

AUSTRALIA'S NOTIFIABLE DISEASE STATUS, 2011: ANNUAL REPORT OF THE NATIONAL NOTIFIABLE DISEASES SURVEILLANCE SYSTEM

NNDSS Annual Report Writing Group

Abstract

In 2011, 65 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 238,158 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, an increase of 14% on the number of notifications in 2010. This increase was largely due to the ongoing pertussis epidemic and higher than usual inter-season notifications of influenza. In 2011, the most frequently notified diseases were sexually transmissible infections (95,456 notifications, 40.1% of total notifications), vaccine preventable diseases (81,872 notifications, 34.4% of total notifications), and gastrointestinal diseases (32,784 notifications, 13.8% of total notifications). There were 17,123 notifications of bloodborne diseases; 8,306 notifications of vectorborne diseases; 1,928 notifications of other bacterial infections; 681 notifications of zoonoses and 8 notifications of quarantinable diseases. *Commun Dis Intell* 2013;37(4):E313–E393.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2011, 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 the national level includes:

- identifying national trends;
- providing guidance for policy development and resource allocation at the national level;
- monitoring the need for and impact of national disease control programs;
- coordinating the response to national or multi-jurisdictional outbreaks;
- describing 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

- supporting quarantine activities, which are the responsibility of the Commonwealth 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 2007¹ 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 signed by Health Ministers in April 2008, establishes the operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. Under the Agreement, in 2011 states and territories forwarded de-identified data on the nationally agreed set of 65 communicable diseases to the Australian Government Department of Health for the purposes of national communicable disease surveillance, although not all 65 diseases were notifiable in each jurisdiction. Data were updated electronically from states and territories, daily or several times a week. The system was complemented by other surveillance systems, which provided information on various diseases, including four that are not reported to National Notifiable Diseases Surveillance System (NNDSS) (HIV, AIDS and the classical and variant forms of Creutzfeldt-Jakob disease (CJD)).

In 2011, the NNDSS core dataset included the following 5 mandatory data fields: unique record reference number; notifying state or territory; disease code; confirmation status and the date when the jurisdictional health department 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 the 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. These data, along with influenza enhanced data, are reported in individual annual reports. 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 2011. These 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 CJD in Australia has been conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) since its establishment in October 2003. CJD is a nationally notifiable disease and by June 2006, CJD was notifiable in all states and territories. Further surveillance information on CJD can be found in surveillance reports from the ANCJDR.⁵

Information on communicable disease surveillance is communicated through several avenues. The most up-to-date information on topics of interest is provided at the fortnightly teleconferences of the Communicable Diseases Network Australia (CDNA). A summary of these reports is available on the [CDNA web site](http://www.health.gov.au/internet/main/publishing.nsf/Content/cdnareport.htm) (<http://www.health.gov.au/internet/main/publishing.nsf/Content/cdnareport.htm>). The *Communicable Diseases Intelligence* (CDI) quarterly journal publishes surveillance data, annual surveillance reports, short reports, and articles on the epidemiology and control of communicable diseases.

Notification rates for each notifiable disease were calculated using the estimated 2011 mid-year resident population supplied by the Australian Bureau of Statistics (Appendix 1 and Appendix 2).⁶ 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 unless stated otherwise.

Notes on interpretation

This report is based on 2011 data from each state and territory, agreed upon in August 2012, and represents a snap shot of the year after duplicate records and incorrect or incomplete data were removed. Totals in this report may vary slightly from the totals reported in CDI quarterly publications.

Analyses in this report were based on the date of disease diagnosis in an attempt to estimate disease activity within the reporting period. The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. As considerable time may have elapsed between the onset and diagnosis dates for hepatitis B (unspecified), hepatitis C (unspecified) and tuberculosis, the earliest of specimen date, health professional notification date or public health unit notification receive date was used for these conditions.

Notified cases can only represent a proportion (the 'notified fraction') of the total incidence (Figure 1) and this has to be taken into account when interpreting NNDSS data. Moreover, the notified fraction varies by disease, by jurisdiction and over 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 List² was established, some diseases are not notifiable in all 8 jurisdictions (Table 1).

Changes in surveillance practices may have been introduced in some jurisdictions and not in others, and must be taken into consideration when comparing data between jurisdictions.

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

Figure 1: Communicable diseases notifiable fraction

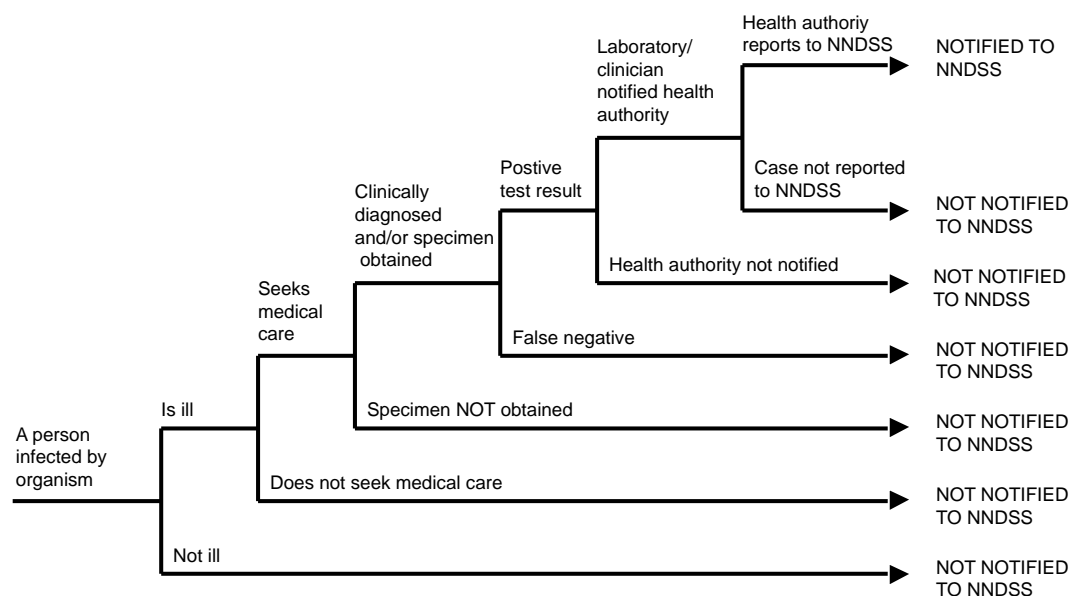


Table 1: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2011

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

Table 1 continued: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2011

Disease	Data received from
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
<i>Haemophilus influenzae</i> 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 disease (invasive)	All jurisdictions
Tuberculosis	All jurisdictions

* Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

NEC Not elsewhere classified.

Data completeness was assessed for cases' 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:⁷

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

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

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

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

9=Not stated

Notes on 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 in 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) as required.

The national surveillance case definitions and their review status are available from the [Australian Government Department of Health's web site](http://www.health.gov.au/casedefinitions) (<http://www.health.gov.au/casedefinitions>).

Results

There were 238,158 communicable disease notifications received by NNDSS in 2011 (Table 2)

In 2011, the most frequently notified diseases were sexually transmissible infections (95,456 notifications, 40.1% of total notifications), vaccine preventable diseases (81,872 notifications, 34.4% of total notifications), and gastrointestinal diseases (32,784 notifications, 13.8% of total notifications).

Table 2: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2011, by disease category rank order

Disease category	Number	%
Sexually transmissible infections	95,456	40.1
Vaccine preventable diseases	81,872	34.4
Gastrointestinal diseases	32,784	13.8
Bloodborne diseases	17,123	7.2
Vectorborne diseases	8,306	3.5
Other bacterial infections	1,928	0.8
Zoonoses	681	0.3
Quarantinable diseases	8	0.0
Total	238,158	100.0

There was an increase of 14% compared with the total number of notifications in 2010 but numbers were similar to those in 2009 (Figure 2). This increase in total notifications was largely due to the ongoing pertussis epidemic and higher than usual inter-season notifications of influenza.

Notifications and notification rates per 100,000 for each disease by state or territory, in 2011, are shown in Tables 3 and 4 respectively. Trends in notifications and rates per 100,000 for the period 2006 to 2011 are shown in Table 5.

Figure 2: Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2011

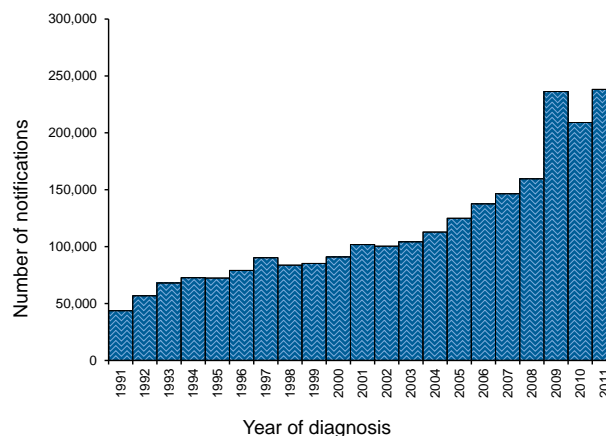


Table 3: Notifications of communicable diseases, Australia, 2011, by state or territory

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)*	2	31	4	46	9	13	67	18	190
Hepatitis B (unspecified)†	93	2,501	159	859	403	40	1,915	659	6,629
Hepatitis C (newly acquired)*	9	45	3	NN	33	27	163	120	400
Hepatitis C (unspecified)†	182	3,281	206	2,435	425	202	2,174	956	9,861
Hepatitis D	0	9	0	7	8	0	17	2	43
Gastrointestinal diseases									
Botulism	0	2	0	0	0	0	0	0	2
Campylobacteriosis	496	NN	160	5,134	2,121	864	6,766	2,176	17,717
Cryptosporidiosis	13	359	94	465	128	42	259	448	1,808
Haemolytic uraemic syndrome	0	4	1	1	3	0	4	0	13
Hepatitis A	3	57	3	25	6	4	34	12	144
Hepatitis E	2	20	0	6	0	0	8	4	40
Listeriosis	1	21	1	10	6	2	22	7	70
Salmonellosis	161	3,480	403	2,923	1,055	195	2,732	1,318	12,267
Shigellosis	9	131	77	63	34	2	94	84	494
STEC,VTEC‡	5	10	1	16	49	2	9	3	95
Typhoid	2	45	3	21	9	3	36	15	134
Quarantinable diseases									
Cholera	0	0	0	5	0	0	0	1	6
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	2	0	0	0	0	2
Sexually transmitted infections									
Chlamydial infection§,	1,261	20,495	2,630	18,649	5,128	1,779	19,184	11,674	80,800
Donovanosis	0	0	0	0	0	0	0	0	0
Gonococcal infection	128	2,880	1,956	2,960	445	19	1,879	1,820	12,087
Syphilis – congenital	0	3	0	4	0	0	0	0	6
Syphilis – all ,¶	33	730	89	553	47	26	862	223	2,563
Syphilis < 2 years duration	9	422	30	332	47	6	330	127	1,303
Syphilis > 2 years or unspecified duration†,	24	308	59	221	NN	20	532	96	1,260
Vaccine preventable diseases									
Diphtheria	0	0	1	3	0	0	0	0	4
<i>Haemophilus influenzae</i> type b	0	4	2	5	0	0	1	1	13
Influenza (laboratory confirmed)	270	5,700	597	10,409	4,738	364	3,208	1,863	27,149
Measles	21	90	5	17	4	0	39	17	193
Mumps	1	67	0	38	7	4	24	14	155
Pertussis	829	13,065	378	8,987	2,351	354	8,649	3,989	38,602
Pneumococcal disease (invasive)	27	530	129	341	143	47	427	243	1,887
Poliomyelitis	0	0	0	0	0	0	0	0	0

Table 3 continued: Notifications of communicable diseases, Australia, 2011, by state or territory

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases, cont'd									
Rubella	2	17	0	10	3	0	11	15	58
Rubella – congenital	0	0	0	0	0	0	0	0	0
Tetanus	0	1	0	1	0	0	0	1	3
Varicella zoster (chickenpox)	11	NN	148	302	477	34	688	434	2,094
Varicella zoster (shingles)	28	NN	186	75	1,614	202	993	901	3,999
Varicella zoster (unspecified)	99	NN	3	4,002	116	79	2,409	1,007	7,715
Vectorborne diseases									
Arbovirus infection (NEC)	0	0	1	9	0	0	14	0	24
Barmah Forest virus infection	2	459	63	872	130	2	187	155	1,870
Dengue virus infection	15	137	25	188	22	3	106	321	817
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0
Kunjin virus infection**	0	1	1	0	0	0	0	0	2
Malaria	3	77	23	137	4	9	95	63	411
Murray Valley encephalitis virus infection**	0	3	2	0	2	0	0	9	16
Ross River virus infection	8	577	184	1,220	979	7	1,312	879	5,166
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australia bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	6	0	30	0	0	2	1	39
Leptospirosis	1	40	2	157	2	1	11	3	217
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	19	0	1	0	1	58	6	85
Q fever	1	131	1	164	7	0	24	10	338
Tularaemia	0	0	0	0	0	2	0	0	2
Other bacterial diseases									
Legionellosis	4	95	5	45	40	7	74	78	348
Leprosy	0	3	0	0	1	0	3	1	8
Meningococcal infection††	2	72	4	61	21	10	50	21	241
Tuberculosis	20	470	33	223	73	17	371	124	1,331
Total	3,744	55,668	7,583	61,481	20,643	4,363	54,981	29,696	238,158

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months. South Australia does not provide data on unspecified syphilis cases.

‡ Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral 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 and the Australian Capital Territory also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

Table 4: Notification rates of nationally notifiable communicable diseases, Australia, 2011, by state or territory, per 100,000

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Bloodborne diseases									
Hepatitis (NEC)	–	–	–	–	–	–	–	–	–
Hepatitis B (newly acquired)*	0.5	0.4	1.7	1.0	0.5	2.5	1.2	0.8	0.8
Hepatitis B (unspecified)†	25.4	34.3	69.0	18.8	24.3	7.8	34.1	28.1	29.3
Hepatitis C (newly acquired)*	2.5	0.6	1.3	NN	2.0	5.3	2.9	5.1	2.2
Hepatitis C (unspecified)†	49.8	44.9	89.4	53.2	25.7	39.6	38.7	40.7	43.6
Hepatitis D	–	0.1	–	0.2	0.5	–	0.3	0.1	0.2
Gastrointestinal diseases									
Botulism	–	<0.1	–	–	–	–	–	–	<0.1
Campylobacteriosis	135.7	NN	69.5	112.1	128.1	169.2	120.4	92.6	115.7
Cryptosporidiosis	3.6	4.9	40.8	10.2	7.7	8.2	4.6	19.1	8.0
Haemolytic uraemic syndrome	–	0.1	0.4	<0.1	0.2	–	0.1	–	0.1
Hepatitis A	0.8	0.8	1.3	0.5	0.4	0.8	0.6	0.5	0.6
Hepatitis E	0.5	0.3	–	0.1	–	–	0.1	0.2	0.2
Listeriosis	0.3	0.3	0.4	0.2	0.4	0.4	0.4	0.3	0.3
Salmonellosis	44.0	47.7	174.9	63.8	63.7	38.2	48.6	56.1	54.2
Shigellosis	2.5	1.8	33.4	1.4	2.1	0.4	1.7	3.6	2.2
STEC,VTEC‡	1.4	0.1	0.4	0.3	3.0	0.4	0.2	0.1	0.4
Typhoid	0.5	0.6	1.3	0.5	0.5	0.6	0.6	0.6	0.6
Quarantinable diseases									
Cholera	–	–	–	0.1	–	–	–	<0.1	<0.1
Highly pathogenic avian influenza in humans	–	–	–	–	–	–	–	–	–
Plague	–	–	–	–	–	–	–	–	–
Rabies	–	–	–	–	–	–	–	–	–
Severe acute respiratory syndrome	–	–	–	–	–	–	–	–	–
Smallpox	–	–	–	–	–	–	–	–	–
Viral haemorrhagic fever	–	–	–	–	–	–	–	–	–
Yellow fever	–	–	–	<0.1	–	–	–	–	<0.1
Sexually transmitted infections									
Chlamydial infection§,	344.9	280.7	1141.6	407.2	309.6	348.5	341.3	496.9	357.2
Donovanosis	–	–	–	–	–	–	–	–	–
Gonococcal infection	35.0	39.4	849.1	64.6	26.9	3.7	33.4	77.5	53.4
Syphilis – congenital	–	<0.1	–	0.1	–	–	–	–	<0.1
Syphilis – all ,¶	9.0	10.0	38.6	12.1	2.8	5.1	15.3	9.5	11.3
Syphilis < 2 years duration	2.5	5.8	13.0	7.2	2.8	1.2	5.9	5.4	5.8
Syphilis > 2 years or unspecified duration†,	6.6	4.2	25.6	4.8	NN	3.9	9.5	4.1	6.0
Vaccine preventable diseases									
Diphtheria	–	–	0.4	0.1	–	–	–	–	<0.1
<i>Haemophilus influenzae</i> type b	–	0.1	0.9	0.1	–	–	<0.1	<0.1	0.1
Influenza (laboratory confirmed)	73.8	78.1	259.1	227.3	286.1	71.3	57.1	79.3	120.0
Measles	5.7	1.2	2.2	0.4	0.2	–	0.7	0.7	0.9
Mumps	0.3	0.9	–	0.8	0.4	0.8	0.4	0.6	0.7
Pertussis	226.7	178.9	164.1	196.2	141.9	69.3	153.9	169.8	170.7
Pneumococcal disease (invasive)	7.4	7.3	56.0	7.4	8.6	9.2	7.6	10.3	8.3
Poliomyelitis	–	–	–	–	–	–	–	–	–

Table 4 continued: Notification rates of nationally notifiable communicable diseases, Australia, 2011, by state or territory, per 100,00

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases, cont'd									
Rubella	0.5	0.2	–	0.2	0.2	–	0.2	0.6	0.3
Rubella – congenital	–	–	–	–	–	–	–	–	–
Tetanus	–	<0.1	–	<0.1	–	–	–	<0.1	<0.1
Varicella zoster (chickenpox)	3.0	NN	64.2	6.6	28.8	6.7	12.2	18.5	13.7
Varicella zoster (shingles)	7.7	NN	80.7	1.6	97.4	39.6	17.7	38.4	26.1
Varicella zoster (unspecified)	27.1	NN	1.3	87.4	7.0	15.5	42.9	42.9	50.4
Vectorborne diseases									
Arbovirus infection (NEC)	–	–	0.4	0.2	–	–	0.2	–	0.1
Barmah Forest virus infection	0.5	6.3	27.3	19.0	7.8	0.4	3.3	6.6	8.3
Dengue virus infection	4.1	1.9	10.9	4.1	1.3	0.6	1.9	13.7	3.6
Japanese encephalitis virus infection	–	–	–	–	–	–	–	–	–
Kunjin virus infection**	–	<0.1	0.4	–	–	–	–	–	<0.1
Malaria	0.8	1.1	10.0	3.0	0.2	1.8	1.7	2.7	1.8
Murray Valley encephalitis virus infection**	–	<0.1	0.9	–	0.1	–	–	0.4	0.1
Ross River virus infection	2.2	7.9	79.9	26.6	59.1	1.4	23.3	37.4	22.8
Zoonoses									
Anthrax	–	–	–	–	–	–	–	–	–
Australia bat lyssavirus	–	–	–	–	–	–	–	–	–
Brucellosis	–	0.1	–	0.7	–	–	<0.1	<0.1	0.2
Leptospirosis	0.3	0.5	0.9	3.4	0.1	0.2	0.2	0.1	1.0
Lyssavirus (NEC)	–	–	–	–	–	–	–	–	–
Ornithosis	–	0.3	–	<0.1	–	0.2	1.0	0.3	0.4
Q fever	0.3	1.8	0.4	3.6	0.4	–	0.4	0.4	1.5
Tularaemia	–	–	–	–	–	0.4	–	–	<0.1
Other bacterial diseases									
Legionellosis	1.1	1.3	2.2	1.0	2.4	1.4	1.3	3.3	1.5
Leprosy	–	<0.1	–	–	0.1	–	0.1	<0.1	<0.1
Meningococcal infection††	0.5	1.0	1.7	1.3	1.3	2.0	0.9	0.9	1.1
Tuberculosis	5.5	6.4	14.3	4.9	4.4	3.3	6.6	5.3	5.9

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months. South Australia does not provide data on unspecified syphilis cases.

‡ Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral 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 and the Australian Capital Territory also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

– A rate could not be calculated as there were no notifications.

Table 5: Notifications and notification rate for communicable diseases, Australia, 2006 to 2011, per 100,000

Disease	Number of notifications						Ratio	Notification rate per 100,000 population					
	2006	2007	2008	2009	2010	2011		2006	2007	2008	2009	2010	2011
Bloodborne diseases													
Hepatitis (NEC)	1	0	1	0	0	0	<0.1	<0.1	<0.1	<0.1	–	–	
Hepatitis B (newly acquired)*	291	296	259	241	228	190	0.7	263.0	1.4	1.2	1.1	1.0	
Hepatitis B (unspecified)†	6,168	6,783	6,444	7,015	6,960	6,629	1.0	6674.0	29.8	30.0	32.0	31.2	
Hepatitis C (newly acquired)*	437	379	363	398	401	400	1.0	395.6	2.6	2.1	2.3	2.2	
Hepatitis C (unspecified)†	11,689	11,675	10,956	10,871	10,916	9,861	0.9	11221.4	56.5	51.0	49.5	49.0	
Hepatitis D	29	33	41	35	34	43	1.3	34.4	0.1	0.2	0.2	0.2	
Gastrointestinal diseases													
Botulism	1	1	0	1	0	2	3.3	0.6	<0.1	<0.1	<0.1	–	
Campylobacteriosis	15,416	16,980	15,539	16,075	16,968	17,717	1.1	16,195.6	111.1	107.3	108.4	112.5	
Cryptosporidiosis	3,201	2,808	2,003	4,624	1,478	1,808	0.6	2,822.8	15.5	9.3	21.1	6.6	
Haemolytic uraemic syndrome	14	19	32	13	9	13	0.7	17.4	0.1	0.1	0.1	<0.1	
Hepatitis A	281	166	277	564	267	144	0.5	311.0	1.4	0.8	2.6	1.2	
Hepatitis E	24	18	44	33	37	40	1.3	31.2	0.1	0.2	0.2	0.2	
Listeriosis	61	50	68	92	71	70	1.0	68.4	0.3	0.3	0.4	0.3	
Salmonellosis	8,215	9,461	8,289	9,509	11,924	12,267	1.3	9,479.6	39.7	44.9	43.3	53.5	
Shigellosis	544	596	828	616	551	494	0.8	627.0	2.6	3.9	2.8	2.5	
STEC,VTEC†	67	105	98	128	80	95	1.0	95.6	0.3	0.5	0.6	0.4	
Typhoid	77	90	105	115	96	134	1.4	96.6	0.4	0.4	0.5	0.4	
Quarantinable diseases													
Cholera	3	4	4	5	3	6	1.6	3.8	<0.1	<0.1	<0.1	<0.1	
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	–	0.0	–	–	–	–	
Plague	0	0	0	0	0	0	–	0.0	–	–	–	–	
Rabies	0	0	0	0	0	0	–	0.0	–	–	–	–	
Severe acute respiratory syndrome	0	0	0	0	0	0	–	0.0	–	–	–	–	
Smallpox	0	0	0	0	0	0	–	0.0	–	–	–	–	
Viral haemorrhagic fever	0	0	0	0	0	0	–	0.0	–	–	–	–	
Yellow fever	0	0	0	0	0	2	–	0.0	–	–	–	<0.1	

Table 5 continued: Notifications and notification rate for communicable diseases, Australia, 2006 to 2011, per 100,000

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2006	2007	2008	2009	2010	2011			2006	2007	2008	2009	2010	2011
Sexually transmitted infections														
Chlamydia infection [§]	47,414	51,947	58,431	62,954	74,266	80,800	59,002.4	1.4	229.1	246.5	271.8	286.8	333.1	357.2
Donovanosis	6	3	2	1	1	0	2.6	<0.1	0.03	<0.1	<0.1	<0.1	<0.1	–
Gonococcal infection	8,598	7,646	7,679	8,044	10,020	12,087	8,397.4	1.4	41.5	36.3	35.7	36.6	44.9	53.4
Syphilis – congenital	11	7	6	3	3	6	6.0	1.0	0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Syphilis – all [¶]	2,209	2,778	2,697	2,731	2,398	2,563	2,562.6	1.0	10.7	13.2	12.5	12.4	10.8	11.3
Syphilis < 2 years duration	892	1,425	1,328	1,331	1,135	1,303	1,222.2	1.1	4.3	6.8	6.2	6.1	5.1	5.8
Syphilis > 2 years or unspecified duration	1,317	1,353	1,369	1,400	1,263	1,260	1,340.4	0.9	6.9	6.9	6.9	6.9	6.1	6.0
Vaccine preventable diseases														
Diphtheria	0	0	0	0	0	4	0.0	–	–	–	–	–	–	<0.1
<i>Haemophilus influenzae</i> type b	22	17	25	19	24	13	21.4	0.6	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)	3,322	10,585	9,178	59,018	13,467	27,149	19,114.0	1.4	16.0	50.2	42.7	268.9	60.4	120.0
Measles	125	12	65	105	69	193	75.2	2.6	0.6	0.1	0.3	0.5	0.3	0.9
Mumps	275	582	285	165	97	155	280.8	0.6	1.3	2.8	1.3	0.8	0.4	0.7
Pertussis	9,759	4,861	14,287	29,769	34,785	38,602	18,692.2	2.1	47.1	23.1	66.5	135.6	156.0	170.7
Pneumococcal disease (invasive)	1,448	1,468	1,628	1,554	1,639	1,887	1,547.4	1.2	7.0	7.0	7.6	7.1	7.4	8.3
Poliomyelitis	0	1	0	0	0	0	0.2	<0.1	–	<0.1	–	–	–	–
Rubella	59	34	36	27	44	58	40.0	1.5	0.3	0.2	0.2	0.1	0.2	0.3
Rubella – congenital	0	2	0	0	0	0	0.4	0.0	–	<0.1	–	–	–	–
Tetanus	3	3	4	3	2	3	3.0	1.0	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Varicella zoster (chickenpox)	NN	1,667	1,799	1,754	1,747	2,094	NN	NN	NN	18.6	19.6	11.8	11.6	13.7
Varicella zoster (shingles)	NN	1,562	2,326	2,718	2,985	3,999	NN	NN	NN	17.5	25.4	18.3	19.8	26.1
Varicella zoster (unspecified)	NN	4,284	4,413	6,784	7,145	7,715	NN	NN	NN	47.9	48.2	45.8	47.4	50.4

Table 5 continued: Notifications and notification rate for communicable diseases, Australia, 2006 to 2011, per 100,000

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2006	2007	2008	2009	2010	2011			2006	2007	2008	2009	2010	2011
Vectorborne diseases														
Arbovirus infection (NEC)	30	17	12	8	24	24	18.2	1.3	0.1	0.1	0.1	0.0	0.1	0.1
Barmah Forest virus infection	2,129	1,709	2,085	1,477	1,470	1,870	1,774.0	1.1	10.3	8.1	9.7	6.7	6.6	8.3
Dengue virus infection	189	314	560	1,406	1,220	817	737.8	1.1	0.9	1.5	2.6	6.4	5.5	3.6
Japanese encephalitis virus infection	0	0	1	0	0	0	0.2	<0.1	–	–	<0.1	–	–	–
Kunjin virus infection**	3	1	1	2	2	2	1.8	1.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Malaria	768	564	523	503	394	411	550.4	0.7	3.7	2.7	2.4	2.3	1.8	1.8
Murray Valley encephalitis virus infection**	1	0	2	4	0	16	1.4	11.4	<0.1	–	<0.1	<0.1	–	0.1
Ross River virus infection	5,529	4,175	5,659	4,787	5,152	5,166	5,060.4	1.0	26.7	19.8	26.3	21.8	23.1	22.8
Zoonoses														
Anthrax	1	1	0	0	1	0	0.6	<0.1	<0.1	<0.1	–	–	<0.1	–
Australia bat lyssavirus	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Brucellosis	50	37	45	32	21	39	37.0	1.1	0.2	0.2	0.2	0.1	0.1	0.2
Leptospirosis	145	108	111	142	131	217	127.4	1.7	0.7	0.5	0.5	0.6	0.6	1.0
Lyssavirus (NEC)	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Ornithosis	165	93	102	65	59	85	96.8	0.9	0.8	0.4	0.5	0.3	0.3	0.4
Q fever	411	448	378	310	329	338	375.2	0.9	2.0	2.1	1.8	1.4	1.5	1.5
Tularaemia	0	0	0	0	0	2	0.0	–	–	–	–	–	–	<0.1
Other bacterial diseases														
Legionellosis	349	306	272	301	299	348	305.4	1.1	1.7	1.5	1.3	1.4	1.3	1.5
Leprosy	7	14	11	4	12	8	9.6	0.8	<0.1	0.1	<0.1	<0.1	0.1	<0.1
Meningococcal infection††	317	305	286	259	229	241	279.2	0.9	1.5	1.4	1.3	1.2	1.0	1.1
Tuberculosis	1,209	1,133	1,214	1,313	1,312	1,331	1,236.2	1.1	5.8	5.4	5.6	6.0	5.9	5.9
Total	131,073	146,148	159,474	236,597	209,370	238,158								

Table 5 continued: Notifications and notification rate for communicable diseases, Australia, 2006 to 2011, per 100,000

*	Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.
†	Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months. South Australia does not provide data on unspecified syphilis cases.
‡	Infection with Shiga toxin/verotoxin-producing <i>Escherichia coli</i> .
§	Includes <i>Chlamydia trachomatis</i> identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral 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 and the Australian Capital Territory also report conjunctival cases.
NEC	Not elsewhere classified.
NN	Not notifiable.
–	A rate could not be calculated as there were no notifications.

The year in which diseases became notifiable to NNDSS in each jurisdiction is shown in Table 6.

Table 6: 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	1991	NN	1991 to present	WA do not report
Hepatitis B (newly acquired)	1995	1993	1993	1994	1993	1993	1993	1993	1994	1995 to present	ACT did not report 1994
Hepatitis B (unspecified)	1991	1991	2004	1994	1991	1991	1991	1991	1991	1991 to present	Qld do not report
Hepatitis C (newly acquired)	1995	1993	2005	NN	1993	1995	1997	1995	1995	1993 to present	Includes reports of incident hepatitis C, 1991 to 1994
Hepatitis C (unspecified)	1991	1991	1991	1991	1994	1991	1991	1991	1993	1995 to present	WA did not report 1999–2000
Hepatitis D	1999	1999	1999	1997	1999	1999	1999	1999	2001	1999 to present	
Gastrointestinal diseases											
Botulism	1992	1998	1998	1997	1993	1992	1992	1992	2001	1992 to present	
Campylobacteriosis	1991	NN	1991	1991	1991	1991	1991	1991	1991	1991 to present	NSW do not report
Cryptosporidiosis	2001	2001	2001	1996	2001	2001	2001	2001	2001	2001 to present	
Haemolytic uraemic syndrome	1999	1999	1999	1997	1999	1999	1999	1999	1999	1999 to present	
Hepatitis A	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Hepatitis E	1999	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	1991 to present	SA did not report 1991 NT did not report 1991–1993
Salmonellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Shigellosis	1991	2001	1991	1997	1991	1991	1991	1991	1991	1991 to present	
STEC, VTEC†	1999	1999	1999	2002	1999	1999	1999	1999	2001	1999 to present	
Typhoid‡	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Quantifiable diseases											
Cholera	1991	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	2004 to present	
Plague	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rabies	1993	1997	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Severe acute respiratory syndrome	2003	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Smallpox	2004	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Viral haemorrhagic fever	1993	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Yellow fever	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

Table 6 continued: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth						Period of national reporting	Exceptions to national reporting	
	ACT	NSW	NT	Qld	SA	Tas.			Vic.
Sexually transmissible infections									
Chlamydial infection (NEC)	1993	1991	1991	1991	1993	1991	1991	1993	NSW did not report 1994–1998
Donovanosis	1991	2002	1991	1991	2002	1993	1991	1991	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Gonococcal infection ^s	1991	1993	1991	1991	1991	1991	1991	1991	NSW did not report 1994–1998
Syphilis – all ^l	1991	1991	1991	1991	1991	1991	1991	1991	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Syphilis < 2 years	2004	2004	2004	2004	2004	2004	2004	2004	NSW did not report 1994–1998
Syphilis > 2 years or unspecified duration	2004	2004	2004	2004	–	2004	2004	2004	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Syphilis – congenital	2003	2003	2003	2003	2003	2003	2003	2003	South Australia do not report
Vaccine preventable diseases									
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991	NSW did not report 1994–1998
<i>Haemophilus influenzae</i> type b	1991	1991	1991	1991	1991	1991	1991	1991	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Influenza (laboratory confirmed)	2001	2001	2001	2001	2001	2001	2001	2001	NSW did not report 1994–1998
Measles	1991	1991	1991	1991	1991	1991	1991	1991	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Mumps	1992	1992	1995	1997–1998; 2002	1994	1995	1992	1994	NSW did not report 1994–1998
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Pneumococcal disease (invasive)	2001	2001	2001	1997	2001	2001	2001	2001	NSW did not report 1994–1998
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Rubella ^l	1991	1991	1993	1991	1993	1995	1992	1994	NSW did not report 1994–1998
Rubella – congenital	2003	2003	2003	1997	2003	2003	2003	2003	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Tetanus	1991	1991	1991	1985	1991	1991	1991	1991	NSW did not report 1994–1998
Varicella zoster (chickenpox)	2006	NN	2006	2006	2006	2006	2008	2006	NSW did not report 1994–1998
Varicella zoster (shingles)	2006	NN	2006	2006	2006	2006	2008	2006	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Varicella zoster (unspecified)	2006	NN	2006	2006	2006	2006	2008	2006	NSW did not report 1994–1998

Table 6 continued: Earliest notification year for which NNDSS contains disease data, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth							Period of national reporting	Exceptions to national reporting		
	ACT	NSW	NT	Qld	SA	Tas.	Vic.			WA	
Vectorborne diseases											
Barmah Forest virus infection	1995	1995	1997	1995	1995	1995	1995	1995	1995	1995 to present	
Dengue virus infection	1993	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	ACT did not report 1991–1992
Arbovirus infection (NEC)**†	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	Includes JEV, MVEV and Kunjin 1991–2000
Japanese encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Reported under MVEV in the ACT
Kunjin virus	2001	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Malaria	1991	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	2001 to present	Combined with Kunjin in the ACT
Ross River virus infection	1993	1993	1991	1991	1993	1993	1991	1991	1991	1993 to present	
Zoonoses											
Anthrax	2001	2001	2001	1991	2002	2001	2001	2001	2001	2001 to present	
Australian bat lyssavirus	2001	2001	2001	1998	2001	2001	2001	2001	2001	2001 to present	
Brucellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Leptospirosis	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Lyssavirus (NEC)	2001	2001	2001	1998	2001	2001	2001	2001	2001	2001 to present	
Ornithosis	1991	2001	1991	1992	1991	1991	1991	1991	1991	1991 to present	NSW did not report 1991–2000 Queensland did not report 1997–2001
Q fever	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Tularaemia	2004	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Other bacterial infections											
Legionellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Leprosy	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Meningococcal infection	1991	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Tuberculosis	1991	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.

† Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

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

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

|| Includes syphilis – congenital from 1991 to 2002.

¶ Includes rubella – congenital from 1991 to 2002.

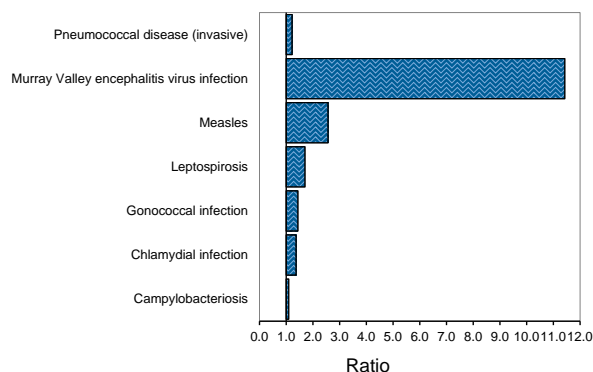
** Before 1997, includes Ross River virus infection, dengue virus infection and Barmah Forest virus infection.

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

NN Not notifiable

The major changes in communicable disease notifications in 2011 are shown in Figure 3 as the ratio of notifications in 2011 to the mean number of notifications for the previous 5 years. Pneumococcal disease (invasive), Murray Valley encephalitis virus (MVEV) infection, measles, leptospirosis, gonococcal infection, chlamydial infection and campylobacteriosis all surpassed the expected range (5-year mean plus 2 standard deviations). MVEV infection is very rare, and therefore any increase in case numbers leads to a large change in the ratio compared with the 5-year mean. Pertussis did not exceed the 5-year mean plus 2 standard deviations but experienced epidemic level activity in 2011.

Figure 3: Comparison of total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2011, with the previous 5-year mean



Data completeness

The case's sex and age at onset was complete in 99.9% of notifications (Table 7). In 2011, Indigenous status was complete in 80% of notifications, and varied by jurisdiction. Indigenous status was complete for 97% of data reported in the Northern Territory and Western Australia, and 93% in South Australia. In the remaining jurisdictions, less than 76% of data were complete for Indigenous status.

Data completeness on Indigenous status also varied by disease as summarised in Appendix 3. In 2011, CDNA set target thresholds of 95% completeness for key diseases and 80% completeness for the remainder of the notifiable diseases. There were 8 diseases for which notifications were 100% complete for Indigenous status. A further 22 diseases equalled or exceeded 80% completeness for Indigenous status. Of the 18 priority diseases agreed to by CDNA and the NSC in 2011 for improving Indigenous identification, seven had an Indigenous completeness that exceeded 95% (*Haemophilus influenzae* type b, hepatitis A, meningococcal infection, congenital syphilis, syphilis < 2 years duration, leprosy, and tuberculosis). The diseases for which there was less than 95% Indigenous completeness included hepatitis C (newly acquired), hepatitis B (newly acquired), dengue virus (DENV) infection, measles, gonococcal infection, pneumococcal disease (invasive), and shigellosis.

Table 7: Completeness of National Notifiable Diseases Surveillance System data received, Australia, 2011, by state or territory*

	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total notifications	3,743	55,668	7,583	61,481	20,643	4,363	54,981	29,696	238,158
Sex									
Unknown/ missing	0	182	0	7	14	0	218	0	421
Per cent complete	100	99.9	100	100	99.4	100	99.8	100	99.9
Age at onset									
Unknown/ missing	0	26	0	0	12	0	152	0	190
Per cent complete	100	100	100	100	99.4	100	99.9	100	99.9
Indigenous status									
Unknown/ missing	2,551	45,163	331	33,542	2,663	2,047	26,410	2,040	114,747
Per cent complete	75.3	69.7	96.7	60.9	93.1	76	76.7	97.3	80.4

* 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.

Bloodborne diseases

In 2011, the bloodborne viruses reported to the NNDSS were hepatitis B, C, and D. Both hepatitis B and C cases were 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 being 'newly acquired' was heavily reliant on public health follow-up, with the method and intensity of follow-up varying by jurisdiction and over time.

In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence or incidence. Testing policies⁸ 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 were also dependant on the level of public health follow-up, which is variable by jurisdiction and over time.

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

Notifications of HIV and AIDS diagnoses are reported directly to the Kirby Institute, formerly the National Centre in HIV Epidemiology and Clinical Research, which maintains the National HIV Registry and the National AIDS Registry. Information on national HIV/AIDS surveillance can be obtained from the [Kirby Institute web site](http://hiv.cms.med.unsw.edu.au/) (<http://hiv.cms.med.unsw.edu.au/>).

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.

Epidemiological situation in 2011

In 2011, there were 6,819 notifications of hepatitis B (both newly acquired and unspecified),

equating to a rate of 30.1 per 100,000 (Figure 4). The Northern Territory recorded the highest hepatitis B rate in 2011 (70.8 per 100,000), followed by Victoria (35.3 per 100,000) and New South Wales (34.7 per 100,000).

Between 2001 and 2011 unspecified hepatitis B rates decreased by 22% from 37.7 to 29.3 per 100,000 and newly acquired hepatitis B rates decreased from a rate of 2.2 to 0.8 per 100,000 (Figure 4). The continued decline in hepatitis B notifications may be attributed to the ongoing hepatitis B vaccination program introduced nationally for infants in 2000. Approximately 92% of the 2012 Australian birth cohort received the full primary course of the hepatitis B vaccine by 15 months of age.⁹ The decline may also be attributable to the adolescent program introduced in 1997.

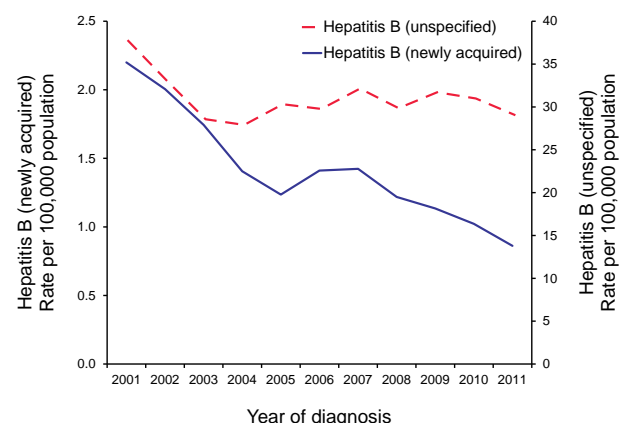
Newly acquired hepatitis B

Epidemiological situation in 2011

In 2011, there were 190 notifications of newly acquired hepatitis B (0.8 per 100,000), a 17% decrease compared with the 228 cases (rate of 1.0 per 100,000) reported in 2010 and a continuation of a downward trend in notifications. (Figure 4).

Nationally, the proportion of all hepatitis B cases in 2011 that were documented as newly acquired continued to trend downward and was 2.8%, compared with 3.2% in 2010 and 5.5% in 2001.

Figure 4: Notification rate for newly acquired hepatitis B* and unspecified hepatitis B,† Australia, 2001 to 2011, by year‡



* Data for newly acquired hepatitis B for the Northern Territory (2001–2004) includes some unspecified hepatitis B cases.

† Data for unspecified hepatitis B for all jurisdictions except the Northern Territory between 2001 and 2004.

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

The identification and classification of newly acquired hepatitis B is reliant upon public health follow-up of laboratory diagnoses, the extent of which varies between jurisdictions and over time.

Geographic distribution

The proportion of newly acquired infections compared with total hepatitis B infections varied substantially between jurisdictions, ranging from 1.2% in Tasmania and 24.5% in New South Wales.

Notification rates varied in states and territories: Tasmania (2.5 per 100,000), the Northern Territory (1.7 per 100,000), Victoria (1.2 per 100,000), Queensland (1.0 per 100,000), Western Australia (0.8 per 100,000), the Australian Capital Territory and South Australia (0.5 per 100,000) and New South Wales (0.4 per 100,000).

Age and sex distribution

Overall, notifications of newly acquired hepatitis B were more frequently reported amongst males. The highest rate of newly acquired hepatitis B infection was observed in males in the 30–34 and 35–39 year age groups (3.1 and 3.2 per 100,000 respectively) (Figure 5).

Between 2001 and 2011, most age group rates have been trending down with the most marked decrease occurring amongst the 20–29 year age range (Figure 6). Changes in hepatitis B notifications may be attributable to variations in levels of testing. Changes in immigration of people from countries where there is higher prevalence of hepatitis B may also impact on the number of cases diagnosed.¹⁰

Figure 5: Notification rate for newly acquired hepatitis B, Australia, 2011, by age group and sex

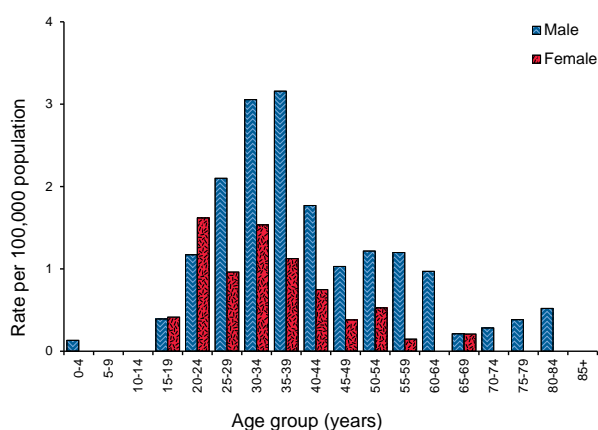
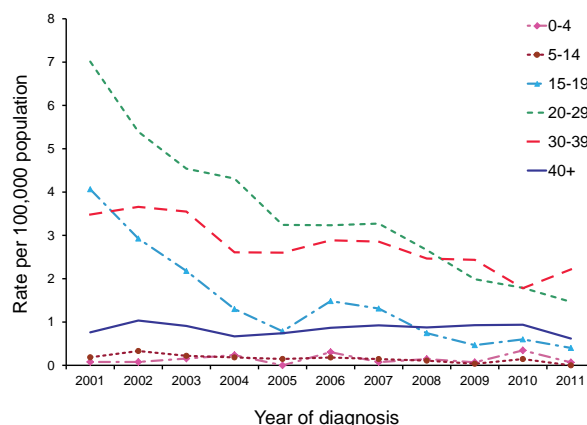


Figure 6: Notification rate for newly acquired hepatitis B,* Australia, 2001 to 2011, by year and age group



* Data for newly acquired hepatitis B for the Northern Territory (2001–2004) includes some unspecified hepatitis B cases.

Risk groups

Exposure histories were assessed for 126 of the 190 cases reported in 2011 (Table 8). In 2011, 72.2% (n=91) of these cases had at least 1 risk factor recorded, with the source of exposure not recorded or not determined for the remainder (Table 8). Injecting drug use remained the most frequently reported source of infection in 2011 (reported as a risk factor for 31% of cases) but has declined from 2007, when it was reported as a risk factor for 47% of cases. Skin penetration procedures were the next most frequently reported risk factor for infection in 2011 (34%), the majority of which were reported as tattoos.

Additional information was collected on the country of birth (COB) from all jurisdictions except Queensland. Of the 116 cases for which COB was reported, the majority occurred amongst Australian-born persons (69%, 80 cases) with the remaining 36 cases being born overseas.

Unspecified hepatitis B notifications

Epidemiological situation in 2011

In 2011, there were 6,629 notifications of unspecified hepatitis B infection, a rate of 29.3 per 100,000, compared with 6,878 cases (and a rate of 31.2 per 100,000) in 2010.

The overall rate of hepatitis B (unspecified) has been trending downward over the past 10 years with the majority of this decrease occurring between 2001 and 2004. Between 2006 and 2011

Table 8: Newly acquired hepatitis B cases, selected jurisdictions,* 2011, by sex and exposure category^{†,‡}

Exposure category	Number of exposure factors reported			Percentage of total cases* (n=126) %
	Male	Female	Total	
Injecting drug use	28	11	39	31.0
Imprisonment	8	0	8	6.3
Skin penetration procedures				
Tattoos	12	5	17	13.5
Ear or body piercing	4	3	7	5.6
Acupuncture	4	1	5	4.0
Healthcare exposure				
Surgical work	2	5	7	5.6
Major dental surgery work	4	3	7	5.6
Blood/tissue recipient (overseas)	0	1	1	0.8
Sexual contact – hepatitis B positive partner				
Opposite sex	6	6	12	9.5
Same sex	6	0	6	4.8
Other				
Household contact	3	3	6	4.8
Needlestick/biohazardous injury	4	0	4	3.2
Perinatal transmission	1	0	1	0.8
Other	11	1	12	9.5
Cases with at least 1 risk factor	65	26	91	72.2
Undetermined	2	1	3	2.4
Unknown (not recorded)	18	14	32	25.4
Total exposure factors reported [†]	93	39	132	–
Total number of cases	85	41	126	–

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

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

‡ Analysis and categorisation of these exposures are subject to interpretation and may vary.

§ The denominator used to calculate the percentage is based on the total number of cases from all jurisdictions (New South Wales, the Northern Territory, the Australian Capital Territory, Tasmania, South Australia and Victoria). As more than 1 exposure category for each notification could be recorded, the total percentage does not equate to 100%.

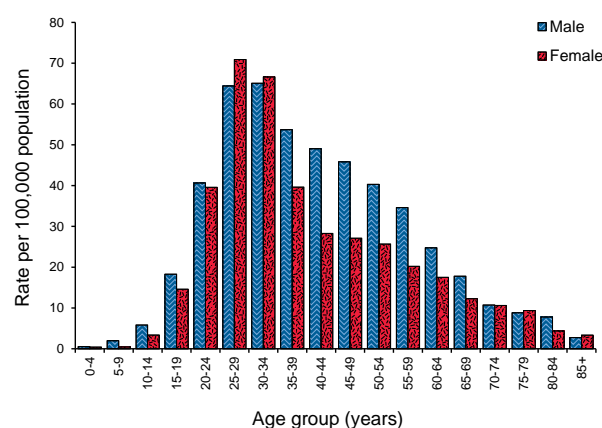
|| Includes both occupational and non-occupational exposures.

the rate has remained relatively stable with an average annual rate of 31 per 100,000 during this time. (Figure 4).

Age and sex distribution

In 2011, the overall male rate (32.2 per 100,000) was higher than for females (26.0 per 100,000), a rate ratio of 1.2:1, but females had the highest age specific rate amongst those in the 25–29 year age group (71 per 100,000) compared with the highest age specific rate amongst males of 65 per 100,000 in the 30–34 years age group (Figure 7).

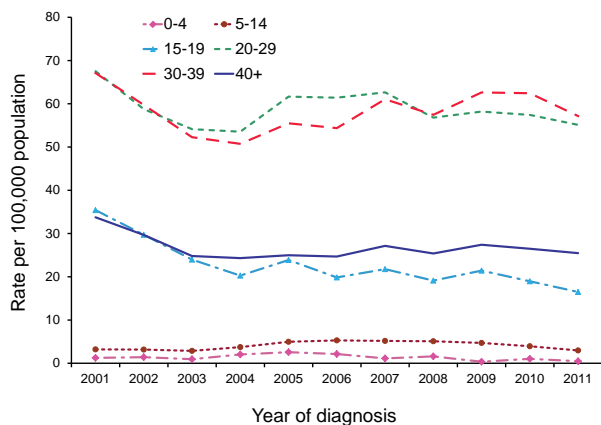
Rates of hepatitis B unspecified have declined across all age groups since 2001 with the majority of this decrease occurring in the first 3 years before stabilising (Figure 8). The biggest decrease

Figure 7: Notification rate for unspecified hepatitis B,* Australia, 2011, by age group and sex

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

(53%) occurred amongst the 15–19 year age group declining from a rate of 35 per 100,000 in 2001 to 16.5 per 100,000 in 2011.

Figure 8: Notification rate for unspecified hepatitis B,*† Australia, 2001 to 2011, by year and age group



* Data for hepatitis B (unspecified) from all states except the Northern Territory between 2001 and 2004.

† Excludes notifications for whom age was not reported.

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 month 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. Although initial infection with the hepatitis C virus is asymptomatic or mildly symptomatic in more than 90% of cases, approximately 50%–80% of cases will go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.⁴

Epidemiological situation in 2011

Between 2001 and 2011, total hepatitis C notification rates declined by 51% (93 to 45 per 100,000), with the greatest reductions observed in the earlier years, (a 16% decline between 2001 and 2002) (Figure 9). In 2011, it was estimated that 304,000

people living in Australia had been exposed to the hepatitis C virus. Of these, approximately 179,900 had chronic hepatitis C infection and early liver disease, 49,500 had chronic hepatitis C infection with moderate liver disease, 6,300 were living with hepatitis C related cirrhosis and 77,300 had cleared their infection.⁴

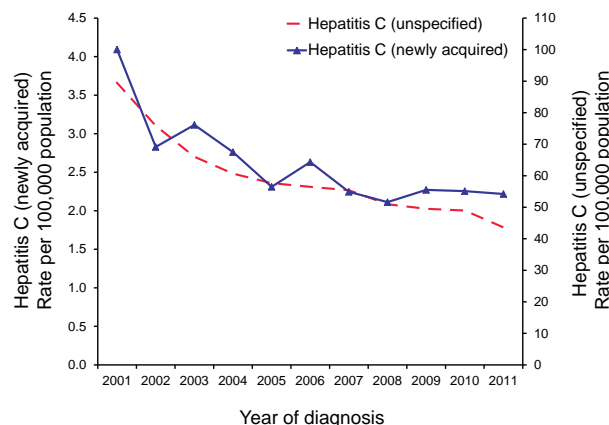
Newly acquired hepatitis C notifications

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.

Epidemiological situation in 2011

There were 400 notifications in 2011 compared with 401 in 2010, giving a rate of 2.2 per 100,000 (Figure 9). Of all hepatitis C cases in 2011, 3.9% were identified as newly acquired infections, which is comparable with previous years.

Figure 9: Notification rate for newly acquired hepatitis C* and unspecified hepatitis C,† Australia, 2001 to 2011



* Data for newly acquired hepatitis C from all states and territories except Queensland 2001–2011 and the Northern Territory 2001–2002.

† Data for unspecified hepatitis C provided from Queensland (2001–2011) and the Northern Territory (2001–2002) includes both newly acquired and unspecified hepatitis C cases.

Geographic distribution

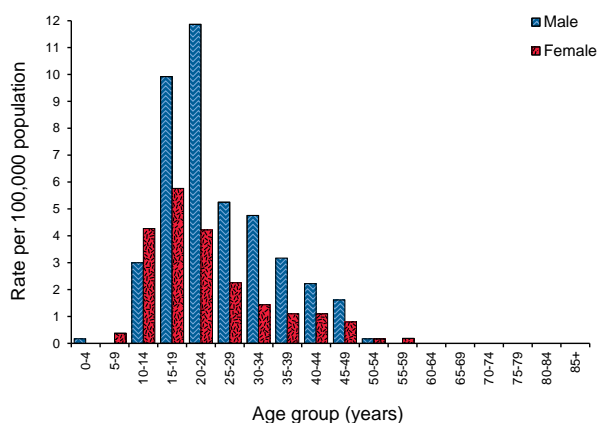
The proportion of infections that were newly acquired compared with total hepatitis C diagnoses varied substantially between the states and territories ranging from 1.4% in the Northern Territory to 11.8% in Tasmania. The highest rates of newly acquired hepatitis C infection were reported in Tasmania (5.3 per 100,000), followed by Western Australia (5.1 per 100,000) and Victoria

(2.9 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 between 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.

Age and sex distribution

The male to female ratio was 2.1:1. Age group specific rates for males were highest in the 20–24 year age group followed by the 15–19 year age group (Figure 10). Age group specific rates for females were highest in the 15–19 year, 10–14 year and 20–24 year age groups (Figure 10).

Figure 10: Notification rate for newly acquired hepatitis C, Australia,* 2011, by age group and sex



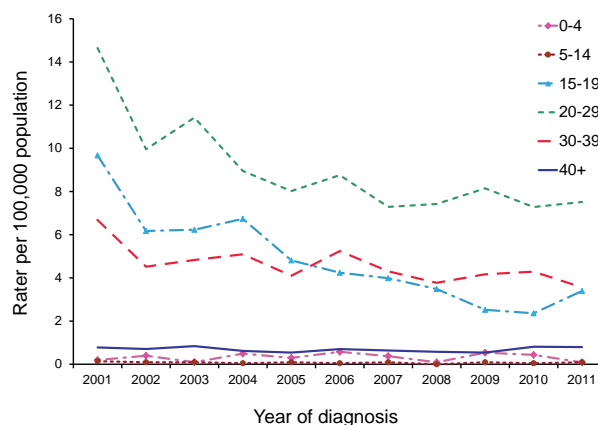
* Data from all states and territories except Queensland.

Between 2001 and 2011, rates of newly acquired hepatitis C infection in the 15–19 year, 20–29 year and 30–39 year age groups have been trending down (Figure 11). Rates amongst other age groups have remained relatively stable over the same period.

Risk groups

Exposure history for all newly acquired hepatitis C cases reported in 2011 was assessed from all jurisdictions except Queensland (Table 9). In 2011, 72% of these cases had at least 1 risk factor recorded, with the source of exposure not recorded or unable to be determined for the remainder of these cases. Approximately 60% of cases had a history of injecting drug use (62% of which reported injecting drug use in the 24 months prior to diagnosis). Skin penetration procedures and imprisonment accounted for approximately 32% and 21% of reported risk

Figure 11: Notification rate for newly acquired hepatitis C, Australia,* 2001 to 2011, by age group and year



* Data from all states and territories except Queensland (2001–2011) and the Northern Territory (2001–2002).

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 2010 found that the prevalence of hepatitis C based on hepatitis C antibody detection was 22%, a decrease compared with the 35% reported in 2007.¹¹

Unspecified hepatitis C notifications

Epidemiological situation in 2011

In 2011, there were 9,861 notifications of unspecified hepatitis C infections, a rate of 43.6 per 100,000 compared with 10,916 cases in 2010 and a rate of 49.0 per 100,000. This continues a downward trend and represents a 51% decline compared with 2001 when the rate was 89.5 per 100,000 (Figure 9).

Several factors may account for the decrease: changes in surveillance practices, including duplicate notification checking; a gradual decline in the prevalent group of hepatitis C cases accumulated prior to the introduction of hepatitis C testing in the early 1990s; general reductions in risk behaviours relating to injecting drug use, particularly amongst young people; and increased access to sterile injecting equipment through need exchange programs.^{10–13}

Geographic distribution

In 2011, the Northern Territory continued to have the highest rate of unspecified hepatitis C infections (89.4 per 100,000) followed by Queensland (53.2 per 100,000) and the Australian Capital Territory (49.8 per 100,000), noting that Queensland's rate includes both newly acquired and unspecified cases. The lowest rate was in South Australia (25.7 per 100,000).

Table 9: Newly acquired hepatitis C notifications, selected jurisdictions,* 2011, by sex and exposure category^{†,‡}

Exposure category	Number of cases with exposure factor			Percentage of total cases*§(n=400) %
	Male	Female	Total	
Injecting drug use	175	65	240	60.0
Imprisonment	80	5	85	21.3
Skin penetration procedures				
Tattoos	50	12	62	15.5
Ear or body piercing	23	17	40	10.0
Acupuncture	3	2	5	1.3
Healthcare exposure				
Surgical work	6	4	10	2.5
Major dental surgery work	10	1	11	2.8
Blood/tissue recipient (overseas)	1	0	1	0.3
Sexual contact – hepatitis C positive partner				
Opposite sex	26	20	46	11.5
Same sex	1	1	2	0.5
Other				
Household contact	13	7	20	5.0
Needlestick/biohazardous injury	6	3	9	2.3
Perinatal transmission	15	5	20	5.0%
Other	15	7	22	5.5
Cases with at least 1 risk factor	207	82	289	72.3
Undetermined	3	3	6	1.5
Unknown (not recorded)	60	45	105	26.3
Total exposure factors reported [†]	424	149	573	–
Total number of cases	270	130	400	–

* Includes diagnoses in all states and territories except Queensland as newly acquired cases are reported as unspecified cases

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

‡ Analysis and categorisation of these exposures are subject to interpretation and may vary.

§ The denominator used to calculate the percentage is based on the total number of notifications from all jurisdictions, except Queensland. As more than 1 exposure category for each case could be recorded, the total percentage does not equate to 100%.

|| Includes both occupational and non-occupational exposures.

Age and sex distribution

The male to female ratio remained consistent with historical trends at 1.8:1 in 2011. Amongst males, rates were highest across age groups between 30 and 54 years ranging from 102 to 118 per 100,000. Similarly, rates for females were highest amongst adults in the 30–34 year age group (64 per 100,000) followed by the 25–29 year age group (57 per 100,000) and the 35–39 year age group (56 per 100,000) (Figure 12).

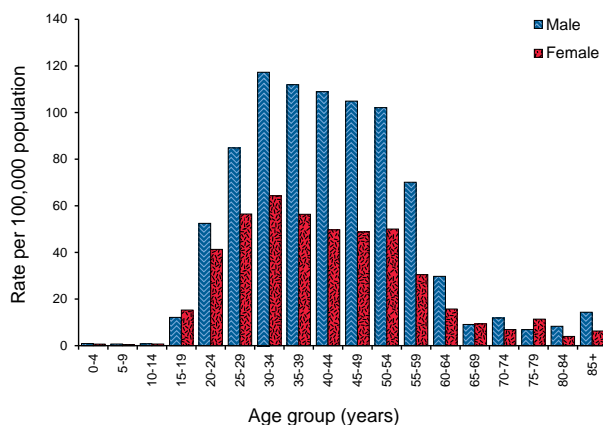
Between 2001 and 2011 the rate of unspecified hepatitis C has declined in all age groups with the biggest decreases occurring in the 15–19 year (81%), 20–29 year (70%) and the 30–39 year (50%) age

groups; the majority of this decline occurred in the early part of the decade (Figure 13). Trends in the 0–4, 5–14 and the 40 years or over age groups have remained relatively stable over this time (Figure 13).

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 superinfection with chronic hepatitis B infection.¹⁴ The modes of hepatitis D transmission are similar to those for hepatitis B. In countries with low hepatitis B prevalence, injecting drug users are the main group at risk for hepatitis D.

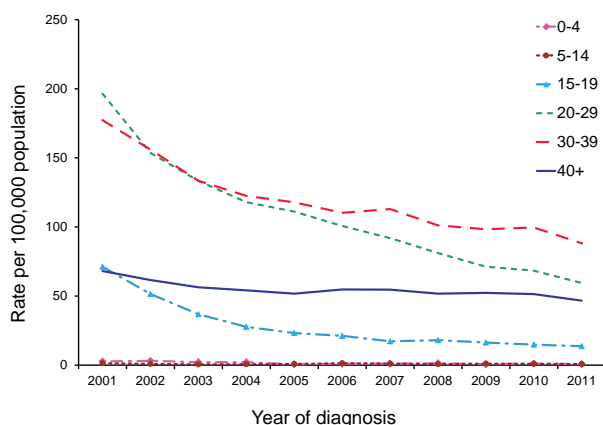
Figure 12: Notification rate for unspecified hepatitis C,*† Australia, 2011, by age group and sex



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

† Excludes notifications for whom age and/or sex were not reported.

Figure 13: Notification rate for unspecified hepatitis C,*† Australia, 2001 to 2011, by age group



* Data provided from Queensland (2001–2011) and the Northern Territory (2001–2002) includes both newly acquired and unspecified hepatitis C cases.

† Excludes notifications for whom age was not reported.

Epidemiological situation in 2011

In Australia, the rate of hepatitis D remains low. In 2011, there were 43 notifications of hepatitis D, a rate of 0.2 per 100,000. Over the past 5 years, the number of notifications of hepatitis D has remained relatively stable with an average of 37 cases notified per year (range 33–43).

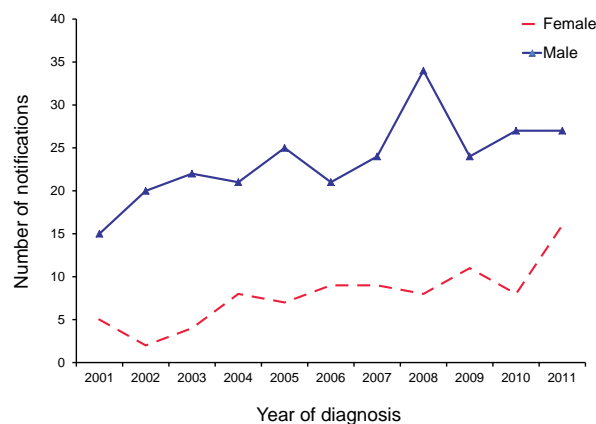
Geographic distribution

Victoria reported the highest number of cases (n=17) followed by New South Wales (n=9), South Australia (n=8), Queensland (n=7) and Western Australia (n=2).

Sex distribution

The male to female ratio in 2011 was 1.7:1 which was lower than the average ratio of 3.0:1 in the preceding 5 years (Figure 14).

Figure 14: Notifications of hepatitis D, Australia, 2001 to 2011, by sex



Gastrointestinal diseases

In 2011, gastrointestinal diseases notified to NNDSS and discussed in this section were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga toxin-producing *Escherichia coli* (STEC) infections and typhoid.

Overall notifications of gastrointestinal diseases increased from 31,483 in 2010 to 32,784 in 2011. Notifications of typhoid and *Campylobacter* infections were notably higher compared with the 5-year historical mean (exceeded the mean by more than 2 standard deviations).

Surveillance system overview

The Australian Government established OzFoodNet—Australia's enhanced foodborne disease surveillance system—in 2000 as a collaborative network of epidemiologists and microbiologists who conduct enhanced surveillance, epidemiological outbreak investigations and applied research into foodborne disease across Australia. OzFoodNet's mission is to apply concentrated effort at the national level to investigate and understand foodborne disease, to describe its epidemiology more effectively and to identify ways to minimise foodborne illness in Australia. The data and results summarised in the following sections will be reported in more detail in the OzFoodNet annual report 2011.

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.¹⁴

Epidemiological situation in 2011

There were 2 notifications of botulism in 2011. Both were infant botulism. There were no notifications reported in 2010 and 1 case reported in 2009.

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.¹⁴ Campylobacteriosis is notifiable in all Australian jurisdictions, except New South Wales.

Epidemiological situation in 2011

Campylobacteriosis was the most frequently notified enteric infection with 17,717 notifications; a rate of 116 per 100,000. This is an increase of 4% on the number of notifications received for 2010 (n=16,969) and a 9% increase on the 5-year historical mean (n=16,196). Notification rates ranged from 69.5 per 100,000 in the Northern Territory to 169.2 per 100,000 in Tasmania.

Age and sex distribution

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 year age group (221.6 and 165.2 per 100,000, respectively) with secondary peaks occurring in the 20–24 year and 70 years or over age groups (Figure 15).

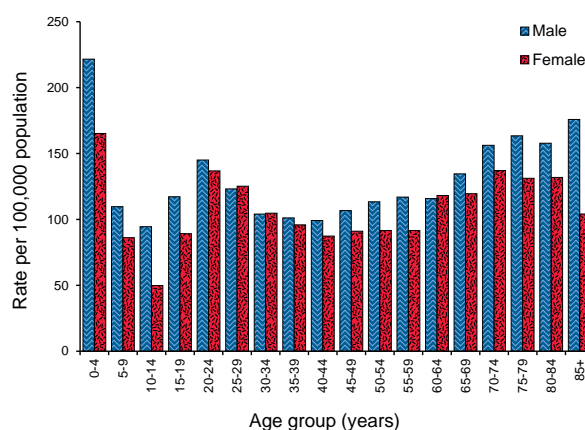
Cryptosporidiosis

Cryptosporidiosis is a parasitic infection characterised by abdominal cramping and usually large-volume watery diarrhoea. Ingesting contaminated water, typically from a recreational source like a community swimming pool or lake, is a major risk factor for infection.¹⁴

Epidemiological situation in 2011

In 2011, there were 1,808 notifications of cryptosporidiosis; a national rate of 8 per 100,000. This represents a 22% increase over the 1,479 notifications reported in 2010; however it is below the

Figure 15: Notification rate for campylobacteriosis, Australia, 2011, by age group and sex



5-year historical mean of 2,823 notifications. Notification rates ranged from 3.6 per 100,000 in the Australian Capital Territory to 40.8 per 100,000 in the Northern Territory.

Age and sex distribution

Notifications for cryptosporidiosis were most frequently in the 0–4 year age group (43%, n=780). Of these, 57% (n=446) were male.

Haemolytic uraemic syndrome

HUS is a rare but serious illness that is characterised by acute renal impairment, and results in chronic complications in 40% of cases.¹⁴ Not all diagnoses of HUS are related to enteric pathogens, but Australian cases are commonly associated with STEC infection.¹⁵

Epidemiological situation in 2011

In 2011, there were 13 notifications of HUS compared with 9 in 2010 and a mean of 17.4 notifications per year between 2006 and 2010.

Age and sex distribution

The median age of notified cases of HUS between 2006 and 2011 was 11 years (range 0–89 years). Cases were most frequently reported amongst children in the 0–4 year age group (n=38).

Hepatitis A

Hepatitis A is an acute viral infection primarily of the liver that can develop into chronic liver disease including liver failure. Infection is usually spread by person-to-person transmission via the faecal-oral route but can be foodborne or waterborne.¹⁴

Epidemiological situation in 2011

There were 144 notifications of hepatitis A in Australia; a rate of 0.65 notifications per 100,000. This was a 46% decrease in the number of cases compared with the 267 notifications in 2010.

Age and sex distribution

Hepatitis A was most frequently notified amongst the 25–34 year age range (n=40) in 2011. The median age of notified cases was 29 years (range 0–97 years), and 59% (n=85) were male.

Indigenous status

Indigenous status was known for 94% (135/144) of notified cases of hepatitis A. Of these, 2 cases were identified as being of Indigenous origin.

Place of acquisition

Overseas travel was the primary risk factor for notified cases in 2011. Infection was acquired overseas in 68% (n=96) of notified cases, compared with 54% (n=143) in 2010.

In 2011, 39 notified cases were locally acquired. This was a decrease from 2010 where 111 notified cases were locally acquired (Table 10). In 2009–2010 an outbreak associated with the consumption of semi-dried tomatoes contributed to an increase in locally acquired hepatitis A cases in both 2009 and 2010.¹⁶

Hepatitis E

Hepatitis E is an acute viral infection primarily of the liver that is transmitted by the faecal-oral route, most often via food or water.¹⁴ The infection is usually acquired overseas amongst travellers to endemic areas.

Epidemiological situation in 2011

There were 40 notifications of hepatitis E in 2011, compared with a 5-year historical mean of 31.2 notifications.

Age and sex distribution

Hepatitis E was most frequently notified amongst the 20–39 year age group (n=27), the median age of notified cases was 29 years (range 5–63 years), and 70% (n=28) were male.

Place of acquisition

Hepatitis E in Australia is associated with overseas travel. In 2011, 80% of cases (n=32) were known to have been acquired overseas, and of those, 66% (n=21) were acquired in India. The place of acquisition for the remaining 8 cases was not supplied or was unknown. No cases were reported to have been locally-acquired.

Listeriosis

Invasive listeriosis is caused by a bacterial infection that commonly affects the elderly or immunocompromised, and typically occurs amongst people with serious 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.

Epidemiological situation in 2011

There were 70 notifications of invasive *Listeria monocytogenes* infection in 2011 compared with a 5-year mean of 68 notifications. This represented a national rate of 0.3 per 100,000.

Age and sex distribution

Notifications for listeriosis were highest in the 80–84 year age group (23%, n=16), and 59% (n=41) of notified cases were male (Figure 16).

Enhanced surveillance in 2011

OzFoodNet collects enhanced surveillance data on all notified cases of listeriosis in Australia. Enhanced surveillance commenced in 2010. It collects detailed information on the characterisation

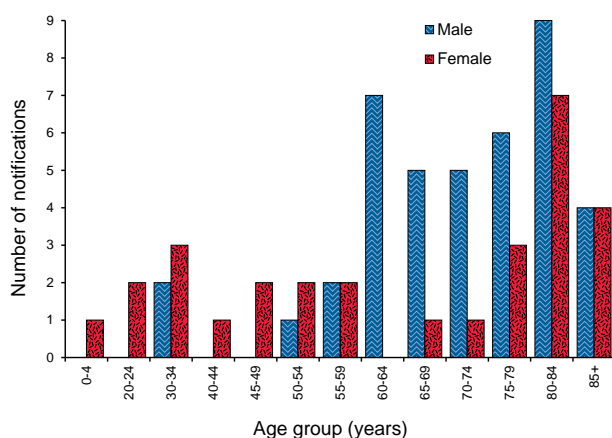
Table 10: Notifications of hepatitis A, Australia, 2006 to 2011, by place of acquisition

Year	Locally acquired		Overseas acquired		Unknown		Total
	n	%	n	%	n	%	
2006	164	58.4	47	16.7	70	24.9	281
2007	63	38.0	50	30.1	53	31.9	166
2008	91	32.9	82	29.6	104	37.5	277
2009	349	61.9	69	12.2	146	25.9	564
2010	111	41.6	143	53.6	13	4.9	267
2011	39	27.1	96	66.7	9	6.3	144

of *Listeria monocytogenes* isolates by molecular subtyping methods, food histories and exposure data on all notified listeriosis cases in Australia. The overall aim of this enhanced surveillance is to enable timely detection of illness and subsequent public health response.¹⁵

Analysis of the enhanced data is covered in the OzFoodNet annual reports from 2010 onwards.

Figure 16: Notifications for listeriosis, Australia, 2011, by age group and sex



Salmonellosis (non-typhoidal)

Salmonellosis is a bacterial disease characterised by the 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.¹⁴

Epidemiological situation in 2011

There were 12,267 notifications of salmonellosis in Australia in 2011; a rate of 54.2 notifications per 100,000, compared with the 5-year historical mean of 9,479.8 notifications. In 2011, notifications continued to rise with a 2.9% increase over the 11,924 notifications in 2010. The number of notifications for 2011 was the highest recorded in NNDSS since 1991. Notification rates ranged from 38.2 per 100,000 in Tasmania to 174.9 per 100,000 in the Northern Territory.

Age and sex distribution

In 2011, 51% (n=6,213) of notifications were in females, with the greatest proportion of notifications in the 0–4 year age group (25%, n=3,118).

Shigellosis

Shigellosis is a 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 through contaminated food.¹⁴

Epidemiological situation in 2011

There were 494 notifications of shigellosis in 2011; a national rate of 2.2 per 100,000, with notifications being less than the 5-year historical mean of 627 notifications. As in previous years, the highest notification rate was in the Northern Territory (33.4 per 100,000).

Age and sex distribution

Notifications for shigellosis were highest in the 0–4 year age group (21%, n=102), and 55% (n=270) of all notified cases were male.

Indigenous status

Information on Indigenous status was available for 89% (n=429) of shigellosis notifications. This proportion varied by state or territory, with New South Wales, Queensland and Victoria 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 55% (114/206).

Place of acquisition

Twenty-seven per cent (n=133) of notified cases of shigellosis were reported as being acquired overseas. The most frequently reported countries of acquisition for imported cases were Indonesia (33%, n=43) and India (17%, n=23).

Shiga toxin-producing *Escherichia coli* infections

Shiga toxin-producing *Escherichia coli* are types of toxin-producing *E. coli* that cause diarrhoeal illness in humans. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact or from environmental exposures. Severe illness can progress to HUS. Children under 5 years of age are most frequently diagnosed with infection and are at greatest risk of developing HUS.¹⁴

Epidemiological situation in 2011

There were 95 notifications of STEC in Australia in 2011; a rate of 0.4 per 100,000 population. Detection of STEC infection is strongly influenced

by jurisdictional practices regarding the screening of stool specimens.¹⁵ In South Australia, and more recently the Australian Capital Territory, single pathology providers are participating in screening studies of bloody stools using polymerase chain reaction (PCR) for genes coding for Shiga toxins and other virulence factors. Notification rates for these jurisdictions are the highest in the country (Table 3). These differences mean that meaningful comparison of notification data by jurisdiction and over time are not valid.

Age and sex distribution

In 2011, 57% (n=54) of notified STEC cases were male. The median age of notified cases was 26 years (range 0–85 years).

Typhoid

Typhoid is a disease caused by *S. enterica* serotype Typhi. The transmission mode is the same as for salmonellosis, however typhoid differs in that humans are the reservoir for the bacterium.¹⁴

Epidemiological situation in 2011

There were 134 notifications of typhoid (0.6 per 100,000) in 2011, compared with the 5-year historical mean of 97 cases. This was a 42% increase compared with the 96 notifications in 2010.

Age and sex distribution

Typhoid was most frequently notified amongst the 20–34 year age range (n=57), the median age of notified cases was 25 years (range 1–88 years), and 60% (n=81) were male.

Place of acquisition

As in previous years, overseas travel was the primary risk factor for notified cases of typhoid in 2011, with 87% (n=117) of notified cases known to have been acquired overseas. India continues to be the most frequently reported country of acquisition, accounting for 50% (n=67) of overseas-acquired cases in 2011.

Quarantinable diseases

Human diseases covered by the Quarantine Act 1908, and notifiable in Australia and to the WHO in 2011 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean–Congo). These diseases are of international public health significance.

Travellers are advised to seek information on the risk of contracting these diseases at their destinations and to take appropriate measures to avoid infection. More information on quarantinable diseases and travel health can be found on the [Department of Health's web site](http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm) (www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm) and the [Department of Foreign Affairs and Trade's Smartraveller web site](http://www.smartraveller.gov.au) (www.smartraveller.gov.au).

There were no cases of plague, rabies, smallpox, SARS, HPAIIH or viral haemorrhagic fevers reported in Australia in 2011. While there were notifications of imported cases of cholera (n=6) and yellow fever (n=2) in 2011, Australia remains free of all the listed quarantinable diseases (Table 11).

Table: 11 Australia's status for human quarantinable diseases, 2011

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
Plague	Free	Last case recorded in Australia in 1923 ¹⁷
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990 ¹⁸
Smallpox	Free	Last case recorded in Australia in 1938, last case world-wide in 1977, declared eradicated by the World Health Organization 1980 ^{19, 20}
Yellow fever	Free	Two cases in 2011 are the first recorded, related to overseas travel
SARS	Free	Last case recorded in Australia in 2003 ²¹
HPAIIH	Free	No cases recorded ²²
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 6 notifications of cholera in Australia in 2011, five from Queensland and one from Western Australia. The 5 cases notified in Queensland all acquired their infection in Papua New Guinea and were overseas residents, while the case in Western Australia was acquired in the Philippines. There were 19 cases of cholera in Australia between 2006 and 2010 (Table 5).

All cases of cholera reported since the commencement of the NNDSS in 1991 were acquired outside Australia except for 1 case of laboratory-acquired cholera in 1996²³ and 3 cases in 2006 linked to imported whitebait.²⁴

Yellow fever virus infection

There were 2 notifications of yellow fever in 2011, both from Queensland. The cases had recently returned from travel to yellow fever endemic areas (one from Colombia and the other from Ghana), were IgM positive, had a clinically-compatible illness and had received yellow fever vaccine in the 3 months prior to onset. Treating clinicians considered that both were likely to be vaccine related, but met the national case definition, and the possibility that they were true cases could not be excluded.

Sexually transmissible infections

Introduction

In 2011, 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.

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,^{25,26} 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, with low testing rates resulting in an underestimation of disease and increased testing potentially causing an increase in notifications.²⁷ For some diseases, changes in surveillance practices may also need to be taken into account when interpreting national trends.

It is important to note that data is reported by diagnosis date, a derived field, with the exception of syphilis in Queensland which is reported by notification receive date. These data may not be directly comparable with data in state and territory communicable disease reports, which report by onset date or notification date, but the trends that they highlight should be comparable.

Direct age standardised notification rates, using the method described by the Australian Institute of Health and Welfare²⁸ were calculated for Aboriginal and Torres Strait Islander and non-Indigenous notifications for jurisdictions that had Indigenous status data completed for more than 50% of notifications over the period 2006–2011. Where the Indigenous status of a case 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 Aboriginal and Torres Strait Islander populations. 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.²⁹

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).³⁰ 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

Genital chlamydia infection is caused by the bacterium *Chlamydia trachomatis* serogroups D–K. The infection is asymptomatic in approximately 1%–25% of sexually active men and up to 70% of sexually active women.¹⁴ Men with asymptomatic infection act as carriers transmitting the infection while only rarely suffering from long term health problems. However, in women there is a high risk of developing severe health conditions as a result of the infection, including infertility and pelvic inflammatory disease.^{14,31}

Epidemiological situation in 2011

Chlamydial infection continued to be the most commonly notified disease in 2011. Since chlamydial infection became a nationally notifiable disease in 1991 (1997 in New South Wales), the rate has increased in each consecutive year. In

2011, there were 80,800 notifications of chlamydial infection, equating to a rate of 357.2 per 100,000. This represents an increase of 7.2% compared with the rate reported in 2010 (333.1 per 100,000). Between 2006 and 2011, chlamydial infection rates increased by 56%, from 229.1 to 357.2 per 100,000.

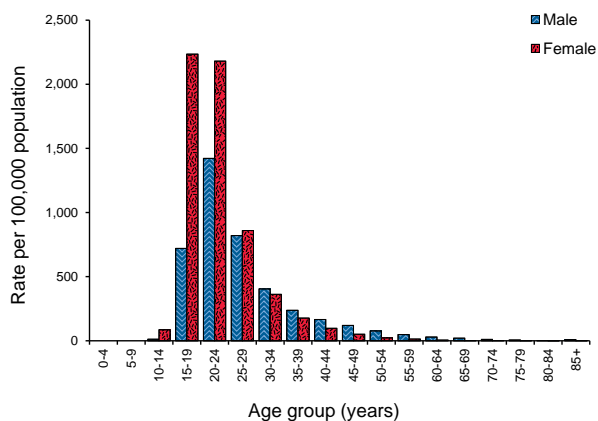
Geographical distribution

Increasing rates of chlamydia were reported from all states and territories with the Northern Territory (1,141.6 per 100,000), Western Australia (496.9 per 100,000) and Queensland (407.2 per 100,000) substantially higher than the national rate (Table 4).

Age and sex distribution

In 2011, rates of chlamydial infection in males and females were 296 and 416 per 100,000 respectively. When compared with 2010, rates increased by 6% in males and 8% in females. Rates in females exceeded those in males prior to the age of 30 years, especially in the 10–14 year age group with a male to female ratio of 0.1:1, while males have higher rates in older age groups (Figure 17). The overall rate in females exceeded those in males with a ratio of 0.7:1, which may be partly attributable to preferential testing of women attending health services compared with men.^{10,32}

Figure 17: Notification rate for chlamydial infection, Australia, 2011, by age group and sex*

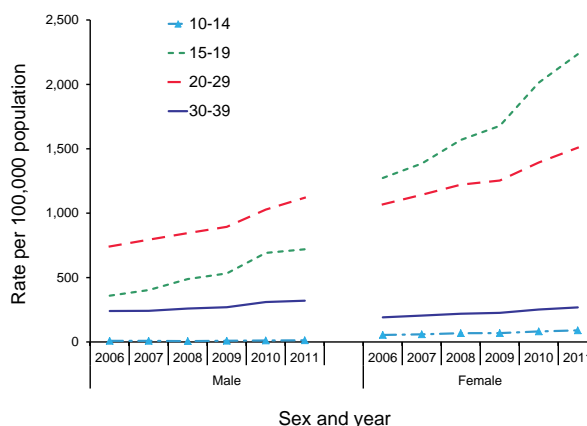


* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Between 2006 and 2011, there was an increasing trend in chlamydia notification rates across both sexes and in all age groups (Figure 18). The greatest increase in rates amongst those aged between 15 and 39 years occurred in both males and females

in the 15–19 (100% and 76% respectively) age group. Those between 15 and 29 years of age accounted for approximately 80% of the annual number of reported cases during the period 2006–2011.

Figure 18: Notification rate for chlamydial infection in persons aged 10–39 years, Australia, 2006 to 2011, by age group and sex*



* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Indigenous populations

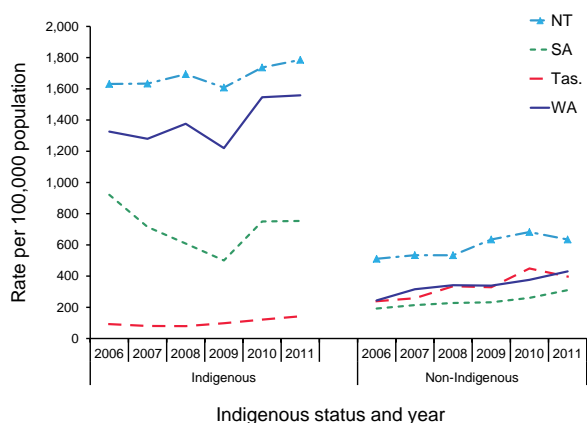
Data on Indigenous status were complete in 51% of notifications in 2011, higher than the preceding 5-year average of 47% (range: 44%–51%). It should be noted that the completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Four jurisdictions had greater than 50% completeness of the Indigenous status field across the 2006 to 2011 period: the Northern Territory, South Australia, Tasmania and Western Australia. Amongst these jurisdictions, the combined age standardised notification rate ratio between Aboriginal and Torres Strait Islander and non-Indigenous populations in 2011 was 3.5:1.

Rates amongst the Aboriginal and Torres Strait Islander population remained fairly consistent between 2006 and 2009, with an average rate during this period of 1,205 per 100,000. Between 2010 and 2011 there was a 21% increase to 1,366 per 100,000. Rates amongst the non-Indigenous population have been trending upwards from a rate of 235 per 100,000 in 2006 to 393 per 100,000 in 2011, a 67% increase over this period.

In 2011 chlamydia rates increased compared with 2010 in all 4 states and territories in which Indigenous status was more than 50% complete, the proportion of cases that were of Aboriginal or Torres Strait Islander origin ranged from 1% in

Western Australia to 17% in Tasmania. Amongst the non-Indigenous population chlamydia rates decreased in Tasmania (11%) and the Northern Territory (7%) and increased in South Australia (18%) and Western Australia (14%) (Figure 19). The overall high Aboriginal and Torres Strait Islander rates observed in the Northern Territory may be partly explainable by the high levels of screening that take place in remote Aboriginal and Torres Strait Islander communities.

Figure 19: Age standardised rates for chlamydial infection, selected states and territories,* 2006 to 2011, by Indigenous status



* Includes notifications in the Northern Territory, South Australia, Tasmania and Western Australia where Indigenous status was reported for more than 50% of cases between 2006 and 2011.

Donovanosis

Donovanosis, caused by the bacterium *Klebsiella granulomatis*, is a chronic, progressively destructive infection that affects the skin and mucous membranes of the external genitalia, inguinal and anal regions.³³ Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project, which commenced in 2001.³⁴ It predominantly occurred in rural and remote Aboriginal and Torres Strait Islander communities in central and northern Australia and is now relatively uncommon.

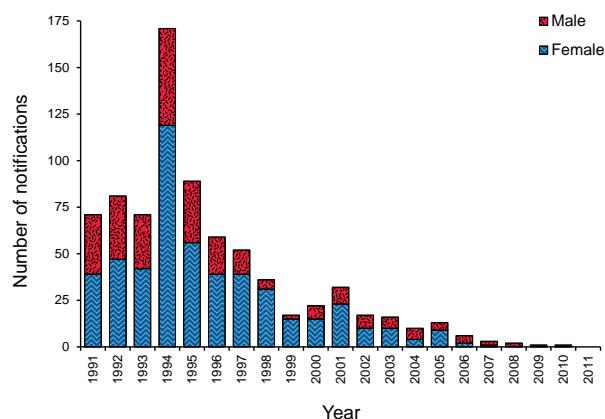
Epidemiological situation in 2011

There were no notifications of donovanosis in 2011 (Figure 20).

Gonococcal infections

Gonorrhoea is caused by the bacterium *Neisseria gonorrhoeae* which affects the mucous membranes causing symptomatic and asymptomatic genital and extragenital tract infections.¹⁴

Figure 20: Notifications of donovanosis, Australia, 1991 to 2011, by sex



Epidemiological situation in 2011

In 2011, there were 12,087 notifications of gonococcal infection, a rate of 53 per 100,000. This was an 18.9% increase compared with 2010.

Geographical distribution

The highest rate in 2011 was in the Northern Territory (849 per 100,000), which was approximately 16 times higher than the national rate (Table 5). All states and territories except Tasmania and South Australia reported increases in rates ranging from 1% in the Northern Territory to 124% in the Australian Capital Territory compared with 2010.

Age and sex distribution

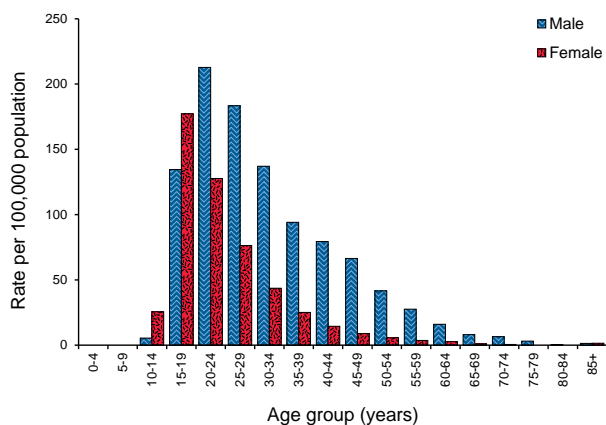
Nationally, there was an increase in gonococcal infection rates in both males (18%) and females (27%) compared with 2010. The male to female rate ratio in 2011 was 2.0:1 (72 and 35 per 100,000 respectively), which is similar to the previous 5 years. The rate of gonococcal infection in males exceeded those in females in all age groups except those less than 20 years of age (Figure 21).

Age specific rates amongst males and females increased in all age groups with a marked increase amongst the 15–19 year age group reported for males and females. (Figure 22).

Indigenous populations

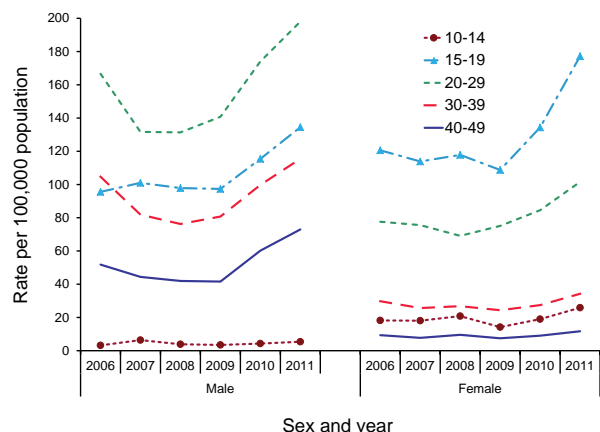
In 2011, the data completeness of the Indigenous status field for gonococcal infection notifications was 68%, slightly higher than 2010 (65%) but a decrease compared with the previous few years (average completeness 69%). All jurisdictions except New South Wales and the Australian Capital Territory had greater than 50% complete-

Figure 21: Notification rate for gonococcal infections, Australia, 2011, by age group and sex*



* Excludes notifications for whom age or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Figure 22: Notification rate for gonococcal infection in persons aged 10–49 years, Australia, 2006 to 2011, by age group and sex*

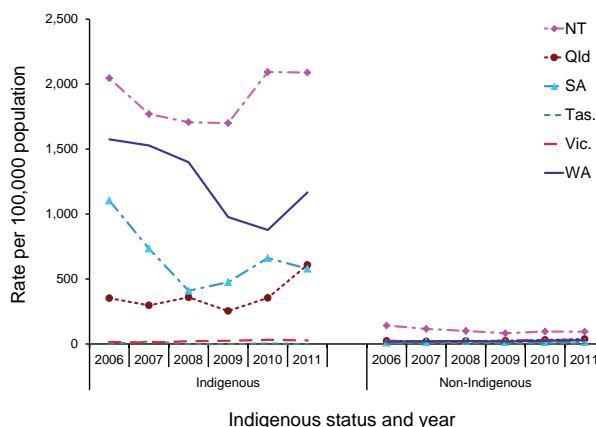


* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

ness of the Indigenous status field. Amongst these jurisdictions with adequate completeness, the combined age standardised notification rate for gonococcal infection in the Aboriginal and Torres Strait Islander population had been steadily declining from 923 per 100,000 in 2006 to 642 per 100,000 in 2009 before increasing to 746 per 100,000 in 2010 and 894 per 100,000 in 2011. In the non-Indigenous population, rates remained stable at around 22 to 23 per 100,000 between 2006 and 2009 before also increasing by 35% to 31 per 100,000 in 2011. Between 2006 and 2011 the Aboriginal and Torres Strait Islander to non-Indigenous rate ratio

decreased 30% from 41:1 to 28:1. In 2011, rates of gonococcal infection in the Aboriginal and Torres Strait Islander and non-Indigenous populations decreased or remained relatively stable compared with 2010 in all jurisdictions except Queensland and Western Australia (Figure 23).

Figure 23: Age standardised rates for gonococcal infection, selected states and territories,* 2006 to 2011, by Indigenous status



* 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.

The overall high Aboriginal and Torres Strait Islander rates observed in the Northern Territory may be partly explained by the high levels of screening that take place in remote Aboriginal and Torres Strait Islander communities.

Microbiological trends

The AGSP is the national surveillance system for monitoring the antimicrobial resistance of *N. 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 2011, the AGSP annual report for 2011³⁵ reported a total of 4,230 gonococcal isolates were tested for antibiotic susceptibility, representing approximately 35% of gonococcal infection notifications, which was a slightly lower proportion to 2010 (41%) and 2009 (40%).

Of the isolates collected through the AGSP in 2011, the majority (3,403) were from males with the remaining 827 being from females (ratio 4:1). In males, 68% of isolates were obtained from the

urethra, 18% from the rectum and 12% from the pharynx. In females, the majority of isolates (86%) were obtained from the cervix.

In 2011, approximately 25% of gonococcal isolates had some level of resistance to the penicillins, a decrease from the 29% in 2010. In addition, 27% had some level of resistance to quinolones, a further decrease in proportion of quinolone resistance from 35% in 2010 and 43% detected in 2009. Since 2001, low but increasing numbers of isolates with decreased susceptibility to ceftriaxone have been identified in Australia with 3.2% observed nationally in 2011. For more details see the AGSP annual report series published in CDI.

Discussion

From 2006 to 2011 there was an increase in the notification rate of gonorrhoea. Preliminary findings from analysis of notification data from 2007 to 2011 indicated that there is no evidence to suggest the overall increase in notifications was due to an increase in a particular sub-group (e.g. Indigenous populations). These analyses also suggest there may be 2 separate epidemics occurring in men who have sex with men in eastern states and amongst women in more remote areas with greater Indigenous populations (unpublished analysis).

Syphilis (non-congenital categories)

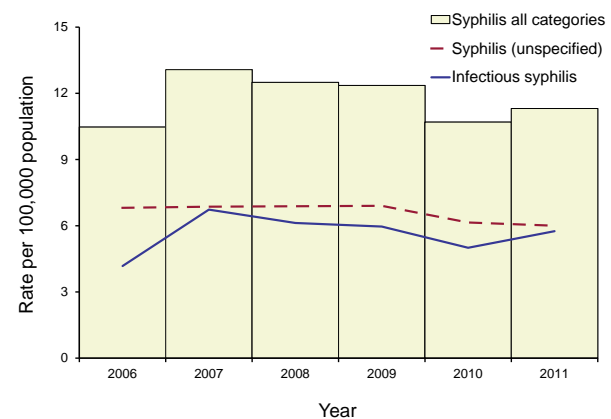
Syphilis, caused by the bacterium *Treponema palladium*, is characterised by a primary lesion, a secondary eruption involving skin and mucous membrane, long periods of latency and late lesions of skin, bone, viscera, cardiovascular and nervous systems.¹⁴

In 2004, all jurisdictions except South Australia began reporting to the NNDSS of 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 2 categories, as well as for syphilis of the combined categories (syphilis – all categories) for the purpose of showing trends in previous years. Data for all states and territories are reported by diagnosis date, except Queensland, which is reported by notification receive date. During this reporting period, the syphilis case definition was applied differently in Queensland compared with the rest of Australia. The aggregated data are presented with this variation in place.

Epidemiological situation in 2011

In 2011, there were 2,563 notifications of syphilis in all non-congenital categories, representing a rate of 11.3 per 100,000, a 4.6% increase compared with 2010 (10.8 per 100,000) (Table 5, Figure 24).

Figure 24: Notification rate for non-congenital syphilis infection (all categories), Australia, 2006 to 2011



* Excludes notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Geographical distribution

The Northern Territory continued to have the highest rate of syphilis (38.6 per 100,000), however this was a marked decline compared with 2010 (61 per 100,000). New South Wales was the only other state to report a decrease in rates. The remaining states and territories reported an increase in rates ranging from 3% in Victoria to 94% in South Australia.

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

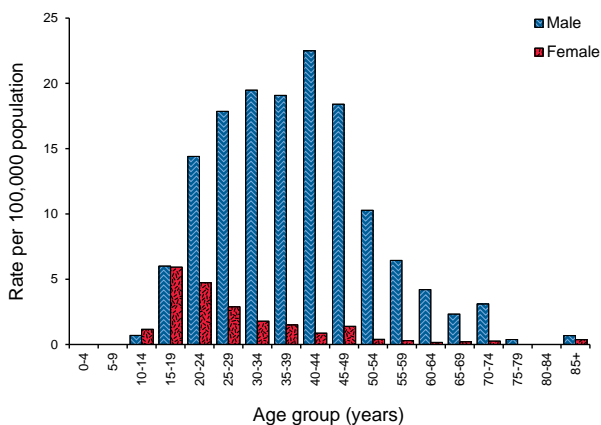
Epidemiological situation in 2011

In 2011, there were 1,303 notifications of infectious syphilis (primary, secondary and early latent), of less than 2 years duration. This represents a notification rate of 5.8 per 100,000, an increase of 13.7% compared with 2010 (5.1 per 100,000) (Table 5). The rate of infectious syphilis notifications increased from 4.3 per 100,000 in 2006 to a peak of 6.8 per 100,000 in 2007 and gradually declined until 2010 with an increase in 2011. The Northern Territory had the highest notification rate at 13.0 per 100,000 in 2011, a 31% decrease compared with 2010.

Age and sex distribution

Rates of infectious syphilis for males and females were 10.0 and 1.5 per 100,000 respectively, representing a male to female ratio of 7:1 (Table 12). Rates in males were highest in the 40–44 year age group (22.5 per 100,000), followed by the 30–34 and 35–39 year age groups (19.5 and 19.1 per 100,000 respectively), whereas in females the highest notification rates were observed in the 15–19 year age group followed by the 20–24 and 25–29 year age groups (5.9, 4.7 and 2.9 per 100,000 respectively) (Figure 25).

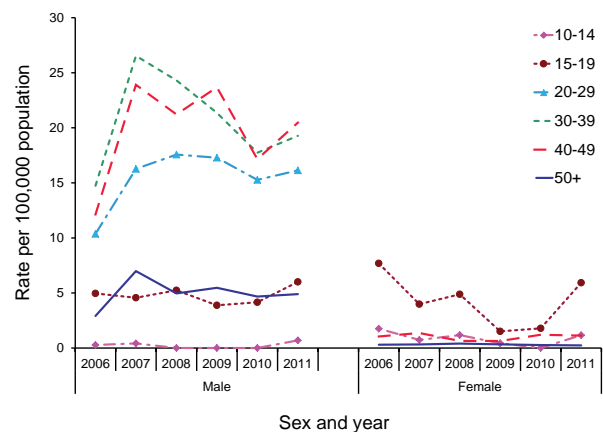
Figure 25: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2011, by age group and sex*



* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

From 2006 to 2007 notification rates amongst males increased substantially in the 20–49 year age range. Since 2007, 20–29 and 30–39 year age groups have either decreased or remained relatively stable. The 40–49 year age group experienced a marked increase between 2010 and 2011. In females, for the 2006 to 2011 period, rates remained relatively steady, except in the 15–19 year age group where they decreased from a peak of 7.7 per 100,000 in 2006 to 1.8 per 100,000 in 2010 and then increased to 5.9 per 100,000 in 2011 (Figure 26).

Figure 26: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, in persons aged 10 years or over, Australia, 2006 to 2011, by age group and sex*



* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Table 12: Number* and rates† of notifications of infectious syphilis (less than 2 years duration), Australia, 2011, by state or territory and sex

State or territory	Male		Female		Total‡	
	Count	Rate†	Count	Rate†	Count	Rate†
ACT/NSW	416	10.9	14	0.4	431	5.6
NT	14	11.7	16	14.4	30	13.0
Qld	262	11.4	70	3.1	332	7.2
SA	24	3.1	12	1.4	45	2.8
Tas.	5	2.0	1	0.4	6	1.2
Vic.	295	10.6	31	1.1	330	5.9
WA	105	8.8	22	1.9	127	5.4
Total	1,121	10.0	166	1.5	1,301	5.8

* Excludes notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

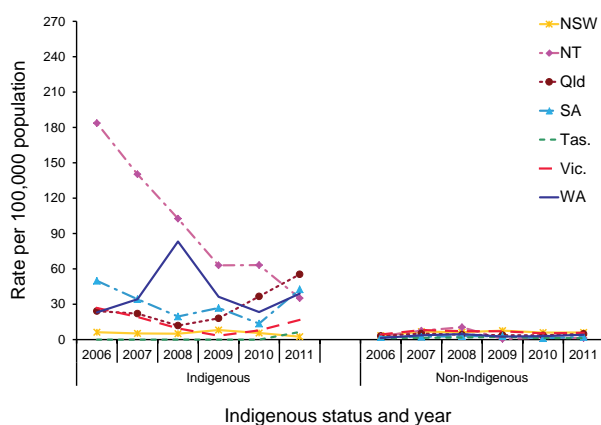
† Notification rate per 100,000 population.

‡ Includes notifications that have missing sex.

Indigenous populations

In 2011, data on Indigenous status was complete for 94% of infectious syphilis notified cases. All jurisdictions except the Australian Capital Territory had greater than 50% completeness of the Indigenous status field between 2006 and 2011. The age standardised notification rate was 29 per 100,000 in the Aboriginal and Torres Strait Islander population and 5.0 per 100,000 in the non-Indigenous population, representing a rate ratio of 6:1. In 2011, Aboriginal and Torres Strait Islander rates in all states and territories, except New South Wales and the Northern Territory, increased when compared with 2010. The increase evident in Aboriginal and Torres Strait Islander rates in Western Australia in 2008 was largely attributable to an outbreak that occurred in 2008 in the Pilbara region amongst Aboriginal people (Figure 27).³⁶ Nationally, there has been a 28% decrease in Aboriginal and Torres Strait Islander rates (from 40 to 29 per 100,000) between 2006 and 2011.

Figure 27: Age standardised rates for infectious syphilis, selected states and territories,* 2006 to 2011, by Indigenous status



* 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

Epidemiological situation in 2011

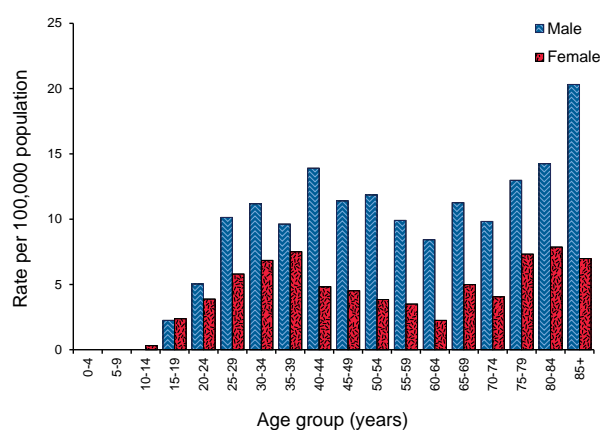
In 2011, there were 1,260 notifications of syphilis of more than 2 years or unknown duration, a rate of 6.0 per 100,000, which is similar to the rate in 2010 (6.1 per 100,000).

Age and sex distribution

In 2011, 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 13), with a male to female rate ratio of 2.0:1 (Figure 28). Age group specific rates in males were higher than in females except in the 10–14 and 15–19 age groups, and were more than 3 times higher amongst males in the 50–54 and 60–64 year age groups than in females. The distribution of notification rates across age groups in females was bimodal, with the highest rate (7.9 per 100,000) amongst those in the 80–84 year age group, followed by those in the 35–39 year age group (7.5 per 100,000). In males, rates remained high in those aged 25 years or over with peaks occurring in the 40–44 year and 85 years or over age groups (13.9 and 20.3 per 100,000 respectively).

Figure 28: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2011, by age group and sex†



* Data from all states and territories except South Australia.

† Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Over the period 2006 to 2011, rates amongst most age groups in males have decreased with a substantial decrease of 42% observed amongst males in the 15–19 year age group. Over the same period, rates in females have decreased in all age groups with the largest declines reported amongst the 15–19 and 20–29 year age groups (56% and 37% respectively) (Figure 29).

Congenital syphilis

Epidemiological situation in 2011

Following a peak of 19 notifications in 2001, notifications of congenital syphilis have continued to decline (Figure 30). Antenatal screening for congenital syphilis is considered to be a contributor to this decline.³⁷ There were 6 notifications of con-

Table 13: Notifications* and notification rate† for syphilis of more than 2 years or unknown duration, Australia,‡ 2011, by state or territory and sex

State or territory	Male		Female		Total§	
	n	Rate	n	Rate	n	Rate
ACT/NSW	220	5.8	112	1.5	332	4.3
NT	41	34.4	18	7.8	59	25.6
Qld	132	5.8	87	1.9	221	4.8
SA	NDP	–	NDP	–	NDP	–
Tas.	12	4.8	8	1.6	20	3.9
Vic.	365	13.1	158	2.8	530	9.4
WA	58	4.9	38	1.6	96	4.1
Total	829	7.9	421	4.0	1,258	6.0

* Excludes notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

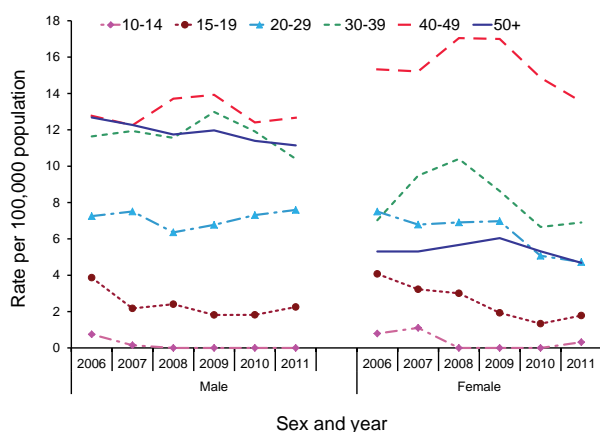
† Notification rate per 100,000 population.

‡ Data from all states and territories except South Australia.

§ Includes notifications missing sex.

NDP No data provided.

Figure 29: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2006 to 2011, by age group and sex†



* Data from all states and territories except South Australia.

† Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

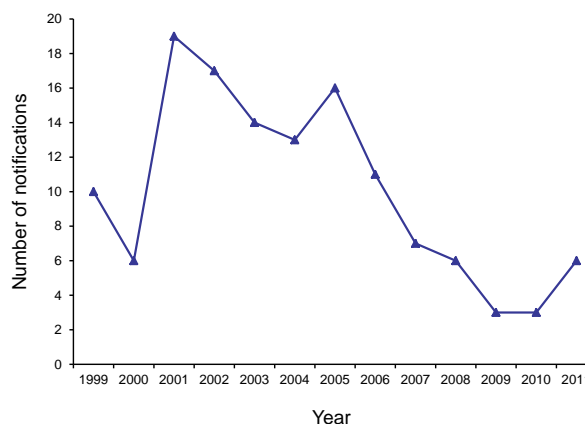
genital syphilis in 2011; 2 females and 1 male from Queensland and 2 males and 1 female from New South Wales. Three of the cases were reported as Aboriginal and Torres Strait Islander and three were non-Indigenous.

Vaccine preventable diseases

Introduction

This section summarises the national surveillance data for notifiable diseases targeted by the

Figure 30: Trends in notifications of congenital syphilis, Australia, 1999 to 2011



National Immunisation Program (NIP) in 2011. 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 detailed 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.³⁸ Additionally, a similar report which analyses the impacts of vaccines on the health of Aboriginal and Torres Strait Islander people between 2007 and 2010 was published in the November 2013 supplement to CDI.³⁹

In 2011, there were 81,872 VPD cases reported to the NNDSS, representing 34% of all reported cases and a 32% increase compared with 2010 (62,009 cases). Pertussis was the most commonly notified VPD with 38,602 cases (47%) reported, reflecting the continued high levels of pertussis activity in 2011; followed by influenza (27,149, 33%). The number of notifications and notification rates for VPDs in Australia are shown in Tables 3 and 4.

New vaccines added to the National Immunisation Program in 2011

In 2011, a pneumococcal conjugate 13-valent (13vPCV) vaccine for infants aged 2, 4 and 6 months and medically at-risk children was introduced onto the NIP. A single catch-up dose was also funded from 1 October 2011 until 30 September 2012 for children aged between 12 months and 35 months who had already completed a primary pneumococcal vaccination.

Vaccination coverage is an important factor influencing the incidence of vaccine preventable diseases. Since the commencement of the Australian Childhood Immunisation Register in 1996, immunisation coverage in Australian 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. 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 the severity of the disease.

Information on the receipt of vaccines has historically been recorded in NNDSS using the 'vaccination status' field (fully or partially vaccinated for age 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 2011, 4 jurisdictions were using the new fields (the 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.

Diphtheria

Diphtheria is an acute toxin-mediated systemic disease caused by the bacterium *Corynebacterium diphtheriae*. Infection is usually localised to the throat (pharyngeal diphtheria) in which a membranous inflammation of the upper respiratory tract can cause airway obstruction, or the skin (cutaneous diphtheria). Systemic complications caused by the bacterium'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.

Epidemiological situation in 2011

There were 4 notifications of diphtheria in 2011. A cluster of 3 pharyngeal cases were diagnosed in Queensland and 1 unrelated case of cutaneous diphtheria was acquired in Indonesia and diagnosed in the Northern Territory. The index case in the Queensland cluster had recently returned from Papua New Guinea, where it is likely that they acquired their infection. This case was confirmed as being a pharyngeal carrier of penicillin resistant *Corynebacterium diphtheriae*. The second case in this cluster, who subsequently died, was a close contact of the index case and the third was an asymptomatic case who was identified through contact tracing. Queensland Health followed up close contacts of the cases and provided prophylactic treatment where required as per their public health guidelines.

Age and sex distribution

The male to female ratio was 1:1 comprising 2 cases each. Three cases, including the fatal case, were in the 20–24 year age group and the fourth was over 85 years of age.

Vaccination status

Two of the 3 cases in the Queensland cluster were vaccinated including the index case and the asymptomatic contact, while the third case, who died, was unvaccinated. The case of cutaneous diphtheria was also vaccinated.

Discussion

In the decade between 1926 and 1935 over 4,000 deaths from diphtheria were reported.³⁸ A vaccine for diphtheria was introduced in Australia in 1932 and since then both cases and deaths have virtually disappeared. Prior to the cases reported in 2011, the last Australian case was one of cutaneous diph-

theria in 2001 acquired in East Timor and the last deaths, of which there were two, occurred between 1986 and 1995. 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.⁴⁰ The 2011 Queensland cluster highlights the importance of maintaining high vaccination coverage to protect against vaccine preventable diseases that remain endemic in many other countries around the world.

Influenza

Epidemiological situation in 2011

Notifications of influenza increased substantially compared with previous years, with the exception of the 2009 pandemic year. There were 27,149 notifications of laboratory-confirmed influenza in 2011, more than twice the number of cases from the previous year (n=13,419).

Notification rates were highest in South Australia (286 per 100,000), followed by the Northern Territory (259 per 100,000) and Queensland (227 per 100,000). Notification rates in the remaining jurisdictions were all substantially lower than the national notification rate of 120 per 100,000. In 2011, Queensland reported a larger number of

influenza cases than any other jurisdiction, comprising 38% of all notifications, which was consistent with previous years with the exception of 2010 (Figure 31).

Age and sex distribution

Females accounted for 14,323 (53%) of the 27,119 influenza notifications for which sex was reported in 2011. Notification rates were higher amongst females in most age groups, with the primary exception of those aged less than 15 years where the rates were higher for males (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.⁴¹

The highest number of influenza notifications occurred in the 0–4 year age group and accounted for 14% of all notifications. Similarly, notification rates were highest in the 0–4 and 5–9 year age groups (255 and 227 notifications per 100,000, respectively) (Figure 33). More than half of all influenza notifications were in persons aged less than 30 years.

Seasonality

Higher than usual numbers of influenza notifications during the 2010–11 inter-seasonal period were reported in all jurisdictions, especially in Queensland and the Northern Territory. The 2011

Figure 31: Notifications of laboratory-confirmed influenza, Australia, 2011, by week and state or territory

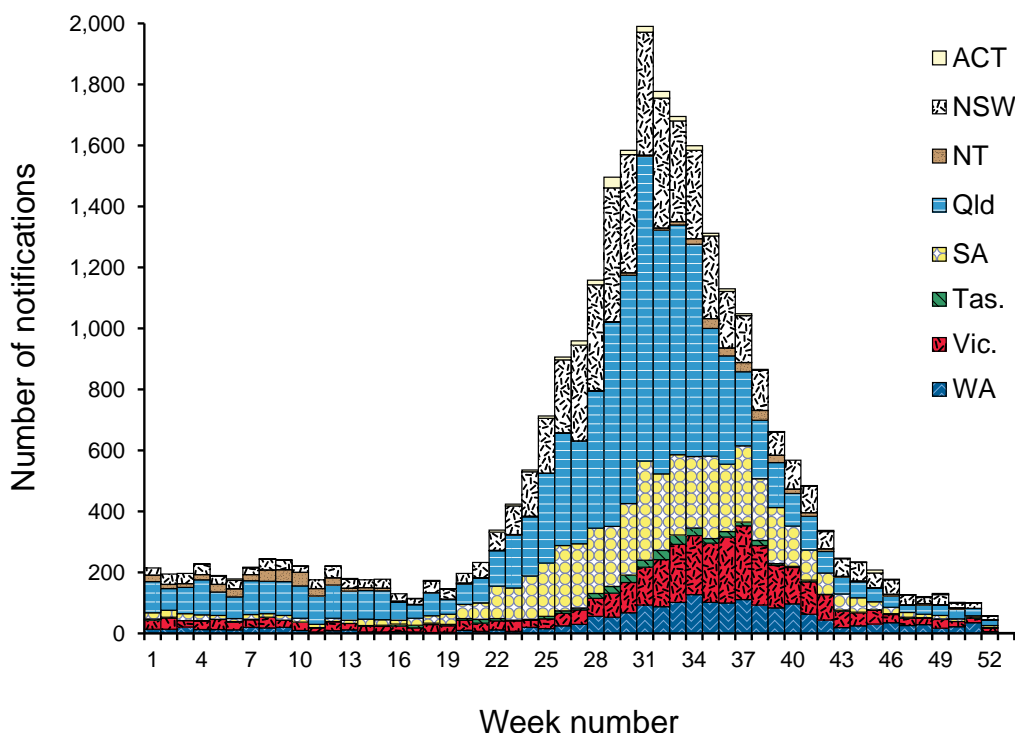
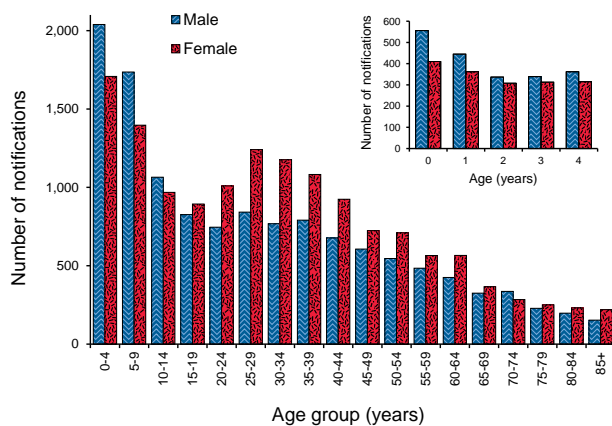
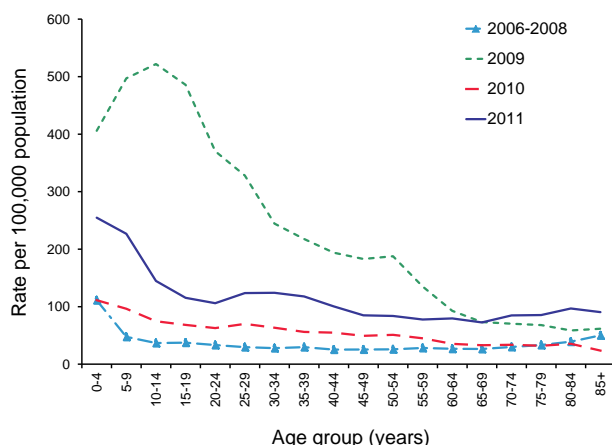


Figure 32: Notifications of laboratory-confirmed influenza, Australia, 2011, by age group and sex*



* Excludes 44 notifications for which age and/or sex were not reported.

Figure 33: Notification rate for laboratory-confirmed influenza, Australia, 2006 to 2011, by age group and year

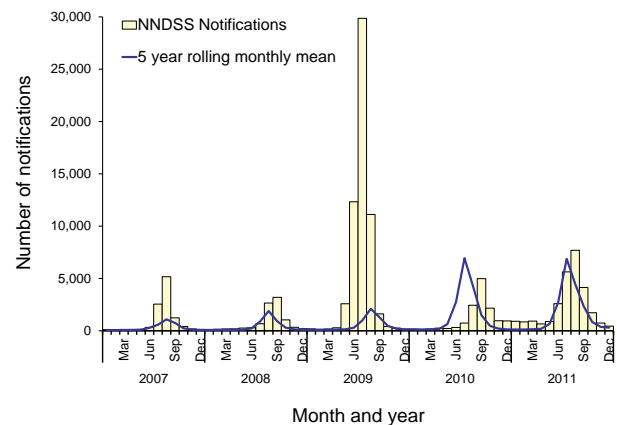


season peaked in August with 7,690 cases for the month, compared with the lower peak of 4,981 notifications during September 2010 (Figure 34). Notifications fell substantially through October and returned to typical inter-seasonal levels by late November 2011.

Virological surveillance

In 2011, almost all (>99%, n=27,049) of the influenza notifications in NNDSS had some level of influenza typing reported. Of those with type information, 73% were type A (40% A (unsubtyped), 26% A(H1N1)pdm09, 7% were A(H3N2)) and almost 27% were type B. Mixed influenza type A and B infections, and influenza type C together accounted for less than 1% of notifications (Figure 35). In comparison, in 2010 the

Figure 34: Notifications of laboratory-confirmed influenza, Australia, 2007 to 2011, by month and year

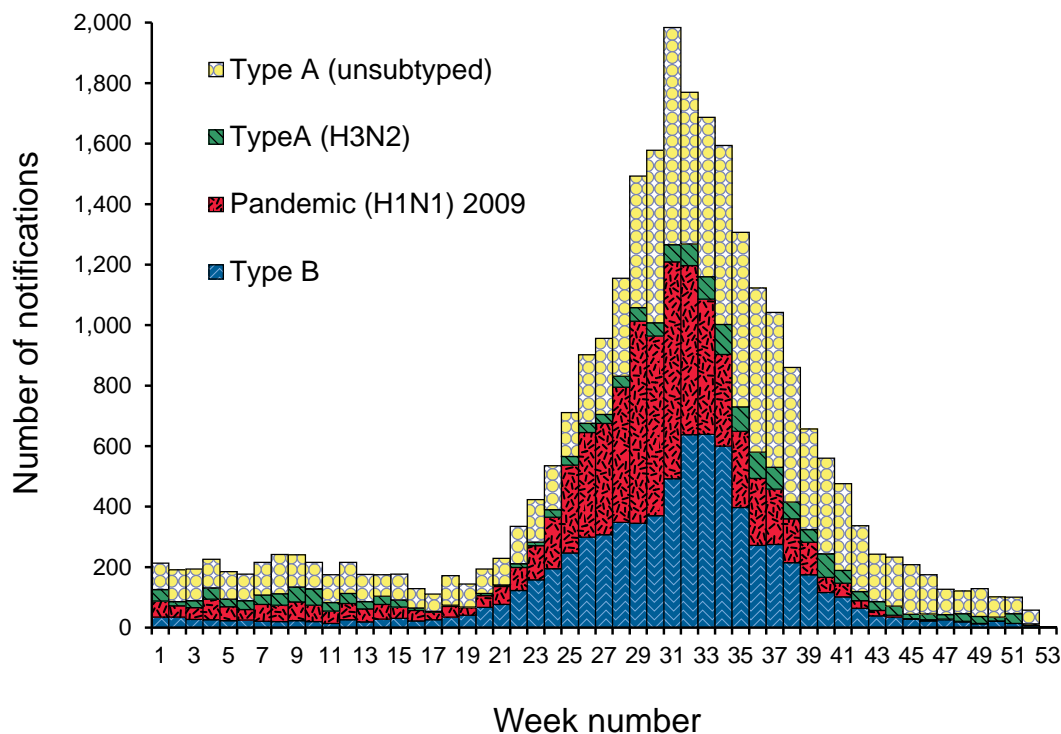


proportion of notifications reported as influenza type A was much higher (90%), with the majority of these (56%) reported as A(H1N1)pdm09, followed by A(unsubtyped) (30%), with very few A(H3N2) (4%). Additionally, the proportion of influenza B notifications in 2010 was substantially less (10%) than in 2011. Mixed influenza type A and B infections also accounted for less than 1% of notifications and typing data were not available for 18 cases in 2010.

The WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) typed and subtyped 2,377 influenza virus samples that were collected in 2011. This represented 8.8% of the 27,149 laboratory confirmed cases reported to the NNDSS. Influenza A(H1N1)pdm09 comprised 46% of influenza viruses, followed by influenza B (29%; consisting of 98.3% B/Victoria lineage and 1.7% B/Yamagata lineage viruses) and influenza A(H3N2) (24%).

All 3 strains of the 2011 Southern Hemisphere influenza vaccine were the same as those previously recommended in the 2010 Southern Hemisphere vaccine. The 2011 Australian influenza vaccine contained an A/California/7/2009 (H1N1)-like virus, an A/Perth/16/2009 (H3N2)-like virus and a B/Brisbane/60/2008-like virus. The WHOCC conducted antigenic characterisation by haemagglutination inhibition assays on 2,177 influenza virus isolates. The majority (79%) of A(H1N1)pdm09 isolates were characterised as A/California/7/2009-like, while the remainder were characterised as 'low reactor' compared with the reference virus. Of the circulating influenza A(H3N2) viruses analysed, nearly all (98%) were antigenically similar to the A/Perth/16/2009 virus. Similarly, most (89%) influenza B viruses detected were closely related to the B/Brisbane/60/2008

Figure 35: Notifications of laboratory-confirmed influenza,* Australia, 2011, by week and subtype



* Excluding mixed type A and B, type C and untyped influenza infections.

virus (a B/Victoria lineage virus). A small number ($n=7$) of influenza B viruses were closely related to the B/Florida/4/2006 virus (B/Yamagata lineage). Thus, the majority of circulating viruses that were isolated in 2011 were antigenically similar to the 2011 vaccine viruses.

Viruses collected in 2011 were also tested for antiviral susceptibility and resistance to the neuraminidase inhibitor class of antiviral drugs (oseltamivir and zanamivir). Neuraminidase inhibition (NAI) assay was performed on 2,173 viral isolates. Twenty-four of the A(H1N1)pdm09 isolates tested showed resistance to oseltamivir and two showed resistance to zanamivir. Pyrosequencing of 157 A(H1N1)pdm09 clinical specimens (these samples were influenza positive but virus was not able to be isolated from them for the NAI assay) found 15 specimens with the H275Y mutation, which is known to confer oseltamivir resistance. Therefore a total of 39 (3.6%) A(H1N1)pdm09 viruses showed oseltamivir resistance. No oseltamivir or zanamivir resistance was detected in any of the A(H3) or influenza B viruses.

Discussion

Higher than usual levels of influenza activity characterised the 2010–11 inter-seasonal period and contributed to the increase in notifications, compared with the previous year. There were 4,207

notifications in the first 5 months of 2011, compared with just 934 in the same period of 2010. Most of the influenza activity during this period was attributed to A(H1N1)pdm09 and A(H3N2) infections. The reason for the unusually high activity is not clear but does not appear to be solely due to increased testing. It is worth noting that the 2010–11 inter-seasonal period was characterised by extensive flooding, particularly in Queensland, which may have been associated with increases in influenza A virus survival rates,⁴² and time spent indoors.

The main 2011 winter season commenced and peaked earlier than the previous year, although the timing of peaks and the distribution of subtypes varied by jurisdiction. While the majority of virus detections were reported as influenza A(unsubtyped), the season was characterised as a A(H1N1)pdm09 season, with co-circulation of influenza B. In comparison to 2010, the proportion of A(H1N1)pdm09 notifications fell from 56% to 26% in 2011. The shift to increasing proportions of influenza A(H3N2) and type B was associated with an increase in notifications rates for people aged 70 years or over. This contrasts with the pandemic dominant year of 2009, which was characterised by decreasing notifications rates by increasing age.

The number of laboratory confirmed notifications of influenza in 2011 was more than twice that of

the previous year. Other influenza surveillance systems indicate that the increase in activity through the main winter season is not significant compared with 2010, and may be a result of, at least in part, increased testing, including differential testing rates between jurisdictions.

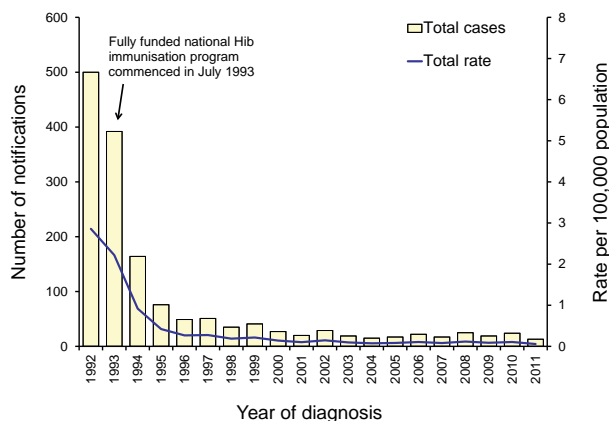
Invasive *Haemophilus influenzae* type b disease

Invasive *Haemophilus influenzae* type b (Hib) bacteria cause disease with symptoms dependant on which part of the body is infected. These include: septicaemia; meningitis; epiglottitis; pneumonia; osteomyelitis and cellulitis.

Epidemiological situation in 2011

There were 13 notifications of Hib disease reported in 2011, a little over half of the 24 reported in 2010 and a ratio of 0.6 compared with the mean notifications during the previous 5 years. The rate in 2011 was 0.1 per 100,000 and consistent with the very low rates since the Hib vaccine was included in NIP in July 1993 (Figure 36). Cases occurred in Queensland (n=5), New South Wales (n=4), the Northern Territory (n=2), and one each in Victoria and Western Australia. Indigenous status was completed for 100% of cases in 2011. Two cases (15%) were reported as Indigenous, both were notified from the Northern Territory.

Figure 36: Notifications and notification rate for invasive *Haemophilus influenzae* type b infection, Australia, 1992 to 2011

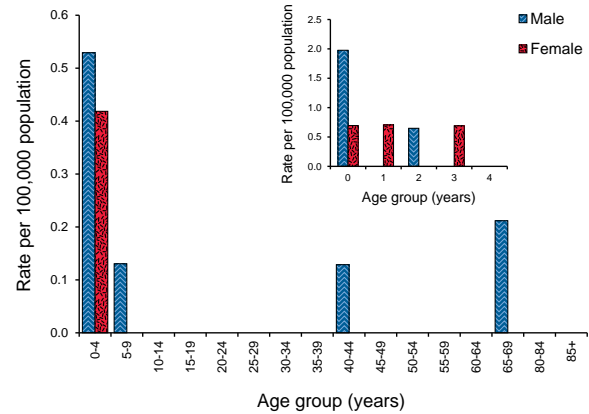


Age and sex distribution

The male to female ratio was 1.6:1 in 2011 with 8 males and 5 females overall. The majority of cases (n=7) were in children less than 5 years of

age, 57% of which were in infants aged less than 1 year. Age group specific rates were highest in the 0–4 year age group (Figure 37).

Figure 37: Notification rate for invasive *Haemophilus influenzae* type b infection, Australia, 2011, by age group and sex



Vaccination status

Since the introduction of the Hib vaccine in 1993, there has been a marked reduction in notified cases of Hib in Australia (Figure 36), which now has one of the lowest rates of Hib in the world.³⁸

In 2011, all children under the age of 19 years were eligible for Hib vaccination in infancy, as Hib vaccines were introduced to the NIP in April 1993 for all children born after February 1993. Of the 8 eligible cases in 2011, 7 were fully vaccinated for age and of these, four had received all scheduled doses as recommended under the NIP.

Invasive pneumococcal disease

Invasive pneumococcal disease (IPD) is a clinical condition in which *Streptococcus pneumoniae* is isolated from a normally sterile site such as blood, cerebrospinal fluid or pleural fluid. A universal pneumococcal vaccination program with the 7-valent pneumococcal conjugate vaccine (7vPCV) was introduced onto the NIP for young children in 2005. This was an expansion of the use of the 7vPCV for Aboriginal and Torres Strait Islander and medically at-risk children that was included on the NIP in July 2001. The 7vPCV targets *S. pneumoniae* serotypes 4, 6B, 9V, 14, 18C, 19F and 23F. From 1 July 2011 a higher valency conjugate vaccine replaced the 7vPCV on the NIP; the 13-valent pneumococcal conjugate vaccine (13vPCV) targets an additional 6 serotypes (1, 3, 5, 6A, 7F, 19A).

From 1 October 2011 until 30 September 2012 a supplementary dose of the 13vPCV was made available under the NIP to eligible children who had completed their primary pneumococcal vaccination course with the 7vPCV. Vaccination with the 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or over in 1999 and non-Indigenous Australians aged 65 years from January 2005.⁴³

Epidemiologic situation in 2011

There were 1,887 notifications of IPD in 2011, representing a rate of 8.3 per 100,000 population.

This was the highest number and rate reported in any 1 year since prior to the introduction of the universal pneumococcal conjugate vaccine program for young children in 2005. The jurisdiction-specific rate of IPD varied from 7.3 per 100,000 in New South Wales to 56.0 per 100,000 in the Northern Territory.

A rise in IPD due to serotype 1 was observed in Central Australia, initially in school-aged Indigenous children in October 2010. The increase continued throughout 2011, spreading throughout the Northern Territory and into Western Australia and Queensland. Nationally, there were 155 cases of IPD due to serotype 1 reported in 2011 and Indigenous Australians accounted for 71% (n=110) of these cases. Excluding these cases reduced the national rate to 7.7 cases per 100,000. Compared with 2010, the overall number of IPD cases increased by 15%. IPD due to serotype 1 accounted for a large proportion (63%), but not all, of this increase.

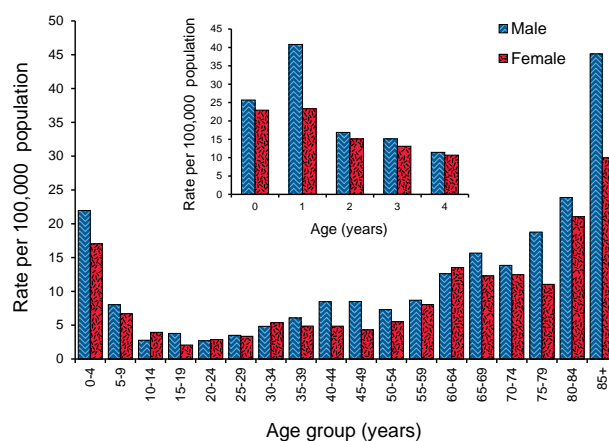
Each jurisdiction experienced an increase on the number of cases reported in the previous year. The largest increase at the jurisdictional level was in cases resident in the Northern Territory, where the number of cases increased by 126%, from 57 cases in 2010 to 129 cases in 2011. Most of this increase (85%, n=61) can be attributed to a large outbreak of serotype 1 cases. The increase in cases reported in Queensland and Western Australia also exceeded the average national increase. Cases resident in Queensland increased by 26% from 271 cases reported in 2010 to 341 cases reported in 2011. Only 23% of this increase can be attributed to serotype 1 cases. Cases resident in Western Australia increased by 23% from 198 cases reported in 2010 to 243 cases reported in 2011. All of this increase can be attributed to serotype 1 cases, with 56 cases reported in Western Australia in 2011.

Age and sex distribution

The rate of IPD distributed by age in 2011 was bimodal, with the highest rates reported in the elderly and young children (Figure 38). In the elderly, the highest rate was in those aged 85 years or over (35.2 per 100,000) and in children aged less than 5 years the rate was highest in those aged 1 year (32.3 per 100,000).

In 2011, males accounted for 54% of cases of IPD, resulting in a male to female ratio of 1.2:1. The rate of disease in males exceeded that in females in almost all age groups (Figure 38).

Figure 38: Notification rate for invasive pneumococcal disease, Australia, 2011, by age group and sex



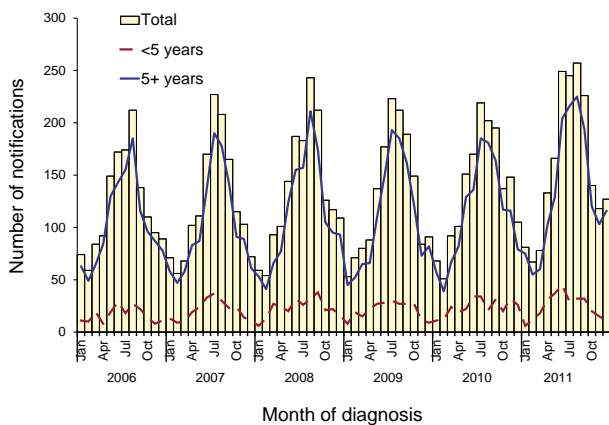
Seasonality

The seasonal trend of IPD in 2011 followed the trend seen in previous years and that of other respiratory diseases (Figure 39). The number of cases was greatest in the winter months, reaching a peak for 2011 in August (n=257). The seasonal trend was more evident in the distribution of cases aged 5 years or over compared with younger children.

Indigenous status

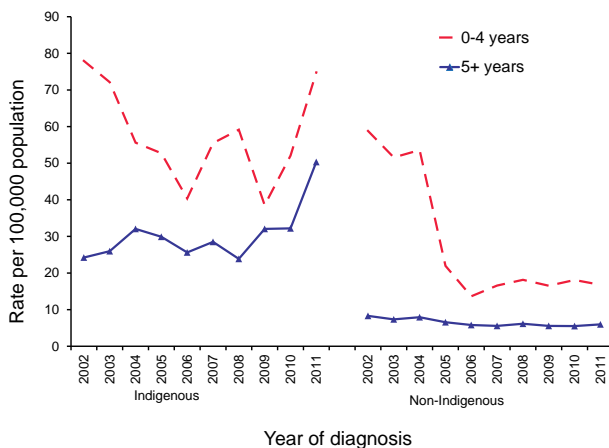
Completeness of Indigenous status reporting in 2011 was high, with 94% (n=1778) of cases reported with a known Indigenous status. Indigenous people made up 17% (n=307) of all notifications. In 2011, the rate of IPD in Indigenous people (53.3 per 100,000) was 8 times the rate of non-Indigenous people (6.7 per 100,000). This is the largest gap in IPD rates since national surveillance commenced in 2002.

Figure 39: Notifications of invasive pneumococcal disease, Australia, 2011, by month of diagnosis



The rate of disease in Indigenous people has steadily increased since 2008 (Figure 40). However, the rate of disease in non-Indigenous people, particularly in the 0–4 years age group, has continued the large decrease observed as a result of the introduction of the universal 7vPCV immunisation program in 2005.

Figure 40: Notification rate for invasive pneumococcal disease, Australia, 2002 to 2011, by Indigenous status and age group

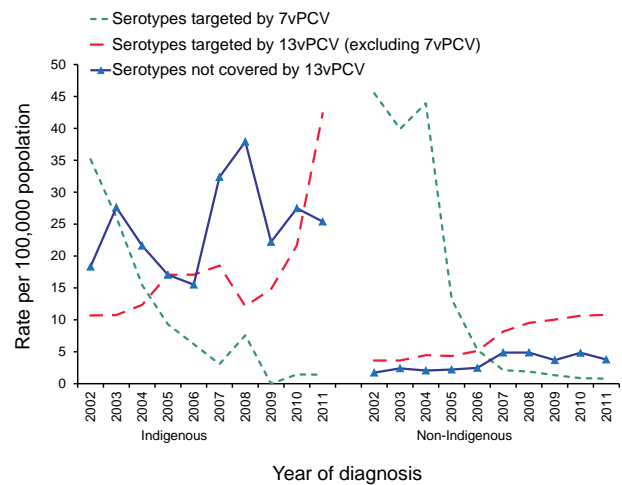


Serotype

An identified serotype was reported in 93% (n=1746) of notified cases of IPD in 2011. In children aged less than 5 there has been a dramatic reduction in disease due to serotypes targeted by the 7vPCV in both the Indigenous and non-Indigenous populations, with this reduction being maintained in 2011 (Figure 41). As few as 5% (n=12) of notifications in children aged less than

5 with a serotype identified were caused by those targeted by the 7vPCV in 2011. In this same age group there was an increase in disease due to the 6 additional serotypes targeted by the 13vPCV, with 63% (n=181) of notifications in 2011 due to one of these 6 serotypes. In Indigenous children, this was largely (67%) due to serotype 1, while in non-Indigenous children serotype 19A was the most common serotype reported (77%).

Figure 41: Notification rate for invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2011, by Indigenous status and serotype group



Discussion

In 2011, IPD reached its highest level since the introduction of the universal pneumococcal conjugate vaccine program in 2005, with much of this increase attributable to an outbreak of IPD due to serotype 1, which began in Central Australia in late 2010. Despite this, a significant reduction in disease due to serotypes targeted in the 7vPCV in both Indigenous and non-Indigenous populations is clearly demonstrated in the notification data. It is important to note that for the Indigenous population national pre-vaccination data are not available as the program was introduced prior to national surveillance commencement in 2002.

The recent increase in disease due to serotypes 1 and 19a indicates potential for the introduction of the 13vPCV to have a significant impact on IPD in Australia. On-going surveillance will be critical to measuring the impact of this and future vaccine programs and detecting the emergence of non-vaccine serotypes.

More detailed analyses can be found in the IPD annual report series published in CDI.

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 is a late, rare (approximately 1 in 100,000 cases) complication of measles, which is always fatal.⁴⁴

Epidemiological situation in 2011

There were 193 notifications of measles in 2011 representing a rate of 0.9 per 100,000 and 2.6 times the mean notification rate of the previous 5 years. This was the highest number of cases since 1999 when 239 cases were reported (Figure 42).

Increases occurred in all states and territories, except Tasmania where no cases were reported. The majority of cases, and largest increases compared with 2010, occurred in New South Wales (n=90), Victoria (n=39) and the Australian Capital Territory (n=21) (Figure 43).

Indigenous status was known for 95% of cases in 2011 (n=183), and of these, 5.5% (n=10) were reported as Indigenous. These 10 cases were all reported from New South Wales where they represented a significantly higher notification rate compared with non-Indigenous people in that state.⁴⁵ In temperate climates where measles transmission remains endemic, the majority of cases occur in late winter or early spring. This seasonal pattern is no longer evident in Australia.

Figure 42: Notifications and notification rate for measles, Australia, 1997 to 2011

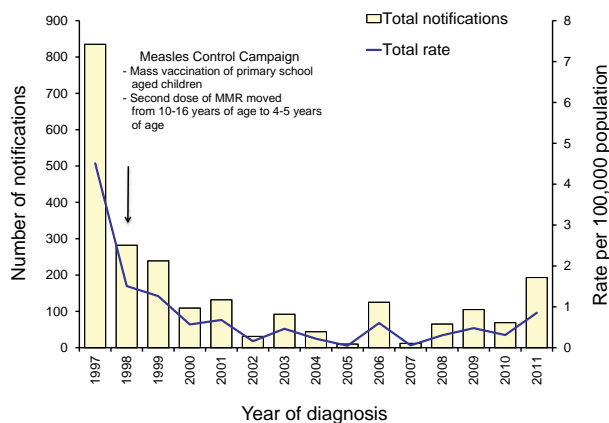
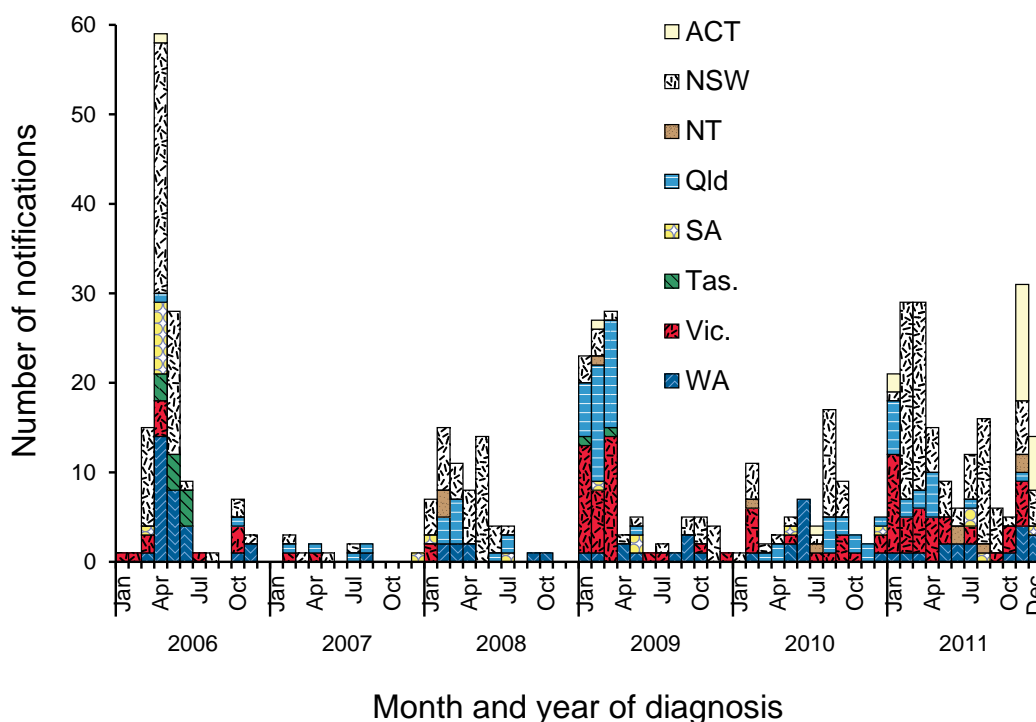


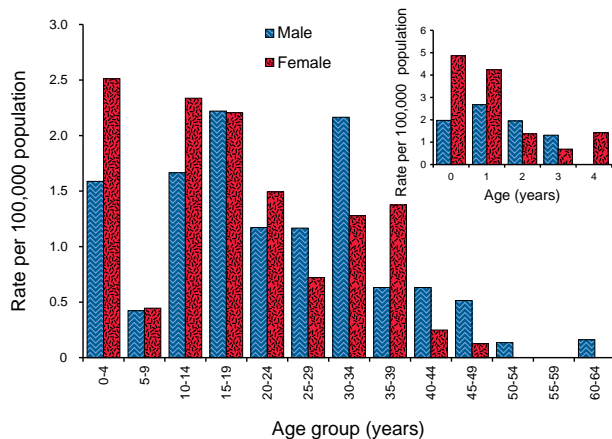
Figure 43: Notifications of measles, Australia, 2006 to 2011, by state and territory and month of diagnosis



Age and sex distribution

The overall male to female ratio was 1:1 in 2011; however, variation in sex distribution occurred across age groups (Figure 44).

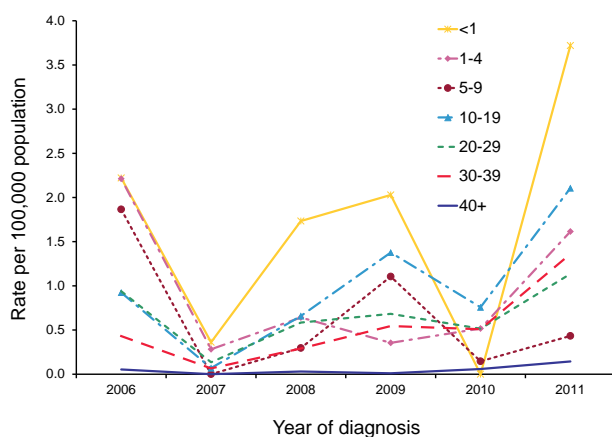
Figure 44: Notification rate for measles, Australia, 2011, by age group and sex



In 2011, age at diagnosis ranged from 0 to 66 years with a median age of 19 years. Rates increased across all age groups in 2011 compared with 2010 (Figure 45). The highest age specific rates occurred in the less than 1 year age group at 3.7 per 100,000. There were 11 cases reported in this group. High rates also occurred in the 10–19 year age group (2.1 per 100,000), reflecting the large number of cases reported in this age group (n=61).

Measles rates remained below 2.5 per 100,000 in all age groups between 2006 and 2011 with the exception of infants aged less than 1 year in 2011 (Figure 45). The fluctuating nature of these rates can be attrib-

Figure 45: Notification rate for measles, Australia, 2006 to 2011, by age group



uted to a general trend of sporadic imported cases that occasionally result in outbreaks of locally acquired infection amongst susceptible contacts.

Vaccination status

Two doses of the measles–mumps–rubella (MMR) vaccine are funded under the NIP for children at 12 months and 4 years of age. The MMR vaccine induces long-term measles immunity in 95% of recipients after a single dose and 99% of recipients after the second dose.⁴⁴

Of the 193 cases notified in 2011, 172 (89%) were born after 31 December 1969 and eligible for a publicly funded measles-containing vaccine. Of the 18 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 and the remaining 17 were not vaccinated. Of the remaining 154 cases 4 years of age or over and eligible for 2 doses, 81 were not vaccinated, 17 were partially vaccinated, 5 were fully vaccinated and 51 were of unknown vaccination status. The 10–19 year age group accounted for 51% (n=41) of the unvaccinated cases amongst those 4 years of age or over. Twenty-two cases occurred amongst those born between 1978 and 1982 (29–33 years in 2011). This cohort has previously been identified as susceptible to measles infection as during their childhood a second dose of a measles containing vaccine was not yet recommended and they were not targeted as part of the 1998 measles control campaign.⁴⁶ Of these cases, 6 were at least partially vaccinated for age, 5 were not vaccinated and 11 were of unknown vaccination status. Eleven cases occurred in adults born before 1968, a cohort that is considered to have high levels of natural immunity,⁴⁷ all of them were either unvaccinated or of unknown vaccination status.

Source of infection and outbreaks

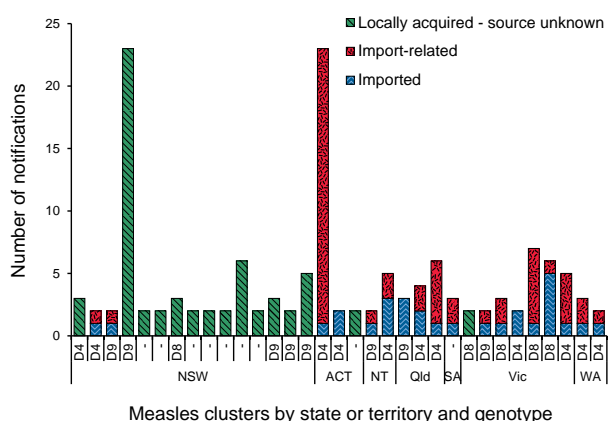
The majority of cases in 2011 were either imported (32%, n=61) or import-related (27%, n=53). The remaining 79 cases (41%) were locally acquired with the original source of infection unknown. Eighty four per cent of all imported cases were either from the South East Asia Region (n=26) or the Western Pacific Region (n=25). There were 9 cases imported from the European Region and one from South East Africa.

There were 33 clusters of two or more epidemiologically linked cases in 2011. In all except two of these, transmission was interrupted quickly resulting in an outbreak size of fewer than 10 cases. There were 2 outbreaks with more than 9 cases, the first of which involved 23 locally-acquired cases in western Sydney for which a definitive source of infection could not be established. The second cluster

was associated with an imported case from New Zealand and resulted in 23 cases predominantly amongst students at a high school in Canberra and their contacts in both the Australian Capital Territory and neighbouring New South Wales.

Genotyping was available for 73% (n=24) of the outbreaks accounting for 85% of all outbreak cases. Genotypes D4, D8 and D9 were identified amongst outbreak cases across Australia (Figure 46).

Figure 46: Measles clusters, Australia, 2011, by state or territory, genotype and source of infection



Discussion

In October 2010, at the Western Pacific Regional Committee meeting the Regional goal of measles elimination was re-affirmed (resolution RC61.R7) and the Regional Director was requested to establish regional measles verification mechanisms. A Regional Verification Commission for Measles Elimination (RVC) was established in December 2011 and Professor David Durrheim from Australia was nominated and accepted as a member of this committee. One of the main terms of reference for the RVC was to establish guidelines and the associated procedures and criteria for verifying elimination of measles at the country and regional level. The WHO proposed definition of measles elimination is the absence of endemic measles transmission in a defined geographical area (e.g. region) for greater than or equal to 12 months in the presence of a well performing surveillance system. Endemic transmission is defined as the existence of continuous transmission of indigenous or imported measles virus that persists for greater than or equal to 12 months in any defined geographical area.⁴⁸

Evidence suggests that endemic measles has been eliminated from Australia, since at least 2005, but possibly earlier.⁴⁹ Based on the WHO definitions, Australia has maintained this in the intervening years. Outbreaks of measles continue to occur mostly related to unvaccinated or partially vaccinated travellers who have been infected in countries where endemic measles transmission continues and then returned to Australia whilst infectious. Due to the highly infectious nature of measles, local transmission can then occur if susceptible individuals, including infants too young to be protected through vaccination, come into contact with the traveller during their infectious period.

In 2011, no outbreak persisted for more than 12 months with the longest lasting approximately 43 days. Ongoing evidence of high population immunity was demonstrated by the rapid cessation of the majority of outbreaks with only three involving more than three generations of transmission (i.e. 35 to 44 days between onset of disease in the first and the last case).⁵⁰ Ninety-five per cent of outbreak cases, in all states except New South Wales, were associated with an index case that was imported from overseas. There was no evidence that a single genotype was continuously circulating for 12 months or more. Of concern in 2011 was that 41% of cases in New South Wales had no link to an imported case able to be established, highlighting that surveillance gaps do occur, either because some cases do not seek medical attention or are not diagnosed with measles when they do.⁵¹ This underlines the difficulty in identifying measles in Australia where the incidence is low and the health system is no longer familiar with this disease.

As part of the regional verification process Australia, along with all Western Pacific Region member countries, will be required to provide epidemiological and virological evidence of sustaining measles elimination on a background of high quality surveillance in order for Australia and the region to be certified measles-free.

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. Mumps is a multi-system infection, with orchitis occurring in 20% to 30% of post-pubertal males.¹⁴

Epidemiological situation in 2011

In 2011, there were 155 notifications of mumps, a rate of 0.7 per 100,000 and a 60% increase compared with the 97 cases reported in 2010. The number of cases remains low compared with the peak of 582 cases reported in 2007 (Figure 47). Cases in 2011 were reported from all states and territories except the Northern Territory. Rates were highest in New South Wales (0.9 per 100,000) followed by Queensland and Victoria (0.8 per 100,000).

Indigenous status was reported for 63% of mumps cases, an increase of 13% compared with 2010, and 2% (2/98 cases) were reported as Indigenous.

Age and sex distribution

In 2011, the overall male to female ratio was 1:1 with some variation in the sex ratio amongst age groups, notably where the numbers were small. The highest rates for males occurred in the 30–34 year age group (1.66 per 100,000) and for females in the 15–19 year age group (1.24 per 100,000). Rates were higher for young adults of both sexes compared with other cohorts and ranged from 0.93 to 1.66 per 100,000 for males between 20 and 39 years of age and 0.96 to 1.24 per 100,000 for females between 15 and 39 years of age (Figure 48).

There were cases of mumps notified across most age groups with age at diagnosis ranging from 3 to

80 years and with a median age of 30 years. All age group rates in 2011 were higher than in 2010 except those less than 1 year of age, in which there were no cases. The biggest increase in age group rates occurred amongst young adults between 20 and 39 years of age, although they remained lower compared with the peak amongst this cohort in 2007–2008 (Figure 49).

Vaccination status

The mumps component of the MMR vaccine has been estimated to be the least effective of the 3 components, providing 62%–88% and 85%–95%

Figure 48: Notification rate for mumps, Australia, 2011, by age group and sex

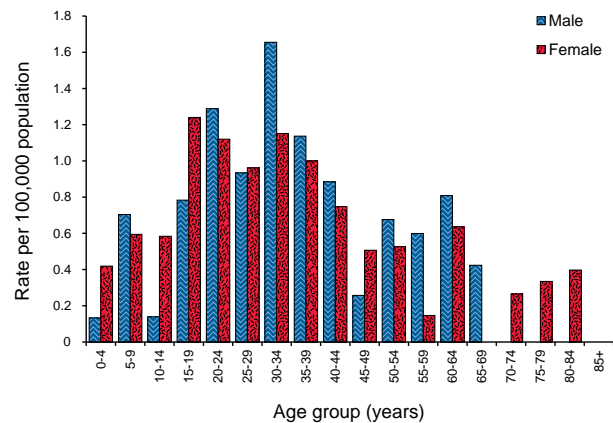


Figure 47: Notifications of mumps, Australia, 2006 to 2011, by state or territory and month of diagnosis

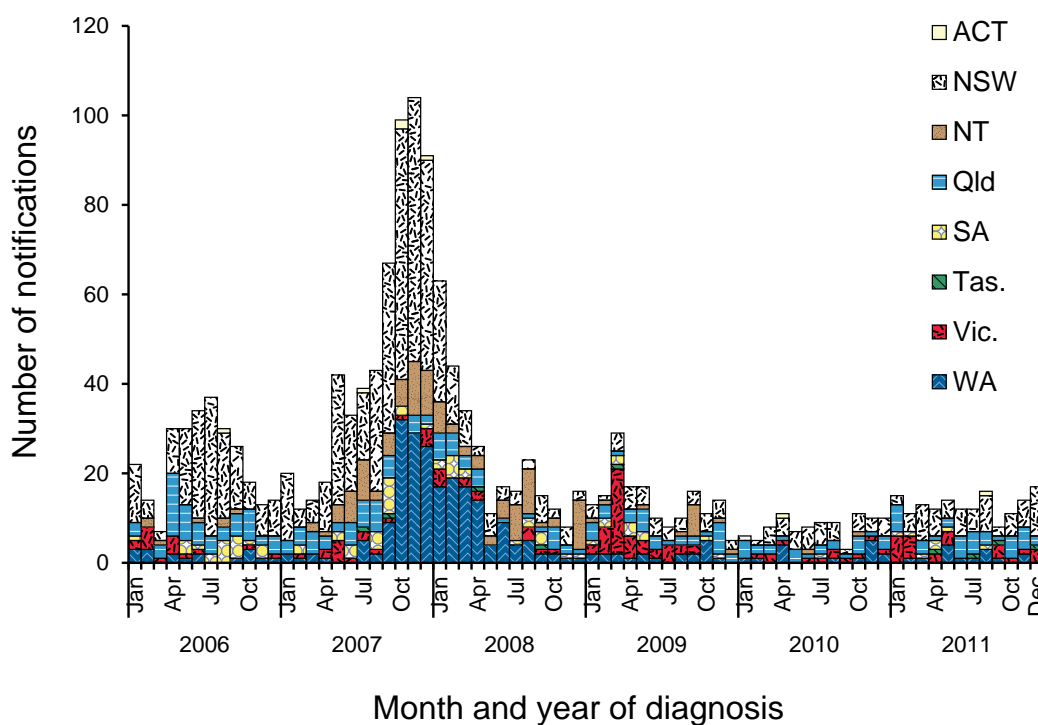
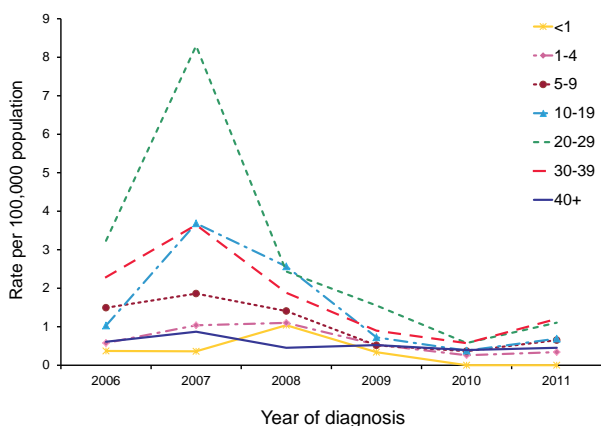


Figure 49: Notification rate for mumps, Australia, 2006 to 2011, by age group



protection for the first and second doses respectively.^{52,53} Reduced effectiveness of the mumps vaccine has been demonstrated over time and this waning immunity may at least partially account for the proportion of vaccinated mumps cases and contribute to mumps outbreaks in older vaccinated populations.⁵³

The mumps vaccine was first funded on the NIP available in Australia in 1981 with people born since then eligible for 2 doses of a mumps-containing vaccine.⁵⁴ In 2011, there were 75 cases of mumps in individuals born after 31 December 1980. One case was aged between 1 and 3 years and eligible for 1 dose and was fully vaccinated

for age. The remaining 74 cases were aged 4 years or over. Of these, 9% (n=7) were fully vaccinated for age, 9% (n=7) were partially vaccinated for age, 15% (n=11) were unvaccinated. As mumps notifications are not routinely followed up by all public health units, a further 66% (n=49) had an unknown vaccination status reported.

Pertussis

Pertussis, commonly known as whooping cough, is a highly infectious disease caused by *Bordetella pertussis* and is spread by respiratory droplets.

Epidemiological situation in 2011

In 2011, there continued to be a large number of cases of pertussis associated with the Australia-wide epidemic that began in mid-2008 (Figure 50). There were 38,602 notifications of pertussis in 2011. This included 3 deaths, all in infants less than 8 weeks of age who were too young to be protected by vaccination. While pertussis remains endemic in Australia with a cyclical pattern of epidemic activity occurring approximately every 3 to 4 years, this latest epidemic has been much larger and more prolonged than earlier outbreaks (Figure 51).

Rates varied considerably by state or territory in 2011 with the Australian Capital Territory (227 per 100,000), Queensland (196 per 100,000) and New South Wales (179 per 100,000) all having rates higher than the national rate (171 per 100,000).

Figure 50: Notifications of pertussis, Australia, 2006 to 2011, by month of diagnosis

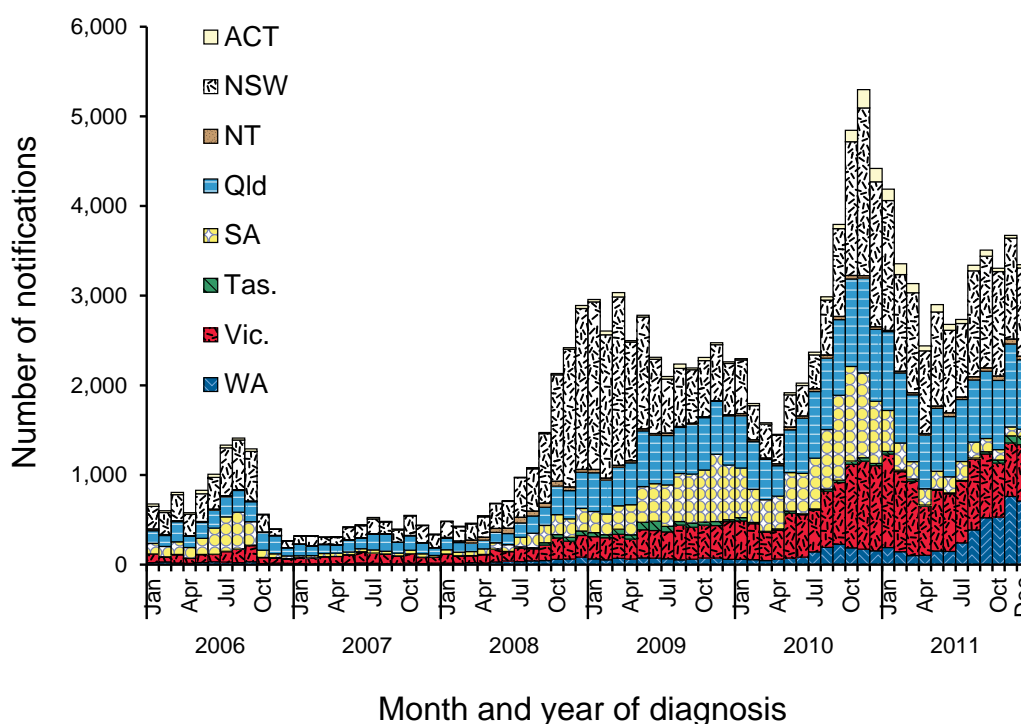
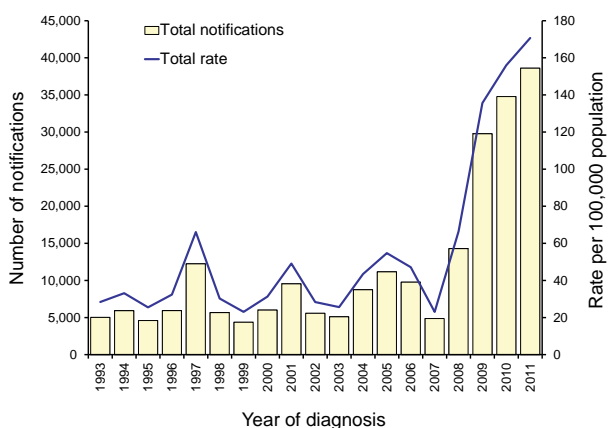
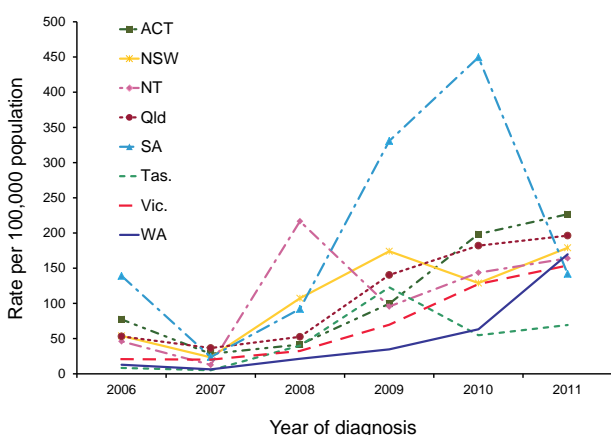


Figure 51: Notifications and notification rate for pertussis, Australia, 1993 to 2011



While the timing of epidemic activity has varied across states and territories, all except South Australia had increased rates in 2011 compared with 2010 and the Australian Capital Territory, New South Wales, Queensland, Victoria and Western Australia all reported their highest rates since the epidemic began. The largest increase in activity in 2011 occurred in Western Australia, which increased from a rate of 63 per 100,000 in 2010 to 170 per 100,000 in 2011. In contrast, rates in South Australia declined sharply from a peak of 450 per 100,000 in 2010 to 142 per 100,000 in 2011 (Figure 52).

Figure 52: Notification rate for pertussis, 2006 to 2011, by state and territory

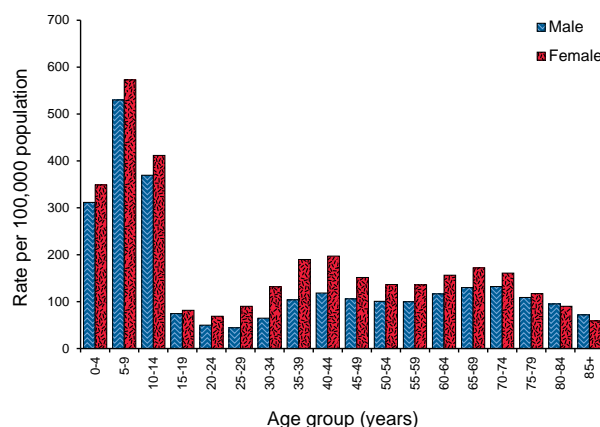


Age and sex distribution

In 2011, females accounted for 56% (n=21,512) of cases, resulting in a male to female ratio of 0.8:1. Forty-one cases had no sex specified and an additional 20 had no age provided. Females had higher rates across all age groups except for those

adults 80 years of age or over (Figure 53). The highest rate in both males and females occurred in the 5–9 year age group (530 and 573 per 100,000 respectively). The largest difference in sex distribution occurred in the 25–29, 30–34 and 35–39 year age groups where rates in females were 2 times that of males, likely representing the increased health seeking behaviour noted in adult females compared with males.⁴¹

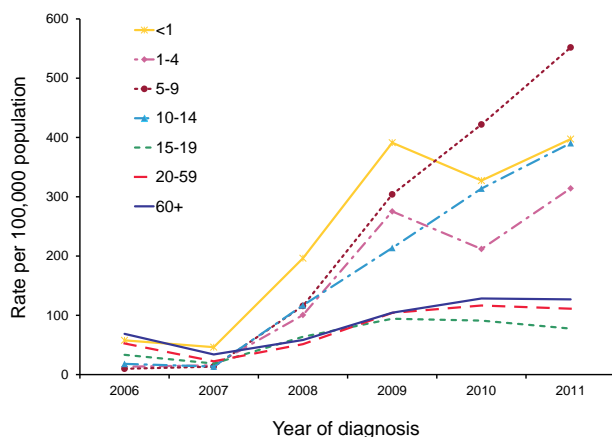
Figure 53: Notification rate for pertussis, Australia, 2011, by age group and sex



Rates in 2011 varied widely with age. Children less than 15 years of age had a higher rate (422 per 100,000) than those adolescents and adults 15 years of age or over (112 per 100,000) representing a rate ratio of 3.8:1. This is consistent with the trend of increasing rates amongst children during this epidemic period but differs with the pre-epidemic years in which adults had a higher rate relative to children (rate ratios of 0.7:1, 0.3:1 and 0.5:1 respectively for 2005, 2006 and 2007).

Between 2006 and 2007, a period inclusive of the last national epidemic in 2005–2006, rates in all age groups were either trending down or remaining relatively constant and were closely clustered. Since 2007, rates have been increasing and most markedly amongst those less than 15 years of age (Figure 54). In 2011, rates increased in all age groups less than 15 years of age compared with 2010, particularly amongst those in the 1–4 year age group whose rate increased by 48% from 212 per 100,000 in 2010 to 314 per 100,000 in 2011. The highest age group rate remained in the 5–9 year age group, 552 per 100,000, a 31% increase compared with the 422 per 100,000 reported in 2010. In contrast, rates decreased amongst all age groups over 15 years of age including a 15% decrease in the 15–19 year age group from 91 per 100,000 in 2010 to 78 per 100,000 in 2011.

Figure 54: Notification rate for pertussis, Australia, 2006 to 2011, by age group



Vaccination status

Pertussis 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 2003.⁵⁵ Immunity to disease decreases over time post-vaccination with estimates of protection remaining for 4–12 years.⁵⁶ 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 2011, some infants were given 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 to those less than 5 years of age. This age group made up 12.6% (n=4,865) of all notified cases in 2011.

Information on vaccination status was available for 92% (n=4,481) of all cases in children less than 5 years of age; 67% (n=2,989) were fully vaccinated for age, 18% (n=797) were partially vaccinated for age and 11% (n=500) were not vaccinated. Four per cent (n=195) were less than 6 weeks of age and therefore too young to be vaccinated.

Discussion

Pertussis was the most commonly notified vaccine preventable illness in Australia in 2011 reflecting the ongoing epidemic activity across the country in this year. Epidemics of pertussis occur at regular intervals of approximately 3 to 5 years on a back-

ground of endemic circulation in Australia.⁵⁷ The timing of this epidemic activity was not uniform across the country. States and territories experienced peak levels of pertussis at varying intervals as evidenced in 2011 when South Australia had its lowest rate since 2008, while the Australian Capital Territory, New South Wales, Queensland, Victoria and Western Australia all experienced the highest rates since the epidemic began.

In vaccinated populations, outbreaks of pertussis tend to be smaller with less mortality and morbidity than in unvaccinated populations.¹⁴ Despite the large number of cases reported in Australia throughout this epidemic period, there does not appear to have been a concurrent increase in pertussis related mortality.⁵⁸ While pertussis can affect people of any age, infants are at highest risk of more severe disease as adequate immunity is not achieved through infant vaccination until at least the second vaccine dose has been administered at 4 months of age.⁵⁹ In Australia during this epidemic period, very young un-immunised infants or incompletely immunised children accounted for the majority of severe disease requiring hospitalisation.⁶⁰

The causes of this epidemic are likely to be multifactorial. A widespread shift in diagnostic practice associated with the increased use of PCR for pertussis diagnosis in all age groups^{61,62} and increased case ascertainment during the epidemic period both serve to amplify the number of reported cases. Additional contributory factors may also include waning immunity levels in the vaccinated population including amongst children following their booster vaccination at 4 years of age,^{63,64} reduced vaccine efficacy of the acellular vaccine compared with the whole cell vaccine,⁶⁵ the removal of the 18-month dose from the routine schedule⁶⁶ and adaptation of *Bordetella pertussis* to the acellular vaccine.⁶⁷

Strategies to reduce pertussis infection in young children, particularly those less than 6 months of age, continued in 2011. In February 2001, the Australian Technical Advisory Group on Immunisation (ATAGI) endorsed recommendations to bring forward the first dose of the pertussis-containing vaccine from 8 weeks to 6 weeks and schedule the fifth (adolescent booster) dose at 11 to 13 years of age to better protect siblings, especially newborns.⁶⁸ States and territories continued to provide ongoing public awareness campaigns and most extended funding during 2011 for booster vaccination programs for parents and carers of infants. ATAGI also discussed the United States Centers for Disease Control and Prevention Advisory Committee on Immunization Practices recommendation to vaccinate pregnant women but concluded that while there is indirect evidence that maternal immunisation would be

beneficial, further data on safety and efficacy would be required before it could recommend this as a routine option.⁶⁹

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 but in less than 1% of cases the virus may invade the nervous system and cause acute flaccid paralysis (AFP).¹⁴

In 2011, there were no notifications 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 2011 Australia achieved for the fourth consecutive year in a row. More details can be found in the annual report of the Australian National Polio Reference Laboratory published in the CDI.

Rubella and congenital 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 that 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.¹⁴

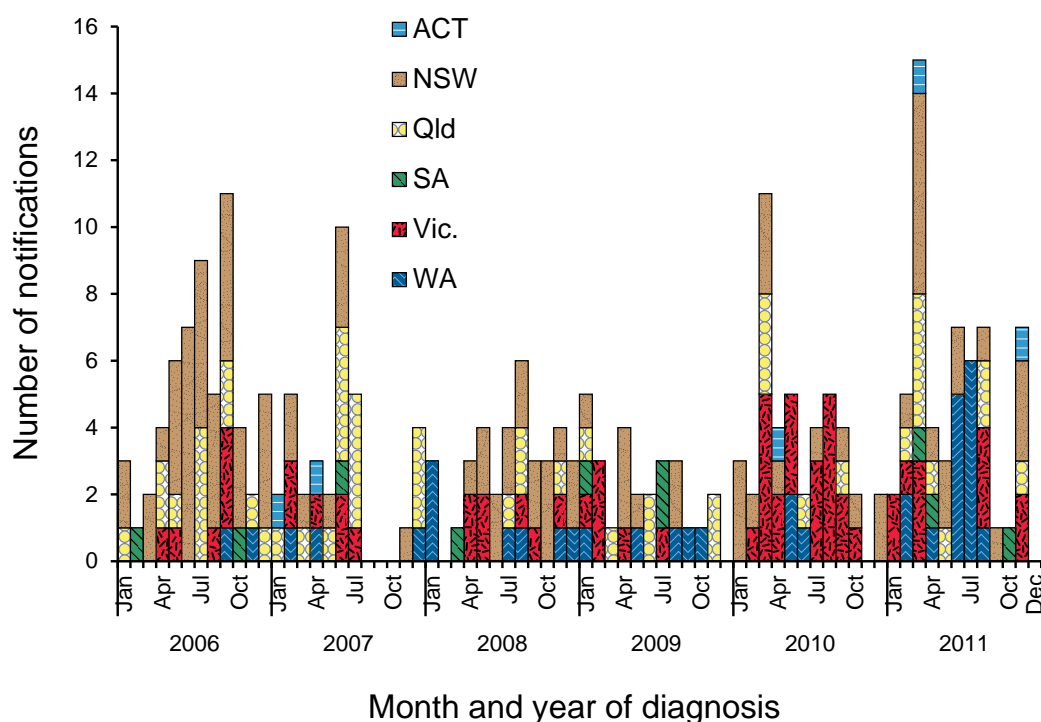
Epidemiological situation in 2011

In 2011, there were 58 notifications of rubella; a rate of 0.3 per 100,000 and 1.5 times the notification rate 5-year mean. The increase in cases in 2011 was not associated with any particular outbreak and was likely due to the sporadic nature and overall small number of cases reported annually (Figure 55). There were no cases of CRS reported in 2011. Indigenous status was recorded in 78% of cases, one of which was reported as Indigenous.

Source of infection

In 2011, a quarter of the cases were reported as being imported from overseas (26%, n=15). The remaining cases (n=43) were reported as being locally acquired with the original source of infec-

Figure 55: Notifications of rubella, Australia, 2006 to 2011, by month of diagnosis

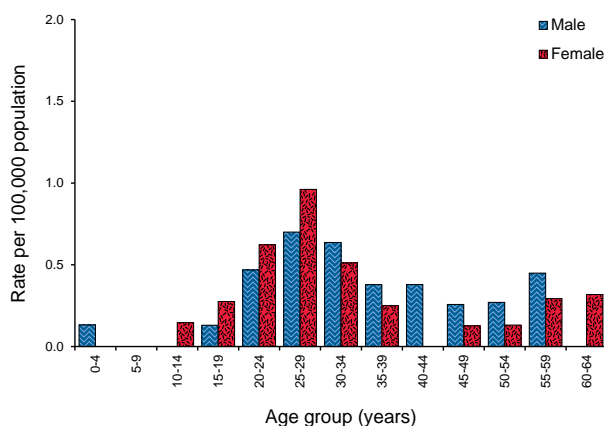


tion unknown. The majority of imported cases were from Asia, South East Asia (n=9), China (n=3) and India (n=1).

Age and sex distribution

The male to female ratio of notified cases in 2011 was 1.1:1 comprising 30 males and 28 females. Females had higher rates than males between 10 and 29 years of age and in the 60–64 year age group but males predominated in all other age groups (Figure 56). The highest rates for both males and females occurred in the 25–29 year age group, 0.7 per 100,000 and 1.0 per 100,000 for males and females respectively.

Figure 56: Notification rate for rubella, Australia, 2011, by age group and sex



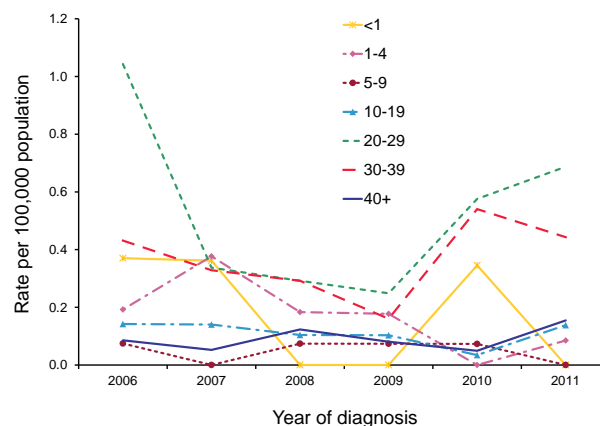
The majority of cases cluster around the young adult age groups with 74% of cases aged between 20 and 49 years of age and a median age of 31 years. The majority (75%) of female cases were notified in women of child-bearing age ranging from 15 to 36 years of age.

In 2011, an increasing trend was evident in the 20–29 year age range where the rate increased from 0.2 per 100,000 in 2009 to 0.7 per 100,000 in 2011, a 176% increase (Figure 57).

Vaccination status

A single dose of rubella vaccine produces an antibody response in more than 95% of recipients and while antibody levels are lower than after natural infection, they are shown to persist for at least 16 years in the absence of endemic disease.⁴⁴ Rubella vaccine is included in the combined MMR vaccine and provided under the NIP schedule at 12 months and 4 years of age.

Figure 57: Notification rate for rubella, Australia, 2006 to 2011, by age group



Information on vaccination was available for 40% (n=23) of rubella cases, 57% (n=13) of which were reported as not vaccinated and 43% (n=10) as vaccinated. Six of the 10 vaccinated cases were reported as receiving 1 dose of a rubella-containing vaccine and 1 case had reportedly received 2 doses. Dose information was not available for the remaining 3 cases.

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.¹⁴ 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.⁴⁴

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. 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 do so.⁴⁴ Results from the 2006 Adult Vaccination Survey indicate that uptake of this booster vaccine is likely to be low and decrease with increasing age

with 67% of adults in the 50–64 year age group (the oldest age group for which data were available) having been vaccinated in the previous 10 years.⁴⁴

Epidemiological situation in 2011

In 2011, there were 3 notifications of tetanus reported, which was consistent with the low numbers of this disease notified in recent years (Table 2). Because laboratory confirmation of tetanus is usually not possible, notification of cases relies on reports from clinicians, resulting in the potential for under reporting.³⁸ There were 2 male and 1 female cases, aged 84, 18 and 75 years respectively. One case had last been vaccinated 63 years earlier, the 18-year-old was of unknown vaccination status and the remaining case was not vaccinated.

Varicella zoster virus infections

The varicella zoster virus (VZV) is a highly contagious member of the herpesvirus family and causes 2 distinct illnesses: chickenpox (or varicella) following initial infection and shingles (or herpes zoster). Shingles occurs following re-activation of latent virus in approximately 20%–30% of cases, most commonly after 50 years of age.¹⁴

In 2006, CDNA agreed to make 3 categories of VZV infection nationally notifiable: chickenpox, shingles and varicella infection unspecified. By 2009 all jurisdictions were notifying VZV to NNDSS with the exception of New South Wales, where VZV is not notifiable.

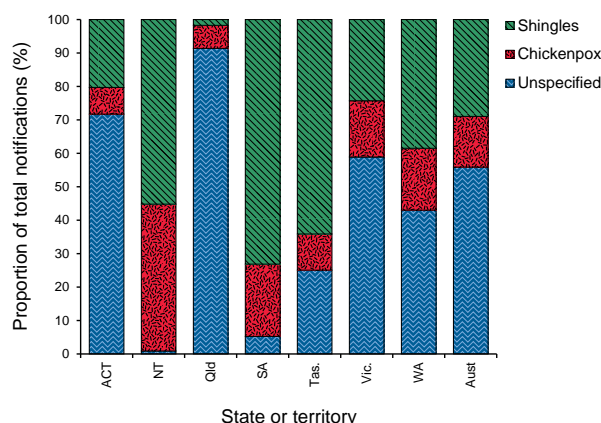
Epidemiological situation in 2011

In 2011, there were 13,808 notifications of VZV infection from the 7 reporting jurisdictions. This was 16% more than the 11,877 notified in 2010 and continues an upward trend in notifications since 2009. In 2011, 56% (n=7,715) of cases were reported as unspecified varicella infection, 29% (n=3,999) as shingles and 15% (n=2,094) as chickenpox (Figure 58). Although varying by jurisdiction, the VZV unspecified proportion of all VZV notified cases continued its downward trend accounting for 56% of cases in 2011 compared with 60% in 2010 and 62% in 2009.

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.

Figure 58: Proportion of notifications of varicella zoster virus unspecified, chickenpox and shingles, 2011, by state or territory*



* Excluding New South Wales.

Epidemiological situation in 2011

There were 7,715 notifications of unspecified VZV infections in 2011; a rate of 50 per 100,000 and an 8% increase in notifications compared with 2010.

The highest rate of unspecified VZV was reported from Queensland at 87 per 100,000 (n=4,002) followed by Western Australia and Victoria with 43 per 100,000 each (n=1,007 and n=2,409 respectively). VZV unspecified rates should be interpreted with caution as they are directly dependent on the jurisdictional practice of following-up laboratory notifications.

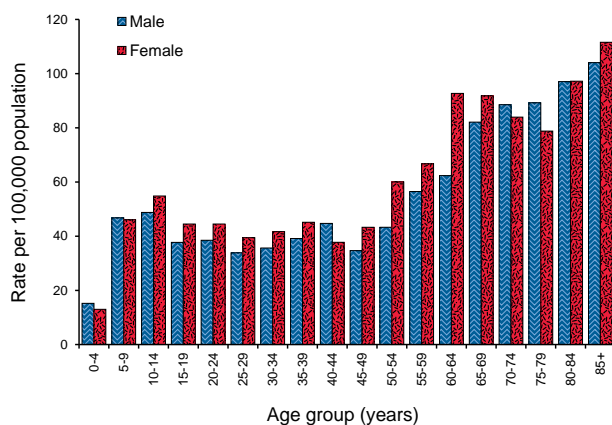
Age and sex distribution

The male to female ratio in the unspecified VZV notifications was 0.9:1 with females having an overall higher rate of notification with 54 cases per 100,000 compared with 47 per 100,000 in males and predominating across the majority of age groups. The highest rates occurred in the 85 years or over age group for both males, 112 per 100,000, and females, 104 per 100,000. The lowest rates were in the 0–4 year age group, likely reflecting the practice of increased follow up amongst children to determine clinical presentation (Figure 59).

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 a patient with chickenpox or shingles infection. Chickenpox is usually a mild disease of childhood; however, complications occur in approximately 1% of cases. It is more severe in

Figure 59: Notification rate for varicella zoster virus infection (unspecified), Australia,* 2011, by age group and sex



* Excluding New South Wales.

adults and in individuals of any age with impaired immunity, in whom complications, disseminated disease, and fatal illness can occur.⁴⁴

Epidemiological situation in 2011

In 2011, there were 2,094 notifications of chickenpox; a rate of 14 per 100,000 and a 20% increase in notifications compared with 2010. The highest rate, 64.2 per 100,000 was reported from the Northern Territory (n=148), followed by South Australia, 29 per 100,000 (n=477) reflecting the increased case ascertainment in these jurisdictions compared with others.

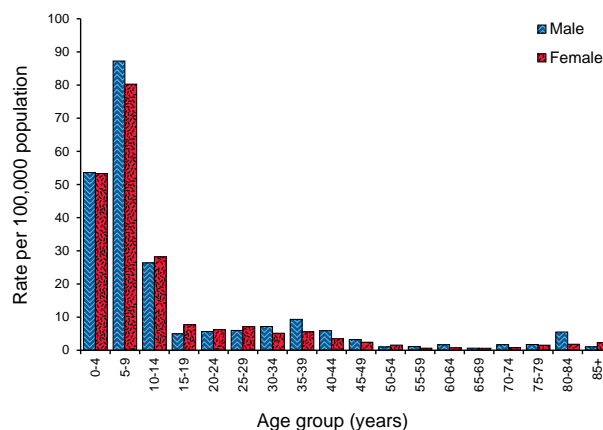
Age and sex distribution

The male to female ratio in 2011 was 1:1 although there was some slight variation, particularly in the older age groups where reported case numbers were smaller. Sixty-three per cent of cases (n=1,328) occurred in children aged less than 10 years. The 5–9 year age group had the highest notification rate amongst both sexes and all age groups, 87 per 100,000 for males and 80 per 100,000 for females (Figure 60). Although higher rates amongst children compared with adults is expected for chickenpox, they also reflect the jurisdictional practice of not following up adult cases.

Vaccination status

In November 2005, the monovalent varicella zoster vaccine was added to the NIP 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 2011, children born in 2004 and eligible for the 18-month dose would be 7 years of age or younger and as follow-up of cases does not routinely occur in

Figure 60: Notification rate for chickenpox, Australia,* 2011, by age group and sex



* Excluding New South Wales.

those older than 7 years, and analysis of vaccination status is restricted to this cohort. Information was available for 51% (n=525) of the 1,028 children less than 8 years of age. Thirty-one per cent (n=165) were vaccinated and 69% were either not vaccinated (n=126) or less than 18 months of age and ineligible for vaccination (n=234).

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 itching, tingling, or severe pain in the affected dermatome. In the majority of patients, shingles is an acute and self-limiting disease but complications develop in approximately 30% of cases, the most common of which is chronic severe pain or post-herpetic neuralgia.¹⁴

Epidemiological situation in 2011

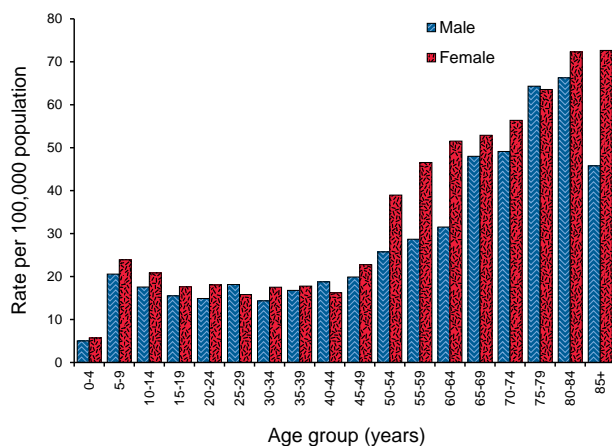
There were 3,999 notifications of shingles reported to NNDSS in 2011; a rate of 26 per 100,000 and a 34% increase compared with 2010. The highest rates of shingles occurred in South Australia, 97 per 100,000 (n=1,614) and the Northern Territory, 81 per 100,000 (n=186). High rates in these jurisdictions likely reflect their increased case ascertainment compared with others.

Age and sex distribution

There were more female cases (n=2,234) than males (n=1,764); a ratio of 0.8:1. As expected, rates

increased with age with the highest rate for males in the 80–84 year age group, 66 per 100,000 and in females in the 85 years or over age group, 73 per 100,000 (Figure 61).

Figure 61: Notification rate for shingles, Australia,* 2011, by age group and sex



* Excluding New South Wales.

Vectorborne diseases

Vectorborne diseases are infections transmitted by arthropods such as mosquitoes and ticks. A vectorborne disease may involve a simple transfer via the arthropod, or may involve replication of the disease-causing organism in the vector.¹⁴ Vectorborne diseases of public health importance in Australia listed in this chapter are; arbovirus not elsewhere classified (NEC), Barmah Forest virus (BFV) infection, dengue virus (DENV) infection, Japanese encephalitis virus (JEV) infection, Kunjin virus (KUNV) infection, malaria, Murray Valley encephalitis virus (MVEV) infection and Ross River virus (RRV) infection. The vectorborne diseases yellow fever virus (YFV) infection, plague and certain viral haemorrhagic fevers are listed under quarantinable diseases. The National Arbovirus and Malaria Advisory Committee (NAMAC) provides expert technical advice on vectorborne diseases to the Australian Health Protection Principal Committee through the CDNA. NAMAC provides a detailed report of vectorborne diseases of public health importance in Australia by financial year.⁷⁰

Alphaviruses

Viruses in the genus *Alphavirus* that are notifiable in Australia are BFV and RRV. These viruses are unique to the Australasian region.⁷¹ Infection can cause a clinical illness, which is

characterised by fever, rash and polyarthrits. The viruses are transmitted by numerous species of mosquito that breed in diverse environments.⁷² The alphavirus chikungunya is not nationally notifiable, and thus not included in this annual report, but it is notifiable in all states and territories except the Australian Capital Territory, and states and territories send information about cases to the Commonwealth for national collation and analysis.^{70,73}

The national case definitions for RRV and BFV require only a single IgM positive test to one of them, in the absence of IgM to the other.⁷⁴ False positive IgM diagnoses for BFV in particular are a known issue, and it is unclear what proportion of notifications might represent true cases. There was a large increase in notifications of BFV nationally subsequent to this reporting period (occurring from October 2012), which is suspected to be due to false positive notifications. This is under investigation and the laboratory case definition is under review.

Barmah Forest virus infection

Epidemiological situation in 2011

In 2011, there were 1,870 notifications of BFV infection, for a rate of 8.3 per 100,000 population. This compares with a 5-year mean of 1774.0 notifications and a 5-year mean rate of 8.3 per 100,000.

Seasonality and place of acquisition

The seasonality of BFV notifications is less marked than for RRV, and a high proportion of interseasonal notifications are thought to be due to false positive diagnoses. Peak notification of BFV during the period 2006 to 2011 was between January and April, and 47% of cases were diagnosed during these months (compared with 57% for RRV).

Most notifications of BFV infection are from Queensland and New South Wales (78% of all cases from 2006 to 2011), but rates are highest in the Northern Territory. The number of BFV notifications increased markedly in Victoria between December 2010 and March 2011, and the notification rate for 2011 was 4.9 times the 5-year mean (Figure 62).

Age and sex distribution

BFV was most frequently reported in middle aged adults (median 46 years, range 0–90 years). Age specific rates were highest amongst the 60–64 year age group for males and the 55–59 year age group for females (Figure 63).

Figure 62: Notifications of Barmah Forest virus infection, Australia, 2006 to 2011, by month and year and state or territory

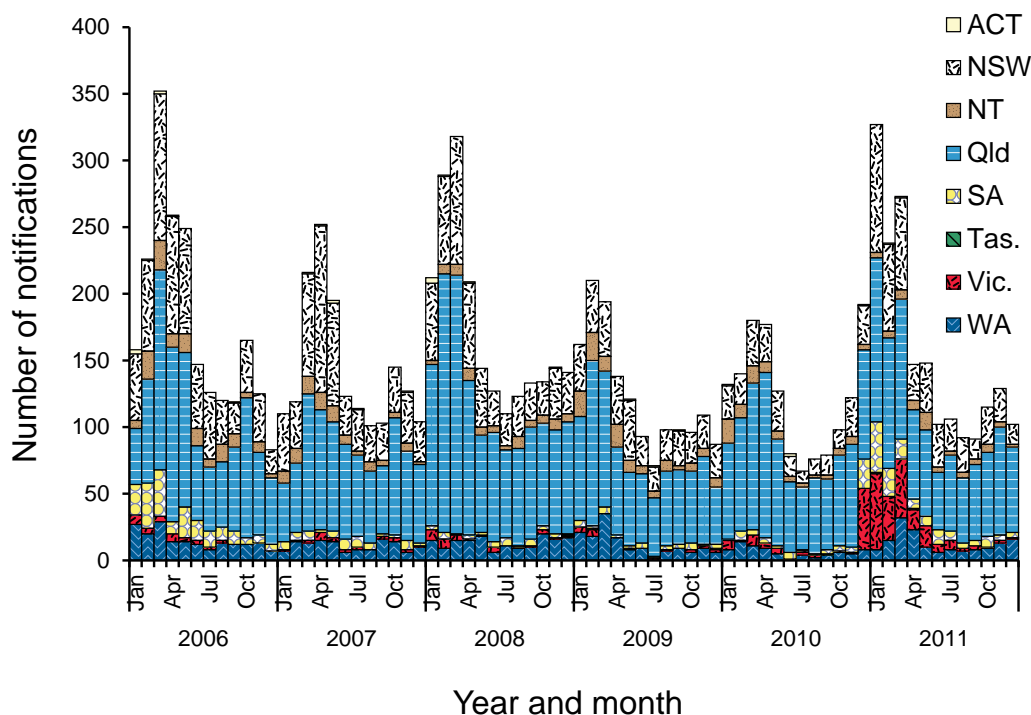
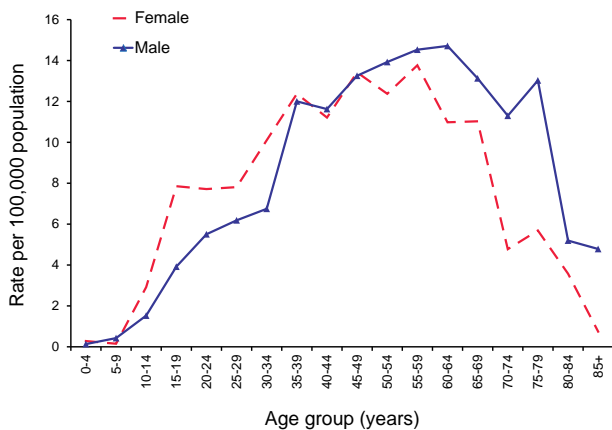


Figure 63: Notification rates for Barmah Forest virus infection, 2011, by age and sex (n=1,869)*



* Sex was not available for 1 case, and this case is excluded from the figure.

Ross River virus infection

Epidemiological situation in 2011

In 2011, there were 5,166 notifications of RRV, giving a rate of 22.8 per 100,000. This compares with a 5-year mean of 5060.4 notifications and a 5-year mean rate of 23.6 per 100,000.

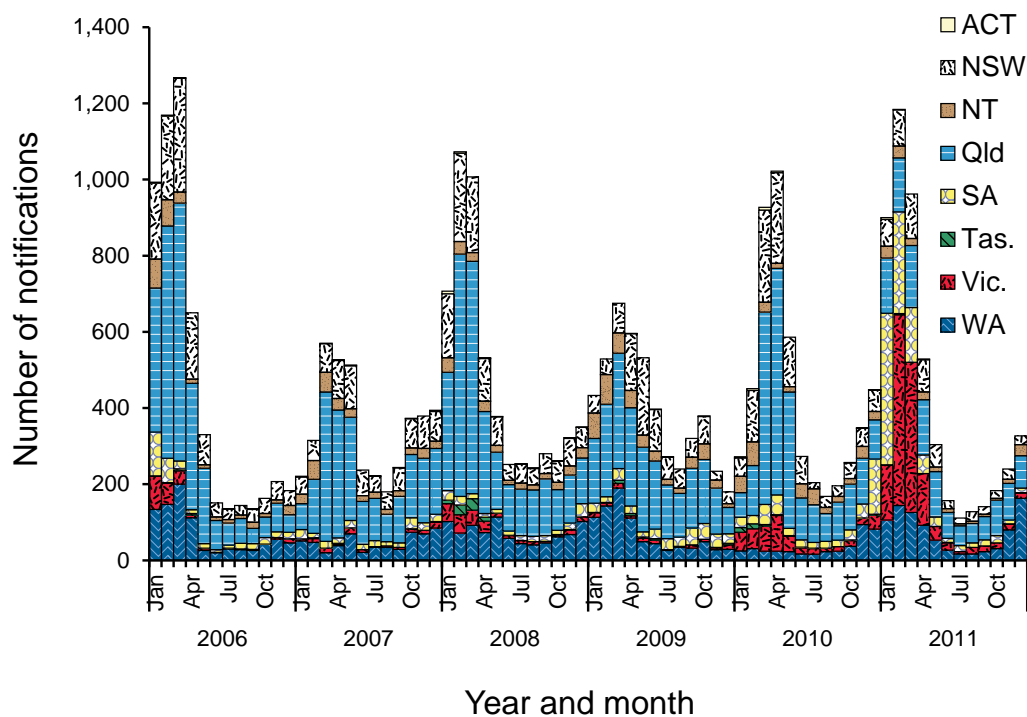
Seasonality

Peak transmission for RRV during the period 2006 to 2011 occurred between January and April, and 57% of cases were diagnosed during these months.

Between 2006 and 2011, nearly half of all RRV infections were from Queensland (44% of all cases), but rates were highest in the Northern Territory. Significant increases in the number and rate of reported cases were noted in Victoria and South Australia (Figure 64), where rates were 6.0 and 3.2 times the 5-year mean, respectively.

Over the spring and summer of 2010–11 the south-east of Australia experienced unusually wet weather and flooding resulting in increased mosquito and wild bird numbers. The noted increases in reporting of RRV occurred in the context of widespread evidence of seroconversions in sentinel chickens to flaviviruses and outbreaks of arboviral disease (KUNV and RRV) in horses, and equine cases were widely distributed across Victoria and New South Wales, and also in south-eastern parts of South Australia and Queensland and south-western parts of Western Australia.⁷⁵ Between January and June 2011, there were 982 clinically apparent cases of arboviral disease in horses and 91 horses died.⁷⁵ RRV infections predominated in equines in Victoria, and formed a significant proportion of infections in South Australia.⁷⁵

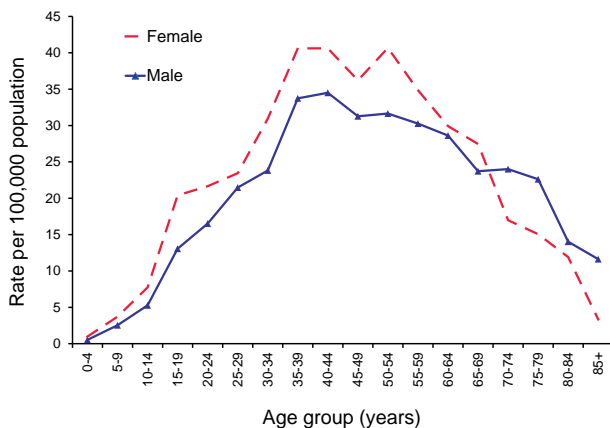
Figure 64: Notifications of Ross River virus infection, Australia, 2006 to 2011, by month and year and state or territory



Age and sex distribution

RRV was most frequently reported in middle aged adults (median 43 years, range 0–92 years). Age specific rates were highest amongst the 50–54 year age group for females, and the 40–44 year age group for males (Figure 65).

Figure 65: Notification rates for Ross River virus infection, 2011, by age and sex (n=5,164)*



* Sex was not available for 2 cases, and these are excluded from the figure.

Flaviviruses

In Australia, flavivirus infections of particular public health importance are DENV, KUNV, MVEV and JEV. YFV is reported under Quarantinable diseases. Unspecified flavivirus infections are reported under arbovirus NEC. These infections are nationally notifiable.

DENV has 4 serotypes, each containing numerous genotypes, and the serotypes isolated from returning travellers (and thus involved in local outbreaks) vary by year and geographical region. Infection with 1 serotype probably confers lifelong immunity to that serotype,¹⁴ but subsequent infection with a different serotype is one factor thought to increase the risk of severe outcomes, along with the infecting serotype and genotype and host factors.^{14,76–78} The clinical illness is characterised by mild to severe febrile illness with fever, headache, muscle/joint pain and sometimes a rash. A minority of cases progress to severe dengue with haemorrhage and shock. *Aedes aegypti* is the major vector of DENV in Australia.

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness, but a small percentage of cases progress to encephalomyelitis of variable severity. *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 available to prevent JEV infection,⁴⁴ but there are no vaccines currently for DENV, MVEV or KUNV infection.

Arbovirus NEC

Epidemiological situation in 2011

In 2011, there were 24 notifications of arbovirus NEC, compared with an average of 18 cases during the previous 5 years. These notifications comprised chikungunya (1 case), flavivirus unspecified (14 cases), Kokobera (1 case), Stratford (1 case), and the infecting organism was unknown or not supplied for a further 6 cases (Table 14).

The majority of notifications in 2011 were from Victoria (14 cases), with the remainder being from Queensland (9 cases) and the Northern Territory (1 case). Information about the place of acquisition was available for 63% of cases (15/24), and all of these were acquired overseas.

The median age of cases was 30 years (range 15–72 years).

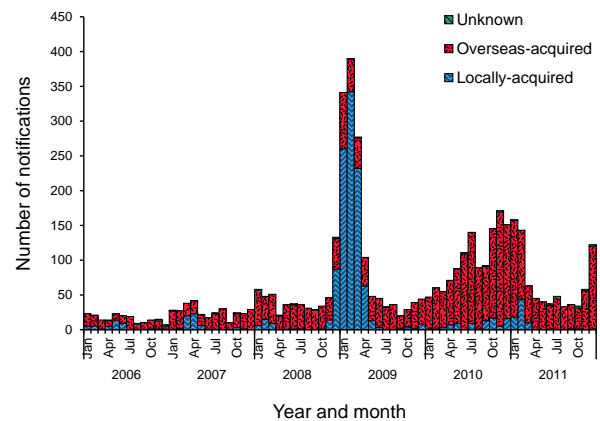
Dengue virus infection

Local transmission of dengue in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.^{44,79,80} Dengue is not endemic in North Queensland, but local transmission can occur upon introduction of the virus to the mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.⁸⁰

Epidemiological situation in 2011

There were 817 notifications of DENV infection in 2011, compared with 1,246 in 2010, and a 5-year mean of 737.8 cases. Most infections were acquired overseas (n=727) (Figure 66). There were 76 infections acquired in Australia. For a small number of cases (n=14), no information was supplied on the place of acquisition.

Figure: 66: Notifications of dengue virus infection, Australia, 2006 to 2011, by month and year and place of acquisition



Serotype of dengue virus infections

Historically, imported and locally-acquired cases of DENV have involved all 4 serotypes. In 2011, serotype information was available for 51% of notifications (413/817), which was unchanged compared with the 5-year mean (51%). In 2011, 37% (130/352) of overseas-acquired cases with a known serotype were DENV serotype 1, and 32% (112/352) were DENV 2, similar to the 5-year mean of 33% for each (Table 15). Locally-acquired cases were most commonly DENV 2 (51%, 39/76) followed by DENV 1 and DENV 4 (each comprising 10%, 10/76), DENV 3 (1%, 1/76), and for 16 notifications, the infecting serotype was unknown.

Seasonality and place of acquisition

There were 727 DENV infections known to have been acquired overseas in 2011, down from 1,104 in 2010, which was the largest number of overseas-acquired cases since the disease was made nationally notifiable in 1991. Between 2006 and 2009, the number of cases known to have been acquired overseas ranged between 142 and 474. In recent years, improved diagnostic techniques, in particular the availability of the rapid NS1 antigen detection kit, have improved detection and would have contrib-

Table: 14: Notifications of arbovirus NEC, Australia, 2011, by infecting organism and state or territory

Virus	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total
Chikungunya	0	0	1	0	0	0	0	0	1
Flavivirus	0	0	0	0	0	0	14	0	14
Kokobera	0	0	0	2	0	0	0	0	2
Stratford	0	0	0	1	0	0	0	0	1
Unknown	0	0	0	6	0	0	0	0	6
Total	0	0	1	9	0	0	14	0	24

Table 15: Serotype of overseas acquired dengue virus cases, 2011, by serotype and place of acquisition

Country	Serotype					Total
	DENV 1	DENV 2	DENV 3	DENV 4	Unknown/ untyped	
Indonesia	107	87	51	29	185	459
Thailand	5	3	17	2	58	85
India	2	2	1	0	22	27
Philippines	3	0	1	2	17	23
Malaysia	4	4	1	0	12	21
Papua New Guinea	3	4	2	0	6	15
Sri Lanka	2	0	1	0	10	13
Vietnam	1	3	1	0	8	13
East Timor	0	2	1	0	9	12
Bangladesh	1	2		0	8	11
Other countries	2	5	1	0	37	45
Unknown countries	0	1	0	0	2	3
Total	130	113	77	33	374	727

uted to the observed increase in reported numbers of overseas-acquired dengue in Australia,⁸¹ along with the dramatic re-emergence and geographical expansion of dengue overseas over the past 50 years and explosive outbreaks.⁷⁸

For 14 cases (2%), no information on the place of acquisition was available (Figure 66). Complete information on the country or region of acquisition was available for 98% (724/727) of overseas-acquired cases in 2011, compared with the 5-year mean of 82%. Cases acquired in Indonesia continue to account for the largest number and proportion of all notifications (Table 15), but in 2011, the number decreased to 459 cases acquired in Indonesia (63% of overseas acquired cases with a known country of acquisition), from 711 (63%) in 2010. Other frequently reported source countries in 2011 included Thailand, India and the Philippines.

Most of the 76 locally-acquired cases in 2011 were known to have been associated with one of 3 outbreaks of locally-acquired infection that occurred in Queensland in 2011. The largest of these was an outbreak of DENV 2 in Cairns and Innisfail, and was related to an importation from Papua New Guinea in 2010.⁷⁰ An outbreak of 13 cases of DENV 4 occurred in Innisfail between January and March, but the source of the outbreak was unknown and an outbreak of 9 cases of DENV 1 in Townsville was linked to an importation from Bali.⁸² One locally-acquired case in Western

Australia was health-care associated; a physician in Perth sustained a needle-stick injury whilst taking blood, 5 days prior to symptom onset.⁸³

The peak months for overseas-acquired dengue in 2011 were December, January and February, together accounting for 49% of cases. For locally-acquired cases, 95% of cases were diagnosed between January and March 2011.

Age and sex distribution

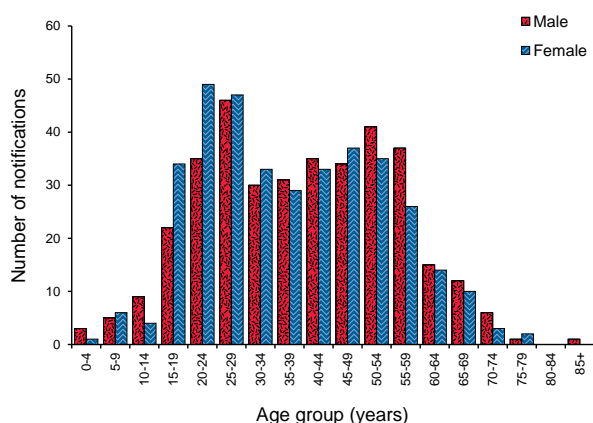
DENV infections acquired overseas in 2011 were most commonly reported amongst younger and middle aged adults (median 37.5 years, range 0–86 years), with a peak of notifications amongst males aged 20–24 and 25–29 years and females aged 25–29 years (Figure 67). Males and females each comprised 50% of overseas acquired cases. For locally-acquired cases, infections were more commonly reported amongst middle aged and older adults (median 43 years, range 2–78 years), with peak notifications amongst males and females aged 40–44 years (Figure 68). Males and females each comprised 50% of locally-acquired cases.

Kunjin virus infection

Epidemiological situation in 2011

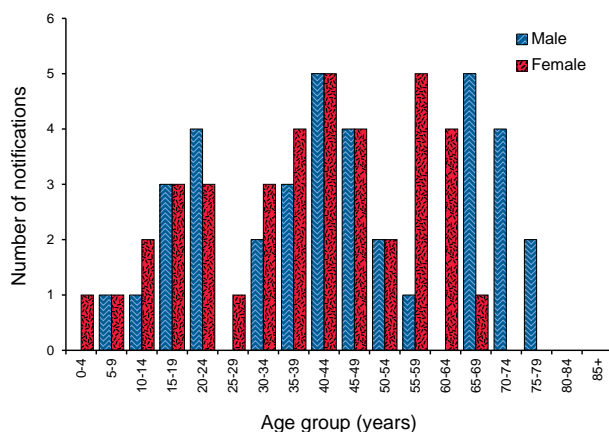
In 2011, there were 2 notifications of KUNV infections in Australia, compared with an average of 1.8 cases per year between 2006 and 2010.

Figure 67: Notifications of overseas-acquired dengue, 2011, by age and sex (n=723)*



* Age was not available for 1 case.

Figure 68: Notifications of dengue virus infection acquired in Australia, 2011, by age and sex (n=76)



The first case, with onset in April 2011, was a 60-year-old man from the Northern Territory who was IgM positive for KUNV and negative for MVEV, BFV and RRV. The infection was acquired in the Barkly region. The case was non-encephalitic, and recovered from infection.⁷⁰

The second case, with onset in December 2011, was in a 44-year-old female from New South Wales who seroconverted to KUNV and was negative for BFV and RRV. The specific region in which the case was likely to have been exposed was unclear, but the case was a resident of the South Coast of New South Wales.

There were only 11 cases of KUNV infection between 2006 and 2011, and the median age of these cases was 41 (range 20–80 years) and 64% of cases (7/11) were male (Table 16).

While there was a large number of equine cases of KUNV infection during the previously mentioned outbreak of arboviruses in horses (see RRV infections, Epidemiological situation), there was only 1 human case during the outbreak period (February to August 2011),⁷⁵ and the human case acquired the infection outside the areas where equine cases were reported.

Table 16: Notifications of Kunjin virus infection, Australia, 2006 to 2011, by month and year

Year	Month	State or territory of residence	Age group	Sex
2006	March	WA	25–29	Female
2006	April	WA	20–24	Male
2006	April	Qld	40–44	Female
2007	October	Vic.	55–59	Male
2008	July	Qld	30–34	Male
2009	February	Qld	25–29	Male
2009	March	NT	35–39	Female
2010	February	Qld	40–44	Male
2010	June	NT	80–84	Male
2011	April	NT	60–64	Male
2011	December	NSW	40–44	Female

Japanese encephalitis virus infection

There were no notifications of JEV infection in 2011. The last notified case was in 2008 and was acquired overseas.

Murray Valley encephalitis virus infection

Epidemiological situation in 2011

In 2011, there were 16 notifications of MVEV infection, compared with a 5-year mean of 1.4 cases. A confirmed case in a Canadian resident that was acquired in Australia and diagnosed in Canada was not notified to the NNDSS. Details of these cases have been reported elsewhere.⁷⁰

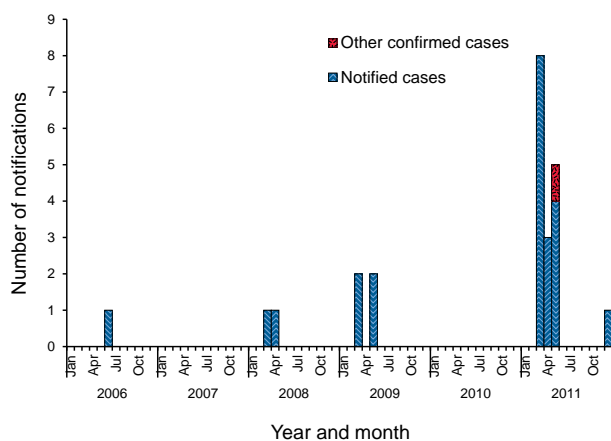
The outbreaks of MVEV infection in humans occurred in the context of the previously mentioned widespread evidence of seroconversions in sentinel chickens to flaviviruses and outbreaks of arboviral disease (KUNV and RRV) in horses (see RRV infections, Epidemiological situation).⁷⁵

Seasonality and place of acquisition

Twelve of the 16 notified cases were acquired in areas of regular enzootic viral activity (the Pilbara and Kimberley regions of Western Australia, and the northern two-thirds of the Northern Territory), or where epizootic disease activity is not unexpected (the Midwest and Goldfields region of Western Australia).⁷⁰ For the remaining 4 cases, the infection was acquired in areas where epizootic activity is rare (New South Wales, South Australia).

Of the confirmed cases, 15 (94%) had dates of onset between March and May 2011 (Figure 69).

Figure 69: Notifications of Murray Valley encephalitis virus infections, Australia, 2006 to 2011, by month and year and notification status



Age and sex distribution

In 2011, the median age of confirmed MVEV cases was 31 years (range 1–67 years) and there were equal numbers of cases in males and females.

Malaria

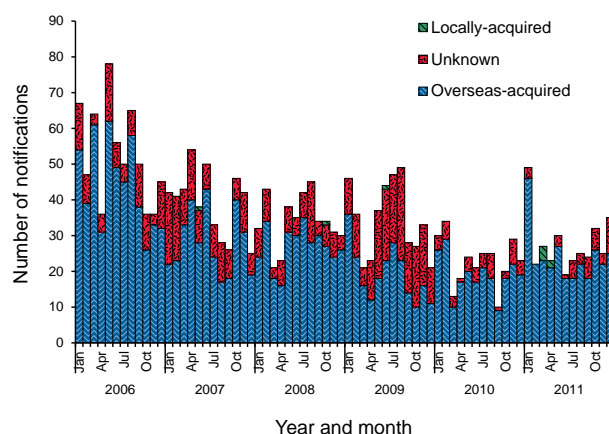
Malaria is caused by a protozoan parasite in the genus *Plasmodium*, and 5 species are known to infect humans; *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale* and *Plasmodium knowlesi*.^{14,84} Malaria is a serious acute febrile illness that is transmitted from person-to-person via the bite of an infected mosquito of the genus *Anopheles*. Malaria is the most frequently reported cause of fever in returned travellers world-wide.⁸⁵ Australia was declared free of malaria in 1981,⁸⁶ but suitable vectors are present in Northern Australia, and the area remains malaria-receptive. A recent case series in the Northern Territory showed that malaria cases

were reported in travellers returning from endemic areas, but also reflected current events such as military operations and increased refugee arrivals from particular areas.⁸⁷

Epidemiological situation in 2011

There were 411 notifications of malaria in Australia in 2011, an 18% decrease compared with a 5-year mean of 550.4 cases, and continuing the trend of decreasing notifications since 2005 (Figure 70). The largest number of cases was reported by Queensland (137 cases), but population rates were highest in the Northern Territory (10.0 per 100,000).

Figure 70: Notifications of malaria, Australia, 2006 to 2011, by month and year and place of acquisition



Seasonality and place of acquisition

Most infections in 2011 were acquired overseas, but 6 locally-acquired cases were associated with an outbreak in the Torres Strait in April 2011.⁸⁸ The last outbreak of locally-acquired infection on the mainland was in North Queensland in 2002.⁸⁹

Complete information on the country or region of acquisition was supplied for all but one of the cases known to have been acquired overseas, and the remaining cases were listed as overseas, country unknown. The most frequent country of acquisition was Papua New Guinea (28% of cases with complete information) and the most frequent infecting species was *P. falciparum* (reported in 54% of cases with complete information) (Table 17). No place of acquisition was supplied for cases that are classified as 'Unknown'.

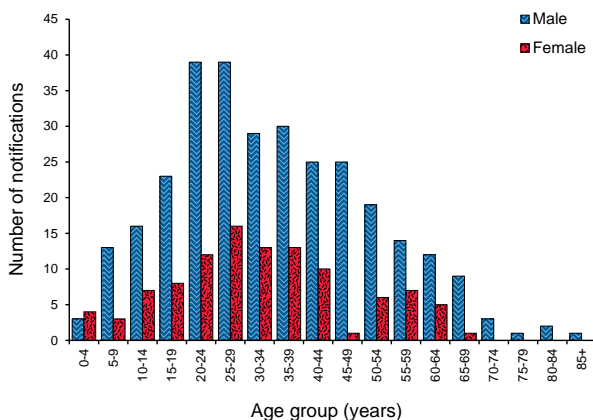
There was no discernible seasonality in notifications between 2006 and 2010, but in 2011, 13% of notifications were for cases diagnosed in January,

compared with 6% to 10% of cases each month over the previous 5 years. This appears to have been due to an increase in cases from Papua New Guinea (23/55 cases notified in January 2011). The outbreak of malaria reported in the Torres Strait related to people moving between Papua New Guinea and the Torres Strait and occurred in March and April 2011,⁸⁸ shortly after the observed increase in imported cases from Papua New Guinea in January 2011.

Infecting species for malaria infections

The infecting species was supplied for 97% (400/411) of cases in 2011 (Table 17). *P. vivax* was associated with Asia and the Pacific, whilst most cases acquired in African countries were *P. falciparum*.

Figure 71: Notifications of malaria, Australia, 2011, by age group and sex (n=409)*



* Two cases for whom sex was not supplied are excluded from the figure.

Age and sex distribution

In 2011, malaria was most commonly reported in males (74%, 303 of the 409 cases for whom sex was stated) with a peak of notifications in males in the 20–24 and 25–29 year age groups (Figure 71). The median age of cases was 32 years (range 2–85 years).

Zoonoses

Zoonoses are those diseases and infections that are naturally transmitted between vertebrate animals and humans.⁹⁰ Approximately 60%–70% of emerging human infectious diseases are zoonoses^{91,92,93} and more than 70% of emerging zoonoses originate from wildlife.⁹² 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’.⁹⁴

The zoonoses notifiable to the NNDSS included in this chapter are: anthrax, Australian bat lyssavirus (ABLV), lyssavirus (unspecified) infection, brucellosis, leptospirosis, ornithosis, Q fever, and tularaemia.

Several zoonoses notifiable to the NNDSS are included under other headings in this report. For example, *Salmonella* and *Campylobacter* infections are typically acquired from contaminated food and are listed under the gastrointestinal diseases section. Rabies is listed under Quarantinable diseases.

Anthrax

Anthrax is caused by the bacterium *Bacillus anthracis* and mainly causes cutaneous infection.

Table 17: Notifications of malaria, Australia 2011, by infecting species and country of acquisition

Place of acquisition	Malaria species					Total	
	<i>P. falciparum</i>	<i>P. malariae</i>	<i>P. falciparum</i> and <i>P. malariae</i>	<i>P. ovale</i>	<i>P. vivax</i>		<i>Plasmodium</i> unspecified
Papua New Guinea	34	4		2	55	3	98
India	2				41	3	46
Sudan	21			1		1	23
Uganda	18		1	3			22
Tanzania	21			1			22
Ghana	20						20
Indonesia	5				12		17
Sierra Leone	14	1			1		16
Australia	7						7
Other countries	55	5	1	4	30	3	98
Unknown	24	1	0	1	15	1	42
Total	221	11	2	12	154	11	411

However, it can also cause gastrointestinal and respiratory infections. Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts. It can be an occupational hazard for veterinarians, and agriculture, wildlife and industry livestock workers who handle infected animals or by-products.

In Australia, the areas of anthrax risk are well defined and include the northern and north-eastern districts of Victoria and central New South Wales.⁹⁵ Anthrax occurs only sporadically in livestock in the at-risk areas, and rare or isolated incidents or cases have historically occurred in Queensland, South Australia, Tasmania and Western Australia.⁹⁵

Epidemiological situation in 2011

There were no notifications of anthrax in 2011. Over the previous 10 years, only 3 human cases of anthrax were reported in Australia; in 2006, 2007 and 2010.^{96–98} All had domestic farm or animal related exposures and all were cutaneous anthrax. Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

There were no reports of anthrax in livestock in Australia in 2011, and the last reported case of anthrax in livestock was in November 2010.⁹⁹

Australian bat lyssavirus and lyssavirus (unspecified) infections

ABLV belongs to the genus lyssavirus, which also includes the rabies virus. Both invariably result in progressive, fatal encephalomyelitis in humans.¹⁰⁰ ABLV was identified in Australia in 1996^{101,102} and is present in some Australian bats and flying foxes. Australia is free of terrestrial rabies.

The best way to prevent ABLV infection is to avoid contact with bats. For people whose occupation (including volunteer work) or recreational activities place them at increased risk of being exposed to ABLV, rabies virus vaccine is effective in preventing infection. Pre-exposure vaccination with rabies virus vaccine is recommended for bat handlers, veterinarians and laboratory personnel working with live lyssaviruses.¹⁰³ Post-exposure prophylaxis for ABLV consists of wound care and administration of a combination of rabies virus vaccine and human rabies virus immunoglobulin (HRIG), depending on exposure category and prior vaccination or antibody status.^{44,103}

Epidemiological situation in 2011

There were no notifications of ABLV or lyssavirus (unspecified) in Australia in 2011. Subsequent to

this reporting period, a fatal case of ABLV infection was reported in Queensland in 2013, for a total of 3 cases of ABLV infection in humans (1996, 1998 and 2013). All cases occurred after close contact with an infected bat and all were fatal.^{104–106} In 2013, the Queensland Department of Agriculture, Fisheries and Forestry confirmed ABLV infection in 2 horses on a Queensland property. These were the first known equine cases of ABLV infection.^{107,108} The Bat Health focus group in the Australian Wildlife Health networks gathers and collates information from a range of organisations on testing of bats for ABLV. In 2011 there were 7 ABLV detections compared with 9 detections in bats during 2010.¹⁰⁹

There were also no notifications of rabies (see Quarantinable diseases chapter).

Brucellosis

Brucella species that can cause illness in humans include *Brucella melitensis* acquired from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989 and *B. melitensis* has never been reported in Australian sheep or goats.⁹⁵ Therefore, all 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. Eales et al (2010) found that feral pig hunting was the most common risk factor for infection for brucellosis cases in Townsville during 1996 to 2009.¹¹⁰

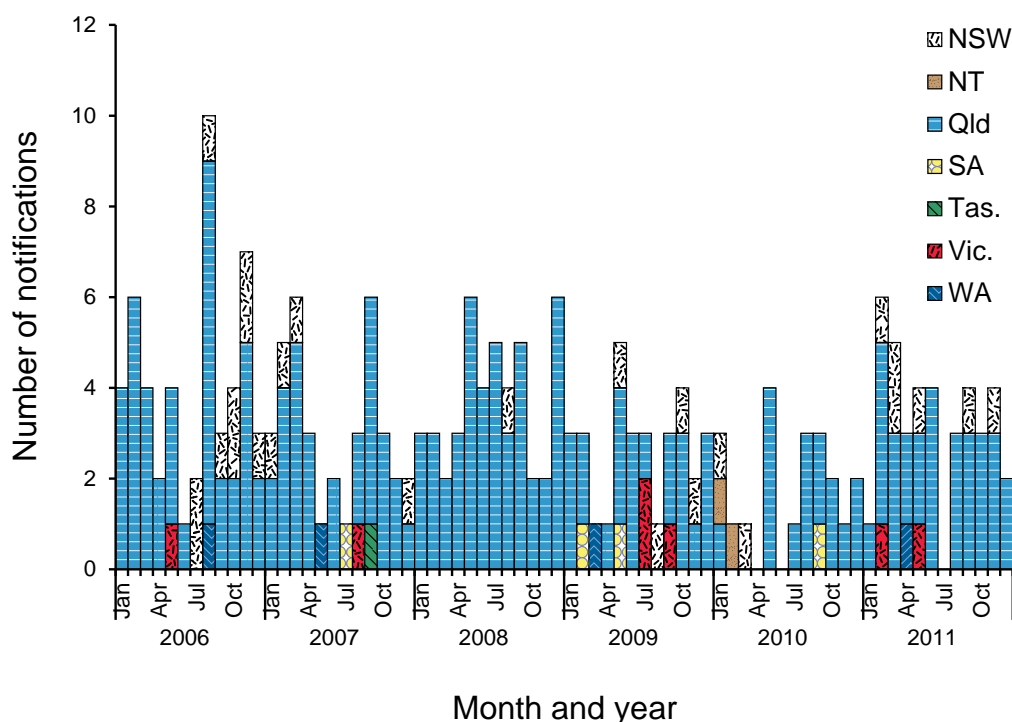
Internationally, brucellosis is mainly an occupational disease of farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.¹⁴

Epidemiological situation in 2011

In 2011, there were 39 notifications of brucellosis (a rate of 0.2 per 100,000) compared with the 5-year mean of 37 notifications between 2006 and 2010. Seventy-seven per cent of notifications were from Queensland (30/39) (Figure 72), a state-specific rate of 0.7 per 100,000. Since 1991, 84% of notifications have been from Queensland.

The species of the infecting organism was available for a third of notifications (n=13). Eight notifications were for *B. suis*, all of them from Queensland, with 7 of 8 males aged between 17 and 45 years. There were 5 overseas-acquired cases of *B. melitensis*, with the country of acquisition listed as India (n=2), Syria (n=1), Turkey (n=1) and an unspecified overseas country (n=1).

Figure 72: Notifications of brucellosis, Australia, 2006 to 2011, by month and year of diagnosis and state or territory*



* No notifications from the Australian Capital Territory, the Northern Territory, South Australia or Tasmania in 2011.

The median age of notified cases of brucellosis was 30 years (range 11–77 years) and 82% of cases (32/39) were male.

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 in certain agricultural sectors and swimming or wading in contaminated water).^{111,112} The last reported death in Australia attributed to leptospirosis was in 2002.¹¹³

Epidemiological situation in 2011

In 2011, there were 217 notifications of leptospirosis (a rate of 1.0 per 100,000), a 71% increase compared with the 5-year mean of 127.4 notifications (2006–2010). Cases were reported in all jurisdictions, with Queensland accounting for 72% (157/217) of notifications (Figure 73). A large increase was observed in leptospirosis notifications from Queensland in the first part of 2011. Much of this increase appears to be associated with extensive flooding experienced in central and southern Queensland between December 2010 and January 2011.^{114,115}

Age and sex distribution

The median age of leptospirosis notifications was 43 years (range 4–79 years) and 91% (197/217) of cases were male. The highest notification rate was observed in the 55–59 year age group for males (Figure 74).

Typing information

The WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis routinely conducts PCR-based serotyping for leptospirosis cases from Queensland (from whence the majority of cases are reported), and collates national data that may be submitted to the laboratory from other states or territories. These data may differ from that submitted to NNDSS. The WHO/FAO/OIE collaborating centre reported on 189 serotyped cases of leptospirosis nationally in 2011, of which 49% (93/189) were serovar Arborea, 10% (19/189) were Zanoni, 11% (22/189) were Australis, 14% (27/189) were Hardjo and the remainder were a range of serotypes, each representing 3% or fewer cases.

Typing information was available for 75% (163/217) of notifications to NNDSS, and of these, 46% were serovar Arborea.

Figure 73: Notifications of leptospirosis, Australia, 2006 to 2011, by month and year of diagnosis and state or territory

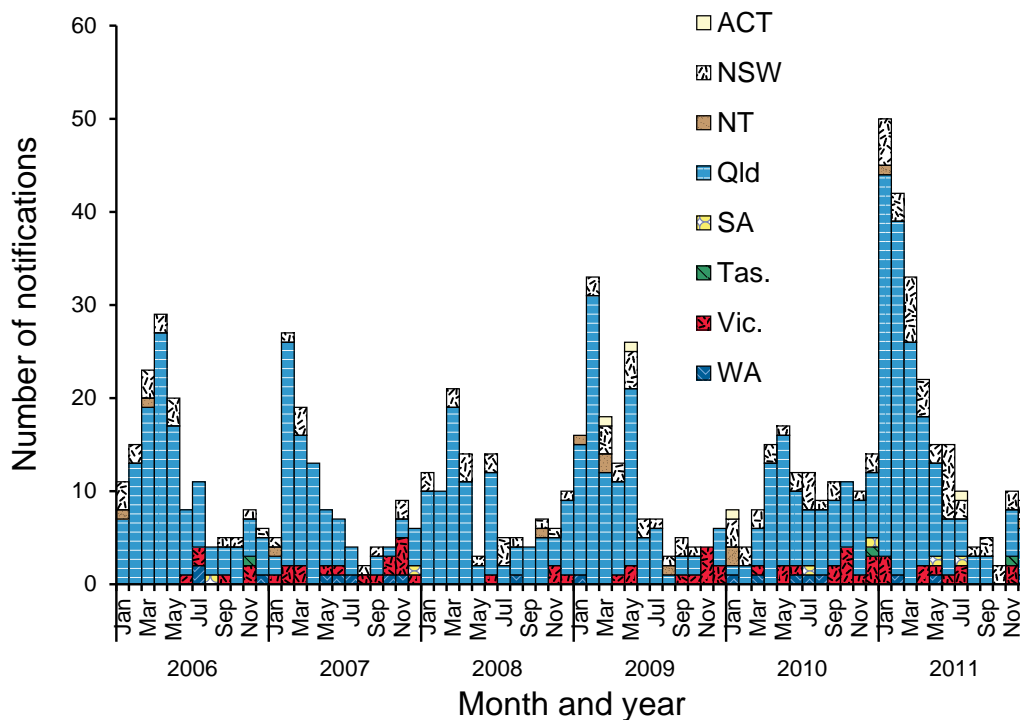
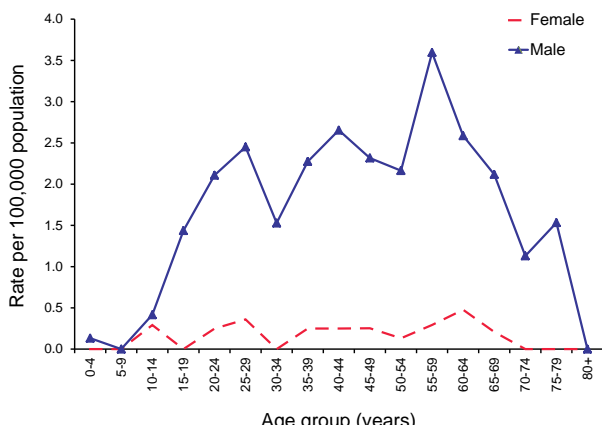


Figure 74: Notification rate for leptospirosis, Australia, 2011, by age group and sex (n=217)



Ornithosis

Ornithosis (or psittacosis) is caused by infection with the bacterium *Chlamydophila psittaci* and is transmitted to humans primarily from infected parrots of many species, but also poultry and a range of other birds.¹¹⁶ Transmission to humans can occur via the inhalation of contaminated dried faeces, nasal or eye secretions and dust from the feathers. Individuals at risk of contracting ornithosis include bird owners and those with occupational exposure to birds.¹¹⁷

Epidemiological situation in 2011

In 2011, there were 85 notifications of ornithosis (a rate of 0.4 per 100,000 population) compared with the 5-year mean of 96.8 notifications (2006 to 2010). The number of ornithosis notifications in 2011 was a 44% increase from 2010 (n=59), which was the lowest since 2001 (Figure 75).

In 2011, there were notified cases of ornithosis in New South Wales, Queensland, Tasmania, Victoria and Western Australia. The majority of notifications in 2011 were from Victoria (68%, 58/85), where a significant increase in case numbers was reported compared with 2010 (n=36) and 2009 (n=40).¹¹⁸

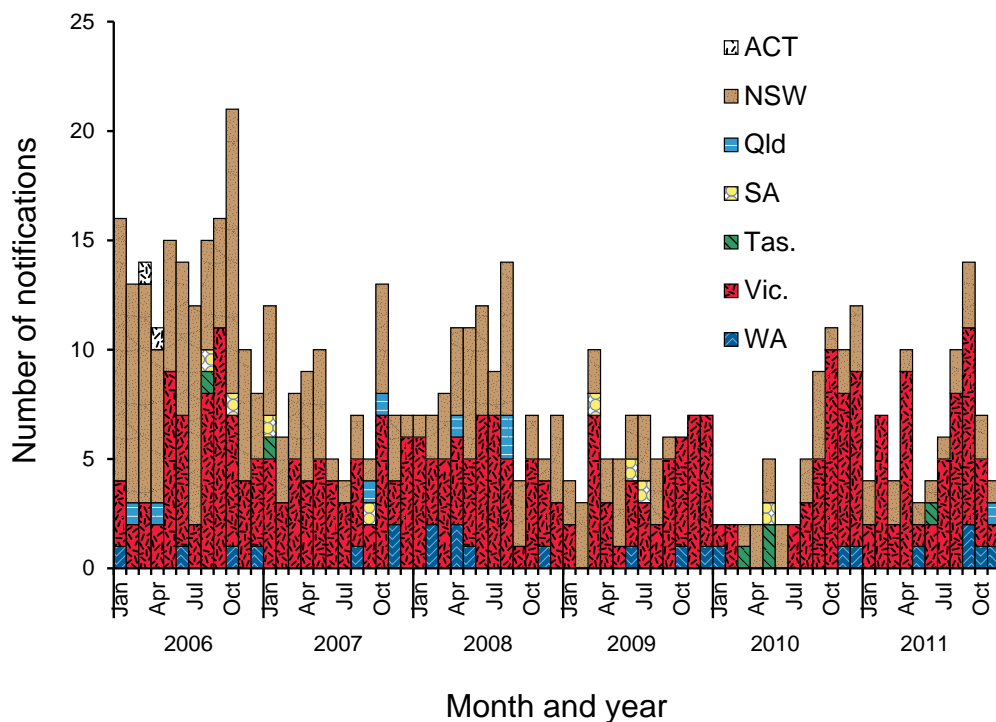
Age and sex distribution

The median age of ornithosis notifications was 54 years (range 0–87 years) and 61% (52/85) of notified cases were male.

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.¹¹⁹ Q fever is most commonly transmitted via the airborne route, where the organism is carried in dust contaminated

Figure 75: Notifications of ornithosis, Australia, 2006 to 2011, by month and year of diagnosis and state or territory*



* No notifications from the Australian Capital Territory, the Northern Territory or South Australia in 2011.

with tissue, birth fluids or excreta from infected animals.¹²⁰ Prior to the commencement of vaccination programs in Australia, approximately half of all cases in New South Wales, Queensland and Victoria were amongst abattoir workers.^{121,122}

The Australian Government funded the National Q Fever Management Program (NQFMP) between 2001 and 2006 for states and territories to provide free vaccine to at-risk groups (such as abattoir workers).¹²³

Adults at risk of Q fever infection, including abattoir workers, farmers, veterinarians, stockyard workers, shearers and animal transporters should be considered for vaccination. The administration of the Q fever vaccine requires a pre-vaccination screening test to exclude those recipients with a previous (unrecognised) exposure to the organism. A Q fever vaccine may cause an adverse reaction in a person who has already been exposed to the bacterium. Vaccination is not recommended for children under 15 years of age.⁴⁴

Epidemiological situation in 2011

In 2011, there were 338 notifications of Q fever (a rate of 1.5 per 100,000), compared with the 5-year mean of 375.2 notifications (2006–2010).

Between 1991 and 2001, and prior to the introduction of the NQFMP, Q fever notification rates ranged from between 2.5 and 4.9 per 100,000.¹²⁴ In 2011, the highest notification rates were from Queensland (3.6 per 100,000, n=164) and New South Wales (1.8 per 100,000, n=131). Cases also occurred in Victoria (n=24), Western Australia (n=10) and South Australia (n=7). There was 1 notification each from the Australian Capital Territory and the Northern Territory and none from Tasmania (Figure 76).

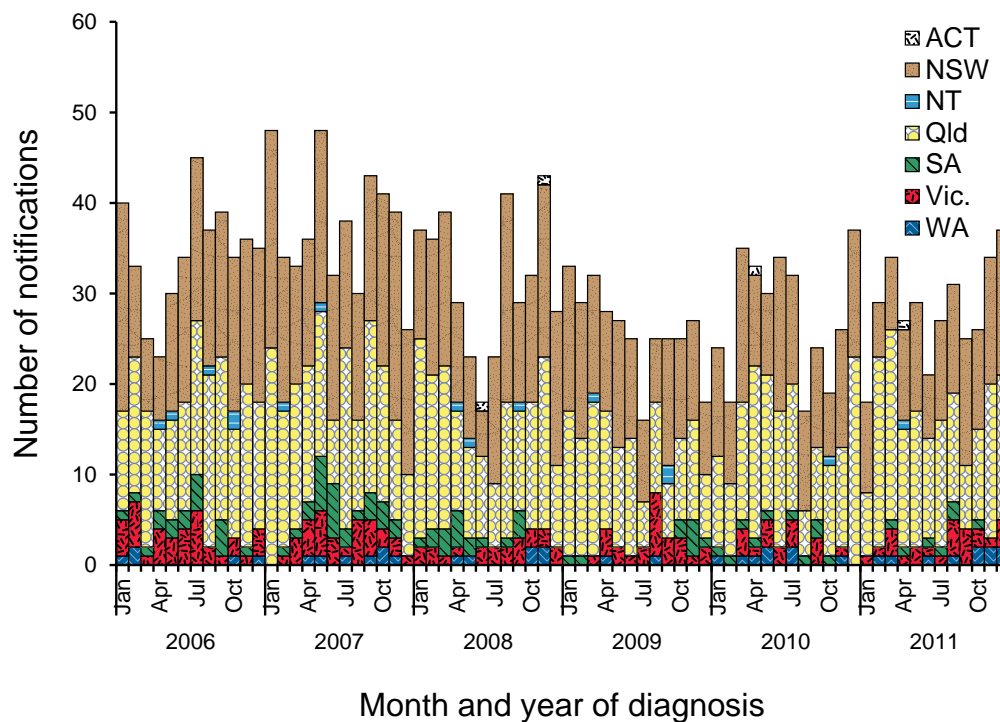
Age and sex distribution

The median age of Q fever notifications was 45 years (range 3–88 years) and 74% of cases (249/338) were male. The highest notification rate was observed in the 40–44 year age group for males (Figure 77).

Tularaemia

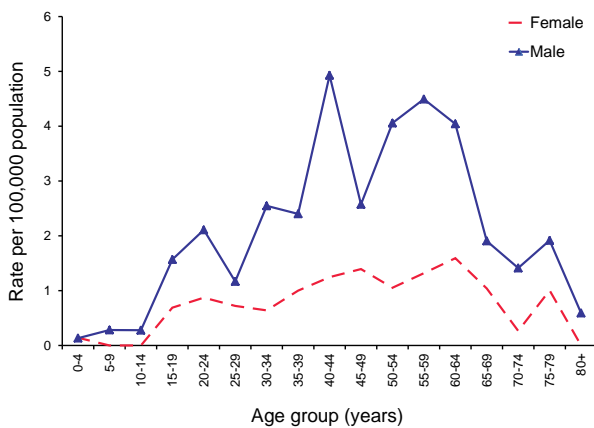
Tularaemia is caused by infection with the bacterium *Francisella tularensis*. The most common modes of transmission are through arthropod bites, handling infected animals, inhalation of infectious aerosols or exposure to contaminated food or water. Small mammals such as rodents, rabbits and hares are often the reservoir.¹²⁵

Figure 76: Notifications of Q fever, Australia, 2006 to 2011, by month and year of diagnosis and state or territory*



* No notifications from Tasmania in 2011.

Figure 77: Notification rate for Q fever, Australia, 2011, by age group and sex (n=338)



Epidemiological situation in 2011

In 2011, there were 2 notifications of tularaemia, both from Tasmania with exposure to sick or injured wildlife. This is the first time that *F. tularensis* type B had been detected in the Southern Hemisphere.¹²⁶ Both cases were in women who had been bitten by possums along the same stretch of road in a remote location, one in February and one in September 2011.¹²⁷

Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2011 and classified as 'other bacterial infections' in the NNDSS. A total of 1,928 notifications were included in this group in 2011, which accounted for less than 1% of all the notifications to NNDSS, an increase in cases and a similar proportion as notified in 2010 (n=1,852 and 1% of total).

Legionellosis

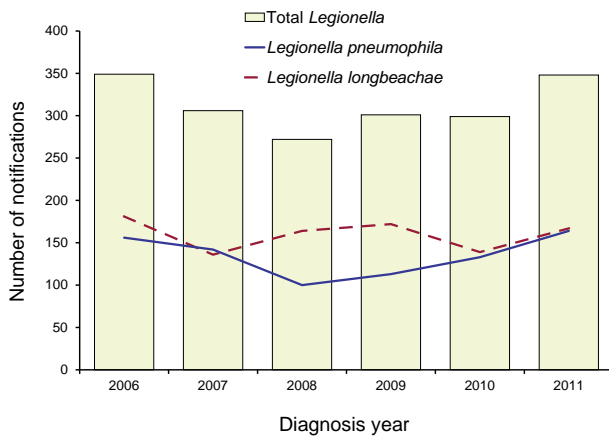
Legionellosis, caused by the bacterium *Legionella*, can take the form of either Legionnaires' disease, a severe form of infection of the lungs or Pontiac fever, a milder influenza-like illness. The species that are most commonly associated with human disease in Australia are *L. pneumophila* and *L. 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.

Epidemiological situation in 2011

In 2011, there were 348 notifications of legionellosis, representing a rate of 1.5 per 100,000.

Compared with the previous reporting period the overall number of legionellosis cases increased in 2011 by 16%. This number of annual notifications was the highest since 2006 (Figure 78).

Figure 78: Notifications of legionellosis, Australia, 2006 to 2011, by species



Data on the causative species were available for 95% (n=332) of cases reported in 2011. Of the cases with a reported species there were roughly equal proportions of *Legionella longbeachae* (n=167) and *L. pneumophila* (n=164) (Table 18). A single case was reported with an infective species of *L. bozemanii*.

Over the period 2006 to 2011, annual notifications of *L. longbeachae* ranged from 136 to 181 cases, while annual notifications of *L. pneumophila* ranged from 100 to 164 cases (Figure 78). Annual notifications of *L. pneumophila* have steadily increased since 2008.

Mortality data were available for 62% of notifications in 2011. There were 8 reported deaths due to legionellosis, which was an increase on the 7 deaths reported in 2010. Half of the deaths were associated with *L. pneumophila* infection and the

remaining half was associated with *L. longbeachae* (Table 18). Mortality data should be interpreted with caution given the large proportion of cases reported without death data to the NNDSS.

Geographical distribution

Jurisdictional-specific rates of legionellosis in 2011 varied from 1.0 per 100,000 in Queensland to 3.3 per 100,000 in Western Australia (Table 18).

Unlike the previous 3 years, the geographic distribution of *L. longbeachae* and *L. pneumophila* in 2011 aligned with longer historical trends.¹²⁴ *L. longbeachae* made up the majority of notifications in South Australia and Western Australia, while *L. pneumophila* was the most common infecting species in the eastern states (New South Wales, Queensland and Victoria).

Age and sex distribution

In 2011, legionellosis was predominantly seen in older males. Males accounted for the majority (67%) of the notifications of legionellosis resulting in a male to female ratio of 2:1. Overall, the age group with the highest notification rate was the 75–79 year age group (7 per 100,000). The highest age and sex specific rates were observed in men aged 75–79 years (11.5 per 100,000 population) and women aged 65–69 years (3.5 per 100,000, n=10) (Figure 79). The 8 cases that were reported to have died due to legionellosis ranged in age between 58 and 95 years (median 77 years); 7 deaths were males and 1 death was a female.

An infecting species analysis by age group shows that 91% of *L. longbeachae* notifications were reported in persons 40 years or over and is most predominant in the 75–79 year age group (3.6 per 100,000). Similarly 90% of *L. pneumophila* infections notified were in persons aged 40 years or over and is most predominant in the 80–84 year age group (3.6 per 100,000).

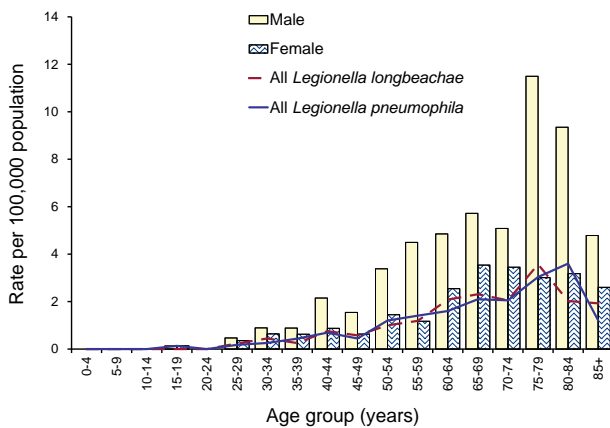
Table 18: Notifications of legionellosis, Australia, 2011, by species and state or territory

Species	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
<i>L. longbeachae</i>	0	32	3	20	30	5	12	65	167*
<i>L. pneumophila</i>	0	59	2	23	10	2	55	13	164†
<i>L. bozemanii</i>	0	0	0	0	0	0	1	0	1
Unknown species	4	4	0	2	0	0	6	0	16
Total	4	95	5	45	40	7	74	78	348
Rate (per 100,000)	1.1	1.3	2.2	1.0	2.4	1.4	1.3	3.3	1.5

* 4 deaths.

† 4 deaths.

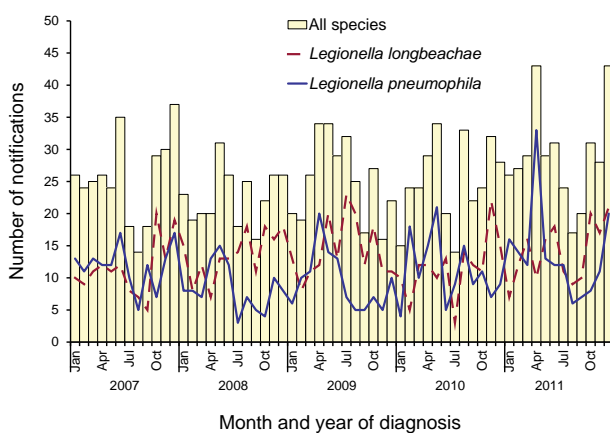
Figure 79: Notification rate for legionellosis, Australia, 2011, by age group and sex



Seasonality

In 2011, diagnoses of legionellosis were highest in April and December, with 43 cases notified in each month (Figure 80). *L. pneumophila* occurred most frequently in the autumn months, with 58 cases reported over the months March to May 2011. *L. longbeachae* cases peaked in spring 2011, with 47 cases reported over the months September to November 2011, the majority (n=20) of which occurred in October. These patterns seen in 2011 are consistent with peaks in notifications experienced in previous years.

Figure 80: Notifications of legionellosis, Australia, 2007 to 2011, by month of diagnosis and species



Place of acquisition

Place of acquisition was reported for 68% (n=235) of legionellosis cases notified in 2011. Of cases with a place of acquisition reported, most (n=218, 93%) were reported as having been acquired within

Australia. A small number (n=17) of cases was reported as acquired overseas. Indonesia (n=9) and China (n=3) were the most commonly reported overseas place of acquisition.

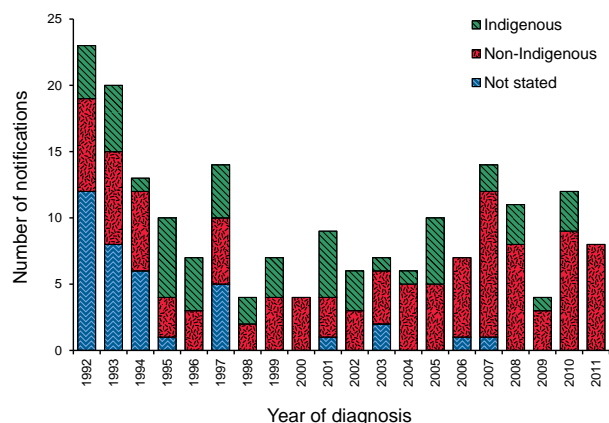
Leprosy

Leprosy is a chronic infection of the skin and peripheral nerves due to the bacterium *Mycobacterium leprae*. Leprosy is a rare disease in Australia. Its incidence world-wide is declining due to factors including economic development, the use of Bacillus Calmette–Guérin vaccine and high coverage of multi-drug therapy.¹⁴

Epidemiological situation in 2011

There were 8 notifications of leprosy in 2011, representing a rate of less than 0.1 per 100,000. All cases of leprosy reported in 2011 were reported as non-Indigenous (Figure 81).

Figure 81: Notifications of leprosy, Australia, 1992 to 2011, by year of diagnosis and Indigenous status



Compared with the previous reporting period the number of leprosy cases decreased in 2011 by a third, from the 12 cases reported in 2010. Since 1992 annual notifications of leprosy ranged from 4 to 23 cases.

Geographical distribution

In 2011, cases of leprosy were notified in New South Wales (n=3), Victoria (n=3), South Australia (n=1) and Western Australia (n=1).

Age and sex distribution

In 2011, notified cases of leprosy were predominantly seen in males, with a male to female ratio of 3:1. The median age of cases was 31 years (range 22–65).

Meningococcal disease (invasive)

Meningococcal disease is caused by the bacterium *Neisseria meningitidis* 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.¹⁴ Historically, *N. meningitidis* serogroups B and C have been the major cause of invasive meningococcal disease (IMD) in Australia.

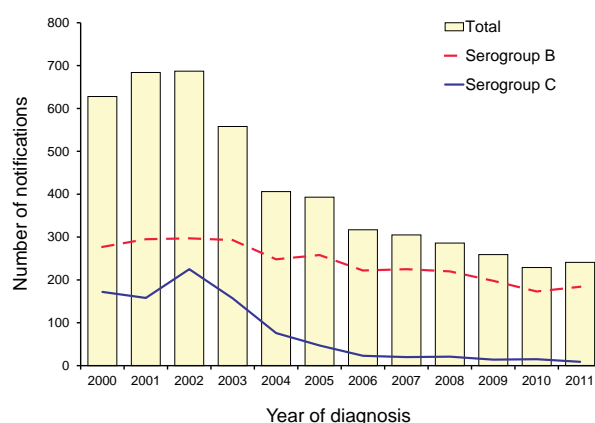
Epidemiological situation in 2011

In 2011, there were 241 notifications of IMD representing a rate of 1.1 per 100,000. While case numbers were 5% higher compared with 2010, they continue the downward trend in overall case numbers and were below the 5-year mean of 279 cases (Figure 82).

Data on serogroup were available for 90% of cases in 2011. Of these, 76% were caused by serogroup B organisms, 5% each by serogroup W135 and serogroup Y and 4% by serogroup C. Notifications of IMD caused by serogroup C organisms have decreased substantially following the introduction of the National Meningococcal C Vaccination Program in 2003, with less than 25 cases reported annually since 2006.

Mortality data were available for 56% of cases reported to the NNDSS in 2011. Of these, 15 were reported as having died from IMD, including 12 due to serogroup B, 2 due to serogroup Y and 1 due to serogroup W135 (Table 19). Mortality data should be interpreted with caution given the large proportion of cases reported without death data to the NNDSS.

Figure 82: Notifications of invasive meningococcal disease, Australia, 2000 to 2011



Geographical distribution

Cases were reported from all states and territories, ranging from 2 cases in the Australian Capital Territory to 72 cases in New South Wales (Table 20). Jurisdictional-specific rates ranged from 0.5 per 100,000 in Australian Capital Territory to 2.0 per 100,000 in Tasmania.

Age and sex distribution

In 2011, sex was evenly distributed with a male female ratio of 1:1; however age specific variations did occur. This was particularly the case in the 0–4 year age group where the male to female ratio was at 1.8:1 and in the 10–14 year age group where it was 4.5:1.

The majority of cases reported (69%) were less than 25 years of age. Of those, the highest proportion (30%) and age specific rate, at 5 per 100,000, were cases less than 5 years of age. High rates also occurred amongst the 15–19 year age group (2.8 per 100,000) followed by the 20–24 year age group (1.6 per 100,000) (Figure 83).

Table 19: Deaths due to invasive meningococcal disease, Australia, 2011, by serogroup and state or territory

Serogroup	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
B	0	4	0	2	2	0	2	2	12
C	0	0	0	0	0	0	0	0	0
W135	0	0	0	0	0	1	0	0	1
Y	0	0	0	1	0	0	1	0	2
Unknown	0	0	0	0	0	0	0	0	0
Total	0	4	0	3	2	1	3	2	15

Table 20: Notifications of invasive meningococcal disease, Australia, 2011, by serogroup and state or territory

Serogroup	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
B	2	44	3	49	18	6	43	19	184
C	0	2	0	3	2	1	1	0	9
W135	0	4	0	2	1	3	2	0	12
Y	0	4	0	3	0	0	3	2	12
Unknown	0	18	1	4	0	0	1	0	24
Total	2	72	4	61	21	10	50	21	241
Rate (per 100,000)	0.5	1.0	1.7	1.3	1.3	2.0	0.9	0.9	1.1

Figure 83: Notification rate for invasive meningococcal disease, Australia, 2011, by age and sex

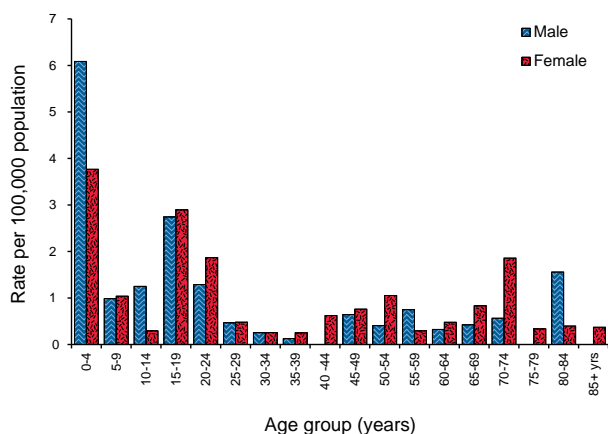
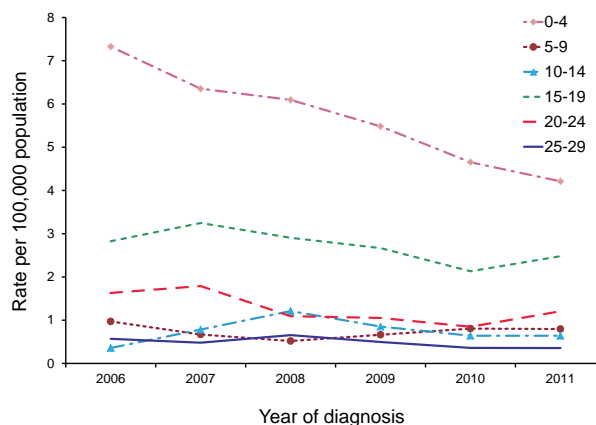


Figure 84: Notification rate for serogroup B invasive meningococcal disease, Australia, 2006 to 2011, by select age group



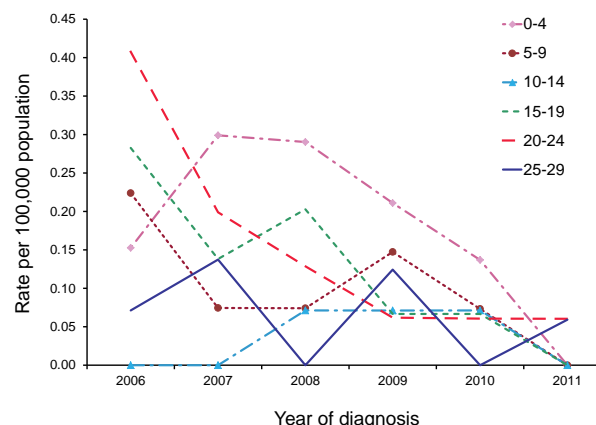
Serogroup B accounted for the majority of cases across all age groups including those less than 25 years of age, where it accounted for 84% of cases. While rates of serogroup B infection remain high compared with the other serogroups, they continue to trend downwards. This was noted in 2011 for the 0–4 year age group where the rate of 4.0 per 100,000 represents a 43% decline from 2006 when the rate was 7.0 per 100,000 (Figure 84).

There were no reported cases of IMD due to serogroup C amongst children and adolescents less than 20 years of age in 2011 and rates continue to be low, at less than 0.2 per 100,000 across all age groups (Figure 85).

Seasonality

In 2011, diagnoses of IMD were highest across the winter months. This was consistent with the normal seasonal pattern of this disease (Figure 86).

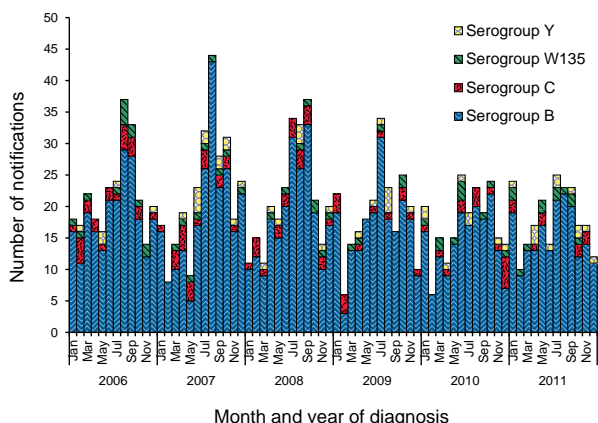
Figure 85: Notification rate for serogroup C invasive meningococcal disease, Australia, 2006 to 2011, by select age group



Vaccination status

Of the 9 cases of IMD due to serogroup C only 1 case was less than 24 years of age (and therefore eligible for vaccination) and this case was reported as not vaccinated.

Figure 86: Notifications of invasive meningococcal disease, Australia, 2006 to 2011, by serogroup and month and year of diagnosis



Laboratory based meningococcal disease surveillance

The Australian Meningococcal Surveillance Programme (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 with the latest report published for 2012.¹²⁸

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 overseas-born and Indigenous communities.

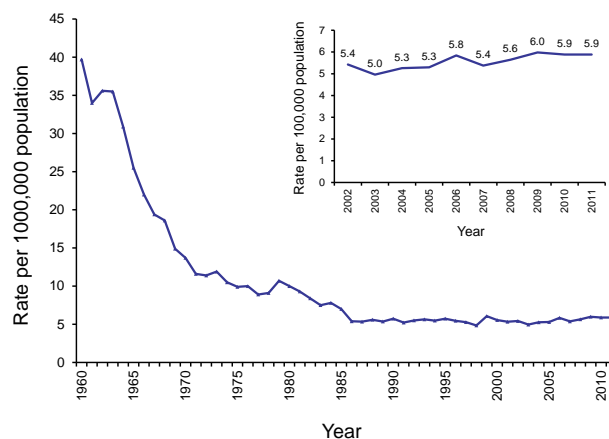
Epidemiological situation in 2011

In 2011, there were 1,331 notifications of TB, a small increase (1%) on the number of cases reported in the previous year (n=1,312). While the substantial decline in the rate of TB since the 1960s has been maintained, notifications in the last decade tend to have increased (Figure 87).

At the time the annual report snap shot was agreed, the New South Wales TB data was affected by a data quality issue, resulting in an undercount of the number of TB cases in New South Wales and

consequently nationally. The issue was identified and subsequently resolved in the NNDSS. For the revised case totals please refer to the NNDSS data on the [Department of Health's web site](http://www.health.gov.au/nndssdata) (www.health.gov.au/nndssdata). The analysis presented in this report relates to data agreed in the creation of the snap shot.

Figure 87: Notification rate for tuberculosis, Australia, 1960 to 2011



Geographical distribution

New South Wales (n=470), Victoria (n=371) and Queensland (n=223) accounted for 80% of all cases of TB diagnosed in Australia. Notification rates were highest in the Northern Territory (14.3 per 100,000), Victoria (6.6 per 100,000) and New South Wales (6.4 per 100,000). Rates in the remaining jurisdictions were all lower than the national notification rate of 5.9 per 100,000.

Age and sex distribution

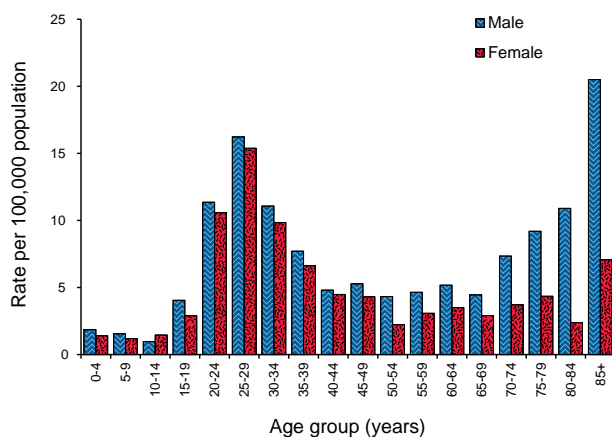
In 2011, TB was predominantly seen in young adults and older males. Males accounted for more than half (56%) of the notifications of TB, resulting in a male to female ratio of 1.3:1. Overall, the age group with the highest notification rate was the 25–29 year age group (15.8 per 100,000). The highest age and sex specific rates were observed in men aged 85 years or over (20.5 per 100,000) and in women aged 25–29 years (15.4 per 100,000) (Figure 88).

Enhanced surveillance

Enhanced data is collected on all cases of TB. These data were not finalised at the time core notification data were finalised for this report. Further analyses, including identification of risk

groups and reporting on treatment outcomes, can be found in the TB annual report series, which is published in CDI.

Figure 88: Notification rate for tuberculosis, Australia, 2011, by age group and sex



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 Communicable Disease Control, South Australian Department of Health, South Australia
 Communicable Diseases Prevention Unit, Department of Health and Human Services, Tasmania
 Health Protection Branch, Department of Health, Victoria
 Communicable Diseases Control Directorate, Department of Health, Western Australia

Abbreviations

7vPCV	7 valent pneumococcal conjugate vaccine
13vPCV	13 valent pneumococcal conjugate vaccine
23vPPV	23 valent pneumococcal polysaccharide vaccine
ABLV	Australian bat lyssavirus
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	<i>Haemophilus influenzae</i> type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HRIG	human rabies immunoglobulin
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
NAI	Neuraminidase inhibition
NAMAC	National Arbovirus and Malaria Advisory Committee
NDP	no data provided
NEC	not elsewhere classified
NIP	National Immunisation Program
NN	not notifiable
NNDSS	National Notifiable Diseases Surveillance System
NQFMP	National Q Fever Management Program
NSC	National Surveillance Committee
PCR	polymerase chain reaction
RRV	Ross River virus
RVC	Regional Verification Commission
SARS	severe acute respiratory syndrome
STEC	Shiga toxin-producing <i>Escherichia coli</i>
STI(s)	sexually transmissible infections(s)
TB	tuberculosis
TSI	Torres Strait Islander
VPD(s)	vaccine preventable disease(s)
VTEC	verotoxigenic <i>Escherichia coli</i>
VZV	varicella zoster virus
WHO	World Health Organization
WHOCC	World Health Organization Collaborating Center
YFV	yellow fever virus

Appendices

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

	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic.	WA	
Males	182,006	3,618,616	119,243	2,288,870	818,621	251,841	2,785,729	1,193,053	11,259,345
Females	183,615	3,683,558	111,126	2,291,412	837,678	258,678	2,835,481	1,156,272	11,358,949
Total	365,621	7,302,174	230,369	4,580,282	1,656,299	510,519	5,621,210	2,349,325	22,618,294

Source: Australian Bureau of Statistics 3101.0 Table 4. Estimated Resident Population, States and Territories. June 2011 population.⁶

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

Age group	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic.	WA	
0-4	24,753	462,145	18,596	316,938	99,846	33,654	358,023	158,329	1,472,401
5-9	21,588	445,890	17,342	293,467	94,291	31,242	333,625	145,446	1,383,048
10-14	21,056	450,244	16,811	299,077	99,255	32,798	335,769	149,972	1,405,184
15-19	24,028	475,159	16,615	314,120	107,459	34,580	362,670	157,551	1,492,373
20-24	31,424	524,682	18,529	334,507	116,656	32,218	424,247	176,077	1,658,472
25-29	32,519	542,391	21,012	338,319	113,523	29,422	431,998	181,711	1,691,066
30-34	29,103	511,983	18,986	309,727	103,279	28,108	401,641	164,792	1,567,777
35-39	27,155	514,410	18,282	323,148	107,375	31,719	401,750	166,709	1,590,705
40-44	25,906	502,205	17,238	323,844	115,013	34,619	403,868	170,951	1,593,846
45-49	25,078	502,570	16,056	316,883	116,426	35,855	386,759	166,010	1,565,859
50-54	24,008	484,798	14,842	300,064	114,784	37,091	367,255	155,889	1,498,929
55-59	21,103	435,632	12,546	269,824	105,590	34,843	330,426	140,104	1,350,263
60-64	18,858	403,654	9,784	251,795	99,883	33,078	304,100	125,789	1,247,102
65-69	12,945	314,996	5,975	192,330	76,706	25,733	233,011	91,071	952,868
70-74	9,233	244,764	3,645	141,270	59,995	19,526	183,133	69,327	730,961
75-79	6,689	190,584	1,917	102,982	47,754	14,654	143,602	51,640	559,851
80-84	5,069	153,094	1,192	79,058	40,124	11,217	115,327	39,330	444,422
85+	4,906	144,489	804	73,372	39,042	10,203	106,886	35,712	415,427
Total	365,421	7,303,690	230,172	4,580,725	1,657,001	510,560	5,624,090	2,346,410	22,620,554

Source: Australian Bureau of Statistics 3201.0 Australian Demographic Statistics Tables. Jun 2011 population.⁶

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2011, 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
Arbovirus infection (NEC)	0	0	0	20	4	0	24	86.5	20	4
Barmah Forest virus infection	37	8	1	623	810	391	1,870	62.4	669	1,201
Botulism	0	0	0	1	0	1	2	50.0	1	1
Brucellosis	0	0	0	23	16	0	39	86.7	23	16
Campylobacteriosis	200	24	16	9,070	8,148	259	17,717	52.3	9,310	8,407
Chlamydial infection	5,938	767	339	33,955	20,882	18,919	80,800	58.7	40,999	39,801
Cholera	0	0	0	2	4	0	6	60.0	2	4
Cryptosporidiosis	208	3	4	992	466	135	1,808	73.9	1,207	601
Dengue virus infection	3	1	1	663	123	26	817	83.1	668	149
Diphtheria	0	0	0	1	3	0	4	50.0	1	3
Gonococcal infection	4,045	345	145	3,705	1,688	2,159	12,087	79.1	8,240	3,847
Haemolytic uraemic syndrome	1	0	0	12	0	0	13	100.0	13	0
<i>Haemophilus influenzae</i> type b	2	0	0	11	0	0	13	100.0	13	0
Hepatitis A	1	0	1	133	9	0	144	95.9	135	9
Hepatitis B (newly acquired)	13	1	1	145	27	3	190	91.0	160	30
Hepatitis B (unspecified)	206	27	6	2,066	1,992	2,332	6,629	65.1	2,305	4,324
Hepatitis C (newly acquired)	67	0	0	287	39	7	400	91.2	354	46
Hepatitis C (unspecified)	550	12	23	3,156	3,497	2,623	9,861	55.9	3,741	6,120
Hepatitis D	1	0	0	32	2	8	43	70.6	33	10
Hepatitis E	0	0	0	37	3	0	40	93.2	37	3
Influenza (laboratory confirmed)	1,033	46	31	10,738	10,233	5,068	27,149	46.9	11,848	15,301
Kunjin virus infection	0	0	0	2	0	0	2	100.0	2	0
Legionellosis	6	0	0	307	30	5	348	87.8	313	35
Leprosy	0	0	0	8	0	0	8	100.0	8	0
Leptospirosis	9	1	0	164	42	1	217	95.1	174	43
Listeriosis	2	0	0	59	8	1	70	92.8	61	9
Malaria	1	9	2	334	59	6	411	91.7	346	65
Measles	10	0	0	173	8	2	193	93.6	183	10
Meningococcal disease (invasive)	22	2	0	206	11	0	241	97.5	230	11

Appendix 3 continued: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2011, 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
Mumps	2	0	0	96	31	26	155	72.8	98	57
Murray Valley encephalitis virus infection	4	0	0	12	0	0	16	100.0	16	0
Ornithosis	0	0	0	76	6	3	85	75.1	76	9
Pertussis	673	55	61	18,771	12,515	6,527	38,602	68.4	19,560	19,042
Pneumococcal disease (invasive)	294	6	7	1,309	162	109	1,887	91.6	1,616	271
Q fever	11	0	1	239	75	12	338	89.0	251	87
Ross River virus infection	79	9	2	2,795	1,711	570	5,166	58.7	2,885	2,281
Rubella	1	0	0	44	7	6	58	76.1	45	13
Salmonellosis	360	15	17	5,915	2,976	2,984	12,267	70.8	6,307	5,960
Shigellosis	133	2	2	293	39	25	494	91.3	430	64
STEC, VTEC	2	0	0	85	5	3	95	92.3	87	8
Syphilis - congenital	3	0	0	3	0	0	6	100.0	6	0
Syphilis < 2 years	191	3	2	1,038	54	15	1,303	95.3	1,234	69
Syphilis > 2 years or unspecified duration	134	7	6	817	284	12	1,260	87.6	964	296
Tetanus	0	0	0	3	0	0	3	100.0	3	0
Tuberculosis	32	3	1	1,277	18	0	1,331	98.5	1,313	18
Tularaemia	0	0	0	2	0	0	2	100.0	2	0
Typhoid	0	0	0	127	6	1	134	90.5	127	7
Varicella zoster (chickenpox)†	145	4	0	1,727	206	12	2,094	76.3	1,876	218
Varicella zoster (shingles)†	110	3	1	3,349	504	32	3,999	73.7	3,463	536
Varicella zoster (unspecified)†	97	12	2	1,844	5,558	202	7,715	30.1	1,955	5,760
Yellow fever	0	0	0	1	1	0	2	50.0	1	1
Total	14,626	1,365	672	106,748	72,262	42,485	238,158	80.4	123,411	114,747

* 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

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