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Annual reports

Australia's notifiable disease status, 2010: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

In 2010, 65 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 209,079 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, a decrease of 12% on the number of notifications in 2009. This decrease was largely due to a reduction of influenza compared with the influenza A(H1N1) pandemic 2009. In 2010, the most frequently notified diseases were sexually transmissible infections (86,620 notifications, 41.4% of total notifications), vaccine preventable diseases (61,964 notifications, 29.6% of total notifications), and gastrointestinal diseases (31,548 notifications, 15.1% of total notifications). There were 18,302 notifications of bloodborne diseases; 8,244 notifications of vectorborne diseases; 1,866 notifications of other bacterial infections; 532 notifications of zoonoses and 3 notifications of quarantinable diseases. *Commun Dis Intell* 2012;35(1):1–69.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2010, 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 respective public health legislation. In September 2007, the *National Health Security Act 2007*[1] received royal assent. This Act provides a legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List,[2] which specifies the diseases about which personal information can be provided. The *National Health Security Agreement,*[3] which was drafted in 2007 and signed by Health Ministers in April 2008, establishes operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. Under the Agreement, in 2010 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.

In 2010, the National Notifiable Diseases Surveillance System (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 central agency in the jurisdiction 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 B, 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 2010 and were forwarded to the Kirby Institute for infection and immunity in society. 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 2010 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 the direct method of standardisation, with 2006 census data as the standard population. All rates are represented as the rate per 100,000 population unless stated otherwise.

The 2 maps produced for this report (chlamydia and pertussis) were created with ArcGIS mapping software (ESRI, Redlands, CA) and based on the NNDSS notifications' residential postcode. Notifications were summed by the postcode weighting calculated by the ABS Postcode Concordance.[8] These ABS concordance data were used to proportionally allocate notifications into SDs/SSDs according to the percentage of the population of the postcode living in the region. The total notifications per region are displayed in the relevant area.

With one exception, all jurisdictions in the Australian map consist of Statistical Divisions (SD) as defined by the Australian Standard Geographical Classification (Map 1, Table 1). 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 SSDs. This combination helps preserve confidentiality while improving legibility at the scale of the maps to be printed. The geocode 77777 for Greater Darwin is only nominal.

Disease rates were calculated per 100,000 population for the relevant areas using ABS population data.[7] Rates were mapped for different SDs and ordered into 5 groups using the Jenks Natural Breaks method 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 6 rate classes per map. Note that the classification is data dependent and changes from map to map.

Notes on interpretation

The present report is based on 2010 'finalised' data from each state or territory agreed upon in June 2011 and represents a snap shot of the year after duplicate records and incorrect or incomplete data were removed. Therefore, totals in this report may vary slightly from the totals reported in *CDI* quarterly publications.

Analyses in this report were based on the date of disease diagnosis in an attempt to estimate disease activity within the reporting period. For the purposes of NNDSS, the date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification

date, or the notification receive date. As considerable time may have elapsed between the onset and diagnosis dates for hepatitis B (unspecified), hepatitis C (unspecified) and tuberculosis, the earliest of specimen date, health professional notification date or public health unit notification receive date was used for these conditions.

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

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] has been 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, which 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 2010 data.

Postcode information usually reflects the residential location of the case, but this does not necessarily represent the place where the disease was acquired.

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:[9]

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

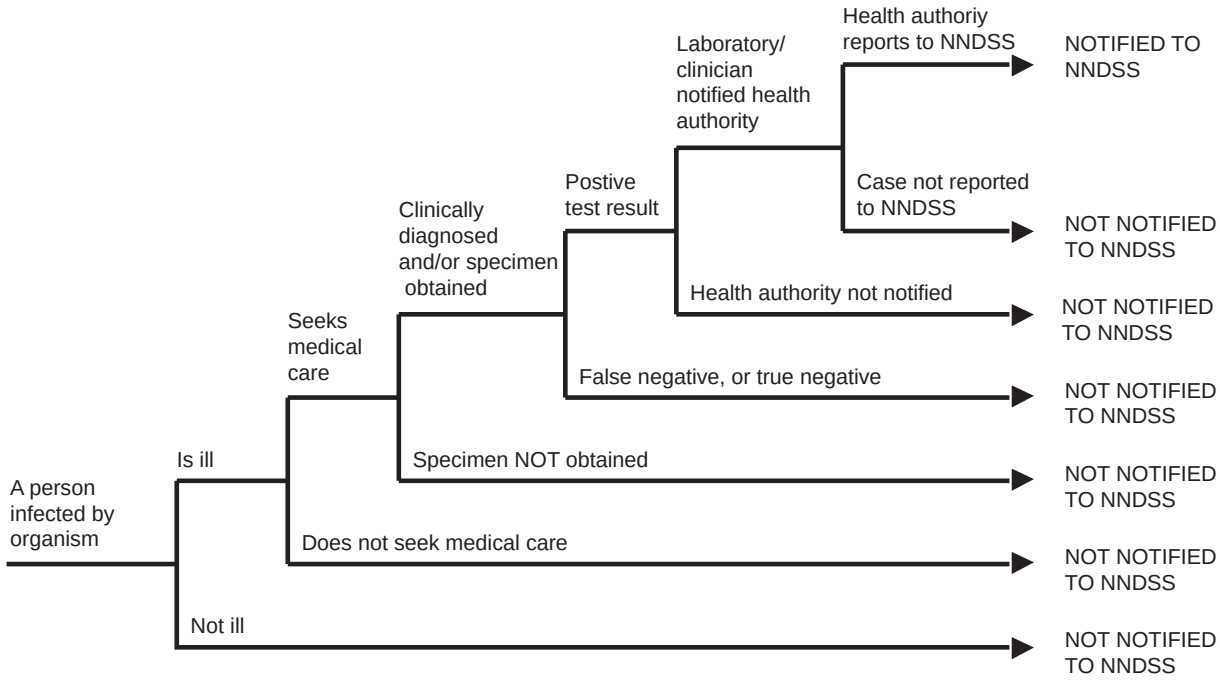
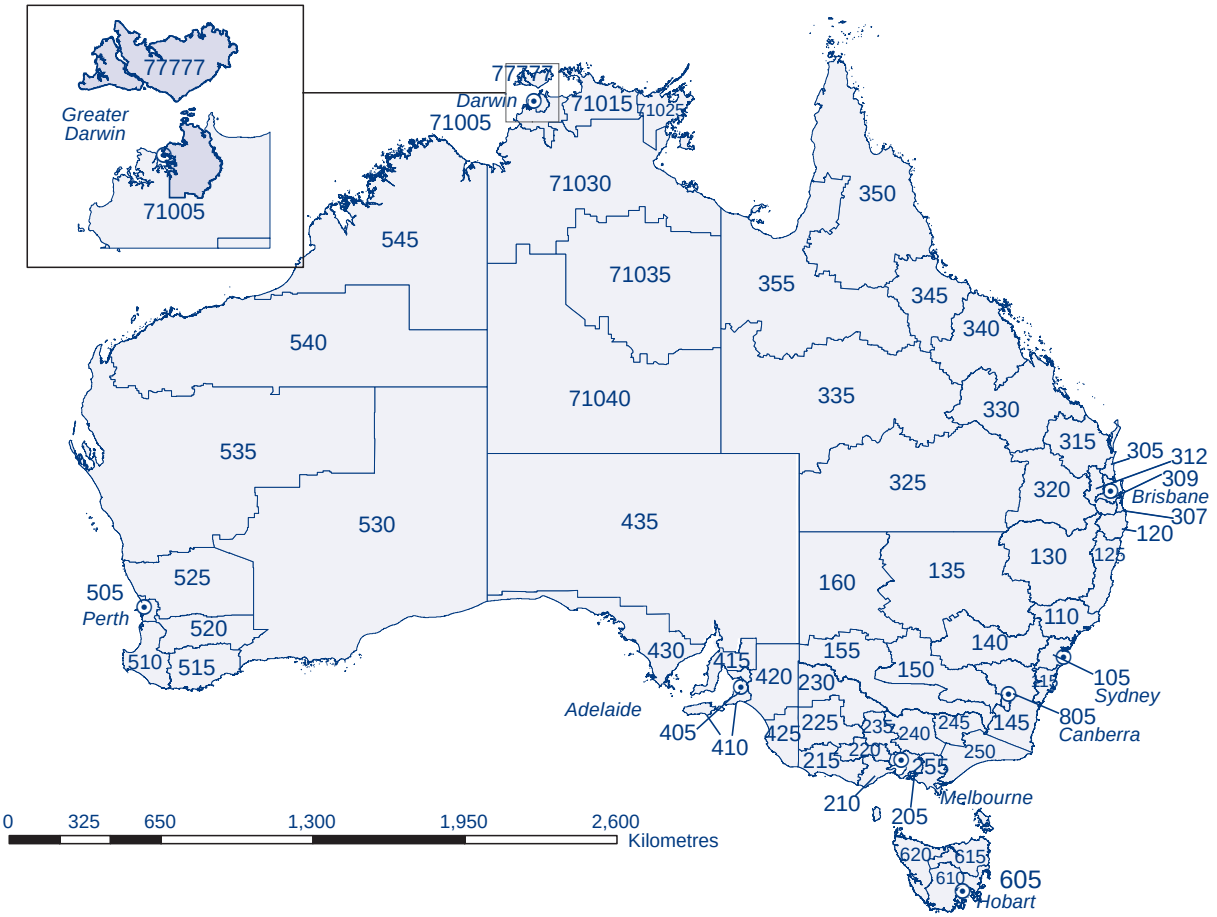


Table 1: Australian population by Statistical Division and Statistical Subdivision for the Northern Territory, 2009

SD code	Statistical Division	Population	SD code	Statistical Division	Population
Australian Capital Territory			**South Australia**		
805	Canberra	351,868	405	Adelaide	1,203,186
810	ACT balance	Included above	410	Outer Adelaide	139,489
New South Wales			415	Yorke and Lower North	47,585
105	Sydney	4,575,532	420	Murray Lands	70,705
110	Hunter	651,622	425	South East	66,724
115	Illawarra	436,117	430	Eyre	35,892
120	Richmond–Tweed	244,085	435	Northern	81,001
125	Mid-North Coast	313,322	**Tasmania**		
130	Northern	186,496	605	Greater Hobart	214,705
135	North Western	119,329	610	Southern	37,838
140	Central West	184,921	615	Northern	142,311
145	South Eastern	219,655	620	Mersey–Lyell	112,789
150	Murrumbidgee	159,624	**Victoria**		
155	Murray	119,302	205	Melbourne	4,077,036
160	Far West	22,584	210	Barwon	290,277
Northern Territory (Subdivisions)			215	Western District	107,072
71005	Finniss	2,906	220	Central Highlands	158,627
71015	Alligator	6,908	225	Wimmera	50,903
71025	East Arnhem	16,252	230	Mallee	95,213
71030	Lower Top End NT	24,170	235	Loddon	186,201
71035	Barkly	8,137	240	Goulburn	212,799
71040	Central NT	41,272	245	Ovens–Murray	101,086
77777	Greater Darwin	130,066	250	East Gippsland	87,872
Queensland			255	Gippsland	178,846
305	Brisbane	2,043,185	**Western Australia**		
307	Gold Coast	527,828	505	Perth	1,696,065
309	Sunshine Coast	330,934	510	South West	253,512
312	West Moreton	97,414	515	Lower Great Southern	59,412
315	Wide Bay–Burnett	293,455	520	Upper Great Southern	19,100
320	Darling Downs	241,537	525	Midlands	56,435
325	South West	26,489	530	South Eastern	59,070
330	Fitzroy	223,516	535	Central	65,600
335	Central West	12,387	540	Pilbara	48,610
340	Mackay	176,236	545	Kimberley	35,706
345	Northern	231,628	**Other territories**		–
350	Far North	275,058	**Australia**	**Total**	22,326,388
355	North West	34,183			

Source: ABS 3235.0 Regional Population Growth, Australia, 4 August 2011.[8]

Map 1: Australian Bureau of Statistics Statistical Division codes, Australia, and Statistical Subdivision codes, Northern Territory, 2010



Notes on case 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) on a regular basis, or earlier if the PHLN laboratory case definitions change, relevant new evidence or guidelines emerge, or other significant issues are identified.

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

* The CDWG is a working group of the CDNA.

Results

There were 209,079 communicable disease notifications received by NNDSS in 2010 (Table 3).

In 2010, the most frequently notified diseases were sexually transmissible infections (n = 86,620, 41.4%), vaccine preventable diseases (n = 61,964, 29.6%), and gastrointestinal diseases (n = 31,548, 15.1%) (Table 3).

There were 18,302 notified cases of bloodborne diseases; 8,244 notified cases of vectorborne diseases; 1,866 notified cases of other bacterial infections; 532 notified cases of zoonoses and 3 notified cases of quarantinable diseases. There was a decrease of 12% compared with the total number of notifications in 2009 (Figure 2). This decrease was largely due to the number of cases of influenza A(H1N1) pandemic 2009 in 2009.

Table 2: Diseases notified to the National Notifiable Diseases Surveillance System, Australia 2010

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
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

Table 2 *cont'd*: Diseases notified to the National Notifiable Diseases Surveillance System, Australia 2010

Disease	Data received from
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

* Infection with Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC).
NEC Not elsewhere classified.

Notifications and notification rates per 100,000 population for each disease by state or territory, in 2010, are shown in Table 4 and Table 5 respectively. Trends in notifications and rates per 100,000 population for the period 2005 to 2010 are shown in Table 6.

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

Table 3: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2010, by disease category rank order

Disease category	Number	%
Sexually transmitted infections	86,620	41.4
Vaccine preventable diseases	61,964	29.6
Gastrointestinal diseases	31,548	15.1
Bloodborne diseases	18,302	8.8
Vectorborne diseases	8,244	3.9
Other bacterial diseases	1,866	0.9
Zoonoses	532	0.3
Quarantinable diseases	3	0.0
Total	209,079	100.0

The major changes in communicable disease notifications in 2010 are shown in Figure 3 as the ratio of notifications in 2009 to the mean number of notifications for the previous 5 years. Pertussis, gonococcal infection, chlamydial infection and salmonellosis all exceeded the expected range (5-year mean plus 2 standard deviations).

Figure 2: Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2010, by year

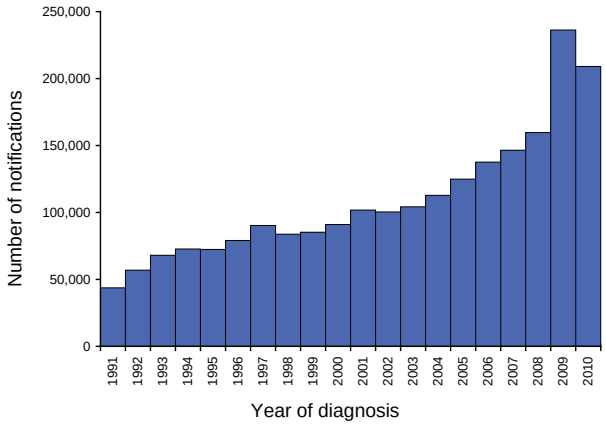


Table 4: Notifications of communicable diseases, Australia, 2010, by state or territory

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0
Hepatitis B (newly acquired)*	3	34	3	59	21	6	69	33	228
Hepatitis B (unspecified)†	93	2,432	157	1,070	409	51	1,891	775	6,878
Hepatitis C (newly acquired)*	12	36	0	NN	46	22	162	80	358
Hepatitis C (unspecified)†,‡	211	3,517	172	2,742	485	241	2,441	994	10,803
Hepatitis D	0	9	0	20	0	0	6	0	35
Gastrointestinal diseases									
Botulism	0	0	0	0	0	0	0	0	0
Campylobacteriosis§	552	NN	165	4,788	1,768	726	6,644	2,323	16,966
Cryptosporidiosis	12	349	97	302	47	100	431	142	1,480
Haemolytic uraemic syndrome	0	3	0	2	0	0	3	0	8
Hepatitis A	5	83	3	41	4	4	91	32	263
Hepatitis E	2	15	0	7	0	0	11	3	38
Listeriosis	2	26	0	9	1	3	27	3	71
Salmonellosis	212	3,822	559	2,940	665	235	2,283	1,277	11,993
Shigellosis	7	117	75	93	54	5	87	114	552
STEC,VTEC‖	0	10	0	18	33	0	12	8	81
Typhoid fever	2	31	2	20	5	1	24	11	96
Quarantinable diseases									
Cholera	0	2	0	0	0	0	0	1	3
HPAIH	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¶,**	1,157	18,278	2,662	19,216	4,330	2,008	16,474	10,180	74,305
Donovanosis	0	0	0	1	0	0	0	0	1
Gonococcal infection**	56	2,322	1,932	2,028	468	21	1,748	1,396	9,971
Syphilis – all**,††	29	746	141	404	45	21	823	155	2,364
Syphilis <2 years duration**	14	416	43	221	21	7	291	86	1,123
Syphilis >2 years or unspecified duration†,**	15	330	98	183	NDP	14	532	69	1,241
Syphilis – congenital**	0	0	0	2	0	0	0	1	3
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	6	2	7	2	0	5	2	24
Influenza (laboratory confirmed)	95	1,592	479	3,202	4,247	103	2,076	1,625	13,419
Measles	1	25	2	14	2	0	15	11	70
Mumps	1	38	2	26	1	0	12	15	95
Pertussis	712	9,288	329	8,216	7,388	281	7,131	1,448	34,793
Pneumococcal disease (invasive)	24	503	56	271	140	46	406	198	1,644
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	1	13	0	5	0	0	22	3	44
Rubella – congenital	0	0	0	0	0	0	0	0	0

Table 4 *cont'd*: Notifications of communicable diseases, Australia, 2010, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *cont'd*									
Tetanus	0	1	0	0	0	0	1	0	2
Varicella zoster (chickenpox)	4	NN	84	351	379	19	506	400	1,743
Varicella zoster (shingles)	31	NN	130	99	1,166	184	650	718	2,978
Varicella zoster (unspecified)	87	NN	3	3,894	298	81	1,912	877	7,152
Vectorborne diseases									
Arbovirus infection (NEC)	0	1	10	1	0	0	12	0	24
Barmah Forest virus infection	4	265	82	908	57	2	76	77	1,471
Dengue virus infection	15	211	42	281	23	7	119	503	1,201
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	2	124	11	126	8	5	67	56	399
Murray Valley encephalitis virus infection¶¶	0	0	0	0	0	0	0	0	0
Ross River virus infection	22	1,073	336	2,383	450	39	422	422	5,147
Zoonoses									
Anthrax	0	1	0	0	0	0	0	0	1
Australia bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	2	2	16	1	0	0	0	21
Leptospirosis	1	21	2	84	2	1	14	6	131
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	13	0	0	1	3	36	3	56
Q fever	1	136	1	151	10	0	16	8	323
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial infections									
Legionellosis	4	93	3	42	29	6	67	54	298
Leprosy	0	1	1	2	0	0	4	3	11
Meningococcal infection***	1	76	3	53	25	6	44	22	230
Tuberculosis	10	478	31	188	72	10	431	107	1,327
Total	**3,371**	**45,793**	**7,580**	**54,083**	**22,658**	**4,237**	**47,271**	**24,086**	**209,079**

* Newly acquired hepatitis and syphilis 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 or is greater than 24 months.

‡ 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.

** The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – 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 of nationally notifiable communicable diseases per 100,000 population, Australia, 2010, by state or territory

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	0.8	0.5	1.3	1.3	1.3	1.2	1.2	1.4	1.0
Hepatitis B (unspecified)†	25.9	33.6	68.4	23.7	24.9	10.0	34.1	33.7	95.0
Hepatitis C (newly acquired)*	3.3	0.5	0.0	NN	2.8	4.3	2.9	3.5	2.0
Hepatitis C (unspecified)†, ‡	58.8	48.6	74.9	60.7	29.5	47.5	44.0	43.3	149.2
Hepatitis D	0.0	0.1	0.0	0.4	0.0	0.0	0.1	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	153.8	NN	71.8	106.0	107.5	143.0	119.8	101.2	112.3
Cryptosporidiosis	3.3	4.8	42.2	6.7	2.9	19.7	7.8	6.2	6.6
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Hepatitis A	1.4	1.1	1.3	0.9	0.2	0.8	1.6	1.4	1.2
Hepatitis E	0.6	0.2	0.0	0.2	0.0	0.0	0.2	0.1	0.2
Listeriosis	0.6	0.4	0.0	0.2	0.1	0.6	0.5	0.1	0.3
Salmonellosis	59.1	52.8	243.4	65.1	40.4	46.3	41.2	55.6	53.7
Shigellosis	2.0	1.6	32.7	2.1	3.3	1.0	1.6	5.0	2.5
STEC,VTEC‖	0.0	0.1	0.0	0.4	2.0	0.0	0.2	0.3	0.4
Typhoid fever	0.6	0.4	0.9	0.4	0.3	0.2	0.4	0.5	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
HPAIH	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¶,**	322.4	252.5	1159.0	425.5	263.3	395.6	297.0	443.3	332.6
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	15.6	32.1	841.2	44.9	28.5	4.1	31.5	60.8	44.6
Syphilis – all**,††	8.1	10.3	61.4	8.9	2.7	4.1	14.8	6.7	10.6
Syphilis <2 years duration**	3.9	5.7	18.7	4.9	2.7	1.4	5.2	3.7	5.0
Syphilis >2 years or unspecified duration†,**	4.2	4.6	42.7	4.1	NDP	2.8	9.6	3.0	6.0
Syphilis – congenital**	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.9	0.2	0.1	0.0	0.1	0.1	0.1
Influenza (laboratory confirmed)	26.5	22.0	208.6	70.9	258.2	20.3	37.4	70.8	60.1
Measles	0.3	0.3	0.9	0.3	0.1	0.0	0.3	0.5	0.3
Mumps	0.3	0.5	0.9	0.6	0.1	0.0	0.2	0.7	0.4
Pertussis	198.4	128.3	143.2	181.9	449.2	55.4	128.5	63.1	155.7
Pneumococcal disease (invasive)	6.7	6.9	24.4	6.0	8.5	9.1	7.3	8.6	7.4
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.3	0.2	0.0	0.1	0.0	0.0	0.4	0.1	0.2
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 5 *cont'd*: Notification rates of nationally notifiable communicable diseases per 100,000 population, Australia, 2010, by state or territory

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *cont'd*									
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	1.1	NN	36.6	7.8	23.0	3.7	9.1	17.4	11.5
Varicella zoster (shingles)	8.6	NN	56.6	2.2	70.9	36.2	11.7	31.3	19.7
Varicella zoster (unspecified)	24.2	NN	1.3	86.2	18.1	16.0	34.5	38.2	47.4
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	4.4	0.0	0.0	0.0	0.2	0.0	0.1
Barmah Forest virus infection	1.1	3.7	35.7	20.1	3.5	0.4	1.4	3.4	6.6
Dengue virus infection	4.2	2.9	18.3	6.2	1.4	1.4	2.1	21.9	5.4
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	0.6	1.7	4.8	2.8	0.5	1.0	1.2	2.4	1.8
Murray Valley encephalitis virus infection¶¶	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	6.1	14.8	146.3	52.8	27.4	7.7	7.6	18.4	23.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.9	0.4	0.1	0.0	0.0	0.0	0.1
Leptospirosis	0.3	0.3	0.9	1.9	0.1	0.2	0.3	0.3	0.6
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	0.1	0.6	0.6	0.1	0.3
Q fever	0.3	1.9	0.4	3.3	0.6	0.0	0.3	0.3	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	0.9	1.8	1.2	1.2	2.4	1.3
Leprosy	0.0	0.0	0.4	0.0	0.0	0.0	0.1	0.1	0.0
Meningococcal infection***	0.3	1.0	1.3	1.2	1.5	1.2	0.8	1.0	1.0
Tuberculosis	2.8	6.6	13.5	4.2	4.4	2.0	7.8	4.7	5.9

* Newly acquired hepatitis and syphilis 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 or is greater than 24 months.

‡ 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.

** The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – 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, 2005 to 2010, (per 100,000 population)

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2005	2006	2007	2008	2009	2010			2005	2006	2007	2008	2009	2010
Bloodborne diseases														
Hepatitis (NEC)	1	1	0	1	0	0	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	251	292	294	256	241	228	312.4	0.7	1.2	1.4	1.4	1.2	1.1	1.0
Hepatitis B (unspecified)†	3,600	3,769	4,291	4,008	4,440	6,878	5,397.2	1.3	26.4	27.2	30.3	27.7	29.9	45.5
Hepatitis C (newly acquired)*	374	442	380	364	385	358	460.6	0.8	2.3	2.7	2.3	2.1	2.2	2.0
Hepatitis C (unspecified)†,‡	7,610	7,569	7,718	7,363	7,175	10,803	9,647.6	1.1	55.8	54.5	54.5	50.8	48.4	71.5
Hepatitis D	32	30	33	42	35	35	34.4	1.0	0.2	0.1	0.2	0.2	0.2	0.2
Gastrointestinal diseases														
Botulism	3	1	1	0	1	0	1.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	16,498	15,423	16,990	15,543	16,081	16,966	16,107.0	1.1	121.0	111.1	119.9	107.3	108.4	112.3
Cryptosporidiosis	3,213	3,201	2,810	2,003	4,626	1,480	3,170.6	0.5	15.8	15.5	13.3	9.3	21.1	6.6
Haemolytic uraemic syndrome	20	14	19	31	13	8	19.4	0.4	0.1	0.1	0.1	0.1	0.1	0.0
Hepatitis A	327	281	165	277	563	263	322.6	0.8	1.6	1.4	0.8	1.3	2.6	1.2
Hepatitis E	30	24	18	44	33	38	29.8	1.3	0.1	0.1	0.1	0.2	0.2	0.2
Listeriosis	54	61	50	68	92	71	65.0	1.1	0.3	0.3	0.2	0.3	0.4	0.3
Salmonellosis	8,422	8,251	9,534	8,333	9,586	11,993	8,825.2	1.4	41.3	39.9	45.2	38.8	43.6	53.7
Shigellosis	729	546	599	830	622	552	665.2	0.8	3.6	2.6	2.8	3.9	2.8	2.5
STEC, VTEC‖	86	70	106	107	130	81	99.8	0.8	0.4	0.3	0.5	0.5	0.6	0.4
Typhoid fever	52	77	90	105	116	96	88.0	1.1	0.3	0.4	0.4	0.5	0.5	0.4
Quarantinable diseases														
Cholera	3	3	4	4	5	3	3.8	0.8	0.0	0.0	0.0	0.0	0.0	0.0
HPAIH	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 *cont'd*: Notifications and notification rate for communicable diseases, Australia, 2005 to 2010, (per 100,000 population)

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2005	2006	2007	2008	2009	2010			2005	2006	2007	2008	2009	2010
Sexually transmissible infections														
Chlamydial infection[¶, **]	41,290	47,434	51,971	58,435	62,632	74,305	52,352.4	1.4	202.5	229.2	246.6	271.8	285.1	332.6
Donovanosis	13	6	3	2	1	1	5.0	0.2	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	8,055	8,597	7,635	7,642	7,963	9,971	7,978.4	1.2	39.5	41.5	36.2	35.5	36.3	44.6
Syphilis – all**,[††]	1,939	2,205	2,769	2,695	2,708	2,364	2,463.2	1.0	9.5	10.7	13.1	12.5	12.3	10.6
Syphilis <2 years duration**	656	890	1,418	1,325	1,310	1,099	1,119.8	1.0	3.2	4.3	6.7	6.2	6.0	5.0
Syphilis >2 years or unspecified duration**	1,283	1,315	1,351	1,370	1,398	1241	1,343.4	0.9	6.3	6.9	6.9	6.9	6.9	6.0
Syphilis – congenital**	17	11	7	6	3	3	8.8	0.3	0.1	0.1	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	0.0	0.0
Haemophilus influenzae type b	17	22	17	25	19	24	20.0	1.2	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)[‡‡]	4,638	3,327	10,600	9,223	59,090	13,419	17,375.6	0.8	22.7	16.1	50.3	42.9	269.0	60.1
Measles	10	125	12	65	104	70	63.2	1.1	0.0	0.6	0.1	0.3	0.5	0.3
Mumps	240	275	582	285	165	95	309.4	0.3	1.2	1.3	2.8	1.3	0.8	0.4
Pertussis	11,167	9,764	4,864	14,292	29,794	34,793	13,976.2	2.5	54.8	47.2	23.1	66.5	135.6	155.7
Pneumococcal disease (invasive)	1,691	1,451	1,476	1,628	1,557	1,644	1,560.6	1.1	8.3	7.0	7.0	7.6	7.1	7.4
Poliomyelitis	0	0	1	0	0	0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	29	59	34	36	27	44	37.0	1.2	0.1	0.3	0.2	0.2	0.1	0.2
Rubella – congenital	1	0	2	0	0	0	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	2	3	3	4	3	2	3.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	16	1,622	1,667	1,799	1,753	1,743	1,710.3	1.0	NN	18.5	18.6	19.6	11.8	11.5
Varicella zoster (shingles)	7	1,178	1,562	2,326	2,716	2,978	1,945.5	1.5	NN	13.5	17.5	25.4	18.3	19.7
Varicella zoster (unspecified)	141	3,764	4,284	4,413	6,775	7,152	4,809.0	1.5	NN	43.0	47.9	48.2	45.7	47.4
Vectorborne diseases														
Arbovirus infection (NEC)	27	30	17	12	8	24	18.8	1.3	0.1	0.1	0.1	0.1	0.0	0.1
Barmah Forest virus infection	1,317	2,130	1,712	2,087	1,480	1,471	1,745.2	0.8	6.5	10.3	8.1	9.7	6.7	6.6
Dengue virus infection	219	189	314	563	1,406	1,201	538.2	2.2	1.1	0.9	1.5	2.6	6.4	5.4
Japanese encephalitis virus infection	0	0	0	1	0	0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection[¶¶]	1	3	1	1	2	2	1.6	1.3	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	816	770	565	524	508	399	636.6	0.6	4.0	3.7	2.7	2.4	2.3	1.8
Murray Valley encephalitis virus infection[¶¶]	2	1	0	2	4	0	1.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	2,538	5,529	4,180	5,663	4,796	5,147	4,541.2	1.1	12.4	26.7	19.8	26.3	21.8	23.0

Table 6 *cont'd*: Notifications and notification rate for communicable diseases, Australia, 2005 to 2010, (per 100,000 population)

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2005	2006	2007	2008	2009	2010			2005	2006	2007	2008	2009	2010
Zoonoses														
Anthrax	0	1	1	0	0	1	0.4	2.5	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	41	50	37	45	32	21	41.0	0.5	0.2	0.2	0.2	0.2	0.2	0.1
Leptospirosis	129	145	108	112	146	131	128.0	1.0	0.6	0.7	0.5	0.5	0.7	0.6
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	164	165	93	102	65	56	117.8	0.5	0.8	0.8	0.4	0.5	0.3	0.3
Q fever	352	411	449	376	310	323	379.6	0.9	1.7	2.0	2.1	1.7	1.4	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	331	349	307	272	302	298	312.2	1.0	1.6	1.7	1.5	1.3	1.4	1.3
Leprosy	10	7	13	11	4	11	9.0	1.2	0.0	0.0	0.1	0.1	0.0	0.0
Meningococcal infection***	393	318	305	286	259	230	312.2	0.7	1.9	1.5	1.4	1.3	1.2	1.0
Tuberculosis	1,078	1,211	1,134	1,196	1,324	1,327	1,188.6	1.1	5.3	5.9	5.4	5.6	6.0	5.9
Total	**117,996**	**131,207**	**139,827**	**153,508**	**230,100**	**209,079**								

* Newly acquired hepatitis and syphilis 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 or is longer than 24 months.

‡ 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.

** The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – 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.

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	

Table 7 *cont'd*: 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		
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	
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

Table 7 *cont'd*: 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		
Vaccine preventable diseases, *cont'd*										
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 JE, MVE 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 MVE 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	
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	

Table 7 *cont'd*: 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		
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.

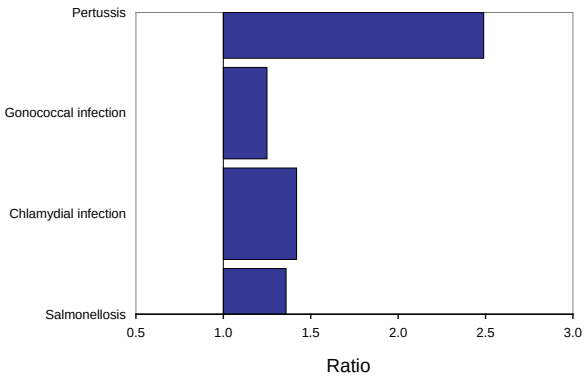
‖ Includes rubella – congenital from 1991 to 2002.

¶ 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

Figure 3: Total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2010, compared with the previous 5-year mean



Data completeness

Data on the sex of cases was complete in 99.8% of notifications and age at onset in close to 100% of notifications (Table 8). In 2010, Indigenous status was complete in 54% of notifications, and varied by jurisdiction. Indigenous status was complete for 94% of data reported in the Northern Territory, 84% in South Australia and 90% in Western Australia. In the remaining jurisdictions, less than 60% of data were complete for Indigenous status.

Data completeness on Indigenous status also varied by disease as summarised in Appendix 3. There were 5 diseases for which notifications were 100% complete for Indigenous status.[9] A further 9 diseases equalled or exceeded 90% completeness for Indigenous status. Of the 18 priority diseases agreed to by CDNA and the NSC in 2010 for improving Indigenous identification, nine had an Indigenous completeness that exceeded 90% (donovanosis, *Haemophilus influenzae* type b, hepatitis A, meningococcal infection, congenital syphilis, syphilis < 2 years duration, leprosy, measles, and tuberculosis). The diseases for which there was less than 90% Indigenous completeness included hepatitis C (newly acquired), hepatitis B (newly acquired), dengue virus infection, gonococcal infection, pneumococcal disease (invasive), and shigellosis. In 2010, CDNA set target thresholds of 90% completeness for priority diseases and 80% completeness for the remainder of the notifiable diseases.

Bloodborne diseases

In 2010, 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 notified cases over time may not solely reflect changes in disease prevalence or incidence. Testing policies such as the National

Table 8: Completeness of National Notifiable Diseases Surveillance System data received, Australia, 2010, by state or territory*

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total notifications	3,371	45,793	7,580	54,083	22,658	4,237	47,271	24,086	209,079
Sex									
Unknown/ missing	1	166	3	17	0	0	389	0	518
Per cent complete	99.9	99.6	99.9	99.9	100.0	100.0	99.2	100.0	99.8
Age at onset									
Unknown/ missing	0	74	0	0	0	1	139	0	200
Per cent complete	100.0	99.8	100.0	100.0	100.0	99.9	99.7	100.0	99.9
Indigenous status									
Unknown/ missing	2,335	33,368	450	30,905	3,747	1,783	22,437	2,386	95,933
Per cent complete	30.7	27.1	94.1	42.9	83.5	57.9	52.5	90.1	54.1

* 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.

Hepatitis C Testing Policy[10] 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 is described within the hepatitis B and hepatitis C sections.

Hepatitis B

Hepatitis B notifications are classified as either 'newly acquired' or 'unspecified' as described above. 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 2010, there were 7,106 diagnoses of hepatitis B (both newly acquired and unspecified) reported, equating to a rate of 31.9 cases per 100,000 population (Figure 4). The Northern Territory recorded the highest hepatitis B diagnosis rate in 2010 (69.7), followed by Victoria (35.3) and Western Australia (35.2).

Since the introduction of the adolescent hepatitis B vaccination program for children aged between 10 and 13 years in 1997 and the universal infant program in 2000,[11] there has been a general decline in overall rates of hepatitis B. Between 2000 and 2010 unspecified hepatitis B rates decreased 22% from 39.5 to 30.8 and newly acquired hepatitis B rates decreased from a rate of 2.2 to 1.0 (Figure 4). Approximately 92% of the 2010 Australian birth cohort received the full primary course of the hepatitis B vaccine by 15 months of age.[12]

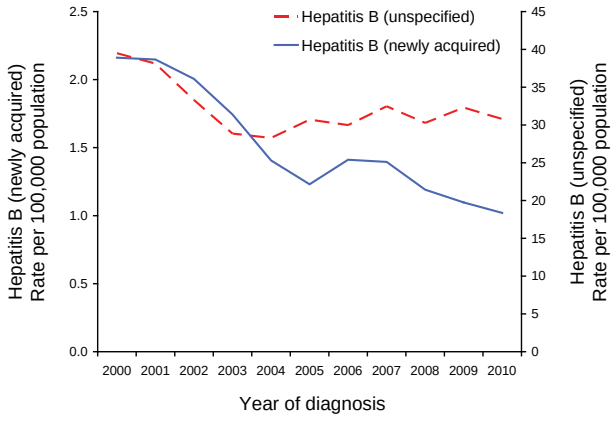
Newly acquired hepatitis B

In 2010, there were 228 notified cases of newly acquired hepatitis B (1.0 per 100,000 population) reported to the NNDSS; a 4% decrease compared with the 238 cases (rate of 1.1) reported in 2009 and a continuation of the downward trend in notified cases (Figure 4).

Nationally, the proportion of all hepatitis B cases in 2010 that were documented as newly acquired continued to trend downward and was 3.2%, compared with 3.3% in 2009 and 5.2% in 2000. The proportion of newly acquired infections compared to total hepatitis B infections varied substantially: Tasmania (11%); Queensland (5.2%), South Australia (4.9%); Western Australia (4.1%); Victoria (3.5%); the Australian Capital Territory (3.1%); the Northern Territory (1.9%) and New South Wales (1.4%). The highest rates were reported from Western Australia (1.4), closely followed by the Northern Territory, Queensland and South Australia (all 1.3) and Tasmania and Victoria (1.2).

Overall, cases were more common amongst males, with a male to female ratio of 1.9:1. In 2010, the highest rate of newly acquired hepatitis B infection was observed amongst males 35–39 and 40–44 years (3.0 and 3.1 respectively) (Figure 5).

Figure 4: Rate for newly acquired hepatitis B* and unspecified hepatitis B,† Australia, 2000 to 2010, by year‡

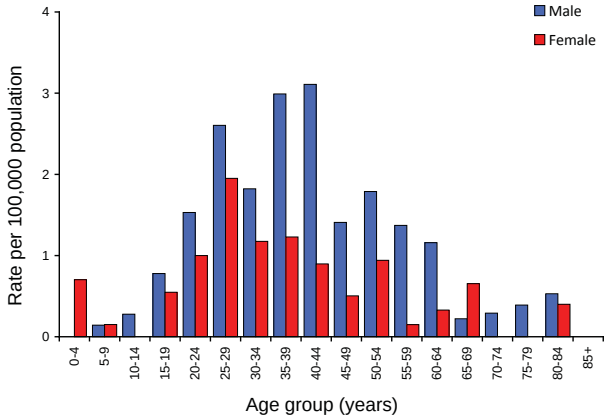


* Data for newly acquired hepatitis B for the Northern Territory (2000–2004) includes some unspecified hepatitis B cases.

† Data for unspecified hepatitis B for all jurisdictions except the Northern Territory between 2000 and 2004.

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

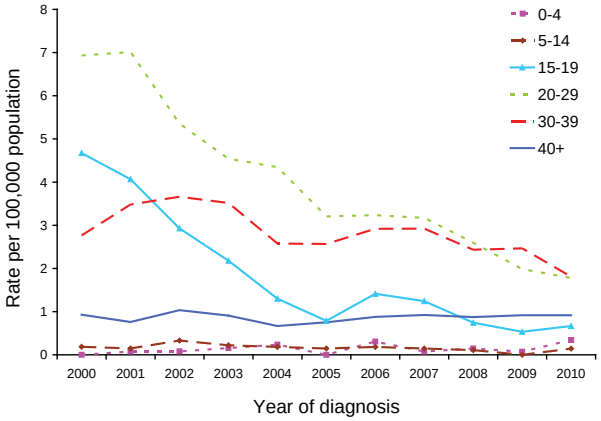
Figure 5: Rate for newly acquired hepatitis B,* Australia, 2010, by age group and sex



Trends in newly acquired hepatitis B infection by year and age group are shown in Figure 6. Between 2000 and 2010, most age group rates have been trending down with the most marked decreases occurring among the 15–19 year and 20–29 year age groups. There were 5 cases, all female, in the 0–4 year age group in 2010, the highest number since 4 cases were reported in 2006 and well above the average of 1.6 for the previous 5 years.

Of the 228 cases reported in 2010, the exposure history of 120 cases from New South Wales, Victoria, South Australia and the Northern Territory were assessed† (Table 9). In 2010, 73% (n = 87) of these

Figure 6: Rate for newly acquired hepatitis B,* Australia, 2000 to 2010, by year and age group



† Prior to 2009 enhanced hepatitis B surveillance data were reported to Kirby from health authorities in the states and territories.

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

Table 9: Notified cases of newly acquired hepatitis B cases,* selected jurisdictions, 2010, by sex and exposure category†

Exposure category	Number of exposure factors reported†			Percentage of cases* (n=120)
	Male	Female	Total	
Injecting drug use	29	18	47	39.2
Imprisonment	9	1	10	8.3
Skin penetration procedure	10	8	18	15.0
Tattoos	8	4	12	10.0
Ear or body piercing	2	3	5	4.2
Acupuncture	0	1	1	0.8
Healthcare exposure	9	2	11	9.2
Surgical work	6	1	7	5.8
Major dental surgery work	1	1	2	1.7
Blood/tissue recipient	0	0	0	0.0
Haemodialysis	2	0	2	1.7
Sexual contact – hepatitis B positive partner	5	7	12	10.0
Opposite sex	4	6	10	8.3
Same sex	1	1	2	1.7
Household contact	4	5	9	7.5
Needlestick/biohazardous injury§	2	0	2	1.7
Perinatal transmission	1	1	2	1.7
Other	10	7	17	14.2
Sexual contact – unknown HBV status‖	6	4	10	8.3
Cases with at least one risk factor	56	31	87	72.5
Undetermined	11	6	17	14.2
Unknown (not recorded)	10	6	16	13.3
Total exposure factors reported†	100	61	161	–
Total number of cases*	77	43	120	–

* Cases from New South Wales, the Northern Territory, South Australia, and Victoria.

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

‡ The denominator used to calculate the percentage is based on the total number of cases from all jurisdictions (New South Wales, the Northern Territory, South Australia, and Victoria). As more than one exposure category for each notification could be recorded, the total percentage does not equate to 100%.

§ Includes both occupational and non-occupational exposures.

‖ Established through analysis of free text field.

cases had at least one risk factor recorded, with the source of exposure not recorded or unable to be determined for the remainder. Injecting drug use remains the most frequently reported source of infection in 2010 but has declined as a proportion of reported cases from 52% in 2006 to 39% in 2010. Skin penetration procedures were the next most frequently reported source of infection (15%), the majority of which were reported as tattoos.

Additional information was also collected on the country of birth (COB) from all jurisdictions except Queensland. Of the 137 cases in which COB was reported, the majority occurred amongst Australian born persons (66%, n = 90) with the remaining 47 cases amongst those born overseas. The proportion of overseas-born people with hepatitis B was similar to the proportion of overseas born people in the Australian population for Europe and the Americas and higher for those born in North Africa and the Middle East and Asia.[13]

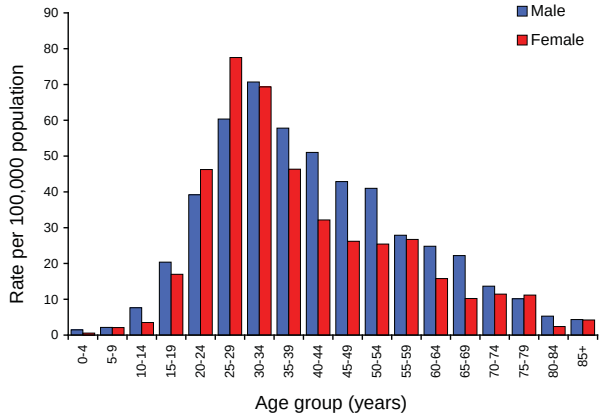
Unspecified hepatitis B notifications

In 2010, there were 6,878 notified cases of unspecified hepatitis B infection reported to the NNDSS, a rate of 31 per 100,000 population, compared with 7,094 cases (and a rate of 32) in 2009.

The overall rate of hepatitis B (unspecified) has been trending downward over the past 11 years with the majority of this decrease occurring between 2000 and 2004. Between 2005 and 2010 the rate has remained relatively stable with an average annual rate of 31 during this time (Figure 4).

In 2010, the overall male rate (33) was higher than for females (28), a rate ratio of 1.2:1, but females had the highest age specific rate amongst those in the 25–29 year age group (78) compared with the highest age specific rate amongst males of 71 in the 30–34 year age group (Figure 7).

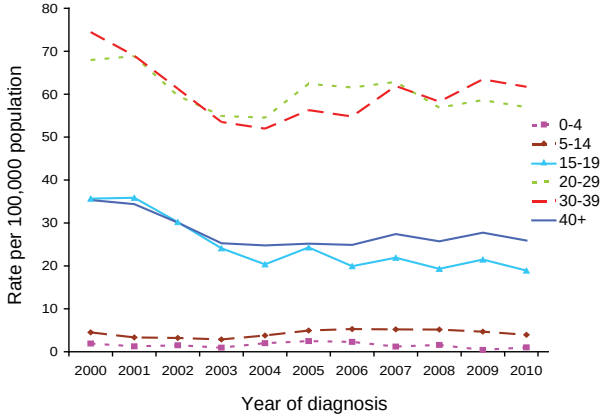
Figure 7: Rate for unspecified hepatitis B, Australia, 2010, by age group and sex



Trends in hepatitis B (unspecified) infection by year and age group are shown in Figure 8. Rates across all age groups have declined since 2000 with the majority of this decrease occurring in the first 3 years, before stabilising. The biggest decrease (47%) has occurred amongst the 15–19 year age group declining from a rate of 36 in 2000 to 19 in 2010.

The Northern Territory recorded the highest rate (68), followed by Victoria, New South Wales and Western Australia (all 34).

Figure 8: Rate for unspecified hepatitis B,* Australia, 2000 to 2010, by year and age group



* Data for hepatitis B (unspecified) from all states and territories except the Northern Territory between 2000 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 2000 and 2010, total hepatitis C notification rates declined by 50% (101 to 50 per 100,000), with the greatest reductions observed in the earlier years, (a 16% decline between 2001 and 2002) (Figure 9). These reductions followed a peak in notified cases associated with the detection and

notification of prevalent cases that occurred in the late 1990s through the expansion of testing in high risk groups.[14] 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.[14,15]

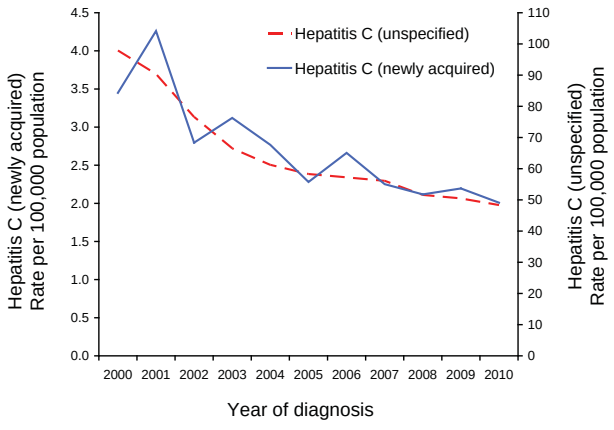
Although initial infection with the hepatitis C virus is asymptomatic or mildly symptomatic in more than 90% of cases, approximately 50%–80% of cases will go on to develop a chronic infection. Of these, half will eventually develop cirrhosis or cancer of the liver.[16] In 2010, it was estimated that 297,000 people living in Australia had been infected with the hepatitis C virus. Of these, approximately 168,000 had chronic hepatitis C infection and early liver disease, 48,000 had chronic hepatitis C infection with moderate liver disease, 6,100 were living with hepatitis C related cirrhosis and 76,000 had cleared their infection.[13]

Newly acquired hepatitis C notifications

Cases of newly acquired hepatitis C were reported from all states and territories except Queensland, where all cases of hepatitis C are reported as unspecified. There were 358 notified cases reported in 2010 compared with 401 notified cases in 2009, giving a rate of 2.0 per 100,000 population (Figure 9).

Of all hepatitis C cases in 2010, 3% were identified as newly acquired infections, which is comparable to previous years. The proportion of newly acquired infections compared with total hepatitis C diagnoses

Figure 9: Rates for newly acquired hepatitis C* and unspecified hepatitis C,† Australia, 2000 to 2010, by year



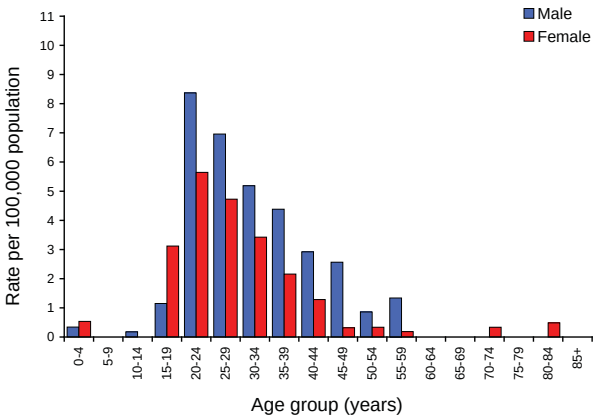
* Data for newly acquired hepatitis C from all states and territories except Queensland 2000–2010 and the Northern Territory 2000–2002.

† Data for unspecified hepatitis C provided from Queensland (2000–2010) and the Northern Territory (2000–2002) includes both newly acquired and unspecified hepatitis C cases.

varied substantially amongst the states and territories with 9% in South Australia; 8% in Tasmania; 7% in Western Australia; 6% in Victoria; 5% in the Australian Capital Territory; and 1% in New South Wales. No newly acquired cases were recorded for the Northern Territory or Queensland. The highest rates of newly acquired hepatitis C infection were reported in Tasmania (4.3 per 100,000), followed by Western Australia (3.5) and the Australian Capital Territory (3.3 per 100,000). 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 amongst states and territories, with newly acquired infection more likely to be detected in population groups that are tested frequently, such as those in prison settings.

The male to female ratio was 1.6:1 with the highest rate amongst males in the 20–24 year age group followed by the 25–29 year age group and the 30–34 year age group (8.4, 7.0 and 5.2 respectively). The highest age group rates for females were consistent with males, occurring in the 20–24 year age group followed by the 25–29 and 30–34 year age groups (5.6, 4.7 and 3.4 respectively) (Figure 10).

Figure 10: Rate for newly acquired hepatitis C, Australia,* 2010, by age group and sex

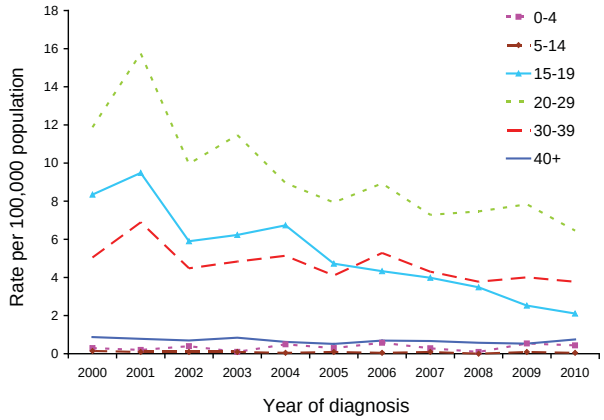


* Data from all states and territories except Queensland.

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 year age groups. Annual rates in the other age groups continued to be relatively stable over the 2000 to 2010 period.

Exposure history surveillance data for 91% of newly acquired hepatitis C cases reported in 2010 were

Figure 11: Rate for newly acquired hepatitis C, Australia* 2000 to 2010, by year and age group



* Data from all states and territories except Queensland (2000–2010) and the Northern Territory (2000–2002).

assessed from all jurisdictions except Queensland (Table 10). In 2010, 75% of these cases 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. Seventy-nine per cent of notifications had a history of injecting drug use (57% of which reported injecting drug use in the 24 months prior to diagnosis). Skin penetration procedures and imprisonment accounted for 26% and 22% of reported risk factors respectively, noting that screening rates are generally higher in the prison entry population than the general population. A screening survey of prison entrants conducted over a 2-week period in 2007 found that the prevalence of hepatitis C, based on hepatitis C antibody detection, was 35%.[17]

Table 10: Notified cases of newly acquired hepatitis C, selected jurisdictions,* 2010, by sex and exposure category†

Exposure category	Number of exposure factors reported†			Percentage‡ of cases* (n=325)
	Male	Female	Total	
Injecting drug use	156	102	258	79.4
Imprisonment	64	7	71	21.8
Skin penetration procedure	56	28	84	25.8
Tattoos	34	15	49	15.1
Ear or body piercing	21	12	33	10.2
Acupuncture	1	1	2	0.6
Healthcare exposure	12	13	25	7.7
Surgical Work	5	10	15	4.6
Major dental surgery work	4	2	6	1.8
Blood/tissue recipient	3	0	3	0.9
Haemodialysis	0	1	1	0.3
Sexual contact – hepatitis B positive partner	27	15	42	12.9
Opposite sex	22	14	36	11.1
Same sex	5	1	6	1.8
Household contact	12	8	20	6.2
Perinatal transmission	22	7	29	8.9
Needlestick/biohazardous injury§	3	0	3	0.9
Other	8	9	17	5.2
Notifications with at least one risk factor	138	107	245	75.4
Undetermined	46	14	60	18.5
Unknown or missing (not recorded)	11	9	20	6.2
Total exposure factors reported†	417	212	629	–
Total cases*	195	130	325	–

* Includes diagnoses in all states and territories except Queensland.

† 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.

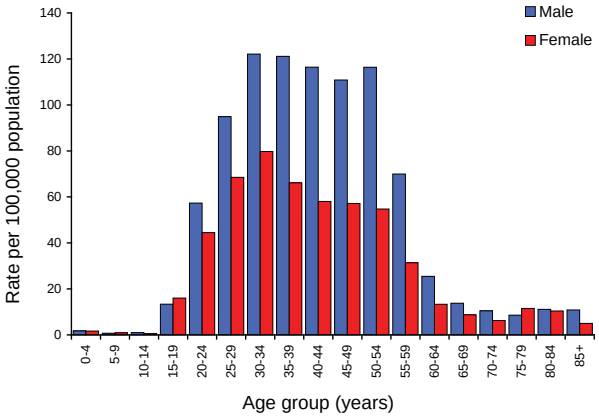
Unspecified hepatitis C notifications

In 2010, there were 10,803 notified cases of unspecified hepatitis C infections (48 per 100,000 population) compared with 11,081 notified cases in 2009 and a rate of 51 per 100,000 population. This continues the downward trend and represents a 51% decline compared with 2000, when the rate was 98 per 100,000 population.

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.[14,15,18]

The male to female ratio remained consistent with historical trends at 1.7:1 in 2010. Amongst males, rates were highest across age groups between 30 and 54 years ranging from 111 to 122. Females rates were similarly highest amongst adults in the 30–34 year age group (80 per 100,000) followed by the 25–29 year (67 per 100,000) and 35–39 year age groups (66 per 100,000) (Figure 12).

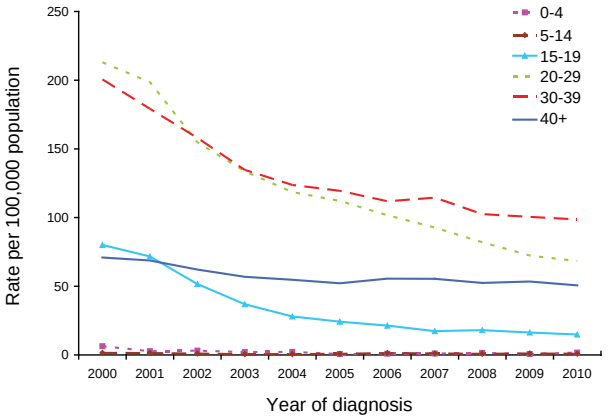
Figure 12: Rate for unspecified hepatitis C,* Australia, 2010 by age group and sex



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

The rate of unspecified hepatitis C declined in all age groups with the biggest decreases occurring amongst the 15–19 year age group (82%), the 20–29 year (68%) and the 30–39 year age groups (51%). The majority of this decline occurred in the early part of the decade. Trends in the 0–4, 5–14 and the 40 years or over age groups have remained relatively stable over this time (Figure 13).

Figure 13: Rate for unspecified hepatitis C,* Australia, 2000 to 2010, by year and age group



* Data provided from Queensland (2000–2010) and the Northern Territory (2000–2002) includes both newly acquired and unspecified hepatitis C cases.

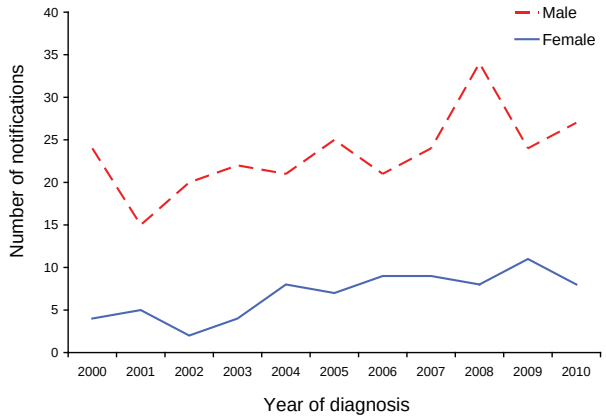
In 2010, the Northern Territory continued to have the highest rate (75 per 100,000) followed by Queensland (61 per 100,000) and the Australian Capital Territory (59 per 100,000), noting that Queensland's rate includes both newly acquired and unspecified cases. The lowest rate was in South Australia (30 per 100,000) (Table 5).

Hepatitis D

Hepatitis D is a defective single-stranded RNA virus 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.[16] The modes of hepatitis D transmission are similar to those for hepatitis B, and in countries with low hepatitis B prevalence such as Australia, injecting drug users are therefore likely to be the main group at risk for hepatitis D.

In Australia, the rate of hepatitis D remains low. In 2010, there were 35 notified cases of hepatitis D, a rate of 0.2 per 100,000 population, reported from Queensland (n = 20), New South Wales (n = 9) and Victoria (n = 6). Reported cases of hepatitis D have had a slight increasing trend with case numbers in 2010 above the average of 30 cases notified per year (range: 20–42) between 2000 and 2009. The male female ratio in 2010 was 3.4:1 consistent with the average ratio of 3:1 in the preceding 5 years (Figure 14).

Figure 14: Notified cases of hepatitis D, Australia, 2000 to 2010, by year and sex



Gastrointestinal diseases

In 2010, 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 decreased slightly from 31,695 in 2009 to 31,548 in 2010. Only notifications of salmonellosis were notably increased compared with the 5-year mean (exceeding 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. 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; botulism, campylobacteriosis, HUS, hepatitis A, listeriosis, non-typhoidal salmonellosis, STEC infection, shigellosis, and typhoid. The data and results from these analyses are summarised in the following sections and are reported in more detail in the OzFoodNet annual report 2010.

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.[16] 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.

There were no cases of botulism reported to NNDSS in 2010. There was 1 notified case reported in 2009 and no cases reported in 2008.

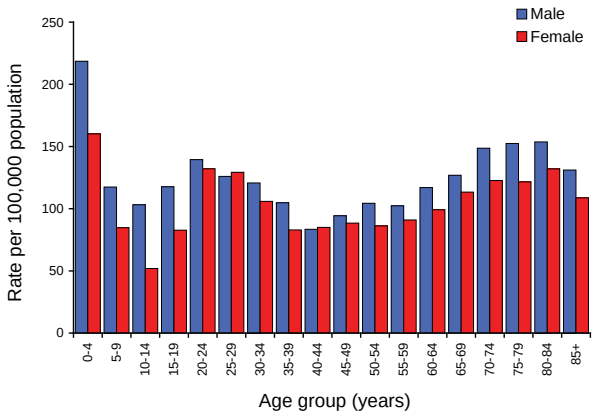
Campylobacteriosis

The bacterium *Campylobacter* is a common cause of foodborne illness (campylobacteriosis) in humans. The severity of this illness varies and is characterised by diarrhoea (often bloody stools), abdominal pain, fever, nausea and or vomiting. Campylobacteriosis is notifiable in all Australian jurisdictions, except New South Wales.

In 2010, there were 16,966 notified cases of campylobacteriosis, a rate of 112 per 100,000, similar to the 16,081 notifications in 2009. Notification rates ranged from 71.8 per 100,000 in the Northern Territory to 153.8 per 100,000 in the Australian Capital Territory.

Notification rates were highest amongst males in nearly all age groups. The highest age-specific rate for both males and females was in the 0–4 age group (218.5 and 160.2 per 100,000, respectively) with additional peaks in the 20–24 and 80–84 year age groups (Figure 15).

Figure 15: Rate for campylobacteriosis, Australia, 2010, by age group and sex



Cryptosporidiosis

Cryptosporidiosis is a parasitic infection of the lower intestine, characterised by abdominal cramping and usually large-volume watery diarrhoea. Ingesting contaminated water is a major risk factor for infection.

In 2010, there were 1,480 notified cases of cryptosporidiosis reported to NNDSS; a rate of 7 cases per 100,000. This represents a 68% decrease over the 4,625 notifications in 2009, which was the largest number reported since the disease became nationally notifiable in 2001.

Cryptosporidiosis notifications fluctuate from year to year, and notifications are most numerous in autumn and summer, with some regional variation.

Haemolytic uraemic syndrome

Haemolytic uraemic syndrome is a rare but serious clinically diagnosed disease, that is characterised by acute renal impairment, and results in chronic complications in 40% of cases.[19] Not all diagnoses of HUS are related to enteric pathogens, but in Australia cases are commonly associated with STEC infection.

In 2010, there were 8 notified cases of HUS (rate 0.05 per 100,000 population) (Table 11), compared with 13 in 2009 and a mean of 19 notifications per year (0.1 per 100,000 population) between 2005 and 2009.

The median age of HUS cases between 2005 and 2010 was 6 years (range 0–89 years) and cases were most frequently reported amongst children in the 0–4 year age group (Table 11).

Hepatitis A

Hepatitis A is a viral disease primarily of the liver that can develop into chronic liver disease including liver failure. Infection is usually spread via the faecal-oral route but can be foodborne or waterborne.

In 2010 there were 263 notified cases of hepatitis A in Australia, a rate of 1.2 notifications per 100,000 population. This was a 53% decrease compared with the 563 notifications in 2009 (Table 6). The increase in 2009 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.[20,21]

In 2010, 40% (106/263) of notified cases were locally-acquired with most of these being part of the 2009–2010 outbreak, and with a 5-year average (2005–2009) of 166 locally-acquired cases per year (Table 12).

Hepatitis A was most frequently notified amongst young adults and in 2010, the median age of notified cases was 27 years (range 0–97 years), and 51% (133/263) were male.

Indigenous status was known for 91% of notifications and of these no cases identified as being Indigenous.

Table 11: Notified cases of haemolytic uraemic syndrome, Australia, 2005 to 2010, by age group

Age group	2005	2006	2007	2008	2009	2010
0–4	12	5	8	11	5	6
5–9	2	3	2	4	2	0
10–14	1	2	0	2	2	1
15–19	0	0	3	2	0	0
20–24	0	1	0	0	0	1
25–29	0	2	0	1	1	0
30–34	0	0	0	2	0	0
35–39	0	0	1	0	0	0
40–44	0	0	2	0	0	0
45–49	0	0	0	3	0	0
50–54	2	0	1	1	0	0
55–59	0	0	0	1	0	0
60–64	1	1	1	0	1	0
65–69	1	0	0	0	0	0
70–74	0	0	1	2	0	0
75–79	0	0	0	1	1	0
80–84	1	0	0	1	0	0
85+	0	0	0	0	1	0
Total	20	14	19	31	13	8

Table 12: Hepatitis A notifications, Australia, 2005 to 2010, by place of acquisition

	Locally acquired		Overseas acquired		Unknown	
Year	n	%	n	%	n	%
2005	140	42.8	151	46.2	36	11.0
2006	101	35.9	69	24.6	111	39.5
2007	74	44.8	35	21.2	56	33.9
2008	99	35.7	52	18.8	126	45.5
2009	416	73.9	68	12.1	79	14.0
2010	106	40.3	131	49.8	26	9.9

Hepatitis E

Hepatitis E is a viral disease primarily of the liver that is transmitted by the faecal-oral route.

In 2010, there were 38 notified cases of hepatitis E; a rate of 0.2 per 100,000, compared with 33 notifications in 2009. Hepatitis E in Australia is associated with overseas travel, with 58% (n = 22) known to have been acquired overseas.

Listeriosis

Invasive listeriosis commonly affects the elderly or immunocompromised, and is most common amongst people with serious or terminal underlying illnesses. Listeriosis can also affect pregnant women and infect their unborn baby. Laboratory-confirmed infections in a mother and unborn child or a neonate are notified separately in the NNDSS. However, OzFoodNet counts such pairs as 1 case, with the mother reported as the primary case, which explains the differences in numbers from those reported in OzFoodNet annual reports.

In 2010, there were 71 notified cases of invasive *Listeria monocytogenes* infection; a rate of 0.3 per 100,000, compared with a 5-year historical mean of 65 notifications. This was a decrease from the 92 notified cases in 2009, when an outbreak associated with chicken wraps was reported.[22]

Salmonellosis (non-typhoidal)

Salmonellosis is a bacterial disease caused by *Salmonella enterica*. The disease is characterised by rapid development of symptoms including abdominal pain, fever, diarrhoea, muscle pain, nausea and/or vomiting. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures.

There were 11,993 notified cases of salmonellosis in Australia in 2010; a rate of 53.7 per 100,000, compared with the 5-year mean of 8,825 notifications. In 2010, salmonellosis notifications continued to increase, with notifications exceeding the 5-year mean by more than 2 standard deviations.

Notification rates ranged from 40.4 per 100,000 in South Australia to 243.4 per 100,000 in the Northern Territory. In 2010, 51% (n = 6,111) of cases were in females, with the greatest proportion of cases in the 0–4 year age group (26%, n = 3,090).

Shigellosis

Shigellosis is bacterial disease characterised by acute abdominal pain and fever, small-volume loose stools, vomiting and tenesmus. *Shigella* is transmitted via the faecal-oral route, either directly (such as male-to-male sexual contact) or indirectly and can be foodborne.

In 2010, there were 552 notified cases of shigellosis; a rate of 2.5 per 100,000, with notifications being less than the 5-year mean of 665 notifications. As in previous years, the highest notification rate was in the Northern Territory (32.7 per 100,000).

Notifications for shigellosis were highest in the 0–4 year age group (21%, n = 115), and 53% (n = 293) of notified cases were female.

Information on Indigenous status was available for 82% (n = 451) of notifications, and this proportion varied by state or territory, with New South Wales, Queensland, South Australia and Tasmania 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 35% (99/283).

Twenty-five per cent (n = 140) of notified cases were reported as being acquired overseas. The most frequently reported countries of acquisition for imported cases were Indonesia (34%, n = 48) and India (11%, n = 15).

Shiga toxin-producing *Escherichia coli* infections

Shiga toxin-producing *E. coli* are species of toxin-producing *E. coli* that cause diarrhoeal illness in humans. Severe cases can progress to HUS.[23]

There were 81 notifications of STEC in Australia in 2010; a rate of 0.4 per 100,000 population.

Rates of STEC infection are strongly influenced by jurisdictional practices regarding the screening of stool specimens.[23] 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 2.0 per 100,000.

In 2010, 62% (n = 50) of notified cases were female. The median age of notified cases was 43 years (range 1–98 years).

Typhoid

Typhoid is a disease caused by *S. enterica* serotype Typhi. Transmission is the same as for salmonellosis, however typhoid differs in that humans are the reservoir for the bacterium.

There were 96 notified cases of typhoid during 2010 (rate 0.4 per 100,000), which was slightly higher than the 5-year mean of 88.

Similar to previous years, overseas travel was the primary risk factor for notified cases in 2010, with 76% (n = 73) of notified cases known to have been acquired overseas, compared with 89% (102/115) in 2009. India continues to be the most frequently reported country of acquisition, accounting for 43% (n = 41) of overseas-acquired cases in 2010, 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.

Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and notifiable in Australia and to the WHO in 2010 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean–Congo).

Cholera, plague, rabies, smallpox, yellow fever, SARS, HPAIH and viral haemorrhagic fevers are of international public health importance. 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 2010. Table 13 provides information on the occurrence of quarantinable diseases in Australia.

Table 13: Australia's status for human quarantinable diseases, 2010

Disease	Status	Date of last record and notes
Cholera	Free	Small number of cases are reported annually and related to overseas travel or imported food products[24]
Plague	Free	Last case recorded in Australia in 1923[25]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990 [26]
Smallpox	Free	Last case recorded in Australia in 1938[27]
Yellow fever	Free	No cases recorded on shore in Australia – 5 occasions on which vessels arrived in Australian ports 1892–1915[25]
SARS	Free	Last case recorded in Australia in 2003[28]
HPAIH	Free	No cases recorded[29]
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded
Marburg	Free	No cases recorded
Lassa	Free	No cases recorded
Crimean–Congo	Free	No cases recorded

Cholera

There were 3 notified cases of cholera in Australia in 2010, two from New South Wales and one from Western Australia. All were acquired overseas. There were 19 cases of cholera in Australia between 2005 and 2009 (Table 7).

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 and 3 cases in 2006.[24]

Sexually transmissible infections

In 2010, 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 2005 and 2010 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 as changes in screening programs,[30,31] 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. Rates for STIs, are particularly susceptible to overall rates of testing as well as targeted testing in certain high risk population sub-groups.[32] 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[33] 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 2005 to 2010. Where the Indigenous status of a notification was not completed, these notifications were counted as non-Indigenous in the analyses. 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 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.[34,35] 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.[32]

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).[36] 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.

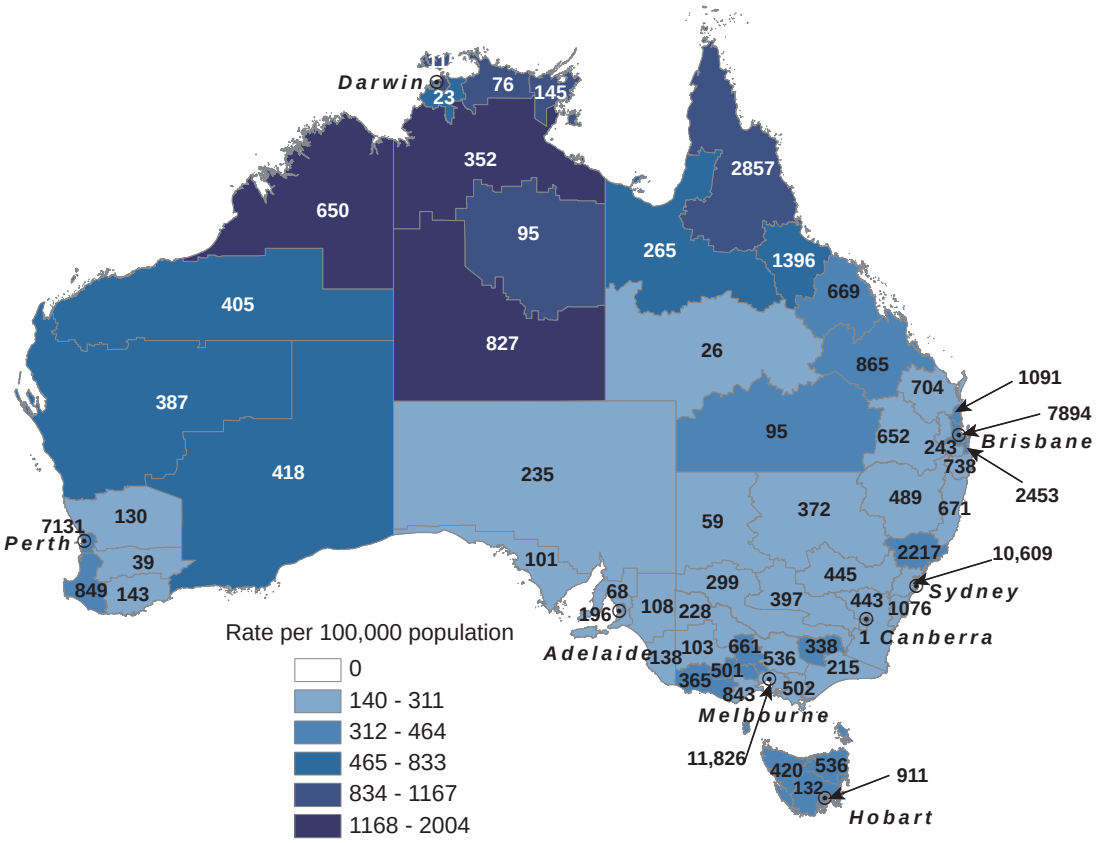
Chlamydial infection

Chlamydial infection continued to be the most commonly notified disease in 2010. Since chlamydial infection became a nationally notifiable disease in 1991 (1997 in New South Wales), the rate has increased in each consecutive year. In 2010, there were 74,305 notified cases of chlamydial infection, equating to a rate of 333 per 100,000 population. This represents an increase of 17% compared with the rate reported in 2009 (285). Between 2005 and 2010, chlamydial infection rates increased by 64%, from 203 to 333 per 100,000 population (Table 6).

Increasing rates of chlamydia were reported from all states and territories with the Northern Territory (1,159 per 100,000), Western Australia (443 per 100,000) and Queensland (426 per 100,000) substantially higher than the national rate (Table 5). At a regional level, chlamydial rates were highest in the Central NT SSD of the Northern Territory (2,004 per 100,000; n = 827) followed by the Kimberley SD of Western Australia (1820 per 100,000; n = 650). However, rates in geographic areas where the estimated residential population and case numbers are small, should be interpreted with caution. Rates were substantially higher than the national rate in the remaining SSDs of the Northern Territory, the North and North West SDs in Queensland and the Pilbara, Central and South Eastern SDs in Western Australia (Map 2).

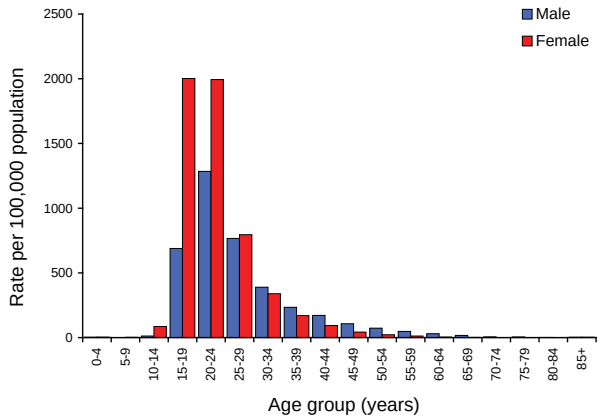
In 2010, rates of chlamydial infection in males and females were 279 and 384 per 100,000 population respectively. When compared with 2009, rates increased by 19% in males and 15% in females. The male to female rate ratio in 2010 was 0.7:1, which was similar to previous years. Rates for females exceeded those for males in the under 30 age range, especially in the 10–14 year age group with a ratio of 0.1:1, while males had higher rates in the older age groups (Figure 16).

Map 2: Rates and counts* for chlamydial infection, Australia, 2010, by Statistical Division and Statistical Subdivision of residence in the Northern Territory



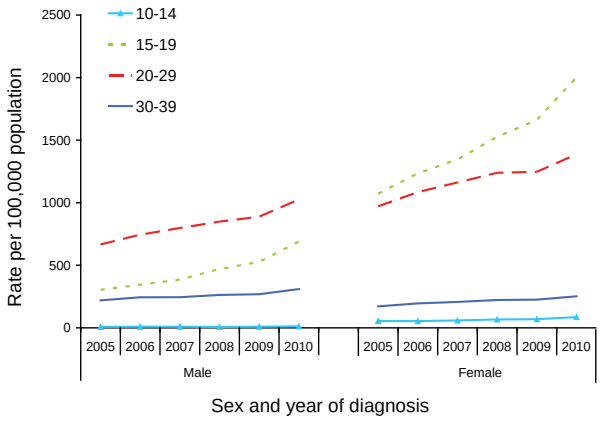
* Numbers in the shaded Statistical Divisions and Statistical Subdivisions represent the count of notifications.

Figure 16: Rate for chlamydial infection, Australia, 2010, by age group and sex*



* Excludes 246 notifications for whom age and or sex were not reported.

Figure 17: Rate for chlamydial infection in persons aged 10–39 years, Australia, 2005 to 2010, by sex, year and age group



Between 2005 and 2010, there was an increasing trend in chlamydia notification rates across both sexes and in all age groups (Figure 17). The greatest increase in rates amongst those aged 15–39 years occurred in both males and females in the 15–19 age group (114% and 75% respectively). Those aged 15–29 years accounted for approximately 80% of the annual number of notified cases during the period 2005 to 2010.

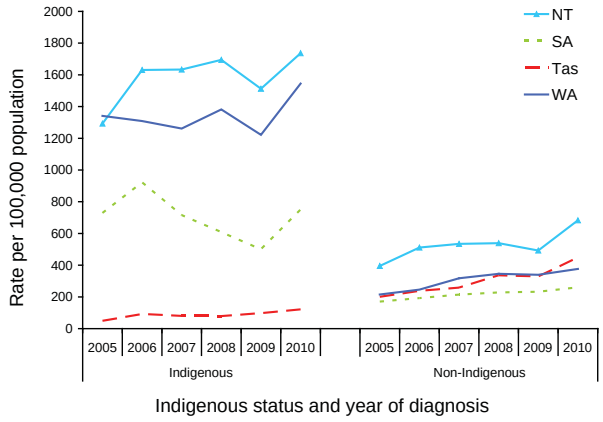
Nationally in 2010, data on Indigenous status were complete for 50% of notifications, which was higher than the preceding 5-year mean of 45% (range:

40%–49%). 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 2005 to 2010 period. These were the Northern Territory, South Australia, Tasmania and Western Australia. Amongst these jurisdictions, the combined age standardised notification rate ratio between Indigenous and non-Indigenous populations in 2010 was 3.8:1, with the disparity in notification rates improving substantially since 2000.

After a 40% increase between 2005 and 2006, rates amongst the Indigenous population remained fairly consistent between 2006 and 2009, with an average rate during this period of 1,193, but in 2010 there was a 12% increase to 1,342 compared with this average. In contrast, rates amongst the non-Indigenous population have been trending upwards from a rate of 205 in 2005 to 356 in 2010, representing a 74% increase over this period.

In 2010, chlamydia rates increased compared with 2009 in all 4 states and territories in which Indigenous status was more than 50% complete, ranging from 14% (Tasmania) to 49% (South Australia) amongst the Indigenous population and 11% (Western Australia) to 39% (Northern Territory) amongst the non-Indigenous population (Figure 18). The overall high Indigenous population rates observed in the Northern Territory, Western Australia and South Australia may be partly explained by the high level of screening, which take place in remote Indigenous communities.

Figure 18: Age standardised rate for chlamydial infection, selected states and territories,* 2005 to 2010, by Indigenous status, year and state or territory



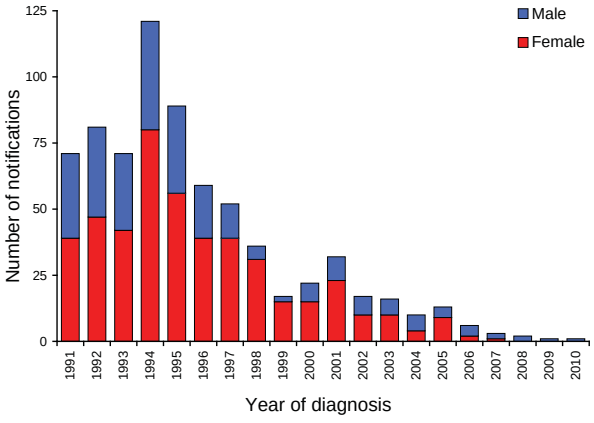
* Includes notifications in the Northern Territory, South Australia, Tasmania and Western Australia where Indigenous status was reported for more than 50% of cases over a 5-year period.

Between May 2007 and June 2010, the Australian Government Department of Health and Ageing funded a pilot program called the Australian Collaboration for Chlamydia Enhanced Sentinel 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 2010, ACCESS identified that chlamydia positivity amongst people who accessed the sentinel sites, was 11% amongst males and 10% amongst females, with positivity highest in the 16–19 year age group across most of the sentinel sites.[13] The chlamydia positivity rate increased between 2% and 3% amongst young heterosexual men and women and amongst men who have sex with men between 2006 and 2010. Between 2007 and 2010, the number of people who accessed these sentinel sites and were tested increased by 21%. Notification rates for chlamydia and other STIs are particularly susceptible to overall rates of testing as well as targeted testing in high-risk groups.

Donovanosis

Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project.[37] The disease predominantly occurred in rural and remote Indigenous communities in central and northern Australia and is now relatively uncommon. In 2010, 1 notified case was reported to the NNDSS of a male from Queensland (Figure 19).

Figure 19: Notified cases of donovanosis, Australia, 1991 to 2010, by year and sex



Gonococcal infection

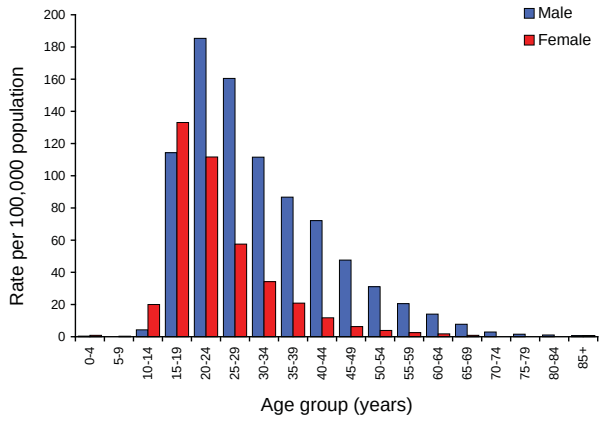
In 2010, there were 9,971 notified cases of gonococcal infection reported to the NNDSS; a rate of 45 per 100,000 population. This was a 23% increase compared with 2009. Due to a technical processing

error, gonococcal data for Queensland were under-reported in 2009 and 2010 and therefore should be interpreted with caution.

The highest rate in 2010 was in the Northern Territory (841 per 100,000 population), which was almost 19 times higher than the national rate (Table 5). Between 2008 and 2009 considerable declines in rates were observed in Western Australia (23%), South Australia (25%) and Tasmania (17%) with increases for the same period reported in Victoria (64%), New South Wales (22%), and the Australian Capital Territory (157%). In 2010, all states and territories except Tasmania and the Australian Capital Territory reported increases ranging from 2% in Western Australia to 38% in New South Wales when compared with 2009.

Nationally, there was an increase in the gonococcal infection rates in both males (26%) and females (17%) compared with 2009. The male to female rate ratio in 2010 was 2.2:1 (61 and 28 respectively), which is similar to the previous 5 years. Nationally, the rate of gonococcal infection in males exceeded those in females in all age groups except those aged less than 20 years (Figure 20). As in previous years, the exception to this pattern was the Northern Territory, where females had an overall higher notification rate than males (889 compared with 797 per 100,000).

Figure 20: Rate for gonococcal infections, Australia, 2010, by age group and sex*



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

Age specific rates amongst males increased in all age groups except the 10–14 year age group in contrast to females for which rates increased in the 15–19 and 20–29 year age groups but otherwise remained relatively stable (Figure 21).

In 2010, the data completeness of the Indigenous status field for gonococcal infection notifications was 65%, the same as in 2009 but a decrease compared with the previous few years (around 70%). All jurisdictions except New South Wales and the Australian Capital Territory had greater than 50% completeness of the Indigenous status field. Amongst these jurisdictions the combined age standardised notification rate for gonococcal infection in the Indigenous population had been steadily declining from 919 per 100,000 in 2006 to 629 per 100,000 in 2009 before increasing to 878 per 100,000 in 2010. In the non-Indigenous population, rates have been stable at around 22 to 23 per 100,000 between 2005 and 2009 before also increasing by 40% to 32 per 100,000 in 2010. Between 2005 and 2010 the Indigenous to non-Indigenous rate ratio has decreased 31% from 40:1 to 27:1. In

Figure 21: Rate for gonococcal infection in persons aged 10–49, Australia, 2005 to 2010, by and sex, year and age group

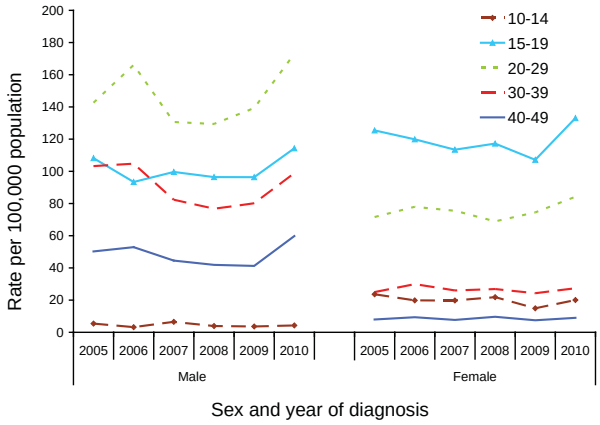
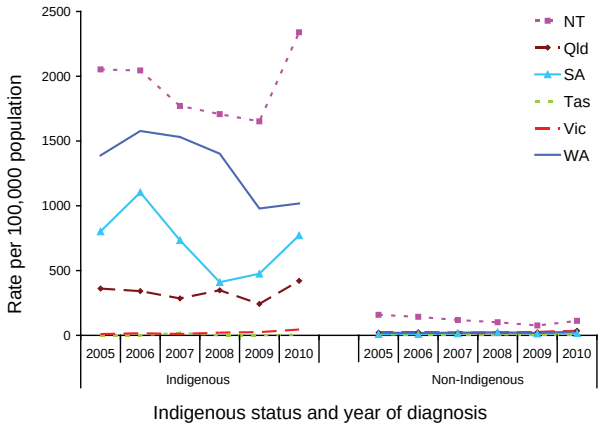


Figure 22: Age standardised rate for gonococcal infection, selected states and territories,* 2005 to 2010, by Indigenous status, year and state or territory



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

2010, rates of gonococcal infection in the Indigenous and non-Indigenous populations increased compared with 2009 in all jurisdictions except Tasmania (Figure 22). The overall high Indigenous population rates observed in the Northern Territory may be partly explained by the high level of screening which take place in remote Indigenous communities.

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 using a standardised methodology is performed on gonococcal isolates to a core group of antibiotics: penicillin, ceftriaxone, spectinomycin, quinolone and tetracycline.

In 2010, the AGSP[38] reported that 4,101 gonococcal isolates were tested for antibiotic susceptibility, representing approximately 41% of notified cases of gonococcal infection and a similar proportion to 2009 (40%) and 2008 (42%).

Of the isolates collected through the AGSP in 2010, the majority (n = 3,381) were from males with the remaining 720 from females (ratio 4.7:1). In males, 65% of isolates were obtained from the urethra, 20% from the rectum and 12% from the pharynx. In females, the majority of isolates (89%) were obtained from the cervix.

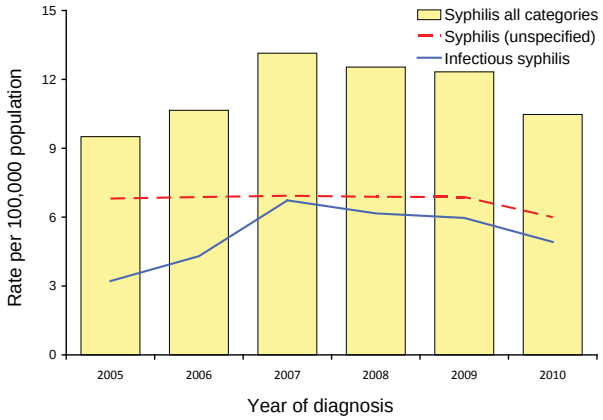
In 2010, approximately 29% of gonococcal isolates had some level of resistance to the penicillins, a decrease from the 36% identified in 2009. In addition, 35% had some level of resistance to quinolones, representing a further decrease in the proportion of quinolone resistance from 43% in 2009 and 54% detected in 2008. Since 2001, low but increasing numbers of isolates with decreased susceptibility to ceftriaxone have been identified in Australia with 4.8% observed nationally in 2010. There were no resistant ceftriaxone isolates reported in 2010. For more details see the AGSP annual report series published in CDI.

Syphilis (non-congenital categories)

In 2004, all jurisdictions except South Australia 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. South Australia, only report cases of infectious syphilis. 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 2010, there were 2,364 notified cases of syphilis infection of all non-congenital categories reported to NNDSS, representing a rate of 10.6 per 100,000 population; a 14% decrease compared with 2009 (12.3 per 100,000 population) (Table 6, Figure 23). The Northern Territory continued to have the highest rate of syphilis (61 per 100,000 population), consistent with the rate in 2009. In 2010, there were decreases in rates from Tasmania (26%), New South Wales (21%), Queensland (19%), Western Australia (17%), South Australia (16%), and Victoria (5%). While national rates have declined since 2007, overall between 2005 and 2010 there has been an 11% increase, and as in other developed countries, predominantly affecting men who have sex with men.[39,40]

Figure 23: Rate for non-congenital syphilis infection (all categories), Australia, 2005 to 2010, by year



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

In 2010, there were 1,099 notified cases of infectious syphilis (primary, secondary and early latent), less than 2 years duration reported to NNDSS. This represents a notification rate of 4.9 per 100,000, a decrease of 18% compared with 2009 (6.0 per 100,000 population) (Table 5). The rate of infectious syphilis notifications increased from 3.2 per 100,000 in 2005 to a peak of 6.7 per 100,000 in 2007 and has been gradually declining since then (Figure 23). The Northern Territory had the highest notification rate at 19 per 100,000 population in 2010, an 11% increase compared with 2009, but an overall 59% decrease compared with 2005.

Nationally, the rates of infectious syphilis for males and females were 8.9 and 1.0 per 100,000 population respectively, representing a male to female ratio of 9:1 (Table 14). Rates in males were highest in the 40–44 year age group (19 per 100,000), closely followed

Table 14: Notified cases and rates* for infectious syphilis (less than 2 years duration), Australia, 2010, by state or territory†

State or territory	Male		Female		Total	
	Count	Rate*	Count	Rate*	Count	Rate*
ACT/NSW	410	10.9	19	0.5	430	5.7
NT	29	24.4	14	12.7	43	18.7
Qld	192	8.5	29	1.3	221	4.9
SA	16	2.0	5	0.6	21	1.3
Tas	6	2.4	1	0.4	7	1.4
Vic	264	9.6	26	0.9	291	5.2
WA	71	6.1	15	1.3	86	3.7
Total	988	8.9	109	1.0	1,099	4.9

* Notification rate per 100,000 population.

by the 30–34 and 35–39 year age groups (18 and 17 per 100,000 respectively), whereas in females the highest notification rates were observed in the 20–24 year age group followed by the 25–29 and 30–34 year age groups (2.8, 2.2 and 2.1 per 100,000) (Figure 24).

Over the period 2005 to 2007, notification rates amongst males increased substantially, in the 20–29, 30–39 and 40–49 year age groups but since then have either decreased or remained relatively stable. The overall increases observed during this period were mainly attributed to men who have sex with men.[18] In females, for the 2005 to 2010 period, rates remained relatively steady, except in the 15–19 year age group where they decreased from a peak of 7.5 per 100,000 in 2006 to 1.6 per 100,000 in 2010 (Figure 25).

In 2010, data on Indigenous status were complete for 95% of cases. All jurisdictions except the Australian Capital Territory had greater than 50% completeness of the Indigenous status field between 2005 and 2010. The age standardised notification rate was 23 per 100,000 in the Indigenous population and 5.2 in the non-Indigenous population, representing a rate ratio of 5:1. Nationally, there was a 29% decrease in rates for the Indigenous population (from 32.6 per 100,000 to 23.2 per 100,000) between 2005 and 2010 in contrast to the 80% increase (2.5 to 4.5 per 100,000) in the non-Indigenous population during the same period. However, rates varied widely across jurisdictions. In 2010, Indigenous rates in Queensland increased by 34% compared with 2009 but were 20% lower than in 2005, while in the remaining states and territories rates either stayed relatively stable or decreased when compared with 2009. The increase evident in Indigenous rates in Western Australia in 2008 was largely attributable to an outbreak that occurred in 2008 in the Pilbara region amongst Indigenous people (Figure 26).[41] In 2010, rates of

Figure 24: Rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2010, by age group and sex

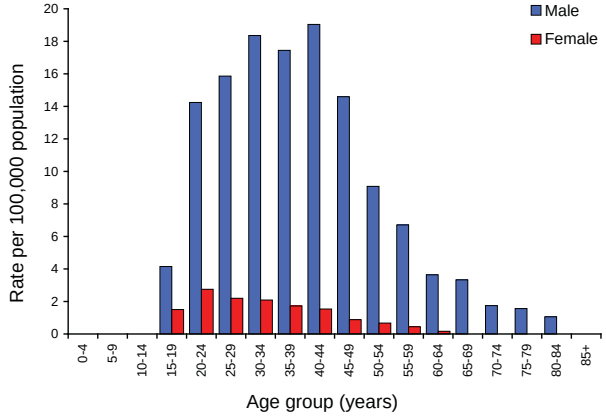
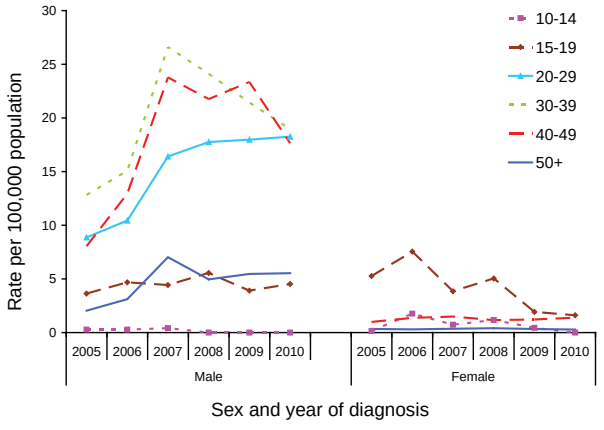
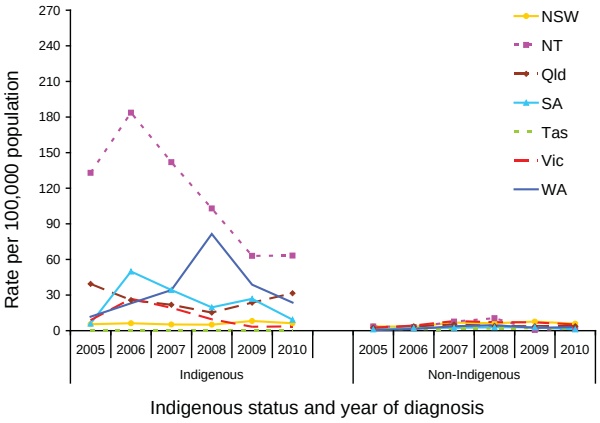


Figure 25: Rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, in persons aged 10 years or over, Australia, 2005 to 2010, by sex, year and age group



infectious syphilis in the Indigenous population were highest in the 20–24 year age group, while in the non-Indigenous population the highest rates were amongst the 30–34 and 40–44 year age groups.

Figure 26: Age standardised rate for infectious syphilis, selected states and territories,* 2005 to 2010, by Indigenous status, year and state or territory



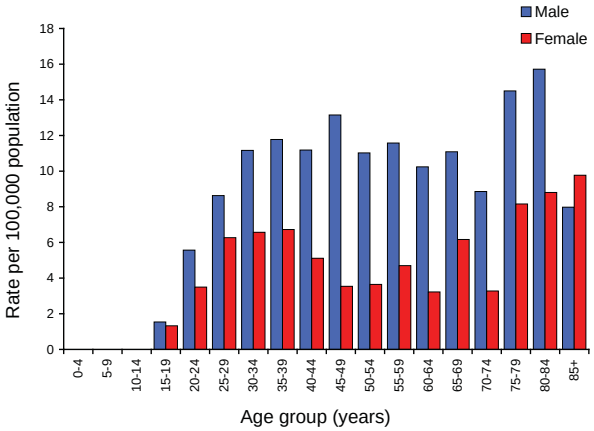
* Includes notifications in the Northern Territory, Queensland, South Australia, Tasmania, Victoria, Western Australia and New South Wales where Indigenous status was reported for more than 50% of cases over a 5-year period.

Syphilis of more than 2 years or unknown duration

In 2010, there were 1,241 notified cases of syphilis of more than 2 years or unknown duration reported to the NNDSS, giving a rate of 6.0 per 100,000 population, which was similar to the rate in 2009 (6.8 per 100,000). The Northern Territory continued to have the highest notification rate (42.7 per 100,000), consistent with 2009 but was a 43% decrease compared with 2008.

In 2010, notification rates of syphilis of more than 2 years or unknown duration in males and females were 7.9 and 4.0 per 100,000, respectively (Table 15), representing a male to female ratio of 1.9:1 (Figure 27). Rates in males were higher than in females across all ages, except in the 85 years or over age group, and were 3 times higher amongst those in the 45–54 and 60–64 year age groups. The distribution of notification rates across age groups in females was bimodal, with the highest rate (9.8 per 100,000) in the 85 years or over age group, followed by those in the 30–34 year age group (6.7 per 100,000). In males, rates remained high in those aged 30 years or over and peaks occurred in the 45–49 and 80–84 year age groups at 13.0 and 16.0 respectively.

Figure 27: Rate for syphilis of more than 2 years or unknown duration, Australia,* 2010, by age group and sex



* Data from all states and territories except South Australia.

Table 15: Notified cases 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	
	n	Rate	n	Rate	n	Rate
ACT/NSW	223	5.9	119	1.6	345	4.5
NT	57	47.9	41	17.9	98	42.7
Qld	114	5.1	69	1.5	183	4.1
SA	NDP	NDP	NDP	NDP	NDP	NDP
Tas	9	3.6	5	1.0	14	2.8
Vic	365	13.3	163	2.9	532	9.6
WA	45	3.9	24	1.0	69	3.0
Total	813	7.9	421	4.1	1,241	6.0

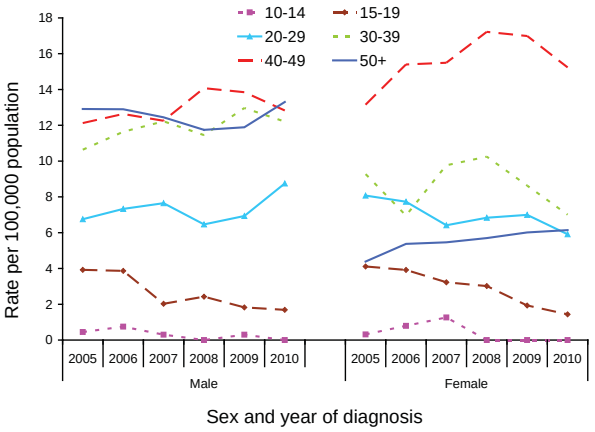
* Notification rate per 100,000 population.

† Data from all states and territories except South Australia.

NDP No data provided.

Over the period 2005 to 2010, rates increased amongst males in all age groups over 19 years but particularly in the 20–29 year age group, which increased by 30% during this time. During this same period a substantial decrease of 57% was observed amongst males in the 15–19 year age group. In contrast, rates for females during this period decreased in all age groups less than 40 years but had a 40% increase amongst those 50 years or older (Figure 28).

Figure 28: Rate for syphilis of more than 2 years or unknown duration, Australia,* 2005 to 2010, by sex, year and age group

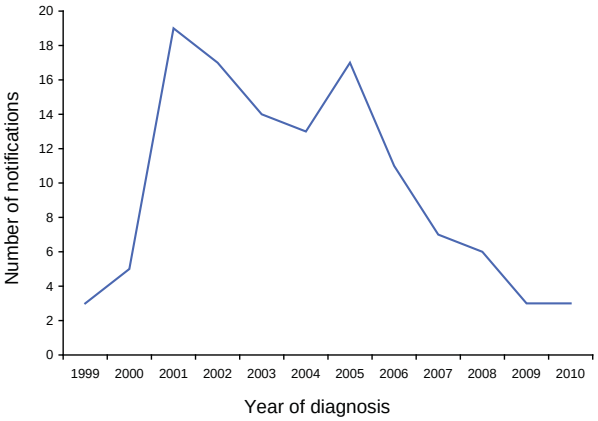


* Data from all states and territories except South Australia.

Congenital syphilis

Following a peak of 19 notified cases in 2001, notifications of congenital syphilis have continued to decline (Figure 29). There were 3 notified cases of congenital syphilis reported in 2010, 2 males from Queensland and 1 female from Western Australia. Two of the cases were reported as Indigenous and one was non-Indigenous.

Figure 29: Trends in notifications of congenital syphilis, Australia, 1999 to 2010, by year



Vaccine preventable diseases

Introduction

This section summarises the national notification surveillance data for notifiable diseases targeted by the National Immunisation Program (NIP) in 2010. 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. 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 was published as the December 2010 supplement issue of CDI. [42]

In 2010, there were 61,964 notified cases of VPDs, representing 30% of all notified cases to NNDSS and a 39% decrease compared with 2009 (n = 102,003). Pertussis was the most commonly notified VPD (n = 34,793, 56% of the total), reflecting the ongoing epidemic of this disease in 2010, followed by influenza (n = 13,419, 22%). The number of notifications and notification rates for VPDs in Australia are shown in Tables 2 and 3.

Whilst there were no new vaccines added to the NIP in 2010, eligibility for the seasonal trivalent influenza vaccine, which included the pandemic (H1N1) 2009 strain was extended to protect a wider range of vulnerable people. Those eligible for the seasonal influenza vaccine under the NIP in 2010 included individuals with medical conditions predisposing them to severe influenza, Aboriginal and Torres Strait Islander people aged 15 years and over, pregnant women, and persons aged 65 years or over. The seasonal influenza vaccine was also available to the rest of the population if they wished to pay for a prescription or were able to obtain the vaccine through workplace or other programs. In addition, the monovalent vaccine developed in response to the 2009 influenza pandemic continued to be available for free to everyone not eligible for the free seasonal vaccine and was distributed through the national Pandemic (H1N1) 2009 Vaccination Program.

Vaccination coverage is an important factor influencing the incidence of vaccine preventable dis-

eases. Since the commencement of the Australian Childhood Immunisation Register in 1996, immunisation coverage in children has been high by international standards, although geographical pockets of lower coverage remain, in which there is an increased potential for VPDs to occur and circulate. These areas 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 can be much higher.[43]

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 for recording 'vaccine type' and 'vaccination date' for each dose. The new fields were intended to replace the old fields, with a transition period allowing either field to be utilised. In 2010, 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 provided by the states and territories 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.

Diphtheria

Diphtheria is an acute toxin-mediated systemic disease caused by the bacterium *Corynebacterium diphtheriae*. Infection is usually localised to the skin (cutaneous diphtheria) or to the throat (pharyngeal diphtheria), in which a membranous inflammation of the upper respiratory tract can cause airway obstruction. Systemic complications caused by the bacteria's exotoxin can occur in both pharyngeal and cutaneous diphtheria. Diphtheria is spread by respiratory droplets or by direct contact with skin lesions or articles soiled by infected individuals.[16] While there are non-toxigenic strains of *C. diphtheriae,* they usually only cause mild throat or skin infection and are not nationally notifiable. In Australia, serosurveillance data indicate that childhood immunity to diphtheria is greater than 99% however, waning immunity amongst adults may result in this population being susceptible with the most likely source of exposure being through overseas travel to countries where diphtheria remains endemic.[44]

There were no notified cases of diphtheria reported to NNDSS in 2010. The last case of diphtheria reported in Australia was a case of cutaneous diphtheria in 2001, the only case reported since 1992.

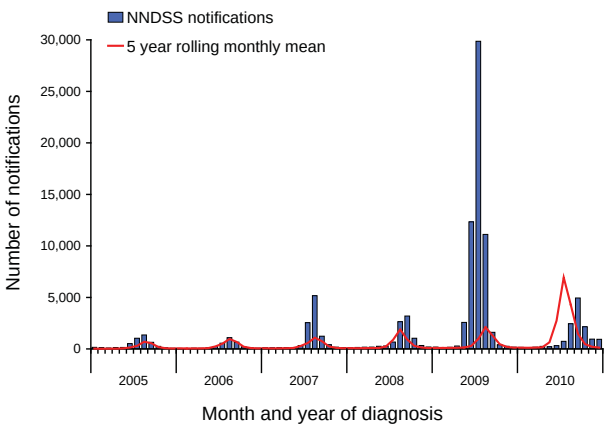
Influenza

Influenza is a viral respiratory infection that causes annual epidemics of respiratory disease. In temperate climates there is usually an increase in influenza transmission during the winter months, from May to October, with the intensity and severity of a season varying from year to year. As only laboratory confirmed cases of influenza are notifiable, it can be difficult to draw conclusions about the true level of influenza activity in the community, due to an unknown proportion of cases where no health care was sought, or no testing performed.

Notifications of influenza decreased substantially in 2010 following the 2009 pandemic year. The 2010 influenza season in Australia was relatively mild, with notification levels comparable to pre-pandemic years. There were 13,419 notified cases of laboratory-confirmed influenza reported to NNDSS in 2010, which was less than a quarter of the number of cases from the previous year. The season peaked in September with 4,944 cases for the month, which was later than in previous years (Figure 30). Higher than usual levels of influenza activity continued across the summer months following the end of the influenza season, and into the following year.

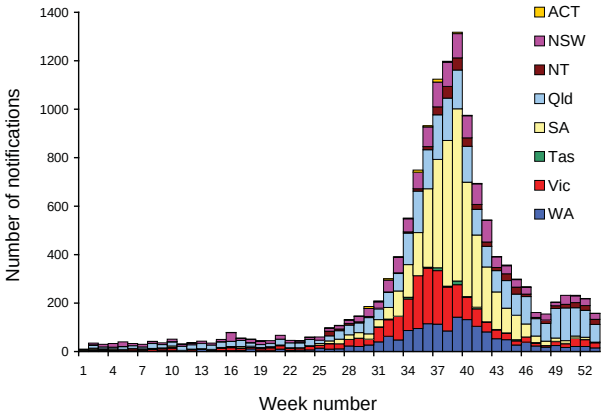
Notification rates were highest in South Australia (258 per 100,000), followed by the Northern Territory (209 per 100,000), with a large gap to the next highest notification rates in Queensland and Western Australian (both 71 per 100,000). Notifications in these jurisdictions were all higher than the national notification rate of 60 per 100,000. Queensland consistently had the highest number of notifications

Figure 30: Notified cases of laboratory-confirmed influenza, Australia, 2005 to 2010, by month and year



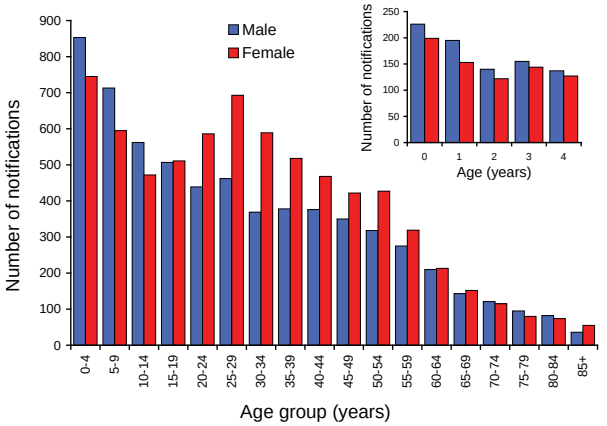
in previous years. In 2010, South Australia replaced Queensland as the jurisdiction with the highest proportion of influenza cases notified (32%), it is thought this increase was due to an actual increase in influenza activity and not an artefact of testing practices (Figure 31).

Figure 31: Notified cases of laboratory-confirmed influenza, Australia, 2010, by week and state or territory



Females accounted for 7,034 (52%) of the 13,419 influenza notifications in 2010. Notifications were higher amongst females than males in most age groups except in the age groups less than 15 years where this was reversed (Figure 32). This likely reflects the health seeking behaviour of adult females, as they tend to account for a greater proportion of encounters in general practice.[45] The highest number of influenza notifications occurred in the 0–4 year age group in both males and females; together they accounted for 12% of all notifications. Over half of the notifications were in people aged less than 30 years.

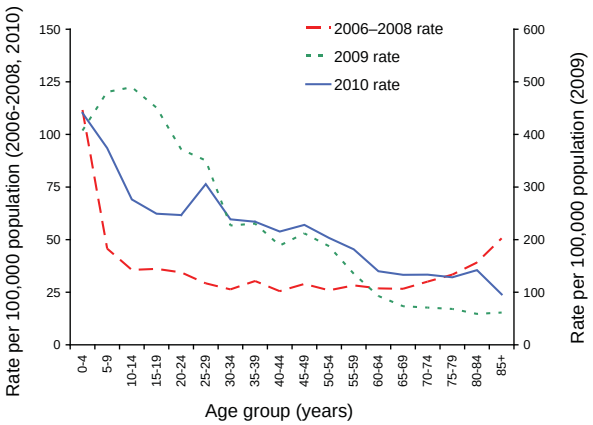
Figure 32: Notified cases of laboratory-confirmed influenza, Australia, 2010, by age group* and sex



* Excludes 96 notifications for whom age or sex were not reported.

Notification rates were highest in the 0–4 year age group (109 notifications per 100,000) with a secondary peak seen in those aged 25–29 years (75 per 100,000), however overall notification rates of influenza decreased with increasing age (Figure 33). Although notifications in 2010 were predominantly the pandemic (H1N1) 2009 strain, the age distribution profile was quite different in the younger age groups in 2010 compared with 2009. In 2009 the highest rates were seen in the 5–9, 10–14 and 15–19 year age groups, with rates in those over 30 years substantially declining relative to the younger age groups. In pre-pandemic seasons, there was typically an increase of notification rates in those aged 70 years and over compared with other adults, this pattern was not observed in 2009 or in 2010 (Figure 33).

Figure 33: Rate for laboratory-confirmed influenza, Australia, 2006 to 2010, by age and year

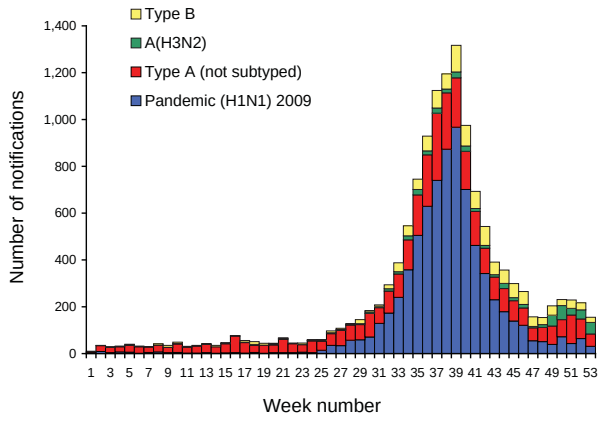


In 2010, almost all (n = 13,402) of the influenza notifications in NNDSS had some level of influenza typing reported. Of those with type information, 90% (n = 12,050) were type A (56% were pandemic (H1N1) 2009, 30% were A (not subtyped) and 4% were A(H3N2)) and 10% (n = 1,301) were type B. Mixed influenza type A and B infections accounted for less than 1% of notifications and typing data were not available for 17 cases (Figure 34).

In 2010, 1,908 Australian influenza viruses were typed and subtyped by the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC). This represented 14% of laboratory-confirmed cases reported to the NNDSS. Pandemic (H1N1) 2009 represented the majority (75%) of viruses, followed by influenza B (14%) and influenza A(H3N2) (11%). Pandemic (H1N1) 2009 replaced previous seasonal A(H1N1) viruses in 2010.

The WHOCC conducted antigenic characterisation on 1,543 of the influenza virus isolates. The vast majority of pandemic (H1N1) 2009 isolates were

Figure 34: Notified cases of laboratory-confirmed influenza, Australia, 2010, by week* and subtype



* Notifications of influenza 'untyped' (n=17) and type A and B (n= 51) were excluded from analysis.

characterised as A/California/7/2009-like. Of the circulating influenza A(H3N2) viruses analysed, most were antigenically similar to the A/Perth/16/2009 virus. Most influenza B viruses detected, were closely related to the B/Brisbane/60/2008 virus.

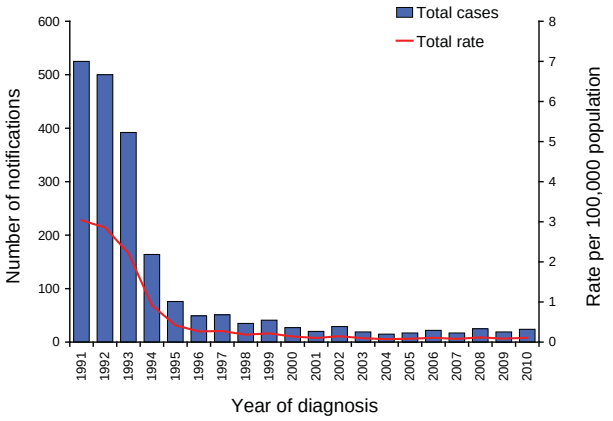
All 3 strains of the 2010 Southern Hemisphere influenza vaccine were different to those previously recommended in the 2009 Southern Hemisphere vaccine. The 2010 vaccine contained A/California/7/2009 (H1N1), an A/Perth/16/2009 (H3N2)-like virus and B/Brisbane/60/2008 (a representative of the B/Victoria/2/87 lineage). Almost all the circulating viruses that were isolated in 2010 were antigenically similar to the 2010 vaccine viruses.[46]

In 2010, the WHOCC conducted antiviral susceptibility testing on 1,320 influenza viruses for resistance to the antiviral drugs oseltamivir and zanamivir. Neuraminidase inhibition assay was performed on 1,277 viral isolates. Four of the pandemic (H1N1) 2009 isolates tested showed resistance to oseltamirvir due to the H275Y neuraminidase mutation. Pyrosequencing of 43 pandemic (H1N1) 2009 clinical specimens found 2 specimens with the same H275Y mutation, which is known to confer oseltamivir resistance. Therefore a total of 6 influenza viruses showed oseltamivir resistance but none were resistant to zanamivir. No oseltamivir or zanamivir resistance was detected in any of the A(H3N2) or influenza B viruses.

Invasive *Haemophilus influenzae* type b disease

Invasive *Haemophilus influenzae* type b (Hib) 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 notified cases in Australia (Figure 35), which now has one of the lowest rates of Hib in the world.[42]

Figure 35: Notified cases and rate for invasive *Haemophilus influenzae* type b infection, Australia, 1991 to 2010, by year



There were 24 notified cases of Hib disease in 2010; a rate of 0.1 per 100,000 population and five more than reported in 2009. The majority of cases (n = 10) were in children aged less than 5 years who had the highest rate of notification (0.7 per 100,000 population), and 60% (n = 6) of which were infants less than one year including 1 case in an infant less than 6 months of age. There were no cases in persons between the ages of 6 and 22 years. The remaining 13 cases ranged in age between 23 and 100 years, and included 4 cases in the 50–54 year age group (Figure 36). The majority, 63% (n = 15) of cases were female, predominately in age groups over 4 years.

Indigenous status was complete for 92% (n = 22) of Hib cases in 2010. Thirty-six per cent (8/22) were reported as Indigenous. The rate for Hib in 2010 was 1.4 in Indigenous people and 0.07 in non-Indigenous people, giving a rate ratio of 20:1. Rates of Hib infection in the Indigenous population fluctuated between 2005 and 2010 from 0.6 to 1.4 and represented a 5– to 27-fold increase compared with rates in the non-Indigenous population. The wide variation in rates was due to the low number of cases. Indigenous status recorded as unknown or missing represented an average of 1.5 cases between 2005 and 2010 and were included in the non-Indigenous category for the purpose of this analysis.

Figure 36: Rate for invasive *Haemophilus influenzae* type b infection, Australia, 2010, by age group and sex

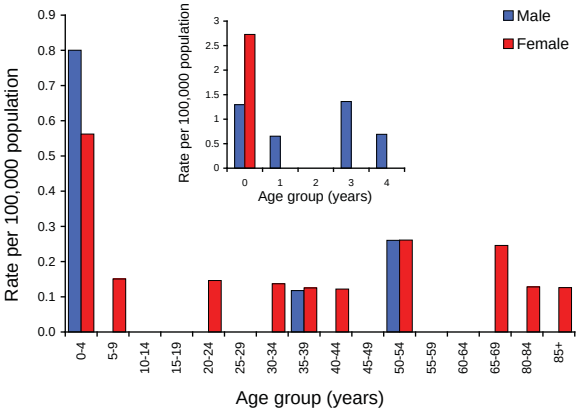
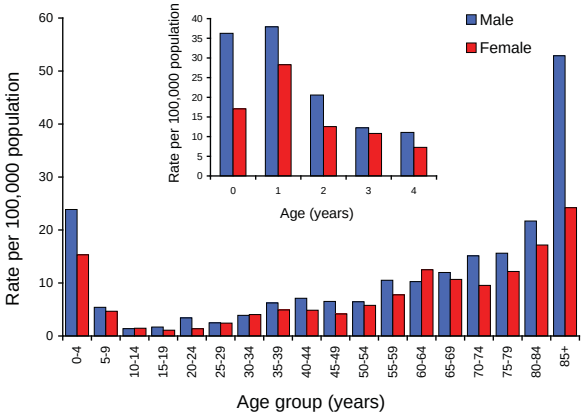


Figure 37: Rate for invasive pneumococcal disease, Australia, 2010, by age group and sex



In 2010, all children under the age of 18 years were eligible for Hib vaccination in infancy. Hib vaccine was introduced to the NIP in April 1993 for all children born after February 1993.

Vaccination status was known for all 10 cases in children aged less than 5 years of which 9 were fully vaccinated for age and 1 was unvaccinated. Of the 9 vaccinated cases, 5 had received all recommended doses of Hib containing vaccine under the NIP.

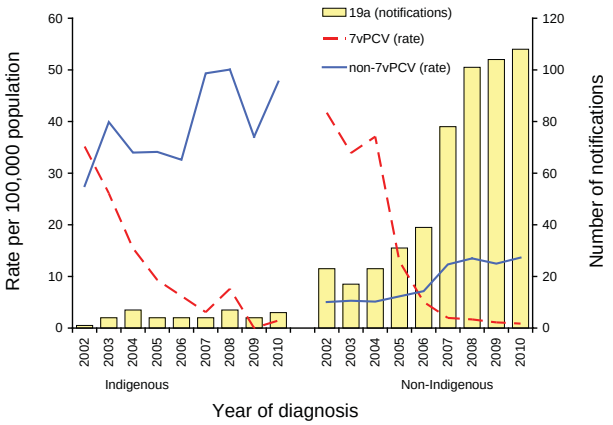
Invasive pneumococcal disease

There were 1,644 notified cases of invasive pneumococcal disease (IPD) in Australia in 2010; a rate of 7.4 per 100,000 population. This was an increase of 6% from the 1,557 reported in 2009 (7.1 per 100,000). An increase in rates in 2010, compared with 2009, was seen in New South Wales (6.9 per 100,000, n = 503) Queensland (6.0 per 100,000, n = 271), Tasmania (9.1 per 100,000, n = 46), Victoria (7.3 per 100,000, 406 cases) and Western Australia (8.6 per 100,000, 198 cases). A decrease in rates was noted in the Australian Capital Territory (6.7 per 100,000, n = 24), the Northern Territory (24 per 100,000, n = 56) and South Australia (8.5 per 100,000, n = 140).

In 2010, males accounted for 56% (914 per 100,000) of the 1,644 notified cases of IPD. In most age groups there were more males than females, resulting in a male to female ratio of 1.3:1. Figure 37 shows that the highest rates of IPD in 2010 were for persons aged 85 years or over (34 per 100,000) and in children aged 1 year (33 per 100,000).

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, older Australians and Aboriginal and Torres Strait Islander people. The 7vPCV vaccine was added to the NIP schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age from January 2005.[11] National pre-vaccination data are not available for the Indigenous population, however since surveillance began in 2002 the rate of disease due to disease caused by serotypes covered by 7vPCV in Indigenous children, aged less than 5 years, decreased from 35 per 100,000 to 1.4 per 100,000 in 2010 (Figure 38). In non-Indigenous children aged less than 5 years, the rates of IPD disease caused by serotypes covered by 7vPCV decreased since the introduction of the vaccine on the NIP in 2005, with a rate of 0.9 per 100,000 reported in 2010. Rates of disease caused by non-7vPCV serotypes over the same period increased for both Indigenous and non-Indigenous children, this included a 5-fold increase in the number of cases due to serotype 19A in non-Indigenous children over the period.

Figure 38: Rate for invasive pneumococcal disease in children aged less than 5 years, 2002 to 2010, by Indigenous status and year



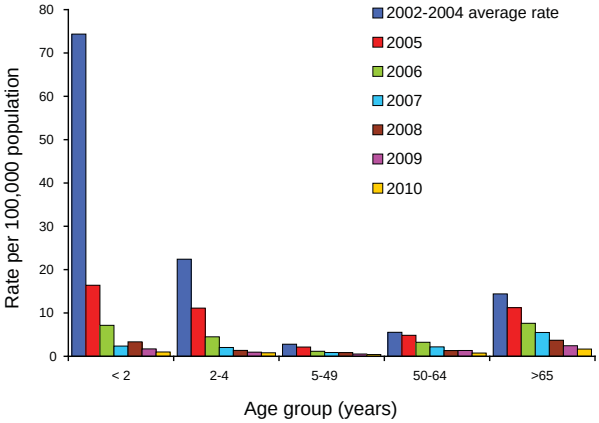
7vPCV	Disease caused by serotypes not covered by the vaccine.
Non-7vPCV	Disease cause by serotypes not covered by the vaccine.

The substantial increases in 19A serotype disease seen in non-Indigenous children were not evident in Indigenous children.

Overall, rates of IPD disease caused serotypes covered by 7vPCV declined between 2004 and 2010 from 7.7 per 100,000 to 0.7 per 100,000 (1,549 to 149 cases). The decline is seen across all age groups (Figure 39).

Enhanced data were collected on cases of IPD in all Australian jurisdictions during 2010. More detailed analyses can be found in the IPD annual report series published in CDI.

Figure 39: Rate for invasive pneumococcal disease caused by 7vPCV serotypes, Australia, 2002 to 2010, by age group and year



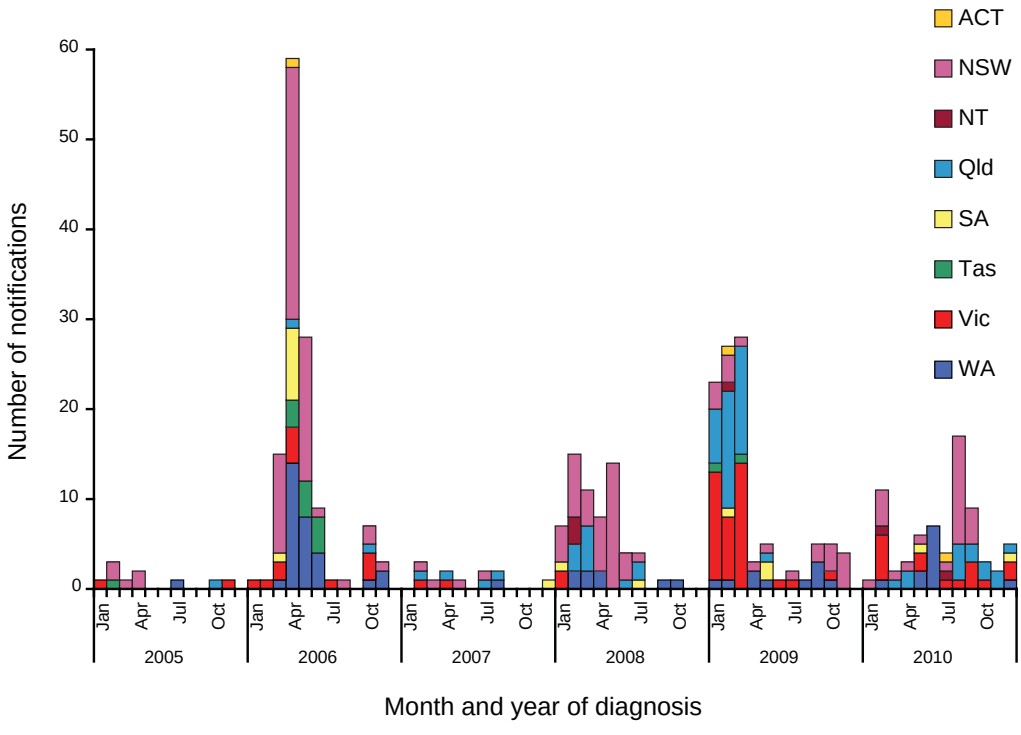
Measles

Measles is a highly infectious, acute viral illness spread by respiratory secretions, including air-borne transmission via aerosolised droplets. The prodrome, lasting 2–4 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,[16] which is always fatal.[11] Evidence suggests that endemic measles has been eliminated from Australia, since at least 2005.[47]

There were 70 notified cases of measles reported to NNDSS in 2010 representing a rate of 0.3 per 100,000 population. Cases were reported from all states and territories with the exclusion of Tasmania. The majority of cases (n = 25) occurred in New South Wales, followed by Victoria (n = 15), Queensland (n = 14), Western Australia (n = 11), the Northern Territory (n = 2), South Australia (n = 2) and the Australian Capital Territory (n = 1). There were no cases in Tasmania (Figure 40).

In 2010, cases were evenly distributed by sex. Age at diagnosis ranged from 1 to 62 years with a median of 23 years and there were no cases amongst infants less than 1 year. The majority of cases (n = 50) were

Figure 40: Notified cases of measles, Australia, 2005 to 2010, by month and year and state or territory



between 10 and 34 years of age. However, 4 cases were amongst those born before 1968, a cohort that is considered to have high levels of natural immunity[48] (Figure 41).

Between 2005 and 2010 there were 386 notified cases, 93% (n = 369) of which were less than 40 years of age. During this 5-year period, rates were highest amongst those less than 5 years of age (0.8 per 100,000 population) followed closely by those in the 10–14 (0.7) and the 25–29 (0.6) year age groups (Figure 42).

In 2010, 46% (n = 32) of notified cases were reported as being acquired from overseas including: South Africa (n = 6), Indonesia (n = 4), France (n = 3), Vietnam (n = 3), Cambodia (n = 2) and 1 importation each from China, Germany, Italy, Malawi, Malaysia, New Zealand, Pakistan, the Philippines, Singapore, Sri Lanka, Thailand, the United Kingdom, France and South Africa. Of the 38 locally-acquired cases, 36 were epidemiologically linked to an imported case in 8 separate clusters and the remaining two were part of a locally-acquired cluster in Victoria, for which the original source of infection could not be determined. There were 9 clusters during 2010, the largest of which occurred in the Tweed River region of New South Wales (n = 14), in an area of low vaccination coverage. Genotyping was available for each cluster with D9 the most common serotype (Figure 43).

Figure 41: Rate for measles, Australia, 2010, by age group and sex

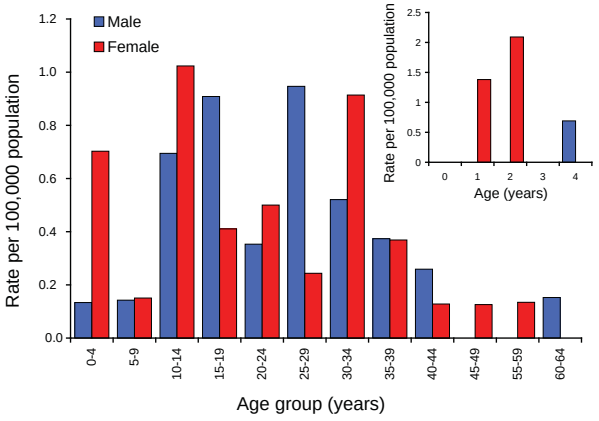


Figure 42: Rate for measles, Australia, 2005 to 2010 combined, by age group

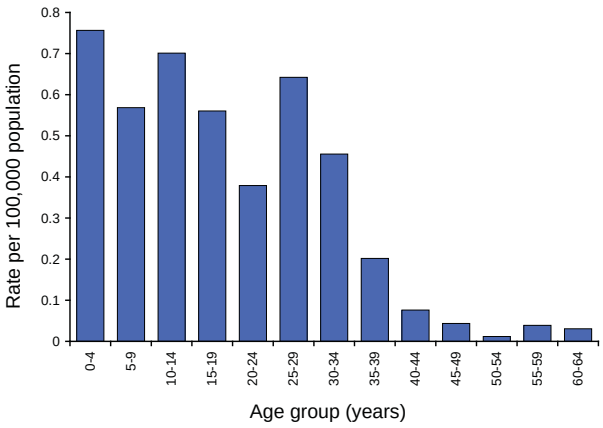
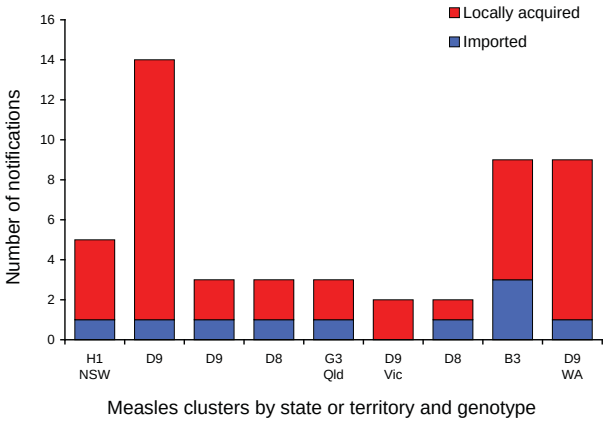


Figure 43: Measles clusters, Australia, 2010, by state or territory, genotype and importation status



Two doses of the measles–mumps-rubella vaccine (MMR) are funded under the NIP for children 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 second dose.[11]

Sixty-four of the 70 cases notified in 2010 were born after 31 December 1969 and therefore eligible for a publicly funded measles-containing vaccine. Of the 5 cases aged between 1 and 3 years of age who were eligible for 1 dose of a measles-containing vaccine, one was fully vaccinated for age, three were not vaccinated and one was of unknown vaccination status. Of the remaining 59 cases, who were aged 4 years or older and eligible for 2 doses, the majority (n = 31) were not vaccinated, nine were partially vaccinated for age, 5 were fully vaccinated for age and 14 were of unknown vaccination status.

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. The characteristic bilateral, or occasionally unilateral, parotid swelling occurs in 60%–70% of clinical cases, however a high proportion have non-specific symptoms including fever, headache, malaise, myalgia and anorexia, with approximately one-third of

infections being asymptomatic.[16] Mumps is a multi-system infection, with 30% of post-pubertal males experiencing epididymo-orchitis.[49]

In 2010, there were 95 notified cases of mumps; a rate of 0.4 per 100,000 population, compared with the 165 cases and a rate of 0.7 per 100,000 reported in 2009. The number of notified cases has continued to decrease nationally since reaching a peak in 2007. Cases in 2010 were reported from all jurisdictions except Tasmania, with 40% (n = 38) occurring in New South Wales followed by 27% (n = 26) in Queensland (Figure 44). The highest rate was in the Northern Territory with 2 reported cases (0.9 per 100,000) followed by Western Australia (0.7 per 100,000) with 15 cases reported in 2010.

In 2010, cases of mumps were notified across all age groups with the majority (n = 45) occurring amongst young adults between the ages of 25 and 44 years, reflecting historical vaccination schedules (Figure 45). Sixty-two per cent of cases were in females; a higher proportion than in the past 5 years and giving a male to female rate ratio of 0.6:1. The highest rates for females occurred in the 25–29, 30–34 and 40–44 year age groups respectively, while for males rates were highest in the 40–44 year age group followed by the 35–39 year age group.

Rates in all age groups have continued to decline in 2010 (Figure 46).

Indigenous status was reported for 50% of mumps cases, of which 4% (n = 2) were reported as Indigenous. In 2009, 10% (n = 11) of cases were reported as Indigenous.

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 first and second dose respectively.[50,51] 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.[51]

Figure 45: Rate for mumps, Australia, 2010, by age group and sex

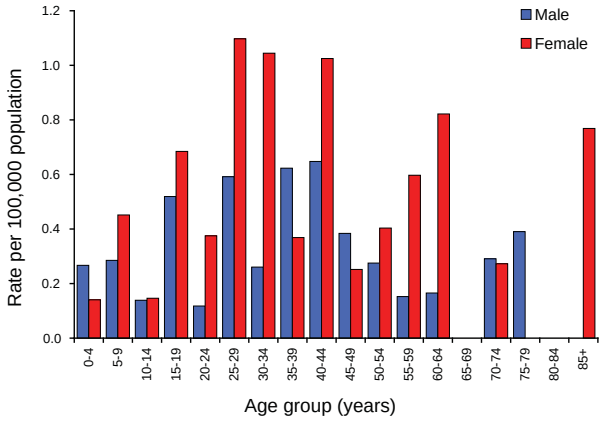
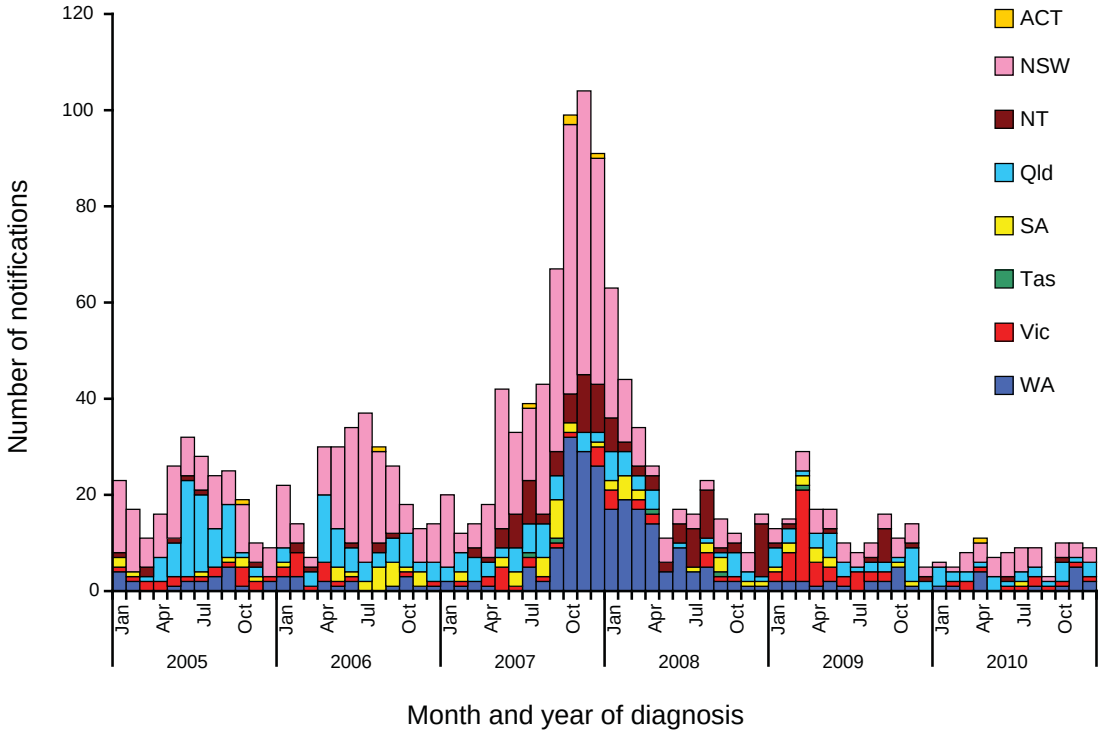
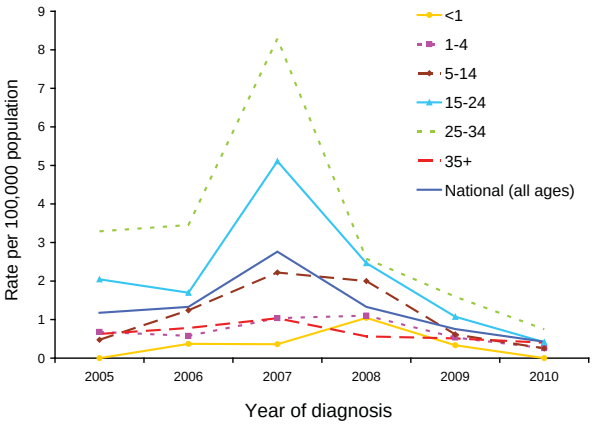


Figure 44: Notified cases of mumps, Australia, 2005 to 2010, by month and year and state or territory



The mumps vaccine was first available in Australia in 1981 with people born after that eligible for 2 doses of a mumps-containing vaccine.[52] In 2010, there were 37 notified cases in individuals born after 31 December 1980. Of these, none were less than 1 year of age, two were aged between 1 and 3 years and eligible for 1 dose and were fully vaccinated for age. The majority of cases (35/37) were aged 4 years or older. Of these, 17% (n = 6) were fully vaccinated for age, 6% (n = 2) were partially vaccinated for age, 11% (n = 4) unvaccinated and the majority, (66%) were of unknown vaccination status.

Figure 46: Rate for mumps, Australia, 2005 to 2010, by year and age group



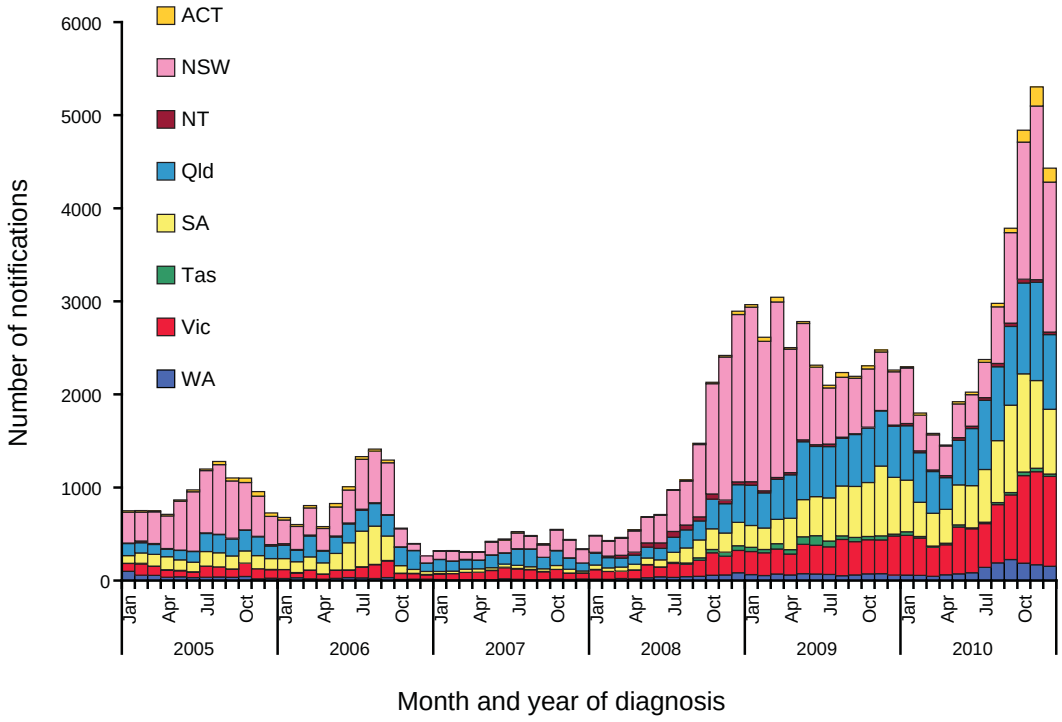
Pertussis

Pertussis was the most commonly notified vaccine preventable illness in Australia in 2010. It is a highly infectious disease caused by *Bordetella pertussis* and is 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.[53] In vaccinated populations these outbreaks tend to be smaller with less mortality and morbidity than in unvaccinated populations.[16] 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 at least the second vaccine dose at 4 months of age.[54] The majority of notifications usually occur in the spring and summer months.

In 2010 there continued to be large numbers of notified cases of pertussis associated with the Australia-wide epidemic which began in mid-2008 (Figure 47). The causes of this epidemic are likely to be multi-factorial with contributing factors including waning immunity levels in the vaccinated population in addition to improved testing methods and better case ascertainment.

In 2009, the Australian Technical Advisory Group on Immunisation (ATAGI) convened a Pertussis Working Party to consider the use of the combined

Figure 47: Notified cases of pertussis, Australia, 2005 to 2010, by month and year and state or territory



diphtheria-tetanus-pertussis (DTPa) vaccine in young children, and the duration of effectiveness of the diphtheria-tetanus-pertussis (dTpa) vaccine in adolescents/adults. On the basis of evidence provided by the working party, ATAGI endorsed recommendations that the first dose of the pertussis-containing vaccine could be brought forward from 8 weeks to 6 weeks,[55] the scheduled fourth dose of vaccine could be administered from the age of 3 years and 6 months, and that the adolescent booster dose could be given from 11 to 13 years of age to better protect siblings, especially newborns.[56] States and territories continued to provide ongoing public awareness campaigns and most extended funding during 2010 for booster vaccination programs for parents and carers of infants, as part of a cocooning strategy to protect vulnerable infants from infection.

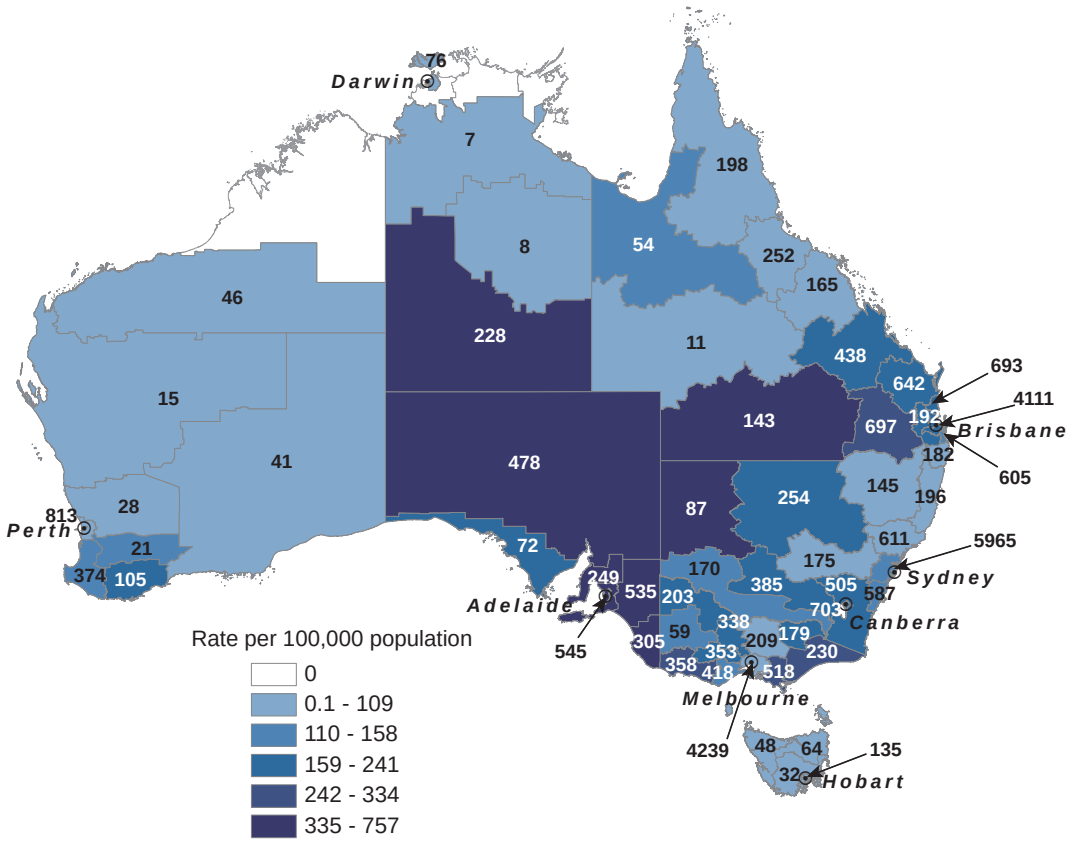
There were 34,793 notified cases of pertussis; a rate of 156 per 100,000 and 2.5 times the 5-year mean. Notifications included 3 pertussis related deaths Two of the deaths were recorded amongst infants less than 2 months of age and too young to be protected by vaccination, while the third death was a person 70 years of age.

In 2010, pertussis rates varied considerably by state or territory and residential location. Rates were highest in South Australia (449 per 100,000; n = 7,388) followed by the Australian Capital Territory (198 per 100,000; n = 712) and Queensland (182 per 100,000; n = 8,216) (Figure 48). Rates by SD varied widely across most jurisdictions except for South Australia where they were uniformly high (Map 3).

The timing of epidemic activity has varied across states and territories with the Northern Territory experiencing its peak rate in 2008 (217 per 100,000; n = 478), New South Wales (175 per 100,000; n = 12,448) and Tasmania (123 per 100,000; n = 618) in 2009 and Australian Capital Territory, Queensland, South Australia, Victoria and Western Australia in 2010 (Figure 48).

In 2010, females accounted for 57% (n = 19,950) of notifications, resulting in a male to female ratio of 0.7:1. Sixty cases had no sex specified. Females had higher rates in all age groups compared with males, except in the 85 years or over age group. Notification rates in 2010 varied widely with age. Children aged less than 15 years had a higher rate (321 per 100,000)

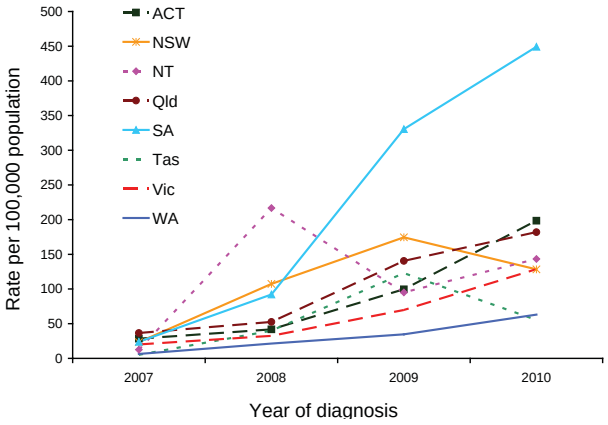
Map 3: Rates and counts* for pertussis, Australia, 2010, by Statistical Division and Statistical Subdivision of residence in the Northern Territory



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

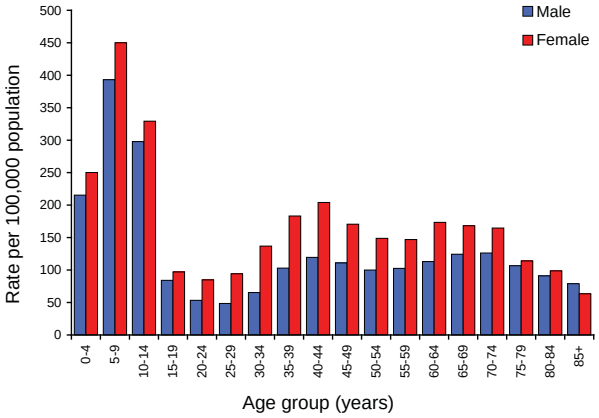
Note that rates by Statistical Division should be interpreted with caution as they can be high or low depending on the size of the population.

Figure 48: Rate for pertussis, Australia, 2007 to 2010, by year and state or territory



than those adolescents and adults 15 years of age or over (117 per 100,000), with a rate ratio of 2.7 and consistent with the rate ratios in 2008 (2.2) and 2009 (2.6). The current epidemic trend of higher rates in children compared with adults is in contrast to the pre-epidemic years in which adults had a higher rate relative to children (rate ratios of 0.7, 0.3 and 0.5 respectively for 2005, 2006 and 2007). The highest rate amongst both males and females occurred in the 5–9 year age group (393 and 450 per 100,000 respectively) (Figure 49).

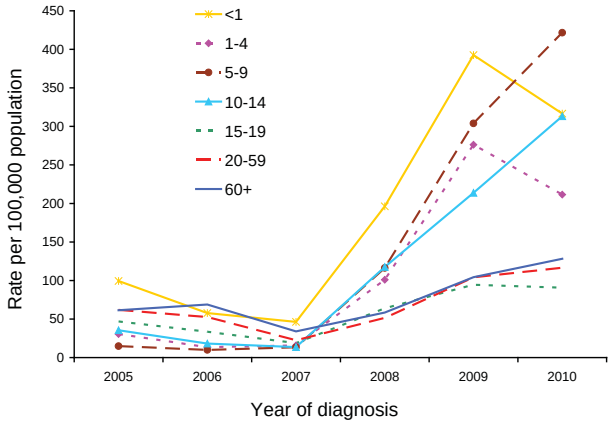
Figure 49: Rate for pertussis, Australia, 2010, by age group and sex



In 2010, rates amongst children in the 5–9 year age group (422 per 100,000) overtook infants aged less than 1 year (317 per 100,000) to have the highest rate for the first time since 2005 (except for 2006 in which adults 60 years or over had the highest rate and infants the second highest).

Between 2005 and 2007, a period inclusive of the last national epidemic in 2005/2006, age group rates either declined or remained relatively constant. Since 2007, rates have been increasing most markedly amongst those less than 15 years of age. In 2010 rates continued to increase all age groups, especially the 5–9 and 10–14 year age groups, compared with 2009, except those less than 5 years of age (Figure 50).

Figure 50: Rate for pertussis, Australia, 2005 to 2010, by year and age group



While the pertussis vaccine is not 100% effective and does not confer life-long immunity, vaccine effectiveness amongst Australian children has been estimated to range from 82% to 89% with the lower figure representing the cohort of children who would not have been eligible for the 18 month booster dose, which was removed from the NIP in 2003.[57] Immunity to the disease decreases over time post-vaccination with estimates of protection remaining for 4–12 years.[58] 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). In response to the ongoing epidemic in 2010, some infants were being provided their first vaccination at 6 weeks of age and their fourth from 3.5 years.

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 for those less than 5 years of age. This age group made up 10% (n = 3,400) of all notified cases in 2010.

Information on vaccination status was available for 89% (3,030/3,400) of all cases in children less than 5 years of age of which 64% (1,944/3,030) were fully vaccinated for age, 15% (447 per 100,000) were partially vaccinated for age and 13% (379 per 100,000) were not vaccinated. Eight per cent (260 per 100,000) of cases were less than 6 weeks of age and therefore too young to be vaccinated.

Poliomyelitis

Poliomyelitis is a highly infectious disease caused by gastrointestinal infection by poliovirus. Transmission occurs primarily 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).[16]

In 2010 there were no notified cases of poliomyelitis in Australia, which along with the Western Pacific Region remained poliomyelitis free. Poliomyelitis is a notifiable disease in Australia with clinical and laboratory investigation conducted for cases involving patients of any age with a clinical suspicion of poliomyelitis. Australia follows the WHO protocol for poliomyelitis surveillance and focuses on investigating cases of 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, which in 2010, Australia achieved for the fourth consecutive year. More details can be found in the annual report of the Australian National Polio Reference Laboratory published in the CDI. A revised national polio case definition was endorsed by CDNA in 2010 and implemented on 1 July 2011. This revised definition is available on the Department of Health and Ageing's web site at http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd_polio.htm. The principal changes were:

- the WHO definition of AFP was adopted under clinical definitive evidence;
- the laboratory definitive evidence was updated to include vaccine derived poliovirus; and
- a new section was added to include non-paralytic cases of poliovirus infection.

Rubella

Rubella is generally a mild and self-limiting viral infectious disease. It is spread person-to-person through contact with respiratory secretions directly or via air-borne droplets. Clinically, rubella can be difficult to distinguish from other diseases which cause a febrile rash, such as measles, and is asymptomatic in up to 50% of cases. Rubella infection in pregnancy can result in 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.[16]

In 2010, there were 44 notified cases of rubella; a rate of 0.2 per 100,000 population and an increase compared with the 27 notifications in 2009. The increase in cases in 2010 was not associated with any particular outbreak and was likely due to the sporadic nature and overall small number of cases reported annually. Notifications were reported from Victoria (n = 22), New South Wales (n = 13), Queensland (n = 5), Western Australia (n = 3) and the Australian Capital Territory (n = 1). The male to female ratio of notified cases in 2010 was 1.9:1, (29 males and 15 females). The majority (87%) of female cases were notified in women of childbearing age (15–44 years of age). The majority (86%) of cases were adults aged between 20 and 49 years with a median age of 29.5 years (Figure 51). There were no notified cases of CRS reported in 2010.

Rubella cases across the age groups have continued to trend at low levels since 2004, except for a spike amongst the 25–34 year age group in 2006 (Figure 52). This spike was primarily due to an increase in cases from South Eastern and Central Sydney, New South Wales for which no single source was identified.[59]

A single dose of rubella vaccine produces an antibody response in more than 95% of recipients and

Figure 51: Rate for rubella, Australia, 2010, by age group and sex

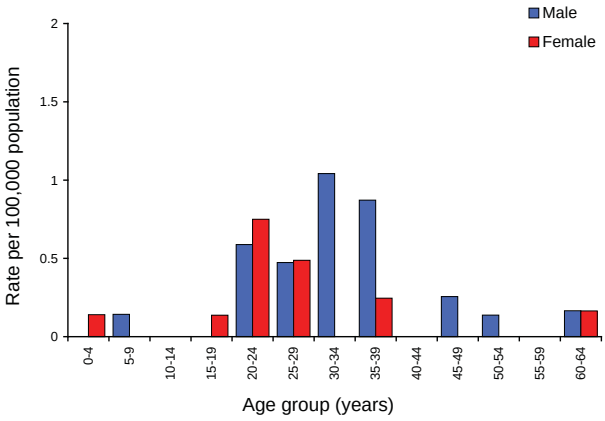
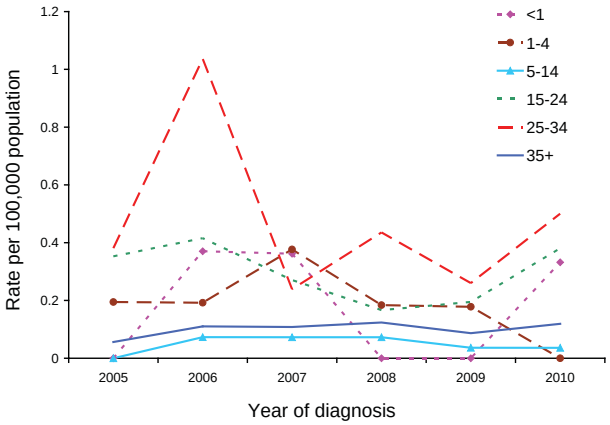


Figure 52: Rate of rubella, Australia, 2005 to 2010, by year and age group



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.[11] Rubella vaccine is included in the combined MMR vaccine and provided under the NIP schedule at 12 months and 4 years of age.

Information on vaccination was available for 25% (n = 11) of rubella cases of which the majority, 73% (n = 8), were not vaccinated, two were reported as fully vaccinated for age and one was too young for routine vaccination.

Indigenous status was recorded for the majority (91%) of cases, all of whom were non-Indigenous.

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.[16] 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.[11]

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 vaccinees have low or undetectable levels of immunity. It is recommended, though not funded under the NIP, that all adults who reach 50 years of age and have not received a booster of a tetanus-containing vaccine in the previous 10 years should have one.[11]

In 2010, there were 2 notified cases of tetanus, 1 male from New South Wales and 1 female from Victoria, both greater than 78 years of age. Neither case had vaccination status recorded.

Varicella zoster virus infections

The varicella zoster virus (VZV) is a highly contagious member of the herpesvirus family and causes two distinct illnesses: chickenpox (or varicella) following initial infection and shingles (or herpes zoster), which occurs following re-activation of latent virus in approximately 20%–30% of cases, most commonly after 50 years of age.[16]

In 2006, CDNA agreed to make 3 categories of VZV infection notifiable: chickenpox, shingles and varicella infection unspecified. With the exception of New South Wales, where VZV is not notifiable, 2010 was the second complete year in which all jurisdictions sent VZV data to NNDSS.

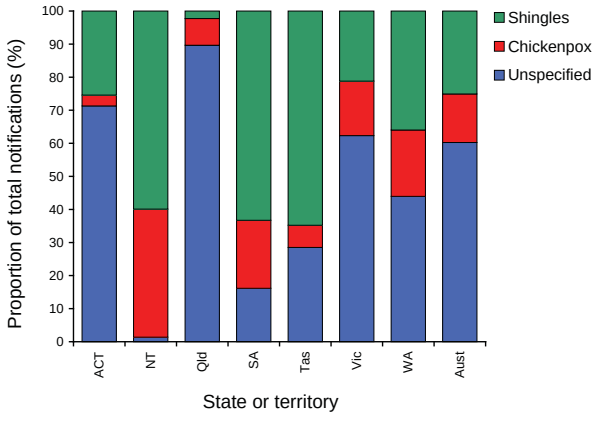
In 2010, there were 11,873 VZV notified cases from the 7 reporting jurisdictions. This was 6% more than in 2009. Sixty per cent (n = 7,152) were reported as unspecified varicella infection, 25% (n = 2,978) as shingles and 15% (n = 1,743) as chickenpox.

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 53), VZV unspecified accounted for 60% of all VZV notified cases in 2010, a decrease compared with 62% of the total in 2009.

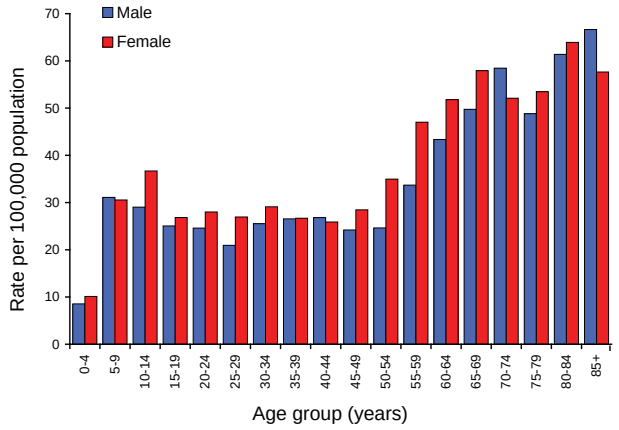
There were 7,152 notified cases of unspecified VZV infections based on laboratory diagnoses compared with 6,977 in 2009 and a rate of 47 per 100,000 population The high proportion of unspecified VZV infection compared to chickenpox or shingles is attributable to the varying capacity of jurisdictions to follow-up on laboratory notifications to determine the clinical presentation of each case. The highest rate was reported from Queensland (86 per 100,000; n = 3,894), followed by Western Australia (38 per 100,000; n = 877) and Victoria (34 per 100,000; n = 1,912).The age and sex distribution of unspecified VZV is shown in Figure 54.

Figure 53: Proportion of notified cases for varicella zoster virus unspecified, chickenpox and shingles, 2010, by state or territory*



* Excluding New South Wales

Figure 54: Rate for varicella zoster virus infection (unspecified), Australia,* 2010, 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.[11]

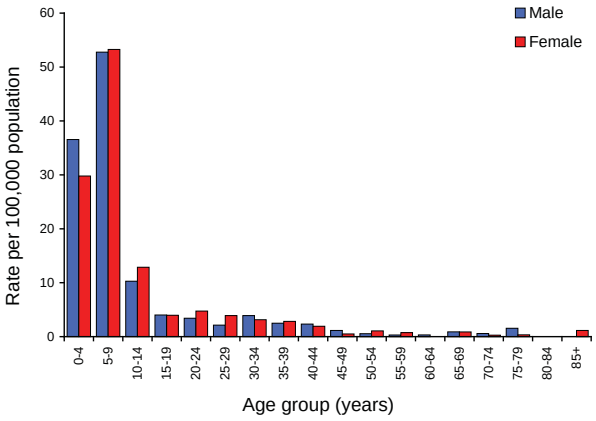
In 2010, there were a total of 1,743 notified cases of chickenpox reported to NNDSS; a rate of 12 per 100,000, compared with 1,599 in 2009. The highest rate was reported from the Northern Territory (37 per 100,000; n = 84) and South Australia (23 per 100,000; n = 379) reflecting the increased case ascertainment in these jurisdictions compared with others.

The male female ratio in 2010 was 1:1 with males and females being fairly consistently represented across the age groups. Seventy per cent of cases (n = 1,212) occurred in children aged less than 10 years. The highest notification rates amongst both sexes and all age groups were amongst the 5–9 year age group (53 per 100,000; n = 725) (Figure 55). Although higher rates amongst children compared with adults is expected, they are likely to be biased by the jurisdictional practice of not following up adult cases.

Indigenous status was recorded for 89% (n = 1,552) of cases. Of these, 93% (n = 1,447) were non-Indigenous.

In November 2005, the varicella zoster vaccine was added to the NIP Schedule as a single dose due at 18 months of age (for children born on or after 1 May 2004), or as a catch-up dose at 10–13 years of age. In 2010, children born in 2004 and eligible for the 18-month dose would be 6 years of age or younger and as follow-up of cases does not routinely occur in those older than 7 years, analysis of vaccination status is restricted to this cohort. Of the 823 children less than 7 years of age, vaccination information was available for 77% (636/823) of cases of whom 44% (n = 277) were vaccinated, 16% (n = 102) were unvaccinated and 40% (n = 257) were aged less than 18 months and therefore ineligible for vaccination.

Figure 55: Rate for chickenpox, Australia,* 2010, by age group and sex



* Excluding New South Wales.

Shingles

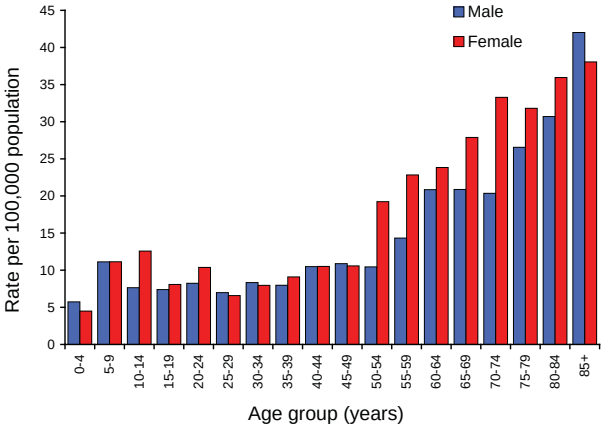
Shingles occurs most commonly with increasing age, impaired immunity, and a history of chickenpox in the first year of life. Reactivation of VZV causing shingles is thought to be due to a decline in cellular immunity to the virus, and in the majority of cases presents clinically as a unilateral vesicular 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.[16]

There were 2,978 notified cases of shingles reported in 2010, a rate of 20 per 100,000 and a 10% increase compared with 2009. The highest rate was in South Australia (71 per 100,000; n = 1,166) followed by the Northern Territory (57 per 100,000; n = 130), reflecting the increased case ascertainment in these jurisdictions compared with others.

There were more female cases (n = 1,670) than males (n = 1,305); a ratio of 0.8:1. As expected, rates

increased with age with the highest rate amongst those over 85 years of age or older (61 per 100,000; n = 159) (Figure 56).

Figure 56: Rate for shingles, Australia,* 2010, by age group and sex



* Excluding New South Wales.

Indigenous status was recorded for 87% (n = 2,579) of notified cases, of whom 97% (n = 2,489) were non-Indigenous.

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 most concern in Australia are typically mosquitoes that are able to transmit viruses or parasites to humans.

There were 8,244 notified cases of mosquito-borne diseases (4% of total notifications), which was similar to the number of cases in 2009 (n = 8,232). 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 (a parasitic disease caused by *Plasmodium* spp). Yellow fever is reported under quarantinable diseases.

Rates of infection for a geographical location 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 (NAMAC) annual report 2009–10.[60]

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).[61] The reservoirs of these viruses are mammals, particularly macropod marsupials. In Australia, BFV and RRV are the alphaviruses of major public health significance, accounting for 80% (n = 6,618) of vectorborne disease notifications.

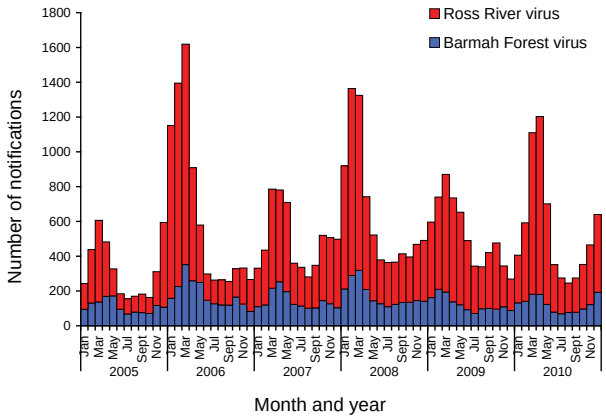
Barmah Forest virus infection

There were 1,471 notified cases of BFV in Australia in 2010; a rate of 6.6 per 100,000 population compared with the 5-year mean of 8.3 per 100,000 population (Figure 57). The highest rates of BFV notifications were reported by the Northern Territory (35.7 per 100,000), and Queensland (20.1 per 100,000). Cases were reported in all jurisdictions. The median age of cases was 47 and 51% of cases were in males.

Ross River virus infection

There were 5,147 notifications of RRV infection in Australia in 2010, a rate of 23 notifications per 100,000 population compared with a 5-year mean of 21.4 per 100,000 population (Figure 57). Nearly half of all cases were from Queensland (46%, n = 2,383), but the highest rate was in the Northern Territory (146.3). The median age of cases was 43 and 45% of notifications were in males.

Figure 57: Notified cases of Barmah Forest and Ross River virus infections, Australia, 2005 to 2010, by month and year



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 virus (DENV).

The Sentinel Chicken Program is a surveillance scheme involving New South Wales, the Northern Territory, Victoria and Western Australia. Chicken flocks are located in strategic locations and are regularly tested for antibodies to MVEV and KUNV. This program is designed to provide early warning of flavivirus activity (excluding DENV and JEV).[62] A sentinel chicken surveillance report was published as part of the NAMAC annual report 2009–10.[60]

Dengue virus infection

There were 1,201 notified cases of DENV infection reported in 2010, which was a 14% decrease from the 1,402 cases reported in 2009 (Figure 58). In 2010, the median age of dengue cases was 39 years and 52% of cases were in males.

Whilst the number of dengue cases overall decreased compared with previous years, the number of overseas-acquired cases was the highest on record (Figure 58) and most cases of dengue in 2010 were acquired overseas (93%, n = 1,119). Where the country of acquisition of overseas cases was provided (85%, n = 1,019), the most frequently reported country was Indonesia (66%, n = 673), followed by Thailand (11%; n = 109).

The increasing number of overseas-acquired dengue cases in Australia is likely to be due in part to the increasing frequency of travel to countries such as Indonesia, but also to the increasing incidence of dengue in the South East Asian region. In 2010, over 2.2 million cases of dengue were reported to the WHO from the Americas, South East Asia and Western Pacific.[63] Dengue is now endemic in more than 100 countries, compared with 40 years earlier, when only 9 countries had experienced severe epidemics.

Overseas acquired cases were most frequently reported amongst younger and middle-aged adults (Figure 59) particularly females in the 20–24 year age group (n = 75), reflecting the frequency of overseas travel for these age groups. Amongst older adults (aged 50+ years), overseas-acquired dengue was more common amongst males (62% of cases were male).

Figure 58: Notified cases of dengue virus infection, Australia, 2005 to 2010, by month and year and place of acquisition

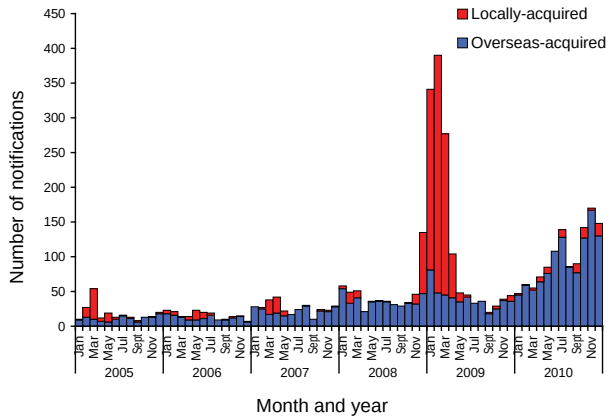
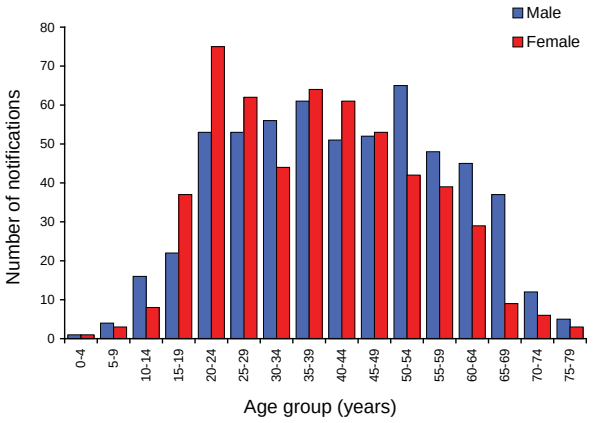


Figure 59: Notified cases of overseas-acquired dengue, Australia, 2010, by age group and sex



Local transmission of dengue virus 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. In 2010, the proportion of cases that were locally acquired was much lower than in 2009 (66%), and there were no significant local outbreaks. The majority of locally-acquired cases were reported from Queensland, with a small number from other states (n = 9), but all were acquired in North Queensland. In 2010, majority of locally-acquired cases that were typed, were due to serotype 2 (91%, 52/57).

No cases in 2010 were reported to have been fatal. Dengue-related deaths have been very rare in Australia. A death reported in March 2009 and 2 deaths in early 2004 were the first deaths attributed to dengue in over 100 years.[64]

Japanese encephalitis virus infections

Japanese encephalitis is the leading cause of childhood encephalitis in Asia.[65] The usual host of the virus is birds or pigs, and it is transmitted to humans through the bite of infected mosquitoes of the genus *Culex*. There were no notified cases of JEV infection reported to the NNDSS in 2010. JEV infection is rare in Australia with only 3 cases reported in the past 10 years, all acquired overseas.

JEV emerged in Australia in 1995 with an outbreak in the Torres Strait. In an outbreak in 1998, a further 2 cases were reported, one of them acquired on the mainland (Cape York Peninsula).[66] Seasonal incursions of JEV have been detected (usually in sentinel pigs) in the Torres Strait every year except 1999.[65] Targeted vaccination programs of residents of the Torres Strait commenced in 1995.

Kunjin virus infection

There were 2 notified cases of KUNV infection reported in 2010; one from Queensland and one from the Northern Territory. Between 2005 and 2009, there were 8 notifications of KUNV infection.

Murray Valley encephalitis virus infection

During 2010, there were no notifications of MVEV infection reported. There were 9 notifications between 2005 and 2009 (Table 6).

Arbovirus infections (NEC)

In 2010, there were 24 notified cases of arbovirus infection not elsewhere classified (NEC). There was 1 notification each from New South Wales and Queensland, 10 from the Northern Territory and 12 from Victoria. The median age of cases was 36 years and 33% of cases were male.

Ten cases were chikungunya virus infection, reported by the Northern Territory, one was unspecified, and the remainder were flavivirus infections (Kokobera [1], not further specified [13]).

Malaria

Malaria is a serious acute febrile illness which can be transmitted to humans through the bite of an infected mosquito. It is caused by parasites of the genus *Plasmodium* that includes 5 species that cause disease in humans – *vivax, falciparum, malariae,knowlesi* and *ovale*.[16] There were 399 notified cases of malaria in Australia in 2010, which was down from 526 in 2009.[67]

All cases in 2010 were acquired overseas. Australia was declared malaria free in 1981, and since then, there have been 2 reported outbreaks of locally-acquired malaria; in 1986 and 2002 with a total of 15 cases. Where the country of acquisition was available (84%, n = 337 notifications), the most frequently reported country was Papua New Guinea (28%, n = 94, with 53 of these reported from Queensland), followed by India (16%, n = 53).

Malaria was most frequently reported amongst males aged 20–29 years with 69% of all malaria cases being males (Figure 60).

The infecting *Plasmodium* species was reported for 97% of malaria notifications in 2010 (Table 16). The predominant infecting species were *P. falciparum* (45%) and *P. vivax* (43%).

Zoonoses

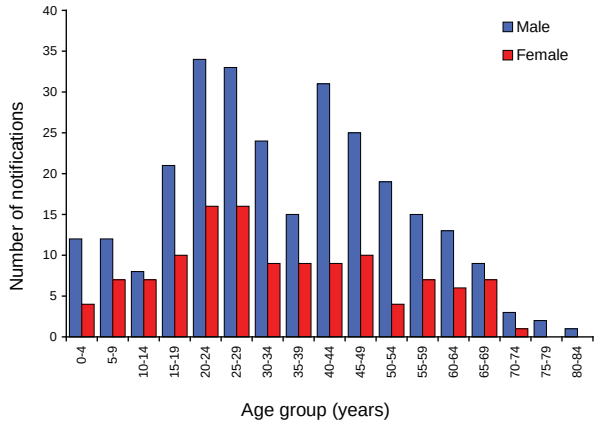
Zoonoses are 'those diseases and infections which are naturally transmitted between vertebrate animals and man'.[68] Approximately 60%–70% of emerging human infectious diseases are zoonoses[69,70] and more than 70%

Table 16: Notified cases of malaria, Australia, 2009, by parasite type and state or territory

	State or territory								Aust	
Plasmodium species	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	n	%
P. falciparum	1	58	3	53	3	2	26	35	181	45
P. vivax	0	44	8	64	4	1	37	15	173	43
P. ovale	0	12	0	3	0	1	2	1	19	5
P. malariae	0	5	0	1	0	0	1	3	10	3
P. falciparum and *P. vivax**	0	0	0	0	0	0	1	1	2	1
P. falciparum and *P. malariae**	0	0	0	0	0	0	0	1	1	0
Plasmodium species	1	5	0	5	1	1	0	0	13	3
Total	2	124	11	126	8	5	67	56	399	100

* 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 60: Notified cases of malaria, Australia, 2010, by age group and sex



of emerging zoonoses originate from wildlife.[69] 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'.[71]

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 2010, 532 notified cases of these zoonotic diseases were reported to the NNDSS. Queensland accounted for 47% (n = 251) and New South Wales 33% (n = 173) of notified cases. Notifications were generally more frequent amongst males (77%, n = 412). There were only 5 notified cases (< 1%) of zoonotic disease in persons aged less than 15 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.[16] Anthrax has a low incidence in animals, and occurs only sporadically in Australia.[72] It can be an occupational hazard for veterinarians, and agriculture, wildlife and industry livestock workers who handle infected animals or animal by-products.

One case of anthrax was reported to NNDSS in 2010. The case occurred in New South Wales in February 2010. Over the previous 10 years, only 2 human cases of anthrax were reported in Australia. Both cases were cutaneous anthrax and were reported in 2006 and 2007.[73,74] Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

In 2010, 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.

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.[75] No notified cases of either Australian bat lyssavirus infection (ABL), rabies or lyssavirus (unspecified) infections were reported to the NNDSS during 2010. 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.[76,77] Surveillance indicates that ABL 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.[78] Testing of bats conducted by the Australian Wildlife Health Network between January and June 2010 yielded 4 ABL detections compared with 12 detections in bats during 2009.[79]

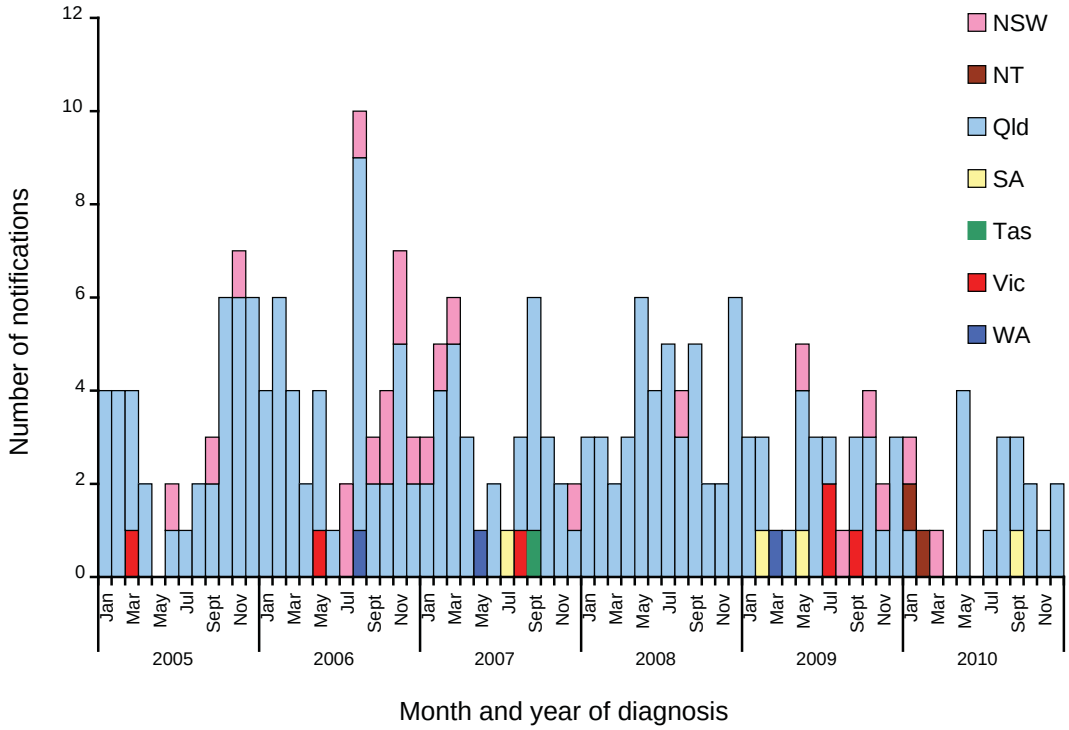
Brucellosis

Several *Brucella* species can infect both animals and humans including *Brucella melitensis* from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989[72] and *B. melitensis* has never been reported in Australian sheep or goats.[72] All human cases of *B. melitensis* or *B. abortus* in Australia are related to overseas travel. *B. suis* is confined to some areas of Queensland, where it occurs in feral pigs.

Internationally, brucellosis is mainly an occupational disease of farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[16] In Australia, 83% of cases since 1991 have been reported from Queensland, where feral pig hunting is the most common risk factor for infection.[80]

In 2010, there were 21 notified cases of brucellosis reported to the NNDSS; a 49% decline in notifications compared with the 5-year average of 41 cases (Figure 61). Seventy-six per cent of notifications

Figure 61: Notified cases of brucellosis, Australia, 2005 to 2010, by month and year and state or territory*



* There have been no cases reported from the Australian Capital Territory.

were from Queensland (n = 16). Most cases were in males (81%, n = 17) aged between 15 and 49 years (85%, n = 18).

The species of the infecting organism was available for 38% of notifications (n = 8), of which seven were *B. suis* (all from Queensland, and all in males aged between 27 and 43 years). There was 1 imported case of *B. melitensis,* which was acquired in Iraq.

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 certain agricultural sectors and swimming or wading in contaminated water).[16]

In 2010, there were 131 notified cases of leptospirosis reported; giving a rate of 0.6 per 100,000 population compared with the 5-year mean of 128.0 notifications. Cases were reported in all jurisdictions, but Queensland accounted for 64% (n = 84) of notifications (Figure 62). Eighty-seven per cent (n = 127) of leptospirosis cases were male and 82% (n = 120) of all cases were aged between 15 and 54 years (Figure 63).

The WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis provides an annual surveillance report of leptospirosis cases that are sent for typing.‡ In 2010, the reference centre typed 94 cases of leptospirosis. The most frequently identified serovars were Arborea (21% n = 20, Australis (16%, n = 15), Zanoni (15%, n = 14), and Hardjo (15%, n = 14).81 In 2009, Serovar Arborea was the most frequently reported serovar, accounting for 29% of all notifications.[82] The last reported death in Australia attributed to leptospriosis was in 2002.

Ornithosis

Ornithosis (or psittacosis) 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 species of parrot. 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.[16] Person-to-person transmission is rare.

‡ Reference laboratory numbers consist of data submitted to/by the reference laboratory and are reported by notification date, thus numbers will not necessarily be the same as those reported from NNDSS in this report.

Figure 62: Notified cases of leptospirosis, Australia, 2005 to 2010, by month and year and state or territory

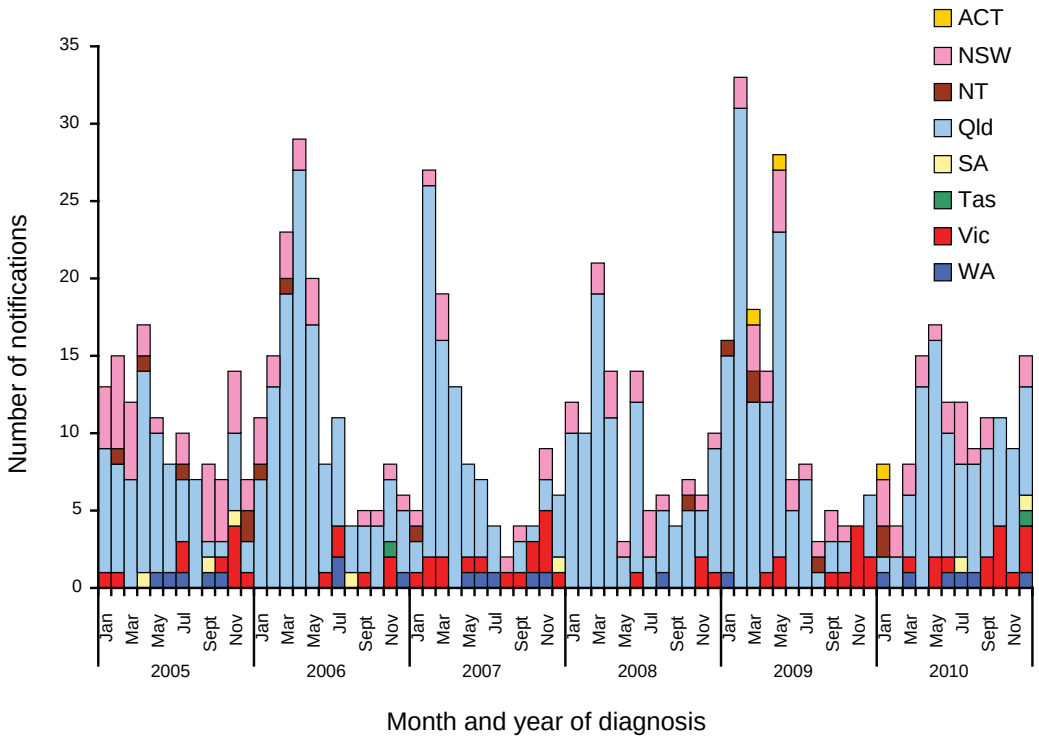
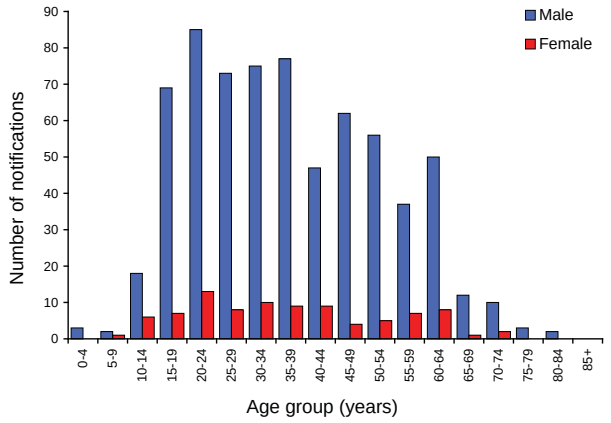


Figure 63: Notified cases of leptospirosis, Australia, 2010, by age group and sex



In 2010, there were 56 notified cases of ornithosis reported; giving a rate of 0.3 per 100,000 population. The number of ornithosis notifications has declined steadily in recent years (Figure 64), and case numbers in 2010 are the lowest since 2001.

Notifications were from all states and territories except the Northern Territory, but the majority of notifications were from Victoria (64%, n = 36). This represents a change from the previous 5 years, where the majority of cases were from New South Wales (53%, 312/589). Sixty-six per cent of cases in 2010 were male (39 cases). All cases were aged 20 years or older and 83% were aged 40 years or older. Cases of ornithosis over the previous 5 years have been mainly in adults, with a median age of 54 years (Figure 65).

Individuals 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.[83]

Q fever

Q fever is caused by infection with the bacterium, *Coxiella burnetii*. The primary reservoirs of these bacteria are cattle, sheep and goats. *C. burnetii* is resistant to environmental conditions and many common disinfectants.[16] Q fever is most commonly transmitted via the airborne route, where the organism is carried in dust contaminated with tissue, birth fluids or excreta from infected animals.[84] It can also occur through 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. Prior to vaccination programs in Australia, approximately half of all cases in New South Wales, Queensland and Victoria were amongst abattoir workers.[16,85,86] The Australian Government previously funded the National Q Fever Management Program between 2001 and 2006 for states and territories to provide

Figure 64: Notified cases of ornithosis, Australia, 2005 to 2010, by month and year and state or territory

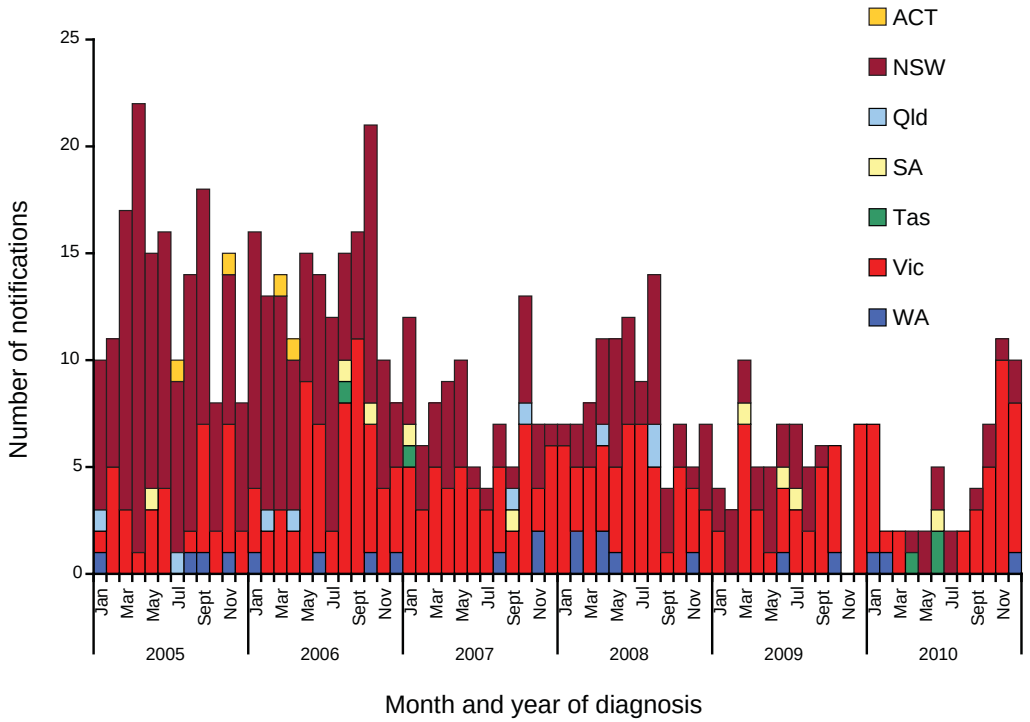


Figure 65: Notified cases of ornithosis, Australia, 2005 to 2010, by age group and sex

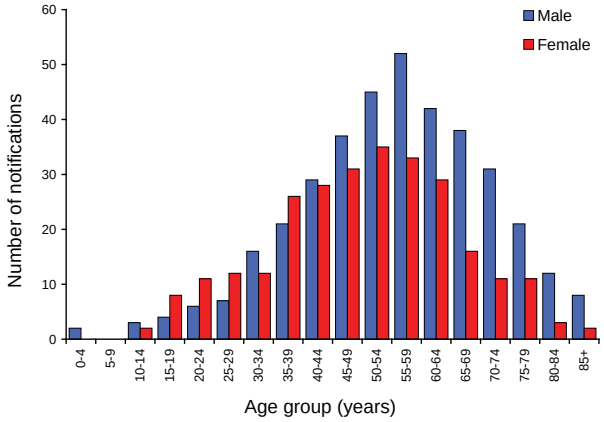
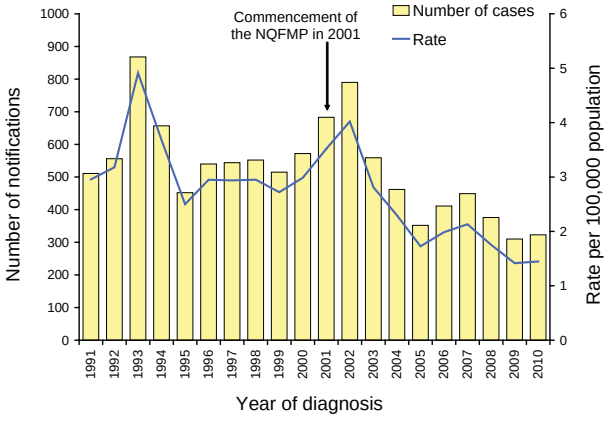


Figure 66: Notified cases of Q fever, Australia, by year



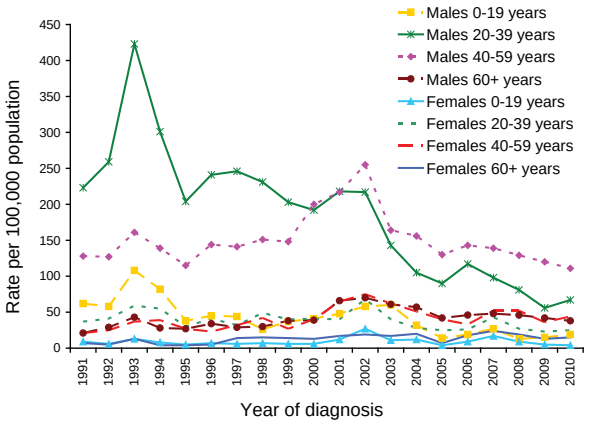
free vaccine to at risk groups (such as abattoir workers). The Australian Government has secured the supply of vaccine through to 2016.

In 2010, there were 323 notified cases of Q fever reported to the NNDSS; a rate of 1.4 per 100,000 population. Between 1991 and 2001, and prior to the introduction of the National Q Fever Management Program, Q fever notification rates ranged between 2.5 and 4.9 per 100,000 population (Figure 66).

In 2010, the highest notification rates were from Queensland (151; 3.3 per 100,000 population) and New South Wales (136; 1.9 per 100,000 population). Cases also occurred in Victoria (n = 16), South Australia (10 cases) and Western Australia (n = 8). There was 1 case each in the Australian Capital Territory and the Northern Territory.

Between 1991 and 2010, Q fever cases have been most frequently reported amongst males aged between 20 and 59 years, and it is in these groups that are likely to be at highest risk of infection where the declines in notifications are most pronounced (Figure 67). Whilst the ending of drought conditions may have contributed to the decrease, it is likely that vaccination programs have been highly effective at preventing Q fever amongst those most at risk.

Figure 67: Notified cases of Q fever, Australia, by year, age group and sex



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. Q fever vaccine may cause an adverse reaction in a person who has already been exposed to the bacterium. Vaccine is not recommended for children under 15 years of age.[11]

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 notified cases of tularaemia in 2010, and no cases in any previous years.

Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2010 and classified as 'other bacterial infections' in the NNDSS. A total of 1,866 notifications were included in this group in 2010, which accounted for less than 1% of all the notifications to NNDSS, a decrease in cases and a similar proportion as in 2009 (n = 1,911 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. There were 298 notified cases of legionellosis reported in 2010, giving a national rate of 1.3 per 100,000 and consistent with the 302 cases reported in 2009 (Figure 68). Rates for states and territories ranged from 0.9 per 100,000 in Queensland to 2.4 in Western Australia in 2010.

Data on the causative species were available for 91% of cases; the majority were *L. longbeachae* (46%) and *L. pneumophila* (45%) (Table 17).

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

Table 17: Cases of legionellosis, Australia, 2010, by species and state or territory

Species	State or territory								Aust	Total %
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Legionella longbeachae	0	47	2	17	20	1	5	46	138*	46
Legionella pneumophila	0	39	1	18	9	5	53	8	133†	45
Legionella bozemanii	0	0	0	0	0	0	1	0	1	0.3
Unknown species	4	7	0	7	0	0	8	0	26	9
Total	4	93	3	42	29	6	67	54	298	100

* Two deaths.

† Five deaths.

eastern states (Queensland, New South Wales and Victoria). However, similar to 2009, *L. longbeachae* was notified more frequently than *L. pneumophila* in New South Wales and almost as frequently in Queensland in 2010.

Six of the 8 *L. pneumophila* cases reported in Western Australia in 2010 acquired their infections in Bali, Indonesia and five of these cases stayed at a particular hotel in Kuta, Bali. An additional 4 cases associated with this hotel or a nearby exposure source were identified in Victoria from travellers recently returned from Bali. Disease onset for these 10 cases ranged between 10 August 2010 and 1 January 2011.

In 2010, diagnoses of legionellosis were highest in May (11%; n = 34,) and August (n = 33) (Figure 68). *L. pneumophila* occurred most frequently in the autumn months, with 46 cases reported over the period March to May 2010 (Figure 69). Twenty-one cases of *L. pneumophila* were reported in May 2010, the largest number of cases diagnosed in a month since 23 cases were reported in March 2006. *L. longbeachae* cases peaked in spring 2010, with 45 cases reported over the period September to November 2010, the majority (n = 21) of which occurred in November.

Males accounted for 65% of legionellosis cases in 2010, with a male to female ratio of 1.9:1. There were no cases in people under the age of 15 years. The notification rate was highest in the 75–79 year age group (5.3). The highest age and sex-specific rates were observed in men aged 80–84 years (7.9) and women aged 75–79 years (4.4) (Figure 70).

Analysis of infecting species by age group showed that 89% (123/138) of *L. longbeachae* notifications were in persons aged 45 years or older, with the highest rate in the 75–79 year age group (2.4). Similarly, the proportion of *L. pneumophila* infections in persons 45 years or older was 87% (116/133), with the highest rate in the 70–74 year age group (2.5).

Figure 69: Notified cases of legionellosis, Australia, 2005 to 2010, by month and year and organism

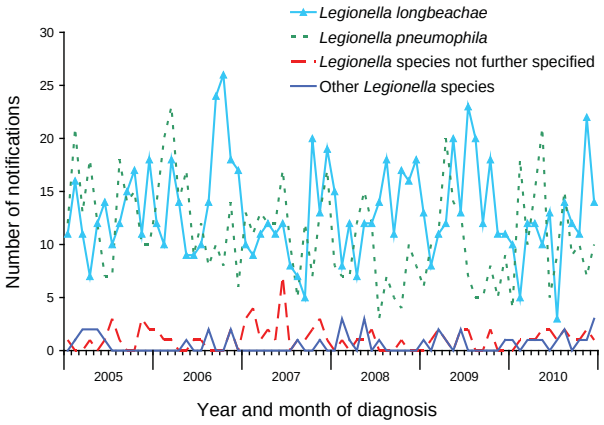


Figure 68: Notified cases of legionellosis, Australia, 2005 to 2010, by month and year and state or territory

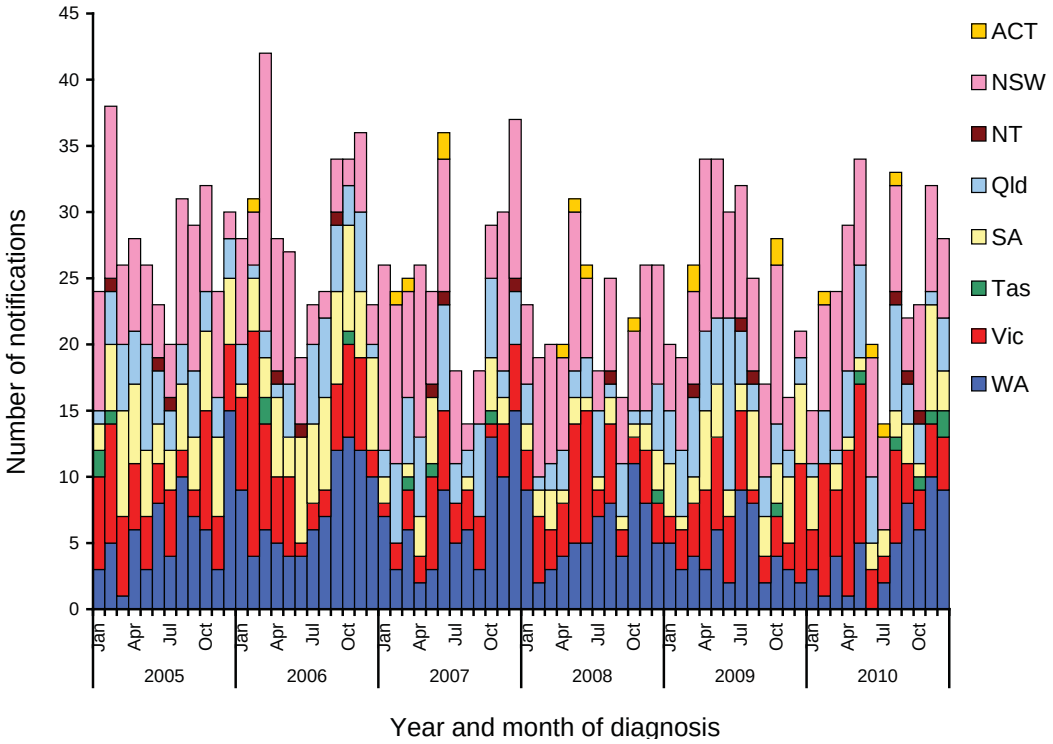


Figure 70: Rate for legionellosis, Australia, 2010, by age group and sex

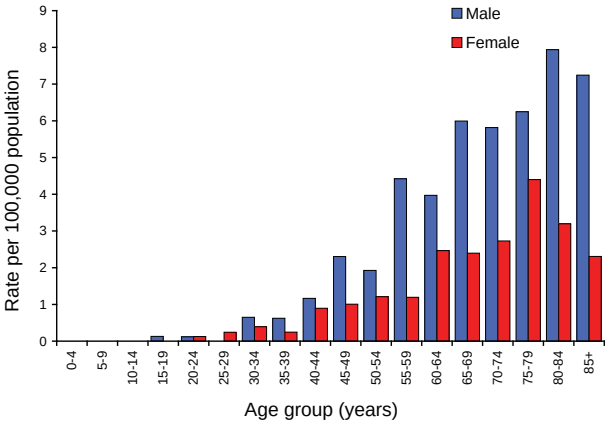
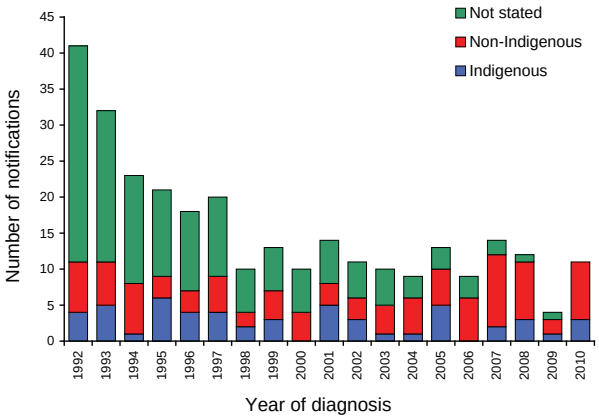


Figure 71: Notified cases of leprosy, Australia, 1992 to 2010, by year and Indigenous status



Mortality data were available for 58% of notifications. There were 7 reported deaths due to legionellosis in Australia in 2010, which was a decrease from 10 reported deaths in 2009. Those who died ranged in age between 55 and 86 years (median 76 years); 5 deaths were males and 2 deaths were females. There were 5 deaths associated with *L. pneumophila* infection and 2 deaths were associated with *L. longbeachae* (Table 17). 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 71.

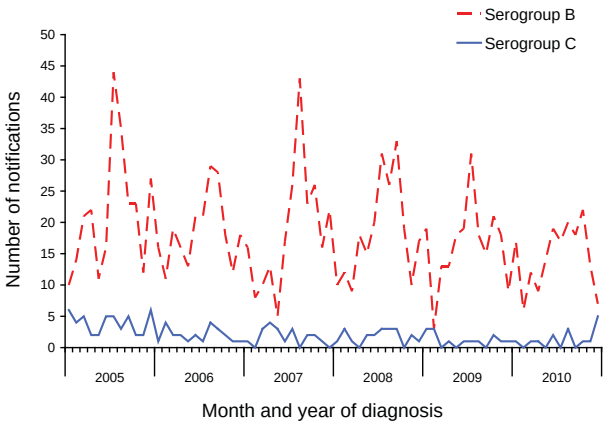
In 2010, 11 notified cases of leprosy were reported (8 male, 3 female), compared with 3 cases in 2009 and 11 in 2008. The majority of cases were reported from Victoria (n = 4) followed by Western Australia (n = 3) and Queensland (n = 2) with one each from New South Wales and the Northern Territory. Three cases were identified as Indigenous. Ten of the 11 cases were adults aged 24 years or older (range 24–55) and the remaining case was a 10-year-old.

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 meningococcus. Globally, serogroups A, B, C, W135 and Y most commonly cause disease.[16] 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 2010, there were 230 notified cases of IMD; an 11% decrease from 259 cases in 2009, and the lowest number since 1996. Rates have halved from 2 to 1 case per 100,000 between 2004 and 2010. During 2010, case numbers started to rise in May and remained elevated over the winter months in a clear seasonal pattern before declining from a peak in October (Figure 72).

Figure 72: Notified cases of invasive meningococcal disease, Australia, 2005 to 2010, by month and year and serogroup



Cases were evenly distributed amongst males and females in 2010 with 116 and 114 cases respectively. Ninety-four per cent of notified cases (n = 217) met the national case definition as 'confirmed' and the remaining 6% (n = 13) were classified as 'probable', based on clinical symptoms alone.

Ninety per cent of IMD cases in 2010 had serogroup data available of which 76% were caused by serogroup B organisms, 7% by C (Figure 72), 4% by W135 and 3% by Y (Table 18); a similar distribution to 2009.

Although there is no vaccine available to protect against serogroup B disease, the rate for IMD due to serogroup B organisms has continued to decline, particularly in the 0–4 and 5–9 year age groups over the period 2005 to 2010 (Figure 73). The highest age-specific IMD rate (6) in 2010 was in children aged 0–4 years. Of the cases reported in this age group, 80% were serogroup B. The highest rate for serogroup B infection in 2010 was 4.7 in the 0–4 year age group (n = 69), representing a 40% rate decline from 2005 (7.8, n = 101). There was a corresponding 50% decline in the 5–9 year age group from a rate of 1.6 (n = 22) in 2005 to 0.8 (n = 11) in 2010.

Notification rates for IMD due to serogroup C infections remained low in most age groups in 2010. The largest decline has been in the 0–4 year age group, decreasing from a rate of 0.6 (n = 8) in 2005 to 0.1 (n = 2) in 2010 (Figure 74).

In 2010, vaccination information was recorded for 3 of the 4 notified cases of serogroup C disease who were eligible for the meningococcal C vaccine (aged between 1 and 26 years in 2010) of which one was vaccinated and two were unvaccinated.

Mortality data for IMD were available for 50% of cases reported to the NNDSS in 2010. Of these, there were 14 deaths due to IMD (10 serogroup B, 1 serogroup C and 1 serogroup W135 and two of unknown serogroup) (Table 19). This was an increase from 10 deaths in 2009 (although mortal-

Figure 73: Rate for serogroup B invasive meningococcal disease, Australia, 2005 to 2010, by year and select age group

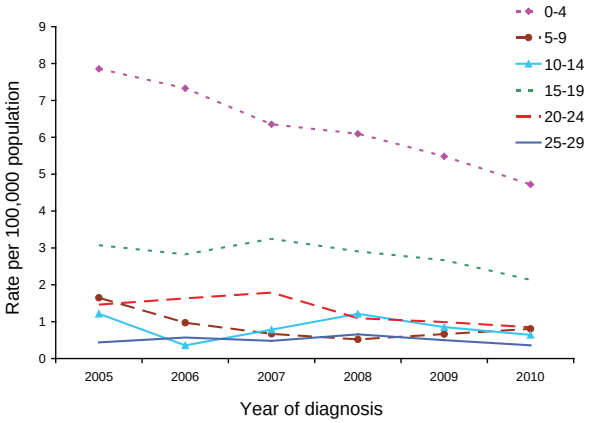


Table 18: Notified cases of invasive meningococcal disease, Australia, 2010, by serogroup and state or territory

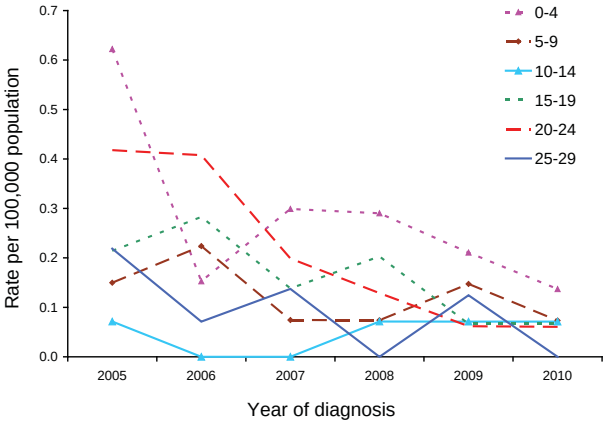
Serogroup	State or territory								Aust	% of total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
B	1	50	2	41	22	5	34	19	174	76
C	0	6	0	5	2	0	1	1	15	7
W135	0	4	1	1	0	0	4	0	10	4
Y	0	3	0	0	1	0	3	1	8	3
Unknown	0	13	0	6	0	1	2	1	23	10

Table 19: Deaths due to invasive meningococcal infection, Australia, 2010, by serogroup and state or territory

Species	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Serogroup B	0	3	0	1	1	0	2	3	10
Serogroup C	0	0	0	0	0	0	0	1	1
Serogroup W135	0	1	0	0	0	0	0	0	1
Serogroup Y	0	0	0	0	0	0	0	0	0
Serogroup unknown	0	1	0	1	0	0	0	0	2
Total deaths	0	5	0	2	1	0	2	4	14

ity data completeness in NNDSS for 2009 was only 38%). 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.

Figure 74: Rate for serogroup C invasive meningococcal disease, Australia, 2005 to 2010, by year and select age group



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. Annual reports of the AMSP are published in CDI.

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 issue in the overseas-born and Indigenous communities. In 2010, 1,327 notified cases of TB were reported to NNDSS, a rate of 5.9 and consistent with the rate reported in 2009 (6) and 2008 (5.6). TB rates were higher than the national average in the Northern Territory (13), Victoria (7.8) and New South Wales (6.6), and the lowest rate occurred in Tasmania (2).

Further details and analysis of TB cases can be found in the tuberculosis annual report series which is published in CDI.

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Australian Meningococcal Surveillance Programme
Australian Sentinel Practice Research Network
Australian Quarantine Inspection Service
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Abbreviations

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

Appendices

Appendix 1: Mid-year estimate of Australian population, 2010, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aus
Males	178,639	3,588,734	119,052	2,257,344	812,591	250,434	2,751,566	1,164,553	11,124,254
Females	180,255	3,650,085	110,623	2,259,017	832,051	257,192	2,795,961	1,131,858	11,218,144
Total	358,894	7,238,819	229,675	4,516,361	1,644,642	507,626	5,547,527	2,296,411	22,342,398

Source: ABS 3201.0 Population by Age and Sex, Australian States and Territories. June 2010 population.[7]

Appendix 2: Mid-year estimate of Australian population, 2010, by state or territory and age

Age group	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aus*
0–4	24,134	462,888	18,761	314,493	98,912	33,655	353,610	154,511	1,461,088
5–9	20,969	441,773	17,556	289,868	93,857	30,803	328,154	142,614	1,365,747
10–14	20,974	450,467	16,762	297,328	100,272	33,168	335,668	148,933	1,403,778
15–19	24,224	479,867	16,844	314,118	107,978	34,983	365,523	157,285	1,501,010
20–24	31,023	523,865	18,952	330,942	116,277	31,807	423,869	172,796	1,649,659
25–29	31,963	539,075	21,122	333,893	111,581	29,236	423,522	174,699	1,665,263
30–34	27,892	502,698	18,884	303,580	101,966	28,255	391,259	159,367	1,534,043
35–39	27,501	522,538	18,579	328,642	110,337	32,838	407,324	168,128	1,616,051
40–44	25,166	489,989	17,003	314,179	113,724	33,936	392,503	165,950	1,552,666
45–49	25,113	508,660	16,133	318,201	117,841	36,908	386,780	165,206	1,575,053
50–54	23,501	475,539	14,554	293,227	113,131	36,589	360,605	153,040	1,470,376
55–59	20,934	428,644	12,422	265,641	104,417	34,231	322,990	136,531	1,326,013
60–64	18,294	394,475	9,380	244,649	97,333	32,177	295,661	120,433	1,212,537
65–69	12,204	301,563	5,683	182,739	72,983	24,543	222,554	87,136	909,512
70–74	8,856	238,870	3,381	135,883	58,841	18,865	178,661	67,030	710,444
75–79	6,507	188,238	1,799	100,842	47,486	14,643	141,709	50,371	551,621
80–84	5,010	151,442	1,131	77,708	40,323	11,076	114,215	38,393	439,306
85+	4,629	138,228	729	70,428	37,383	9,913	102,920	33,988	398,231
Total	358,894	7,238,819	229,675	4,516,361	1,644,642	507,626	5,547,527	2,296,411	22,342,398

Source: ABS 3201.0 Population by Age and Sex, Australian States and Territories. Jun 2010 population.[7]

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2010, 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
Anthrax	0	0	0	1	0	0	1	100.0	1	0
Cholera	1	0	0	2	0	0	3	100.0	3	0
Leprosy	3	0	0	8	0	0	11	100.0	11	0
Syphilis - congenital	2	0	0	1	0	0	3	100.0	3	0
Tetanus	0	0	0	2	0	0	2	100.0	2	0
Tuberculosis	37	3	0	1,266	21	0	1,327	98.4	1,306	21
Meningococcal disease (invasive)	17	2	1	201	9	0	230	96.1	221	9
Syphilis <2 years	108	16	6	912	51	6	1,099	94.8	1,042	57
Typhoid fever	0	1	0	88	7	0	96	92.7	89	7
Haemophilus influenzae type b	8	0	0	14	1	1	24	91.7	22	2
Listeriosis	1	0	1	63	6	0	71	91.5	65	6
Measles	1	0	0	63	6	0	70	91.4	64	6
Hepatitis A	0	0	0	240	22	1	263	91.3	240	23
Rubella	0	0	0	40	3	1	44	90.9	40	4
Varicella zoster (chickenpox)+	95	7	3	1,447	172	19	1,743	89.0	1,552	191
Hepatitis C (newly acquired)	51	0	0	264	36	7	358	88.0	315	43
Arbovirus infection (NEC)	4	1	1	15	2	1	24	87.5	21	3
Haemolytic uraemic syndrome	0	0	0	7	1	0	8	87.5	7	1
Hepatitis E	0	0	0	33	4	1	38	86.8	33	5
Varicella zoster (shingles)+	85	1	4	2,489	364	35	2,978	86.6	2,579	399
Legionellosis	3	0	0	255	33	7	298	86.6	258	40
Pneumococcal disease (invasive)	187	6	4	1,224	143	80	1,644	86.4	1,421	223
STEC, VTEC	1	0	0	66	13	1	81	82.7	67	14
Malaria	2	5	1	321	58	12	399	82.5	329	70
Shigellosis	138	3	5	305	59	42	552	81.7	451	101
Ornithosis	0	1	0	44	10	1	56	80.4	45	11
Hepatitis B (newly acquired)	16	3	0	164	40	5	228	80.3	183	45
Dengue virus infection	3	0	0	930	217	51	1,201	77.7	933	268
Q fever	9	0	0	231	76	7	323	74.3	240	83
Leptospirosis	5	0	2	89	31	4	131	73.3	96	35

Appendix 3 *continued*: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2010, 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
Syphilis >2 years or unspecified duration	134	16	11	748	324	8	1,241	73.2	909	332
Gonococcal infection	3,256	264	82	2,885	1,911	1,573	9,971	65.1	6,487	3,484
Influenza (laboratory confirmed)	605	53	15	6,970	4,241	1,535	13,419	57.0	7,643	5,776
Cryptosporidiosis	139	2	2	684	562	91	1,480	55.9	827	653
Pertussis	488	19	31	17,280	12,432	4,543	34,793	51.2	17,818	16,975
Mumps	2	0	0	46	30	17	95	50.5	48	47
Chlamydial infection	5,524	834	323	30,766	23,839	13,019	74,305	50.4	37,447	36,858
Campylobacteriosis	173	14	9	8,333	8,109	328	16,966	50.3	8,529	8,437
Kunjin virus infection	0	0	0	1	1	0	2	50.0	1	1
Hepatitis D	0	0	0	17	15	3	35	48.6	17	18
Salmonellosis	403	22	14	5,364	3,909	2,281	11,993	48.4	5,803	6,190
Brucellosis	0	0	0	9	12	0	21	42.9	9	12
Ross River virus infection	67	7	7	1,877	2,734	455	5,147	38.0	1,958	3,189
Hepatitis C (unspecified)	536	14	18	3,352	4,706	2,177	10,803	36.3	3,920	6,883
Hepatitis B (unspecified)	211	23	6	2,237	2,611	1,790	6,878	36.0	2,477	4,401
Barmah Forest virus infection	41	4	3	432	858	133	1,471	32.6	480	991
Varicella zoster (unspecified)+	100	19	5	1,705	5,141	182	7,152	25.6	1,829	5,323
Donovanosis	0	0	0	0	1	0	1	0.0	0	1

* 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

Arboviral diseases and malaria in Australia, 2009–10: Annual report of the National Arbovirus and Malaria Advisory Committee

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Abstract

The National Notifiable Diseases Surveillance System received 7,609 notified cases of disease transmitted by mosquitoes for the season 1 July 2009 to 30 June 2010. The alphaviruses Barmah Forest virus and Ross River virus, accounted for 6,546 (79%) of these notifications during the 2009–10 season. There were 37 notifications of dengue virus infection locally-acquired from North Queensland and 581 notified cases in Australia that resulted from overseas travel. This number of overseas acquired cases continues to rise each year due to increasing disease activity in the Asia–Pacific region and increased air travel. Detection of flavivirus seroconversions in sentinel chicken flocks across Australia provides an early warning of increased levels of Murray Valley encephalitis virus and Kunjin virus activity. Flavivirus activity was detected in western and northern Australia in 2009–10, which prompted public health action. No human cases of Murray Valley encephalitis virus infection were notified, while there were 2 cases of Kunjin virus infection notified. There were no notifications of locally-acquired malaria in Australia and 429 notifications of overseas-acquired malaria during the 2009–10 season. This annual report presents information of diseases transmitted by mosquitoes in Australia and notified to the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2012;36(1):70–81.

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

Introduction

This report describes the surveillance of mosquito-borne diseases of public health importance in Australia for the season 1 July 2009 to 30 June 2010. It includes locally and overseas acquired notified cases of disease caused by the alphaviruses (Barmah Forest virus, chikungunya virus and Ross River virus), flaviviruses (dengue virus, Murray Valley encephalitis virus, Kunjin virus, Japanese encephalitis virus and yellow fever virus) and malaria.

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

Methods

Human cases of arbovirus infection and malaria are monitored using the National Notifiable Diseases Surveillance System (NNDSS). All Australian states and territories require doctors and/or pathology laboratories to notify cases of infectious diseases that are important to public health including several arboviruses and malaria. The *National Health Security Act 2007* provides the legislative basis for communicable disease notifications in Australia and authorises the exchange of health information between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be provided. State and territory health departments transfer these notifications regularly to the NNDSS. The primary responsibility for public health action resulting from a notification resides with state and territory health departments. This report presents data extracted from NNDSS during March 2011 and analysed by date of diagnosis. This is a derived field and represents the earliest of the reported fields of notification date and notification received date. The dataset represents a 'snap shot', and numbers

in this report may vary slightly from those reported from other NNDSS sources. Detailed notes on the interpretation of NNDSS are available in the 2009 NNDSS annual report.[1] Case definitions for the diseases included in this report are available from http://www.health.gov.au/casedefinitions The report includes information on the following pathogens transmitted by mosquitoes:

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

To compare notifications in 2009–10 to historical totals, counts and crude rates of notification were compared either with the mean of the previous 5 years or with data from the previous year. The Australian Bureau of Statistics estimated resident population for Australia and each state or territory at June 2009 was used to calculate notification rates.

Additional information was available from a survey conducted with some state and territory public health surveillance managers. The survey sought to confirm cases reported to NNDSS and determine the place of acquisition for locally-acquired cases of dengue virus infections. States and territories may conduct follow-up of arbovirus and malaria cases to determine the likely place of acquisition of infection.

Results

During the 2009–10 season, there were 7,609 notifications of diseases transmitted by mosquitoes. This represented a 6% increase from the mean of 7,207 notifications for the previous 5 years. A summary of the counts and crude rates of these mosquito-borne diseases is shown in Table 1. There were no reported cases of yellow fever during the season.

Alphavirus

The most frequently reported alphaviruses in Australia, Ross River virus (RRV) and Barmah Forest virus (BFV), can cause illness characterised by fever, rash and polyarthritis. These viruses are transmitted by numerous species of mosquitoes that breed in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[2] No specific treatment or vaccine is available for these diseases. The viruses are maintained in a primary mosquito–mammal cycle involving macropods (kangaroos and wallabies), possibly other marsupials (e.g. possums), flying fox and native rodents. Horses, which may act as amplifier hosts, appear to develop joint and nervous system disease after infection with RRV. During the 2009–10 season, there were 6,546 notifications of alphaviruses (BFV and RRV) of which RRV infections accounted for 79% (5,148).

Barmah Forest virus infections

There were 1,398 notifications of BFV infections during the 2009–10 season, representing a rate of 6.4 per 100,000 population, which has decreased from the mean of 8.3 per 100,000 population for the previous 5 years (Table 1). Queensland reported the largest number of notifications of BFV (845) while the highest rate was reported in the Northern Territory (42 per 100,000 population).

Cases were reported in all jurisdictions. Approximately half of cases were male (52%). The median age for cases was 47 years.

As in previous years, there was a marked seasonal trend with the highest number of cases being diagnosed in the months of March (181) and April (180).

Ross River virus infections

There were 5,148 notifications of RRV infection during 2009–10 representing a rate of 23 per 100,000 population (Table 1). This was a 2.7% increase over the mean of the previous 5 years. Queensland reported the largest number of cases of RRV (2,540) while the highest rate was reported in the Northern Territory (143 per 100,000 population).

Cases were reported in all jurisdictions. Just under half of all notifications were male (44%). The median age for cases was 42 years.

As in previous years, there was a marked seasonal trend with the highest number of notifications being diagnosed in the months of March (928) and April (1,020).

South Australia reported a significant increase in RRV notifications when compared with the mean of the previous 5-year period. For the 2009–10 reporting period there were 391 RRV notifications (232 in 2008-09); 49 of which were along the Murray River; with the rest dispersed throughout both regional and metropolitan areas. RRV notifications in 2009–10 alone indicate a high level of virus activity and susceptibility in human populations. Heavy rainfall in South Australia since March 2010 has no doubt played a role in this.[3]

Chikungunya virus infection

Chikungunya virus (CHIKV) is a member of the alphavirus genus in the family *Togaviridae* and is closely related to RRV and BFV. Illness is charac-

Table 1: Number of notified cases, rate and 5-year mean rate per 100,000 population of mosquito-borne diseases, Australia, 2004–05 to 2009–10, by disease and state or territory

Disease		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Arbovirus Infection (NEC*)	Notified cases 2009/10	0	0	3	9	0	0	2	0	14
	Rate, 09/10	0.0	0.0	1.3	0.2	0.0	0.0	0.0	0.0	0.1
	Mean rate, 2004/05 – 08/09	0.0	0.0	0.3	0.5	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	Notified cases 2009/10	5	297	94	845	38	1	32	86	1,398
	Rate, 09/10	1.4	4.2	41.6	19.1	2.3	0.2	0.6	3.8	6.4
	Mean rate, 2004/05 – 08/09	1.4	7.5	41.6	21.5	4.4	0.2	0.5	6.8	8.3
Dengue virus infection†	Notified cases 2009/10	19	117	30	160	11	4	51	226	618
	Rate, 09/10	5.4	1.6	13.3	3.6	0.7	0.8	0.9	10.1	2.8
	Mean rate, 2004/05 – 08/09	1.6	1.3	9.2	7.5	1.1	0.4	0.3	2.7	2.4
Japanese encephalitis virus infection – infection acquired overseas	Notified cases 2009/10	0	0	0	0	0	0	0	0	0
	Rate, 09/10	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
	Mean rate, 2004/05 – 08/09	0.000	0.003	0.000	0.000	0.000	0.000	0.000	0.000	0.001
Kunjin virus infection	Notified cases 2009/10	0	0	1	1	0	0	0	0	2
	Rate, 09/10	0.0	0.0	0.4	0.0	0.0	0.0	0.00	0.0	0.01
	Mean rate, 2004/05 – 08/09	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	Notified cases 2009/10	3	84	13	134	23	3	94	75	429
	Rate, 09/10	0.9	1.2	5.7	3.0	1.4	0.6	1.7	3.3	2.0
	Mean rate, 2004/05 – 08/09	3.5	2.0	18.8	5.5	1.8	3.4	2.1	4.2	3.1
Murray Valley encephalitis virus infection	Notified cases 2009/10	0	0	0	0	0	0	0	0	0
	Rate, 09/10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	Mean rate, 2004/05 – 08/09	0.00	0.00	0.19	0.01	0.00	0.00	0.00	0.04	0.01
Ross River virus infection	Notified cases 2009/10	20	1139	323	2540	391	41	351	343	5148
	Rate, 09/10	5.7	16.0	142.8	57.4	24.1	8.1	6.4	15.3	23.4
	Mean rate, 2004/05 – 08/09	3.0	13.3	131.3	50.2	13.1	5.2	2.7	32.4	20.7

Does not include 33 chikungunya virus infections reported to the National Notifiable Diseases Surveillance System during the 2009–10 season as chikungunya is not reported by all jurisdictions.

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

† Locally acquired and overseas acquired; See Table 2.

NEC Not elsewhere classified.

terised by a sudden onset of fever, rash and severe joint pain. The acute disease lasts 1–10 days, but convalescence may include prolonged joint swelling and pain lasting months. It has clinical similarities to dengue, including occasional cases with haemorrhagic manifestations.[4] CHIKV is of concern given that the virus is transmitted from human to human by infected mosquitoes. Other vertebrates are not required for high levels of transmission to occur. In Australia, the confirmed mosquito vectors for CHIKV include *Aedes aegypti,* which occurs in northern Queensland and *Aedes albopictus,* which is found on Cocos, Christmas and the Torres Strait Islands.[5] Effective surveillance is required as there is the potential for the virus to become established in humans and spread in areas in Australia where the vectors exist. Other Australian mosquitoes that have been shown to be competent laboratory vectors of CHIKV include *Ae. vigilax, Ae. procax, Ae. notoscriptus* and *Coquillettidia linealis.*[6]

CHIKV infection is a notifiable disease in all jurisdictions other than the Australian Capital Territory. There were 33 notifications of overseas-acquired CHIKV infection reported to NNDSS during the 2009–10 season compared with 21 cases in 2008–09 and 3 cases in 2007–08. Twelve of the cases were reported to have acquired their infection during travel to India.

Flaviviruses

This section provides information on several flaviviruses notified to NNDSS including dengue virus (DENV), Murray Valley encephalitis virus (MVEV), Kunjin virus (KUNV) and Japanese encephalitis virus (JEV). Other flaviviruses may be notified under the arbovirus (NEC) category. Dengue is characterised by flu like symptoms (fever, headache, muscle/joint pain) and has 4 distinct serotypes. Infection with MVEV, KUNV and JEV can, in a small percentage of cases, result in illness involving the DENV nervous system including encephalitis of variable severity. *Ae. aegypti* is the major vector of dengue in Australia and *Culex annulirostris* is the major vector of MVEV, JEV and KUNV. No specific treatment is available for these diseases and care is largely supportive. A vaccine is not available for prevention of DENV, MVEV or KUNV infection but a vaccination to prevent JEV infection is available.[7] There were 620 notified flavivirus cases in 2009–10, which included 618 notified cases of DENV and 2 notified cases of KUNV disease (Table 1). There were no notified cases of JEV or MVEV.

Dengue virus infection

There were 618 notified cases of DENV infection during the season of 2009–10. Of these, 37 notified cases were locally-acquired in north Queensland and 581 notified cases acquired their DENV infection while overseas (Table 2).

Locally-acquired dengue virus infection

Local transmission of DENV is restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.[8] Dengue is not endemic in north Queensland, however local transmission can occur upon introduction of the

Table 2: Place of reporting of notified cases of dengue virus infection, Australia, 1 July 2005 to 30 June 2010, by state or territory

Dengue	Year of diagnosis	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Locally-acquired*	2005/06	0	0	0	42	0	0	1	0	43
	2006/07	0	0	1	46	0	0	0	0	47
	2007/08	0	0	0	26	0	0	0	0	26
	2008/09	0	5	0	1,008	0	0	3	1	1,017
	2009/10	0	3	0	33	0	0	1	0	37
Total locally-acquired		0	8	1	1,155	0	0	5	1	1,170
Overseas-acquired	2005/06	7	56	16	33	10	0	12	22	156
	2006/07	2	71	14	67	12	0	9	27	202
	2007/08	4	105	26	84	35	4	15	94	367
	2008/09	13	168	24	121	26	6	18	120	496
	2009/10	19	114	30	127	11	4	50	226	581
Total overseas-acquired		45	514	110	432	94	14	104	489	1,802

* Cases acquired their infection while living in or visiting north Queensland.

virus to the mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.[9] There were 37 notified cases of locally-acquired DENV infection during 2009–10. All cases of infection were acquired in north Queensland. Locally-acquired cases were notified between September 2009 and May 2010 and were associated with a number of outbreaks identified in north Queensland, dominated by 16 cases of DENV-2 from Tully.

Overseas-acquired dengue virus infection

There were 581 notifications of DENV infection acquired overseas during the 2009–10 season (Table 1), which was an increase when compared with the average of 257 over the last 5 years. All jurisdictions reported increased numbers of notifications of overseas-acquired DENV infection since 2005–06 (Figure). Most notably, Western Australia reported 226 cases compared with 22 cases in 2005–06.

Country of acquisition was available for 416 (72%) of the 581 cases of overseas-acquired DENV reported to NNDSS (Table 3). Indonesia (notably Bali) was the country of acquisition for 274 (47%) cases and involved all 4 dengue serotypes. Nineteen other destinations were identified by patients. The infecting DENV serotype was determined for 216 (37%) of the 581 overseas-acquired dengue cases. DENV serotype 2 (n = 98) was the most frequently reported serotype.

Figure: Notified cases of overseas-acquired dengue virus infection, Australia, 1 July 2004 to 30 June 2010, by state or territory

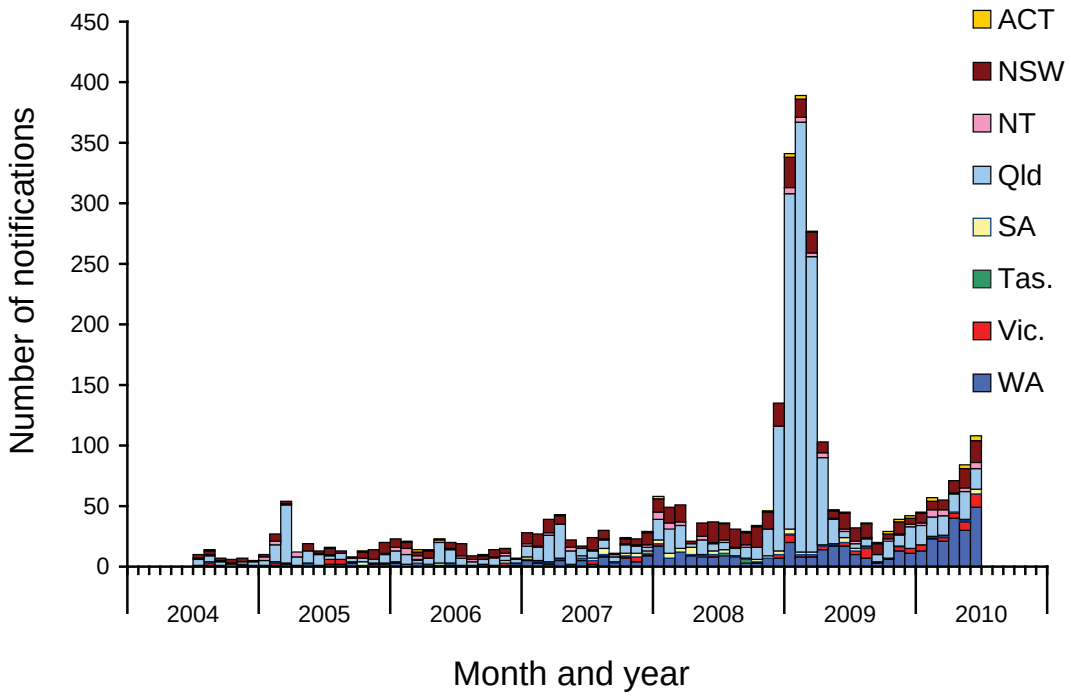


Table 3: Notified cases of overseas-acquired dengue virus infection, Australia, 1 July 2009 to 30 June 2010, by serotype and country of acquisition

Country of acquisition	Dengue serotype					Total cases
	Dengue 1	Dengue 2	Dengue 3	Dengue 4	Untyped	
Indonesia	34	66	22	10	142	274
Thailand	3	6	5	2	20	36
East Timor	5	0	0	2	13	20
Fiji	0	6	3	0	7	16
Vietnam	3	1	0	0	10	14
Other country	11	11	3	2	29	56
Country not listed	6	8	5	2	144	165
Total	62	98	38	18	365	581

Japanese encephalitis virus infections

The last JEV notification in Australia was reported by New South Wales in September 2008 and was acquired overseas. There were no cases of locally-acquired JEV infection notified to NNDSS in Australia during 2009–10. The last case of locally-acquired JEV infection was reported in 1998.[10]

Kunjin virus disease

There were 2 human cases of KUNV disease reported in Australia during 2009–10. One case each was reported by Queensland and the Northern Territory. The Northern Territory case exhibited encephalitic symptoms that are unusual for this disease.

Murray Valley encephalitis virus infection

There were no human cases of MVEV infections reported in Australia during 2009–10. Previous MVEV cases were reported in March and May 2009 with 2 cases each reported from Western Australia and the Northern Territory. Both cases from the Northern Territory were fatal.

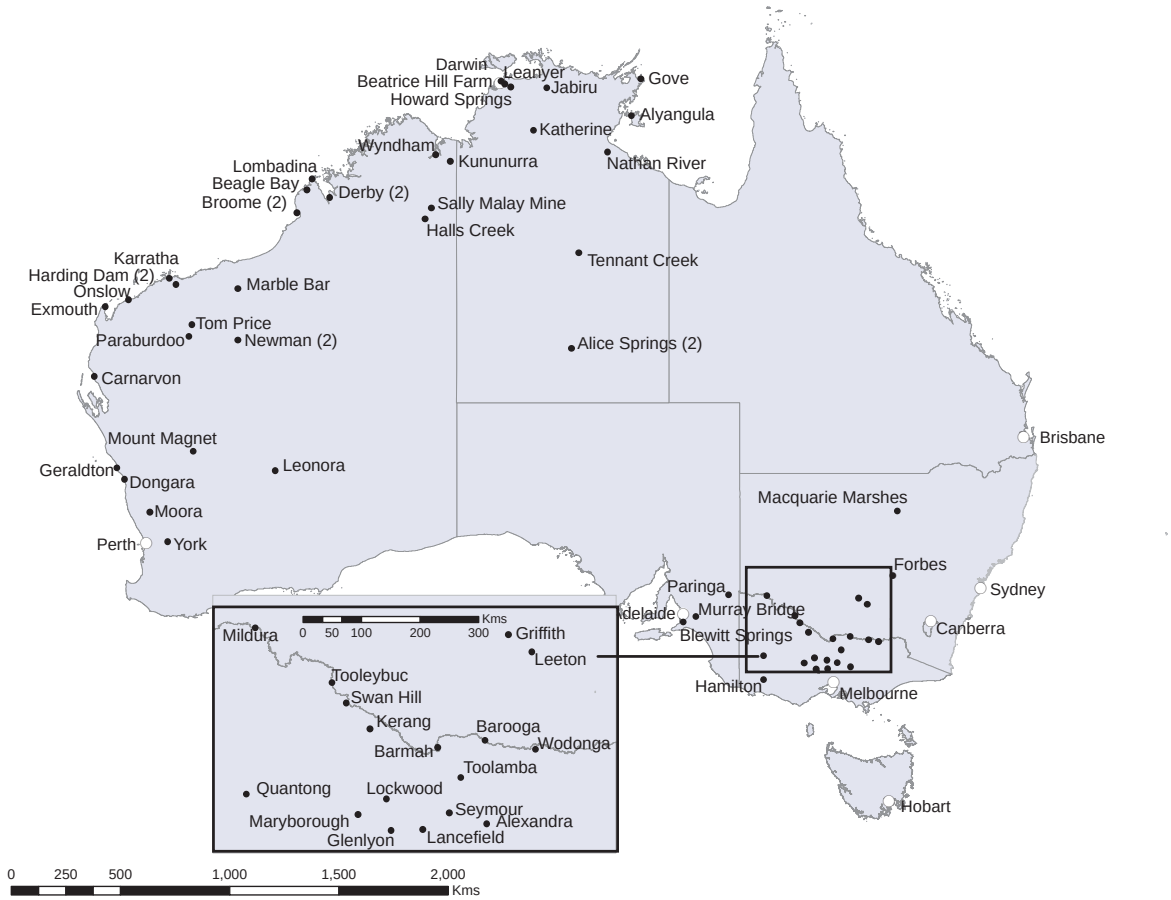
Sentinel chicken flavivirus surveillance programs

The sentinel chicken program is designed to detect flavivirus activity in Western Australia, New South Wales, Victoria, South Australia and the Northern Territory. The program aims to provide early warning of the endemic arboviruses MVEV and KUNV, as well as exotic arboviruses such as JEV.[11] A public health response or warning can be implemented when chickens from a flock develop new antibodies to a flavivirus of interest. These warnings advise residents of the need to take added precautions to avoid mosquito bites and may be used to direct mosquito management programs. Chickens are replaced at least annually and more frequently if birds die or large proportions seroconvert. The flocks are well positioned to detect flavivirus activity and provide a timely and accurate indication of risk to people.[12] The location of sentinel chicken sites during 2009–10 is shown in the Map.

Northern Territory

Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for flavivirus antibodies in a combined program between the Northern Territory Department of Health, Centre for Disease

Map: Sentinel chicken testing sites, Australia, 2009–10



Control Medical Entomology section, the Northern Territory Department of Resources (DoR) Berrimah Veterinary Laboratories, and volunteers.

DoR officers or volunteers usually bleed flocks once a month and the samples are tested by DoR for antibodies to MVEV and KUNV.

In the 2009–10 season, MVEV activity was detected in the flocks at Jabiru in April, and at Tennant Creek in August 2009 and May 2010. The three chickens that seroconverted to MVEV in Tennant Creek in August, outside the main MVE season, are possibly a carry over of MVEV activity from the previous season as the chickens were not bled in July 2009. The results in 2009–10 indicated low MVE virus activity in the Northern Territory, despite above average rainfall in the Top End. There have been no seroconversions to MVEV in the Alice Springs flocks since 2001–02, when the nearby Ilparpa swamp was drained, despite high levels of summer rain and large volumes of effluent released into the swamp this year. The high summer rain indicated an expected seroconversion to MVEV from one of the predictive models[13] and was the impetus for mosquito control precautions and warnings. The absence of any MVEV activity may have been partially due to an extensive aerial larval control of the swamp with methoprene pellets in March, but may also indicate that the local MVE ecology near Alice Springs has changed with the draining of the swamp and now may not be as suitable for MVEV transmission.

The results further indicate that MVEV is endemic as far south as Tennant Creek, and that high levels of activity in the Top End are not necessarily a prerequisite for activity in the semi-arid areas south to Tennant Creek.

KUNV activity occurred in the Top End, with seroconversions to KUNV being detected in the Howard Springs flock in January, and a flavivirus only indication in the Howard Springs flock, and the Coastal Plains flock in June 2010. The sentinel chicken flavivirus only seroconversion in Howard Springs in June 2010 later seroconverted to KUNV in 2010–11. There was a case of KUN encephalitis in the Darwin rural area in June 2010. Health warnings were issued in March and April and after the KUN encephalitis case. This is one of the rare instances of KUN encephalitis, and was reported in the *Northern Territory Disease Control Bulletin*.[14]

Western Australia

The flavivirus sentinel chicken program in Western Australia is undertaken by the Arbovirus Surveillance and Research Laboratory (ASRL) at the University of Western Australia, on behalf of the Western Australian Department of Health. Many state and local government authorities and community volunteers also take part in the program. Thirty sentinel chicken flocks (of up to 12 chickens) are located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Mid West and Central Coastal regions of Western Australia (Map). Blood samples are collected from the chickens by environmental health officers or trained volunteers at fortnightly intervals. Samples are transported to the ASRL where they are tested for antibodies to flaviviruses using an epitope blocking enzyme-linked immunosorbent assay.[15]

Rainfall prior to commencement of the 2009–10 wet season was generally below average in northern Western Australia. Thunderstorms and tropical cyclones (Tropical Cyclone Laurence and Tropical Cyclone Magna) caused above average rainfall in the north from November to January, and temperatures were warmer than usual. Warm dry conditions prevailed in February and March. Heavy rainfall returned to the north in April, and the wet, warm conditions continued into May.

A total of 3,941 serum samples from 30 flocks (including one new flock at One Arm Point near Lombadina) were tested for antibodies to flaviviruses during 2009–10.[16] Seroconversions to flaviviruses were detected in just 16 (0.4%) samples. One KUNV seroconversion was detected at Lombadina in July, 1 MVEV seroconversion at the Harding Dam flock in August, and 1 MVEV seroconversion at Tom Price in August was associated with activity extending from the 2008–09 wet season.

The first activity associated with the 2009–10 wet season occurred in February 2010, when KUNV was detected at Halls Creek in the south-east Kimberley region. Flavivirus activity was subsequently detected in the north-east Kimberley region (1 MVEV and 1 KUNV infection) and Lombadina in the West Kimberley region (5 MVEV infections) in May 2010. Overall, 13 seroconversions were detected through to July 2010. The level of MVEV and KUNV activity in sentinel chickens was substantially lower than the previous season, which was one of the highest on record for flavivirus activity. No flavivirus cases were reported in Western Australia during 2009–10.

The Western Australian Department of Health issued three media statements. The first was released on 24 August 2009, following continuing detections of MVEV and KUNV in sentinel chickens in the Pilbara region (associated with continued activity from the 2008–09 season). The second media statement was issued on 8 April 2010 after KUNV was detected in sentinel chickens in the Kimberley region for the first time in the 2009–10 wet season. The third media release was released on 9 June 2010

after MVEV was detected in sentinel chickens in the Kimberley region, representing the first evidence of MVEV activity in Western Australia for the 2009–10 season.[15,16]

New South Wales

The New South Wales Arbovirus Surveillance and Mosquito Monitoring program at the Institute of Clinical Pathology and Medical Research undertakes the New South Wales sentinel chicken program. The 2009–10 season began on 1 November 2009 with the first bleed and ended on 14 April 2010 with the last. A total of 2,133 samples were received from 7 sentinel chicken flocks in New South Wales during this period in 2009–10. The sentinel chicken flocks were located at Bourke, Deniliquin, Forbes, Griffith, Leeton, Macquarie Marshes and Menindee (Map). There were no seroconversions to MVEV or KUNV (personal communication: Stephen Doggett, New South Wales Health). A description of the bleeding method of the chickens and the testing regime is outlined in the 2003–04 New South Wales Arbovirus Surveillance Program annual report.[17]

Victoria

The Victorian sentinel chicken program is undertaken by the Department of Primary Industries on behalf of the Department of Health. For the period 1 July 2009 to 30 June 2010 sentinel chickens were placed at 10 locations along the Murray River and monitored from November 2009 to April 2010. Blood samples were collected weekly and tested for the presence of flavivirus antibodies. A total of 3,521 individual sentinel chicken samples were received and no samples were confirmed positive for flavivirus antibodies (personal communication: Rod Moran, Victorian Government Department of Health).

South Australia

Sentinel chicken flocks at Blewitt Springs, Murray Bridge and Paringa were screened twice for MVE and KUNV by the Department of Primary Industries South Australia in conjunction with the Department of Health and pathology services. No seroconversions to either virus were detected.[3]

Malaria

Malaria is a serious acute febrile illness that is normally transmitted from person to person through the bite of an infected mosquito. It is caused by a protozoan parasite called *Plasmodium* that includes 4 species that infect humans – *vivax, falciparum*, *malariae* and *ovale*.[18] A 5th species, *Plasmodium knowlesi* has been recently identified as a cause of human malaria occurring predominantly in South East Asia. Infection with this primate malaria has the potential of being fatal if treatment is not given early in the course of an infection.[19]

There were 429 notifications of overseas-acquired malaria during the season 2009–10, representing a rate of 2.0 per 100,000 population (Table 1). This was a decrease when compared with the mean rate of the previous 5 years of 3.1 per 100,000 population. There were no reports of locally-acquired malaria. The last Australian outbreak of locally-acquired malaria occurred in the Torres Strait (Saibai Island) in May 2004 where 3 cases of *P. falciparum* infection were reported.[20] The last outbreak of locally-acquired malaria on the Australian mainland occurred in north Queensland during 2002.[21]

The age group most affected was the 25–29 year age group with 65 notified cases. Approximately two-thirds of the 429 notified cases were male (70%), which was consistent with previous years. Cases were reported in all jurisdictions. No deaths from malaria were reported during the 2009–10 season.

The infecting *Plasmodium* species was reported for 98% of malaria notifications during the 2009–10 season (Table 4). *P. falciparum* and *P. vivax* were the predominant species. New South Wales notified 1 overseas-acquired case of *P. knowlesi* during the 2009–10 season. The infection was acquired in Indonesian Borneo. A man in his 30s attended a suburban hospital in Sydney, New South Wales, with a 2-week history of morning fevers and mild headaches. His symptoms started 13 days after he left Indonesian Borneo (Kalimantan). He had spent an average of 10 days per month for the past 18 months working adjacent to a forest area in South Kalimantan Province, Indonesian Borneo. The most recent visit was toward the end of the rainy season. He did not use any personal protection measures (mosquito nets, long clothing, insect repellent) or malaria chemoprophylaxis. He did not travel to any other malaria-endemic areas during this 18-month period. He was treated with atovaquone/proguanil for 3 days and the fever resolved and his platelet count returned to the reference level within 48 hours. He did not show any complications.

The country of acquisition of infection was available for 240 (56%) cases of malaria reported to NNDSS (Table 5). Papua New Guinea was identified as the place of acquisition for 84 (20%) cases and included both *P. falciparum* and *P. vivax* species. Thirty six other destinations were identified as a place of acquisition, including India (42), Ghana (12) and Uganda (12).

Table 4: Overseas-acquired malaria cases, Australia, 1 July 2009 to 30 June 2010, *Plasmodium* species, by state or territory

Plasmodium species	State or territory									Type (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
Plasmodium falciparum	2	45	2	56	10	3	28	40	186	43
Plasmodium vivax	1	30	10	71	10	0	59	24	205	48
Other *Plasmodium* species	0	6	0	4	1	0	6	5	22	5
Mixed *Plasmodium* species	0	2	1	0	0	0	1	5	9	2
Plasmodium species	0	1	0	3	2	0	0	1	7	2
Total	3	84	13	134	23	3	94	75	429	100

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

Table 5: Overseas-acquired malaria cases, Australia, 1 July 2009 to 30 June 2010, by country of acquisition and *Plasmodium* species

Country of acquisition	Total cases	*Plasmodium* species				
		Not specified	falciparum	vivax	Mixed *Plasmodium* species	Other species
Papua New Guinea	84	3	24	56	1	0
India	42	0	1	41	0	0
Ghana	12	0	9	1	0	2
Uganda	12	0	9	0	0	3
Other country	90	0	51	32	3	4
Country not listed	189	4	92	75	5	13
Total	429	7	186	205	9	22

Arbovirus infection (NEC)

The category includes notifications of arbovirus infections not elsewhere classified (NEC). There were 14 notifications in this category during the 2009–10 season, which was similar when compared with the previous 5 years. Queensland (9) the Northern Territory (3) and Victoria (2) accounted for all notified cases.

Other surveillance and research activities

National Arbovirus Monitoring Program

The National Arbovirus Monitoring Program (NAMP) monitors the distribution of economically important arboviruses of livestock and their vectors in Australia. Important arboviruses include bluetongue virus, Akabane virus and bovine ephemeral fever virus (BEFV) and are further described in the NAMP 2009–2010 annual report.[22]

Northern Australia Quarantine Strategy

The Australian Quarantine and Inspection Service (AQIS) Northern Australia Quarantine Strategy (NAQS) continues to undertake limited surveillance for the presence of JEV in the Torres Strait and mainland Australia. A sentinel pig herd at Injinoo airport near Bamaga in Cape York, Queensland has not shown any serological evidence of mainland presence since early 2004.[23]

Torres Strait Aedes albopictus *Elimination and Control Program*

The Asian Tiger mosquito, *Ae. albopictus,* which was previously exotic to Australia, was found on the outer islands of Torres Strait in April 2005.[24] If this mosquito establishes in mainland Australia, it will increase the number and extent of mosquitoes capable of transmitting dengue and chikungunya, as well as becoming a new serious pest mosquito. Since 2005, the Australian Government provided funding to Queensland Health towards a mosquito elimination program in the Torres Strait. The initial aim of the program was to eliminate *Ae. albopictus* from the Torres Strait islands. The development and implementation of a program based on the 'cordon sanitaire' approach (a barrier designed to prevent a disease or other undesirable condition from spreading) around Thursday and Horn islands

was initiated in May 2008 in an attempt to prevent the spread of *Ae. albopictus* further south, following unsuccessful attempts to eliminate *Ae. albopictus* from the outer islands of the Torres Strait.[25] Multiple incursions of *Ae. albopictus* into the Torres Strait had likely occurred and resulted from human activity or traffic moving these mosquitoes around the Torres Strait. In May 2009, the Australian Government agreed to provide further funding to Queensland Health over 4 years to continue support towards the Torres Strait Health Protection Strategy mosquito program.[26] The focus of the program is surveillance and control of *Ae. albopictus* in the Torres Strait and prevention of the spread of *Ae. albopictus* from the Torres Strait to mainland Australia.

Discussion

This report summarises the surveillance of nationally notifiable mosquito-borne disease in Australia for the season 1 July 2009 to 30 June 2010.

Australia recorded its first overseas-acquired case of *Plasmodium knowlesi* this year. *P. knowlesi* is now established as the fifth *Plasmodium* species to cause malaria in humans. Given the case history, clinicians should now consider the possibility of infection with *P. knowlesi* in patients who have acquired malaria in forest areas of South East Asia.[27]

The 2009–10 dengue fever season, when there were 37 locally-acquired notified cases, was comparatively mild compared with the 2008–09 season. In 2008–09, outbreaks of all 4 serotypes affected several locations with over 1,000 dengue cases in a short period and represented the largest reported annual number of cases in recent times.[28,29]

Much of the increase in overseas-acquired DENV infections over the past few years can be attributed to an increase in disease activity in the Asia/Pacific region as well as increased travel by Australians to some destinations in South East Asia. Dengue is the most rapidly increasing mosquito-borne viral disease in the world. The *Dengue Strategic Plan for the Asia Pacific Region (2008–2015)*[30] (The Strategic Plan) has been prepared by the WHO Regional Offices for South East Asia and the Western Pacific to respond to the increasing threat from dengue, which is spreading to new geographical areas in Member countries of the South-East Asia and the Western Pacific regions. The Strategic Plan provides a framework for national plans and is used to mobilise resources where they are needed. The NAMAC reviewed and endorsed this plan.

The Strategic Plan provides generic recommendations to allow its local adaptation. Effective dengue control is not possible if control efforts are limited to one country or a few countries. It requires the adoption of a regional approach through collaboration among countries and sustained partnerships to enable countries to implement evidence-based interventions and the use of best practices.[30]

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

MVEV and KUNV activity was detected in the sentinel chicken flocks in northern Western Australia and the Northern Territory. This led to public health media releases in both jurisdictions, which warned the public of potential infection and other prevention strategies.

The limitations of surveillance data used in this report are referred to in detailed notes on the interpretation of NNDSS, which is available in the 2009 NNDSS annual report.[1] A specific limitation of the data used in this report relates to the virological testing, which is required to distinguish alphavirus disease from other causes of arthritis. The alphavirus infections notified to NNDSS each season are based on laboratory definitive evidence only and assumes a clinically compatible arthritic infection. A case can still be notified when clinical illness may not be consistent with the diagnosis of alphavirus infection. Furthermore, false positive reactions are an issue in the serological diagnosis of some arboviral infections and cross-reacting IgM can occur, particularly with flavivirus infections.[31] Human surveillance for alphavirus infection enables local authorities to implement public health action and manage local disease outbreaks, but does not necessarily provide a reliable indication of the true incidence of a disease.

Another limitation of surveillance data of this report relates to place or country of acquisition of infection. This information is currently not available for South Australian notifications to NNDSS.

NAMAC provides advice on the strategic approaches to the management of arbovirus diseases and malaria, which continue to pose significant challenges to Australia and the region. This report describes the activities resulting from notified cases of human infections to health authorities and sentinel animal activities for the early detection of serious disease threats. Public Health authorities recognise the importance of vigilance in the surveillance of these diseases, which enables the rapid detection and response to the threat of arborviral disease and malaria.

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Tuberculosis notifications in Australia, 2008 and 2009

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Abstract

The National Notifiable Diseases Surveillance System received 1,194 tuberculosis (TB) notifications in 2008 and 1,322 notifications in 2009. The incidence of TB in Australia was 5.6 cases per 100,000 population in 2008 and 6.0 per 100,000 in 2009, similar to rates since 1986. In both 2008 and 2009, more than 85% of cases occurred in the overseas-born population. The incidence in the Australian-born Indigenous population was 6.2 per 100,000 population in 2008 and 4.8 per 100,000 in 2009. By contrast, the incidence of TB in the Australian-born non-Indigenous population was 0.9 per 100,000 in both 2008 and 2009. Household or other close contact with TB or past residence in a high risk country were the most commonly reported risk factors for TB infection. In 2008, 83 cases of TB were reported in health care workers; this decreased to 75 in 2009. There were no reports of TB transmission in Australian health care settings. Outcome data of the 2007 and 2008 TB cohort indicate that treatment success was attained in more than 95% of cases. As Australia continues to contribute to global TB control it is important to maintain good centralised reporting of TB to identify populations at risk and for early detection of reversal in trends in TB. *Commun Dis Intell* 2012;36(1):82–94.

Keywords: tuberculosis, annual reports

Introduction

The World Health Organization (WHO) has restated its commitment to reducing the global burden of tuberculosis (TB) by 2015 in the *Global Plan to Stop TB 2011–2015*.[1] Through the implementation of the Stop TB Strategy and the Global Plan to Stop TB, the incidence rate of TB worldwide is in gradual decline, with overall prevalence and death rates falling. Despite this, more than 9 million people still develop active TB globally each year and nearly 2 million people die. WHO has called for intensified efforts in scaling up existing interventions for the diagnosis and treatment of TB and research and development to achieve the 2015 targets.[1]

Australia sits within the WHO Western Pacific Region (WPR). The WPR contains four of the 22 high-burden countries that have received particular attention at the global level since 2000. Cambodia, China, the Philippines and Vietnam account for 19% of incident TB cases worldwide, and 95% of the incident TB cases in the WPR.[2] WHO data show that the WPR is on track to meet the goals to halve the TB prevalence and mortality rates from those of the year 2000.[2]

As a large proportion of TB cases diagnosed in Australia are people born outside of Australia, the ongoing success of the Stop TB Strategy in our region and globally has a significant impact on TB control in Australia. Surveillance of TB in Australia is overseen by the National Tuberculosis Advisory Committee (NTAC), a subcommittee of the Communicable Diseases Network Australia (CDNA). NTAC has the key role of providing strategic, expert advice to CDNA on a coordinated national approach to TB control. NTAC also has the role of developing and reviewing nationally agreed strategic and implementation plans for the control of TB in Australia. NTAC relies on quality surveillance data to inform these evidence-based policies.

This report should be considered in conjunction with the Australian Mycobacterium Reference Laboratory Network report on bacteriologically proven cases.[3]

Methods

TB is a nationally notifiable disease in Australia. Medical practitioners, public health laboratories and other health professionals are legally required to report cases of TB to state and territory health authorities. Notifications of TB are reported regularly to the National Notifiable Diseases Surveillance System (NNDSS). The primary responsibility for public health action resulting from notification resides with state and territory health departments.

Data presented in this report represent a point in time analysis of cases of TB notified to the NNDSS. Analyses of these cases were finalised in June 2011. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of TB notifications at the jurisdictional level. Detailed notes on case definition, data collection, quality control and the derivation of population subgroups are available in the 2007 annual report.[4]

Rates of notifications were calculated using the mid-year estimated resident population supplied by the Australian Bureau of Statistics. Rates specific to population subgroups were based on experimental estimated resident population as at 30 June 2006.

TB drug susceptibility data on bacteriologically confirmed cases are collected, analysed and reported by the Australian Mycobacterial Reference Laboratory Network in a companion report.[3]

Results

Overall numbers and rates

In 2009, a total of 1,322 cases of TB were notified in Australia, representing a rate of 6.0 cases per 100,000 population (Figure 1 and Table 1). This was an increase of 11% in the number of notified cases and an increase of 8% in the rate compared with 2008 (1,194 and 5.6 per 100,000 population, respectively). The rate of TB reported in 2009 is the highest rate since 1999.

Of the cases reported with a case classification, the majority of cases in both 2008 (97%, 1,157/1,192) and 2009 (96%, 1,259/1,315) were classified as new cases – a patient who has never been treated for TB or a patient who was treated for less than 1 month (Table 1). Of the 36 cases reported in 2008 as a relapse case—TB after a patient has deemed to have completed a partial or full course of treatment—11 relapsed after full treatment in Australia, one following partial treatment in Australia and 24 following full or partial treatment overseas. Of the 56 cases reported in 2009 as a relapse, 17 relapsed after full treatment in Australia, three following partial treatment in Australia and 36 following full or partial treatment overseas. A small number of cases were notified to the NNDSS without this information, one in 2008 and seven in 2009.

Figure 1: Notification rate for tuberculosis, Australia, 1960 to 2009

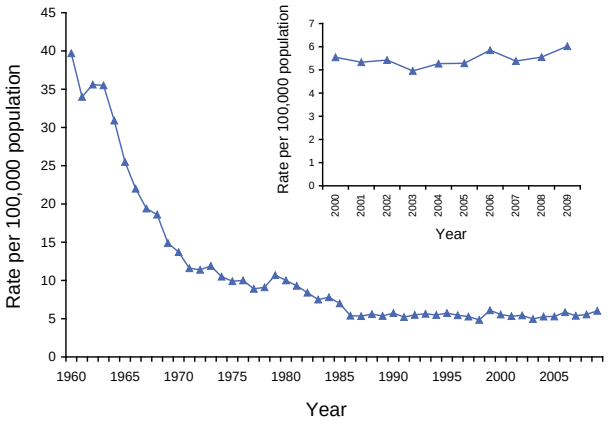


Table 1: Notified cases and notification rates per 100,000 population for tuberculosis, Australia, 2008 and 2009, by case classification and state or territory

	New cases	New cases rate	Relapse cases	Relapse case rate	Total cases*	Total rate
2008						
ACT	13	3.8	0	0.0	13	3.8
NSW	467	6.7	16	0.2	484	6.9
NT	32	14.5	0	0.0	32	14.5
Qld	121	2.8	9	0.2	130	3.0
SA	62	3.9	3	0.2	65	4.1
Tas	8	1.6	0	0.0	8	1.6
Vic	360	6.8	6	0.1	366	6.9
WA	94	4.3	2	0.1	96	4.4
Australia	1,157	5.4	36	0.2	1,194	5.6
2009						
ACT	22	6.2	1	0.3	23	6.5
NSW	492	6.9	28	0.4	523	7.3
NT	27	11.9	0	0.0	27	11.9
Qld	148	3.3	7	0.2	155	3.5
SA	54	3.3	4	0.2	58	3.6
Tas	8	1.6	1	0.2	9	1.8
Vic	409	7.5	8	0.1	417	7.7
WA	99	4.4	7	0.3	110	4.9
Australia	1,259	5.7	56	0.3	1,322	6.0

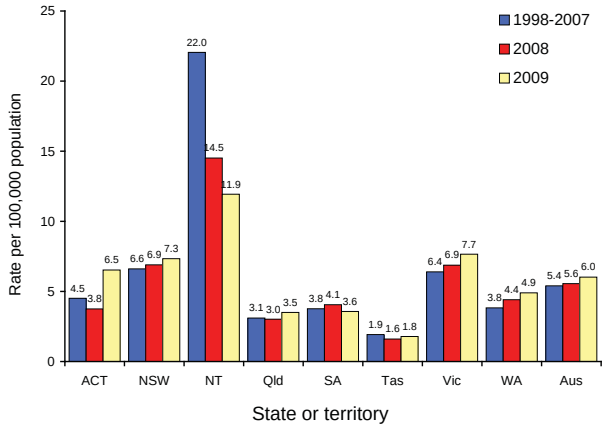
* New or relapse cases were not reported for 1 case in 2008 and 7 cases in 2009.

Tuberculosis cases by state or territory

As in previous years, New South Wales accounted for the largest proportion of cases notified by a state or territory in both 2008 (41%, 484/1,194) and 2009 (40%, 523/1,322). The highest notification rates in 2008 were reported by the Northern Territory (14.5 per 100,000 population), New South Wales (6.9 per 100,000 population) and Victoria (6.9 per 100,000 population). Similarly, the highest notification rates by state and territory in 2009 were reported in these same jurisdictions (Table 1).

Figure 2 presents TB notification rates for 2008 and 2009, compared against the average rate over the preceding 10 years for each state and territory. The Australian Capital Territory, New South Wales, Queensland, Victoria and Western Australia all reported a rate in 2009 that exceeded the rate in 2008 and the average rate of the 10 years preceding.

Figure 2: Rate for tuberculosis, Australia, 1996 to 2009, by state or territory



Tuberculosis in the Australian-born population

Indigenous status was reported for each of the 172 Australian-born cases reported in 2008 and 155 in 2009 (Table 2). The incidence rate of TB in the Australia-born population for 2008 was 1.1 cases per 100,000 population. The rate in the Australian-born Indigenous group was 6.2 cases per 100,000 population (n = 32) and in the Australian-born non-Indigenous group was 0.9 cases per 100,000 population (n = 140). The incident rate in the Australian-born Indigenous population was almost 7 times the rate seen in the Australian-born non-Indigenous population in 2008.

The incidence rate of TB in the Australia-born population for 2009 (1.0 per 100,000 population) was slightly lower than that reported in 2008. The rate in the Australian-born Indigenous group was 4.8 cases per 100,000 population (n = 25) and the Australian-born non-Indigenous population maintained the rate reported in 2008 (0.9 per 100,000 population, n = 130). The disparity in rates experienced in the Australian-born Indigenous and Australian-born non-Indigenous groups narrowed in 2009, with the Australian-born Indigenous rate reported just greater than 5 times the rate in the Australian-born non-Indigenous population.

The rate of TB in the Australian-born non-Indigenous population has remained relatively stable since 2002 (Figure 3), while the rate in the Australian-born Indigenous population tends to be trending downwards over this period. The caseload attributable to the Australian-born non-Indigenous population has decreased from 16% in 2002 to 10% in 2009 (Figure 4).

Figure 3: Notified cases and rate for tuberculosis, Australia, 2002 to 2009, by population subgroup

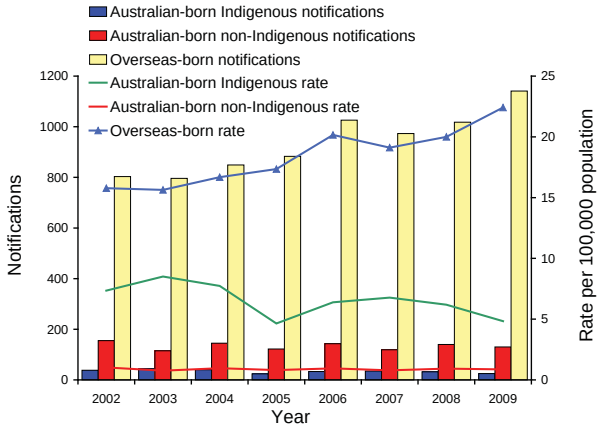
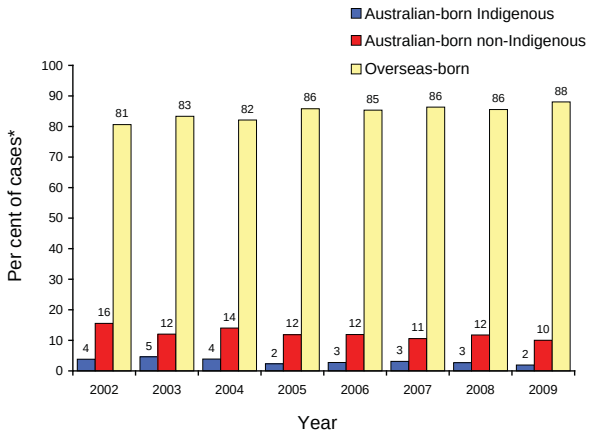


Figure 4: Notified cases of tuberculosis as a percentage of all cases with a reported population subgroup, Australia, 2002 to 2009, by population subgroup



* Where country of birth is reported.

Tuberculosis cases in the overseas-born population

Country of birth was known for nearly all (1,190/1,194) cases in 2008 and 98% (1,296/1,322) in 2009. Of the cases reported with a known place of birth in 2008, 86% (1,018/1,190) were born overseas (Table 2). This proportion of cases reported as overseas-born ranged by state and territory from 38% (3/8) in Tasmania to 92% (88/96) in Western Australia. The rate of TB among the overseas-born population in 2008 was over 18 times the rate in the Australian-born (20.0 per 100,000 population versus 1.1 per 100,000 population).

Of the cases reported with a known place of birth in 2009, 88% (1,141/1,296) were born overseas (Table 2). This proportion of cases reported as overseas-born ranged by state and territory from 67% (6/9) in Tasmania to 95% (55/58) in South Australia. The rate of TB among the overseas-born population in 2009 was almost 23 times the rate in the Australian-born (22.4 per 100,000 population versus 1.0 per 100,000 population).

The number of cases and the rate of TB in the overseas-born population has steadily increased since 2002 (Figure 3), noting that completeness of reporting country of birth and Indigenous status has improved over this period. The case load attributable to the overseas-born population has increased from 81% in 2002 to 88% in 2009 (Figure 4).

Among overseas-born cases, the most frequently reported country of birth was India (2008: 20%, 206/1,018; 2009: 27% 310/1,141; Table 3), followed by Vietnam (2008: 10%, 104/1,018; 2009: 11% 122/1,141), the Philippines (2008: 8%, 84/1,018; 2009: 8% 90/1,141) and China (2008: 6%, 65/1,018; 2009: 6% 74/1,141). These countries have consistently been reported as the most frequent countries of birth in recent years. The number and proportion of cases reported against these countries has remained relatively stable over recent years, except cases reported with a country of birth of India. India has increasingly been reported as a country of birth since 2002, when 13% (107/803) of overseas-born cases were reported as born in India.

Data on the year of arrival were available for 98% of the cases reported as overseas-born in 2008

Table 2: Notified cases and rate for tuberculosis, Australia, 2008 and 2009, by population subgroup and state or territory

	Australian-born						Overseas-born	
	Indigenous cases	Indigenous rate	non-Indigenous cases	non-Indigenous rate	Total Australian-born cases	Total Australian-born rate	Overseas-born cases	Overseas-born rate
2008								
ACT	0	0.0	4	1.6	4	1.6	9	11.4
NSW	5	3.3	57	1.2	62	1.2	422	23.4
NT	16	25.0	2	1.8	18	10.1	14	42.6
Qld	9	6.2	12	0.4	21	0.6	106	13.2
SA	1	3.6	9	0.8	10	0.8	55	16.0
Tas	0	0.0	5	1.2	5	1.2	3	5.3
Vic	0	0.0	44	1.2	44	1.2	321	23.8
WA	1	1.4	7	0.5	8	0.6	88	14.3
Aust	32	6.2	140	0.9	172	1.1	1,018	20.0
2009								
ACT	0	0.0	3	1.2	3	1.2	20	25.4
NSW	5	3.3	48	1.0	53	1.1	470	26.0
NT	8	9.4	2	1.8	10	4.5	17	51.7
Qld	11	7.6	19	0.6	30	0.9	103	12.8
SA	0	0.0	3	0.3	3	0.2	55	16.0
Tas	0	0.0	3	0.7	3	0.7	6	10.7
Vic	0	0.0	38	1.0	38	1.0	379	28.1
WA	1	1.4	14	1.0	15	1.0	91	14.8
Aust	25	4.8	130	0.9	155	1.0	1,141	22.4

Indigenous status and country of birth were not reported for 4 cases in 2008 and 26 cases in 2009.

Table 3: Notifications and notification rates for tuberculosis for selected countries of birth, Australia, 2008 and 2009

Country of birth	Cases				Estimated resident population†	Estimated rate per 100,000 population	
	2008		2009				
	n	%*	n	%*		2008	2009
India	206	20	310	27	180,130	114.4	172.1
Vietnam	104	10	122	11	185,450	56.1	65.8
Philippines	84	8	90	8	140,020	60.0	64.3
China‡	65	6	74	6	259,180	25.1	28.6
Nepal	35	3	53	5	5,020	697.2	1,055.8
Indonesia	52	5	39	3	59,380	87.6	65.7
Papua New Guinea	31	3	36	3	27,800	111.5	129.5
Burma (Myanmar)	21	2	25	2	14,060	149.4	177.8
Sri Lanka	17	2	23	2	71,730	23.7	32.1
Sudan	26	3	22	2	21,550	120.6	102.1
Afghanistan	26	3	21	2	19,610	132.6	107.1
Thailand	14	1	18	2	35,560	39.4	50.6
Ethiopia	22	2	18	2	6,540	336.4	275.2
Somalia	14	1	18	2	5,040	277.8	357.1
Republic of Korea	7	1	17	1	60,310	11.6	28.2
Pakistan	15	1	17	1	19,410	77.3	87.6
Bangladesh	16	2	16	1	18,310	87.4	87.4
Other overseas-born	263	26	222	19	3,960,965	–	–
Total overseas-born	1,018	–	1,141	–	5,090,065	–	–
Australian-born	172	–	155	–	15,607,815	–	–
Country of birth not reported	4	–	26	–	–	–	–
Total	1,194	–	1,322	–	20,697,880	–	–

* Proportion of all overseas-born cases.

† The Australian Bureau of Statistics estimated resident population at 30 June 2006.

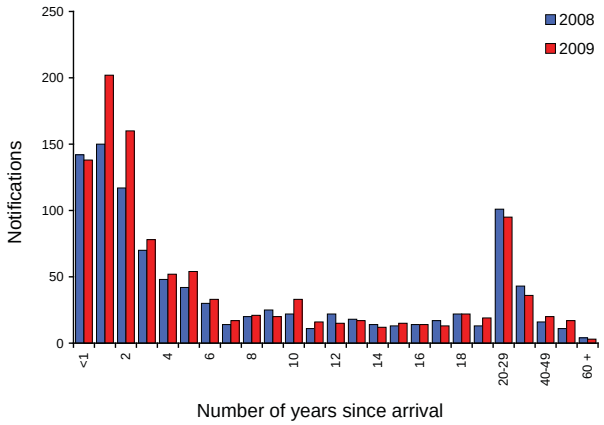
‡ China excludes the Special Administrative Region and Taiwan.

– Not applicable.

(999/1,018) and 2009 (1,122/1,141). Of the overseas-born cases notified in 2008 and 2009 that were reported with a year of arrival, 30% (632/2,121) presented within 2 years of arrival in Australia and 84% (1,776/2,121) within 20 years of arrival (Figure 5). The median length of time in Australia between arrival and diagnosis was 4 years (interquartile range, IQR: 1–14).

The residency status was available for 63% (644/1,018) of the cases reported as overseas-born in 2008 and 91% (1,033/1,141) in 2009. The collection of residency status was introduced in New South Wales during 2008, therefore completeness of these data is expected to improve from 2009 onwards. Residency status should be interpreted with caution as it is self-reported by each case and is not verified. Of the cases reported with a residency status, the majority in both years were reported as permanent residents (2008:

Figure 5: Tuberculosis cases in the overseas-born population, by number of years since arrival, Australia 2008 and 2009



58%, 372/644; 2009: 59%, 610/1,033), followed by overseas students (2008: 18%, 117/644; 2009: 20%, 204/1,033). Refugees (2008: 7%, 48/644; 2009: 5%, 55/1,033) and overseas visitors (2008: 7%, 46/644; 2009: 6%, 66/1,033) represented similar proportions within the subgroup.

There was a total of 21 cases of TB among Papua New Guinea (PNG) nationals accessing health care in the Torres Strait Treaty Zone in 2008 and 19 in 2009. These cases were all reported by Queensland and represented 14% (40/285) of the state's caseload across 2008 and 2009. There were 10 illegal fishermen detained by Australian Customs, diagnosed with TB and commenced on TB treatment in 2008, with only 1 case reported with this residency status in 2009. These cases were all reported by the Northern Territory and represented 19% (11/59) of the Northern Territory caseload across the 2 years.

Tuberculosis cases by age and sex

Information on the sex and age of TB cases was available for all cases reported to the NNDSS in 2008 and all but 1 case reported in 2009. The male to female ratio for both 2008 and 2009 was 1:0.8, a similar ratio as reported in previous years.

The age group incidence rates for TB in overseas-born, Australian-born Indigenous and Australian-born non-Indigenous populations are shown in Table 4 and Figure 6. The rate in the overseas-born population was highest in the 25–34 years age group (45.8 per 100,000 population, n = 665). In the Australian-born Indigenous group the highest age-specific rate was reported in the 45–54 years age group (13.4 per 100,000 population, n = 12), while in the Australian-born non-Indigenous group the age-specific rate increased throughout adult life. The highest notification rate in this group was reported in the 65 years or over age group (2.3 per 100,000 population, n = 79). The median age in the overseas-born cases was 32 years (IQR 25–50)

Table 4: Notified cases and notification rate per 100,000 population for tuberculosis, Australia, 2008 and 2009, by population subgroup and age group

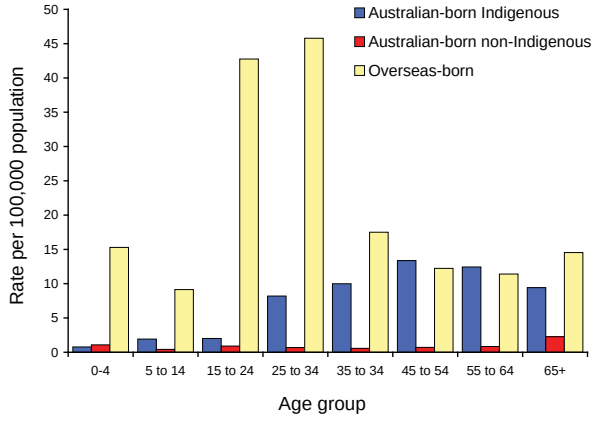
	Australian-born Indigenous		Australian-born non-Indigenous		Total Australian-born		Overseas-born		Total	
	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases*	Rate
2008										
0–4	1	1.6	13	1.1	14	1.1	10	20.4	24	1.8
5–14	2	1.5	11	0.5	13	0.5	17	7.2	30	1.1
Subtotal <15 years	3	1.5	24	0.7	27	0.7	27	9.5	54	1.3
15–24	3	3.0	18	0.8	21	0.9	220	42.9	241	8.3
25–34	8	10.9	11	0.5	19	0.9	288	39.7	307	10.6
35–44	7	10.8	10	0.5	17	0.8	153	16.8	171	5.6
45–54	6	13.4	14	0.7	20	1.0	105	11.5	127	4.4
55–64	5	20.7	17	1.2	22	1.5	97	12.0	119	5.3
65+	0	0.0	46	2.6	46	2.6	128	13.7	175	6.5
Total	32	6.2	140	0.9	172	1.1	1,018	20.0	1,194	5.8
2009										
0–4	0	0.0	13	1.1	13	1.0	5	10.2	18	1.4
5–14	3	2.3	9	0.4	12	0.5	26	11.1	39	1.4
Subtotal <15 years	3	1.5	22	0.6	25	0.7	31	10.9	57	1.4
15–24	1	1.0	23	1.0	24	1.0	219	42.7	245	8.5
25–34	4	5.5	18	0.9	22	1.0	377	51.9	406	14.0
35–44	6	9.2	14	0.7	20	0.9	165	18.2	192	6.3
45–54	7	15.6	13	0.7	20	1.0	118	12.9	142	5.0
55–64	1	4.1	7	0.5	8	0.6	88	10.9	98	4.3
65+	3	18.9	33	1.9	36	2.0	144	15.4	182	6.8
Total	25	4.8	130	0.9	155	1.0	1,142	22.4	1,322	6.4

* Country of birth and Indigenous status not reported for 4 cases in 2008 and 26 cases in 2009.

compared with 40 years in the Australian-born Indigenous cases (IQR 28–48) and the Australian-born non-Indigenous cases (IQR 19–68).

One of the most important measures of TB control is the incidence in children aged less than 15 years because these cases represent recent TB infection. TB was notified in 54 children aged less than 15 years in 2008, which represented 5% of the total number of notified cases (Table 4). Of these, 27 were Australian-born and 27 were born overseas. In 2009, 57 children aged less than 15 years were notified with TB; 4% of the total number of notified cases. Of these, 25 were Australian-born and 31 were born overseas. Of the Australian-born children three were identified as Indigenous Australians.

Figure 6: Average rate for tuberculosis, Australia, 2008 and 2009, by population subgroup and age group



Tuberculosis and selected risk factors

Information on selected risk factors for TB, excluding HIV, is reported in Table 5. Selected risk factor data were provided for 76% (913/1,194) of cases in 2008 and 74% (982/1,322) of cases reported in 2009. Of the cases with completed selected risk factor, 91% (828/913) of cases in 2008 and 98% (964/982) of cases in 2009 were reported with at least one risk factor.

Household member or other close contact with TB was a common risk factor in all 3 population subgroups in both 2008 and 2009. Past travel to or residence in a high-risk country was a common risk factor in both the Australian-born non-Indigenous and overseas-born cases. Interpretation of this risk factor in overseas born subjects is difficult, because at the time of this data collection there was no clarification as to whether this risk factor included a country of birth that has high TB incidence, or exclusively those subjects that visit such a country. NTAC has now agreed to record this risk factor only in those visiting a new high incidence setting, aiming to identify travel related TB, and therefore subsequent reports of this risk factor will be clearer.

A total of 83 cases of TB in 2008 were reported in people who had previously worked or were currently working in a health care setting either in Australia or overseas: 17 of these cases were known to have worked in an Australian health care setting at some point within the 12 months prior to diagnosis. In 2009, the total number of cases who had previously worked or were currently working in a health care setting decreased to 75. Of these cases, 24 cases were known to have worked in an Australian health care setting at some point within the 12 months prior to diagnosis. None of the cases were deemed to have acquired TB in an Australian health care setting, nor were there any reports of active TB transmission to patients from health care workers in Australia in 2008 and 2009.

The number of health care workers reported has increased since 2000 (Figure 7). The majority of these cases are overseas-born.

Tuberculosis and HIV status

HIV status reporting was complete in 94% (1,127/1,194) of cases in 2008 and 95% (1,257/1,322) of cases in 2009. A case was considered to have complete HIV status data if it was reported as HIV positive, HIV negative, HIV tested – result unknown, not tested or refused testing.

Testing was made available to 80% (906/1,127) of cases in 2008 and 81% (1,024/1,257) in 2009. Testing was reported to be refused by a small number of cases – 5 cases each in 2008 and 2009. Of the cases nationally with a known HIV test outcome, 3% of cases were reported as HIV positive in both 2008 (13/506) and 2009 (17/635).

Figure 7: Tuberculosis cases reported in health care workers, Australia, 2002 to 2009, by country of birth

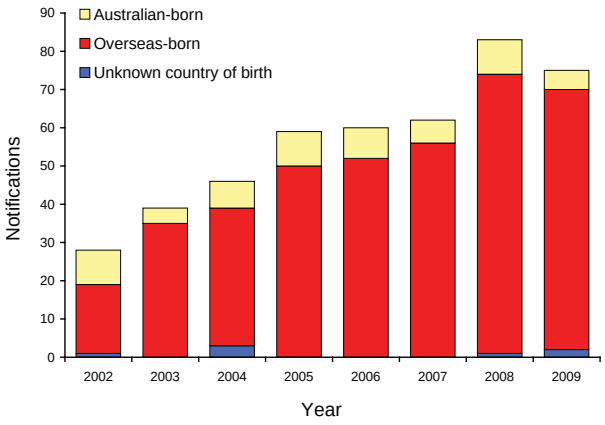


Table 5: Notifications of tuberculosis, Australia, 2008 and 2009, by selected risk factors*,† and population subgroup

Risk factor	Australian-born Indigenous cases	Australian-born non-Indigenous cases	Overseas-born cases	Total‡	Australian-born Indigenous cases	Australian-born non-Indigenous cases	Overseas-born cases	Total‡
	2008				2009			
Household or other close contact with TB	19	37	133	191	11	38	127	178
Ever resided in a correctional facility§	0	0	5	5	1	1	0	2
Ever resided in an aged care facility§	0	1	0	1	0	2	2	4
Ever employed in an institution§	0	2	11	13	2	0	14	16
Currently or previously employed in health industry in Australia or overseas§	0	9	73	83	0	5	68	75
Ever homeless	2	3	9	14	2	0	3	5
Past travel to or residence in a high-risk country	0	21	563	587	2	23	610	657
Chest X-ray suggestive of old untreated TB	1	2	6	9	0	1	10	11
Currently receiving immunosuppressive therapy	0	3	8	11	0	3	4	8
Australian-born child with one or more parent born in a high-risk country	0	5	n.a.	5	0	9	n.a.	9
None of the above risk factors	10	44	31	85	7	36	37	80
Not assessed/ no risk factor reported	1	28	252	281	2	17	317	340

* Excludes HIV status.

† More than 1 risk factor may be reported for each notified case.

‡ Country of birth and Indigenous status not reported for 4 cases in 2008 and 26 cases in 2009.

§ Within the preceding 5 years.

n.a. Not applicable.

Anatomical site of disease

The anatomical site of TB infection was recorded in all notified cases in 2008 and in all but 5 cases in 2009 (Table 6). In 2008, pulmonary disease was reported in 718 cases, of which 80% (572/718) were reported as having pulmonary disease only and the remaining 20% (146/718) were reported as having pulmonary disease plus disease at an extrapulmonary site. Of the pulmonary TB cases, the smear positive proportion was 36% (255/718). Extrapulmonary disease was reported in 40% (476/1,194) of cases, with lymph nodes reported as the most frequent extrapulmonary site (n = 185).

In 2009, pulmonary disease was reported in 757 cases, of which 78% (587/757) were reported as having pulmonary disease only and the remaining 23% (170/757) were reported as having pulmonary disease plus disease at an extrapulmonary site. Of the pulmonary TB cases, the smear positive proportion was 39% (298/757). Extrapulmonary disease was reported in 42% (560/1,322) of cases, with lymph nodes reported as the most frequent extrapulmonary site (n = 256).

Treatment outcomes of 2007 and 2008 tuberculosis patient cohort

Table 7 presents treatment outcome data for all TB cases reported in 2007 and 2008. Treatment success, including those with bacteriologically confirmed cure and those who completed treatment, was reported in 95% of cases with assessable outcomes reported in 2007 (964/1,010) and 2008 (1,018/1,068). Only 84% (27/32) of Australian-born Indigenous cases were reported with treatment success in 2007, however this increased to 90% (27/30) of Australian-born Indigenous cases in 2008. There were no cases of a treatment failure reported in 2007 and 2008.

Table 6: Notified cases of tuberculosis, Australia, 2008 and 2009, by site of disease and case classification

Site	New cases	Relapse cases	Total cases*	Per cent of cases
2008				
Total pulmonary disease	696	22	718	60.1
Pulmonary only	552	20	572	47.9
Pulmonary plus other sites	144	2	146	12.2
Extrapulmonary only*	461	14	476	39.9
Pleural	86	0	86	7.2
Lymph nodes	177	8	185	15.5
Bone/joint	29	0	30	2.5
Genito/urinary	27	1	28	2.3
Miliary	28	1	29	2.4
Meningeal	4	0	4	0.3
Peritoneal	37	0	37	3.1
Other	104	6	110	9.2
2009				
Total pulmonary disease	713	42	757	57.3
Pulmonary only	552	33	587	44.4
Pulmonary plus other sites	161	9	170	12.9
Extrapulmonary only*	545	14	560	42.4
Pleural	72	1	73	5.5
Lymph nodes	246	10	256	19.4
Bone/joint	48	2	50	3.8
Genito/urinary	33	1	34	2.6
Miliary	24	2	26	2.0
Meningeal	9	0	9	0.7
Peritoneal	29	3	32	2.4
Other	84	2	87	6.6

* New/relapse case was not reported in 1 case in 2008 and 7 cases in 2009.

Table 7a: Notified cases of tuberculosis, Australia, 2007, by treatment outcome and population subgroup

Assessable outcomes	Australian-born Indigenous		Australian-born non-Indigenous		Overseas-born		Total cases‖	
	Cases	%	Cases	%	Cases	%	Cases	%
Treatment success	27	84.4	94	93.1	839	96.2	964	95.4
Cured (bacteriologically confirmed)*	10	31.3	3	3.0	46	5.3	59	5.8
Completed treatment	17	53.1	91	90.1	793	90.9	905	89.6
Interrupted treatment†	0	0.0	0	0.0	4	0.5	4	0.4
Died of tuberculosis	2	6.3	3	3.0	6	0.7	11	1.1
Defaulted‡	3	9.4	2	2.0	16	1.8	21	2.1
Failure§	0	0.0	0	0.0	0	0.0	0	0.0
Not followed up, outcome unknown	0	0.0	2	2.0	7	0.8	10	1.0
Total assessable	32	100.0	101	100.0	872	100.0	1,010	100.0
Non-assessable outcomes								
Transferred out of Australia	1	2.9	0	0.0	70	7.2	72	6.3
Died of other causes	2	5.7	17	14.3	28	2.9	48	4.2
Still under treatment	0	0.0	1	0.8	3	0.3	4	0.4
Total	35	100.0	119	100.0	973	100.0	1,134	100.0

* Cured is defined as the bacteriologically confirmed sputum smear and culture positive at the start of treatment and culture negative in the final month of treatment and on at least 1 previous occasion.

† Interrupted treatment means treatment interrupted for two months or more but completed.

‡ Defaulted means failed to complete treatment.

§ Failed means sputum culture positive at 5 months or later.

‖ Population subgroup unknown in 7 cases.

Table 7b: Notifications of tuberculosis, Australia, 2008, by treatment outcome and population subgroup

Assessable outcomes	Australian-born Indigenous		Australian-born non-Indigenous		Overseas-born		Total cases‖	
	Cases	%	Cases	%	Cases	%	Cases	%
Treatment success	27	90.0	125	94.7	863	95.6	1,018	95.3
Cured (bacteriologically confirmed)*	3	10.0	10	7.6	41	4.5	56	5.2
Completed treatment	24	80.0	115	87.1	822	91.0	962	90.1
Interrupted treatment†	0	0.0	2	1.5	3	0.3	5	0.5
Died of tuberculosis	2	6.7	1	0.8	13	1.4	16	1.5
Defaulted‡	0	0.0	3	2.3	17	1.9	20	1.9
Failure§	0	0.0	0	0.0	0	0.0	0	0.0
Not followed up, outcome unknown	1	3.3	1	0.8	7	0.8	9	0.8
Total assessable	30	100.0	132	100.0	903	100.0	1,068	100.0
Non-assessable outcomes								
Transferred out of Australia	0	0.0	0	0.0	71	7.0	72	6.0
Died of other causes	2	6.3	7	5.0	38	3.7	47	3.9
Still under treatment	0	0.0	1	0.7	6	0.6	7	0.6
Total	32	100.0	140	100.0	1,018	100.0	1,194	100.0

* Cured is defined as the bacteriologically confirmed sputum smear and culture positive at the start of treatment and culture negative in the final month of treatment and on at least 1 previous occasion.

† Interrupted treatment means treatment interrupted for 2 months or more but completed.

‡ Defaulted means failed to complete treatment.

§ Failed means sputum culture positive at 5 months or later.

‖ Population subgroup unknown in 4 cases.

National performance indicators

Performance criteria for incidence (less than 1 per 100,000 population) were met only for the crude incidence rates in Australian-born non-Indigenous cases (Table 8). Incidence rates in the age groups under 15 years exceeded the performance criteria (less than 0.1 case per 100,000 population) in all population groups. Outcome reporting came close to meeting the target of 100% for the 2007 and 2008 patient cohorts, with less than 1% of cases with assessable outcomes reported with an unknown outcome. The performance indicator for cases that reported treatment success was met in both 2007 and 2008. Additionally this performance indicator was met in each of the population subgroups, including Australian-born Indigenous cases.

Discussion

While the overall rate of TB in Australia remains low by global standards, the modest rate increases in 2008 and 2009 suggest that we still face challenges to maintaining TB control. The current epidemiology of TB in Australia is largely a direct effect of the global TB situation with overseas-born persons contributing to a steadily increasing number and proportion of notifications since 2002 (Figure 3). Based on feedback from state TB services, the increase in skilled migrants and students from high burden countries is likely to be a key contributing factor to the recent trend.

It is important to identify target populations for whom specific strategies are required to better control TB in the community. Two populations that experience greater morbidity from TB than the overall Australian population, are migrants from high TB incidence countries and Indigenous Australians. There are smaller subgroups identified in Table 5 that also warrant further evaluation. Table 5 provides data on risk factors selected for further evaluation by NTAC. The data do not reveal homelessness or residence within correctional facilities to be significant risk factors for TB in Australia, unlike the experience of other countries.[5,6] Similarly, there is no strong evidence that TB is a significant problem in people with HIV infection.

Over the past decade there has been a decline in the rate of TB in Indigenous Australians but their rates are still more than 5 times those of Australian-born non-Indigenous persons. This suggests that despite access to treatment and prevention, social and economic conditions likely remain a significant barrier to closing the gap. However the data for Indigenous Australians need to be interpreted with caution due to the small case numbers. By contrast rates for Australian-born non-Indigenous persons have remained relatively static since 2002 at less than 1 per 100,000 population. The highest proportion of cases, as expected, is in the older age groups reflecting reactivation of remote infection.

Table 8: National tuberculosis performance indicators, performance criteria* and the current status of tuberculosis, Australia, 2007, 2008 and 2009

National tuberculosis performance indicator	Performance criteria	2007†	2008	2009
Annual incidence of TB (cases per 100,000 population)				
Australian-born Indigenous Australians	<1	6.8	6.2	4.8
Australian-born non-Indigenous Australians	<1	0.8	0.9	0.9
Overseas-born persons	*	19.1	20.0	22.4
Incidence in children <15 years, by risk group (per 100,000 population)				
Australian-born Indigenous Australians	<0.1	3.6	1.5	1.5
Australian-born non-Indigenous Australians	<0.1	0.4	0.7	0.7
Overseas-born persons	*	12.0	9.5	10.9
Collection of HIV status in tuberculosis cases‡	100%	87%	94%	95%
Treatment outcome measures (%)				
Cases evaluated for outcomes	100%	99.0	99.2	TBA
Cases that have treatment completed and are cured	>90%	95.4	95.3	TBA
Cases recorded as treatment failures	<2%	0.0	0.0	TBA

* Performance criteria currently under review.

† Evaluation of indicators for the 2007 patient cohort was re-assessed in August 2011.

‡ HIV status is considered complete if case is reported as HIV positive, HIV negative, HIV tested-result unknown, not tested or refused testing.

TBA To be assessed: 2009 patient cohort outcomes to be reported in 2010 annual report.

Specific overseas-born population groups that do not have the same privileges of access to public health care as the general population and who also have a greater incidence of TB can experience diagnostic delays. This can result in personal suffering for them as individuals and also increase the risk for transmission of TB to the general public. One particular group of temporary visa entrants that has been highlighted by studies in Victoria and Queensland is international students (Brown L, personal communication, 24 March 2011; Walpola H, personal communication, 24 March 2011). In addition to experiencing diagnostic delays these students are often in settings of large classrooms resulting in large contact screenings. Given that international students are a significant source of revenue for educational institutions and the Australian economy, it is important to ensure they have equitable access to health care. Among other things, this would then ensure they do not suffer more from TB than the general population, as well as decrease the need for costly contact screening by ensuring earlier diagnosis of TB.

While refugees are screened for active TB disease before coming to Australia, they are at high risk for latent TB infection and are resettled to diverse settings throughout Australia. It remains a challenge to ensure they are adequately integrated into the health care system. For those unauthorised entrants seeking asylum in Australia an additional challenge has been to ensure communication between detention centres and TB programs and ensuring continuity of care once they are released into the community. One of the future priorities for NTAC is to work with the Department of Immigration and Citizenship to improve communication between TB services and/or refugee health services and the detention centres from which such subjects are released to community detention to ensure these people are adequately integrated into supportive health services.

PNG nationals from selected villages in the South Fly District of the Western Province, who access health care in the Torres Strait Treaty Zone, have been highlighted in previous reports.[4] A provision of the Torres Strait Treaty allows free movement between Australia and Papua New Guinea for traditional activities in the Protected Zone and nearby areas.[7] Some of these visitors from PNG access Australian health care centres while visiting the outer Torres Strait Islands. In 2008 and 2009, there were 40 cases of TB among such PNG patients reported to the NNDSS. Multi-drug resistance is a particular concern in this population.[3] It is important that these patients are effectively managed to minimise the further development of drug resistance and prevent transmission to the local Australian population. NTAC supports the view that the best long-term solution is to assist PNG in establishing effective TB treatment and control services so that residents can get the health care they need at home. TB clinicians from both sides of the border are working together towards a staged, safe and ethical approach in handing over both sensitive and multi-drug resistant TB patients to PNG. NTAC advises that there be some provision for TB care of PNG nationals presenting to Australian clinics in the Torres Strait until such time that PNG can ensure adequate management of all cases, and that the transitional arrangement be closely monitored to ensure successful treatment outcomes.

A final target population is health care workers. Figure 7 shows that the number of TB cases in health care workers has been increasing and the majority is overseas born. These data must be interpreted with caution as changes in surveillance practices may contribute to different risk factor reporting over time. Surveillance suggests that many of these cases reactivate remote infection that has been acquired overseas, not from within Australian health care settings. The low incidence of TB in Australia has resulted in relaxation of health care worker screening for TB in many low risk settings throughout Australia. However, the increasing reliance in overseas-born health care workers at high risk for TB highlights the importance for health care settings to remain vigilant in screening their work force.

The data here are reported against key performance indicators established in the *National Strategic Plan For TB Control Beyond 2000,*[8] and at the end of the period addressed by this plan. The criteria for the crude incidence rates in the Australian-born population, by international standards, are very low. Yet the Australian-born non-Indigenous rate has continued to fall, and is now consistently below the indicator. The Australian-born Indigenous rate is also low and has fallen, such that it is approaching the non-Indigenous rate. However, it remains above the indicator and has not reached equivalency with non-Indigenous Australians. By contrast, the rate in overseas-born subjects continues to climb, and this is both indicative of the main source of TB, and the on-going challenge in TB control in Australia.

The indicator for crude incidence rates in children (< 15 years of age) is that of elimination, which would in turn indicate that recent infection is not occurring in Australia. While rates are stable and very low, especially in Australian-born children, this indicator has not been achieved. There are low proportions of cases in children under 15 years (2008 4.5%; 2009 4.2%) particularly those less than 5 years (2008 1.9%; 2009 1.37%). Although the rates observed in overseas-born children are higher (Table 4), the absolute numbers of cases are low. In Australian-born children the rates are less than 1 per 100,000 population and approaching elimination. In addition, the number of meningeal cases in chil-

dren less than 5 years is another key marker of TB control and such cases are rare averaging 1 case only per year since 2002.

Completeness of reporting both of HIV status and outcomes has improved considerably, and both are now close to the 100% benchmark.

In terms of treatment outcomes, high rates of treatment success in reportable cases have consistently exceeded the performance indicator. Relapse and treatment failure rates are important indicators of the quality of treatment programs. Relapse rates in people previously treated in Australia remain low (< 5%) and no treatment failure cases were reported.

In summary, by the performance indicators set in the strategic plan, Australia maintains excellent TB control. At the time of writing this report NTAC is awaiting final endorsement of a new strategic plan (2011–2015). This will include considerably widened strategic goals. With respect to reporting of notification data this will include achieving real time national reporting of combined clinical and laboratory data. Development of epidemiological performance indicators beyond those reported here will include measures of diagnostic delay, incidence of relapsed TB that was treated in Australia within 5 years, occurrence of TB in specific high risk subgroups (in addition to crude rates) and a number of laboratory performance criteria (e.g. proportion of cases confirmed by culture, proportion of laboratories meeting recommended turn-around-time of results, and time to identification of drug resistant TB etc). Given the rise in the TB incidence rate in Australia that is identified in this report, and especially in the face of increasing immigration from countries with high burdens of TB, the implementation of this strategic plan will be important in the future control of TB in Australia.

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Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific and South East Asian Regions, 2010

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Abstract

The World Health Organization (WHO) Gonococcal Antimicrobial Surveillance Programme (GASP) has conducted continuous surveillance of antimicrobial resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region (WPR) to optimise antibiotic treatment and control of gonococcal disease since 1992. From 2007, this has been enhanced by the inclusion of data from the WHO South East Asian Region (SEAR). Over time, there has been recruitment of additional centres in both regions. This report provides an analysis of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR and SEAR derived from results of the 2010 GASP surveillance. In 2010 there were 9,744 *N. gonorrhoeae* isolates examined for their susceptibility to one or more of the antibiotics used for the treatment of gonorrhoea, incorporating External Quality Assurance controlled methods, from reporting centres in 19 countries and/or jurisdictions. A high proportion of penicillin and quinolone resistance was again detected amongst isolates tested in the 'Asian' countries of WHO WPR and SEAR. In contrast, lower levels of penicillin and quinolone resistance were reported from the Pacific Islands of Fiji and New Caledonia. The proportion of gonococci reported as having 'decreased susceptibility' to the third-generation cephalosporin antibiotic ceftriaxone varied widely, ranging from 1.3% to 55.8%. There is a continued need for revision and clarification of some of the *in vitro* criteria that are currently used to categorise the clinical importance of gonococci with different ceftriaxone and oral cephalosporin MIC levels, and to relate these to treatment outcome. Azithromycin resistance was very low in most countries reporting, except in Mongolia where it was 34%. The number of instances of spectinomycin resistance remained low. A high proportion of strains tested continued to exhibit high-level plasmid mediated resistance to tetracyclines. The continuing emergence and spread of antibiotic resistant gonococci in and from the WHO WPR and SEAR underlines the importance of the maintenance and expansion of surveillance programs such as GASP, which are essential for disease control. *Commun Dis Intell* 2012;36(1):95–100.

Keywords: annual reports; antimicrobial resistance; *Neisseria gonorrhoeae*, World Health Organization, Western Pacific Region, South East Asia Region

Introduction

The progressive development of antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* within and across antibiotic classes has, over many years, compromised the treatment and public health management of gonococcal disease in the World Health Organization (WHO) Western Pacific Region (WPR) and South East Asian Region (SEAR), where there continues to be a high incidence of this sexually transmitted disease.

The treatment of gonorrhoea by the public sector in the 'Asian' countries of the WHO WPR, and in the WHO SEAR is substantially based on single-dose treatment regimens of the third-generation cephalosporin agents, predominantly the injectable ceftriaxone, however there are a wide range of dosing regimens used. The oral third-generation cephalosporin most commonly used is cefixime, but dosing regimens are more uniform. Other injectable and oral cephalosporins are also used in some jurisdictions.[1]

Resistance to penicillin, early generation cephalosporin and quinolone antibiotics in the 'Asian' group of WPR and in SEAR countries is widespread.[2,3] However, in the 'Pacific Island' or 'Oceania' group of countries within the WHO WPR, there are a small number of settings where antibiotic resistance continues to be low and the penicillin group of agents continues to be the recommended treatment.[2]

Other antibiotics such as spectinomycin and azithromycin are recommended and used in some countries, although availability and cost limits their wider use. There are few reliable data on antibiotic usage and availability in the private sector in the WHO WPR and SEAR, but anecdotally, a wide variety of antibiotics are used, often in suboptimal doses.[1]

It is recommended by the WHO[4] and others[5,6] that therapeutic regimens be supported by data from

surveillance of AMR in *N. gonorrhoeae,* and further that routine use of an antibiotic for treatment be discontinued when treatment failure occurs and/or AMR reaches a level of 5%. The WPR Gonococcal Antimicrobial Surveillance Programme (GASP) has documented the emergence and spread of AMR in *N. gonorrhoeae* in the WHO WPR from 1992 [2,7] to provide information for action and to optimise the antibiotic treatment for gonorrhoea. The WHO SEAR GASP has published similar data intermittently.[3]

Significant concerns have been expressed following the appearance and spread of gonococci with 'decreased-susceptibility' to the later-generation cephalosporins in the WHO WPR.[8–11] This was followed by reports of treatment failures with several oral third-generation cephalosporins.[8,10,12] The gonococci involved would be classified as 'multi-drug resistant gonococci' by recently proposed criteria.[4] This report provides an analysis of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR and SEAR derived from the results of the GASP surveillance for the calendar year 2010. The difficulties currently experienced with reliable detection and reporting of gonococci with altered susceptibility to cephalosproins[4] and strategies implemented to address this in these settings are discussed.

Methods

The methods used by the WHO WPR GASP and more recently by WHO SEAR, have been publishedand provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs (EQA) used to generate these data.[7] These general principles were unaltered in 2010. The expansion of the reference panel of *N. gonorrhoeae* control strains used in WHO WPR and SEAR EQA programs continues.[13] This is to monitor the impact of emerging resistance (initially with the quinolones and, latterly, the third-generation cephalosporins) and address issues related to the detection of these forms of resistance.[13,14]

Results

In 2010, there were 9,744 *N. gonorrhoeae* examined for their susceptibility to one or more antibiotics used for the treatment of gonorrhoea using EQA controlled methods. These were reported from 21 centres in 19 countries and jurisdictions; 15 in the WHO WPR and 4 from the WHO SEAR. Other centres were unable to supply data for 2010 but maintained contact with the program through participation in the EQA program. In 2010, data were not available from Laos, Papua New Guinea, Tonga and Myanmar.

Quinolone resistant *Neisseria gonorrhoeae*

In 2010, quinolone resistance (QRNG)or reduced susceptibility was in excess of 90% of all *N. gonorrhoeae* examined in Brunei, Cambodia, China, Hong Kong SAR, Korea, the Philippines and Vietnam (WHO WPR) and in Bhutan, India, Sri Lanka and Thailand (WHO SEAR). Rates between 70% and 90% were reported from Japan, Malaysia and Singapore. Lower, but still substantial, proportions of QRNG were present in Australia, Mongolia, and New Zealand. Quinolone resistance remained below 1% in Fiji and New Caledonia (Table 1).

Penicillin resistance

Penicillin resistance rates were lower than those for the quinolone antibiotics, and in a similar pattern to that of previous years. Not all jurisdictions monitored penicillin resistance because treatment of gonorrhoea with this group of antibiotics has long been discontinued. Even where this surveillance was performed, it was sometimes limited to detection of beta-lactamase production (Table 2).

Decreased susceptibility and resistance to third-generation cephalosporins

Regionally, 9,282 isolates of *N. gonorrhoeae* were examined for cephalosporin susceptibility in 2010, and WHO EQA validated data were available for 7,024 of these isolates at the time of this report. Most of these centres tested isolates for susceptibility to ceftriaxone only and the proportion of gonococci reported with 'decreased susceptibility' to ceftriaxone varied widely. Singapore reported 1.3% and Australia reported 4.8% of isolates with decreased susceptibility to ceftriaxone; whereas China (55.8%), Korea (29.3%), Japan (20.3%), Hong Kong SAR (23.3%) and India (10.8%) reported gonococci with decreased susceptibility to ceftriaxone in much larger proportions. There were no EQA validated data reporting resistance in either region.

Spectinomycin resistance

In 2010, as in previous years, there were only a few sporadic cases of resistance to spectinomycin from a limited number of settings reported from the 15 centres testing 9,315 isolates for resistance to this antibiotic. There were low numbers of isolates (12 or less) with *in vitro* resistance or decreased susceptibility to spectinomycin reported from Mongolia; China and Bhutan, similar to the GASP data for 2009.

Tetracycline resistant *Neisseria gonorrhoeae*

Tetracyclines are not a recommended treatment for gonorrhoea in the WHO WPR or SEAR, but historical data on the spread of high-level plas-

mid mediated tetracycline resistant *N. gonorrhoea* (TRNG) continues to be monitored in some countries. Fifteen centres tested gonococci for TRNG in 2010, and up to 70% of gonococci exhibited this form of resistance. The proportion of TRNG has been high in some parts of the WPR for many years, with Brunei reporting TRNG in the percentage range of 71%–100%. Mongolia, China, Hong Kong SAR, Singapore and Vietnam reported proportions of TRNG in the range 35%–70%. Proportions in the range 10%–34% were reported from Australia, India, Korea and New Zealand, Papua New Guinea, Sri Lanka and the Philippines. The number of strains tested in the countries and jurisdictions mentioned above are shown in Tables 1 and 2.

Azithromycin resistance

Azithromycin AMR data are reported for the first time in this GASP report. This antibiotic can be used either as a primary treatment for gonorrhoea or as adjunctive treatment for other pathogens, and resistance to this antibiotic is known to occur in the WHO WPR. In 2010, 6 countries (four in the WPR and two in SEAR) tested 5,295 *N. gonorrhoea* isolates for susceptibility. There was no resistance (0%) reported from Cambodia; Vietnam and India and very low rates (< 1%) from Australia. In contrast 34% resistance was reported from Mongolia.

Discussion

This paper reports the findings of the WHO Gonococcal Antimicrobial Surveillance Programme for the Western Pacific and South East Asian Regions for 2010. In this calendar year there were 9,744 *N. gonorrhoeae* isolates examined for their susceptibility to one or more of the antibiotics used for the treatment of gonorrhoea, incorporating external quality assurance controlled methods, from reporting centres in 19 countries and/or jurisdictions. Important limitations apply to data generated from surveys of this kind. Inevitably, only low sample numbers were available in some centres for reasons including the absence, abandonment or inability to perform laboratory-based diagnostic culture and where syndromic management is used. Further, there is increasing substitution of diagnostic nucleic amplification assays replacing culture. Resource restrictions in many settings in the region limit the capacity for the 'gold standard' of susceptibility testing based on minimum inhibitory concentra-

Table 1: Quinolone resistant *Neisseria gonorrhoeae* (QRNG) in the World Health Organization Western Pacific Region and the South East Asia Region, 2009 (n = 9,744 strains)

		Less susceptible		Resistant		All QRNG	
Country	n	n	%	n	%	n	%
Western Pacific Region							
Australia	3,997	43	1.1	1,342	33.6	1,385	34.7
Brunei	396	127	32.1	242	61.1	369	93.2
Cambodia	76	2	2.6	73	96.1	75	98.7
China	1,398	38	2.7	1,250	89.4	1,288	92.1
Fiji	336	0	0.0	2	0.6	2	0.6
Hong Kong SAR	947	18	1.9	916	96.7	934	98.6
Japan	403	3	0.7	292	72.5	295	73.2
Korea	82	2	2.4	76	92.7	78	95.1
Malaysia	17	3	17.6	12	70.6	15	88.2
Mongolia	690	7	1.0	231	33.5	238	34.5
New Caledonia	197	0	0.0	1	0.5	1	0.5
New Zealand	72	0	0.0	21	29.2	21	29.2
Philippines	59	0	0.0	57	96.6	57	96.6
Singapore	160	2	1.3	117	73.1	119	74.4
Vietnam	86	3	3.5	83	96.5	86	100.0
South East Asia Region							
Bhutan	179	0	0.0	172	96.1	172	96.1
India	37	1	2.7	36	97.3	37	100.0
Sri Lanka	72	0	0.0	65	90.3	65	90.3
Thailand	540	111	20.6	416	77.0	527	97.6
Total	9,744	360	3.7	5,404	55.5	5,764	59.2

Table 2: Penicillin resistance in *Neisseria gonorrhoeae* in the World Health Organization Western Pacific Region and the South East Asia Region, 2010 (n = 9,702 strains)

Country	n	PPNG		CMRP		All penicillin resistance	
		n	%	n	%	n	%
Western Pacific Region							
Australia	3,997	462	11.6	699	17.5	1,161	29.0
Brunei	397	210	52.9	71	17.9	281	70.8
Cambodia	76	–	–	–	–	59	77.6
China	1,398	534	38.2	NS	–	NS	–
Fiji	336	16	4.8	12	3.6	28	8.3
Hong Kong SAR	947	304	32.1	182	19.2	486	51.3
Japan	403	1	0.2	158	39.2	159	39.5
Korea	82	14	17.1	29	35.4	43	52.4
Malaysia	17	1	5.9	3	17.6	4	23.5
Mongolia	605	–	–	–	–	361	59.7
New Caledonia	197	1	0.5	0	0.0	1	0.5
New Zealand	72	0	0.0	13	18.1	13	18.1
Philippines	59	57	96.6	0	0.0	57	96.6
Singapore	160	57	35.6	22	13.8	79	49.4
Vietnam	86	27	31.4	15	17.4	42	48.8
South East Asia Region							
Bhutan	179	–	–	–	–	178	99.4
India	37	14	37.8	4	10.8	18	48.6
Sri Lanka	43	28	65.1	4	9.3	32	74.4
Thailand	611	503	82.3	88	14.4	591	96.7
Totals	9,702	2,229	23.0	1,300	13.4	3,593	37.0

PPNG Penicillinase producing *Neisseria gonorrhoeae* (β-lactamase positive).
CMRP Chromosomally mediated resistance to penicillin.
NS Data not supplied (Gonococci in China were examined for penicillinase production only).

tions (MIC) methodology, even when gonococcal isolates are available, so that disc testing procedures with methods incorporating standardised control strains remain the only practical means of *in vitro* assessment of gonococcal antibiotic susceptibility in many situations.[14] Despite this, in the absence of other surveillance data sources, the WHO WPR GASP has been conducted for more than 20 years, under the same conditions and the annual WHO WPR gonococcal surveillance reports continue to provide reliable trend data for the region as a whole. Since 2007, the addition of quality controlled information has been available from the WHO SEAR. The consistent results that have been obtained over time in similar countries in the WPR reinforce the significance of the findings. This allows inferential extrapolation of the data obtained to those countries that are unable to participate fully in each surveillance period.

The patterns of resistance to the quinolone and penicillin groups of antibiotics by jurisdiction for the year 2010 are shown in Tables 1 and 2. The WHO recommends that use of an antibiotic for routine treatment be removed from standard treatment schedules when therapeutic failure reaches a level of 5%. The previously described patterns of resistance to these groups of antibiotics across the WHO WPR and SEAR[2,7] were again evident in 2010. Whilst a high proportion of both penicillin and quinolone resistance was detected amongst isolates tested in most reporting centres, from the Pacific Island states, New Caledonia continues to report low levels of both penicillin and quinolone resistance and Fiji low levels of quinolone resistance and low but increased penicillin resistance.

N. gonorrhoeae in the WPR and SEAR with decreased susceptibility to third-generation cephalosporins have been reported for a number of years.[4,7–12] This has been accompanied by reports of treatment failure with oral third-generation cephalosporins in a significant number of cases.[6,8,10,12] Regionally, surveillance of gonococcal AMR to the third-gener-

ation cephalosporins (ESCs) focuses on ceftriaxone because of its widespread use,[1] and data reported in 2010 are based primarily on testing of the *in vitro* susceptibility of gonococcal isolates to ceftriaxone. However, there are ongoing concerns regarding assessment of *N. gonorrhoeae* with altered susceptibility to the ESCs. The mechanisms of resistance in *N. gonorrhoeae* to the ESCs are multiple and complex, involving the aggregation and expression of a number of different genes within *N. gonorrhoeae,*[15–17] and further, other important mechanisms of gonococcal cephalosporin resistance exist, but are yet to be fully elucidated.[16] The effects of the polygenic involvement on *in vitro* susceptibility of the injectable agents such as ceftriaxone and on the oral cephalosporins such as cefixime and ceftibuten differ considerably, indicating that susceptibility data for ceftriaxone cannot reliably predict the outcomes of treatment with oral cephalosporins.[4,12] To address this there is ongoing revision and clarification of some of the *in vitro* criteria that are currently used to categorise and report on the different MIC levels that arise with both the injectable and oral cephalosporins through WHO working groups.[4] In 2010, with the use of the WHO reference panel[13] in particular 'WHO K' the ceftriaxone control for decreased susceptibility, laboratories have the measure to correctly interpret MIC results of test isolates, however, some limitations continue to be evident in reporting AMR and in EQA performance data.[14]

In 2010, the revised panel of *N. gonorrhoeae* WHO control strains was further developed and distributed in the WPR and SEAR and widespread incorporation of these has better defined 'decreased susceptibility' and 'resistance' to the different third-generation cephalosporin antibiotics.[13,14,18] This is not an easy task because of the need to define 'clinical' as opposed to *in vitro* resistance through better and more complete examination of gonococci isolated from documented treatment failures, and also by use in various circumstances of the different treatment doses, especially for ceftriaxone.[1] It is also established that elimination of *N. gonorrhoeae* from some sites is also more difficult, e.g. extra-genital tract infections are harder to eradicate.[19] The 2010 data are indicative of a well documented increase in the MIC values of cephalosporins in gonococci found in both regions[15–17] and are alarming in terms of the proportion of isolates with decreased susceptibility and the absence of alternative therapies on the horizon. Very few isolates were tested separately for their susceptibility to the oral cephalosporin agents. It is thus not possible at present to interpret the *in vitro* data in terms of likely clinical outcome other than in general terms.

Spectinomycin resistance has been only infrequently reported in GASP from the WPR and latterly the SEAR. A form of high-level resistance due to a single-step ribosomal mutation has been described,[20] and there are other reports of unexplained low-level resistance or decreased susceptibility. The availability of spectinomycin as a treatment option has been significantly reduced following a lack of reliable supplies of the drug. Spectinomycin resistance has not been detected in WHO WPR or SEAR for many years and overall resistance to this antibiotic remains low in both regions.

In the 6 countries reporting testing for azithromycin AMR there was low or no resistance reported from Cambodia; Vietnam, India and Australia, and 34% resistance was reported from Mongolia. There are recent reports elsewhere of high-level azithromycin resistance following widespread use of this antibiotic [21] and it is now recommended as part of a dual therapy strategy in the United Kingdom National Guideline for treatment of gonorrhoea in adults.[22] Azithromycin has not been a part of the WHO GASP core group of antibiotics tested in the past, however it is evident that its inclusion is necessary and AMR data will be reported where available in the GASP.

Increased and improved surveillance of gonococcal antibiotic resistance in the WHO WPR and SEAR is urgently required and this has long been evident.[4–6] Further to this, expanding surveillance of resistance to include other antibiotics is imperative as therapeutic options diminish, and enhancement of surveillance should also include test of cure studies, which are crucial to determine both the clinical correlates of surveillance data, and for disease control. The emergence and spread of antibiotic resistant gonococci from the WHO WPR and SEAR to other parts of the world has been documented,[4] and there is a high likelihood that, unless better disease control becomes a reality, new forms of resistance will continue to appear and spread well beyond these regions. A suggested approach to the closely related issues of gonococcal disease control and AMR control in *N. gonorrhoeae* has recently been published from WHO sources.[4] Implicit in these recommendations is the availability of reliable and verifiable antibiotic resistance surveillance data.

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Peer-reviewed articles

An outbreak of *Salmonella* Typhimurium linked to a kebab takeaway shop

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Abstract

This paper describes the public health investigation and response to a *Salmonella* Typhimurium outbreak in June 2010 in the Central Coast of New South Wales. Two complaints from people with acute gastrointestinal illness pointed to food from a kebab takeaway shop as the cause of their illness. Liaison between public health and food authorities ensured timely epidemiological and environmental investigations leading to prompt identification and elimination of the point source. A case series investigation identified 45 outbreak cases including 31 laboratory-confirmed and 14 epidemiologically-linked cases. The food vehicles identified were hommus and tabouli – 93% of cases reported having one or both items in their kebab. *S.* Typhimurium with the same MLVA type was found in stool specimens from outbreak cases and in food (including hommus and tabouli) and environmental samples collected at the kebab takeaway shop. Education of commercial food handlers, reduction of poultry meat contamination and collaboration between public health and food authorities to ensure prompt identification and control of outbreaks are important strategies to reduce *Salmonella* related illness. *Commun Dis Intell* 2012;36(1):101–106.

Keywords: Salmonella, outbreak, foodborne, kebab, cross-contamination

Introduction

Foodborne gastroenteritis is a significant public health problem in Australia, incurring substantial societal and health care costs. Even though the vast majority of the estimated 5.4 million cases of foodborne disease which occur in Australia each year experience a mild and self-limiting illness and do not seek medical attention, foodborne gastroenteritis is estimated to result in 2.1 million days of work lost, 1.2 million doctor visits, 18,000 hospital admissions and over 100 deaths annually.[1]

In Australia, non-typhoidal *Salmonella* is responsible for a significant proportion of foodborne gastroenteritis cases[1] and is also the most commonly implicated aetiological agent in outbreaks of foodborne illness.[2] In the quarter from April to June 2010 *Salmonella* was the aetiological agent for 10 of the 35 confirmed or suspected foodborne disease outbreaks reported in Australia.[3]

On Tuesday 8 June 2010, the Central Coast office of the Northern Sydney Central Coast Public Health Unit (PHU) received notification of 2 complaints to the New South Wales Food Authority (NSWFA). The complaints referred to 5 people who had developed acute gastrointestinal symptoms and who had pointed to food from a kebab takeaway shop in the Central Coast (the shop) as the cause of their illness. The shop had been implicated in a *S.* Typhimurium outbreak in April 2007 involving 44 laboratory-confirmed cases. This paper describes the public health response to the notifications received in June 2010.

Methods

Epidemiological investigation

Following receipt of the NSWFA notifications, staff from the PHU interviewed the 5 cases reported. The PHU obtained details of food eaten by the cases over the 3 days prior to the onset of symptoms to determine common exposures. The information obtained supported the hypothesis that the shop that had been identified by the complainants was the likely source of their illness. The PHU requested that the 5 cases attend their general practitioner (GP) to provide stool specimens for testing.

Further cases were identified from laboratory notifications of salmonellosis received by the PHU, which had a specimen collection date in June 2010, or from reports to the PHU of persons meeting the case definition. Enhanced surveillance was initiated by alerts to hospital emergency and pathology departments in the Central Coast about the outbreak and requests to report any cases meeting the case definition.

The NSWFA investigated the presence of illness amongst staff at the shop and their exposure to food from the shop.

Case definition

An outbreak case was defined as any person with acute gastrointestinal illness with onset after eating food from the shop from 30 May. As the investiga-

tion progressed, the case definition became more specific to only include exposures between 30 May and 15 June 2010.

Persons with laboratory-confirmed salmonellosis were interviewed using a suspected foodborne illness investigation form to determine their exposure and to obtain demographic and illness information. Persons with laboratory-confirmed salmonellosis who met the case definition were categorised as outbreak laboratory-confirmed cases. Persons with laboratory-confirmed salmonellosis who were not able to be interviewed after 3 attempts were excluded from the study.

Persons reported as meeting the case definition but who were not tested were categorised as outbreak epidemiologically-linked cases.

Environmental investigation

Based on the initial epidemiological information, the NSWFA conducted an inspection of the shop on Wednesday 9 June. At this time the NSWFA collected food and environmental samples for testing, including cooked and uncooked food items, and swabs of surfaces, equipment and utensils used for food storage and preparation. The NSWFA assessed hygiene and food safety controls and discussed these with the proprietor.

On Tuesday 15 June the NSWFA obtained further food and environmental samples from the shop for testing. The NSWFA requested information from the proprietor of the shop about numbers of kebabs sold in the week up to 9 June. The NSWFA also investigated the suppliers of the various food ingredients used at the shop.

Laboratory investigation

Stool specimens were cultured at several laboratories in the Central Coast and Sydney. Serotyping and multiple locus variable number of tandem repeat analysis (MLVA) were conducted at the Institute of Clinical Pathology and Medical Research (ICPMR) at Westmead, Sydney. Phage typing was conducted at the Microbiological Diagnostic Unit Public Health Laboratory (MDUPHL) at the University of Melbourne. NSWFA samples were cultured at the Division of Analytical Laboratories (DAL) at Lidcombe, Sydney, serotyped and phage typed at the Australian Salmonella Reference Centre in Adelaide and MLVA typed at ICPMR.

Data analysis

Data were entered into and analysed using a Microsoft® Excel spreadsheet.

Results

Epidemiological investigation

The 5 initial cases belonged to 2 unrelated groups. Three cases were part of a group of 4 including 2 residents of the Central Coast and 2 visitors to the Central Coast. Three members of the group ate kebabs from the shop and became sick, while the 4th member ate food from elsewhere and remained well; no other common exposures for the cases could be identified. The 2 cases in the other group belonged to a household of five; the 2 cases shared a kebab from the shop, the other members of the household did not eat any food from the shop and remained well; all other foods consumed by the cases were also eaten by the other members of the household.

Salmonella notifications

The PHU received 52 *Salmonella* notifications for the month of June 2010 including the notifications for two of the initial complainants.

Of the 52 notifications, 29 (55.8%) were identified as outbreak cases. Twenty (38.5%) notifications were not directly linked to the shop and no common exposure was identified for these *Salmonella* infections. Three (5.8%) laboratory-confirmed *Salmonella* notifications could not be contacted after 3 attempts and were excluded from the study.

A further 2 laboratory-confirmed outbreak cases were identified by the PHU at an adjacent region among residents of that region who had visited the Central Coast and consumed food from the shop. Overall, 31 laboratory-confirmed outbreak cases were identified.

Epidemiologically-linked cases

Fourteen cases of acute gastrointestinal illness that met the outbreak case definition but who had not submitted a stool specimen for testing were identified. Thirteen were identified through interviews with laboratory-confirmed outbreak cases and one through a hospital in the Central Coast.

All cases

All 45 cases in this outbreak (31 laboratory-confirmed and 14 epidemiologically-linked) were identified as customers of the food outlet. No cases were reported among staff – staff reported to the NSWFA that they regularly consumed kebabs, including hommus and tabouli, from the shop.

The majority (31/45, 69%) of outbreak cases were female. Age was known for 35 cases and ranged from 7 to 70 years (median = 28).

Illness

Symptom data were available for 43 of the 45 outbreak cases. All but 1 (42/43, 98%) reported diarrhoea, with 4 cases reporting bloody diarrhoea. The second most commonly reported symptom was abdominal cramps (74%), followed by fever (63%), nausea (63%) and vomiting (53%). Sixty per cent of cases reported additional symptoms, such as dizziness and headache.

In those who reported that they had recovered by the time they were interviewed (21 cases), the duration of illness ranged from 1 to 18 days (median = 8). Many cases reported having to take time off work or study for a length of 1 to 10 days (median = 5) because of their illness.

Thirty-one cases sought medical assistance either from a GP (13), hospital (11) or both (7). Eight cases required hospital admission; half of these had visited a GP prior to visiting the hospital and being admitted, while the other four had gone directly to a hospital emergency department. Length of stay in hospital ranged from 2 to 7 days; 3 cases were admitted for 3 days and three for 4 days (median length of stay = 3.5).

A secondary salmonellosis infection occurred in a child who had not consumed food from the shop. The child's parent had developed acute gastrointestinal illness following consumption of a kebab from the shop on 4 June 2010 and cared for the child while ill. The child was admitted to hospital with salmonellosis for 5 days.

Food exposures

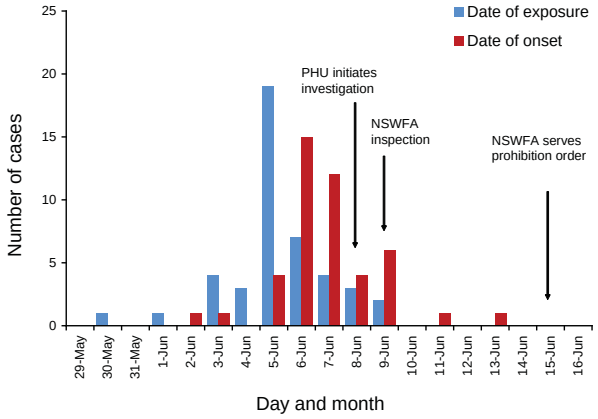
Information on the date of purchase of food at the shop was available for 44 cases. Cases purchased food between 30 May and 9 June 2010 with 59% purchasing food during the weekend of 5–6 June (Figure). Incubation periods ranged from 8 to 100 hours (median = 24.5 hours). The earliest onset date reported was 2 June and the latest was 13 June; the outbreak peaked on 6 June (Figure).

Food consumed

The shop served kebab rolls, vegetarian rolls, falafel rolls and savoury crepes. Chicken and a mix of beef and lamb were the meats used for the kebab rolls and, if requested by the customers, were also used in crepes. A variety of salad items, sauces and other accompaniments were available to be served in rolls and crepes.

All but 1 case reported eating kebabs. Table 1 lists the food items consumed by the 45 cases. Forty-two cases (93%) reported having either hommus or tabouli or both in their kebab. Of the cases who did not have hommus nor tabouli, one reported having a beef/lamb kebab, one reported having a chicken crepe (the case with the earliest exposure and onset date), and one reported having a chicken kebab with salad.

Figure: Number of laboratory-confirmed and epidemiologically-linked cases, by date of exposure* and date of onset of symptoms



* Excludes 1 laboratory-confirmed case with unknown date of exposure.

Environmental investigation

The shop was situated in the main food court of a busy shopping centre. The shop consisted of a food display and serving area at the front and a rear food preparation and storage area that were not visible to the public. Chicken meat, marinated in-house, was stacked onto a large metal skewer to assemble a chicken meat kebab log; the mixed lamb and beef kebab logs were purchased already assembled; the kebab logs were then cooked in vertical rotisseries in the front area of the shop. The proprietor stated that over 1,000 kebab rolls had been sold in the week up to 9 June 2010.

The initial inspection and interview with staff working at the shop on Wednesday 9 June 2010 identified only minor hygiene defects and potential risks (for example, lack of evidence of consistent use of sanitising step following washing of food utensils and other food contact equipment; storage of sealed bag of raw minced meat alongside bags of vegetables and fruits) but did not reveal any substantial breaches to food handling practices and sanitation. However, the results received on Tuesday 15 June for the tests conducted on samples collected on 9 June 2010 showed the presence of *S.* Typhimurium in food and environmental samples (Table 2).

On Tuesday 15 June 2010, on the basis of these test results, the NSWFA issued an immediate prohibition order to operate and obtained further food and

Table 1: Food items consumed by outbreak cases

	Cases consuming the specific food item N=45	
Food items consumed	**Number**	**Percentage**
Kebab	44	98
Crepe	1	2
Kebab/crepe fillings:		
Hommus	40	89
Tabouli	36	80
Tomato	38	84
Lettuce	35	78
Chicken	26	58
Beef/lamb	25	56
Onion	21	47
Cheese	13	29
BBQ sauce	8	18
Garlic sauce	7	16
Tomato sauce	4	9
Hot chilli sauce	3	7
Sour cream	3	7
Sweet chilli sauce	4	9

Table 2: Environmental investigation test results received for samples collected on 9 June 2010

Samples	Results
Food	
Hommus Tabouli Chicken kebab roll (purchased, contained hommus and tabouli)	*S.* Typhimurium PT 170/108 MLVA type 3-9-7-15-523
Marinated raw chicken	*Salmonella* Infantis
Cooked chicken meat Cooked beef and lamb meat Tahini Pepper Garlic powder Paprika	No pathogens
Environmental	
Coolroom door handle Plastic cabbage leaves (used to line trays containing ready to eat vegetables) Chilled display unit	*S.* Typhimurium PT 170/108 MLVA type 3-9-7-15-523
Lower shelf of food preparation bench	*S.* Typhimurium PT 170/108 and 193 MLVA type 3-9-7-15-523
Slicing machine Knives Metal tongs Stick blender Support table for chicken Plastic apron	No pathogens

environmental samples for testing. No *Salmonella* was detected in any of the samples obtained on 15 June. No issues were identified by the traceback investigation of suppliers of food items. The food outlet remained closed for several weeks during which time the proprietor renovated the premises. The prohibition order was lifted on 30 July after renovations were completed and the business was able to demonstrate adequate food safety skill and knowledge. The business also undertook to cease in-house preparation of chicken kebab logs to minimise future risk of cross-contamination.

Laboratory investigation

The 31 samples from outbreak cases, which had initially tested positive for *Salmonella* species, were serotyped and found to be *S.* Typhimurium. The MLVA types were 3-9-7-14-523 (16 cases), 3-9-7-15-523 (14 cases), and 3-9-7-13-523 (1 case); MLVA typing results were received several weeks after receiving the respective salmonellosis notification. Phage typing of isolates showed them to be phage type (PT) 170/108.

Three food samples and 4 environmental surface swabs tested positive for *S.* Typhimurium MLVA type 3-9-7-15-523 and PT 170/108 (Table 2).

MLVA types with variation of 1–2 digits (repeat differences) at one of the three inner loci (the 4th locus in these isolates) combined with a clear epidemiological link, as found in this occasion between human cases and the implicated food and environment, indicate that this was the same infectious agent.[4] The secondary case was *S.* Typhimurium MLVA type 3-9-7-15-523.

Discussion

This investigation identified a point source *S.* Typhimurium PT 170/108 outbreak resulting from consumption of contaminated food from a kebab takeaway shop. A case series study identified 45 outbreak cases including 31 laboratory-confirmed cases of salmonellosis and 14 epidemiologically-linked (not tested) cases. One secondary case of salmonellosis was identified. Compelling laboratory evidence supported the initial suspicion, which arose from the notification of 2 complaints to the NSWFA, that the shop was the source of the cases' illness. The same infectious agent was found in specimens from outbreak cases and in environmental and food samples collected at the shop, in particular, hommus and tabouli that were consumed by 93% of cases.

This study highlights the importance of timely investigation and liaison between agencies to ensure prompt control of foodborne outbreaks. In this particular incident, the first notifications were received in the week that preceded the 12–14 June 2010 long weekend; the PHU suspected that more people would be consuming food from the implicated shop over a long weekend than on an ordinary weekend. As the shop had been implicated in a relatively large *Salmonella* outbreak 3 years before, there were concerns about a repetition of an incident of similar scale. Close liaison between the PHU and NSWFA from the outset of the incident ensured a timely inspection was conducted, with a food preparation review and cleaning and sanitation undertaken by the implicated shop likely removing the source of infection. The effectiveness of these actions is supported by the observation that no cases reported an exposure after the initial inspection on 9 June.

The outbreak attack rate was estimated at less than 5% (over 1,000 kebabs were sold in the week to 9 June and the investigation identified 43 outbreak cases with exposure in the same period) and was surprisingly low. However, it is likely that the study identified only the cases with more severe illness while milder cases remained unreported. Despite the outbreak's low attack rate, the burden of illness was significant, with almost a fifth of cases being admitted to hospital and many others reporting taking time off work or study because of their illness.

Kebabs, which are a popular takeaway food in Australia, have been identified as potentially posing a food safety risk for consumers.[5] The mechanisms by which this could occur are many and it is possible that their prevalence has changed over time. A survey of kebab businesses conducted by Food Safety Victoria in 2001 concluded that some of the food handling and meat kebab cooking practices observed could result in foodborne illness.[6] Education of food industry workers following the survey and implementation of the survey recommendations, such as use of thinner cuts of meat and use of a secondary cooking step, are likely to have had a positive effect on practice and to have prevented potential outbreaks. More recently, poor refrigeration and non-effective sanitation practices and cross-contamination issues between raw and prepared foods were identified as issues of concern by a food safety survey of retail doner kebabs conducted by the NSWFA in 2004.[7]

Salmonellosis resulting from cross-contamination of ready-to-eat foods has been reported in the literature.[8] Cross-contamination is the most likely explanation for the outbreak being reported here, where hommus and tabouli were found to be positive for *S.* Typhimurium with the same MLVA types as found in the outbreak cases. *Salmonella* is a common contaminant of chicken meat [9] and it is possible that the hommus and tabouli were cross-contaminated. Uncooked chicken meat samples obtained at the shop on 9 June 2010 tested positive for *Salmonella* Infantis but not for *S.* Typhimurium and no pathogens were identified in cooked chicken

meat sampled as part of this investigation. However, it is possible that chicken meat contaminated with *S.* Typhimurium was present at the shop prior to 9 June 2010 and, as a result of cross-contamination was transferred, possibly by spraying of raw meat juices, to surfaces and equipment and via these to the hommus and tabouli.

The outbreak reported here is unusual in that it was the 2nd *S.* Typhimurium outbreak at the same shop in a period of just over 3 years. The April 2007 outbreak, due to *S.* Typhimurium PT U302, was also suspected to have resulted from cross-contamination of ready-to-eat foods from raw meats. At that time the NSWFA provided guidance to the same proprietor who adopted the recommended steps to minimise the risk of an outbreak occurring again. It is likely that the standards of practice at the shop deteriorated since then, even though this was not evident at the time of the inspection on 9 June 2010. No prosecution action was taken in 2007, however, following the 2010 outbreak the proprietor of the shop was prosecuted, convicted of breaches of the relevant legislation and fined accordingly.[10]

Further education of commercial food handlers to improve food handling practice and reduction of poultry meat contamination would help reduce the risk to consumers. The recent introduction in New South Wales of food laws, which require retail businesses handling potentially hazardous foods to appoint a Food Safety Supervisor,[11] are welcome. Furthermore, the Primary Production and Processing Standard for Poultry Meat, which becomes enforceable in May 2012, will introduce new requirements for the poultry industry, with a view to reducing contamination of poultry meat with *Salmonella* and other pathogens.[12]

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Imported malaria in the Northern Territory, Australia – 428 consecutive cases

Timothy J Gray, James M Trauer, Merv Fairley, Vicki L Krause, Peter G Markey

Abstract

Malaria is a notifiable disease in Australia with an average of 600 notifications per year in returned travellers or newly arrived refugees, migrants and visitors. Although endemic disease has been eliminated from the tropical north of Australia, the region remains malaria receptive due to the presence of efficient mosquito vectors. This study analyses enhanced surveillance data collected by the Centre for Disease Control on all cases of malaria notified in the Northern Territory from 1 January 2000 to 31 December 2010. There were 428 malaria episodes notified that occurred in 391 individuals with a median age of 26 years. Of these, 71.4% were male, 40.5% were Australian nationals and 38.0% were prescribed chemoprophylaxis. Primary infection consisted of 196 (51.3%) cases of *Plasmodium falciparum,* 165 (43.2%) *P. vivax,* 2 (0.5%) *P. ovale,* 1 (0.3%) *P. malariae* and 18 were mixed infections. There were 46 episodes of relapsed infection. Residents of non-malarious countries were most likely to have acquired primary infection in East Timor (40.6%), Papua New Guinea (27.8%), Indonesia (18.7%) and Africa (6.4%). Primary infection was diagnosed after a median 19 days (interquartile range (IQR) 7–69) after arrival in Australia for cases of *P. vivax* compared with 4 days for *P. falciparum* (IQR 2–11). Screening protocols led to the diagnosis of 27.2% of cases. Eighty-seven per cent of patients were admitted to hospital at the time of their malaria diagnosis with median duration of 3 days (IQR 2–4) and one patient died. Resettlement of people from endemic countries, as well as military and civilian activities, influences the prevailing notification rates and *Plasmodium* species type. *Commun Dis Intell* 2012;36(1):107–113.

Keywords: malaria, surveillance, Northern Territory, screening

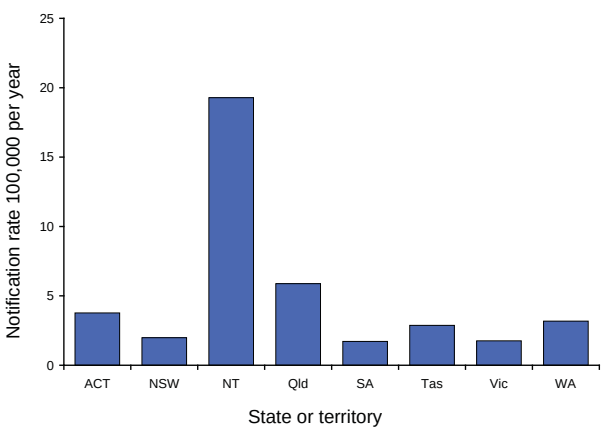
Background

Malaria remains the most frequent cause of fever in returned travellers, being the specific diagnosis in 21% of returned travellers with fever as reported across a worldwide surveillance network, including Australia.[1] Recent migrants and refugees are also at risk of malaria, with a hospital-based survey in Australia reporting 12% of confirmed malaria cases between 1997 and 2001 being from these groups.[2] The last documented case of endemic malaria in the Northern Territory occurred at Roper River in 1962.[3] Subsequently, the World Health Organization (WHO) reported the eradication of malaria from Australia in 1981,[4] but the tropical north remains susceptible to the re-establishment of the disease due to the presence of efficient *Anopheles* mosquito vectors.[5] Two outbreaks of *Plasmodium vivax* infection in northern Queensland[6,7] as well as sporadic introduced cases[8,9] emphasise the public health importance of malaria control in the tropical north.

In the 11 years from January 2000 to December 2010, 6,856 cases of malaria were imported into Australia, with 6.4% of these cases notified in the Northern Territory.[10] The Northern Territory has the highest rate of malaria notifications of all states and territories with 19.3 per 100,000 per year, compared with the national rate of 3.0 per 100,000 per year (Figure 1).[10] Under legislative requirements, laboratories and physicians notify the Northern Territory Centre for Disease Control (NT CDC) of all confirmed or probable cases of malaria. CDC staff routinely interview all individuals diagnosed with malaria and review all hospital medical charts.

Staff from the NT CDC, including those from Medical Entomology, carry out public health risk assessments for each notification and advise on the need for infection control measures such as hospitalisation or avoidance of mosquito exposure.

Figure 1: Average annual notification rates per 100,000 population, Australia, 2000 to 2010[10]



Frequently, mosquito trapping is performed in the vicinity of the case's residence and insecticide fogging is used if necessary.

In addition to the core data items applicable to all notifiable diseases, the NT CDC staff prospectively record additional malaria-specific data incorporating epidemiological, treatment and outcome variables. This report summarises these data for the 428 consecutive episodes of malaria diagnosed in the Northern Territory from January 2000 to December 2010.

Methods

Cases were defined according to the national case definition of laboratory-confirmed malaria, which requires the specific identification of malaria parasites by microscopy on blood film with confirmation of species or the detection of *Plasmodium* species by nucleic acid testing.[11] Probable cases diagnosed by the presence of malaria antigen in whole blood were also included in this analysis.

Malaria data of cases notified between 1 January 2000 to 31 December 2010 were extracted from the Northern Territory Notifiable Diseases System (NTNDS), de-identified and imported into Stata version 11.1 (StataCorp, Texas, USA). Comparisons of the effect of binary exposures on binary outcomes (including gender, chemoprophylaxis use and presence of gametocytes) were performed using the Pearson χ^2 test. Comparisons of non-normally distributed numerical variables (including time to diagnosis, duration of inpatient stay) were performed using the Mann-Whitney U-test (Wilcoxon rank-sum).

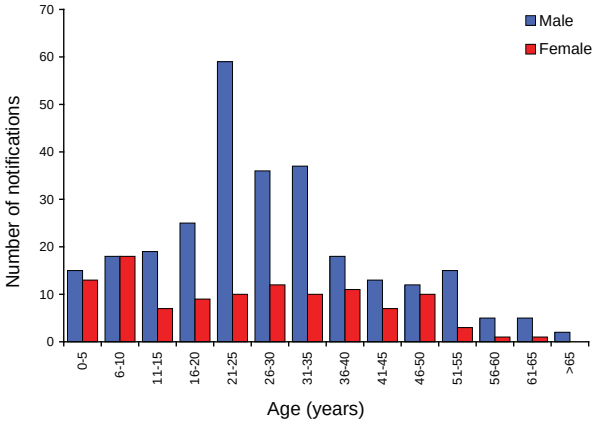
Where two countries are considered possible for acquisition, the most likely was judged at the time of interview. The recorded, but less likely secondary area of acquisition, was excluded from analysis in these cases. Summary demographic statistics were calculated on data from the primary infection or first relapse notified for each individual during the study period. Episodes of infection were judged to be relapses if they had previously been diagnosed with *P. vivax* or *P. ovale* and had not travelled again to a malaria endemic region. When the date of diagnosis was greater than 30 days after the reported date of entry into Australia, original case notes held by the CDC were reviewed to support the documentation of relapsed infection.

Results

Demographics

There were 428 episodes of malaria notified during the study period of which 425 were microscopy confirmed cases and three were probable cases. Of these 428 episodes, 382 were primary infections and 46 were relapse infections. The mean number of primary infections notified per year was 34.7 with a range from 13 to 65. No cases were acquired locally. The 428 episodes occurred in 391 individuals, of whom 279 (71.4%) were males and 112 (28.6%) were females ($P < 0.0001$). The median age was 26 years for males (range 0–80 years) and 25 years for females (range 0–65 years) (Figure 2). Three individuals (0.8%) identified as Indigenous. Over $^2/_5$ of cases were Australian nationals and 24.5% were African (Table 1).

Figure 2: Malaria cases, Northern Territory, January 2000 to December 2010 (n = 391), by age and sex



Classification of species

Primary malaria infections consisted of 196 cases (51.3%) of *P. falciparum,* 165 (43.2%) of *P. vivax,* 2 (0.5%) of *P. ovale* and 1 (0.3%) of *P. malariae.* There were 18 cases (4.7%) of mixed infection proven on microscopy; all were *P. falciparum* co-infection with either *P. vivax* (15 cases) or *P. malariae* (3 cases) (Table 1). There was marked temporal variation in species notified (Figure 3).

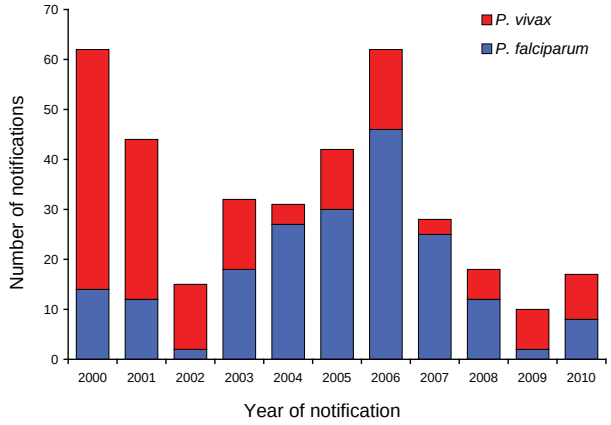
Region of acquisition for primary infections

Throughout the study period *P. falciparum* was the dominant species causing infection in individuals having travelled in, or arrived from Africa (92.0%) and Indonesia (53.8%), while *P. vivax* was the dominant infection in patients travelling to East Timor (68.0%) and Papua New Guinea (PNG) (66.2%). Since 2005 however, *P. falciparum* has been the dominant infection diagnosed in individuals arriving from East Timor (76.9%), leaving only the Pacific Islands and PNG where *P. vivax* continues to dominate in returned travellers.

Table 1: Characteristics of individuals diagnosed with malaria, Northern Territory, 2000 to 2010

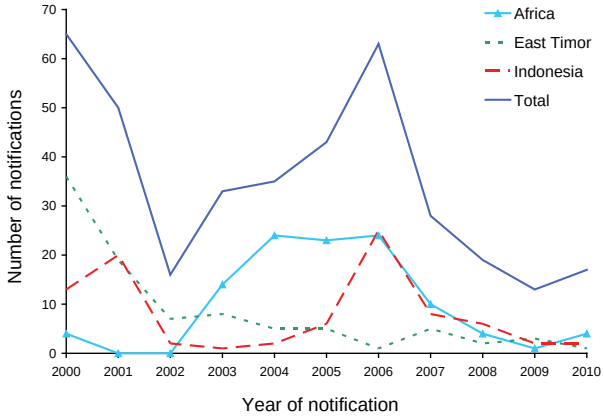
Gender (n=391)	**n**	**%**
Male	279	71.4
Female	112	28.6
Indigenous status (n=390)		
Indigenous	3	0.8
Non-Indigenous	387	99.2
Nationality by region (n=388)		
Australian	157	40.5
African	95	24.5
Indonesian	49	12.6
European	25	6.4
Papua New Guinean	17	4.4
New Zealand	7	1.8
East Timorese	6	1.5
Other	32	8.2
Civilian vs military (n=373)		
Civilian	306	82.0
Military (n=67)		
Australian	49	13.1
International	18	4.8
Region of acquisition *primary* infection (n=382)		
Africa	108	28.3
East Timor	92	24.1
Indonesia	87	22.8
Papua New Guinea	74	19.4
Pacific Islands (other than Papua New Guinea)	8	2.1
Other	13	3.4
***Plasmodium* species in *primary* infection (n=382)**		
P. falciparum	196	51.3
P. vivax	165	43.2
P. ovale	2	0.5
P. malariae	1	0.3
Mixed	18	4.7
Region of acquisition for *relapsed* infection (n=37)		
Africa	2	5.4
East Timor	10	27.0
Indonesia	5	13.5
Papua New Guinea	16	43.2
Other	4	10.8
***Plasmodium* species in *relapsed* infection (n=37)**		
P. vivax	36	97.3
P. ovale	1	2.7
Diagnosed by screening (n=104)		
Refugee arrival	71	68.3
Co-traveller of malaria case	18	17.3
Apprehended persons in Australian water	6	5.8
Students arriving from endemic country	3	2.9
Other	6	5.8

Figure 3: Primary *Plasmodium vivax* and *Plasmodium falciparum* infections,* Northern Territory, 2000 to 2010



* Other malaria species and mixed infections were excluded.

Figure 4: Primary malaria infections* acquired in Africa, East Timor, Indonesia and total of all regions notified in the Northern Territory, 2000 to 2010



* Includes all species and mixed infections.

Of the 187 individuals (including 150 Australian nationals) who reported a nationality from non-malarious regions, 116 (62.0%) were diagnosed with *P. vivax,* 60 (32.1%) with *P. falciparum* and 11 (5.9%) with mixed *P. falciparum/P. vivax* infection. This group acquired infection in East Timor (40.6%), PNG (27.8%), Indonesia (18.7%), Africa (6.4%), South East Asia (2.1%), Pacific Islands (2.7%) and Central and South America (1.1%). For the 191 individuals who were nationals of countries with endemic malaria, 135 (70.7%) were diagnosed with *P. falciparum,* 46 (24.1%) with *P. vivax,* 7 (3.7%) with mixed infection and 3 (1.6%) with *P. ovale* or *P. malariae* infection.

Notification rates by country of acquisition varied considerably during the study period. East Timor contributed the most cases in 2000 and 2001, Africa dominated in the years 2003 to 2007 and Indonesia contributed a significant number of cases in 2000, 2001 and 2006 (Figure 4).

Chemoprophylaxis

Chemoprophylaxis was prescribed in 145 cases (38.0%) of primary infection. Compliance data were available for 120 of these cases with 71 (59.2%) reporting completion of all doses as per accepted chemoprophylaxis guidelines. Ninety-two of 150 (61.3%) Australian nationals with primary malaria were prescribed chemoprophylaxis. The most commonly prescribed medication was doxycycline (57.2%). Of the 51 individuals who acquired malaria despite reporting compliance with doxycycline, 43 developed *P. vivax* malaria and 8 developed *P. falciparum* malaria. A further 18 cases reported full compliance to chemoprophylaxis agents other than doxycycline, of these, 11 developed *P. vivax* and 7 developed *P. falciparum* infection. Military personnel were more likely to have been prescribed chemoprophylaxis compared with civilians (51/64 vs 94/300, $P < 0.001$). When prescribed prophylaxis, military personnel were more likely to report full compliance compared to civilians (33/45 vs 38/75, $P = 0.014$). Civilians from non-malarious countries were more likely to utilise prophylaxis compared with individuals from malarious countries (105/187 vs 29/191, $P < 0.001$).

Presentation and screening

The date of arrival in Australia was recorded in 368 of the 382 primary malaria infections. The use of prophylaxis had a marked effect on the delay to the date of the first diagnostic test. In the case of *P. vivax* the median time from arrival to diagnosis was 9 days (IQR 4 to 23) in the absence of prophylaxis compared with 32 days (IQR 10 to 95) when prophylaxis of any type was prescribed ($P < 0.0001$). Similarly, *P. falciparum* cases presented after a median of 4 days (IQR 2 to 10) in the absence of prophylaxis compared with 6 days (IQR 3 to 21) when prophylaxis use was reported ($P < 0.0001$) (Table 2). The latest primary presentation of *P. falciparum* was 92 days after arrival in Australia in an African refugee migrating to Australia, who did not report using prophylaxis. The latest presentation of *P. vivax* was 333 days after arrival in Australia, in a member of the military who reported compliance with prophylaxis while deployed in East Timor.

Malaria was diagnosed by screening procedures in 104 of the primary infection presentations (27.2%). Reasons for screening included recently arrived refugee status (71 cases), being identified as a co-traveller of an individual diagnosed with malaria (18 cases), individuals apprehended for illegally fishing or arriving in Australian waters (6 cases) and

Table 2: Showing median days (interquartile range) from arrival into Australia until diagnosis of malaria for all primary malaria infections (n = 347)

	All patients	No prophylaxis	Prophylaxis prescribed
P. vivax	19 days (7–69)	9 days (4–23)	32 days (10–95)
P. falciparum	4 days (2–11)	4 days (2–10)	6 days (3–21)

The use of prophylaxis significantly delayed the diagnosis of *P. vivax* and *P. falciparum*.

students arriving or returning from malarious countries (3 cases) in schools participating in screening programs (Table 1).

The *Plasmodium* gametocyte is the life cycle stage that infects the feeding mosquito vector. Microscopy results reporting the presence or absence of gametocytes on blood films were available in 423 of the 428 episodes of malaria. Gametocytes were less likely to be identified on the films of patients infected with *P. falciparum* compared with *P. vivax* (69/195 vs 110/206, $P = 0.003$).

Hospitalisation, treatment and outcomes

Hospitalisation data were available for 395 of the 428 episodes of malaria with 344 (87.1%) individuals admitted to hospital at the time of their malaria diagnosis. The proportion of patients admitted to hospital varied with year (Figure 5). Infection with *P. falciparum* was associated with overall longer hospital admission with 43 of 183 patients with *P. falciparum* admitted for 5 days or greater compared with 15 of 174 *P. vivax* infected patients ($P < 0.001$). Despite this significant difference the median duration of hospital admission was 3 days, irrespective of the infecting species.

Treatment data were available for 413 of the 428 episodes of malaria. Prior to 2006, artemisinin combination therapy was used to treat 11.8% of cases, chloroquine 57.9%, quinine 15.5% and mefloquine 8.5%. Since 2006, artemisinin combination therapy was used in 77.5% of cases, chloroquine 12.0%, quinine 0.7% and mefloquine 1.4% (Figure 6).

There was a single death attributed to malaria in the Northern Territory during the study period. This occurred in a 32-year-old Malaysian resident evacuated from East Timor in May 2000 with mixed *P. falciparum* and *P. vivax* infection.

Figure 5: Proportion of patients diagnosed with malaria admitted to hospital, Northern Territory, 2000 to 2010

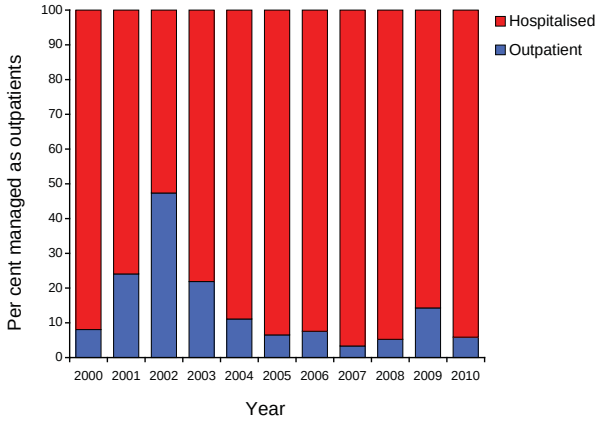
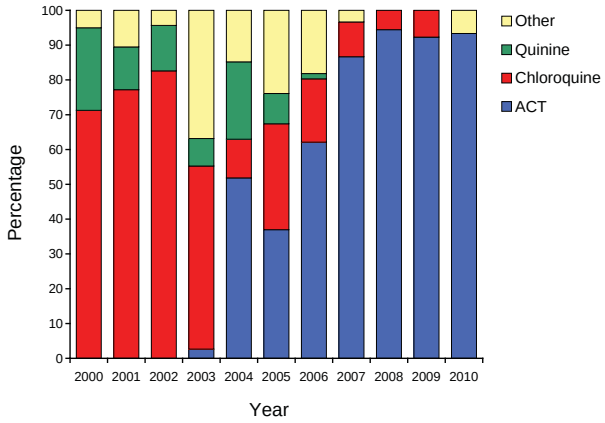


Figure 6: Proportion of malaria cases treated with different antimalarial medication or different antimalarials, Northern Territory, by year



Relapsed infections

There were 46 episodes of malaria identified as relapsed infection in 37 individuals. Microscopy confirmed 45 episodes due to *P. vivax* and 1 episode as *P. ovale*. A single episode of relapse occurred in 31 individuals, 2 relapse episodes occurred in 4 individuals, 3 relapse episodes occurred in 1 individual and 4 episodes in another individual. This group of 37 individuals were compared with the 167 who presented with primary *P. vivax* or *P. ovale* infection. There was no significant difference between the groups (Table 1). Assuming there is a steady state of relapsing individuals moving in and out of the Northern Territory, the approximate proportion of *P. vivax* relapse is 17.9% of primary infections. A 14 day course of primaquine was prescribed for 29 individuals following diagnosis with

relapsed disease, 6 individuals were not prescribed primaquine and the use was unknown in the 2 other cases. Dosage of primaquine and treatment history on cases prior to relapse is not recorded in the NTNDS.

A further 12 patients with previous malaria where categorised as primary infection (presumed reinfection) having travelled back to a malarious area between episodes.

Discussion

This study describes important epidemiological features of 428 consecutively notified cases of malaria in the Northern Territory of Australia. This series includes both hospital and community managed malaria cases and because of legislative requirements requiring laboratories and clinicians to notify the CDC it is unlikely cases have been missed. The surveillance data were collected at the time of diagnosis by chart review and interview of individuals, thus reducing the recall bias of retrospective analysis. Nevertheless, the authors needed to consult original case notes for a small number of incomplete data fields. In particular, case notes were necessary to classify infections as relapsed or reinfection as the NTNDS fails to make a distinction between these cases.

This analysis reveals that the origin of malaria infections in the Northern Territory varies over time and reflects the movement of people from endemic countries to Australia, as shown in the African refugee arrivals from 2005 to 2008 and large scale military and civilian activities following the political transition in East Timor in 2000 and 2001. These same factors have likely contributed to the increased proportion of *P. falciparum* diagnosed in the study period, not just in the Northern Territory but Australia-wide.[12,13] In 2009 and 2010, *P. vivax* was again the predominant species notified in the Northern Territory (Figure 3) as well as nationally, possibly reflecting shifts in refugee populations and implementation of pre-departure screening programs.[14,15]

It is not possible to draw conclusions from this study as to the effectiveness of chemoprophylaxis as the number of persons at risk during the observation period is not known. A number of individuals who reported compliance with appropriate chemoprophylaxis still acquired malaria infection with both *P. vivax* and *P. falciparum,* emphasising that mosquito avoidance and protection is also required.

Males outnumber female malaria cases over 10 years of age, but particularly in those aged between 11 and 35 years. While this may reflect the larger number in this demographic exposed, clinicians should nevertheless target this group with chemoprophylaxis and mosquito avoidance and protection. Current guidelines for prescribing prophylaxis can be found in the *Australian Therapeutic Guidelines,* Version 14.[16]

This study highlights the importance of the CDC policy of screening high risk groups for malaria with 27.2% of all diagnoses arising from screening procedures.[17] Prior to the introduction of refugee predeparture screening and treatment, the incidence of malaria in recently arrived refugees from Africa was reported between 5%–16%.[18] Pre-departure screening programs promoted through the Department of Immigration and Citizenship (DIAC) have reduced malaria diagnosis in refugees, but some still arrive in Australia with malaria[15] and may present later, presumably because of partial immunity or relapse of subclinical disease. The utilisation of screening in the Northern Territory may explain the shorter period from arrival in Australia to diagnosis of malaria when compared with other Australian and international series.[19,20]

There are minimal available data to compare hospitalisation rates for patients with malaria in other jurisdictions. The seemingly high rate of hospitalisation for patients diagnosed with malaria in the Northern Territory may reflect local treatment guidelines, which aim to reduce the risk of transmission to local mosquitoes and the re-establishment of malaria. Admission is recommended where species identification cannot be made within 24 hours, for all *P. falciparum* disease, if gametocytes are seen in the blood film or screened accommodation is not available.[17] The temporal variation in the hospitalised proportion of malaria infected individuals may reflect the increased number of *P. falciparum* infections between 2003–2008. The observation that hospital duration was longer in patients with *P. falciparum* has been described in other jurisdictions in Australia and is in keeping with the well described natural history of this more pathogenic species.[12,19]

Increasing resistance of *P. vivax* malaria to chloroquine in Indonesia, East Timor and Pacific Island nations has led to local Northern Territory and national guidelines recommending the use of artemisinin-based combination therapy (ACT) or mefloquine, for *P. vivax* infection acquired in these regions.[16,17] This analysis shows there has been a clear shift to using ACT and provides evidence that Northern Territory clinicians are implementing these guidelines (Figure 6).

P. vivax and *P. ovale* are characterised by relapsing infection, which arise from the hypnozoite stage of the life cycle within human hepatocytes. The proportion of infections that relapse is variably reported and is probably a function of the malaria

"strain" as well as sporozoite inoculums and host immunity.[21] The proportion of relapses reported in this study population can only be seen as a snapshot as the diagnosis of primary infections in some cases occurred outside the Northern Territory and also it is possible that some individuals may have relapsed after moving out of the Northern Territory jurisdiction. It is also possible that a proportion of these cases classified as relapse were recrudescent infections.

In conclusion, the use of enhanced surveillance data in the Northern Territory has allowed clinicians and health officials to better understand who is at risk of malaria. This analysis provides compelling support for the Northern Territory active screening program and shows that there are significant temporal shifts in malaria rates and species reflecting movement of people from endemic countries into Australia, influenced by military and civilian activities and changing refugee settlement programs. There is recognition that a close working relationship with DIAC and the Department of Defence would benefit diagnostic possibilities and prevention strategies. Enhanced malaria surveillance to some extent is already operational in Victoria,[22] Western Australia,[12] New South Wales and the Australian Capital Territory.[23] Consideration should be given to the gaps in our present knowledge and what might be gained from timely national enhanced data collection and analysis.

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Surveillance summaries

Supplementary report: surveillance of adverse events following immunisation among children aged less than seven years in Australia, 1 January to 30 June 2011

Deepika Mahajan, Jane Cook, Peter McIntyre, Kristine Macartney, Rob Menzies

Introduction

This report summarises national passive surveillance data reported to the Therapeutic Goods Administration (TGA) to 31 August 2011 for adverse events following immunisation (AEFI) reported for children aged less than 7 years who received vaccines between 1 January and 30 June 2011. The report includes all vaccines administered to children in this age group with a focus on the vaccines included in the funded National Immunisation Program (NIP) schedule.[1]

At the time of this report, the most recent change to the NIP schedule occurred in 2010 when annual seasonal trivalent influenza vaccine (TIV with 3 strains: A/H1N1, A/H3N2 and B) was funded for people aged ≥6 months with medical risk factors (previously subsidised through the Pharmaceutical Benefits Scheme).[2] A number of other important changes to vaccine funding and availability also occurred in 2009. From October 2009, the Northern Territory started using a new 10-valent pneumococcal conjugate vaccine (Synflorix®) at 2, 4, 6 and 12 months of age instead of the 3-dose 7-valent pneumococcal conjugate vaccine (Prevenar®) and a 23-valent pneumococcal polysaccharide booster for Indigenous children at 18 months of age. By late 2009, all states and territories were using the hexavalent DTPa-IPV-Hib-HepB (Infanrix hexa®) vaccine for all children at 2, 4 and 6 months of age,[3–5] due to an international shortage of PedvaxHib® (monovalent) and Comvax® (Hib-HepB) *Haemophilus influenzae* type b (Hib) vaccines.[6] To assist readers a glossary of the abbreviations of the vaccines referred to in this report is at the end of this report.

Methods

Case definition and coding

The data reported here are provisional only. It is important to note that an AEFI is defined as a medical event that is temporally, but not necessarily causally, associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system,[7] methods used to analyse the data and information regarding limitations and interpretation of the data.[7–11] Often, several vaccines and reaction codes are listed in an AEFI record so the number of vaccines and reaction codes will exceed the total number of AEFI records. For the purpose of this report, an AEFI is defined as 'serious' if it is life-threatening, had recovery with sequelae, or was associated with admission to hospital, prolongation of hospitalisation, or death. In addition to the standard presentation of numbers and rates, in this report comparisons with previous years' data were made by whether reports included co-administration of influenza vaccines. This was done in order to facilitate comparisons with 2010 where there were a large number of AEFI reports for seasonal and pandemic influenza vaccines.

Denominator calculations from Australian Childhood Immunisation Register

Average annual population-based AEFI reporting rates were calculated using mid-2010 population estimates. Reporting rates per 100,000 doses were calculated for 10 vaccines on the NIP schedule for which reliable dosing data were available from the Australian Childhood Immunisation Register (ACIR), for children aged from birth to < 7 years.

Results

There was a total of 490 AEFI records (annualised reporting rate of 50.0 per 100,000 population) for vaccines administered to children aged < 7 years in the first 6 months of 2011. This was a 78% decrease on the 2,225 records (227.1 per 100,000 population) for the corresponding period in 2010. Forty-four (9%) were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event or death). A total of 86 (18%) AEFI records were assigned a causality rating of 'certain' (n = 78, 16%) or 'probable' (n = 8, 2%). Thirty-four per cent (n = 167) of the 490 AEFI records for the 2011 reporting period were for children aged < 1 year; 13% (n = 66) for those aged 1 to < 2 years; and 52%

(n = 257) were for the 2 to < 7 year age group. The male to female ratio was 1.2: 1, which was similar to previous years.[8,9]

Eighty-seven per cent of AEFI (n = 427) were reported to TGA via states and territories and the remainder were reported direct to TGA: 9% (n = 46) by doctors or health care providers; 2% (n = 8) by hospitals; 1% (n = 4) by pharmaceutical companies; and 1% (n = 5) by members of the public. This is a sharp contrast to the same period in 2010 where 17% of cases were reported to TGA directly by members of the public, mainly because of the active promotion of the reporting of AEFI following the monovalent pandemic H1N1 influenza (pH1N1) vaccine directly to TGA, as well as a high level of public interest in both the pH1N1 and seasonal TIV vaccines.

Sixty-three reports listed one or more vaccines for which accurate dose denominator data were not available from the ACIR. These were influenza (n = 47), 23-valent pneumococcal polysaccharide (n = 7), Bacille Calmette-Guérin (n = 4), hepatitis B (n = 3), and hepatitis A (n = 2) vaccines. AEFI reporting rates per 100,000 doses were calculated for the remainder of records (n = 427) (Table).

Table: Reporting rates of adverse events following immunisation per 100,000 vaccine doses,* children aged less than 7 years, January to June 2011*

	Jan–Jun 2011			Reporting rate per 100,000 doses†*					
	AEFI records‡		Vaccine doses§	Jan–June 2011		Jan–June 2010		Jan–June 2009	
	n	n*	n	Rate	Rate*	Rate	Rate*	Rate	Rate*
Vaccine (NIP vaccines)‖									
DTPa-containing vaccines	332	331	529,539	63	63	37	32	44	44
DTPa-IPV	185	185	142,367	130	130	78	58	82	80
Pentavalent (DTPa-IPV-HepB)	0	0	103	0	0	NA		47	47
Hexavalent (DTPa-IPV-HepB-Hib)	147	146	387,069	38	38	23	22	30	30
Haemophilus influenzae type b	33	33	134,462	25	25	50	21	19	18
Haemophilus influenzae type b-hepatitis B	0	0	197	0	0	185	185	112	112
Measles-mumps-rubella	151	150	279,883	54	54	53	26	39	37
Meningococcal C conjugate	34	34	140,947	24	24	43	18	20	20
Pneumococcal conjugate	139	137	380,482	37	36	22	21	31	31
Varicella	30	30	133,815	22	22	58	14	9	9
Rotavirus	127	127	315,270	40	40	26	25	39	39
Age group									
< 1 year	161	160	1,028,266	16.0	16.0	10.0	9.0	13.0	13.0
1 to < 2 years	60	59	503,873	12.0	12.0	28.0	9.0	8.1	7.9
2 to < 7 years	206	205	309,905	67.0	66.0	42.0	30.0	41.0	40.0
AEFI category‖									
Total	427	424	1,842,044	23.0	23.0	20.0	12.0	17.0	16.0
'Certain' or 'probable' causality rating	74	74	1,842,044	4.0	4.0	1.2	1.4	2.5	2.5
'Serious' outcome	38	38	1,842,044	2.1	2.1	2.2	1.3	2.0	2.0

Source: Therapeutic Goods Administration database.

* Excludes any reports where 2010 seasonal TIV or pH1N1 were co-administered with the National Immunisation Program vaccines.

† Number of adverse events following immunisation (AEFI) records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2011. More than one vaccine may be coded as 'suspected' if several were administered at the same time.

‡ Records where at least one of the 10 vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[7] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.

§ Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2011.

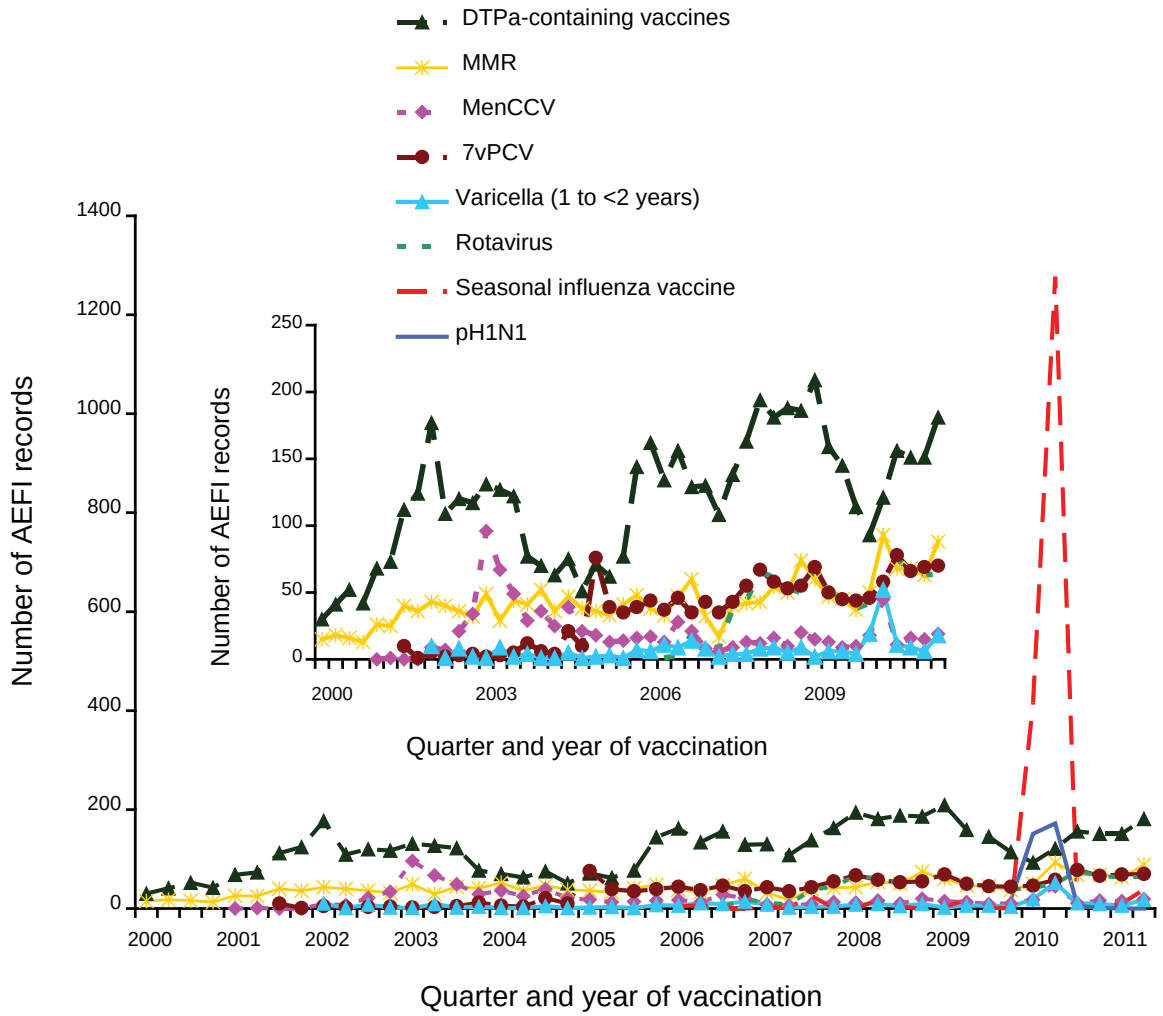
‖ The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the ACIR.

NA Very few pentavalent (DTPa-IPV-HepB) doses.

The overall AEFI rate for those reports, for which accurate dosage data were available, was 23.2 per 100,000 doses, with 2.1 per 100,000 classified as 'serious'. Excluding any reports where seasonal TIV or pH1N1 were co-administered with other childhood vaccines made little difference to the reporting rates for 2011 (23.0 and 2.1), but they were higher than the corresponding rates for 2010 of 12.3 and 1.3, respectively. Reporting rates (excluding reports with influenza vaccine co-administration) were higher in 2011 for all age groups and vaccine types. The largest percentage increase in the AEFI reporting rates was observed for children aged 2 to <7 years (120%), followed by children aged <1 years (72%) and 1 to <2 years (30%). Increases were observed in reporting rates excluding any influenza-containing vaccines, following receipt of measles-mumps-rubella (MMR) (106%), DTPa-containing vaccines (98%), 7vPCV (69%), varicella (63%), and rotavirus (58%) (Figure). No AEFI reports for pentavalent (DTPa-IPV-HepB) and Hib-HepB during the period January to June 2010 were received; there were very few doses administered for these latter 2 vaccines.

The most commonly reported reaction categories were injection site reaction (ISR) (n = 202; 41%), fever (n = 133; 27%), allergic reactions (n = 89; 18%), rash (n = 56; 11%), gastroenteritis following rotavirus vaccination (n = 42; 9%), screaming (n = 33; 7%) and seizure (n = 20; 4%). The largest number of reports were from Victoria (41%) followed by Queensland (19%), New South Wales (13%), South Australia (11%), and Western Australia (9%). There were relatively more reports of ISR in 2011 compared with 2010 (189 in 2011 compared with 85 in 2010). In 2010, 31% reports

Figure: Reports of adverse events following immunisation, 1 January 2002 to 30 June 2011, for vaccines recently introduced into the funded National Immunisation Program*



Inset excludes pH1N1 and seasonal influenza vaccine.

* Meningococcal C conjugate vaccine (MenCCV) was introduced into the National Immunisation Program schedule on 1 January 2003; 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005; DTPa-IPV and DTPa-IPV-HepB-Hib vaccines in November 2005; and Rotavirus (RotaTeq® and Rotarix®) vaccines 1 July 2007. In early 2008, Queensland, South Australia and Victoria changed from DTPa-IPV to DTPa-IPV-HepB-Hib for children at 2, 4 and 6 months of age.

were reported by Victoria followed by Queensland (28%), South Australia (13%) and New South Wales (10%). Compared with 2010, there was a substantial increase in reports of ISR in 2011 by all jurisdictions except the Australian Capital Territory and South Australia. The highest per cent increase was from Western Australia (200%), followed by Victoria and Queensland (180% each), the Northern Territory (170%), Tasmania (150%), and New South Wales (140%). A large number of ISR reports (n = 160; 85%) were from children aged 2 to < 7 years and 87% of these reports were associated with DTPa/IPV.

Nine per cent (n = 44) of the 490 AEFI records had outcomes defined as 'serious', however, there were no reports of life-threatening events, or deaths; all the children with AEFI defined as 'serious' were admitted to hospital. Thirty-four per cent (n = 15) of the 'serious' reports were following vaccination with hexavalent DTPa-IPV-HepB-Hib, 7vPCV, and rotavirus vaccines co-administered together. Serious and other significant AEFI included convulsions (n = 20 of which 6 were associated with hospitalisation), hypotonic-hyporesponsive episodes (HHE); (n = 19; 5 hospitalised), intussusception (n = 4; 3 hospitalised) and one case of idiopathic thrombocytopenic purpura (ITP). Of the 6 cases of seizure requiring hospitalisation, three were febrile convulsions (1 was following seasonal influenza vaccine while 2 others were following vaccination with varicella vaccine). There were a total of 14 reports of febrile convulsions; 43% were reported from Victoria. The most common individual vaccines in reports of convulsions were varicella (n = 5), seasonal influenza vaccine (n = 2), DTPa/HepB/IPV/Hib (n = 1), and DTPa/IPV (n = 1). The other reports of convulsions were following co-administration of Hib/MenC/MMR (n = 5), DTPa/HepB/IPV/Hib/7vPCV/rotavirus (n = 2), and one each of DTPa/HepB/IPV/Hib/7vPCV, 7vPCV/seasonal influenza, DTPa/IPV/MMR, and 23vPPV/HepA vaccines.

The majority of HHE (13/19) were notified by Victoria. Sixteen reports were following receipt of DTPa-containing vaccines, with hexavalent DTPa-IPV-HepB-Hib/ 7vPCV/rotavirus given conjointly in 15 reports and DTPa-IPV in one. Other vaccines in reports of HHE included Hib/MenC/MMR, Hib/MenC/varicella and rotavirus vaccine. Three of the 4 reports of intussusception in 2011 occurred following receipt of DTPa-IPV-HepB-Hib/7vPCV/rotavirus administered together and 1 report was following varicella vaccine. The only case of ITP was an infant following administration of Hib/MenC/MMR vaccine 18 days post-vaccination. There was no known medical history and the child fully recovered.

Discussion

There was a substantial decrease in the total number of AEFI records (78%) and population-based reporting rates (4.5 times lower) for the first 6 months of 2011 compared with the corresponding period in 2010. This appears to have been due to the drop in AEFI reporting following vaccination with seasonal TIV and pH1N1 influenza vaccines. The high number of reports associated with seasonal TIV and pH1N1 influenza vaccines in 2010 has been described previously.[8] High rates of fever and febrile convulsions were reported in association with one brand of the 2010 seasonal influenza vaccine used in children; this vaccine was withdrawn from use in young children from 2010.[12] The higher overall numbers of reports in 2011 (for non- influenza vaccines) is suggestive of generally increased propensity to report by providers in 2011, and may also reflect changes in the proportion of reports that were sent to TGA from individual state or territory surveillance systems. For example, in 2011, Victoria changed to submitting all reports to TGA, irrespective of severity, whereas previously minor/expected AEFI reports had not been submitted (personal communication: Dr Nigel Crawford, SAEFVIC, Victoria).

By age group, reporting rates per 100,000 doses, excluding vaccines co-administered with influenza, were higher in the first half of 2011 for all age groups, but more so in children aged 2 to < 7 years (66 vs 30) compared with children aged < 1 year (15.6 vs 9.0) and 1 to < 2 years (11.7 vs 9.0). The increase in reporting of AEFI in children aged 2 to < 7 years in 2011 was primarily because of increased reporting of ISR following vaccination with DTPa-IPV. The increase was largely seen from Victoria followed by Queensland and New South Wales.

The reporting rate of ISR in children aged 2 to < 7 years has declined in recent years, as was expected following the removal of the dose of DTPa-IPV due at 18 months of age from the NIP schedule in September 2003.[10] The reasons for the increase in 2011 are not entirely clear but at least partly due to general changes in AEFI surveillance stated above. One additional suggested hypothesis is that some ISR's are 'Arthus reactions' caused by the presence of high levels of prevaccination IgG antibody in the vaccinees, which have been associated with higher rates of ISR.[13,14] Possible causes of higher pre-vaccination antibody levels include immunity induced from natural infection in the pertussis epidemic from 2008, which was notable for high notification rates in pre-school aged children,[15] as well as the earlier age of administration of the pre-school DTPa-IPV booster since the change of eligibility rules for provider and parent incentive payments.[16]

Conclusion

The total number of AEFI reported in children aged < 7 years in the first half of 2011 was reduced by 78% compared with the same period in 2010 when a large number of reports were submitted in association with influenza vaccines. However, reporting rates for other vaccines were higher in all age groups in 2011, after excluding vaccines co-administered with influenza. This may reflect a greater propensity by vaccine providers to report in 2011 as well as changes in surveillance and reporting procedures at health departments at the jurisdictional level to report all minor events to TGA. This increase was greater in the 2 to < 7 year age group, particularly for ISR following receipt of DTPa-IPV. If a real increase in ISR incidence has occurred, one possible explanation is higher pre-vaccination antibody levels, due to the recent pertussis epidemic and possibly also earlier receipt of the pre-school booster.

The majority of AEFIs reported to the TGA were mild transient events and the data reported here are consistent with an overall high level of safety for vaccines included in the NIP schedule.

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Abbreviations of vaccine types

7vPCV	7-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
BCG	Bacille Calmette-Guérin (i.e. tuberculosis)
DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
dTpa	diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation
DTPa-IPV	combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus (quadrivalent)
DTPa-IPV-HepB	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus and hepatitis B (pentavalent)
DTPa-IPV-HepB-Hib	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus, hepatitis B and Haemophilus influenzae type b vaccine (hexavalent)
HepA	hepatitis A
HepB	hepatitis B
Hib	*Haemophilus influenzae* type b
IPV	inactivated poliovirus vaccine
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
pH1N1	pandemic H1N1 influenza 2009
TIV	seasonal trivalent influenza vaccine (with 3 strains: A/H1N1, A/H3N2 and B)

Surveillance systems reported in CDI, 2012

This article describes the surveillance schemes that are routinely reported on in *Communicable Diseases Intelligence* (CDI).

Communicable disease surveillance in Australia operates at the national, state and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

- detecting outbreaks and 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; and
- support for quarantine activities, which are the responsibility of the national government.

State and territory health departments collect notifications of communicable diseases under their public health legislation. In September 2007, the *National Health Security Act 2007* (*National Health Security Act*, No 174) 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* (NNDL), which specifies the diseases about which personal information can be provided. The *National Health Security Agreement,* which was drafted in 2007 and signed by Health Ministers in April 2008, establishes operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. States and territories voluntarily forward de-identified data on a nationally agreed set of communicable diseases to the Department of Health and Ageing for the purposes of national communicable disease surveillance.

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control.' It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.'[1] Although some surveillance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

The major features of the surveillance schemes for which CDI publishes regular reports are described below.

Other surveillance schemes for which CDI publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2008;32:1–11), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2008;32:12–17), invasive pneumococcal disease surveillance (*Commun Dis Intell* 2008;32:18–30), the National Arbovirus and Malaria Advisory Committee (*Commun Dis Intell* 2008;32:31–47), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2008;32:425–429).

Australian Childhood Immunisation Register

Accurate information on the immunisation status of children is needed at the community level for program management and targeted immunisation efforts. A population-based immunisation register can provide this need. The Australian Childhood Immunisation Register (ACIR) commenced operation on 1 January 1996 and is now an important component of the *Immunise Australia Program*. It is administered and operated by Medicare Australia. The Register was established by transferring data on all children under the age of 7 years enrolled with Medicare to the ACIR. This constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age. Children who are not enrolled in Medicare are added to the Register when a recognised immunisation provider supplies details of an eligible immunisation. Immunisations are generally notified to Medicare Australia either by electronic means, the Internet or by paper ACIR notification forms. Immunisations recorded on the Register must have been given in accordance with the guidelines for immunisation determined by the National Health and Medical Research Council.

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method described in *Commun Dis Intell* 1998;22:36–37. With this method, a cohort of children is defined by date of birth in 3-month groups. This birth cohort has the immunisation status of its members assessed at the 3 key milestones of 12 months, 24 months and 5 years of age. Analysis of coverage is undertaken 3 months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included in order to minimise inaccuracies in coverage estimates due to duplicate records.

Medicare Australia coverage reports for the 3 milestones are published in CDI each quarter. Coverage estimates are provided for each state and territory and Australia as a whole and for each individual vaccine assessed at each milestone. Changes in 'fully immunised' coverage from the previous quarter are also included in the tables.

A commentary on ACIR immunisation coverage estimates is included with the tables in each issue and graphs are used to provide trends in immunisation coverage.

An Immunisation Coverage Report is also published in CDI on an annual basis and provides more detailed data on immunisation coverage for all recommended vaccines by age group which are funded by the Immunise Australia Program, timeliness of immunisation, small area coverage estimates and data on conscientious objection to immunisation.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in CDI (*Commun Dis Intell* 2008;32:227–231). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. One main purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of MIC testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data of laboratory-confirmed cases confirmed either by culture or by non-culture techniques. Culture-positive cases where a *Neisseria meningitidis* is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions.

Data are reported annually and quarterly in CDI. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2009;33(1):1–9).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is an active surveillance mechanism for prospective, national identification and study of children (< 15 years) with uncommon conditions of childhood, including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, and rare injuries. Each month the APSU sends an e-mail or paper report card to approximately 1,360 paediatricians and other child health clinicians. Clinicians are asked to indicate whether or not they have seen a child newly diagnosed with any of the listed conditions listed. Clinicians reporting cases are asked to provide details about demographics, diagnosis, treatments and short-term outcomes. All negative and positive reports are logged into a database and the report card return rate has been maintained at over 90% over the last 19 years.

Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV and HIV infection, neonatal herpes simplex virus infection; neonatal

varicella, congenital varicella, severe complications of varicella, and juvenile onset recurrent respiratory papillomatosis.

After demonstrating feasibility in 2007, APSU continues to conduct surveillance for severe complications of influenza during the influenza season each year. In 2009 APSU contributed to the national surveillance effort during the Influenza H1N1 09 pandemic.

The activities of the APSU are funded in part by the Australian Government Department of Health and Ageing, NHMRC Practitioner Fellowship No: 1021480 (E Elliott). The Faculty of Medicine, The University of Sydney, and the Royal Australasian College of Physicians, Division of Paediatrics and Child Health, and the Kids Research Institute, Sydney Children's Hospitals Network provide in-kind support.

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Australian National Creutzfeldt-Jakob Disease Registry

The surveillance for CJD in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD has been scheduled as a notifiable disease in all Australian states and territories. The ANCJDR is under contract to the Commonwealth to identify and investigate all suspect cases of transmissible spongiform encephalopathies (TSE) in Australia. An annual update is published in CDI (*Commun Dis Intell* 2009;33(2):188–191).

Australian Sentinel Practice Research Network

The Royal Australian College of General Practitioners and the Department of General Practice at the University of Adelaide operate the Australian Sentinel Practices Research Network (ASPREN). ASPREN is a national network of general practitioners who report presentations of defined medical conditions each week. The main aims of ASPREN are to provide an indicator of the burden of disease in the primary health care setting and to act as an early warning indicator in the event of an influenza pandemic.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2012, 4 conditions are being monitored; all of which are related to communicable diseases. These include influenza like illness, gastroenteritis, chickenpox and shingles.

Laboratory testing of ILI cases was implemented in 2010, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and H1N1(2009).

There are currently 170 general practitioners registered the network from all jurisdictions. Sixty-one per cent of these are in metropolitan areas, 29% in rural and 10% in remote areas of Australia. Approximately 9,000 consultations are recorded each week.

Data for communicable diseases are published in CDI every quarter. Data are presented in graphic format as the rate of reporting per 1,000 consultations per week. The conditions are defined as follows:

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue

Gastroenteritis – record once only per patient

Three or more loose stools, and/or 2 vomits in a 24 hour period excluding cases who have a known cause, for example bowel disease, alcohol, pregnancy.

Chickenpox – record once only per patient

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for three to 4 days and leaves a granular scab.

Shingles – record once only per patient

Recurrence, recrudescence or re-activation of chickenpox infection. Vesicles with any erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Note: Those conditions which show 'record once only per patient' are to have each occurrence of the condition only recorded on 1 occasion no matter how many patient contacts are made for this condition. If the condition occurs a second or subsequent time, it is to be recorded again. Conversely, for other conditions each attendance at which they are addressed in some way is to be recorded.

HIV and AIDS surveillance

National surveillance for HIV and AIDS is coordinated by the Kirby Institute in collaboration with state and territory health authorities, the Australian Government Department of Health and Ageing, the Australian Institute of Health and Welfare and other collaborating networks in surveillance for HIV, viral hepatitis and sexually transmissible infections.

Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, either by the diagnosing laboratory (Australian Capital Territory and Tasmania), by doctor notification (Western Australia) or by a combination of laboratory and doctor sources (New South Wales, Northern Territory, Queensland, South Australia and Victoria). 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.

Currently, 2 tables presenting the number of new diagnoses of HIV infection, AIDS and deaths following AIDS are published in each issue of CDI. The tabulations are based on data available 3 months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

The document *viral hepatitis and sexually transmissible infections in Australia Annual Surveillance Report* has been published by the Kirby Institute from 1997. The Annual Surveillance Report, available through www.kirby.unsw.edu.au provides a comprehensive analysis and interpretation of surveillance data on HIV, viral hepatitis and sexually transmissible infections in Australia. The report *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander people: Surveillance and Evaluation Report* has been published from 2007, as an accompanying document to the *Annual Surveillance Report*. The *Surveillance and Evaluation Report* provides detailed analysis and interpretation of the occurrence of these infections in Aboriginal and Torres Strait Islander communities in Australia.

National Influenza Surveillance Scheme

Australian influenza activity and severity in the community are monitored using a number of indicators and surveillance schemes:

- Notifications of laboratory-confirmed influenza are reported from all Australian states and territories and reported in the National Notifiable Diseases Surveillance System.
- Community level influenza-like illness (ILI) are monitored through two sentinel systems, Flutracking, a weekly online survey integrating syndromic information with participant influenza immunity status; and data from the National Health Call Centre Network.
- Reports on general practice ILI consultations are provided through the Australian Sentinel Practice Research Network and the Victorian Sentinel General Practice Scheme. Additionally, data on ILI presentations to hospital emergency departments are collected from sentinel hospitals sites in Western Australia and New South Wales.
- Hospitalised cases of laboratory-confirmed influenza are reported through the Influenza Complications Alert Network (FluCAN); and severe complications in children are monitored by the Australian Paediatric Surveillance Unit.
- Information on influenza subtypes and positivity are provided from sentinel laboratories, including the National Influenza Centre laboratories and some state public health laboratories. Additional virology and antiviral resistance data are also provided from the World Health Organization Collaborating Centre for Reference and Research on Influenza.

During the influenza season, data from each of these surveillance systems are compiled and published fortnightly in the Australian Influenza Report, which is available generally from May to October on the department's web site. These reports include the above data as well as additional mortality and international surveillance data.

Annual reports on the National Influenza Surveillance Scheme are published in CDI each year (*Commun Dis Intell* 2010;34(1):8–22).

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[2] The National Notifiable Diseases Surveillance System (NNDSS) was established in 1990 under the auspices of the Communicable Diseases Network Australia (CDNA).

Sixty-five communicable diseases agreed upon nationally are reported to NNDSS, although not all 65 are notifiable in each jurisdiction. Data are sent electronically from states and territories daily or several times a week. The system is complemented by other surveillance systems, which provide information on various diseases, including four that are not reported to NNDSS (AIDS, HIV, and the classical and variant forms of Creutzfeldt-Jakob disease).

The NNDSS core dataset includes data fields for a unique record reference number; notifying state or territory; disease code; age; sex; Indigenous status; postcode of residence; date of onset of the disease; death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case is collected. Data quality is monitored by DoHA and the National Surveillance Committee (NSC) and there is a continual process of improving the national consistency of communicable disease surveillance.

While not included in the core national dataset, enhanced surveillance information for some diseases (hepatitis B [newly acquired], hepatitis C [newly acquired], invasive pneumococcal disease and tuberculosis) is obtained from states and territories.

Aggregated data are presented on the department's Internet site under *Communicable Diseases Surveillance* and updated daily (www.health.gov.au/nndssdata). A summary report and data table are also published on the Internet each fortnight (www.health.gov.au/cdnareport).

Data are published in CDI every quarter and in an annual report. The reports include numbers of notifications for each disease by state and territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous 5 years in the same period. A commentary on the notification data is included with the tables in each issue of CDI and graphs are used to illustrate important aspects of the data.

OzFoodNet: enhanced foodborne disease surveillance

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally in the investigation of foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease.

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in CDI (*Commun Dis Intell* 2009;33(4):389–413) since 2001. Data are reported from all Australian jurisdictions.

Sentinel Chicken Surveillance Programme

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVEV) and Kunjin viruses. MVEV causes the disease Murray Valley encephalitis (formerly known as Australian encephalitis), a potentially fatal disease in humans. Encephalitis is less frequent in cases of Kunjin virus infection and these encephalitis cases have a lower rate of severe sequelae.

These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne Murchison and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVEV is also responsible for occasional epidemics of encephalitis in eastern Australia. Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVEV activity. These programs are supported by individual state health departments. Each state has a contingency plan that will be implemented if one or more chickens in a flock seroconverts to MVEV.

Currently, flocks are maintained in the north of Western Australia, the Northern Territory, New South Wales and in Victoria. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales and Victoria are tested only in the summer months, during the main MVEV risk season. Results are posted on the National Arbovirus and Malaria Advisory Committee web site. A yearly summary is presented in CDI (*Commun Dis Intell* 2012;36(1):70–81).

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Guidelines

Position statement on interferon-γ release assays in the detection of latent tuberculosis infection

National Tuberculosis Advisory Committee

Summary

In vitro T-cell based interferon-*y* (IFN-*y*) release assays (IGRAs), the QuantiFERON-TB Gold In-Tube test (QFN-GIT) (Cellestis Limited, Carnegie, Victoria, Australia) and the T-SPOT. TB test (T-Spot) (Oxford Immunotec Limited, Abingdon, United Kingdom), are marketed as a substitute for the tuberculin skin test (TST). The specificity of these immunoassays has been optimised by using *Mycobacterium tuberculosis*-specific antigens. IGRAs are more specific in patients with previous Bacille Calmette-Guérin (BCG) immunisation or exposure to non-tuberculous mycobacteria (NTM).

There have been a plethora of comparative studies of TST and IGRAs, several meta-analyses in specific patient groups, and a few longitudinal studies of the predictive ability of IGRA-positive results for the development of active tuberculosis (TB) disease. A summary of these studies is that IGRAs have not been clearly demonstrated to be superior to TST. The National Tuberculosis Advisory Committee (NTAC) also notes a continuing absence of cost-effectiveness studies of IGRAs under Australasian TB program conditions. Furthermore, TST remains a familiar test with a long history of use and longitudinal data that provides important predictive information that is not yet available with IGRA.

TST therefore remains the preferred test for latent tuberculosis infection (LTBI) in most patient groups. IGRAs may be used as supplemental tests to improve specificity in screening immunocompetent subjects and in addition to TST in immunocompromised patients considered at high risk of LTBI. The specific recommendations in various patient groups are listed in the body of the text.

Background

Detection and treatment of LTBI is considered to be an increasingly important element of TB control efforts in Australia and other low-incidence countries. IGRAs are marketed as a substitute for the TST for the detection of LTBI.

NTAC has released position statements on the use of these assays (the last statement being in September 2009) and has undertaken to revise the recommendations on a regular basis. A MedLine search for 'interferon gamma release assay tuberculosis' articles in English between August 2009 and August 2011 found 197 new publications. To address this large body of literature, the Committee has followed a template recommended in a survey of international IGRA guidelines by Denkinger et al.[1] Each Committee member reviewed one of the following sub-sections. The Committee then discussed the member's proposed recommendation for each sub-section before reaching a consensus position.

Denkinger et al[1] suggested using an evidence-based grading system though the ability to grade the quality of research studies remains controversial.[2] The quality of the IGRA literature is disparate and some of the publications are not relevant to a high-income country such as Australia with a low incidence of TB. The Committee therefore has not formally graded the quality of the evidence but has cited meta-analyses where possible and has provided a few key references for each sub-section.

Summary of available commercial interferon-*y* release assays

Tuberculin (or purified protein derivative-PPD) has been used as an *in vivo* test for LTBI for over 50 years.[3] Tuberculin is injected intra-cutaneously on the volar aspect of the forearm; the diameter of induration is read 48 hours later. Disadvantages of the TST include that the patient must return to the clinic for the result to be read (leading to large drop-out rates) and that the TST lacks specificity because the tuberculin preparation contains antigens that cross-react with BCG and NTM.[3,4] However, TST's long history of use has provided valuable research data and experience, particularly longitudinal data that provide important predictive information that is not yet available with IGRAs.[3]

The United States' Food and Drug Administration have approved three *in vitro* IGRAs that attempt to address these disadvantages of the TST. The spe-

cificity of these immunoassays has been optimised by utilising pooled synthetic antigens, such as early secretory protein 6 [ESAT-6] and culture filtrate protein 10 [CFP-10], from the *M. tuberculosis*-specific region of difference 1 (RD1).[5,6] The assay formats have been summarised in the 2010 United States Centers for Disease Control and Prevention and the 2011 European Centre for Disease Control and Prevention guidelines.[7,8] The currently-available assay, the QFN-GIT, comprises three tubes: a test tube containing antigens from ESAT-6, CFP-10 and part of the sequence of TB7.7; a positive control tube (containing phytohaemagglutinin); and a negative control tube. The three tubes are inoculated with the patient's blood; incubated for 16–24 hours; the plasma is separated; and the IFN-γ concentration measured by an ELISA.

An alternate commercial assay, the T-Spot test, is available but has not been marketed widely in Australia. In the T-Spot test, peripheral blood mononuclear cells (PBMCs) are separated from whole blood and distributed to a microtitre plate (250,000 cells/well) containing test wells (ESAT-6 and CFP-10), and positive- and negative-control wells. Following 16–20 hours incubation, an enzyme-linked immunospot assay (ELISpot) is used to detect increases in the number of cells that secrete IFN-γ (represented as spots in each test well) after stimulation with/without antigen. The T-Spot test is technically demanding requiring PBMC separation and a subjective reading of the ELISpot assay by a technician. However, some studies suggest that the T-Spot test is more sensitive than the Quantiferon tests, particularly in immunocompromised individuals.

The antigens employed in both IGRA formats are absent from BCG and most NTM, but present in *M. marinum, M. kansasii,* and *M. szulgai*.[4] The antigens may also be present in other unrecognised un-sequenced NTM. A small potential for cross-reaction with NTM therefore remains even with the IGRAs.

Diagnosis of active tuberculosis in adults

The previous NTAC statement recommended that TST and IGRAs had no place in the initial investigation of active TB disease. There are limited new data that have bearing on the role of IGRAs in the diagnosis of active TB.

A meta-analysis of the role of IGRAs (i.e. the T-Spot and QFN-GIT assays) for diagnosing active TB disease found the pooled sensitivity of 69%–83% in HIV non-infected subjects and 60%–76% in HIV co-infected patients (i.e. equivalent to prior results for TST).[9] Also, like TST, IGRAs cannot distinguish between LTBI, active TB or past infection. Hence, specificity for active TB is low: 52%–61% in HIV non-infected and 50%–52% in HIV infected subjects. Anecdotal experience amongst TB physicians in Australia and limited published experience[10] suggest that IGRAs are over-used in acute clinical settings where the diagnosis of active TB is being considered.

Recommendation

TST and IGRAs have no place in the initial investigation of active TB disease.

IGRA (like TST) cannot and should not be used to exclude suspected TB disease in adults

Contact investigation in adults

Contact tracing and identification of LTBI following an exposure to active, infectious TB is an important component of TB control, particularly in low-TB incidence settings.[11] Various studies have provided different estimations for the progression rate to active disease two years after TST/IGRA conversion but the overall lifetime risk is generally described as 10%–15%. Treatment of LTBI with isoniazid reduces risk of future disease by 75%–90%.[12] Early identification of infected contacts and appropriate preventive treatment therefore has the potential to minimise future incident cases and ongoing transmission of infection. The limitation for effective contact investigation is the lack of a gold standard test that can identify LTBI, differentiate between active and latent infection, or predict patients at highest risk of progressing to active disease.

Both TST and IGRAs detect a cellular immune response to *M. tuberculosis* antigens as an imperfect surrogate marker for LTBI. There have been two recent meta-analyses comparing the ability of TST and IGRAs to predict progression to active TB disease in patients without active disease at baseline.[13,14] The analysis by Rangaka et al[14] included 15 studies from countries with a low- and high-incidence of TB. The association of a positive IGRA result with subsequent development of active TB disease was weak, with a relative risk of 2.1 (95% CI 1.42–3.08) and similar to a positive TST result, which had a relative risk of 1.60 (95% CI 0·94–2·72) at the 10 mm cut-off. Only four studies fulfilled the inclusion criteria in the meta-analysis by Diel et al.[13] two studies involved screening HIV patients, while the other two consisted of large contact investigations among local and immigrant populations in Germany and The Netherlands. In one contact investigation study, a positive IGRA result had a positive predictive value (PPV) for progression to active TB disease of 14.6% (95% CI 6–29%) compared with TST's PPV of 2.3% (95% CI 0.7–5.2%). The other contact investigation study found no dif-

ference between QFN-GIT, T-Spot or TST with the respective PPVs being: 2.8% (95% CI 0.9–6.4%), 3.3% (95% CI 1.2–7.6%) and 3.1 (1.4–5.8%).

IGRAs therefore have not been clearly demonstrated to be superior to TST for detection of LTBI in contact investigations. In the absence of a clear choice between IGRAs and TST for contact investigations, a number of different approaches have been suggested ranging from TST alone to IGRA as the sole test with a variety of intermediary recommendations. Many guidelines recommend a sequential approach with TST performed as the first test, followed by IGRA as a confirmatory test in the event of a positive TST test. This approach may limit the costs associated with follow up of false-positive TST and unnecessary treatment of LTBI. Logistics and patient preferences must also be considered.

Recommendation

TST remains the test of choice for investigation of contacts of active TB. TST has similar specificity to IGRAs in a non-BCG vaccinated cohort, therefore IGRAs do not add additional value in this group.

In TST-positive subjects at low risk of LTBI and at low risk of progressing to active disease, an IGRA may be used as a supplementary test in a two-step process to confirm LTBI. The improved specificity of IGRA in this circumstance in subjects who have had previous BCG or NTM exposure may allow better targeting of preventative therapy.

IGRAs may be a preferred option where resources, distance or other factors make TST impractical to administer.

Diagnosis of active tuberculosis in children

The 2007 NTAC statement made no specific recommendations regarding the use of IGRA in children. As of July 2011, over 30 guidelines (some including children) that incorporate IGRA in diagnostic algorithms for either LTBI or TB disease are available worldwide.[1]

In children with confirmed TB disease in low TB endemic settings, studies suggest a similar sensitivity of IGRA and TST of between 50% and 90%.[15,16] Therefore, IGRA and TST cannot and should not be used to exclude TB disease. Combining the results of IGRA and TST is associated with a small overall increase in sensitivity in several studies.[17] Given the difficulty of establishing an accurate diagnosis of TB disease in children, results of IGRA (and/or TST) may provide additional evidence of *M. tuberculosis* infection in a child with suspected TB disease. A positive IGRA or TST result does not, however, discriminate between TB disease and LTBI. Neither test should be used as a replacement for standard microbiological and radiological investigations.

Recommendation

IGRA (like TST) should only be used as an adjunctive test in addition to standard microbiological and radiological investigations.

IGRA (like TST) cannot and should not be used to exclude suspected TB disease in children.

Contact tracing in children

Given the absence of a recognised gold standard, estimating the 'true' sensitivity and specificity of IGRA or TST for the detection of LTBI in children is difficult. However, a recently published hierarchy of reference standards for the evaluation of IGRA for the detection of LTBI is informative.[18] Within this hierarchy, the weakest standard is concordance with the TST. Analysis of results of IGRA and TST with respect to defined exposure to *M. tuberculosis* is a better method to assess the accuracy of these tests. Although not all individuals exposed to a smear-positive TB contact will subsequently become infected, this has become an accepted quasi 'gold standard' on which to base comparative evaluations between TST and IGRA. The predictive ability of IGRA for the development of TB disease and the likely efficacy of preventive treatment based on the results of IGRA represent the highest quality standards but remain largely unstudied in children. A negative TST or IGRA does not exclude LTBI.

Almost 40 studies have compared the performance of IGRA with TST as a marker of LTBI in children. The design of most studies has been cross sectional, comparing results of IGRA with the TST in children screened for LTBI for a variety of indications. Results suggest that discordance between IGRA and TST results are common in children (most often TST positive/IGRA negative in the low TB endemic setting) and this may be due to false negative IGRA results. A high rate of indeterminate IGRA results has been reported in young children (< 5 years) in several recent studies.[19] Further, as with TST, the timing of the IGRA is likely to be important (e.g. may be false negative if the contact is less than 1 weeks ago). Therefore, a negative IGRA alone should not be used to exclude LTBI, especially in young children.

In settings with low rates of BCG immunisation such as Australia, IGRA add little over TST in the context of TB screening or contact investigation. In BCG immunised children (usually immigrants), IGRA may have an advantage as the TST can yield false positive results as a result of prior BCG

immunisation. A positive TST is highly specific for LTBI when the child received BCG immunisation as a neonate, which is usual practice. The majority of false positive TST results occur in children immunised after one year of age.[20,21,22]

Infectiousness of the source case and risk of disease in the child contact remain the most important factors in deciding the need for preventive therapy, irrespective of the IGRA or TST result.

Recommendation

IGRA does not replace TST for detection of LTBI in children and (like TST) cannot be used to exclude LTBI. IGRA may have additional value over TST in children that received BCG vaccination after the first year of life.

Screening of immigrants

The evolving epidemiology of TB in Australia is driven mostly by migration of individuals from countries with a high burden of disease. Following arrival in Australia, disease amongst immigrants occurs most commonly as a result of reactivation of latent TB. In 2008, overseas-born people contributed 86% of the total TB case-load. The TB incidence rate in the overseas-born population was 20.4 cases per 100,000 population. This rate is almost 19 times the incidence rate experienced in the Australian born population.[23]

Identification and treatment of people with LTBI to prevent disease is a key component of TB control within Australia. Post-arrival screening and treatment of LTBI in newly-arrived refugees has been shown to be a cost-effective measure, due to the prevention of TB transmission in the community and number of cases and deaths from TB averted.[24]

In 2011, the NICE Clinical Guideline on TB diagnosis and management addressed the issue of diagnosis of LTBI in people who are recent arrivals from countries of high TB prevalence.[25] The conclusion was that IGRAs in this group appeared to be the most cost-effective diagnostic strategy, however, a dual testing strategy utilising both tuberculin skin testing and supplemental IGRA assessment was recommended as TST was a less expensive strategy that would be more effective in low incidence areas and, in particular, there were still issues over the operation of the IGRA tests and inter-subject variability.[25]

Recommendation

TST and supplemental IGRA assessment for people identified with a positive TST is the recommended diagnostic strategy in immunocompetent immigrants from countries where TB is highly prevalent.

Immunocompromised individuals with HIV infection

The utility of IGRAs in HIV-infected individuals has not been clarified. Their use is potentially hampered by the relative or absolute anergy demonstrated by patients with CD4 cell counts < 200 cells per mm^3, although some studies have suggested that IGRAs (especially the T-Spot test) may be less affected by CD4 count than the TST. A recent meta-analysis failed to come to a conclusion regarding the superiority or inferiority of IGRA in comparison to TST.[26]

Furthermore it is unclear whether the QFN-GIT and T-Spot test are equivalent. In the context of diagnosis of active TB (using *M. tuberculosis* culture positivity as the gold standard), the above meta-analysis found the sensitivity of the T-Spot test was 72% (95% CI, 62–81%) and of QFN-GIT was 61% (95% CI, 47–75%) in low–middle-income countries, and 94% (95% CI, 73–100%) and 67% (95% CI, 47–83%) respectively in high-income countries.[26] Thus the sensitivity and specificity of IGRAs vs TST in HIV-infected individuals is unclear. Furthermore, none of these tests (including TST) can be considered definitive for proving or discounting latent or active TB in HIV-infected individuals.

Recommendation

TST remains the test of choice for detection of LTBI in HIV-infected individuals. However, recognising the lowered sensitivity of TST in immunocompromised patients, an IGRA may be used as a supplementary test. An HIV-infected individual would be diagnosed with LTBI if either the TST or IGRA is positive.

Immunocompromised individuals receiving anti-tumour necrosis factor-α therapy

Patients with immune-mediated inflammatory diseases (IMID)—such as rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, ulcerative colitis and Crohn's disease—are at increased risk of developing active TB disease due to their traditional immunosuppressive therapy (e.g. prednisolone) and particularly when receiving the newer immunomodulatory biological agents, such as tumour necrosis factor-α (TNF-α) inhibitors.[27] Jick et al[28] reported that 'low-dose' (< 15 mg per day) and 'high dose' (≥ 15 mg per day) prednisolone was associated with active tuberculosis with an odds ratios of 2.8 (95% CI 1.0–7.9) and 7.7 (95% CI 2.8–21.4), respectively. Five TNF-α inhibitors are available in Australia: infliximab, adalimumab, etanercept, certolizumab, golimumab. The TNF-α inhibitors have been associated with 4–20-fold increases in active TB disease with infliximab and adalimumab carrying a greater TB risk than etanercept.[27]

The 'standard of care' is therefore to screen for LTBI before beginning treatment with TNF- α inhibitors. LTBI screening in IMID patients is problematic because they are often already on prednisolone therapy (which can confound LTBI screening) and controversy surrounds the choice of screening test (i.e. TST or IGRA). Smith et al[29] recently summarised 14 studies comparing TST and IGRAs in a total of 1,630 patients with a variety of IMIDs. The lack of a 'gold standard' for LTBI again confounded these studies, which therefore relied upon correlating TST and IGRA results; five publications also studied the association of test results with TB risk factors by multivariate analysis. The summary of these 14 studies was that IGRAs could not be demonstrated to be superior to TST for LTBI screening in IMID patients. Higher-level evidence of the efficacy of IGRAs in IMIDs is also lacking (such as a formal meta-analysis or longitudinal studies of the risk of active TB in IGRA-positive and -negative patients).

Several societies and organisations in high-income countries with a low incidence of TB have published guidelines for LTBI screening in IMID patients.[27,29] These guidelines generally recommend TST and/or IGRA. Emphasis is also placed upon the importance of an extensive clinical history looking for TB risk factors (e.g. exposure to a TB patient; residence in a TB-endemic country; working or living in congregate settings such as hospitals, jails or homeless shelters) and on a chest x-ray (looking for fibronodular opacities suggestive of inactive TB). For example, the Australian Rheumatology Association[30] recommends a case history risk assessment, chest X-ray within last three months, and either two step TST skin test or IGRA.

Recommendation

Either TST or IGRA are acceptable for LTBI screening in IMID patients. IGRA may be preferred if there is a history of BCG immunisation after one year of age. Both TST and IGRA may be performed if the risk of LTBI is considered high; a diagnosis of LTBI would be made by a positive result in either test.

The TB exposure history and chest X-ray are central in interpreting the TST/IGRA result and in determining the overall risk of LTBI in IMID patients.

Other immunocompromised individuals

Other immunocompromised populations (e.g. pre-organ transplantation, patients with end-stage renal failure on dialysis) are also at increased risk of TB reactivation. For example, the incidence of post-transplant TB is 1.2%–6.4% in non-endemic countries, which is 20–74-fold higher than the general population.[31] Screening for LTBI is therefore indicated in these groups. Unfortunately, published comparisons of IGRAs and TST in these populations are limited and there is a high rate of indeterminate IGRA results in these groups.[31–33] There is also a lack of higher-level evidence of the efficacy of IGRAs in these 'other immunocompromised patient groups'. Hence, NTAC makes the same recommendations for LTBI screening in these 'other immunocompromised' individuals as for IMID patients pre-anti-tumour necrosis factor-α therapy.

Recommendation

Either TST or IGRA are acceptable for LTBI screening in other immunocompromised patients. IGRA may be preferred if there is a history of BCG immunisation after age one year. Both TST and IGRA may be performed if the risk of LTBI is considered high; a diagnosis of LTBI would be made by a positive result in either test.

The TB exposure history and chest X-ray are central in interpreting the TST/IGRA result and in determining the overall risk of LTBI in immunocompromised patients.

Serial testing of healthcare workers

Screening for LTBI in a low-prevalence the healthcare-worker (HCW) population using the traditional TST can be problematic due to the potential confounding effects from previous BCG vaccination and the booster effect from repeat tests in the previously sensitised that can result in false conversions. The IGRA offers improved specificity in relation to the BCG vaccinated and lack of a booster or sensitising effect from repeat testing. IGRAs therefore have potential advantages in HCW screening over the TST.

A recent systematic review of IGRA testing in HCWs from low-prevalence countries found that the IGRA predicts lower rates of LTBI than TST when used in a single screening situation.[34] The higher specificity of the IGRA in the BCG vaccinated is the suggested explanation and may result in fewer HCWs being recommended preventive therapy.

However, interpretation of IGRA results when used for serial testing has raised several questions, particularly regarding the threshold to distinguish new infection from non-specific variation.[35,36] Studies using IGRA in low prevalence populations suggest that the use of a single cut point (0.35 IU/ml) to separate negative from positive is problematic.[34,37–39] The IGRA can vary non-specifically close to this cut-point resulting in high conversion and reversion rates being observed. Gandra et al[37] from the University of Illinois College of Medicine at Peoria, report that screening 6,530 HCWs by QFN-GIT cost $436,096 compared with $78,360 by TST. The increased expense was caused by direct screening costs and additional indirect costs such as extra follow-up visits and investigations for HCWs with borderline-positive QFN-GIT test results and additional chest radiographs.

The dilemma of conversion/reversion results with IGRAs has prompted consideration of alternative definitions for a 'new infection' in HCWs including an absolute increase over baseline or use of a 'grey zone' with a higher cut-point.[37,38,39]

Recommendation

The problem of defining an appropriate cut-off point has resulted in a trend towards more cautious use of IGRAs for HCW screening. For the present, TST remains the preferred test for HCW screening in Australia with IGRA's role limited to supplementary testing as a specificity tool.

Indeterminate results

IGRAs can produce uninterpretable (termed 'indeterminate') results either due to inappropriately high or low IFN-γ response in the negative or positive controls, respectively. The rate of indeterminate results has varied between studies, between populations (i.e. more common in children and immunosuppressed patients) and between assays.[40,41] Some international guidelines provide suggestions on the management of indeterminate reactions. For example, the Canadian guidelines recommend repeat testing of immunocompromised patients with an initial-indeterminate result.[40] Repeated indeterminate results are considered a marker of anergy. The clinician must then determine the patient's LTBI status based on TB exposure history and other results.

The handling of indeterminate results highlights an important principle. IGRAs should only be carried out by clinicians experienced in the diagnosis and management of TB and LTBI. The investigation and management of such patients should occur in liaison with the relevant state or territory TB service. Problematic IGRA results, including indeterminate reactions, can then be assessed expertly in the patient's clinical setting.

Concluding remarks

While international studies have attempted to define the performance and utility of IGRAs, NTAC notes a continuing absence of cost-effectiveness studies of IGRAs under Australasian TB program conditions. Both NTAC and the state-based TB services encourage further clinical and economic evaluation of IGRAs, particularly independent cost-benefit analyses on the use of IGRAs using states' and territories' preferred protocols of investigating LTBI in Australia. Such analyses are needed to determine the relative economic outcomes of changing from TST to immunoassays taking into account the structure of TB services and program delivery in Australia.

The World Health Organization has released recently a policy statement on the use of IGRAs in low– and middle-income countries.[41] This document was based on commissioned systematic reviews of studies from low- and middle-income countries supplemented by the input of an Expert Group. The recommendations are therefore not directly applicable to high-income low-incidence countries such as Australia. However, NTAC notes that the WHO IGRA recommendations match these NTAC guidelines.

This NTAC position statement and recommendations will remain under ongoing review and will be revised as new peer-reviewed published data becomes available. NTAC is committed to ongoing monitoring of new diagnostic tests that may be of value in TB control.

Transparency declaration

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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 55,105 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 October and 31 December 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 - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia

Table 1 *continued*: Reporting of notifiable diseases by jurisdiction

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 October to 31 December 2011, by date of diagnosis

Disease	State or territory								Total 4th quarter 2011	Total 3rd quarter 2011	Total 4th quarter 2010	Last 5 years mean 4th 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	0.4
Hepatitis B (newly acquired)*	0	4	1	14	4	1	25	3	52	46	38	62.0	0.8	189	263.0
Hepatitis B (unspecified)†	23	602	38	182	98	6	463	174	1,586	1,743	1,624	1,645.2	1.0	6,662	6,708.2
Hepatitis C (newly acquired)*,‡	3	8	2	NN	5	4	0	22	44	58	82	95.6	0.5	280	385.8
Hepatitis C (unspecified)†	41	759	61	584	102	41	554	222	2,364	2,586	2,646	2,740.0	0.9	9,986	11,301.6
Hepatitis D	0	1	0	2	0	0	1	0	4	10	10	7.0	0.6	35	34.8
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0.2	0.0	2	0.6
Campylobacteriosis§	126	NN	24	1,187	550	282	1,664	536	4,369	4,447	5,394	4,568.0	1.0	17,725	16,201.6
Cryptosporidiosis	3	83	43	158	8	8	63	45	411	288	308	471.8	0.9	1,807	2,823.4
Haemolytic uraemic syndrome	0	0	0	0	1	0	3	0	4	4	1	6.4	0.6	13	17.4
Hepatitis A	2	14	0	5	2	2	9	6	40	28	52	86.4	0.5	144	311.0
Hepatitis E	0	2	0	2	0	0	2	3	9	6	6	5.0	1.8	40	31.2
Listeriosis	0	6	0	4	1	1	8	2	22	10	17	15.4	1.4	70	68.4
STEC, VTEC‖	5	4	0	6	18	1	2	0	36	27	17	31.8	1.1	98	98.6
Salmonellosis	65	720	113	726	271	54	638	332	2,919	2,012	2,920	2,348.6	1.2	12,286	9,502.4
Shigellosis	4	42	36	11	13	0	21	14	141	95	129	145.4	1.0	496	627.6
Typhoid	1	6	1	6	2	1	10	4	31	22	17	21.8	1.4	134	96.4
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	0	1.8	0.0	6	3.8
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	2	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2011, by date of diagnosis

Disease	State or territory								Total 4th quarter 2011	Total 3rd quarter 2011	Total 4th quarter 2010	Last 5 years mean 4th quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[¶,**]	297	5,002	636	4,521	1,205	453	4,837	2,651	19,602	20,254	17,963	14,425.0	1.4	80,827	59,012.0
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	2.6
Gonococcal infection**	20	890	482	751	115	6	444	504	3,212	2,867	2,565	2,002.2	1.6	12,124	8,390.6
Syphilis – congenital**	0	0	0	1	0	0	0	0	1	2	0	1.4	0.7	7	6.0
Syphilis <2 years duration**	2	101	3	70	12	1	71	29	289	301	275	291.4	1.0	1,259	1,218.6
Syphilis >2 years or unspecified duration**	5	83	16	58	NDP	9	111	31	313	317	268	319.4	1.0	1,252	1,338.6
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	4	0.0
Haemophilus influenzae type b	0	0	0	1	0	0	0	1	2	3	8	5.4	0.4	13	21.4
Influenza (laboratory confirmed)	34	583	59	640	510	28	594	451	2,899	17,375	4,048	1,514.8	1.9	27,018	19,119.6
Measles	19	11	2	1	1	0	8	8	50	34	10	6.4	7.8	193	75.2
Mumps	0	21	0	12	0	1	5	2	41	35	30	87.0	0.5	153	280.6
Pertussis	105	3,270	140	2,473	292	204	1,829	1,999	10,312	9,572	14,568	6,322.8	1.6	38,579	18,698.0
Pneumococcal disease (invasive)	6	121	27	62	24	17	83	43	383	724	391	330.2	1.2	1,879	1,547.4
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.3
Rubella	1	3	0	1	1	0	2	0	8	14	4	6.6	1.2	58	40.0
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	3	3.0
Varicella zoster (chickenpox)[††]	3	NN	44	89	164	5	218	128	651	658	564	608.6	1.1	2,092	1,717.6
Varicella zoster (shingles)[††]	10	NN	48	20	396	64	265	261	1,064	1,008	831	618.0	1.7	3,986	2,153.6
Varicella zoster (unspecified)[††]	30	NN	1	1,075	65	28	723	257	2,179	2,004	1,893	1,457.4	1.5	7,732	5,278.0
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	1	4	0	0	3	0	8	6	15	4.4	1.8	24	18.2
Barmah Forest virus infection	0	68	12	208	16	0	4	38	346	289	409	374.0	0.9	1,867	1,773.0
Dengue virus infection	3	46	7	38	7	1	27	84	213	118	467	181.2	1.2	820	738.4
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Kunjin virus infection[‡‡]	0	1	0	0	0	0	0	0	1	0	0	0.2	5.0	2	1.8
Malaria	0	18	11	36	3	3	22	17	110	95	103	121.0	0.9	413	552.0
Murray Valley encephalitis virus infection[‡‡]	0	1	0	0	0	0	0	0	1	0	0	0.0	0.0	16	1.4
Ross River virus infection	0	68	46	273	41	2	44	274	748	378	1,051	895.6	0.8	5,161	5,058.6

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2011, by date of diagnosis

Disease	State or territory								Total 4th quarter 2011	Total 3rd quarter 2011	Total 4th quarter 2010	Last 5 years mean 4th 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	0	0.0	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	1	0	8	0	0	0	0	9	7	5	9.0	1.0	39	37.0
Leptospirosis	0	6	1	10	0	1	2	1	21	19	36	22.2	0.9	217	127.6
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	6	0	1	0	0	14	4	25	20	30	25.6	1.0	85	96.8
Q fever	0	35	0	43	1	0	6	6	91	80	78	92.4	1.0	328	374.8
Tularaemia	0	0	0	0	0	1	0	0	1	0	0	0.0	0.0	2	0.0
Other bacterial infections															
Legionellosis	0	21	1	12	8	2	26	31	101	60	83	82.2	1.2	343	305.4
Leprosy	0	1	0	0	1	0	1	0	3	2	4	2.6	1.2	7	9.4
Meningococcal infection§§	0	16	1	7	6	0	11	8	49	77	56	65.2	0.8	241	279.2
Tuberculosis	9	93	8	59	22	6	108	35	340	350	382	368.0	0.9	1,263	1,236.4
Total	817	12,717	1,865	13,361	3,965	1,233	12,921	8,226	55,105	68,053	59,398			237,982	

* 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 October to 31 December 2011, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	0.0	0.2	1.7	1.2	1.0	0.8	1.8	0.5	0.9
Hepatitis B (unspecified)†	25.6	33.3	66.2	16.1	23.8	4.7	33.4	30.3	28.4
Hepatitis C (newly acquired)*	3.3	0.4	3.5	NN	1.2	3.2	0.0	3.8	1.0
Hepatitis C (unspecified)†,‡	45.7	41.9	106.2	51.7	24.8	32.3	39.9	38.7	42.3
Hepatitis D	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	140.4	NN	41.8	105.1	133.8	222.2	120.0	93.4	115.7
Cryptosporidiosis	3.3	4.6	74.9	14.0	1.9	6.3	4.5	7.8	7.4
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.2	0.0	0.2	0.0	0.1
Hepatitis A	2.2	0.8	0.0	0.4	0.5	1.6	0.6	1.0	0.7
Hepatitis E	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.5	0.2
Listeriosis	0.0	0.3	0.0	0.4	0.2	0.8	0.6	0.3	0.4
STEC,VTEC‖	5.6	0.2	0.0	0.5	4.4	0.8	0.1	0.0	0.6
Salmonellosis	72.4	39.8	196.8	64.3	65.9	42.6	46.0	57.8	52.3
Shigellosis	4.5	2.3	62.7	1.0	3.2	0.0	1.5	2.4	2.5
Typhoid fever	1.1	0.3	1.7	0.5	0.5	0.8	0.7	0.7	0.6
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¶,**	331.0	276.4	1,107.7	400.4	293.1	357.0	348.8	461.8	350.9
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	22.3	49.2	839.4	66.5	28.0	4.7	32.0	87.8	57.5
Syphilis – congenital**	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Syphilis <2 years duration**	2.2	5.6	5.2	6.2	2.9	0.8	5.1	5.1	5.2
Syphilis >2 years or unspecified duration†,**	5.6	4.6	27.9	5.1	-	7.1	8.0	5.4	6.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0
Influenza (laboratory confirmed)	37.9	32.2	102.8	56.7	124.0	22.1	42.8	78.6	51.9
Measles	21.2	0.6	3.5	0.1	0.2	0.0	0.6	1.4	0.9
Mumps	0.0	1.2	0.0	1.1	0.0	0.8	0.4	0.3	0.7
Pertussis	117.0	180.7	243.8	219.0	71.0	160.7	131.9	348.2	184.6
Pneumococcal disease (invasive)	6.7	6.7	47.0	5.5	5.8	13.4	6.0	7.5	6.9
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	1.1	0.2	0.0	0.1	0.2	0.0	0.1	0.0	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 3 *continued*: Notification rates of diseases, 1 October to 31 December 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, *cont'd*									
Varicella zoster (chickenpox)	3.3	NN	76.6	7.9	39.9	3.9	15.7	22.3	17.2
Varicella zoster (shingles)	11.1	NN	83.6	1.8	96.3	50.4	19.1	45.5	28.2
Varicella zoster (unspecified)	33.4	NN	1.7	95.2	15.8	22.1	52.1	44.8	57.7
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	1.7	0.4	0.0	0.0	0.2	0.0	0.1
Barmah Forest virus infection	0.0	3.8	20.9	18.4	3.9	0.0	0.3	6.6	6.2
Dengue virus infection	3.3	2.5	12.2	3.4	1.7	0.8	1.9	14.6	3.8
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	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	0.0	1.0	19.2	3.2	0.7	2.4	1.6	3.0	2.0
Murray Valley encephalitis virus infection††	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	3.8	80.1	24.2	10.0	1.6	3.2	47.7	13.4
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.7	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.3	1.7	0.9	0.0	0.8	0.1	0.2	0.4
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	0.1	0.0	0.0	1.0	0.7	0.4
Q fever	0.0	1.9	0.0	3.8	0.2	0.0	0.4	1.0	1.6
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	1.2	1.7	1.1	1.9	1.6	1.9	5.4	1.8
Leprosy	0.0	0.1	0.0	0.0	0.2	0.0	0.1	0.0	0.1
Meningococcal infection‡‡	0.0	0.9	1.7	0.6	1.5	0.0	0.8	1.4	0.9
Tuberculosis	10.0	5.1	13.9	5.2	5.4	4.7	7.8	6.1	6.1

* 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).

†† 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.

Additional reports

Australian childhood immunisation coverage

Tables 1, 2 and 3 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months of age, for 3-month birth cohorts of children at the stated ages between 1 July and 30 September 2011. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, or meningococcal C conjugate vaccines, and is outlined in more detail below.

A full description of the basic methodology used can be found in Commun Dis Intell *1998;22:36–37.*

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of 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' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP containing 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 (MMR)-containing vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.*

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1435, E-mail: brynleyh@chw.edu.au

The percentage of children 'fully immunised' by 12 months of age for Australia decreased marginally from the previous quarter by 0.3 of a percentage point to 91.8% (Table 1). Important changes in coverage were seen only in the Northern Territory with coverage for 'fully immunised', DTP, polio, Hib and Hep B vaccines decreasing by almost 6 percentage points. However, this apparent decrease in coverage was due to an administrative delay in data reported to the ACIR from the Northern Territory.

The percentage of children 'fully immunised' by 24 months of age for Australia decreased marginally from the previous quarter by 0.3 of a percentage point to 92.6% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 60 months of age for Australia increased from the previous quarter by 0.6 of a percentage point to 89.9% (Table 3). This is the highest coverage has been for this milestone since coverage was first calculated at the 72-month age milestone in March 2002. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage at 5 years of age is close to the coverage levels attained at 12 and 24 months.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2010; assessment date 31 December 2011

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,284	24,804	964	15,655	4,905	1,534	18,354	7,935	75,435
Diphtheria, tetanus, pertussis (%)	94.2	92.1	87.7	92.0	92.1	93.0	93.5	91.3	92.3
Poliomyelitis (%)	94.1	92.0	87.7	91.9	92.0	93.0	93.5	91.2	92.3
Haemophilus influenzae type b (%)	93.7	91.9	87.7	91.9	91.9	92.9	93.3	91.1	92.1
Hepatitis B (%)	93.5	91.7	87.6	91.6	91.9	92.8	93.1	90.7	91.9
Fully immunised (%)	93.3	91.6	87.5	91.6	91.7	92.8	92.9	90.6	91.8
Change in fully immunised since last quarter (%)	-0.3	-0.3	-5.9	-0.3	-1.3	+1.5	+0.2	-0.2	-0.3

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2009; assessment date 31 December 2011*

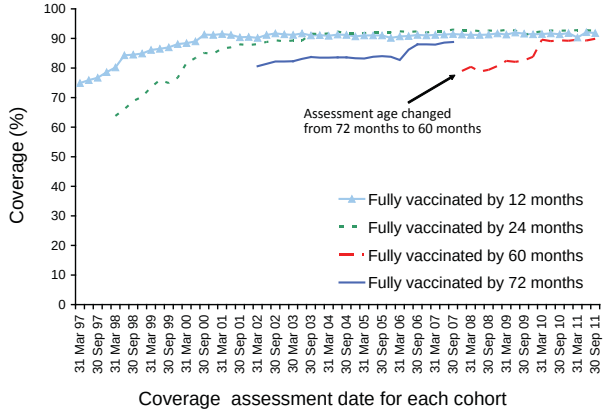
Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,325	25,138	908	15,694	4,999	1,625	18,463	7,983	76,135
Diphtheria, tetanus, pertussis (%)	96.2	94.7	95.5	94.4	94.7	95.2	95.2	93.4	94.7
Poliomyelitis (%)	96.1	94.7	95.6	94.4	94.7	95.2	95.2	93.4	94.7
Haemophilus influenzae type b (%)	96.5	95.2	96.0	94.5	94.8	95.5	95.4	93.7	95.0
Measles, mumps, rubella (%)	95.2	93.9	95.6	93.8	93.8	94.4	94.5	92.4	93.9
Hepatitis B (%)	95.2	94.4	95.5	94.0	94.3	94.8	94.7	92.9	94.3
Fully immunised (%)	93.7	92.6	94.6	92.5	92.6	93.4	93.1	90.9	92.6
Change in fully immunised since last quarter (%)	-0.9	+0.3	+0.9	-0.5	-0.6	-0.9	-0.5	-0.8	-0.3

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2011;35(1):48.

Table 3. Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2006; assessment date 31 December 2011

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,281	25,166	851	15,791	5,080	1,758	18,844	8,276	77,047
Diphtheria, tetanus, pertussis (%)	93.3	90.2	88.0	91.0	88.7	91.8	92.1	87.5	90.5
Poliomyelitis (%)	93.3	90.1	88.0	90.9	88.6	91.6	92.1	87.4	90.4
Measles, mumps, rubella (%)	92.7	90.1	87.7	90.6	88.4	91.5	91.8	87.4	90.3
Fully immunised (%)	92.5	89.7	87.5	90.3	88.1	91.1	91.6	86.8	89.9
Change in fully immunised since last quarter (%)	+1.2	+0.1	-0.9	+1.2	+1.5	+0.9	+0.4	+1.2	+0.6

Figure: Trends in vaccination coverage, Australia, 1997 to 30 September 2011, by age cohorts



Australian Sentinel Practices Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government 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 influenza-like illness (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 influenza A H1N1(2009).

The list of conditions is reviewed annually by the ASPREN management committee. In 2012, 4 conditions are being monitored. They include 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 *2012;36(1):122.*

Reporting period 1 October to 31 December 2011

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 125 general practitioners contributed data to ASPREN in the fourth quarter of 2011. Each week an average of 106 general practitioners provided information to ASPREN at an average of 9,177 (range 6,006–10,209) consultations per week and an average of 138 (range 113–198) notifications per week.

ILI rates reported from 1 October to 31 December 2011 averaged 8 cases per 1,000 consultations (range 6–13 cases per 1,000 consultations). The reported rates in October, November and December 2011 (7–13 cases per 1,000 consultations, 6–8 cases per 1,000 consultations and 6–8 cases per 1,000 consultations respectively) were lower compared with rates in the same reporting period in 2010 (13–22 cases per 1,000 consultations, 13–17 cases per 1,000 consultations and 13–15 cases per 1,000 consultations respectively) (Figure 1).

ILI swab testing has continued through 2011. The most commonly reported virus during this reporting period was rhinovirus (17% of all swabs performed), with the second most common virus being influenza B (13% of all swabs performed).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2010 to 31 December 2011, by week of report

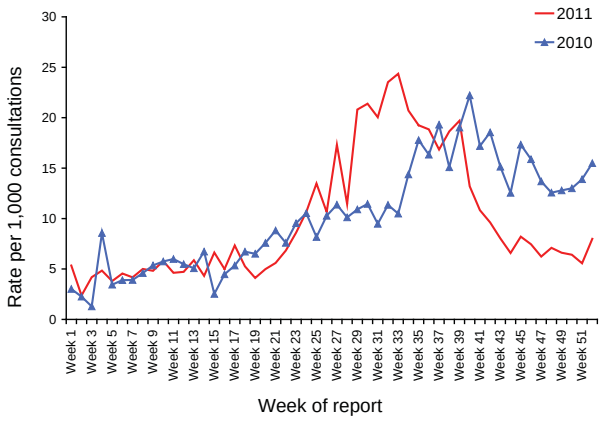
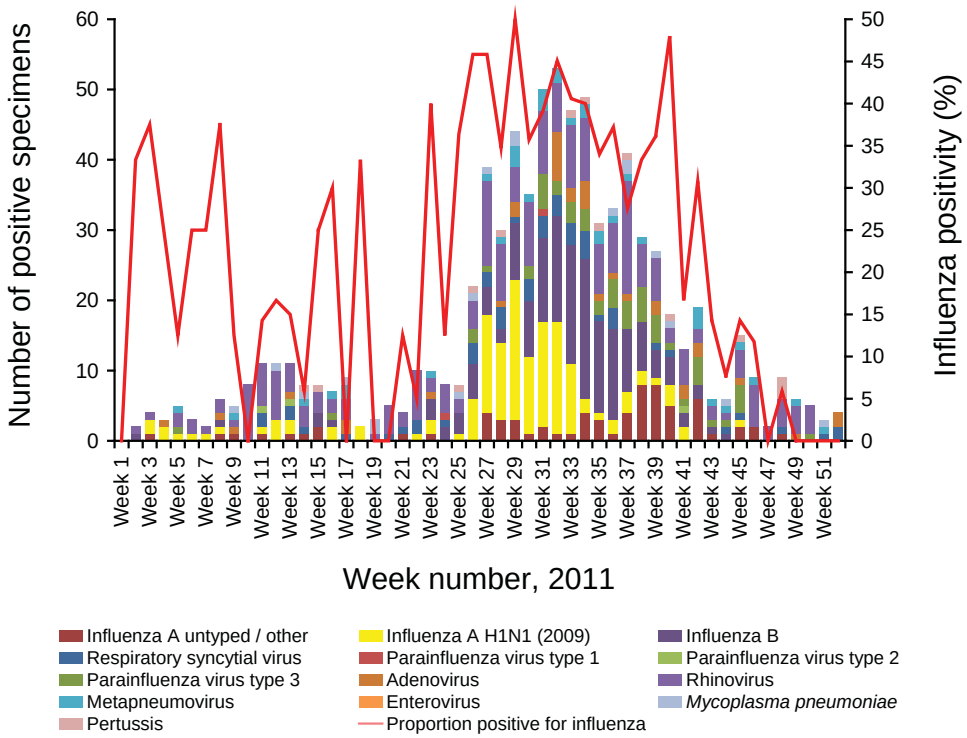


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January 2010 to 31 December 2011, by week of report



From the beginning of 2011 to the end of week 52, 372 cases of influenza were detected, the majority of these being influenza B (13% of all swabs performed), H1N1(2009) (11% of all swabs performed) and the remainder influenza A untyped or other (6% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 6 cases per 1,000 consultations (range 3–12 cases per 1,000, Figure 3), which corresponds with rates in the same reporting period in 2010 where the average was 6 cases per 1,000 consultations (range 4–7 cases per 1,000).

Varicella infections were reported at a slightly higher rate for the fourth quarter of 2011 compared with the same period in 2010. From 1 October to 31 December 2011, recorded rates for chickenpox averaged 0.33 cases per 1,000 consultations (range 0.1–0.83 cases per 1,000 consultations) (Figure 4).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2010 to 31 December 2011, by week of report

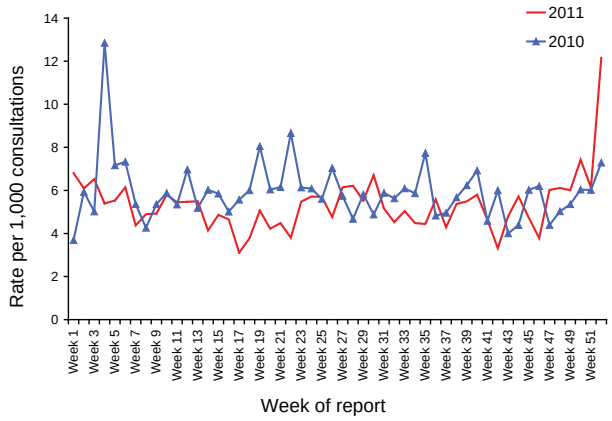
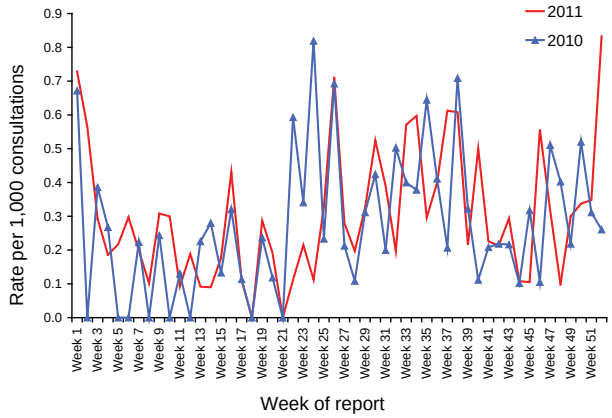
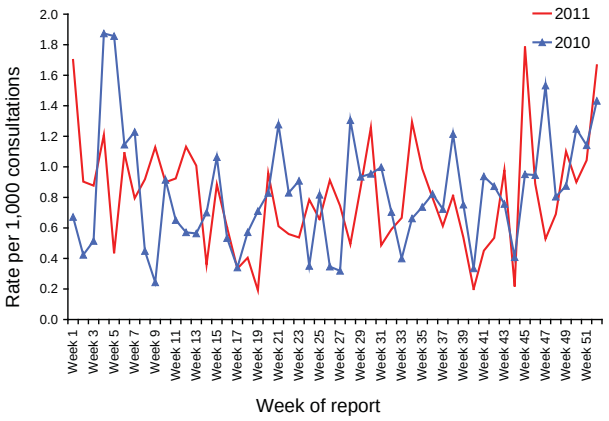


Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2010 to 31 December 2011, by week of report



In the fourth quarter of 2011, reported rates for shingles averaged 0.85 cases per 1,000 consultations (range 0.2–1.79 cases per 1,000 consultations, Figure 5), which was lower compared with the same reporting period in 2010 where the average shingles rate was 0.94 cases per 1,000 consultations (0.34–1.53 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 1 January 2010 to 31 December 2011, by week of report



Gonococcal surveillance

(Dr Monica M Lahra, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme)

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro *resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.*[1] *Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see* Commun Dis Intell *2012;36(1):121.*

Reporting period 1 July to 30 September 2011

The AGSP laboratories received a total of 985 gonococcal isolates and 963 (98%) remained viable for susceptibility testing. This represented a 3% decrease from the 1,014 gonococci referred in this same quarter in 2010. Of the total gonococci referred, 34% were from New South Wales; 21% from Victoria; 19% from Queensland, 12% from the Northern Territory; 11% from Western Australia 3% from South Australia; 1% from the Australian Capital Territory and 1 isolate (0.1%) from Tasmania.

Penicillins

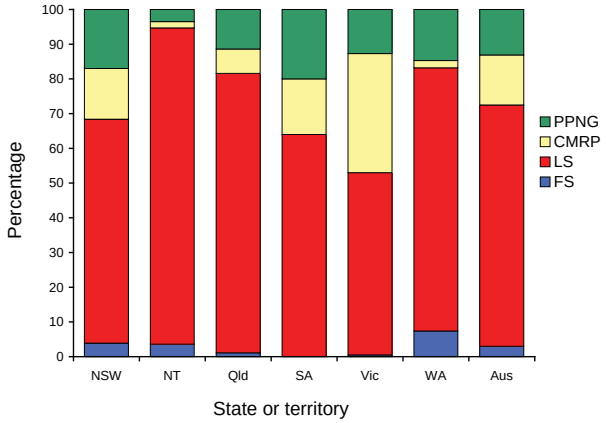
Two hundred and sixty-five (28%) of the 963 isolates examined were penicillin resistant by one or more mechanisms: 126 (13%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 139 (14%) had chromosomally mediated resistance to penicillin (CMRP). Compared with the same quarter in 2010, this represents a small increase in the proportion of PPNG isolates (to 10% in 2010) and a decrease in CMRP (from 16% in 2010). The proportion of all strains resistant to the penicillins by any mechanism ranged from 5% (6/113 isolates) in the Northern Territory to 47% in Victoria. The penicillin resistance rate in South Australia was 36%; in New South Wales 32%, in Queensland 18%, and in Western Australia 17%. There were no penicillin resistant gonococci reported from the Australian Capital Territory or from Tasmania. Whilst the national proportion of PPNG has remained stable at 11%–13% over the period 2007–2011, the proportion of gonococci with CMRP has decreased in the same quarter from 22% in 2009 to 16% in 2010 to 14% in this quarter of 2011.

In Figure 1 the proportion of gonococci fully sensitive (FS) [minimum inhibatory concentration (MIC) ≤0.03 mg/L]; less sensitive (LS) (MIC 0.06–0.5 mg/L); CMRP (MIC ≤ 1 mg/L) and PPNG are shown by state and territory and as aggregated for Australia. A high proportion of strains classified as PPNG or CMRP fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporin antibiotics.

Penicillin resistance by CMRP predominated over PPNG only in Victoria (34% CMRP: 13% PPNG). In the remaining laboratories, PPNG predominated: New South Wales (17% PPNG: 15% CMRP); Western Australia (15% PPNG: 2% CMRP); Queensland (11% PPNG: 7% CMRP); South Australia (20% PPNG:16% CMRP).

In the Northern Territory where 6 of 113 isolates were resistant to penicillin, 4 (3.5%) were PPNG and 2 (1.8%) were CMRP. Information on the geographic acquisition was available for 1 PPNG isolate, acquired overseas from Papua New Guinea. For the remaining 5 penicillin resistant isolates, information on geographic acquisition was not available.

Figure 1: Categorisation of gonococci isolated in Australia, 1 July to 30 September 2011, by penicillin susceptibility and state or territory



FS	Fully sensitive to penicillin, MIC ≤0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L.
CMRP	Chromosomally mediated resistant to penicillin, MIC ≥1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae.*

Ceftriaxone

From 2001 onwards, gonococcal isolates with raised ceftriaxone MIC values have been increasingly reported in Australia. Decreased susceptibility to ceftriaxone has been defined as the MIC range 0.06–0.12 mg/L. In 2010 the proportion of gonococci with an MIC value in the range 0.03–0.12 mg/L was reported from the second quarter AGSP. The rationale for this was to improve the detection of gonococci with decreased susceptibility to ceftriaxone by documenting the right shift in MIC range to ceftriaxone in this organism.

In this quarter, data for decreased susceptibility to ceftriaxone (MIC ≥ 0.06 mg/L) were contributed by all jurisdictions, with 963 isolates examined. Nationally, 36 of the 963 isolates (3.7%) were reported as having decreased susceptibility to ceftriaxone. There were 16 of 204 (7.8%) reported from Victoria; 12 of 330 (3.6%) from New South Wales; 5 of 185 (2.7%) from Queensland; 2 of 95 (2.1%) from Western Australia and there was 1 isolate (0.9%) in the Northern Territory. There were no gonococci with decreased susceptibility to ceftriaxone reported from South Australia, the Australian Capital Territory or Tasmania.

Data for ceftriaxone MIC ≥ 0.03 mg/L were contributed by all jurisdictions (963 isolates). There were 150 isolates (16%) reported with ceftriaxone MIC ≥ 0.03 mg/L: 63 of 204 (31%) isolates reported from Victoria; 5 of 25 (20%) in South Australia; 57 of 330 (17%) in New South Wales; 20 of 185 (11%) in Queensland; 3 of 95 (3.2%) in Western Australia and 2 of 112 (1.8%) in the Northern Territory. There were none in the Australian Capital Territory or Tasmania. In 2011 the overall proportion of isolates in this MIC range was similar to the 15% reported in this same quarter in 2010. By state and territory in 2011, there was an increase from 21% in 2010 to 30% in Victoria in 2011, and from 11% in 2010 to 20% in South Australia; and from none reported in 2010 to 1.8% in the Northern Territory. The proportion was similar in New South Wales (18% in 2010) and Queensland (11% in 2010) and decreased from 17% in 2010 to 3.2% in Western Australia and from one to none in the Australian Capital Territory. Again none were reported from Tasmania.

Spectinomycin

All isolates were susceptible to this injectable agent.

Quinolone antibiotics

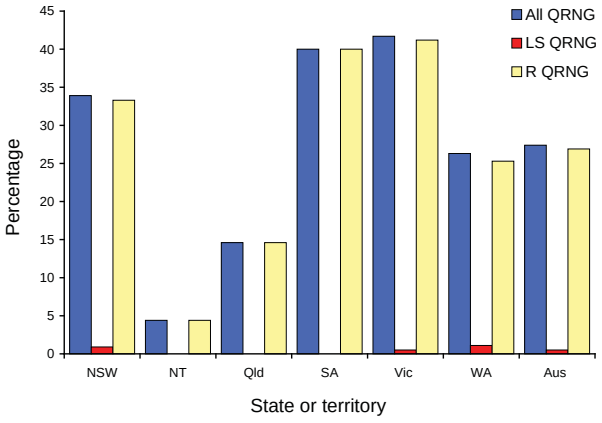
Nationally, the 264 quinolone resistant *N. gonorrhoeae* (QRNG) detected in this quarter represented 27% of all isolates tested. This shows a continuing decrease in the proportion of QRNG from 33% from the same quarter in 2010; 41.3% from the same quarter in 2009; 50.6% QRNG recorded in the third quarter of 2008 and 50.5% in 2007. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups. The majority of QRNG (209/264, 79%) had higher-level resistance to ciprofloxacin: MIC 4 mg/L or more.

QRNG were detected in high proportions in Victoria 85 of 204 (42%); South Australia 10 of 25 (40%); New South Wales 112 of 330 (34%); Western Australia 25 of 95 (26%); and in Queensland 27 of 185 (15%) (Figure 2). There were 5 QRNG detected in the Northern Territory (4%), and none in Tasmania or the Australian Capital Territory.

High level tetracycline resistant *Neisseria gonorrhoeae*

The number (169) and proportion (18%) of high level tetracycline resistant *N. gonorrhoeae* (TRNG) detected, was less than that recorded in this quarter in 2010 (21%) and 2009 (21%). TRNG were found in all states and territories except for Tasmania and the Australian Capital Territory and represented between 16% (in both New South Wales and Western Australia) and 25% (Queensland) of all isolates tested.

Figure 2: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 July to 30 September 2011, by state or territory



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥1 mg/L.

Reference

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

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health and Ageing. 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 *2012;36(1):123.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2011, are included in this issue of Communicable Diseases Intelligence *(Tables 1, 2, 3 and 4).*

Table 1: Number of new diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 January to 31 March 2011, 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 2011	This period 2010	YTD 2011	YTD 2010
HIV diagnoses	Female	0	3	0	7	1	1	7	3	22	42	22	42
	Male	1	86	2	60	9	1	72	14	245	236	245	236
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	89	2	67	10	2	79	17	267	282	267	282
AIDS diagnoses	Female	0	4	0	0	0	0	0	0	4	5	4	5
	Male	0	11	1	1	0	0	12	0	25	29	25	29
	Total*	0	15	1	1	0	0	12	0	29	34	29	34
AIDS deaths	Female	0	1	0	0	0	0	0	0	1	0	1	0
	Male	0	0	0	0	0	0	4	0	4	7	4	7
	Total*	0	1	0	0	0	0	4	0	5	7	5	7

* Totals include people whose sex was reported as transgender.

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 2011, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	39	1,047	31	405	131	23	509	305	2,490
	Male	295	14,915	165	3,508	1,111	142	6,329	1,493	27,958
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	334	16,224	196	3,922	1,243	165	6,884	1,805	30,773
AIDS diagnoses	Female	10	286	6	80	32	4	128	49	595
	Male	95	5,647	52	1,114	427	55	2,218	469	10,077
	Total*	105	5,952	58	1,196	460	59	2,359	520	10,709
AIDS deaths	Female	7	143	1	44	20	2	66	30	313
	Male	73	3,612	33	687	281	34	1,467	301	6,488
	Total*	80	3,766	34	733	301	36	1,542	332	6,824

* Totals include people whose sex was reported as transgender.

Table 3: Number of new diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2011, 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 2011	This period 2010	YTD 2011	YTD 2010
HIV diagnoses	Female	1	3	0	10	9	1	9	8	41	36	63	78
	Male	3	76	1	48	18	5	77	19	247	277	492	486
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	4	79	1	58	27	6	86	27	288	277	555	559
AIDS diagnoses	Female	0	2	0	0	0	0	2	0	4	3	8	8
	Male	0	13	0	1	0	0	9	0	23	26	48	55
	Total*	0	15	0	1	0	0	11	0	27	29	56	63
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	1	1
	Male	0	2	0	0	0	0	1	0	3	4	7	11
	Total*	0	2	0	0	0	0	1	0	3	5	8	12

* Totals include people whose sex was reported as transgender.

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 2011, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	40	1,050	31	415	140	24	518	313	2,531
	Male	298	14,991	166	3,556	1,129	147	6,406	1,512	28,205
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	338	16,303	197	3,980	1,270	171	6,970	1,832	31,061
AIDS diagnoses	Female	10	288	6	80	33	4	130	49	600
	Male	95	5,660	52	1,115	428	55	2,227	469	10,101
	Total*	105	5,967	58	1,197	462	59	2,370	520	10,738
AIDS deaths	Female	7	143	1	44	20	2	66	30	313
	Male	73	3,614	33	687	281	34	1,468	301	6,491
	Total*	80	3,768	34	733	301	36	1,543	332	6,827

* Totals include people whose sex was reported as transgender.

Administration

COMMUNICABLE DISEASES INTELLIGENCE
INSTRUCTIONS FOR AUTHORS

Communicable Diseases Intelligence (CDI) is published quarterly (March, June, September and December) by the Health Protection and Surveillance Branch, Office of Health Protection, Australian Government Department of Health and Ageing.

The aim of (CDI) is to disseminate information on the epidemiology of communicable disease in Australia, including surveillance, prevention and control.

The objectives of CDI are to:

- report on surveillance of communicable diseases of relevance to Australia;
- publish other articles relevant to communicable disease epidemiology in Australia; and
- provide information on other activities relevant to the surveillance, prevention and control of communicable disease in Australia.

CDI invites contributions dealing with any aspect of communicable disease epidemiology, surveillance, prevention or control in Australia. Submissions can be in the form of original articles, short reports, or letters to the editor.

CDI will invite guest editorials and review articles on occasion and publish guidelines and position papers from the Communicable Diseases Network Australia (CDNA) and its expert sub-committees.

Manuscripts for submission

Manuscripts submitted to CDI must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- a list of all authors;
- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, facsimile and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgment. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to printing.

Authorship

Authorship should be based on substantial contribution to the article. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Types of manuscript

Original Articles

The text of articles must be structured to contain an abstract, introduction, methods, results, discussion, acknowledgments and references. Manuscripts submitted as articles must be 3,000 words or less and are peer-reviewed. Occasionally, reports of urgent public health importance may be published immediately, at the discretion of the Editor.

Short reports

Short reports are not subject to peer review and should be of less than 2,000 words. Types of short reports include:

Surveillance summaries

A report of 1,000 words or less which briefly reports on changes in the local epidemiology of communicable disease, changes in surveillance systems, or new interventions, such as implementing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

Outbreak reports

Unstructured reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

Case reports

Brief unstructured reports of 500 to 1,000 words on unique cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (see Ethics committee approvals and patient's right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Letters to the Editor

The editors welcome comments on articles published in CDI in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than six references.

Document preparation

Authors are asked to provide an electronic copy of the manuscripts. Microsoft Word for Windows 2003 or an earlier version is preferred. Alternatively files should be saved as Rich Text Format (rtf).

In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts. Structured abstracts are not acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations.
- Do not use numbered paragraphs.
- Do not use page numbering.
- Do not use headers or footers.

Final manuscripts should not include any field codes such as automatic numbering for references. Electronic referencing software (e.g. Endnote) field codes should be embedded before submission of the final version.

Tables

- Tables and table headings should be provided in the manuscript at the end of the text and should be referred to within the results section.
- Information in tables should not be duplicated in the text.
- Headings should be brief.
- Simplify the information as much as possible, keeping the number of columns to a minimum.
- Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).
- If abbreviations are used these should be explained in a footnote.
- Footnotes should use the following symbols in sequence: * † ‡ § || ¶ ** †† ‡‡
- Do not use borders, or blank rows or blank columns for spacing.

Figures and illustrations

Figures and illustrations, including headings, should be provided in the manuscript at the end of the text and should be referred to within the results section. In addition, they should also be provided as a separate file in accordance with the following requirements.

Examples of each of the following can be found in the on-line version of Instructions to authors at: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm

Charts

- Use Microsoft Excel for Windows.
- Each figure should be created on a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).
- The numerical data used to create each figure must be included on a separate worksheet.
- Worksheets should be appropriately titled to distinguish each graph.
- Do not include the graph heading on the Excel worksheet.

Illustrations

- Illustrations or flow charts can be included if required.
- Images should preferably be at least 300 dpi.
- Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator but other sim-

ilar graphic software is acceptable. Files should be saved in one of the following graphic formats (in preferential order): AI, TIFF, EPS, or GIF.

- Use a sans serif font for figures (e.g. arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Photographs

- Photographs may be submitted if required.
- Photos need to be at least 300 dpi.
- Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order): PSD, TIFF, EPS or JPEG (JPG).

Maps

- Electronic copies of black and white (outline) maps should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order): PSD, TIFF, EPS, or GIF.
- Thermal maps created by mapping programs such as MapInfo or Arc GIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order): TIFF, EPS, or JPEG (JPG). Shading of map areas should be distinguishable when printed in black and white.
- Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

References

References should be identified consecutively in the text by the use of superscript numbers without brackets. Any punctuation should precede the reference indicators.

The accuracy of references is the responsibility of authors. Use the Vancouver reference style (see International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. Ann Intern Med 1997;1126:36–47 available from: http://www.nlm.nih.gov/bsd/uniform_requirements.html) and abbreviate journal names as in Medline (e.g. Commun Dis Intell). The PubMed journal database is available from: http://www.ncbi.nlm.nih.gov/nlmcatalog/journals Include the surnames and initials of all authors (or only the first six authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. a letter, an editorial, an abstract, or supplement).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

Ethics committee approvals and patients' rights to privacy

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should appear in the manuscript.

When informed consent has been obtained this should be included in the text.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Review process

Articles provisionally accepted for publication undergo a peer review process. Manuscripts are reviewed by two experts in the topic area. Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Occasionally, reports of urgent public health importance may be published immediately without peer review, at the discretion of the Editor. Articles may also be rejected without peer review.

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Requests for further information can be obtained either by telephone to (02) 6289 2717, by facsimile: (02) 6289 2600 or by email to the address above.