Australia among children aged less than 5 years; with the majority of these cases occurring among the less than 1 year age group (Table 3). There wasn't sufficient data to examine in detail the distribution within the less than 1 year age group as would have been ideal given that the literature suggest a high disease burden observed in the less than 3 month age group.

Applying the average cost of $5,245 per hospital admission, the 2,773 to 5,710 hospital episodes estimated per year for the < 5 year age group (Table 3) will cost the Australian health system between $14.544 m and $29.949 m per year in hospital costs (Table 4). If, as suggested by USA and Canadian literature, hospital costs account for around 60% of the total direct health care costs of RSV infections in young children, the cost of RSV on the Australian health system is estimated to be between $24 and $50 million annually (in 2005 value). No information is available to estimate non-health care or indirect costs attributable to RSV.

Table 2: Age-specific respiratory syncytial virus (RSV) related-hospitalisation rates per 1,000 population for Australia and the United States of America (USA) as estimated by various sources

	LabVISE* (1993–1997) NSW	LabVISE (1991–2000) Australia	NHMD† (1999/2000) Australia	Paramore (2001) USA (national estimate)
RSV hospitalisations (n)	770–1,131 per year‡	2,555–4,640 per year§	5,244	87,105 annually
Hospitalisation rate by age group (per 1,000 population)				
All ages	0.1–0.2‖	0.1–0.3‖	–	–
< 1 year	8.7‖	10.6‖	16.8	17.4
< 5 years	2.2‖	2.8‖	3.9	4.5

* Laboratory Virology and Serology Reporting Scheme.

† National Hospital Morbidity Dataset.

‡ Number of cases in New South Wales as reported in Lister et al, 2000.[8]

§ Number of cases across Australia as reported in Roche et al, 2002.[9]

‖ From Table 1.

Comparing the estimated RSV-related disease burden with those attributed to the influenza virus and rotavirus suggest that in Australia amongst children, RSV is responsible for a much higher disease burden than the routinely monitored influenza virus and rotavirus (Figure). A Sydney-based study reports influenza-related hospitalisation rates varying from 0.95–6.94 per 1,000 population among children aged less than 1year (the group with the highest rate of hospitalisation),[16] whereas this study's estimates of RSV-related hospitalisation rates for the less than 1 year age group were almost 10 times higher (8.7 and 16.8 per 1,000 age-specific population) (Tables 1 and 2). The annual number of cases reported to LabVISE is also lower for influenza with approximately 200–1,000 cases per month of influenza A and 10–300 cases per month of influenza B reported during peak influenza season.[9] This is in contrast to about 1,000 cases of RSV reported per month as the usual number, with up to about 1,600 cases a month reported in one year.[9] A total of 13,191 cases of influenza A and 3,614 cases of influenza B cases was reported to LabVISE over the 10-year period (from 1991 to 2000), whereas a total of 36,346 cases of RSV was reported to LabVISE during the same 10 year period.[9] Hospitalisation rates in Australia for rotavirus among children less than 5 years of age have been estimated previously to be between 3.0 per 1,000 (for 1–4 year age group) and 4.1 per 1,000 (for the less than 1 year age group),[11] which once again is less than the 2.2–4.5 per 1,000 that was estimated for this same age group for RSV (Table 2).

Discussion

Our analysis based, on limited available data on RSV in Australia, suggests that RSV potentially causes a higher disease burden compared with viruses such as influenza or rotavirus that are monitored through surveillance programs and are vaccinated against as part of the national immunisation program in Australia. The lack of systematic monitoring systems in Australia for RSV meant that this study was limited to using data from published sources. This meant that the data are not the most recent and are subject to systematic errors when used to estimate the burden of disease for Australia.

The limitations of using a passive surveillance system as the primary source of data are recognised, and it

Table 3: Estimated incidence of respiratory syncytial virus infections, Australia, based on hospitalisation rates

Age group	Hospitalisation rate per 1,000 population (A)		Age-specific Population† (B)	Expected number of hospitalisations (C) = (A)x(B)	Number of RSV cases /year ‡ (D)=(C) x 50	Incidence per 1,000 population§ [(D) / (B)] x 1,000
<1 year	16.8	(Table 2 NHMD)*	260,101	4,362	218,095	838.5
<1 year	8.7–17.4	(Table 2 range)	260,101	2,263–4,521	113,144–226,028	435.0–869.0
<5 years	3.9	(Table 2 NHMD)*	1,260,403	4,916	245,779	195.0
<5 years	2.2–4.5	(Table 2)	1,260,403	2,773–5,710	138,644–285,481	110.0–226.5

* National Hospital Morbidity Dataset (NHMD) derived hospitalisation rates have been applied separately as these rates were higher than the Laboratory Virology and Serology Reporting Scheme–derived rate estimates for Australia (Table 2).

† Australian Bureau of Statistics Census 2006 accessed via the Australian Bureau of Statistics web site, Census data, 2006 Census (Australia, quick statistics).

‡ Assumes that hospitalisation = 2% of incidence.[3]

§ Per 1,000 age-specific population (Australia).

Table 4: Estimated cost of hospitalisation and direct health care cost for respiratory syncytial virus infections, Australia, (2005 value)

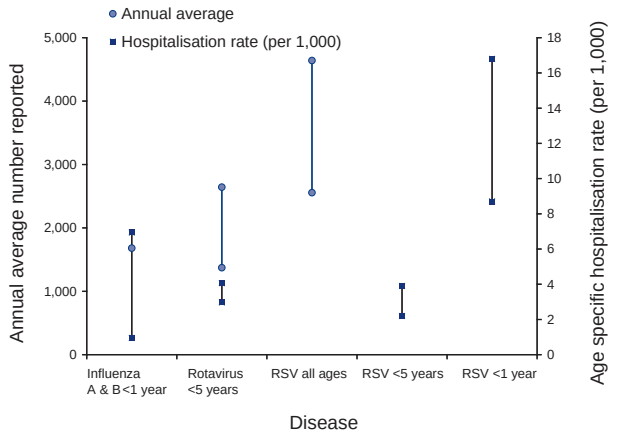
Age group	<1 year	<5 years
Estimated number of RSV hospitalisations (annual)	2,263–4,521*	2,773–5,710*
Direct cost of hospitalisation per year ($ million)†	$11.869–$23.713	$14.544–$29.949
Total direct health care cost per year ($ million)‡	$19.782–$39.521	$24.241–$49.915

* From Table 3.

† Unit cost = average cost of influenza/pneumonia as estimated by Newall et al = $5,245.[13]

‡ Total healthcare costs estimated based on reports that hospitalisation costs in respiratory syncytial virus (RSV) account for 60% of all direct health care costs associated with RSV.[5]

Figure: Disease burden caused by influenza A and B, rotavirus, and respiratory syncytial virus in Australia



Reference period for annual average and hospitalisation rates, respectively:

Influenza A & B:	2001 and 1994-2001.
Rotavirus:	1991-2002 and 1999-2000.
RSV all ages:	1991-2000.
RSV <5years:	1993-2000.
RSV <1 year:	1993-2000.

is acknowledged that such surveillance systems only provide a means of examining overall trends.[9] The implication of LabVISE being a passive surveillance system on this study is that the derived estimates underestimate the true disease burden. This fact is supported to some extent by the finding that the LabVISE estimated RSV hospitalisation rates were less than the NHMD derived rates (Table 2). The use of LabVISE to estimate hospitalisation has been considered previously by Roche and colleagues who state that the 'number of LabVISE reports of RSV was of the same order as total admissions for bronchiolitis.'[20] They go on to say that 'It is likely (that) LabVISE captures the majority of hospitalised cases of RSV through its networks or tertiary hospital laboratories in major Australian cities.'[20] For the purpose of this study, it is believed that LabVISE and the use of NHMD jointly provide information to demonstrate the high disease burden caused by RSV, particularly in relation to the influenza viruses and rotavirus.

Another limitation is the absence of a recent and definite estimate of the proportion of RSV cases that require hospitalisation, and the consequent use of statistics from a text book quoted in a journal paper. The implication of using the 2% upper end of the quoted range is that the incidence would be lower than had the 0.5% lower end of the range been used. The 0.5%–2% hospitalisation rate also relates to infants, and since infancy is a significant risk factor for hospitalisation,[21] it is reasonable to assume that the hospitalisation rates across all ages would be lower. The decreasing hospitalisation rate with age was also demonstrated in a conference paper presented by the Centre for Disease Control personnel in 2010 presenting national estimates of RSV-related hospitalisations for the period 1997 to 2006 for the USA. The estimates presented for the less than 1 age group were 2.63% in 1997–1999 and 2.34% in 2004–2006. The rates reduced with age, from 1.19% in the 1 to less than 2 year age group, to 0.19% in the 2 to less than 5 year age group.[22]

Counter arguments may also exist to explain higher rates of RSV being reported when in fact RSV infection rates are no more than the occurrence of influenza or rotavirus. For example, it may be possible that the diagnosis of influenza or rotavirus is often made based on clinical judgement not requiring serological assay, while testing for RSV may be undertaken more frequently. This may result in a higher number of RSV reportings that do not necessarily reflect the true disease burden.

Notwithstanding these limitations and possible explanations, the finding in this study that RSV is possibly contributing to a significant disease burden in Australia, especially when compared with influenza and rotavirus that are considered sufficiently significant to warrant monitoring and vaccination, needs further investigation. If RSV is a significant contributor to influenza-like illness and the associated economic burden, there is a need to accurately assess and understand the burden and to identify goals and strategies to protect and reduce morbidity, especially among high-risk groups.

The benefits of preventing RSV extend beyond reducing morbidity and mortality directly related to RSV among the high-risk groups. In common with other respiratory tract viral pathogens, RSV is associated with exacerbation of asthma.[23] While the majority of infants and young children infected with RSV experience a full recovery, there is evidence to suggest an association between RSV infection in early childhood and the likelihood of wheezing, or developing asthma.[3,24–27] This association is of particular relevance for Australia given that the prevalence of asthma in Australia is among the highest in the world, affecting 14%–16% children and 10%–12% adults.[28]

To date, no vaccine has been proved to be safe and effective in the control of RSV, and treatment is largely symptomatic. Prophylactic monoclonal antibody therapy has been shown to be effective in reducing the morbidity associated with RSV, however given the significant cost of the intervention, the current recommendation by the National Health amd Medical Research Council is to limit its use to high risk groups.[29]

Given that this study suggests a disease burden higher than those attributed to the influenza viruses or rotavirus prior to the introduction of vaccines, there is a need to further assess the disease burden and explore the value of monitoring RSV activity in Australia, primarily to establish a baseline level of activity, but also to assist in prioritising, implementing, and assessing control measures that may be required.

Ethical consideration

This study utilised aggregated data available in the public domain and did not require approval from an ethics committee.

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Using HIV notification data to identify priority migrant groups for HIV prevention, New South Wales, 2000–2008

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Abstract

Non-Australian-born people comprise a third of HIV notifications in Australia. With increasing numbers of immigrants in Australia, public health and health promotion programs will need to adapt to the emerging epidemic of HIV among people from culturally and linguistically diverse (CALD) backgrounds. This study uses HIV notification data to compare Australian-born and non-Australian-born cases in New South Wales and aims to determine if income of source country is useful in identifying high priority CALD groups. Notified cases of newly diagnosed HIV between 2000 and 2008 in New South Wales were divided into Australian-born, persons born in high-income countries and persons born in middle and low-income countries based on World Bank classifications. These three groups were then compared to determine their risk factors for HIV infection. Of the 3,397 newly diagnosed HIV infections in New South Wales, 2,906 (86%) had a country of birth reported from 102 different countries. Cases born in high-income countries were similar to Australian-born cases; predominantly men reporting homosexual acquisition. Both these groups were different to cases born in middle and low-income countries; they were younger, more commonly female and reported heterosexual acquisition of HIV. Using income from source countries is useful as a model to better understand and target responses to HIV in non-Australian-born populations in New South Wales as it suggests that the public health and health promotion response in New South Wales and Australia should also focus on the priority communities drawn from low and middle income countries. *Commun Dis Intell* 2011;35(2):185–191.

Keywords: HIV, immigrants, income, epidemiology

Introduction

There are 33 million people living with HIV worldwide and an estimated 2.7 million people were newly infected with HIV in 2008. sub-Saharan Africa has by far the highest prevalence of HIV in the world, at 5.2% in 2008, with the Caribbean, Eastern Europe, Central Asia, North America and Latin America having prevalence's between 0.5 and 1%, the remaining regions have less than this.[1] Australia has an estimated 17,444 people living with HIV or a prevalence of 0.1%, and between 2004 and 2008, non Australian-born people accounted for 33% of HIV notifications. With the exception of 2005, the highest annual incident rate over this period was among people born in sub-Saharan Africa.[2] Increasing cases of HIV among immigrants have been reported in other high-income countries in the European Union,[3,4] other European countries,[5] the United States of America (USA),[6,7] Canada,[8] Israel[9] and New Zealand.[10]

In New South Wales, where 54% of all newly diagnosed cases of HIV in Australia are located, HIV infection occurs predominately in men who have sex with men, although there has been a modest increase in the number of cases reporting heterosexual acquisition since the mid-1990s.[11] Non-Australian-born cases of HIV are drawn from multiple countries, but are concentrated in the countries of Asia and sub-Saharan Africa.[12] Surveys among these communities have found high levels of knowledge and awareness of HIV but variable practice to HIV prevention and poor health service access.[13] Recent migrants living with HIV are often faced with negotiating two major life disruptions simultaneously: an HIV diagnosis and the stressors of migration.[14]

Global population mobility and accelerating international migration has transformed the demography of most Western countries over the past 50 years,[15] facilitated by increased air travel and temporary migration.[16] Population mobility and migration have long been implicated in the history of the spread of infectious diseases like tuberculosis and hepatitis B, by bringing populations with disparate prevalence rates of disease into closer proximity with each other.[17]

Australia's annual migrant intake comprises 150,000 permanent settlers and more than 600,000 temporary residents per year.[18] The health requirements for permanent settlers aged over 15 years includes an HIV test,[14] which can occur in Australia or offshore.[19] For temporary visa applications, the health requirements vary depending on personal circumstance, intended activities in Australia, and country of origin or residence.[20] Applicants with health conditions that do not meet the health requirements (including HIV-positive applicants) may be granted a health

waiver. For the 2004/2005 financial year 156 such health waivers were granted from a total of almost 4.5 million visa applications to the Department of Immigration and Citizenship.[19] New South Wales is the most popular destination for both permanent settlers and temporary entrants[18] and has one of the most culturally diverse communities in Australia with 24% of the population born overseas and 16% speaking a language other than English at home.[21]

People from culturally and linguistically diverse (CALD) backgrounds are recognised as a priority population in the *NSW HIV/AIDS Strategy.*[22] According to this strategy, high priority groups within this population can be identified through HIV notifications in New South Wales, census and immigration data, and the prevalence of HIV in the countries-of-origin.[22] This study uses HIV notification data to compare Australian-born and non-Australian-born cases in New South Wales and aims to determine if income of source country is useful in identifying these high priority CALD groups.

Methods

Under the *Public Health Act 1991,* diagnosing laboratories are required to report all confirmed HIV infections that meet the national case definition[23] with further demographic, clinical, risk factor and testing history information collected from the diagnosing doctor. A case was defined as a person newly diagnosed with HIV between 2000 and 2008, who was a New South Wales resident at the time of diagnosis. Cases were excluded if they had a previous HIV positive test reported in New South Wales, they were not residents of New South Wales or reported a previous positive HIV test outside New South Wales.

Region of birth categories and New South Wales population by country of birth were obtained from the Australian Bureau of Statistics.[24] The two European regions were combined, and North Africa, the Middle East, and North East, Central and Southern Asia were combined due to small numbers of HIV cases. As population by country of birth was available for census years only (2001 and 2006), data were analysed in three 3 year groups: 2000–2002, 2003–2005 and 2006–2008 and the 2006 population data used as the denominator for the 2 later periods. Tests for trends were conducted using Poisson regression for rates and linear regression for proportions.

Gross national income as per the World Bank country classifications[25] were used to categorise the non-Australian-born into high and middle and low-income groups, and then compared these with Australian-born cases. Several categorical logistic regression models using backward elimination were constructed to compare these 3 groups: Australian-born with cases from other high-income countries (Model 1), Australian-born with cases from middle and low-income countries (Model 2), and cases from high-income countries with cases from middle and low-income countries (Model 3). Factors included in these models were age group, residence at diagnosis, exposure category and stage of HIV diagnosis. Gender was excluded from the models due to its high correlation with homosexually acquired exposure. Data were analysed using SAS version 9.1.3.[26]

Risk exposure information was provided by the treating medical practitioner on the notification form. Where more than one risk exposure was reported, a hierarchy of risk was used to designate the case's primary risk exposure; that most strongly associated with transmission of HIV. Where homosexually acquired infection was reported, it was considered the primary risk exposure. All heterosexually-acquired cases were combined in the models due to a small numbers in some categories.

Early diagnosis was defined as cases with either a negative or indeterminate HIV antibody test or a seroconversion illness in the 12 months prior to testing positive and late diagnosis was defined as having either a CD4 count less than 200 in the absence of a seroconversion illness or an AIDS defining illness within 3 months of HIV diagnosis. A third category of those with a CD4 count greater that 200 was also included. Residence at diagnosis, based on address information on the notification form, was categorised into Inner Sydney, comprising central and south-eastern Sydney, metropolitan comprising the remainder of Sydney, the Hunter and Illawarra areas and non-metropolitan comprising the rest of the State. Likely country of HIV acquisition has not been included in this analysis as it has only been collected since mid-2007.

Results

Between 2000 and 2008, there were 3,367 newly diagnosed HIV infections in New South Wales. The rate of newly diagnosed HIV in New South Wales remained stable over the study period, with an annual average rate between 5.1 per 100,000 in 2006–2008 to 6.1 per 100,000 in 2003–2005 ($P = 0.69$). Country of birth was reported for 2,906 newly diagnosed HIV cases (86%) and this proportion did not differ over the three periods ($P = 0.22$).

New HIV diagnoses by region and country of birth

There were 102 countries of birth reported for newly diagnosed HIV cases in New South Wales, with almost two-thirds (62%) of the study population

being Australian-born. Ten per cent were born in Europe, 9% in South East Asia, 5% in sub-Saharan Africa, 4% in Oceania (excluding Australia), 4% in North Africa, Middle East and Other Asia, 2% in Central and South America and 2% in North America.

Cases born in sub-Saharan Africa had the highest rates of new HIV diagnoses in New South Wales for each of the three periods, at over 30 per 100,000 population. This was followed by North America, where rates decreased over the study period, Central and South America, where the later period had the highest rates, and South East Asia. Although there was some fluctuation in the average annual rates by region of birth over the three periods for most regions, these were not significantly different (Figure).

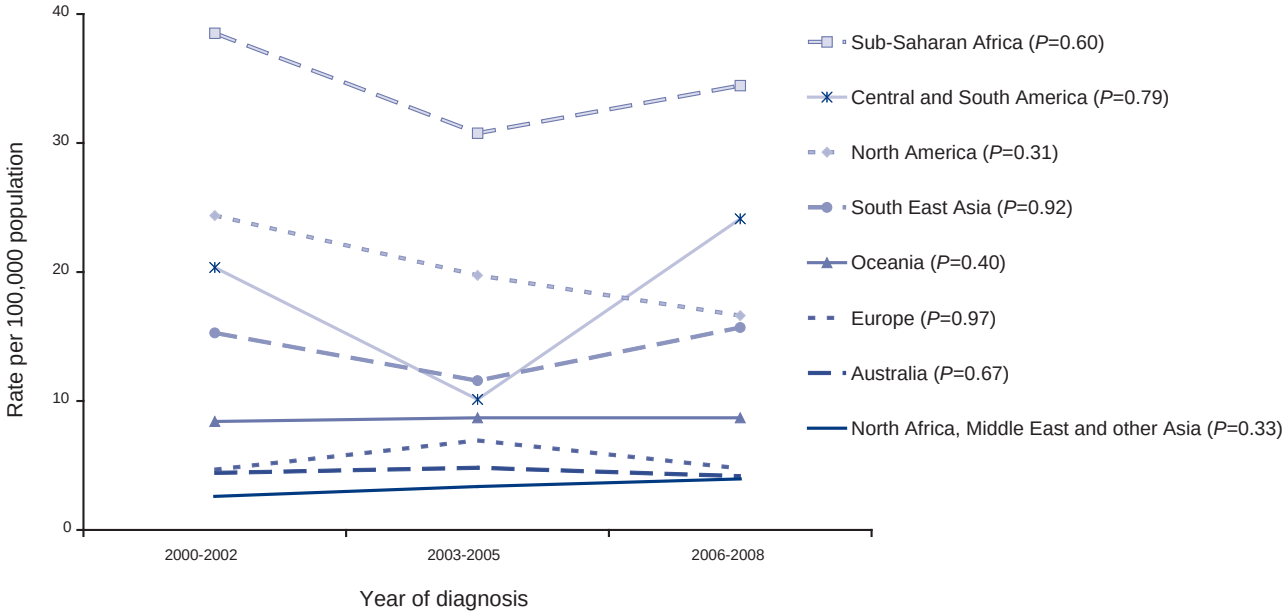
Countries of birth with more than 5 new HIV diagnoses in all three periods included Thailand, the United States of America (USA), New Zealand and the United Kingdom (UK). These rates remained stable for the USA and UK over the three periods at around 25 and 11 per 100,000 respectively. Rates for Thailand fluctuated over the three periods between 119.4, 57.0 and 95.0 per 100,000 respectively, and decreased for New Zealand from 6.2 per 100,000 during 2000–2002 to 4.8 per 100,000 in 2006–2008. During the last period Indonesia, Brazil and Zimbabwe had more than 5 cases per year at 27.4, 121.7 and 133.5 per 100,000 respectively, and South Africa had more than 5 cases in the earliest period at 16.3 per 100,000 population.

Comparison of country of birth income groups

There were 479 (17%) newly diagnosed HIV cases born in high-income countries and 614 (21%) born in middle and low-income countries. The average annual rate of new HIV diagnoses was consistent for the three groups over the three periods ($P = 0.67$, 0.96 and 0.74 for Australian-born, high-income and middle and low-income, respectively). These rates ranged from 4.2 per 100,000 for Australia-born in 2006–2008 to 11.0 per 100,000 for middle and low-income groups in the same period.

When comparing the epidemiology of cases by country of birth income group, the Australian-born and cases from high-income countries were similar to each other, yet both were different to cases from middle and low-income countries (Table 1). In terms of both gender and acquisition, males and homosexually-acquired exposure accounted for most new HIV diagnoses in the Australian-born and for cases from high-income countries, whereas almost a third of cases from middle and low-income countries were female and almost half were heterosexually acquired. In middle and low-income countries, a third of cases reported heterosexual acquisition from high prevalence countries (34%), compared with 0.5% of the Australian and none of the high-income groups. Cases from middle and low-income countries were also younger than the other two groups, with 73.9% aged less than 39 years, compared with 64.2% and 64.7% of the Australian-born and cases from high-income countries respectively.

Figure: Rate of newly diagnosed HIV, New South Wales, 2000 to 2008, by period of diagnosis and region of birth



There were differences between all three groups with respect to residence at diagnosis. Although the highest proportion for each group resided in Inner Sydney at diagnosis, this proportion was higher for the Australian-born and high-income countries than for the middle and low-income countries (64.3% and 77.0% compared with 51.0%). Australian-born cases had the highest proportion residing in non-metropolitan New South Wales at diagnosis at 8.8%.

As expected, language spoken at home also differed amongst the three groups, with half of cases from middle and low-income countries, 14.8% of cases from high-income countries and 0.2% of Australian-born cases reporting speaking a language other than English (LOTE) in the home (Table 1). The proportion reporting speaking a LOTE at home decreased from 20% in 2000–2002 to 11% in the two later periods and this was most marked in cases from middle and low-income countries, in which the proportion reporting speaking a LOTE at home decreased from 79% during 2000–2002 to 38% during 2006–2008.

The type of doctor diagnosing HIV also differed between the three groups, with a higher proportion

Table 1: Characteristics of Australian and non-Australian-born persons with newly diagnosed HIV in New South Wales, 2000–2008

Characteristic*	Income group		
	Australian-born	High-income	Middle and low-income
Total	1,813 (100.0)	479 (100.0)	614 (100.0)
Sex			
Female	107 (5.9)	22 (4.6)	169 (27.5)
Male	1,699 (93.7)	453 (94.6)	439 (71.5)
Age group			
<30 years	455 (25.1)	112 (23.4)	229 (37.3)
30–39 years	708 (39.1)	198 (41.3)	225 (36.6)
40–49 years	443 (24.4)	105 (21.9)	115 (18.7)
>50 years	207 (11.4)	64 (13.4)	45 (7.3)
Exposure			
Heterosexually	188 (10.4)	49 (10.2)	291 (47.4)
High prevalence†	9 (0.5)	0 (0.0)	207 (33.7)
Homosexually	1,479 (81.6)	387 (80.8)	261 (42.5)
Intravenous drug users	73 (4.0)	10 (2.1)	23 (3.7)
Other	8 (0.4)	0 (0.0)	4 (0.7)
Stage at diagnosis			
Late	236 (13.0)	62 (12.9)	127 (20.7)
CD4 >200	539 (29.7)	161 (33.6)	214 (34.9)
Early	699 (38.6)	168 (35.1)	104 (16.9)
Residence at diagnosis			
Inner Sydney	1,165 (64.3)	369 (77.0)	313 (51.0)
Metropolitan	447 (24.7)	86 (18.0)	254 (41.4)
Non-metropolitan	159 (8.8)	15 (3.1)	26 (4.2)
Language spoken at home			
English	1,779 (98.1)	399 (83.3)	230 (37.5)
LOTE	3 (0.2)	71 (14.8)	334 (54.4)
Diagnosing doctor			
General practitioner	1,131 (62.4)	220 (45.9)	256 (41.7)
Publicly funded clinic	606 (33.4)	214 (44.7)	261 (42.5)
Immigration /occupational	1 (0.1)	25 (5.2)	71 (11.6)
Other	47 (2.6)	10 (2.1)	20 (3.3)

LOTE Language other than English

* May not add to total due to missing responses.

† Exposure reported from countries or regions are those where the prevalence of HIV in the population is more than 1%

of Australian-born cases being diagnosed by general practitioners (62.4%) compared with less than half of cases from high-income and middle and low-income countries (45.9% and 41.7% respectively). Cases from high-income and middle and low-income countries had higher proportions being diagnosed at publicly funded clinics or immigration and occupational services (Table 1).

The multivariate models confirmed these observations. In model 1, where Australian-born cases were compared with cases from high-income countries, there were only two differences. Australian-born cases were more likely to have resided in non-metropolitan areas at diagnosis and report exposure through injecting drug use (Table 2).

In the comparison of Australian-born cases with cases from middle and low-income countries (Model 2), there were several differences. Australian-born cases were more likely to be aged more than 50 years, reside in non-metropolitan areas at diagnosis, and report exposure as either homosexually acquired or acquired through IDU. Results from the comparison between cases from high-income and cases from middle and low-income countries (Model 3) were similar to that of Model 2, as cases from high-income countries were also more likely be aged more than 50 years, reside in non-metropolitan areas at diagnosis, and have either homosexually or IDU acquired exposure compared with cases from middle and low-income countries. The odds ratios were higher in Model 2 compared with Model 3, suggesting that the difference between Australian-born cases and cases from middle and low-income countries was more marked than the differences between cases from high-income countries and cases from middle and low-income countries (Table 2).

Discussion

Almost two-thirds of cases newly diagnosed with HIV in New South Wales between 2000 and 2008 were Australian-born, with the remainder being born in one of 101 other countries. Cases from sub-Saharan Africa had the highest rates in New South Wales over the study period, followed by North America, Central and South America and South East Asia.

Cases born in other high-income countries were similar to Australian-born cases, predominantly men reporting homosexual-acquisition of their HIV infection. These two groups were different

Table 2: Multivariate analyses comparing Australian-born with those born in high-income countries, Australian-born with those born in middle and low-income countries and those born in high-income countries with those born in middle and low-income countries

Characteristic	Model 1: Australian-born vs high-income	Model 2: Australian-born vs middle and low-income	Model 3: High vs middle and low-income
Period			
2000–2002	–	1.0	1.0
2003–2005	–	1 (0.8–1.4)	1.3 (0.8–1.9)
2006–2008	–	0.6 (0.4–0.8)	0.7 (0.5–1.1)
Age group			
<30 years	–	0.7 (0.5–1)	0.7 (0.5–1.1)
30–39 years	–	1.0	
40–49 years	–	1.3 (1–1.9)	1 (0.7–1.6)
>50 years	–	2.5 (1.5–4.1)	2.7 (1.5–5)
Residence at diagnosis			
Metropolitan	1.0	1.0	–
Inner Sydney	0.6 (0.4–0.8)	0.8 (0.6–1.1)	–
Non-metropolitan	1.9 (1–3.5)	3.7 (2.1–6.8)	–
Exposure			
Heterosexually	1.0	1.0	1.0
Homosexually	1.5 (1.0–2.2)	7.9 (5.8–10.7)	6.1 (4.1–9.2)
Intravenous drug users	2.8 (1.1–7.1)	9.7 (4.5–20.9)	3.5 (1.1–10.9)
Stage at diagnosis			
Late	–	1.0	1.0
CD4 >200	–	1.4 (1–1.9)	1.5 (1–2.4)
Early	–	3.0 (2.1–4.3)	2.6 (1.6–4.2)

to cases born in middle and low-income countries as they were younger, more commonly female and reported heterosexual acquisition of their HIV infection. This reflects the global pattern of HIV epidemics in their source countries. In the high-income countries of North America and Western and Central Europe, national epidemics are concentrated among key populations including men who have sex with men, injecting drug users and immigrants.[1] In contrast, in sub-Saharan Africa, the region most heavily affected by HIV worldwide, HIV affects all social and economic groups, women and children disproportionally, with transmission predominantly through heterosexual contact. In Asia, the epidemic has long been concentrated in specific populations, namely injecting drug users, sex workers and their clients, and men who have sex with men. However, more recently, the epidemic in many parts of Asia is steadily expanding into low-risk populations through transmission to sexual partners of those most at risk. In some countries, like China, heterosexual contact is now the most predominant mode of transmission.[1]

Although most cases from each of the three groups resided in inner and metropolitan Sydney at diagnosis, Australian-born cases were more likely to reside in non-metropolitan New South Wales. This also reflects migration patterns as cases from middle and low-income countries typically settle in more affordable suburbs whereas those from high-income countries are able to settle in inner Sydney.

Cases from middle/low-income countries were also more likely to be diagnosed late in their infection in New South Wales. This is consistent with national data, which indicates people born in Asia and sub-Saharan Africa had the highest rates of late HIV diagnosis in Australia from 2003–2008.[2] Local social research in New South Wales supports this. Asian gay men in Sydney had much lower rates of prior testing for HIV compared with Anglo-Celtic gay men[27,28] and surveys among wider Asian and sub-Saharan Africa communities in New South Wales have indicated high levels of HIV knowledge and awareness but limited use of health services for HIV despite being eligible for Medicare.[13] Immigrants living with HIV in New South Wales have reported experiencing their diagnosis as a 'death sentence'.[14] These barriers have been reported in other high-income countries with multiple disincentives for immigrants to present earlier for HIV testing and treatment[29] and should be considered in any public health action aimed at reducing the proportion of late diagnoses in these groups.

The variety in countries of birth reported in this study reflects how multicultural New South Wales is and the nature of temporary and permanent migration flows in a globalised world. With increasing numbers of permanent and temporary immigrants to Australia,[18] HIV infections among non-Australian-born populations will continue. Using income of home countries as a predictor for risk factors for new HIV diagnoses in these groups was useful as it showed that immigrants from high-income countries are similar to Australian-born cases and that immigrants from middle and low-income countries are different in terms of risk factors. This suggests that this simple methodology could be used as a proxy measure to identify high priority groups for health promotion efforts where information on HIV prevalence in the home countries is unavailable.

Using surveillance data to describe HIV in New South Wales has limitations. It counts the first positive test in New South Wales and therefore cannot report on the total number of people infected with HIV. Cases need to be tested and diagnosed for HIV before they are included in surveillance data. Also, as HIV is a chronic infection with a long latent period, many persons who are newly infected in a given year may not be diagnosed until years later. Cases were excluded from this analysis if they have been previously diagnosed outside New South Wales and this includes immigrants that tested positive before their arrival in Australia. This group would potentially require the same services as those newly diagnosed in New South Wales, but are left out of any analysis using surveillance data that is aimed at tracking new infection. The number of new HIV infections reported in this study is therefore likely to be an underestimation of the total number of overseas-born people living with HIV in New South Wales.

Conclusion

Permanent and temporary migration and mobility to and from Australia is likely to continue to have an effect on the HIV situation in New South Wales in the years ahead. HIV health promotion and access to testing, treatment, care and support will need to continue to adapt to the emerging epidemic of HIV among overseas-born populations. The use of high, middle and low-income countries as a model to understand and target responses to HIV in non-Australian-born populations in New South Wales facilitates prioritisation to ensure that the most affected communities are reached. This study suggests that the public health response in New South Wales should focus on communities drawn from low- and middle-income countries from South East Asia and sub-Saharan Africa. The study also shows that the income of the country of birth is predictive of the dominant pattern of HIV transmission among these communities, as it largely mirrors the HIV patterns in their home country and/or region of birth. Similarly, income of country of birth is predictive of stage of presentation with HIV infection.

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Outbreak of *Salmonella* Typhimurium phage type 44 infection among attendees of a wedding reception, April 2009

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Abstract

On 30 April 2009, the Communicable Disease Control Branch (CDCB) South Australia was notified of a *Salmonella* infection in a person who attended a wedding reception on 25 April 2009. Several other attendees reported becoming unwell with a similar gastrointestinal illness. The CDCB commenced an investigation to: characterise the outbreak in terms of person, place and time; identify probable source or sources; and implement control measures. A retrospective cohort study was undertaken among wedding reception attendees. A questionnaire collecting information on demographics, illness and menu items consumed was given to the majority of attendees. An environmental inspection of the wedding reception premise and food supplier premise, including food sampling was conducted to identify plausible sources of infection. The questionnaire response rate was 77%, from which an attack rate of 20% was calculated. There was a significant association between consumption of garlic aioli and illness (OR 5.4, 95% CI: 1.6, 18.1). Nine wedding reception attendees' stool samples tested positive for *Salmonella* Typhimurium phage type 44. A sample of garlic aioli also tested positive for *Salmonella* Typhimurium phage type 44. The ingredients of the garlic aioli included raw egg yolk, roasted garlic, Dijon mustard, vinegar and vegetable oil. The raw egg yolk was identified as a high risk food item; however no eggs tested positive for *Salmonella*. *Commun Dis Intell* 2011;35(2):192–196.

Keywords: *Salmonella*, outbreak, eggs, garlic aioli, cohort study

Introduction

Salmonella gastroenteritis is characterised by fever, headache, abdominal pain, diarrhoea, nausea and sometimes vomiting.[1] Infection occurs after ingestion of the organism, typically from food derived from infected animals or contaminated by the faeces of an infected animal or human.[1] The incubation period ranges from 6 to 72 hours, however longer incubation periods of up to 16 days have been described associated with low infectious doses.[1]

Salmonellosis is the 2nd most commonly reported notifiable infectious gastrointestinal disease in Australia after campylobacteriosis, with 9,335 cases of *Salmonella* infection recorded by the National Notifiable Diseases Surveillance System (NNDSS) in 2009.[2] The national notification rate of salmonellosis in 2009 was 42.7 per 100 000 population.[2] Within South Australia, in 2009 there were 683 notifications, compared with the 5-year (2004 –2008) mean of 631.[3]

In 2006 and 2005, *S.* Typhimurium 44 was the 7th most common *Salmonella* isolate notified to NNDSS.[4,5] Australia recorded 211 cases in 2006 (16 reported from South Australia) and 228 cases in 2005 (28 reported from South Australia).[4,5]

On 30 April 2009 a doctor contacted the Communicable Disease Control Branch (CDCB) in South Australia to advise that a 24-year-old male patient had been diagnosed with *Salmonella* infection. His onset of illness was 27 April 2009 and he presented with fever, diarrhoea, abdominal cramps and myalgia. The doctor also reported that several other people who had attended the same wedding reception on 25 April 2009 had become unwell with a similar gastrointestinal illness. The *Salmonella* was further characterised as *Salmonella* Typhimurium phage type 44.

As a result of these reports of illness at the wedding reception, an investigation was conducted to characterise the outbreak, identify the source of infection and to prevent further cases of illness.

Methods

Epidemiological investigation

A cohort study was initiated to identify the source of infection. The cohort was defined as those who consumed food at the wedding reception at Venue A on Saturday 25 April 2009. A questionnaire for attendees of the wedding reception was developed. The questionnaire sought demographic and illness (prior, during and after the reception) information. It also asked attendees about the consumption of food and drink items at the function. The questionnaire, with an accompanying cover letter explaining the outbreak investigation and a reply-paid envelope, was mailed to the majority of attendees. Those who failed to reply, could not read English, or for whom an address was not supplied, were scheduled to be interviewed by telephone.

The case definition for the outbreak included any person who consumed food at the wedding reception on 25 April 2009 at Venue A and subsequently reported gastrointestinal illness (three or more loose bowel movements in a 24 hour period), or a laboratory confirmed *Salmonella* infection, with an onset on or after 26 April 2009.

The questionnaire data were collated in Microsoft Access 2003 software. Descriptive analysis was conducted using Microsoft Excel 2003 software and Stata 10. Univariate analysis was performed, calculating relative risk and 95% confidence intervals (CI) for individual exposures for illness. A multivariate analysis was conducted using logistic regression to identify independent variables that correlated with illness. All foods from the univariate analysis that had a relative risk of two or greater with the 95% confidence interval not including the null, were added to the model. Statistical analyses were conducted using Stata 10.

Laboratory investigation

Faecal samples from attendees at the wedding reception that were requested by a medical practitioner were sent to various accredited laboratories in South Australia. On confirmation of a positive *Salmonella* sample, the isolate was then referred to the Australian *Salmonella* Reference Laboratory for further typing.

Environmental investigation

An environmental inspection of Venue A was conducted by a local council environmental health officer on 30 April and 6 May 2009. The inspection sought details on menu items served, food ingredients, food suppliers and staff illness prior to the function.

Food samples of the garlic aioli served on the night of the wedding, and eggs from Venue A were collected by the environmental health officer and were sent to the Food and Environmental laboratory of the Institute of Medical and Veterinary Science for bacterial testing.

The Food Policy and Programs Branch of the Department of Health also conducted an inspection of the egg and garlic suppliers on 8 May 2009. Food samples of peeled garlic and eggs were collected for bacterial testing.

Results

Epidemiological investigation

One hundred and ninety attendees consumed food at the wedding reception at Venue A on 25 April 2009. Completed questionnaires or interviews were obtained from 147 attendees. This represented a response rate of 77%. Of the respondents, 30 met the case definition, representing an attack rate of 20% (30/147) among responders. Dates of onset of illness ranged between 26 April and 3 May 2009 (Figure). The incubation period ranged between 1 day and 8 days (median 2 days).

The median age of wedding reception attendees who consumed food was 33 years (range 2 years to 90 years) with a male to female ratio of 1:1.3. In those who met the case definition, the median age was 30.5 years (range 8 years to 60 years) with a male to female ratio of 1:1.

Diarrhoea was reported in all 30 cases, 10 of these cases reported bloody diarrhoea. Other common symptoms included abdominal pain (77%) and chills (60%). Four people (13%) experienced vomiting.

The analysis identified a significant association between consumption of garlic aioli and prawns and illness (Table 1).

The significant variables of prawns and garlic aioli were added into a multivariate analysis model. The results identified a significant association between the consumption of the garlic aioli and illness (OR 5.4, 95% CI: 1.6, 18.1).

Eight people, two of whom were laboratory confirmed *S.* Typhimurium 44, did not show symptoms during the typical incubation period of 6–72 hours.[1] All of the 8 cases with an onset after 28 April 2009 (i.e. an incubation period of greater than 72 hours) had other household members that attended the same function. Of these, three had family members who had become unwell prior to their own illness. It is possible that the 8 cases were infected by person-to-person transmission from sick or

Figure: Wedding reception attendees that meet the case definition for gastrointestinal illness, 25 April to 2 May 2009, by date of onset

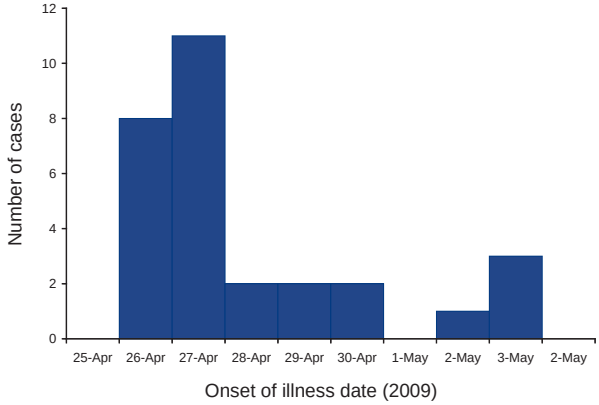

Table 1: Univariate analysis for a selection of food and drinks consumed exposure status, attack rates and risk ratio analysis

	Person who ate item			Person who did not eat item					
Food or drink item	Number ill	Total number	Attack rate (%)	Number ill	Total number	Attack rate (%)	Relative risk	95% CI	*P* value
Octopus	21	86	24	9	61	15	1.66	0.8,3.4	0.15
Feta	22	89	25	8	58	14	1.79	0.9, 3.8	0.11
Prosciutto	25	106	24	5	41	12	1.93	0.8, 4.7	0.12
Prawns	26	95	27	4	52	8	3.56	1.3, 9.7	0.00
Garlic aioli	24	68	35	6	79	8	4.64	2.0, 10.7	0.00
Warm potato and red onion salad	14	78	18	16	69	23	0.77	0.4, 1.5	0.43
Sweet potato	10	57	18	20	90	22	0.79	0.4, 1.5	0.49
Potato	8	64	13	22	83	27	0.47	0.2, 1.0	0.04
Zucchini	10	69	14	20	78	26	0.57	0.3, 1.1	0.09
Asparagus	11	64	17	19	83	23	0.75	0.4,1.5	0.39
Vegetarian curry with couscous	0	9	0	30	138	22	undefined	undefined	0.12
Salt	9	34	26	21	113	19	1.42	0.7, 2.8	0.31
Wedding cake	6	55	11	24	92	26	0.42	0.2, 1.0	0.03
White wine	14	55	25	16	92	17	1.5	0.8, 2.8	0.24
Orange juice	6	22	27	24	125	19	1.4	0.7, 3.1	0.38
Spirits	12	44	27	18	103	17	1.6	0.8, 3.0	0.18

CI Confidence interval.

asymptomatic household members. To account for this possibility, the analysis was repeated excluding the 8 cases with onset after the 28 April 2009. The outcome was the same, a significant association being demonstrated between consumption of garlic aioli and illness (RR 4.4, 95% CI: 1.2, 17.0). As the infection could have occurred from low dose ingestion, and the outcome did not change, all cases were included in the final analysis.

Laboratory investigation

Ten people with gastrointestinal symptoms submitted a stool sample for laboratory testing. Nine specimens were positive for *S*. Typhimurium 44.

Environmental investigation

Two environmental inspections of Venue A were conducted by the local council environmental health officer on 30 April and 6 May 2009. No issues were noted during these inspections. Hygiene and food handling preparation were found to be of a high standard. No staff members reported illness. The egg samples collected tested negative for *Salmonella*. A sample of garlic aioli that was served on the night of the wedding was positive for *S*. Typhimurium 44.

An inspection of the premises of both the supplier of eggs and of the garlic was conducted by the Food Policy and Programs Branch of the Department of Health. No significant hygiene issues were identified. Samples of peeled garlic and eggs were negative for *Salmonella*. The supplier had inadequate traceability records to confirm the source of the eggs purchased.

Discussion

The investigation of this outbreak has demonstrated epidemiological and statistical links between illness due to *S*. Typhimurium 44 and consumption of garlic aioli by attendees of a wedding reception. The ingredients in the garlic aioli consisted of raw eggs, vinegar, roasted garlic, mustard and oil.

It is plausible that the raw eggs may have been responsible for the contamination. Eggs have been associated with numerous *Salmonella* outbreaks in Australia.[7–11] An analysis of the OzFoodNet outbreak register data from January 2001 to December 2008 identified 23 outbreaks of *S*. Typhimurium 44 associated with egg consumption.[12] In particular, Victoria has experienced several outbreaks during recent years, predominantly at the end of 2006 and early in 2007. Six of these outbreaks showed a probable association with the consumption of contaminated eggs.[12]

Based upon this evidence the raw egg yolk in the garlic aioli was identified as a high risk food item; however no eggs tested positive for *Salmonella*. To reduce the risk of further infections, it was recommended to the catering company to consider amending their food preparation techniques and use pasteurised eggs instead of raw eggs for products not subject to cooking.

The other ingredients of the garlic aioli, including roasted garlic, Dijon mustard, vinegar and vegetable oil, cannot be excluded as the contaminated food item in the garlic aioli. As the garlic was roasted it reduces the risk of it being the responsible agent. Poor hygiene measures including cross contamination by kitchen staff or an infected food handler also cannot be excluded. The inspection indicated that no staff reported illness and the garlic aioli was appropriately prepared, handled and stored prior to serving.

The raw egg was hypothesised to be the most plausible source of the contamination in the aioli. Further investigations into the eggs could have determined that the other ingredients or cross contamination was even less likely to have caused the outbreak. However, this was not possible as the egg supplier to Venue A did not have adequate traceability records to confirm the source of the eggs purchased. A trace back to the egg farm may have provided further evidence in the investigation e.g. the same *Salmonella* strain could have been found on the farm. This investigation has highlighted the need for improved record keeping and supports the introduction of stamping eggs to allow their source to be identified.

Prawns also had an elevated risk ratio that was statistically significant on univariate analysis. However this was no longer statistically significant once adjustment for consumption of garlic aioli was made during multivariate analysis. It is therefore likely that this apparent association between prawns and illness was due to confounding by garlic aioli consumption as prawns were served with the garlic aioli as a dipping sauce. It is also more plausible that eggs were the source of infection than prawns as a single egg could contaminate the aioli that was served to a number of guests, while a large number of prawns would need to be contaminated in order to cause illness in a group of people.

There was potential for selection bias during the investigation as some attendees did not receive a questionnaire as their address was unknown or they were unable to speak English. Some of these people (16%) were interviewed by telephone, which could have introduced interviewer bias. This was limited by having one interviewer involved in interviewing attendees.

Ten people responded 'unknown' for consumption of aioli. These people may have been unfamiliar with the terminology of 'aioli' and therefore answered unknown, resulting in reporting bias. However this seems less likely given that other food items, including those that are commonly understood, particularly those served as a complement to the main dish e.g. sweet potato, warm potato and red onion salad, and asparagus had a higher 'unknown' response rate (average 18 'unknown').

It is possible that illness affected the likelihood that someone would respond to the questionnaire. Although the attack rate was found to be 20%, as the response rate was 77%, the attack rate could have been as low as 16% or as high as 38% using the extreme assumptions that those of unknown illness status were all not ill or all were ill. However, non-response would only bias the strength or direction of the association between food consumed and illness if the likelihood of responding varied with food consumed, which is less likely.

Individuals who have experienced gastrointestinal symptoms may be more likely to think about the foods eaten prior to the onset of their illness than those unaffected by illness, resulting in recall bias. This could lead to either an underestimation or overestimation of the effect of the association between exposure and disease, depending on whether the cases recall their exposure to a greater or lesser extent than non-cases.

Whilst the investigation could not determine the source of the *Salmonella* in the garlic aioli, the vehicle was successfully identified with both epidemiological and microbiological evidence. Raw eggs, as a high risk food item for *Salmonella* are a plausible cause of the contamination in this outbreak. This outbreak has highlighted the need for more stringent regulations on the production and sale of eggs, including product traceability. Commercial food outlets should reduce the risk to the public and cease serving dishes that contain raw eggs. The public would also benefit from being better informed on the risks of consuming raw egg products.

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Surveillance summaries

Catching up with the catch–up: HPV vaccination coverage data for Australian women aged 18–26 years from the National HPV Vaccination Program Register

Julia Brotherton, Dorota Gertig, Genevieve Chappell, Lesley Rowlands, Marion Saville

Abstract

This report describes human papillomavirus (HPV) vaccine coverage data for Australian women 18–26 years of age, as notified to the National Human Papillomavirus Vaccination Program Register. A cross-sectional analysis was conducted of notifications to the Register of HPV vaccine doses delivered as part of the National HPV Vaccination Program, which provided free catch-up vaccination to women 18–26 years of age across Australia between 2007 and 2009. HPV vaccination coverage estimates were calculated by age, state or territory of residence and dose number, using the Australian Bureau of Statistics population estimates as the denominator. As at March 2011, approximately 4.49 million doses had been notified to the Register of females of all ages, and 1.7 million of these were for women aged 18–26 years in 2007. Vaccination coverage was highest for females aged 18 years and lowest in females aged 26 years. For the entire 18–26 years cohort, coverage was estimated at 55.2% for dose 1, 44.8% for dose 2 and 31.7% for dose 3. Notified dose 1 coverage rates by single year of age and state or territory ranged between 22% for females aged 26 years in the Northern Territory and 76% in females aged 18 years in Queensland, with dose 1 coverage highest across the age range in the Northern Territory, Queensland and South Australia. These data suggest that over half of Australian women aged 18–26 years commenced HPV vaccine courses and about one-third are fully vaccinated. Some of the differences in the coverage observed between states and territories likely reflect differing mechanisms for notifying to the Register. *Commun Dis Intell* 2011;35(2):197–201.

Keywords: human papillomavirus, immunisation, vaccination coverage

Introduction

Both currently registered prophylactic human papillomavirus (HPV) vaccines are highly efficacious at preventing persistent infection with, and cervical lesions due to, targeted HPV types.[1] Both vaccines protect against the high risk HPV types 16 and 18, which are detected in 70%–80% of cervical cancers in Australia,[2] and the quadrivalent vaccine also provides protection against HPV types 6 and 11, which are detected in 90%–95% of genital warts.[3] As part of the National Immunisation Program, in 2007 the Australian Government funded a Human Papillomavirus Vaccination Program at a cost of $632.9 million over 4 years. A catch-up program using a 3-dose course of quadrivalent vaccine for women 12–26 years of age was delivered through school-based programs for girls at school and through general practice and community-based programs, until 30 June 2009. Women who had started the 3-dose course were provided the opportunity to complete the course by 31 December 2009. The Program now provides HPV vaccine on an ongoing basis through schools for girls aged 12–13 years.

As HPV vaccines are prophylactic only, the primary target group for HPV vaccination is non-sexually active, pre-adolescent females (i.e. before exposure to HPV). Based on trial evidence and policy considerations, many countries have made national recommendations for the inclusion of HPV vaccine in their vaccination programs, with differing target populations according to local data about age of sexual debut, delivery strategies and resources.[5,6] Whilst both Canada and the United States of America recommend vaccination through to age 26 years (the oldest age included in the pre-registration vaccine trial population), to date only Australia has implemented a national, funded vaccination catch-up program for women aged 18–26 years.

As part of the National HPV Vaccination Program, the establishment of an HPV Vaccination Register was enabled through legislation. The National HPV Vaccination Program Register (NHVPR) is Australia's first national adult vaccination register. Consent (verbal or written) is required to provide details to the Register of doses administered, and an individual may opt-off at any time by writing to the Register. Because of the tight timelines for

the initial roll-out of the vaccination program by April 2007, there was a relative delay in the establishment of the NHVPR, which commenced operations in June 2008. This report presents vaccine coverage data for women 18–26 years of age as notified to the NHVPR.

Methods

Collection of notifications from general practitioners and other immunisation providers

In July 2008, all practitioners identified as practising general practitioners (GPs) through Medicare Australia (Medicare) claims data, were invited by mail to register with the NHVPR. However, GPs were only required to complete a registration form if notification payments were requested. GPs were paid $6 per dose notified as having been administered to an age-eligible woman. To encourage notification, the NHVPR provides a range of options for notification to the Register. In most states and territories, immunisation providers notify the Register directly using one of several methods: using automated print-outs from any of the commonly used practice management software packages, by completing a notification form; or via any other paper-based recording of notifications as long as the required fields are present. Notifications can be mailed or faxed to the Register. Both Queensland and the Northern Territory have state-based immunisation registers to which immunisation providers are accustomed to reporting. Providers are not required to directly notify the Register but rather to notify their health department, which then notifies the Register. In South Australia, at the commencement of the catch-up program, their health department established central reporting of GP delivered HPV vaccinations to the department, which then notified the Register once it was established. Since late 2008, South Australian providers have notified the Register directly.

State and territory health departments are responsible for providing notifications from their school-based programs to the NHVPR. Most states and territories maintain state immunisation databases and notify episodes to the Register by electronic upload.

Data entry and processing

Vaccination episodes notified to the NHVPR undergo validation, where the data are checked for mandatory fields, correct data formats and correct data. This validation process checks gender, Medicare number algorithms, presence of localities and postcodes, vaccination dates within a defined range and validity of provider details. Records failing validation are rejected and require review and follow-up. This involves contacting GP practices to check details submitted and to collect missing information. Records that pass the initial validation then undergo consumer matching and further data quality checks. Specialised matching software attempts to match the incoming records with existing consumers on the Register. Where the system is able to locate an exact match, the incoming record is then added to the existing consumer's record. Where the system is not able to find an exact match, it will either create a new record or look for possible matches, which are then reviewed by a data processing operator. The records are also checked for data anomalies and inconsistencies, such as invalid combinations of state, suburb and postcode or GP details inconsistent with Medicare Provider files. These anomalies are presented to an operator for resolution.

Analysis of notifications

Data were extracted from the NHVPR as at 22 March 2011, for all valid doses (i.e. administered according to the minimum dose intervals specified in *The Australian Immunisation Handbook* 9th edition (2008)[6] or constituting part of a clinically complete course, as designated by the Guidelines of the Chief Medical Officer[7]) in women aged 18–26 years in 2007. Coverage was calculated as valid doses notified, divided by estimated resident population expressed as a percentage. Coverage was calculated by dose number, consumer state of residence and age in years as at mid-2007, using mid-year 2007 Australian Bureau of Statistics population estimates as the denominator.

Results

As at 28 January 2011, 22,899 GPs had registered to notify doses with the NHVPR. By March 2011, approximately 4.5 million doses had been notified to the Register from females of all ages, with 1.7 million of these valid doses in women aged 18–26 years in 2007.

National notified coverage for women aged 18–26 years was 55.2% for dose 1, 44.8% for dose 2 and 31.7% for dose 3. Women aged 26 years in 2007, only half of whom were eligible for vaccination given that the GP or community program started in July 2007 and women who had already turned 27 were ineligible to commence the course, had substantially lower notified coverage than other ages. Coverage for women aged 18–25 years is therefore slightly higher at 58.3%, 47.4% and 33.5% for doses 1, 2, and 3 respectively. Coverage rates by dose number, age in 2007 and state are given in the Table. The Figure presents dose 1 coverage data by age in years and state of residence, and graphically indicates that the 2 states and 1 territory with central reporting of HPV doses have the highest documented vaccine coverage.

Table: Notified Australian human papillomavirus vaccination coverage on the National HPV Vaccination Program Register for women aged 18 to 26 years in 2007, as at 22 March 2011,* by dose number, age, and state or territory of residence

State (total doses)	Dose	Age in years (as at mid-2007)									
		18 (%)	19 (%)	20 (%)	21 (%)	22 (%)	23 (%)	24 (%)	25 (%)	26 (%)	18–26 (%)
ACT	1	64	59	56	58	58	56	54	46	31	53
n=33,066	2	54	48	45	47	45	45	43	39	26	43
	3	41	35	31	33	33	33	33	29	19	32
NSW	1	60	55	53	52	50	50	49	43	26	48
n=474,102	2	49	43	41	40	39	38	38	34	20	38
	3	37	30	29	27	27	26	26	24	15	27
NT	1	74	69	69	64	66	64	61	48	22	59
n=20,826	2	63	54	55	50	53	51	48	40	18	47
	3	49	40	41	34	38	37	35	29	13	34
Qld	1	76	74	72	68	67	67	66	58	33	64
n=394,872	2	63	61	60	56	56	55	55	47	26	53
	3	40	38	37	36	35	36	35	29	13	33
SA	1	69	67	64	61	60	60	59	52	32	59
n=132,837	2	57	55	52	50	49	49	49	43	26	48
	3	42	39	36	35	34	34	34	31	18	34
Tas	1	65	60	63	60	59	57	54	48	31	56
n=37,326	2	55	49	51	49	48	47	45	40	25	46
	3	43	36	38	36	35	35	33	30	18	34
Vic	1	70	63	62	60	60	60	58	52	35	58
n=470,004	2	61	53	52	50	50	50	48	44	29	48
	3	49	41	39	37	37	38	37	34	23	37
WA	1	58	56	55	54	53	52	51	41	25	50
n=155,461	2	47	46	44	43	43	41	41	33	20	40
	3	35	34	32	31	31	30	30	25	15	30
Australia	1	67	62	61	59	58	57	56	49	30	55
n=1,718,494	2	55	50	49	47	46	46	45	40	24	45
	3	41	36	34	33	32	32	32	28	17	32

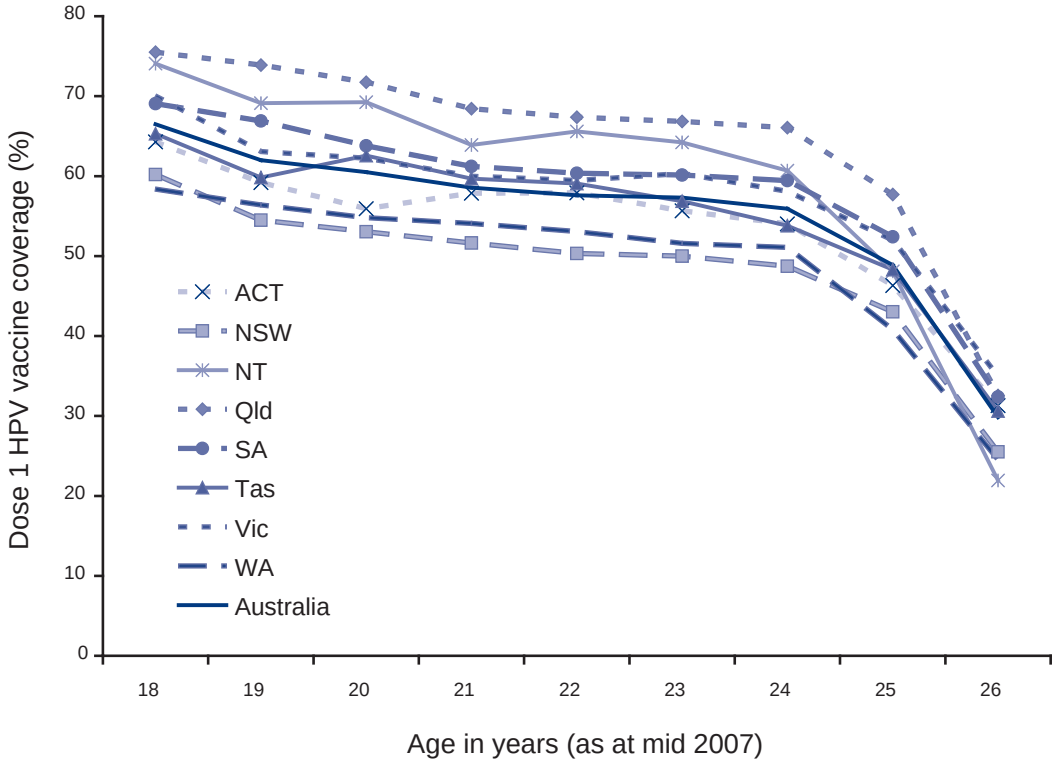
* The catch up component of the National HPV Vaccination Program concluded on 31 December 2009. Notification of doses to the National HPV Vaccination Program Register (NHVPR) is ongoing. Excludes consumers who do not wish their details to be recorded on the Register.

Discussion

Based on these estimates from HPV vaccine doses notified to the NHVPR, vaccine coverage achieved in Australian young adult women is higher than was anticipated based on previous efforts to vaccinate this age group (e.g. young adult measles-mumps-rubella vaccine campaign) and Australia has achieved the highest coverage in young adult women in the world to date. It is estimated that around 60% of all Australian women 18–26 years of age received at least 1 HPV vaccine dose, based on estimates from those states and territories with central notification of administered doses.

The Northern Territory, Queensland and South Australia, the jurisdictions with centralised mechanisms for notification of vaccine doses, have the highest reported HPV vaccine coverage estimates to date (10%–20% higher than in other states and territories). Because promotion of the program was conducted at a national level, it is likely that this difference reflects relative under-notification from other states and territories rather than truly higher coverage in these areas. This relative under-notification is apparent despite the very high number of GPs who registered with the NHVPR to notify. A national study of the Divisions of General Practice Immunisation Coordinators conducted in 2010

Figure: Notified dose 1 human papillomavirus vaccine coverage in women aged 18–26 years on the National HPV Vaccination Program Register, Australia, as at 22 March 2011,* by state or territory of residence



* Excludes consumers who do not wish their details to be recorded on the Register.

found that 50% thought that more than 90% GPs in their Division had notified but that several felt that reporting rates were closer to 50%.[8] A particular challenge for providers was the requirement to retain data and notify doses previously administered, as the NHVPR was not operational at the time of program commencement. Some providers did not initially gain consent for the notification of administered doses to the NHVPR, delaying or preventing subsequent reporting. Concerns about consent gained traction in some states more than others and may explain why New South Wales has apparently lower HPV vaccination uptake. [9] This is supported by NHVPR records which show that 48% of all consent-based queries received by the telephone information service requiring follow up were from New South Wales GPs.

Calculating the percentage of notified doses against dose distribution data for the period 2007–2009 by state and territory shows a range by jurisdiction of 74% to 86% (data not shown, in-confidence). In reconciling these data it was important to account for the number of doses purchased but not yet distributed by the end of 2009, which differed substantially by jurisdiction: these doses were used for the on-going program. As accurate HPV vaccine coverage data are essential for monitoring and evaluating the program, as well as for the future records of women and their doctors, an estimate of the extent of under-notification is vital to adequately adjust for this factor in future assessments of vaccine effectiveness: failure to do so will bias estimates towards the null (i.e. no effect) when outcomes are compared in vaccinated vs apparently unvaccinated women. As well as comparisons against dose distribution data, independent population based coverage estimates are important to accurately assess the extent of non-notification. A population based computer-assisted telephone interview (CATI) in Victoria in May 2009 found coverage of 74%, 69%, and 56% for doses 1, 2, and 3, respectively, among 234 women aged 16–26 years in 2007[10] and a national survey of Year 10 and 12 school girls in 2009 found that 86% reported receipt of at least 1 dose.[11] The Victorian Cytology Service, operator of the NHVPR, is currently undertaking a national CATI survey, in conjunction with the Kirby Institute, to provide independent national estimates of HPV vaccine coverage in young adult women by age and State. Because the survey relies on self reporting, a validation sub-study will compare self-reported doses against the NHVPR and where necessary, provider's records.

All immunisers are strongly encouraged to notify the NHVPR of all HPV vaccinations to make the information on the Register as complete as possible. This

will not only ensure accurate vaccination records are available for women and their health care providers in the future but also improve the quality of data available to measure the impact of Australia's world leading HPV vaccination program.

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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 60,971 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January and 31 March 2011 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions except South Australia
Syphilis - congenital	All jurisdictions

Table 1: Reporting of notifiable diseases by jurisdiction, *continued*

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2011, by date of diagnosis

Disease	State or territory								Total 1st quarter 2011	Total 4th quarter 2010	Total 1st quarter 2010	Last 5 years mean 1st quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Hepatitis B (newly acquired)*	1	9	2	12	3	2	15	3	47	36	73	65.2	0.7	47	65.2
Hepatitis B (unspecified)†	20	714	50	247	79	7	444	157	1,718	1,648	1,962	1,718.8	1.0	1,718	1,718.8
Hepatitis C (newly acquired)*,‡	1	15	1	NN	7	7	47	29	107	80	100	99.0	1.1	107	99.0
Hepatitis C (unspecified)†	43	876	47	600	106	60	597	252	2,581	2,716	3,029	3,005.2	0.9	2,581	3,005.2
Hepatitis D	0	3	0	3	0	0	4	0	10	8	4	9.2	1.1	10	9.2
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.4
Campylobacteriosis§	149	NN	45	1,382	500	216	1,953	641	4,886	5,247	4,234	4,312.8	1.1	4,886	4,312.8
Cryptosporidiosis	9	104	30	115	57	3	46	262	626	297	525	1,356.0	0.5	626	1,356.0
Haemolytic uraemic syndrome	0	2	1	1	0	0	0	0	4	1	3	5.2	0.8	4	5.2
Hepatitis A	1	23	0	3	1	1	13	2	44	49	101	81.4	0.5	44	81.4
Hepatitis E	1	8	0	3	0	0	4	0	16	6	12	11.0	1.5	16	11.0
Listeriosis	0	6	1	3	2	1	4	2	19	16	34	26.4	0.7	19	26.4
STEC, VTEC‖	0	0	0	4	7	0	6	0	17	16	33	33.8	0.5	17	33.8
Salmonellosis	65	1,560	116	1,132	403	74	981	443	4,774	2,873	4,097	3,386.4	1.4	4,774	3,386.4
Shigellosis	4	47	24	20	7	0	25	37	164	128	165	190.4	0.9	164	190.4
Typhoid	1	25	0	8	4	1	11	6	56	17	31	32.4	1.7	56	32.4
Quarantinable diseases															
Cholera	0	0	0	1	0	0	0	0	1	0	0	0.6	1.7	1	0.6
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2011, by date of diagnosis

Disease	State or territory								Total 1st quarter 2011	Total 4th quarter 2010	Total 1st quarter 2010	Last 5 years mean 1st quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[¶,**]	346	5,267	679	4,872	1,289	435	4,775	2,943	20,606	17,635	19,073	14,977.4	1.4	20,606	14,977.4
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	0	0.6
Gonococcal infection**	45	615	521	722	135	3	457	435	2,933	2,521	2,376	2,184.8	1.3	2,933	2,184.8
Syphilis <2 years duration**	2	92	16	88	4	0	78	34	314	250	314	303.2	1.0	314	303.2
Syphilis >2 years or unspecified duration**	9	64	13	48	NDP	5	150	26	315	256	330	332.6	0.9	315	332.6
Syphilis – congenital**	0	2	0	2	0	0	0	0	4	0	1	1.4	2.9	4	1.4
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Haemophilus influenzae type b	0	2	0	1	0	0	0	0	3	8	3	4.0	0.8	3	4.0
Influenza (laboratory confirmed)	17	350	306	1,232	175	9	341	176	2,606	3,843	468	381.6	6.8	2,606	381.6
Measles	2	46	0	10	0	0	20	3	81	10	14	29.2	2.8	81	29.2
Mumps	0	13	0	9	3	0	11	2	38	29	19	61.2	0.6	38	61.2
Pertussis	347	3,598	53	2,388	951	63	2,773	425	10,598	14,155	5,679	3,742.4	2.8	10,598	3,742.4
Pneumococcal disease (invasive)	4	63	23	29	23	7	37	33	219	390	211	203.2	1.1	219	203.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	1	7	0	5	1	0	6	2	22	4	16	8.8	2.5	22	8.8
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Tetanus	0	1	0	0	0	0	0	0	1	0	1	1.6	0.6	1	1.6
Varicella zoster (chickenpox)[††]	3	NN	14	43	105	9	118	78	370	551	279	294.0	1.3	370	294.0
Varicella zoster (shingles)[††]	5	NN	45	25	390	40	272	208	985	812	781	536.6	1.8	985	536.6
Varicella zoster (unspecified)[††]	18	NN	0	994	29	19	492	239	1,791	1,872	1,772	1,281.0	1.4	1,791	1,281.0
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	0	0	0	4	0	4	15	4	6.8	0.6	4	6.8
Barmah Forest virus infection	2	233	16	326	74	2	129	53	835	406	454	604.2	1.4	835	604.2
Dengue virus infection	3	35	13	110	12	0	37	148	358	449	161	295.6	1.2	358	295.6
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Kunjin virus infection[‡‡]	0	0	0	0	0	0	0	0	0	0	1	0.8	0.0	0	0.8
Malaria	0	19	8	41	0	2	28	20	118	99	94	142.8	0.8	118	142.8
Murray Valley encephalitis virus infection[‡‡]	0	1	2	0	2	0	0	3	8	0	0	0.6	13.3	8	0.6
Ross River virus infection	5	277	80	453	805	4	1,042	372	3,038	1,031	1,653	2,123.4	1.4	3,038	2,123.4

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2011, by date of diagnosis

Disease	State or territory								Total 1st quarter 2011	Total 4th quarter 2010	Total 1st quarter 2010	Last 5 years mean 1st quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	1	0.6	0.0	0	0.6
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	2	0	8	0	0	1	0	11	5	5	9.8	1.1	11	9.8
Leptospirosis	0	12	1	99	0	0	2	1	115	36	20	46.0	2.5	115	46.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	3	0	0	0	0	18	1	22	28	11	23.8	0.9	22	23.8
Q fever	0	17	0	46	1	0	3	2	69	75	77	99.2	0.7	69	99.2
Tularaemia	0	0	0	0	0	1	0	0	1	0	0	0.0	0.0	1	0.0
Other bacterial infections															
Legionellosis	0	23	3	15	10	2	18	8	79	81	61	72.8	1.1	79	72.8
Leprosy	0	0	0	0	0	0	0	0	0	2	3	2.6	0.0	0	2.6
Meningococcal infection§§	1	22	3	13	5	2	8	4	58	53	46	51.0	1.1	58	51.0
Tuberculosis	6	87	6	72	13	3	84	28	299	375	293	286.0	1.0	299	286.0
Total	**1,111**	**14,449**	**2,119**	**15,430**	**5,276**	**1,000**	**15,058**	**7,131**	**60,971**	**58,129**	**48,624**			**60,971**	

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

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

‡ In Queensland, includes incident hepatitis cases.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‖ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens. The Northern Territory and Western Australia, exclude ocular infections.

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Ratio of current quarter total to the mean of last 5 years for the same quarter. Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 4 years of data.

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

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Table 3: Notification rates of diseases, 1 January to 31 March 2011, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	1.1	0.5	3.6	1.1	0.7	1.6	1.1	0.5	0.9
Hepatitis B (unspecified)†	22.8	40.4	89.3	22.4	19.7	5.7	32.8	28.0	31.5
Hepatitis C (newly acquired)*	1.1	0.8	1.8	NN	1.7	5.7	3.5	5.2	2.5
Hepatitis C (unspecified)†,‡	49.1	49.6	83.9	54.5	26.4	48.5	44.1	45.0	47.4
Hepatitis D	0.0	0.2	0.0	0.3	0.0	0.0	0.3	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	170.2	NN	80.3	125.5	124.6	174.5	144.3	114.4	132.6
Cryptosporidiosis	10.3	5.9	53.6	10.4	14.2	2.4	3.4	46.8	11.5
Haemolytic uraemic syndrome	0.0	0.1	1.8	0.1	0.0	0.0	0.0	0.0	0.1
Hepatitis A	1.1	1.3	0.0	0.3	0.2	0.8	1.0	0.4	0.8
Hepatitis E	1.1	0.5	0.0	0.3	0.0	0.0	0.3	0.0	0.3
Listeriosis	0.0	0.3	1.8	0.3	0.5	0.8	0.3	0.4	0.3
STEC,VTEC‖	0.0	0.0	0.0	0.4	1.7	0.0	0.4	0.0	0.3
Salmonellosis	74.3	88.4	207.1	102.8	100.5	59.8	72.5	79.1	87.6
Shigellosis	4.6	2.7	42.8	1.8	1.7	0.0	1.8	6.6	3.0
Typhoid fever	1.1	1.4	0.0	0.7	1.0	0.8	0.8	1.1	1.0
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Human 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¶,**	395.3	298.3	1,212.1	442.3	321.3	351.3	352.9	525.4	378.1
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	51.4	34.8	930.1	65.5	33.7	2.4	33.8	77.7	53.8
Syphilis <2 years duration**	2.3	5.2	28.6	8.0	1.0	0.0	5.8	6.1	5.8
Syphilis >2 years or unspecified duration†,**	10.3	3.6	23.2	4.4	NDP	4.0	11.1	4.6	6.2
Syphilis – congenital**	0.0	0.1	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	19.4	19.8	546.3	111.8	43.6	7.3	25.2	31.4	47.8
Measles	2.3	2.6	0.0	0.9	0.0	0.0	1.5	0.5	1.5
Mumps	0.0	0.7	0.0	0.8	0.7	0.0	0.8	0.4	0.7
Pertussis	396.4	203.8	94.6	216.8	237.1	50.9	204.9	75.9	194.5
Pneumococcal disease (invasive)	4.6	3.6	41.1	2.6	5.7	5.7	2.7	5.9	4.0
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	1.1	0.4	0.0	0.5	0.2	0.0	0.4	0.4	0.4
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	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 January to 31 March 2011, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Varicella zoster (chickenpox)	3.4	NN	25.0	3.9	26.2	7.3	8.7	13.9	10.0
Varicella zoster (shingles)	5.7	NN	80.3	2.3	97.2	32.3	20.1	37.1	26.7
Varicella zoster (unspecified)	20.6	NN	0.0	90.2	7.2	15.3	36.4	42.7	48.6
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.1
Barmah Forest virus infection	2.3	13.2	28.6	29.6	18.4	1.6	9.5	9.5	15.3
Dengue virus infection	3.4	2.0	23.2	10.0	3.0	0.0	2.7	26.4	6.6
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection††	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	0.0	1.1	14.3	3.7	0.0	1.6	2.1	3.6	2.2
Murray Valley encephalitis virus infection††	0.0	0.1	3.6	0.0	0.5	0.0	0.0	0.5	0.1
Ross River virus infection	5.7	15.7	142.8	41.1	200.7	3.2	77.0	66.4	55.7
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.7	0.0	0.0	0.1	0.0	0.2
Leptospirosis	0.0	0.7	1.8	9.0	0.0	0.0	0.1	0.2	2.1
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.0	0.0	0.0	1.3	0.2	0.4
Q fever	0.0	1.0	0.0	4.2	0.2	0.0	0.2	0.4	1.3
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.8	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.3	5.4	1.4	2.5	1.6	1.3	1.4	1.4
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‡‡	1.1	1.2	5.4	1.2	1.2	1.6	0.6	0.7	1.1
Tuberculosis	6.9	4.9	10.7	6.5	3.2	2.4	6.2	5.0	5.5

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

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

‡ In Queensland, includes incident hepatitis C cases.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

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

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

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

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

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Laboratory Serology and Virology Reporting Scheme

There were 11,057 reports received by the Laboratory Virology and Serology Reporting Scheme (LabVISE) in the reporting period, 1 January and 31 March 2011 (Tables 4 and 5).

Table 4: Laboratory Virology and Serology reports, 1 January to 31 March 2011 and total reports for the year,* by state or territory†

	State or territory								This period 2011	This period 2010	Year to date 2011	Year to date 2010
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	11	–	–	–	–	5	1	17	8	17	8
Mumps virus	–	–	–	2	1	–	1	1	5	6	5	6
Rubella virus	–	–	–	–	–	–	–	4	4	9	4	9
Hepatitis viruses												
Hepatitis A virus	–	1	–	3	1	1	–	2	8	16	8	16
Hepatitis D virus	–	1	–	–	1	–	–	–	2	4	2	4
Hepatitis E virus	–	–	–	1	–	–	–	–	1	1	1	1
Arboviruses												
Ross River virus	–	3	12	113	404	–	4	42	578	354	578	354
Barmah Forest virus	–	10	–	67	44	–	–	6	127	69	127	69
Alphavirus (unspecified)	–	–	1	–	–	–	–	1	2	–	2	–
Dengue type 1	–	–	5	–	–	–	–	37	42	–	42	–
Dengue type 2	–	–	–	–	–	–	–	16	16	–	16	–
Dengue type 3	–	–	2	–	–	–	–	28	30	–	30	–
Dengue type 4	–	–	1	–	–	–	–	10	11	–	11	–
Dengue not typed	–	–	10	–	–	–	–	111	121	–	121	–
Flavivirus (unspecified)	–	6	–	28	–	–	5	–	39	59	39	59
Adenoviruses												
Adenovirus type 40	–	–	–	–	–	–	–	2	2	–	2	–
Adenovirus type 41	–	–	–	–	–	–	–	17	17	–	17	–
Adenovirus not typed/ pending	2	94	10	109	258	1	2	62	538	226	538	226
Herpesviruses												
Herpes virus type 6	–	2	–	–	–	–	–	1	3	1	3	1
Cytomegalovirus	–	73	–	108	98	3	6	–	288	394	288	394
Varicella-zoster virus	2	62	–	483	161	1	21	96	826	815	826	815
Epstein-Barr virus	–	16	32	302	152	1	6	119	628	866	628	866
Other DNA viruses												
Molluscum contagiosum	–	–	–	–	–	–	–	14	14	–	14	–
Parvovirus	–	1	–	28	16	1	16	6	68	73	68	73
Picornavirus family												
Rhinovirus (all types)	2	114	–	–	409	–	–	21	546	61	546	61
Enterovirus type 71 (BCR)	–	1	–	–	–	–	–	–	1	–	1	–
Enterovirus not typed/ pending	–	35	–	–	1	1	–	6	43	25	43	25
Picornavirus not typed	–	–	7	–	–	1	–	39	47	2	47	2
Ortho/paramyxoviruses												
Influenza A virus	6	40	8	132	99	–	11	38	334	149	334	149
Influenza A virus H3N2	–	–	56	1	–	–	–	28	85	–	85	–
Influenza B virus	–	8	4	21	28	–	3	12	76	25	76	25
Parainfluenza virus type 1	–	5	–	1	1	–	–	1	8	70	8	70
Parainfluenza virus type 2	–	3	–	2	7	–	–	–	12	18	12	18
Parainfluenza virus type 3	–	30	2	16	61	–	–	46	155	34	155	34

Table 4 *continued*: Laboratory Virology and Serology reports, 1 January to 31 March 2011, and total reports for the year,* by state or territory†

	State or territory								This period 2011	This period 2010	Year to date 2011	Year to date 2010
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses, *continued*												
Respiratory syncytial virus	–	103	4	113	31	–	–	33	284	265	284	265
Paramyxovirus (unspecified)	–	10	–	–	–	–	–	–	10	–	10	–
Other RNA viruses												
HTLV-1	–	–	–	1	10	–	–	–	11	17	11	17
Rotavirus	–	14	–	–	7	–	–	11	32	18	32	18
Calicivirus	–	–	–	–	–	–	–	105	105	–	105	–
Norwalk agent	2	30	–	–	111	–	–	–	143	305	143	305
Other												
Chlamydia trachomatis not typed	5	357	4	1,644	532	17	4	451	3,014	3,145	3,014	3,145
Chlamydia pneumoniae	–	–	–	–	–	–	2	–	2	29	2	29
Chlamydia psittaci	–	–	–	1	–	–	13	–	14	8	14	8
Chlamydia spp typing pending	–	15	–	–	–	–	–	–	15	6	15	6
Chlamydia species	–	–	–	–	–	–	1	–	1	1	1	1
Mycoplasma pneumoniae	–	13	7	80	138	2	44	64	348	276	348	276
Mycoplasma hominis	–	4	–	–	–	–	–	–	4	–	4	–
Coxiella burnetii (Q fever)	1	7	–	9	5	–	6	1	29	51	29	51
Rickettsia prowazeki	–	–	–	–	1	–	–	–	1	1	1	1
Orientia tsutsuganushi	–	1	–	1	–	–	–	–	2	–	2	–
Rickettsia - spotted fever group	–	8	–	15	4	2	4	–	33	16	33	16
Rickettsia spp - other	–	1	–	–	–	1	3	1	6	–	6	–
Streptococcus group A	–	11	–	131	–	–	25	–	167	176	167	176
Brucella species	–	–	–	20	–	–	–	–	20	3	20	3
Bordetella pertussis	4	96	–	676	663	1	111	100	1,651	1,573	1,651	1,573
Legionella pneumophila	1	2	1	5	1	1	2	4	17	4	17	4
Legionella longbeachae	–	–	–	–	1	–	–	4	5	5	5	5
Legionella species	1	4	–	9	–	–	1	3	18	10	18	10
Cryptococcus species	–	–	–	2	5	–	–	–	7	15	7	15
Leptospira species	–	–	1	30	3	–	–	1	35	15	35	15
Treponema pallidum	1	48	2	194	83	–	30	15	373	528	373	528
Entamoeba histolytica	–	–	–	–	1	–	–	–	1	3	1	3
Toxoplasma gondii	–	3	–	1	4	2	1	1	12	5	12	5
Echinococcus granulosus	–	–	–	–	3	–	–	–	3	1	3	1
Total	**27**	**1,243**	**169**	**4,349**	**3,345**	**36**	**327**	**1,561**	**11,057**	**9,761**	**11,057**	**9,761**

* Data presented are for reports with report dates in the current period.

† State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

– No data received this period.

Table 5: Laboratory Virology and Serology reports, 1 January to 31 March 2011,* by laboratory

State or territory	Laboratory	January 2011	February 2011	March 2011	Total
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	141	258	276	675
	New Children's Hospital, Westmead	48	53	76	177
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	37	24	62	123
Queensland	Queensland Medical Laboratory, West End	1,785	1,834	1,039	4,658
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	1,734	1,603	–	3,337
Tasmania	Northern Tasmanian Pathology Service, Launceston	14	10	4	28
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	43	1	–	44
	Monash Medical Centre, Melbourne	–	–	–	–
	Royal Children's Hospital, Melbourne	107	38	3	148
	Victorian Infectious Diseases Reference Laboratory	68	–	64	132
Western Australia	PathWest Virology, Perth	742	793	27	1,562
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	43	57	73	173
Total		4,762	4,671	1,624	11,057

* The complete list of laboratories reporting for the 12 months, January to December 2011, 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 Practices Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Commonwealth's Department of Health and Ageing, owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic, web-based data collection was established in 2006.

In June 2010, ASPREN's laboratory ILI testing was implemented, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and H1N1(2009).

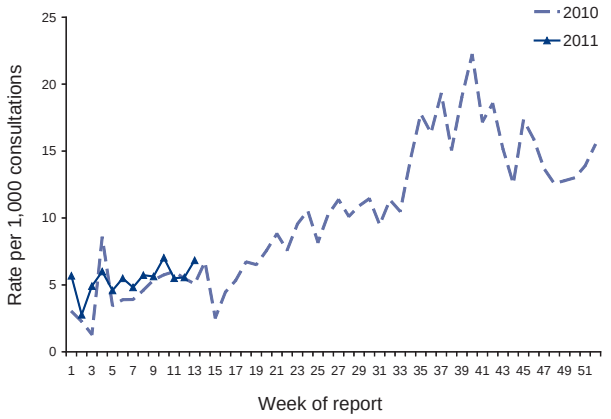
The list of conditions is reviewed annually by the ASPREN management committee. In 2011, 4 conditions are being monitored. They include influenza-like illness (ILI), gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2011;35(1):57–58.*

Reporting period 1 January to 31 March 2011

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 102 general practitioners contributed data to ASPREN in the 1st quarter of 2011. Each week an average of 94 general practitioners provided information to ASPREN at an average of 8,125 (range 3,872–9,362) consultations per week and an average of 105 (range 60–133) notifications per week.

ILI rates reported from 1 January to 31 March 2011 averaged 5 cases per 1,000 consultations (range 3–7 cases per 1,000 consultations). The reported rates in January, February and March 2011 (3–6 cases per 1,000 consultations, 5–6 cases per 1,000 consultations and 6–7 cases per 1,000 consultations respectively) were relatively consistent compared with rates in the same reporting period in 2010 (1–9 cases per 1,000 consultations, 4–5 cases per 1,000 consultations and 5–6 cases per 1,000 consultations, respectively).

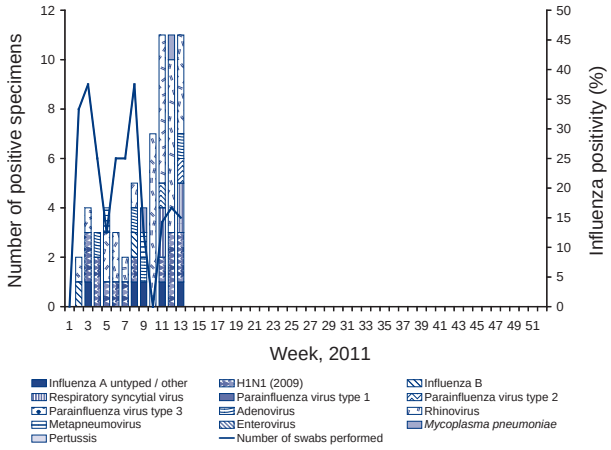
Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2010 to 31 March 2011, by week of report



ILI swab testing has continued through 2011. The most commonly reported virus during this reporting period was rhinovirus (27% of all swabs performed), with the second most common virus being influenza A H1N1(2009) (12% of all swabs performed).

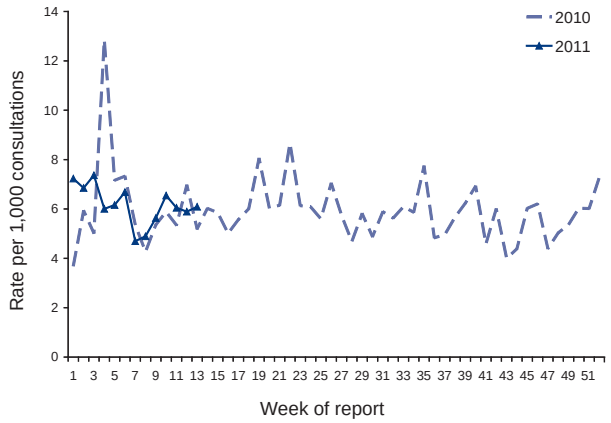
From the beginning of 2011 to the end of week 13, 21 cases of influenza have been detected, the majority of these being H1N1(2009) (12% of all swabs performed) and the remainder were influenza A untyped or other (4%) and influenza B (2%) (Figure 2).

Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January 2010 to 31 March 2011, by week of report



During this reporting period, consultation rates for gastroenteritis averaged 6 cases per 1,000 consultations (range 5–7 cases per 1000, Figure 3). This was relatively consistent compared with rates in the same reporting period in 2010 where the average was 6 cases per 1,000 consultations (range 4–13 cases per 1,000).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2010 to 31 March 2011, by week of report



Varicella infections were reported at a slightly higher rate for the 1st quarter of 2011 compared with the same period in 2010. From 1 January to 31 March 2011, recorded rates for chickenpox averaged 0.3 cases per 1,000 consultations (range 0.1 to 0.8 cases per 1,000 consultations, Figure 4).

In the 1st quarter of 2011, reported rates for shingles averaged 1.1 cases per 1,000 consultations (range 0.4–1.8 cases per 1,000 consultations, Figure 5), slightly higher than the same reporting period in 2010 where the average shingles rate was 0.9 cases per 1,000 consultations (0.2–1.9 cases per 1,000 consultations).

Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2010 to 31 March 2011, by week of report

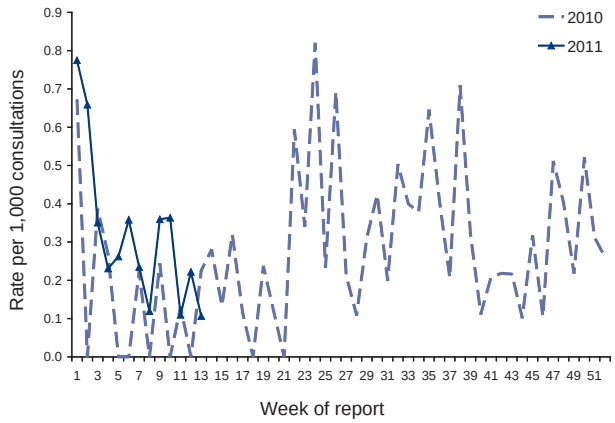
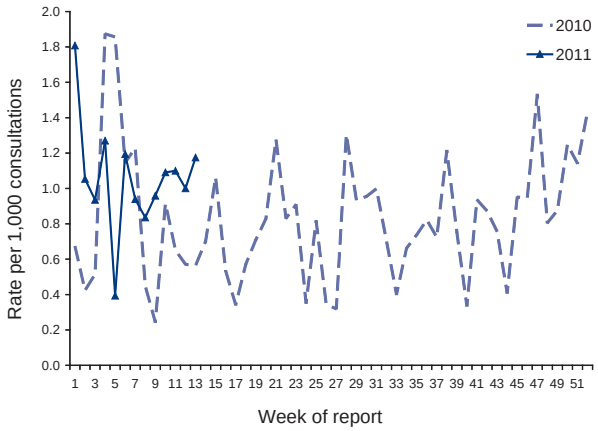


Figure 5: Consultation rates for shingles, ASPREN, 1 January 2010 to 31 March 2011, by week of report



Meningococcal surveillance

(Dr Monica M Lahra, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme)

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where Neisseria meningitidis *is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays (NAA) and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the numbers of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the annual reports of the Programme is published in Communicable Diseases Intelligence. For more information see* Commun Dis Intell *2011;35(1):57.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2011, are included in this issue of Communicable Diseases Intelligence *(Table 1).*

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2011, 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	11			3	3	0	0	0	0	0	0	0	0	3	3
	10			0	0	0	0	0	0	0	0	0	0	0	0
New South Wales	11			10	10	0	0	3	3	1	1	3	3	17	17
	10			13	13	0	0	0	0	1	1	1	1	15	15
Northern Territory	11			0	0	0	0	0	0	0	0	0	0	0	0
	10			0	0	0	0	0	0	0	0	0	0	0	0
Queensland	11			8	8	1	1	1	1	0	0	0	0	10	10
	10			6	6	0	0	0	0	0	0	0	0	6	6
South Australia	11			3	3	0	0	0	0	1	1	0	0	4	4
	10			4	4	0	0	1	1	0	0	0	0	5	5
Tasmania	11			0	0	1	1	0	0	1	1	0	0	2	2
	10			0	0	0	0	0	0	0	0	0	0	0	0
Victoria	11			10	10	0	0	0	0	0	0	0	0	10	10
	10			3	3	0	0	1	1	1	1	0	0	5	5
Western Australia	11			4	4	0	0	0	0	0	0	0	0	4	4
	10			2	2	1	1	0	0	0	0	0	0	3	3
Total	11			38	38	2	1	4	4	3	3	3	3	50	50
	10			28	28	1	1	2	2	2	2	1	1	34	34

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the Kirby Institute (formerly the National Centre in HIV Epidemiology and Clinical Research), in collaboration with state and territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the Kirby Institute, CFI Building, Cnr Boundary and West Streets, Darlinghurst NSW 2010. Internet: http://hiv.cms.med.unsw.edu.au/ Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see Commun Dis Intell *2011;35(1):58.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2010, and 1 April to 30 June 2010, are included in this issue of Communicable Diseases Intelligence *(Tables 1, 2, 3 and 4).*

Table 1: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 January to 31 March 2010, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2010	This period 2009	YTD 2010	YTD 2009
HIV diagnoses	Female	0	8	0	19	2	0	7	0	36	41	36	41
	Male	0	87	4	48	12	0	55	5	211	211	211	211
	Not reported	0	1	0	0	0	0	2	0	3	0	3	0
	Total*	0	97	4	67	14	0	64	5	251	252	251	252
AIDS diagnoses†	Female	0	–	0	1	0	0	1	0	2	6	2	6
	Male	0	–	2	3	1	0	8	0	14	21	14	21
	Total*	0	–	2	4	1	0	9	0	16	27	16	27
AIDS deaths†	Female	0	–	0	0	0	0	0	0	0	0	0	0
	Male	0	–	0	1	1	0	3	0	5	3	5	3
	Total*	0	–	0	1	1	0	3	0	5	3	5	3

* Totals include people whose sex was reported as transgender.

† AIDS cases and deaths following AIDS occurring in New South Wales from January 2008 are not included.

Table 2: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 31 March 2010, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	37	1,018	30	374	127	17	472	266	2,341
	Male	283	14,573	162	3,299	1,078	127	6,067	1,405	26,994
	Not reported	0	229	0	0	0	0	22	0	251
	Total*	320	15,853	192	3,682	1,206	144	6,585	1,678	29,660
AIDS diagnoses†	Female	10	265	6	78	32	4	127	48	570
	Male	95	5,513	50	1,101	427	55	2,162	458	9,861
	Total*	105	5,796	56	1,181	460	59	2,302	508	10,467
AIDS deaths†	Female	7	138	1	43	20	2	66	30	307
	Male	73	3,597	33	682	281	34	1,452	301	6,453
	Total*	80	3,746	34	727	301	36	1,527	332	6,783

* Totals include people whose sex was reported as transgender.

† AIDS cases and deaths following AIDS occurring in New South Wales from January 2008 are not included.

Table 3: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2010, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2010	This period 2009	YTD 2010	YTD 2009
HIV diagnoses	Female	1	5	0	7	2	0	9	8	32	35	71	76
	Male	4	73	0	52	10	0	68	21	228	238	453	450
	Not reported	0	0	0	0	0	0	0	0	0	1	1	1
	Total*	5	78	0	59	12	0	77	29	260	274	528	527
AIDS diagnoses†	Female	0	–	0	0	0	0	0	1	1	2	3	8
	Male	0	–	0	5	0	0	9	1	15	23	30	44
	Total*	0	–	0	5	0	0	9	2	16	25	33	52
AIDS deaths†	Female	0	–	0	1	0	0	0	0	1	1	1	1
	Male	0	–	0	1	0	0	2	0	3	2	8	5
	Total*	0	–	0	2	0	0	2	0	4	3	9	6

* Totals include people whose sex was reported as transgender.

† AIDS cases and deaths following AIDS occurring in New South Wales from January 2008 are not included.

Table 4: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 30 June 2010, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	38	1,025	30	381	129	17	481	281	2,382
	Male	287	14,656	162	3,349	1,088	127	6,136	1,443	27,248
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	325	15,942	192	3,739	1,218	144	6,663	1,731	29,954
AIDS diagnoses†	Female	10	265	6	78	32	4	127	49	571
	Male	95	5,513	50	1,106	427	55	2,171	461	9,878
	Total*	105	5,796	56	1,186	460	59	2,311	512	10,485
AIDS deaths†	Female	7	138	1	44	20	2	66	30	308
	Male	73	3,597	33	683	281	34	1,454	301	6,456
	Total*	80	3,746	34	729	301	36	1,529	332	6,787

* Totals include people whose sex was reported as transgender.

† AIDS cases and deaths following AIDS occurring in New South Wales from January 2008 are not included.